

UNIT 7 Immunologic Function

Case Study

UTILIZING A TEAM APPROACH TO CARE FOR THE PATIENT WITH HIV



You are working in a community health center with an interdisciplinary team to provide care for patients with human immune deficiency virus (HIV). The team consists of a physician,

nurse, pharmacist, social worker, mental health counselor, and a registered dietician. The team is discussing the interdisciplinary management plan for a 21-year-old Hispanic male who recently became positive for HIV infection. The goal is to develop strategies for him to treat his HIV infection and prevent progression to acquired immune deficiency syndrome (AIDS). How can you, as the sole nurse on the team, foster open communication, mutual respect, and shared decision-making within the team to achieve quality patient care?

QSEN Competency Focus: **Teamwork and Collaboration**

The complexities inherent in today's health care system challenge nurses to demonstrate integration of specific interdisciplinary core competencies. These competencies are aimed at ensuring the delivery of safe, quality patient care (Institute of Medicine, 2003). The Quality and Safety Education for Nurses project (Cronenwett, Sherwood, Barnsteiner, et al., 2007; QSEN, 2020) provides a framework for the knowledge, skills, and attitudes (KSAs) required for nurses to demonstrate competency in these key areas, which include ***patient-centered care, interdisciplinary teamwork and collaboration, evidence-based practice, quality improvement, safety, and informatics.***

Teamwork and Collaboration Definition: Function effectively within nursing and interprofessional teams, fostering open communication, mutual respect, and shared decision-making to achieve quality patient care.

SELECT PRE-LICENSURE KSAs

APPLICATION AND REFLECTION

Knowledge

Describe scopes of practice and roles of health care team members	Describe the various roles of the team in managing a patient with HIV. How does your role complement the other members of the team? Why does this patient require a variety of team members to facilitate his disease management?
Describe strategies for identifying and managing overlaps in team member roles and accountabilities	
Recognize contributions of other individuals and groups in helping patient/family achieve health goals	

Skills

Demonstrate awareness of own strengths and limitations as a team member	Discuss how the team approaches the management of the laboratory findings for this patient who is newly diagnosed with HIV. Describe how each member can provide education to him regarding the antiretroviral medications and how they can be used to manage the abnormal laboratory results related to this disease process. Describe how the role of the team changes during the various phases of the disease process.
Initiate plan for self-development as a team member	

Attitudes

Value teamwork and the relationships	After the members of the team meet with the patient, what are ways each member can advocate for this patient's needs? How can you ensure all team
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upon which it is based	members are communicating and collaborating together as a team?
Value different styles of communication used by patients, families, and health care providers	

Cronenwett, L., Sherwood, G., Barnsteiner, J., et al. (2007). Quality and safety education for nurses. *Nursing Outlook*, 55(3), 122–131; Institute of Medicine. (2003). *Health professions education: A bridge to quality*. Washington, DC: National Academies Press; QSEN Institute. (2020). *QSEN competencies: Definitions and pre-licensure KSAs; Teamwork and collaboration*. Retrieved on 8/15/2020 at: qsen.org/competencies/pre-licensure-ksas/#teamwork_collaboration

31 Assessment of Immune Function

LEARNING OUTCOMES

On completion of this chapter, the learner will be able to:

1. Describe the body's general immune responses and the stages of the immune response.
2. Differentiate between cellular and humoral immune responses.
3. Specify the effects of select variables on function of the immune system.
4. Use assessment parameters for determining the status of patients' immune function.

NURSING CONCEPTS

Assessment
Cellular Regulation
Immunity
Infection
Nutrition

GLOSSARY

agglutination: clumping effect occurring when an antibody acts as a cross-link between two antigens

antibody: a protein substance developed by the body in response to and interacting with a specific antigen

antigen: substance that induces the production of antibodies

antigenic determinant: the specific area of an antigen that binds with an antibody-combining site and determines the specificity of the antigen–antibody reaction

apoptosis: programmed cell death that results from the digestion of deoxyribonucleic acid by end nucleases

B cells: cells that are important for producing a humoral immune response

cellular immune response: the immune system's third line of defense, involving the attack of pathogens by T cells

complement: series of enzymatic proteins in the serum that, when activated, destroy bacteria and other cells

cytokines: generic term for nonantibody proteins that act as intercellular mediators, as in the generation of immune response

cytotoxic T cells: lymphocytes that lyse cells infected with virus; also play a role in graft rejection

epitope: any component of an antigen molecule that functions as an antigenetic determinant by permitting the attachment of certain antibodies

genetic engineering: emerging technology designed to enable replacement of missing or defective genes

helper T cells: lymphocytes that attack foreign invaders (antigens) directly

humoral immune response: the immune system's second line of defense (*synonym:* antibody response)

immune response: the coordinated response of the components of the immune system to a foreign agent or organism

immune system: the collection of organs, cells, tissues, and molecules that mediate the immune response

immunity: the body's specific protective response to a foreign agent or organism; resistance to disease, specifically infectious diseases

immunopathology: study of diseases resulting in dysfunctions within the immune system

immunoregulation: complex system of checks and balances that regulates or controls immune responses

immunosenescence: the gradual deterioration of the immune system brought on by the aging process

interferons: proteins formed when cells are exposed to viral or foreign agents; capable of activating other components of the immune system

lymphokines: substances released by sensitized lymphocytes when they come in contact with specific antigens

memory cells: cells that are responsible for recognizing antigens from previous exposure and mounting an immune response

natural killer (NK) cells: lymphocytes that defend against microorganisms and malignant cells

null lymphocytes: lymphocytes that destroy antigens already coated with the antibody

opsonization: the coating of antigen–antibody molecules with a sticky substance to facilitate phagocytosis

phagocytic cells: cells that engulf, ingest, and destroy foreign bodies or toxins

phagocytic immune response: the immune system's first line of defense, involving white blood cells that have the ability to ingest foreign particles

stem cells: precursors of all blood cells; reside primarily in the bone marrow

suppressor T cells: lymphocytes that decrease B-cell activity to a level at which the immune system is compatible with life

T cells: cells that are important for producing a cellular immune response

Immunity is the body's specific protective response to a foreign agent or organism. The **immune system** functions as the body's defense mechanism against invasion and allows a rapid response to foreign substances in a specific manner. Genetic and cellular responses result. Any qualitative or quantitative change in the components of the immune system can produce profound effects on the integrity of the human organism. Immune function is affected by a variety of factors, such as central nervous system integrity, general physical and emotional status, medications, dietary patterns, and the stress of illness, trauma, or surgery. Immune memory is a property of the immune system that provides protection against harmful microbial agents despite the timing of re-exposure to the agent. Tolerance is the mechanism by which the immune system is programmed to eliminate foreign substances such as microbes, toxins, and cellular mutations but maintains the ability to accept self-antigens. Some credence is given to the concept of surveillance, in which the immune system is in a perpetual state of vigilance, screening and rejecting any invader that is recognized as foreign to the host. The term **immunopathology** refers to the study of diseases that result from dysfunctions within the immune system. Immune system dysfunctions can occur across the lifespan; many are

genetically based, others are acquired. Disorders of the immune system may stem from excesses or deficiencies of immunocompetent cells, alterations in the function of these cells, immunologic attack on self-antigens, or inappropriate or exaggerated responses to specific antigens ([Table 31-1](#)).

Primary immunodeficiencies and acquired immune disorders affect large numbers of the population. Thus, nurses in many practice settings need to understand how the immune system functions as well as immunopathologic processes. In addition, knowledge about assessment and care of people with immunologic disorders enables nurses to make appropriate management decisions.

TABLE 31-1 Immune System Disorders

Disorder	Description
Autoimmunity	Normal protective immune response paradoxically turns against or attacks the body, leading to tissue damage
Hypersensitivity	Body produces inappropriate or exaggerated responses to specific antigens
Gammopathies	Overproduction of immunoglobulins
Immune deficiencies	
Primary	Deficiency results from improper development of immune cells or tissues; usually congenital or inherited
Secondary	Deficiency results from some interference with an already developed immune system; usually acquired later in life

Anatomic and Physiologic Overview

Accurate assessment of immune function necessitates the nurse having a good working knowledge of the anatomy and physiology of the immune system.

Anatomy of the Immune System

The immune system is composed of an integrated collection of various cell types, each with a designated function in defending against infection and invasion by other organisms. Supporting this system are molecules that are responsible for the interactions, modulations, and regulation of the system. These molecules and cells participate in specific interactions with immunogenic **epitopes** (antigenic determinants) present on foreign materials, initiating a series of actions in a host, including the inflammatory response, the lysis of microbial agents, and the disposal of foreign toxins. The major components of the immune system include central and peripheral organs, tissues, and cells ([Fig. 31-1](#)).

Bone Marrow

The white blood cells (WBCs) involved in immunity are produced in the bone marrow ([Fig. 31-2](#)). Like other blood cells, lymphocytes are generated from **stem cells** (undifferentiated cells). There are two types of lymphocytes—**B cells**, also called B lymphocytes, and **T cells**, also called T lymphocytes ([Fig. 31-3](#)).

Lymphoid Tissues

The spleen, composed of red and white pulp, acts somewhat like a filter. The red pulp is the site where old and injured red blood cells (RBCs) are destroyed. The white pulp contains concentrations of lymphocytes. The lymph nodes, which are connected by lymph channels and capillaries, are distributed throughout the body. They remove foreign material from the lymph system before it enters the bloodstream. The lymph nodes also serve as centers for immune cell proliferation. The remaining lymphoid tissues contain immune cells that defend the body's mucosal surfaces against microorganisms (Klimov, 2019).

Function of the Immune System



The basic function of the immune system is to remove foreign antigens such as viruses and bacteria to maintain homeostasis. There are two general types of immunity: natural (innate) and acquired (adaptive). Natural immunity or nonspecific immunity is present at birth. Acquired or specific immunity develops after birth. Each type of immunity has a distinct role in defending the body against harmful invaders, but the various components are usually interdependent (Klimov, 2019).

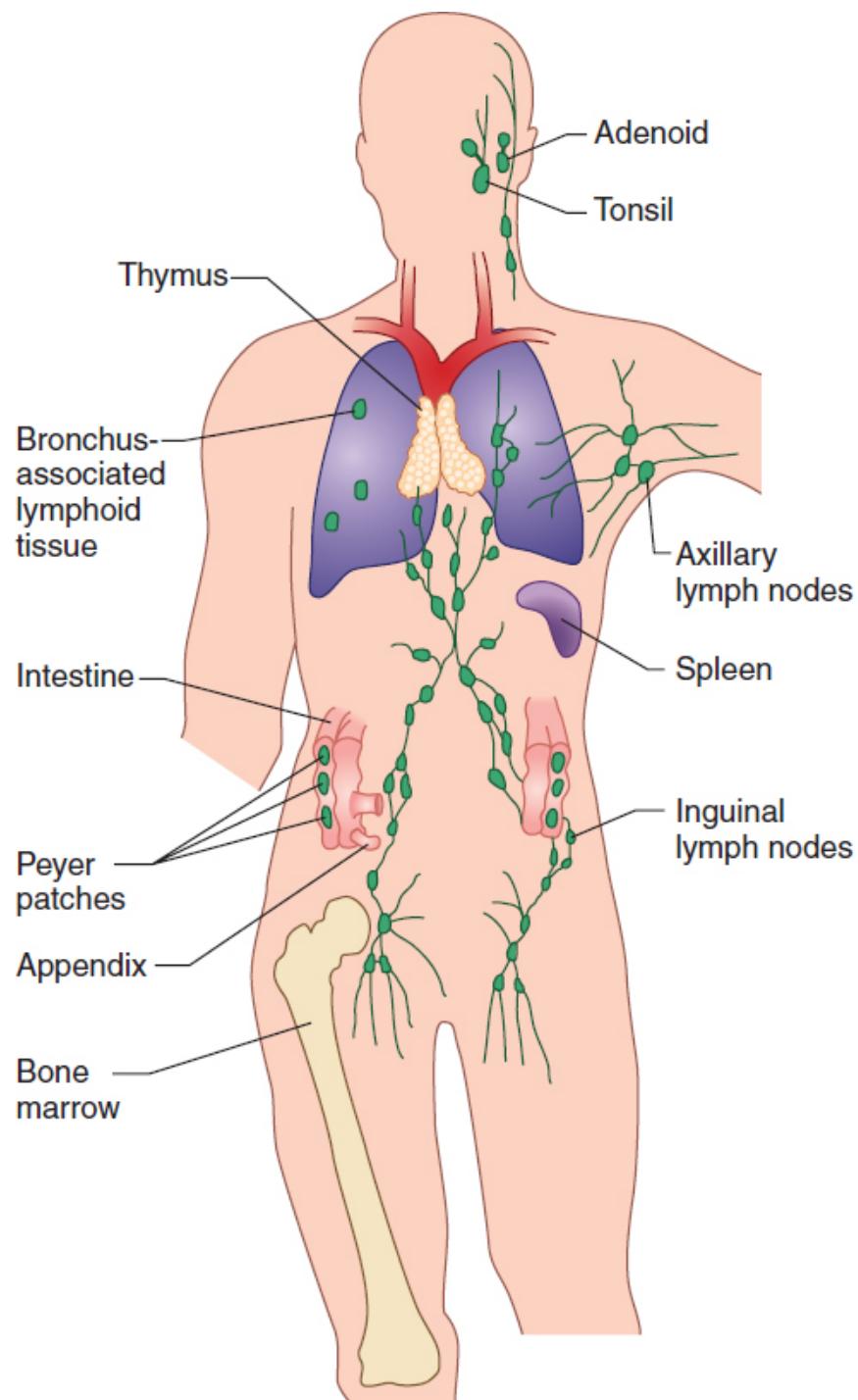


Figure 31-1 • Central and peripheral lymphoid organs, tissues, and cells. Reprinted with permission from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health states* (10th ed., Fig. 11.12, p. 296). Philadelphia, PA: Wolters Kluwer.

Physiology/Pathophysiology

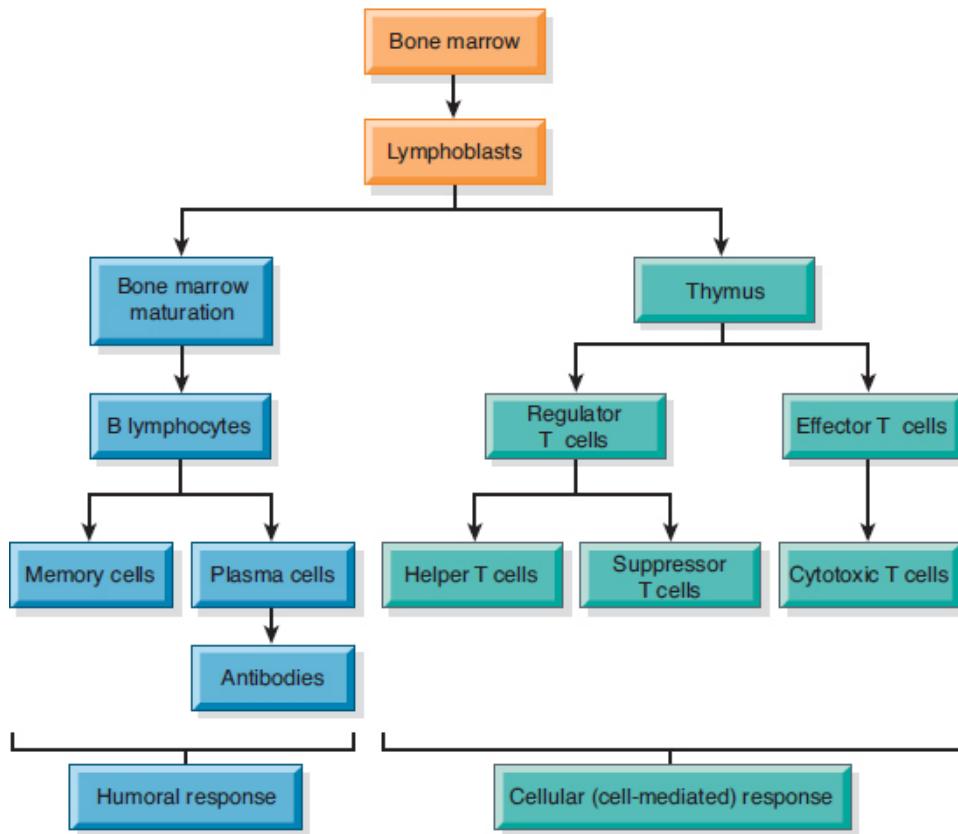


Figure 31-2 • Development of cells of the immune system.

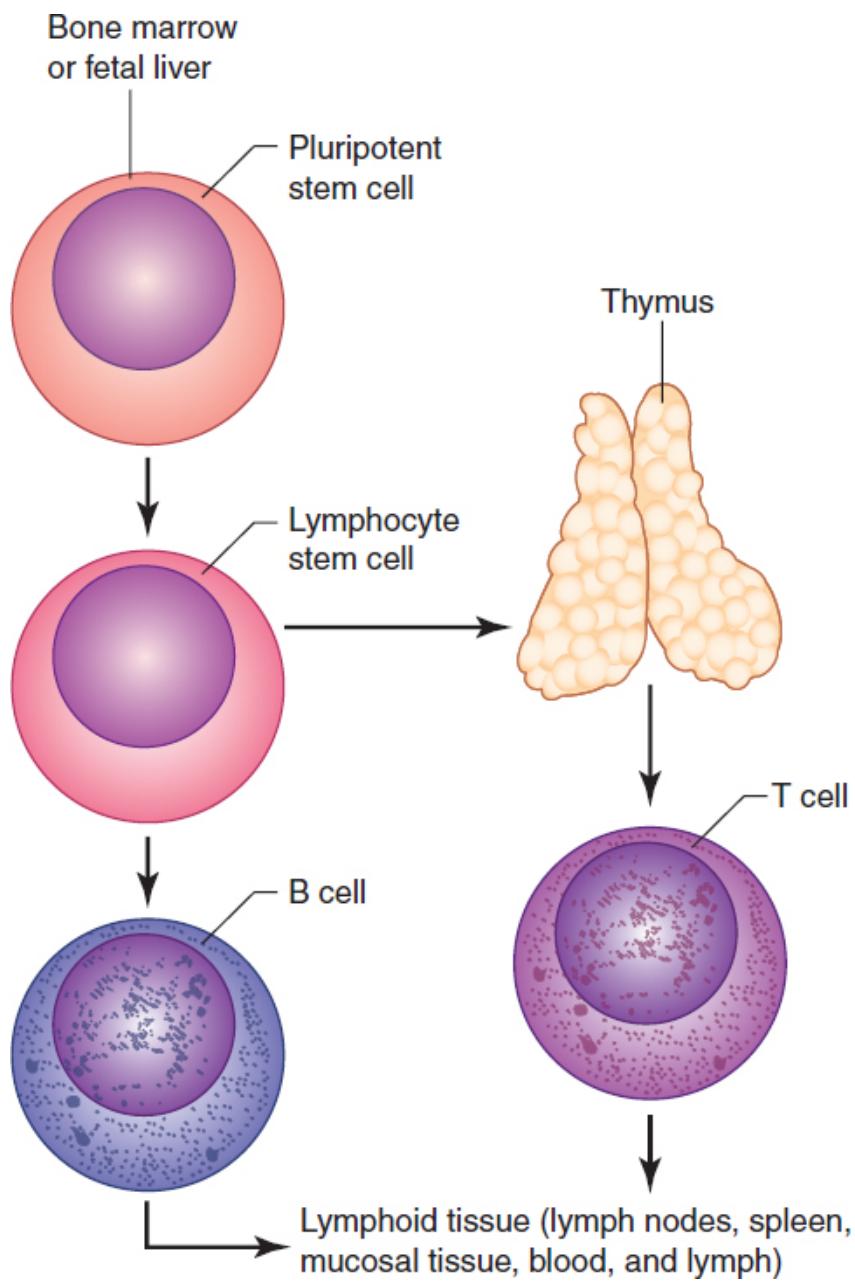


Figure 31-3 • Pathway for T- and B-cell differentiation. Reprinted with permission from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health states* (10th ed., Fig. 11.5, p. 288). Philadelphia, PA: Wolters Kluwer.

Natural Immunity

Natural immunity, which is nonspecific, provides a broad spectrum of defense against and resistance to infection. It is considered the first line of host defense following antigen exposure, because it protects the host without remembering prior contact with an infectious agent (Norris, 2019). Responses to a foreign invader are very similar from one encounter to the next, regardless of the

number of times the invader is encountered. Natural (innate) immunity coordinates the initial response to pathogens through the production of cytokines and other effector molecules, which either activate cells for control of the pathogen (by elimination) or promote the development of the acquired **immune response**. The cells involved in this response are monocytes, macrophages, dendritic cells, **natural killer (NK) cells**, basophils, eosinophils, and granulocytes. The early events in this process are critical in determining the nature of the adaptive immune response. Natural immune mechanisms can be divided into two stages: immediate (generally occurring within minutes) and delayed (occurring within several days after exposure) (Norris, 2019).

White Blood Cell Action

The cellular response is the key to the effective initiation of the immune response. WBCs, or leukocytes, participate in both the natural and the acquired immune responses. Granular leukocytes, or granulocytes (so called because of granules in their cytoplasm), fight invasion by foreign bodies or toxins by releasing cell mediators, such as histamine, bradykinin, and prostaglandins, and by engulfing the foreign bodies or toxins. Granulocytes include neutrophils, eosinophils, and basophils.

Neutrophils (polymorphonuclear leukocytes) are the first cells to arrive at the site where inflammation occurs. Eosinophils and basophils, other types of granulocytes, increase in number during allergic reactions and stress responses. Nongranular leukocytes include monocytes or macrophages (referred to as histiocytes when they enter tissue spaces) and lymphocytes. Monocytes are the first to arrive on the scene and function as **phagocytic cells**, engulfing, ingesting, and destroying greater numbers and quantities of foreign bodies or toxins than granulocytes do. Lymphocytes, consisting of B cells and T cells, play major roles in humoral and cell-mediated immune responses. About 70% to 80% of lymphocytes in the blood are T cells, and about 10% to 15% are B cells (Haynes, Soderberg, & Fauci, 2018).

Inflammatory Response

The inflammatory response is a major function of the natural immune system that is elicited in response to tissue injury or invading organisms. Chemical mediators assist this response by minimizing blood loss, walling off the invading organism, activating phagocytes, and promoting formation of fibrous scar tissue and regeneration of injured tissue. The inflammatory response (discussed further in [Chapter 5](#)) is facilitated by physical and chemical barriers that are part of the human organism.

Physical and Chemical Barriers

Activation of the natural immunity response is enhanced by processes inherent in physical and chemical barriers. Physical surface barriers include intact skin,

mucous membranes, and cilia of the respiratory tract, which prevent pathogens from gaining access to the body. The cilia of the respiratory tract, along with coughing and sneezing responses, filter and clear pathogens from the upper respiratory tract before they can invade the body further. Chemical barriers, such as mucus, acidic gastric secretions, enzymes in tears and saliva, and substances in sebaceous and sweat secretions, act in a nonspecific way to destroy invading bacteria and fungi. Viruses are countered by other means, such as interferon (see discussion later in chapter).

Immune Regulation

Regulation of the immune response involves balance and counterbalance. Dysfunction of the natural immune system can occur when the immune components are inactivated or when they remain active long after their effects are beneficial. A successful immune response eliminates the responsible antigen. If an immune response fails to develop and clear an antigen sufficiently, the host is considered immunocompromised or immunodeficient. If the response is overly robust or misdirected, allergies, asthma, or autoimmune disease results. The immune system's recognition of one's own cells or tissues as "foreign" rather than as self is the basis of many autoimmune disorders (Norris, 2019). While the immune response is critical to the prevention of disease, it must be well controlled to curtail immunopathology. Most microbial infections induce an inflammatory response mediated by T cells and cytokines, which, in excess, can cause tissue damage (Haynes et al., 2018). Therefore, regulatory mechanisms must be in place to suppress or halt the immune response. This is mainly achieved by the production of cytokines and transformation of growth factor that inhibit macrophage activation. In some cases, T-cell activation is so acute that these mechanisms fail, and pathology develops. Ongoing research on **immunoregulation** holds the promise of preventing graft rejection and aiding the body in eliminating cancerous or infected cells (Chae & Bothwell, 2018; Romano, Fanelli, Albany, et al., 2019).

Although natural immunity can often effectively combat infections, many pathogenic microbes have evolved that resist natural immunity. Acquired immunity is necessary to defend against these resistant agents.

Acquired Immunity

Acquired (adaptive) immunity usually develops due to prior exposure to an antigen through immunization (vaccination) or by contracting a disease, both of which generate a protective immune response. Weeks or months after exposure to a disease or vaccine, the body produces an immune response that is sufficient to defend against the disease on re-exposure. In contrast to the rapid but nonspecific natural immune response, this form of immunity relies on the recognition of specific foreign antigens. The acquired immune response

is broadly divided into two mechanisms: (1) the cell-mediated response, involving T-cell activation, and (2) effector mechanisms, involving B-cell maturation and production of antibodies (Haynes et al., 2018).

The two types of acquired immunity are known as active and passive and are interrelated. Active acquired immunity refers to immunologic defenses developed by the person's own body. This immunity typically lasts many years or even a lifetime. Passive acquired immunity is temporary immunity transmitted from a source outside the body that has developed immunity through previous disease or immunization. Examples include immunity resulting from the transfer of antibodies from the mother to an infant in utero or through breast-feeding or receiving injections of immune globulin. Active and passive acquired immunity involve humoral and cellular (cell-mediated) immunologic responses (described later).

Response to Invasion

When the body is invaded or attacked by bacteria, viruses, or other pathogens, it has three means of defense:

- The phagocytic immune response
- The humoral or antibody immune response
- The cellular immune response

The first line of defense, the **phagocytic immune response**, primarily involves the WBCs (granulocytes and macrophages), which ingest foreign particles and destroy the invading agent; eosinophils are only weakly phagocytic. Phagocytes also remove the body's own dying or dead cells. Cells in necrotic tissue that are dying release substances that trigger an inflammatory response. **Apoptosis**, or programmed cell death, is the body's way of destroying worn-out cells such as blood or skin cells or cells that need to be renewed (Norris, 2019).

A second protective response, the **humoral immune response** (*synonym: antibody response*), begins with the B lymphocytes, which can transform themselves into plasma cells that manufacture antibodies. An **antibody** is a protein substance developed by the body, transported in the bloodstream, and attempts to disable invaders. The third mechanism of defense, the **cellular immune response**, involves the T lymphocytes, which can turn into special cytotoxic (or killer) T cells that can attack the pathogens.

The structural part of the invading or attacking organism that is responsible for stimulating antibody production is called an **antigen** (or an immunogen). For example, an antigen can be a small patch of proteins on the outer surface of a microorganism. Not all antigens are naturally immunogenic; some must be coupled to other molecules to stimulate the immune response. A single bacterium or large molecule, such as a diphtheria or tetanus toxin, may have several antigens, or markers, on its surface, thus inducing the body to produce many different antibodies. Once produced, an antibody is released into the

bloodstream and carried to the attacking organism. There, it combines with the antigen, binding with it like an interlocking piece of a jigsaw puzzle ([Fig. 31-4](#)). There are four well-defined stages in an immune response: recognition, proliferation, response, and effector ([Fig. 31-5](#)).

Recognition Stage

Recognition of antigens as foreign, or non-self, by the immune system is the initiating event in any immune response. Recognition involves the use of lymph nodes and lymphocytes for surveillance. Lymph nodes are widely distributed internally throughout the body and in the circulating blood, as well as externally near the body's surfaces. They continuously discharge small lymphocytes into the bloodstream. These lymphocytes patrol the tissues and vessels that drain the areas served by that node. Lymphocytes recirculate from the blood to lymph nodes and from the lymph nodes back into the bloodstream in a continuous circuit. Lymphocytes and other cells have "microbial sensors" that identify molecules on microbes and other microorganisms. The interaction of these sensors with the offending agent sets off a cascade aimed at destroying the microbe. Invading organisms have pathogen-associated molecular patterns (PAMPs) contained in their cell membranes that are recognized by the immune system cells. Once the receptors on the immune cells reach the PAMPs, the immune response is triggered. Macrophages play an important role in helping the circulating lymphocytes process the antigens. Both macrophages and neutrophils have receptors for antibodies and complement; as a result, they coat microorganisms with antibodies, complement, or both, thereby enhancing phagocytosis (Norris, 2019).

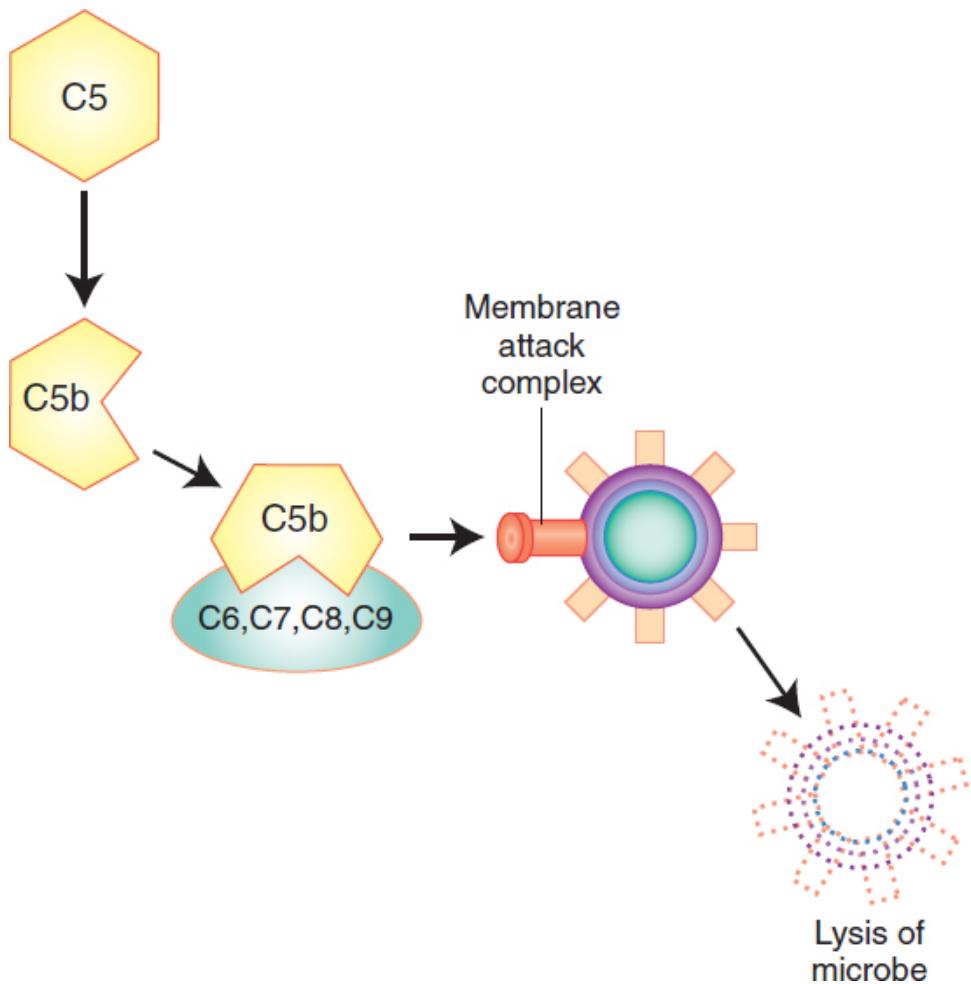


Figure 31-4 • Complement-mediated immune responses.
Reprinted with permission from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health states* (10th ed., second figure in Understanding box, p. 286). Philadelphia, PA: Wolters Kluwer.

In a streptococcal throat infection, for example, the streptococcal organism gains access to the mucous membranes of the throat. A circulating lymphocyte moving through the tissues of the throat comes in contact with the organism. The lymphocyte recognizes the antigens on the microbe as different (non-self) and the streptococcal organism as antigenic (foreign). This triggers the second stage of the immune response—proliferation.

Proliferation Stage

The circulating lymphocytes containing the antigenic message return to the nearest lymph node. Once in the node, these sensitized lymphocytes stimulate some of the resident T and B lymphocytes to enlarge, divide, and proliferate. T lymphocytes differentiate into cytotoxic (or killer) T cells, whereas B

lymphocytes produce and release antibodies. Enlargement of the lymph nodes in the neck in conjunction with a sore throat is one example of the immune response.

Response Stage

In the response stage, the differentiated lymphocytes function in either a humoral or a cellular capacity. This stage begins with the production of antibodies by the B lymphocytes in response to a specific antigen. The cellular response stimulates the resident lymphocytes to become cells that attack microbes directly rather than through the action of antibodies. These transformed lymphocytes are known as cytotoxic (killer) T cells.

Viral antigens induce a cellular response. This response is manifested by the increasing number of T lymphocytes (lymphocytosis) seen in the blood tests of people with viral illnesses such as infectious mononucleosis. (Cellular immunity is discussed later in this chapter.) Most immune responses to antigens involve both humoral and cellular responses, although one usually predominates. For example, during transplant rejection, the cellular response involving T cells predominates, whereas in the bacterial pneumonias and sepsis, the humoral response involving B cells plays the dominant protective role ([Chart 31-1](#)).

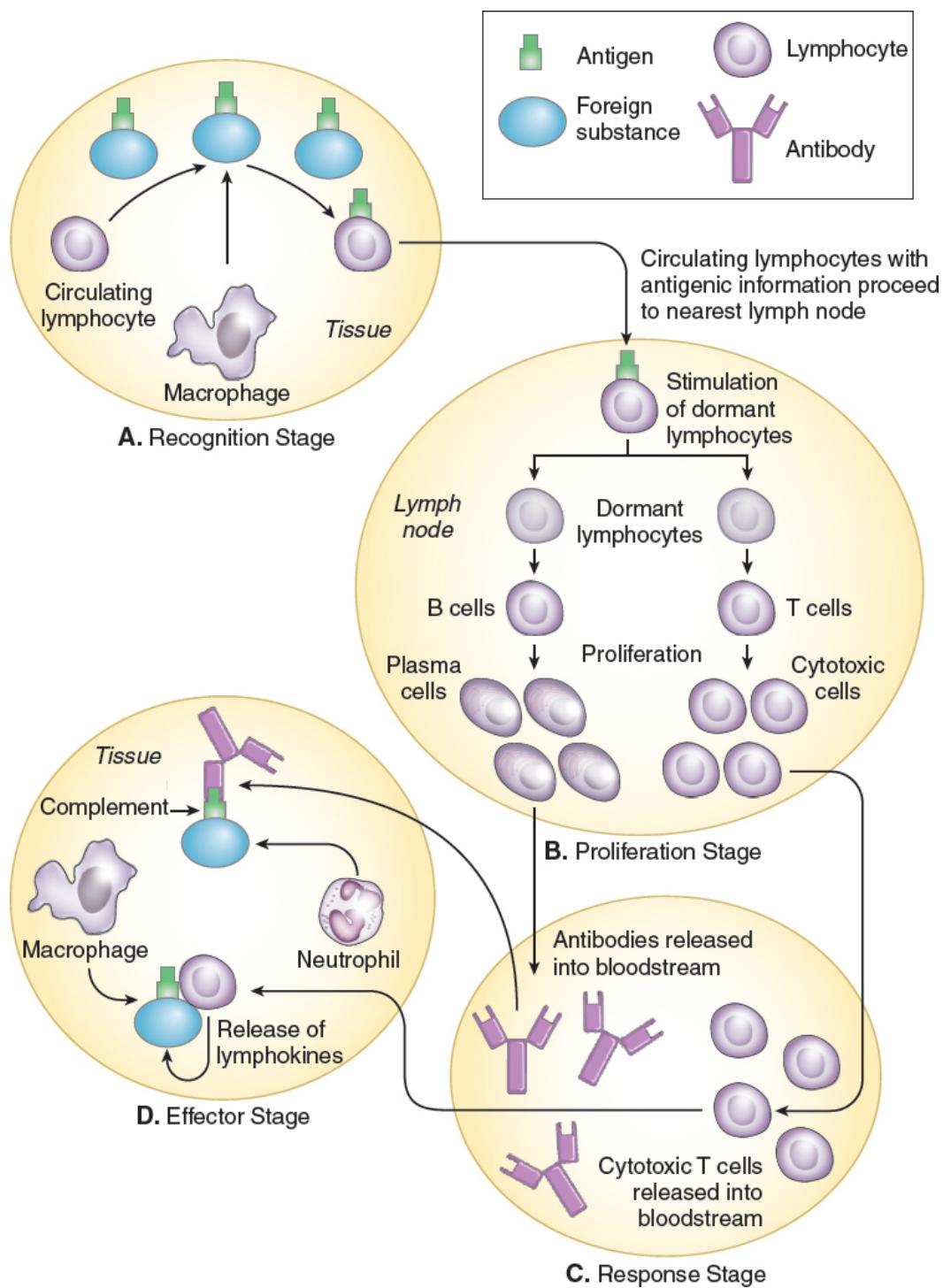


Figure 31-5 • Stages of the immune response. **A.** In the *recognition stage*, antigens are recognized by circulating lymphocytes and macrophages. **B.** In the *proliferation stage*, the dormant lymphocytes proliferate and differentiate into cytotoxic (killer) T cells or B cells responsible for formation and release of antibodies. **C.** In the *response stage*, the cytotoxic T cells and the B cells perform cellular and humoral functions, respectively. **D.** In the

effector stage, antigens are destroyed or neutralized through the action of antibodies, complement, macrophages, and cytotoxic T cells.

Chart 31-1

Comparison of Humoral and Cellular Immune Responses

Humoral Responses (B Cells)

- Bacterial phagocytosis and lysis
- Anaphylaxis
- Allergic hay fever and asthma
- Immune complex disease
- Bacterial and some viral infections

Cellular Responses (T Cells)

- Transplant rejection
- Delayed hypersensitivity (tuberculin reaction)
- Graft-versus-host disease
- Tumor surveillance or destruction
- Intracellular infections
- Viral, fungal, and parasitic infections

Effector Stage

In the effector stage, either the antibody of the humoral response or the cytotoxic (killer) T cell of the cellular response reaches and connects with the antigen on the surface of the foreign invader. This action initiates activities involving an interplay of antibodies (humoral immunity), complement, and action by the cytotoxic T cells (cellular immunity).

Humoral Immune Response

The humoral response is characterized by the production of antibodies by B lymphocytes in response to a specific antigen. Following antibody production, the macrophages of natural immunity and the special T lymphocytes of cellular immunity are involved in antigen recognition.

Antigen Recognition

Several theories explain the mechanisms by which B lymphocytes recognize the invading antigen and respond by producing antibodies. It is known that B lymphocytes recognize and respond to invading antigens in more than one way.

The B lymphocytes respond to some antigens by directly triggering antibody formation; however, in response to other antigens, they need the assistance of T cells to trigger antibody formation. With the help of macrophages, the T lymphocytes are believed to recognize the antigen of a foreign invader. The T lymphocyte picks up the antigenic message, or “blueprint,” of the antigen and returns to the nearest lymph node with that message. B lymphocytes stored in the lymph nodes are subdivided into thousands of clones, which are stimulated to enlarge, divide, proliferate, and differentiate into plasma cells capable of producing specific antibodies to the antigen. Other B lymphocytes differentiate into B-lymphocyte clones with a memory for the antigen. These memory cells are responsible for the more exaggerated and rapid immune response in a person who is repeatedly exposed to the same antigen.

Role of Antibodies

Antibodies are large proteins, called *immunoglobulins*, that consist of two subunits, each containing a light and a heavy peptide chain held together by a chemical link composed of disulfide bonds. Each subunit has one portion that serves as a binding site for a specific antigen and another portion that allows the antibody molecule to take part in the complement system.

Antibodies defend against foreign invaders in several ways, and the type of defense used depends on the structure and composition of both the antigen and the immunoglobulin. The antibody molecule has at least two combining sites, or Fab fragments ([Fig. 31-6](#)). One antibody can act as a cross-link between two antigens, causing them to bind or clump together. This clumping effect, referred to as **agglutination**, helps clear the body of the invading organism by facilitating phagocytosis. Some antibodies assist in the removal of offending organisms through **opsonization**. In this process, the antigen–antibody molecule is coated with a sticky substance that also facilitates phagocytosis.

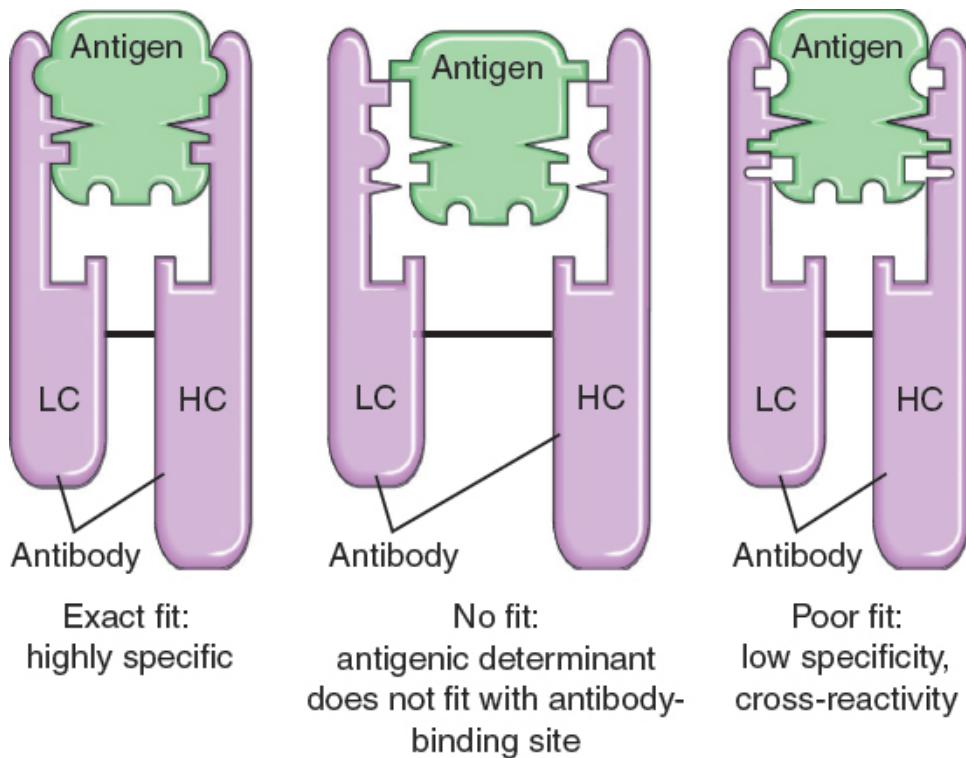


Figure 31-6 • Antigen–antibody binding. (**Left**) A highly specific antigen–antibody complex. (**Center**) No match and, therefore, no immune response. (**Right**) Poor fit or match with low specificity; antibody reacts to antigen with similar characteristics, producing cross-reactivity. HC, heavy chain; LC, light chain.

Chart 31-2

Major Characteristics of the Immunoglobulins

IgG (75% of Total Immunoglobulin)

- Appears in serum and tissues (interstitial fluid)
- Assumes a major role in bloodborne and tissue infections
- Activates the complement system
- Enhances phagocytosis
- Crosses the placenta

IgA (15% of Total Immunoglobulin)

- Appears in body fluids (blood, saliva, tears, and breast milk, as well as pulmonary, gastrointestinal, prostatic, and vaginal secretions)
- Protects against respiratory, gastrointestinal, and genitourinary infections
- Prevents absorption of antigens from food
- Passes to neonate in breast milk for protection

IgM (10% of Total Immunoglobulin)

- Appears mostly in intravascular serum
- Appears as the first immunoglobulin produced in response to bacterial and viral infections
- Activates the complement system

IgD (0.2% of Total Immunoglobulin)

- Appears in small amounts in serum
- Possibly influences B-lymphocyte differentiation, but role is unclear

IgE (0.004% of Total Immunoglobulin)

- Appears in serum
- Takes part in allergic and some hypersensitivity reactions
- Combats parasitic infections

Ig, immunoglobulin.

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health states* (10th ed.). Philadelphia, PA: Wolters Kluwer.

Antibodies also promote the release of vasoactive substances, such as histamine and slow-reacting substances, two of the chemical mediators of the inflammatory response. Antibodies do not function in isolation; rather, they mobilize other components of the immune system to defend against the invader.

The body can produce five different types of immunoglobulin (Ig). Each of the five types, or classes, is identified by a specific letter of the alphabet, IgA,

IgD, IgE, IgG, and IgM. Classification is based on the chemical structure and biologic role of the individual immunoglobulin. Major characteristics of the immunoglobulins are summarized in [Chart 31-2](#). The normal laboratory values for the three major Igs (IgA, IgG, and IgM) can be found in Appendix A on [thePoint](#) at thepoint.lww.com.

Antigen–Antibody Binding

The portion of the antigen involved in binding with the antibody is referred to as the **antigenic determinant**. The most efficient immunologic responses occur when the antibody and antigen fit like a lock and key. Poor fit can occur with an antibody that was produced in response to a different antigen. This phenomenon is known as cross-reactivity. For example, in acute rheumatic fever, the antibody produced against *Streptococcus pyogenes* in the upper respiratory tract may cross-react with the patient's heart tissue, leading to heart valve damage.

Cellular Immune Response

The T lymphocytes are primarily responsible for cellular immunity. Stem cells continuously migrate from the bone marrow to the thymus gland, where they develop into T cells. Despite the partial degeneration of the gland at puberty, T cells continue to develop in the thymus gland. Several types of T cells exist, each with designated roles in the defense against bacteria, viruses, fungi, parasites, and malignant cells. T cells attack foreign invaders directly rather than by producing antibodies.

Cellular reactions are initiated, with or without the assistance of macrophages, by the binding of an antigen to an antigen receptor located on the surface of a T cell. The T cells then carry the antigenic message, or blueprint, to the lymph nodes, where the production of other T cells is stimulated. Some T cells remain in the lymph nodes and retain a memory for the antigen. Other T cells migrate from the lymph nodes into the general circulatory system and ultimately to the tissues, where they remain until they bind with their respective antigens or die (Norris, 2019).

Types of T Lymphocytes

T cells include effector T cells, suppressor T cells, and memory T cells. The two major categories of effector T cells—helper T cells (also referred to as CD4⁺ cells) and cytotoxic T cells (also referred to as CD8⁺ cells)—participate in the destruction of foreign organisms. T cells interact closely with B cells, indicating that humoral and cellular immune responses are not separate, unrelated processes but rather are branches of the immune response that interact.

Helper T cells are activated on recognition of antigens and stimulate the rest of the immune system. When activated, helper T cells secrete **cytokines**, which attract and activate B cells, cytotoxic T cells, NK cells, macrophages, and other cells of the immune system. Cytokines are proteins produced by the cells of the immune system that determine the actions of the immune system cells. Separate subpopulations of helper T cells produce different types of cytokines and determine whether the immune response will be the production of antibodies or a cell-mediated immune response. Helper T cells also produce **lymphokines**, one category of cytokines ([Table 31-2](#)).

Cytotoxic T cells (killer T cells) attack the antigen directly by altering the cell membrane, causing cell lysis (disintegration), and releasing cytolytic enzymes and cytokines. Lymphokines can recruit, activate, and regulate other lymphocytes and WBCs. These cells then assist in destroying the invading organism. Delayed-type hypersensitivity is an example of an immune reaction that protects the body from antigens through the production and release of lymphokines (see later discussion).

Suppressor T cells have the ability to decrease B-cell production, thereby keeping the immune response at a level that is compatible with health (e.g., sufficient to fight infection adequately without attacking the body's healthy tissues). **Memory cells** are responsible for recognizing antigens from previous exposure and mounting an immune response ([Table 31-3](#)).

Null Lymphocytes and Natural Killer Cells

Null lymphocytes and NK cells are other lymphocytes that assist in combating organisms. These cells are distinct from B cells and T cells and lack the usual characteristics of those cells. **Null lymphocytes**, a subpopulation of lymphocytes, destroy antigens already coated with antibody. These cells have special receptor sites on their surface that allow them to connect with the end of antibodies; this is known as antibody-dependent, cell-mediated cytotoxicity.

NK cells are a class of lymphocytes that recognize infected and stressed cells and respond by killing these cells and by secreting macrophage-activating cytokine. The helper T cells contribute to the differentiation of null and NK cells.

Complement System

Circulating plasma proteins, known as **complement**, are made in the liver and activated when an antibody connects with its antigen. Complement plays an important role in the defense against microbes. Destruction of an invading or attacking organism or toxin is not achieved merely by the binding of the antibody and antigens; it also requires activation of complement, the arrival of killer T cells, or the attraction of macrophages. Complement has three major physiologic functions: defending the body against bacterial infection, bridging

natural and acquired immunity, and disposing of immune complexes and the by-products associated with inflammation (Klimov, 2019).

The proteins that comprise complement interact sequentially with one another in a cascading effect. The complement cascade is important to modifying the effector arm of the immune system. Activation of complement allows important events, such as removal of infectious agents and initiation of the inflammatory response, to take place. These events involve active parts of the pathway that enhance chemotaxis of macrophages and granulocytes, alter blood vessel permeability, change blood vessel diameters, cause cells to lyse, alter blood clotting, and cause other points of modification. These macrophages and granulocytes continue the body's defense by devouring the antibody-coated microbes and by releasing bacterial products.

The complement cascade may be activated by any of three pathways: classic, lectin, and alternative. The classic pathway is triggered after antibodies bind to microbes or other antigens and is part of the humoral type of adaptive immunity. The lectin pathway is activated when a plasma protein (mannose-binding lectin) binds to terminal mannose residue on the surface glycoproteins of microbes. The alternative pathway is triggered when complement proteins are activated on microbial surfaces. This pathway is part of natural immunity.

Complement components, prostaglandins, leukotrienes, and other inflammatory mediators all contribute to the recruitment of inflammatory cells, as do chemokines, a group of cytokines. The activated neutrophils pass through the vessel walls to accumulate at the site of infection, where they phagocytize complement-coated microbes. This response is usually therapeutic and can be lifesaving if the cell attacked by the complement system is a true foreign invader. However, if that cell is part of the human organism, the result can be devastating disease and even death. Many autoimmune diseases and disorders characterized by chronic infection are thought to be caused in part by continued or chronic activation of complement, which in turn results in chronic inflammation. The RBCs and platelets have complement receptors and, as a result, play an important role in the clearance of immune complexes that consist of antigen, antibody, and components of the complement system (Norris, 2019).

TABLE 31-2 Cytokines of Innate and Adaptive Immunity

Cytokines	Source	Biologic Activity
Interleukin-1 (IL-1)	Macrophages, endothelial cells, some epithelial cells	Wide variety of biologic effects; activates endothelium in inflammation; induces fever and acute-phase response; stimulates neutrophil production
Interleukin-2 (IL-2)	CD4 ⁺ , CD8 ⁺ T cells	Growth factor for activated T cells; induces synthesis of other cytokines; activates cytotoxic T lymphocytes and NK cells
Interleukin-3 (IL-3)	CD4 ⁺ T cells	Growth factor for progenitor hematopoietic cells
Interleukin-4 (IL-4)	CD4 ⁺ T _H 2 cells, mast cells	Promotes growth and survival of T, B, and mast cells; causes T _H 2 cell differentiation; activates B cells and eosinophils and induces IgE-type responses
Interleukin-5 (IL-5)	CD4 ⁺ T _H 2 cells	Induces eosinophil growth and development
Interleukin-6 (IL-6)	Macrophages, endothelial cells, T lymphocytes	Stimulates the liver to produce mediators of acute-phase inflammatory response; also induces proliferation of antibody-producing cells by the adaptive immune system
Interleukin-7 (IL-7)	Bone marrow stromal cells	Primary function in adaptive immunity; stimulates pre-B cells and thymocyte development and proliferation
Interleukin-8 (IL-8)	Macrophages, endothelial cells	Primary function in adaptive immunity; chemoattracts neutrophils and T lymphocytes; regulates lymphocyte homing and neutrophil infiltration
Interleukin-10 (IL-10)	Macrophages, some T-helper cells	Inhibitor of activated macrophages and dendritic cells; decreases inflammation by inhibiting T _H 1 cells and release of interleukin-12 from macrophages
Interleukin-12 (IL-12)	Macrophages, dendritic cells	Enhances NK cell cytotoxicity in innate immunity; induces T _H 1 cell differentiation in adaptive immunity
Type I interferons (IFN- α , IFN- β)	Macrophages, fibroblasts	Inhibit viral replication, activate NK cells, and increase expression of MHC-I molecules on virus-infected cells
Interferon- γ (IFN- γ)	NK cells, CD4 ⁺ and CD8 ⁺ T lymphocytes	Activates macrophages in both innate immune responses and adaptive cell-mediated immune responses; increases expression of MHC I and II and antigen processing and presentation
Tumor necrosis factor α (TNF- α)	Macrophages, T cells	Induces inflammation, fever, and acute-phase response; activates neutrophils and endothelial cells; kills cells through apoptosis
Chemokines	Macrophages,	Large family of structurally similar cytokines that

	endothelial cells, T lymphocytes	stimulate leukocyte movement and regulate the migration of leukocytes from the blood to the tissues
Granulocyte–monocyte CSF (GM-CSF)	T cells, macrophages, endothelial cells, fibroblasts	Promotes neutrophil, eosinophil, and monocyte maturation and growth; activates mature granulocytes
Granulocyte CSF (G-CSF)	Macrophages, fibroblasts, endothelial cells	Promotes growth and maturation of neutrophils consumed in inflammatory reactions
Monocyte CSF (M-CSF)	Macrophages, activated T cells, endothelial cells	Promotes growth and maturation of mononuclear phagocytes

CSF, colony-stimulating factor; IgE, immunoglobulin E; MHC, major histocompatibility complex; NK, natural killer; T_H1 , T-helper type 1; T_H2 , T-helper type 2.

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health states* (10th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Immunomodulators

Antimicrobial agents and vaccines have yielded considerable therapeutic success and the immune system usually works effectively; however, many infectious diseases remain difficult clinical challenges. Treatment success may be compromised by defects of the immune system; in this case, enhancement of the host immune response may be therapeutically beneficial. An immunomodulator (also known as a biologic response modifier) affects the host via direct or indirect effects on one or more components of the immunoregulatory network. Interferons, colony-stimulating factors, and monoclonal antibodies (MoAbs) are examples of agents used to help enhance the immune system (Davis & Ballas, 2017).

TABLE 31-3 Lymphocytes Involved in Immune Responses

Type of Immune Response	Cell Type	Function
Humoral	B lymphocyte	Produces antibodies or immunoglobulins (IgA, IgD, IgE, IgG, IgM)
Cellular	T lymphocyte	
	Helper T	Attacks foreign invaders (antigens) directly Initiates and augments inflammatory response
	Helper T ₁	Increases activated cytotoxic T cells
	Helper T ₂	Increases B-cell (BG8) antibody production
	Suppressor T	Suppresses the immune response
	Memory T	Remembers contact with an antigen and on subsequent exposures mounts an immune response
	Cytotoxic T (killer T)	Lyses cells infected with virus; plays a role in graft rejection
Nonspecific	Non-T or non-B-lymphocyte null cell	Destroys antigens already coated with antibody
	Natural killer cell (granular lymphocyte)	Defends against microorganisms and some types of malignant cells; produces cytokines

Interferons

Interferon, one type of biologic response modifier, is a nonspecific viricidal protein that is naturally produced by the body and capable of activating other components of the immune system. Interferons continue to be investigated to determine their roles in the immune system and their potential therapeutic effects in disorders characterized by disturbed immune responses. These substances have antiviral and antitumor properties. In addition to responding to viral infection, interferons are produced by T lymphocytes, B lymphocytes, and macrophages in response to antigens. They are thought to modify the immune response by suppressing antibody production and cellular immunity. They also facilitate the cytolytic role of macrophages and NK cells. Interferons are used to treat immune-related disorders (e.g., multiple sclerosis) and chronic inflammatory conditions (e.g., chronic hepatitis). Research continues to evaluate the effectiveness of interferons in treating cancers (Makowska, Braunschweig, Denecke, et al., 2019) and acquired immunodeficiency syndrome.

Colony-Stimulating Factors

Colony-stimulating factors are a group of naturally occurring glycoprotein cytokines that regulate production, differentiation, survival, and activation of hematopoietic cells. Erythropoietin stimulates RBC production. Thrombopoietin plays a key regulatory role in the growth and differentiation of bone marrow cells. Interleukin-5 (IL-5) stimulates the growth and survival of eosinophils and basophils. Stem cell factor and IL-3 serve as stimuli for multiple hematopoietic cell lines. Granulocyte colony-stimulating factor, granulocyte-macrophage colony-stimulating factor, and macrophage colony-stimulating factor all serve as growth factors for specific cell lines. These cytokines have attracted considerable interest for their potential role in immunomodulation (Leleu, Gay, Flament, et al., 2017).

Monoclonal Antibodies

MoAbs have become available through technologic advances, enabling investigators to grow and produce targeted antibodies for specific pathologic organisms. This type of specificity allows MoAbs to destroy pathologic organisms and spare normal cells. The specificity of MoAbs depends on identifying key antigen proteins that are present on the surface of tumors, but not on normal tissues. When the MoAb attaches to the cell surface antigen, it blocks an important signal transduction pathway for communication between the malignant cells and the extracellular environment. The results may include an inability to initiate apoptosis, reproduce, or invade surrounding tissues (Pento, 2017; Singh, Tank, Dwiwedi, et al., 2018).

Advances in Immunology

Important developments in immunology revolve around advances in genetic engineering, use of stem cells, and immunotherapy treatments.

Genetic Engineering

One of the more remarkable evolving technologies is **genetic engineering**, which uses recombinant deoxyribonucleic acid (DNA) technology. Two facets of this technology exist. The first permits scientists to combine genes from one type of organism with genes of a second organism. This type of technology allows cells and microorganisms to manufacture proteins, monokines, and lymphokines, which can alter and enhance immune system function. The second facet of recombinant DNA technology involves gene therapy. If a specific gene is abnormal or missing, experimental recombinant DNA technology may be capable of restoring normal gene function. For example, a recombinant gene is inserted into a virus particle. When the virus particle splices its genes, the virus automatically inserts the missing gene and

theoretically corrects the genetic anomaly. Extensive research into recombinant DNA technology and gene therapy is ongoing (Gonçalves & Paiva, 2017).

Stem Cells

Stem cells are capable of self-renewal and differentiation; they continually replenish the body's entire supply of both RBCs and WBCs. Some stem cells, described as totipotent cells, have tremendous capacity to self-renew and differentiate. Embryonic stem cells, described as pluripotent, give rise to numerous cell types that are able to form tissues. Research has shown that stem cells can restore an immune system that has been destroyed (Haynes et al., 2018). Stem cell transplantation has been carried out in humans with certain types of immune dysfunction, such as severe combined immunodeficiency; clinical trials using stem cells are under way in patients with a variety of disorders having an autoimmune component, including systemic lupus erythematosus, rheumatoid arthritis, scleroderma, and multiple sclerosis. Research with embryonic stem cells has enabled investigators to make substantial gains in developmental biology, gene therapy, therapeutic tissue engineering, and the treatment of a variety of diseases (Haynes et al., 2018).

Cancer and Immunotherapy

It has long been understood that the immune system plays a role in fighting off malignancies. Recent advances in cancer treatment have sought to augment the body's natural antitumor activity and to shut down the pathways that allow malignancies to elude the immune system, which has led to the development of such treatments as MoAbs, cancer vaccines, immune adjuvants, immune checkpoint inhibitors, chimeric antigen receptor (CAR) T-cell therapy and cytokines. These immunotherapeutic treatments are designed to stimulate the patient's immune system to mount its own defense against the cancer and have revolutionized the treatment of many different cancer types. See [Chapter 12](#) for a discussion on the use of immunotherapy in cancer (Gonçalves & Paiva, 2017; Ribas & Wolchok, 2018).

Assessment of the Immune System

An assessment of immune function begins during the health history and physical examination. Areas to be assessed include nutritional status; infections and immunizations; allergies; disorders and disease states, such as autoimmune disorders, cancer, and chronic illnesses; surgeries; medications; and blood transfusions. In addition to inspection of general characteristics,

palpation of the lymph nodes and examinations of the skin, mucous membranes, and respiratory, gastrointestinal, musculoskeletal, genitourinary, cardiovascular, and neurosensory systems are performed ([Chart 31-3](#)).

Health History

The history should note the patient's age along with information about past and present conditions and events that may provide clues to the status of the patient's immune system.

Gender

There are differences in the immune system functions of men and women. For example, many autoimmune diseases have a higher incidence in females than in males, a phenomenon believed to be correlated with sex hormones. In the past two decades, research has revealed that sex hormones are integral signaling modulators of the immune system. Sex hormones play definitive roles in lymphocyte maturation, activation, and synthesis of antibodies and cytokines. In autoimmune disease, expression of sex hormones is altered, and this change contributes to immune dysregulation (Rainville, Tsyglakova, & Hodes, 2018).



Gerontologic Considerations

Immunosenescence is the term for age-related changes in the immune system. These changes have been linked to the increased rates of illness and mortality in older adults (Tariq, Hazeldine, & Lord, 2017). Some of the changes that occur in immunosenescence include, but are not limited to, bone marrow defects, dysfunction of the thymus gland, and impaired lymphocytes. Cellular changes occur as the result of aging and include impaired neutrophil function, decreased circulating macrophages, impaired dendritic cell function, and reduced T-cell activation. As the immune system undergoes age-associated alterations, its response to infections progressively deteriorates. The capacity for self-renewal of hematopoietic stem cells diminishes. There is a notable decline in the total number of phagocytes, coupled with an intrinsic reduction in their activity. The cytotoxicity of NK cells decreases, contributing to a decline in humoral immunity (Tariq et al., 2017). Inflammatory cytokines also tend to increase with age. Acquired immunity may be negatively affected as the efficacy of vaccines is frequently decreased in older adults (Smetana, Chlibek, Shaw, et al., 2018).

Chart 31-3



ASSESSMENT

Assessing for Immune Dysfunction

Be alert for the following signs and symptoms:

Respiratory System

- Changes in respiratory rate
- Cough (dry or productive)
- Abnormal lung sounds (wheezing, crackles, rhonchi)
- Rhinitis
- Hyperventilation
- Bronchospasm

Cardiovascular System

- Hypotension
- Tachycardia
- Arrhythmia
- Vasculitis
- Anemia

Gastrointestinal System

- Hepatosplenomegaly
- Colitis
- Vomiting
- Diarrhea

Genitourinary System

- Frequency and burning on urination
- Hematuria
- Discharge

Musculoskeletal System

- Joint mobility, edema, and pain

Skin

- Rashes
- Lesions
- Dermatitis
- Hematomas or purpura
- Edema or urticaria
- Inflammation
- Discharge

Neurosensory System

- Cognitive dysfunction
- Hearing loss
- Visual changes
- Headaches and migraines
- Ataxia
- Tetany

Older adults have an increased incidence of infections, autoimmune diseases, metabolic diseases, osteoporosis, and neurologic disorders (Eliopoulos, 2018; Tariq et al., 2017). The increased incidence of autoimmune diseases may be from a decreased ability of antibodies to differentiate between self and non-self. Failure of the surveillance system to recognize mutant or abnormal cells may also be responsible, in part, for the high incidence of cancer associated with increasing age.

Age-related changes in many body systems also contribute to impaired immunity ([Table 31-4](#)). For example, postmenopausal females are at a greater risk for urinary tract infections due to residual urine, urinary incontinence, and estrogen deficiency (Jung & Brubaker, 2019). Secondary changes, including malnutrition and poor circulation, as well as the breakdown of natural mechanical barriers such as the skin, place the aging immune system at even greater disadvantage against infection (Tariq et al., 2017).

The effects of the aging process and psychological stress interact, with the potential to negatively influence immune integrity (Gidron, 2019). Consequently, continual assessment of the physical and emotional status of older adults is imperative, because early recognition and management of factors influencing immune response may prevent or mitigate the high morbidity and mortality seen with illness in the older adult population.

Nutrition

Nutritional status is a key determinant of health. Traditionally, the relationship between infection and nutrition focused on the effect of nutrients on host defenses and the effect of infection on nutritional needs (Yaqoob, 2017). This has expanded in scope to encompass the role of specific nutrients in acquired immune function—the modulation of inflammatory processes and the virulence of the infectious agent itself (Lang & Aspinall, 2017). Iron and the immune system are linked in homeostasis and pathology, thus making it essential for maximum function (Martins, Almeida, Lima, et al., 2017). The list of nutrients affecting infection, immunity, inflammation, and cell injury has expanded from traditional proteins to several vitamins, multiple minerals, and, more recently, specific lipid components of the diet. The role of micronutrients and fatty acids on the response of cells and tissues to hypoxic and toxic

damage has been recognized, suggesting that there is another dimension to the relationship. Deficiencies in micronutrients have been connected to impairment in various body functions, including immunity (Carr & Maggini, 2017; Mikkelsen & Apostolopoulos, 2018). Zinc deficiency in particular has been linked to the development of a number of diseases. Zinc plays an important role in homeostasis, immune function, and apoptosis, among other functions (Wessels, Maywald, & Rink, 2017).

TABLE 31-4

Age-Related Changes in Immunologic Function

Body System	Changes	Consequences
Immune	Impaired function of B and T lymphocytes Failure of lymphocytes to recognize mutant or abnormal cells Decreased antibody production Failure of immune system to differentiate “self” from “non-self” Suppressed phagocytic immune response	Suppressed responses to pathogenic organisms with increased risk for infection Increased incidence of cancers Anergy (lack of response to antigens applied to the skin [allergens]) Increased incidence of autoimmune diseases Absence of typical signs and symptoms of infection and inflammation Dissemination of organisms usually destroyed or suppressed by phagocytes (e.g., reactivation or spread of tuberculosis)
Gastrointestinal	Decreased gastric secretions and motility Decreased phagocytosis by the liver’s Kupffer cells Altered nutritional intake with inadequate protein intake	Proliferation of intestinal organisms resulting in gastroenteritis and diarrhea Increased incidence and severity of hepatitis B; increased incidence of liver abscesses Suppressed immune response
Urinary	Decreased kidney function and changes in lower urinary tract function (enlargement of prostate gland, neurogenic bladder); altered genitourinary tract flora	Urinary stasis and increased incidence of urinary tract infections
Pulmonary	Impaired ciliary action due to exposure to smoke and environmental toxins	Impaired clearance of pulmonary secretions; increased incidence of respiratory infections
Integumentary	Thinning of skin with less elasticity; loss of adipose tissue	Increased risk of skin injury, breakdown, and infection
Circulatory	Impaired microcirculation	Stasis and pressure injuries
Neurologic function	Decreased sensation and slowing of reflexes	Increased risk of injury, skin ulcers, abrasions, and burns

Adapted from Eliopoulos, C. (2018). *Gerontological nursing* (9th ed.). Philadelphia, PA: Wolters Kluwer.

The effects exerted by polyunsaturated fatty acids on immune system functions are under investigation. Studies suggest that these elements play a role in diminishing the incidence and severity of inflammatory disorders. Research suggests that diets high in olive oil are not as immunosuppressive as diets rich in fish oil. The contribution of immune modulation by lipids to the high risk of infectious complications associated with the use of parenteral nutrition is unclear (Raman, Almutairdi, Mulesa, et al., 2017).

Depletion of protein reserves results in atrophy of lymphoid tissues, depression of antibody response, reduction in the number of circulating T cells, and impaired phagocytic function. As a result, susceptibility to infection is greatly increased. During periods of infection or serious illness, nutritional requirements may be further altered, potentially contributing to depletion of protein, fatty acid, vitamin, and trace elements and causing even greater risk of impaired immune response and sepsis (Wischmeyer, 2018). Nutritional intake that supports a competent immune response plays an important role in reducing the incidence of infections (Shlisky, Bloom, Beaudreault, et al., 2017); patients whose nutritional status is compromised have a delayed postoperative recovery and often experience more severe infections and delayed wound healing. There is evidence that nutrition plays a role in the development of cancer and that diet and lifestyle can alter the risk of cancer development as well as other chronic diseases (Theodoratou, Timofeeva, Li, et al., 2017). The nurse must assume a proactive role in ensuring the best possible nutritional intake for all patients as a vital step in preventing disease and poor outcomes. The nurse must assess the patient's nutritional status, caloric intake, and quality of foods ingested (see [Chapter 4](#) for further discussion of nutritional assessment).

Immunization

The patient is asked about childhood and adult immunizations, including vaccinations to provide protection against influenza, pneumococcal diseases, herpes zoster, pertussis, and the usual childhood diseases (e.g., measles, mumps). Education about the importance of adhering to the recommended schedule for adult vaccines should be initiated. See [Chapter 3, Table 3-3: Select Health Promotion Screening for Adults](#) for more information about adult immunizations.

Infection

A history of past and present infections and the dates and types of treatments, along with a history of any multiple persistent infections, fevers of unknown origin, lesions or sores, or any type of drainage, as well as the response to treatment, are obtained.

Known past or present exposure to tuberculosis is assessed, and the dates and results of any tuberculin tests (purified protein derivative [PPD] test) and chest x-rays are documented. Recent exposure to any infections, recent travel, and dates are elicited. The nurse must assess whether the patient has been exposed to any sexually transmitted infections (STIs) or bloodborne pathogens such as hepatitis B, C, and D viruses and human immune deficiency virus (HIV). A history of STIs such as gonorrhea, syphilis, human papillomavirus infection, and chlamydia can alert the nurse that the patient may have been exposed to HIV or hepatitis. Herpes simplex virus infections have a significant impact on health, causing a wide range of diseases (e.g., oral and genital herpes).

Allergy

The patient is asked about any allergies, including types of allergens (e.g., pollens, dust, plants, cosmetics, food, medications, vaccines, latex), the symptoms experienced, and seasonal variations in occurrence or severity in the symptoms. A history of testing and treatments, including prescribed and over-the-counter medications that the patient has taken or is currently taking for these allergies and the effectiveness of the treatments, is obtained. All medication and food allergies are listed on paper records with an allergy alert sticker or within the patient's electronic health record (EHR) to make others aware of these allergies. Continued assessment for potential allergic reactions in the patient is vital. See [Chapter 33](#) for more information on allergies.

Disorders and Diseases

Part of the focused immunologic assessment includes determining whether the patient has a history of autoimmune disorders, neoplasms, chronic illnesses, surgeries, or any recent major stressors that may place the patient at increased risk.

Autoimmune Disorders

Autoimmune disorders affect people of both genders of all ages, ethnicities, and social classes. Autoimmune disorders are a group of disorders that can affect almost any cell or tissue in the body (Norris, 2019). As mentioned previously, they tend to be more common in women because estrogen tends to enhance immunity. Androgen, on the other hand, tends to be immunosuppressive. Autoimmune diseases are a leading cause of death by disease in females of reproductive age.

The patient is asked about any autoimmune disorders, such as lupus erythematosus, rheumatoid arthritis, multiple sclerosis, or psoriasis. The onset, severity, remissions and exacerbations, functional limitations, treatments that the patient has received or is currently receiving, and effectiveness of the

treatments are described. Certain autoimmune diseases appear to be genetically linked, so a family history of these is important (Generali, Ceribelli, Stazi, et al., 2017) ([Chart 31-4](#)).

Neoplastic Disease

If there is a history of cancer in the family, more information is obtained, including the type of cancer, age at onset, and relationship (maternal or paternal) of the patient to the affected family members. Dates and results of any cancer screening tests for the patient are documented.

A history of cancer in the patient is also obtained, along with the type of cancer, date of diagnosis, and treatment modalities used. Immunosuppression contributes to the development of cancers; however, cancer itself is immunosuppressive, as are many treatments for cancer. Large tumors can release antigens into the blood, and these antigens combine with circulating antibodies and prevent them from attacking the tumor cells. Furthermore, tumor cells may possess special blocking factors that coat tumor cells and prevent their destruction by killer T lymphocytes. During the early development of tumors, the body may fail to recognize the tumor antigens as foreign and subsequently fail to initiate destruction of the malignant cells. Hematologic cancers, such as leukemia and lymphoma, are associated with altered production and function of WBCs and lymphocytes.

Chart 31-4



GENETICS IN NURSING PRACTICE

Immunologic Disorders

An immunologic disorder is a disorder of a person's immune system, which is a network of cells, tissues, and organs that work together to defend the body against attacks by foreign invaders such as bacteria, parasites, and fungi that can cause infection. A number of immunologic disorders have a known inheritance pattern, while others are noted to have a genetic abnormality that is influenced by environmental exposures. Therefore, the pattern of inheritance is unclear in some immunologic disorders. Examples of immunologic disorders caused by a genetic abnormality include:

- Adenosine deaminase deficiency (autosomal recessive)
- Alopecia areata
- Alopecia totalis
- Asthma
- Ataxia telangiectasia (autosomal recessive)
- Autoimmune polyglandular syndrome
- Bruton agammaglobulinemia (X-linked)
- Burkitt lymphoma
- Crohn's disease
- Diabetes, type 1
- DiGeorge syndrome (Autosomal dominant)
- Familial Mediterranean fever
- Job syndrome (autosomal dominant and recessive)
- Purine nucleoside phosphorylase deficiency (autosomal dominant)
- Severe combined immunodeficiency (primarily X-linked)
- Wiskott–Aldrich syndrome (X-linked)

Nursing Assessments

Refer to [Chapter 4, Chart 4-2: Genetics in Nursing Practice: Genetic Aspects of Health Assessment](#)

Family History Assessment Specific to Immunologic Disorders

- Collect a family history for both maternal and paternal relatives for three generations.
- Assess family history for other family members with histories of immunologic disorders.
- Obtain information about family members with a history of recurrent infections or illness.
- Recognize ethnic risk (non-Ashkenazi Jewish, Armenian, Arab, and Turkish are at greater risk for familial Mediterranean fever; Caucasians have a higher incidence of Crohn's disease)

Patient Assessment

- Assess for symptoms such as changes in respiratory status associated with asthma (e.g., wheezing, or airway hyperresponsiveness; mucosal edema; and mucus production).
- Gather information regarding immunizations and whether an altered response to any immunization has occurred.
- Assess for symptoms of immunodeficiency disorders, such as unexplained weight gain or loss, skin rashes, changes in hair texture or distribution, joint or muscle pain, intolerance to cold, irregular menstrual periods, abdominal discomfort, or the presence of diarrhea.
- Identify pattern of sickness with regard to frequency of colds, respiratory infections, or history of illness that tends to linger.
- Obtain history of childhood illnesses and details of the illness experience.
- Assess for medical history of frequent or recurrent infections.
- Learn about susceptibility to infections, assess for patterns (frequency, length of illness, severity of symptoms), and recognize infections that would be atypical for age.
- Inquire about environmental exposures (e.g., smoke, chloroform, metal or dust particles, paint).
- Ask about exposure to other viruses such as Epstein–Barr or Influenza.

Resources

American Autoimmune Related Diseases Association, www.aarda.org

Genetic and Rare Diseases Information Center, www.rarediseases.info.nih.gov

See Chapter 6, Chart 6-7 for components of genetic counseling.

All treatments that the patient has received or is currently receiving, such as radiation, chemotherapy, and immunotherapy, are recorded in the health history. In addition, the nurse should elicit information related to complementary or alternative modalities that have been used and the response to these efforts. Radiation destroys lymphocytes and decreases the ability to mount an effective immune response. The size and extent of the irradiated area determine the extent of immunosuppression. Whole-body irradiation may leave the patient completely immunosuppressed. Chemotherapy and other cancer treatments also affect bone marrow function, destroying cells that contribute to an effective immune response and resulting in immunosuppression. Immunotherapy can cause inflammatory overreactions of the immune system that mimic autoimmune disorders (Kroschinsky, Stolzel, von Bonin, et al., 2017; Munro, 2019).

Chronic Illness and Surgery

The health assessment includes a history of chronic illness, particularly diabetes, renal disease, or chronic obstructive pulmonary disease (COPD). The

onset and severity of illnesses, as well as treatment that the patient is receiving for the illness, are obtained. Chronic illness may contribute to immune system impairments in various ways. Kidney injury is associated with a deficiency in circulating lymphocytes. In addition, immune defenses may be altered by acidosis and uremic toxins. In diabetes, an increased incidence of infection has been associated with vascular insufficiency, neuropathy, and poor control of serum glucose levels. Recurrent respiratory tract infections are associated with COPD due to altered inspiratory and expiratory function and ineffective airway clearance. In addition, a history of organ transplantation or surgical removal of the spleen, lymph nodes, or thymus should be noted, because these conditions may place the patient at risk for impaired immune function (Bagatini, Cardoso, dos Santos, et al., 2017; Dionne, Dehority, Brett, et al., 2017).

Special Problems

Conditions such as burns and other forms of injury and infection may contribute to altered immune system function. Major burns cause impaired skin integrity and compromise the body's first line of defense. Loss of large amounts of serum occurs with burn injuries and depletes the body of essential proteins, including immunoglobulins.

The physiologic and psychological stressors associated with surgery or injury stimulate cortisol release from the adrenal cortex; increased serum cortisol also contributes to suppression of normal immune responses. The immune system's inflammatory response to surgery is followed by an anti-inflammatory compensatory response, and it is thought that these responses may contribute to postoperative complications (Cerra, 2018).

Patients who have had an ischemic stroke or transient ischemic attack (TIA) are at risk for infection following the event. Evidence suggests that an acute stroke leads to immunosuppression and a subsequently high infection risk; infection is the leading cause of death following a stroke (Hoffmann, Harms, Ulm, et al., 2017). Stroke-induced immunosuppression is linked to the development of stroke-associated pneumonia, which is the most common infection seen in patients who have had a stroke (Liu, Chu, Chen, et al., 2018).

Medications and Blood Transfusions

A list of past and present medications is obtained. In large doses, antibiotics, corticosteroids, cytotoxic agents, salicylates, nonsteroidal anti-inflammatory drugs, and anesthetic agents can cause immune suppression ([Table 31-5](#)).

A history of blood transfusions is obtained; previous exposure to foreign antigens through transfusion may be associated with abnormal immune function. In addition, although the risk of HIV transmission through blood transfusion is extremely low in patients who received a transfusion after 1985

(when testing of blood for HIV was initiated in the United States), a small risk remains.

The patient is also asked about the use of herbal agents and over-the-counter medications. Because many herbal agents have not been subjected to rigorous testing, their effects have not been fully identified. It is important, therefore, to ask patients about their use of these substances, to document their use, and to educate patients about untoward effects that may alter immune responsiveness.

Lifestyle Factors

Personal lifestyle choices have an impact on the immune system. Poor nutritional intake, smoking (Qiu, Liang, Liu, et al., 2017), excessive consumption of alcohol (Rehm, Gmel, Gmel, et al., 2017), illicit drug use, and occupational or residential exposure to environmental radiation and pollutants have been associated with impaired immune function and are assessed in a detailed patient history. Although factors that are not consistent with a healthy lifestyle are predominately responsible for ineffective immune function, positive lifestyle factors can also negatively affect immune function and require assessment. For example, rigorous exercise or competitive exercise—usually considered a positive lifestyle factor—can be a physiologic stressor and cause negative effects on immune response (Aoi & Naito, 2019; Shaw, Merien, Braakhuis, et al., 2018).

TABLE 31-5 Select Medications and Effects on the Immune System

Drug Classification (and Examples)	Effects on the Immune System
Antibiotics (in Large Doses)	
ceftriaxone	Bone Marrow Suppression Eosinophilia, hemolytic anemia, hypoprothrombinemia, neutropenia, thrombocytopenia
cefuroxime sodium	Eosinophilia, hemolytic anemia, hypoprothrombinemia, neutropenia, thrombocytopenia
chloramphenicol	Leukopenia, aplastic anemia
dactinomycin	Agranulocytosis, neutropenia
fluoroquinolones (ciprofloxacin, levofloxacin)	Hemolytic anemia, methemoglobinemia, eosinophilia, leukopenia, pancytopenia
gentamicin sulfate	Agranulocytosis, granulocytosis
macrolides (erythromycin, azithromycin, clarithromycin)	Neutropenia, leukopenia
penicillins	Agranulocytosis
streptomycin	Leukopenia, neutropenia, pancytopenia
vancomycin	Transient leucopenia
Antithyroid Drugs	
propylthiouracil	Agranulocytosis, leukopenia
Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) (in Large Doses)	Inhibit Prostaglandin Synthesis or Release
aspirin	Agranulocytosis
COX-2 inhibitors (celecoxib)	Anemia, allergy, no other major adverse effects
ibuprofen	Leukopenia, neutropenia
indomethacin	Agranulocytosis, leukopenia
phenylbutazone	Pancytopenia, agranulocytosis, aplastic anemia
Adrenal Corticosteroids	Immunosuppression
prednisone	
Antineoplastic Agents (cytotoxic agents)	Immunosuppression
cyclophosphamide	Leukopenia, neutropenia
cisplatin	Leukopenia
cyclosporine	Leukopenia, inhibits T-lymphocyte function
Antimetabolites	Immunosuppression
pyrimidine antagonist	Leukopenia, eosinophilia
folic acid antagonist	Leukopenia, aplastic bone marrow
purine antagonist	Leukopenia, pancytopenia

COX, cyclo-oxygenase.

Adapted from Comerford, K. C., & Durkin, M. T. (Eds.). (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer.

Psychoneuroimmunologic Factors

The bidirectional pathway between the brain and immune system (the mind–body system) is referred to as psychoneuroimmunology. Patient assessment must also address psychoneuroimmunologic factors, such as stress and psychological illness, that may be influencing the patient’s health. The immune response is regulated and modulated in part by neuroendocrine influences. Lymphocytes and macrophages have receptors that can respond to neurotransmitters and endocrine hormones. Lymphocytes can produce and secrete adrenocorticotropic hormone and endorphinlike compounds. Cells in the brain, especially in the hypothalamus, can recognize prostaglandins, interferons, and interleukins, as well as histamine and serotonin, all of which are released during the inflammatory process. Like all other biologic systems functioning to maintain homeostasis, the immune system is integrated with other psychophysiological processes and is regulated and modulated by the brain. These relationships may have immunologic consequences (Lasselin, Schedlowski, Lekander, et al., 2019).

Growing evidence indicates that a measurable immune system response can be positively influenced by biobehavioral strategies such as relaxation, imagery techniques, biofeedback, humor, hypnosis, mindfulness-based strategies, and yoga (Park & Han, 2017; Pascoe, Thompson, & Ski, 2017; Reich, Lengacher, Klein, et al., 2017). Therefore, the assessment should address the patient’s general psychological status and the patient’s use of and response to these strategies. See the Nursing Research Profile in [Chart 31-5](#).

Physical Assessment

During the physical examination (see [Chart 31-3](#)), the skin and mucous membranes are assessed for lesions, dermatitis, purpura (subcutaneous bleeding), urticaria, inflammation, or any discharge. Any signs of infection are noted. The patient’s temperature is recorded, and the patient is observed for chills and sweating. The anterior and posterior cervical, supraclavicular, axillary, and inguinal lymph nodes are palpated for enlargement; if palpable nodes are detected, their location, size, consistency, and reports of tenderness on palpation are noted. Joints are assessed for tenderness, swelling, increased warmth, and limited range of motion. The patient’s respiratory, cardiovascular, genitourinary, gastrointestinal, and neurosensory systems are evaluated for signs and symptoms indicative of immune dysfunction. Any functional limitations or disabilities the patient may have are also assessed.

Diagnostic Evaluation

A series of blood tests and skin tests, as well as bone marrow biopsy, may be performed to evaluate the patient's immune competence. Specific laboratory and diagnostic tests are discussed in greater detail along with individual disease processes in subsequent chapters in this unit. Select laboratory and diagnostic tests used to evaluate immune competence are summarized in [Chart 31-6](#).

Nursing Management

The nurse needs to be aware that patients undergoing evaluation for possible immune system disorders experience not only physical pain and discomfort with certain types of diagnostic procedures but also many psychological reactions. It is the nurse's role to counsel, educate, and support patients throughout the diagnostic process. Many patients may be extremely anxious about the results of diagnostic tests and the possible implications of those results for their future health, employment, and personal relationships. This is an ideal time for the nurse to provide counseling and education, should these interventions be warranted.

Chart 31-5



NURSING RESEARCH PROFILE

Stress Reduction and Effect on Inflammatory Cytokines

Reich, R. R., Lengacher, C. A., Klein, T. W., et al. (2017). A randomized controlled trial of the effects of mindfulness-based stress reduction (MBSR[BC]) on levels of inflammatory biomarkers among recovering breast cancer survivors. *Biological Research for Nursing*, 19(4), 456–464.

Purpose

The purpose of this study was to evaluate the efficacy of the Mindfulness-Based Stress Reduction (Breast Cancer) (MBSR[BC]) program versus usual care in normalizing blood levels of proinflammatory cytokines among breast cancer survivors.

Design

This was a substudy of a large randomized controlled trial. A total of 322 participants were randomized to a 6-wk MBSR(BC) program or usual care. Blood samples, demographic data (age, race, and ethnicity), and clinical data (disease stage and treatments) were collected at baseline, 6 wks, and 12 wks. Plasma cytokines (IL-1 β , IL-6, IL-10), tumor necrosis factor α (TNF- α), transforming growth factor β 1, and soluble tumor necrosis factor receptor 1 (sTNFR1) were assayed. Linear mixed models were used to assess the cytokine levels across the three time points by group.

Findings

Three of the cytokines were not detectable and thus not analyzed further. For the remaining cytokines (TNF- α , IL-6, sTNFR1), TNF α and IL-6 increased during the follow-up period between 6 and 12 wks, but not during the MBSR(BC) training period (baseline to 6 wks). The sTNFR1 levels did not change across the 12-wk study period significantly. The s cytokines TNF- α and IL-6 may be markers for recovery.

Nursing Implications

Patients recovering from cancer treatment are at increased risk for infections and increased levels of psychological distress. Stress reducing activities and programs such as the 2 h per wk mindfulness program can be implemented in clinical practice as part of an interdisciplinary approach to cancer care. Nurses should advise patients that the programs may not take effect immediately; in fact, some of the increases in cytokine levels were not seen until 6 wks after the program ended.

Chart 31-6

Select Tests for Evaluating Immunologic Status

Various laboratory tests may be performed to assess immune system activity or dysfunction. The studies assess leukocytes and lymphocytes, humoral immunity, cellular immunity, phagocytic cell function, complement activity, hypersensitivity reactions, specific antigen–antibodies, or human immune deficiency virus infection.

Humoral (Antibody-Mediated) Immunity Tests

- B-cell quantification with monoclonal antibody
- In vivo immunoglobulin synthesis with T-cell subsets
- Specific antibody response
- Total serum globulins and individual immunoglobulins (electrophoresis, immunoelectrophoresis, single radial immunodiffusion, nephelometry, and isohemagglutinin techniques)

Cellular (Cell-Mediated) Immunity Tests

- Total lymphocyte count
- T-cell and T-cell subset quantification with monoclonal antibody
- Delayed hypersensitivity skin test
- Cytokine production
- Lymphocyte response to mitogens, antigens, and allogeneic cells
- Helper and suppressor T-cell functions

Adapted from Fischbach, F. T., & Fischbach, M. A. (2018). *A manual of laboratory and diagnostic tests* (10th ed.). Philadelphia, PA: Wolters Kluwer.

CRITICAL THINKING EXERCISES

1 pq You are caring for an 80-year-old woman who is admitted to a long-term care facility after having a stroke. What potential immunity-related complications do you need to be aware of for this patient? What are your priority nursing observations and assessments? Identify priorities for this patient's care given her recent stroke.

2 ebp A 40-year-old woman with melanoma is being treated with immunotherapy and is admitted to your hospital for treatment-related pneumonitis. She is prescribed high-dose corticosteroids for treatment of the pneumonitis. The patient has asked you to explain what is happening to her and why this has occurred. How will you educate her and her family about her current condition? Develop an evidence-based education plan for the patient and her family. Discuss the criteria used to assess the strength of the evidence for your education plan.

3 ipc A 35-year-old pregnant woman with systemic lupus erythematosus (SLE) presents with an acute flare-up of her symptoms. How might psychosocial and emotional factors impact her health? What interdisciplinary referrals would you anticipate for this patient's care?

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*Asterisk indicates nursing research.

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Resources

- American Academy of Allergy, Asthma & Immunology, www.aaaai.org
- American Cancer Society, www.cancer.org
- Centers for Disease Control and Prevention, www.cdc.gov
- National Institute of Allergy and Infectious Diseases, www.niaid.nih.gov
- National Institutes of Health, Health Information, www.nih.gov/health/infoline.htm
- National Institutes of Health, National Cancer Institute, www.cancer.gov
- U.S. Department of Health & Human Services, www.hhs.gov

32 Management of Patients with Immune Deficiency Disorders

LEARNING OUTCOMES

On completion of this chapter, the learner will be able to:

1. Identify the pathophysiology, clinical manifestations, and nursing management of patients with primary immune deficiency disorders.
2. Describe the modes of transmission of human immune deficiency virus infection and prevention strategies.
3. Explain the pathophysiology associated with the clinical manifestations of human immune deficiency virus and acquired immune deficiency syndrome and the purpose of antiretroviral therapy.
4. Use the nursing process as a framework for care of the patient with human immune deficiency virus/acquired immune deficiency syndrome.
5. Identify available resources for patients and support systems to promote self-management of immune deficiency disorders.

NURSING CONCEPTS

Family
Immunity
Infection
Nutrition

GLOSSARY

candidiasis: fungal infection, usually of skin or mucous membranes, caused by Candida species

enzyme immunoassay (EIA): a blood test that can determine the presence of antibodies to HIV in the blood or saliva; a variant of this test is called enzyme-linked immunosorbent assay (ELISA)

HIV-1: retrovirus isolated and recognized as the etiologic agent of HIV disease

HIV encephalopathy: clinical syndrome characterized by a progressive decline in cognitive, behavioral, and motor functions

immune reconstitution inflammatory syndrome (IRIS): a syndrome that results from rapid restoration of pathogen-specific immune responses to opportunistic infections

Kaposi sarcoma: malignancy that involves the epithelial layer of blood and lymphatic vessels

latent reservoir: the integrated HIV provirus within the CD4⁺ T cell during the resting memory state; does not express viral proteins and is invisible to the immune system and antiviral medications

Mycobacterium avium complex (MAC): opportunistic infection caused by mycobacterial organisms that commonly causes a respiratory illness but can also infect other body systems

opportunistic infection: illness caused by various organisms, some of which typically do not cause disease in people with normal immune systems

peripheral neuropathy: disorder characterized by sensory loss, pain, muscle weakness, and wasting of muscles in the hands or legs and feet

Pneumocystis pneumonia (PCP): common opportunistic lung infection; pathogen implicated is *Pneumocystis jirovecii*

polymerase chain reaction (PCR): a sensitive laboratory technique that can detect and quantify HIV in a person's blood or lymph nodes

post-exposure prophylaxis (PEP): taking antiretroviral medicines as soon as possible, but no more than 72 hours (3 days) after possible HIV exposure; two to three drugs are usually prescribed which must be taken for 28 days

pre-exposure prophylaxis (PrEP): prevention method for HIV-negative people who are at high risk of HIV infection; involves taking a specific combination of HIV medicines daily; use with condoms and other prevention tools

primary immune deficiency diseases (PIDDs): rare, genetic disorders that impair the immune system

progressive multifocal leukoencephalopathy: opportunistic infection that infects brain tissue and causes damage to the brain and spinal

cord

retrovirus: a virus that carries genetic material in ribonucleic acid (RNA) instead of DNA and contains reverse transcriptase

viral load test: measures the quantity of HIV RNA or DNA in the blood

viral set point: amount of virus present in the blood after the initial burst of viremia and the immune response that follows

wasting syndrome: involuntary weight loss consisting of both lean and fat body mass

The human immune system is complex and multidimensional. It works to protect against invasion by foreign substances, protect against the proliferation of neoplastic cells, and plays a key role in inflammation and healing. Patients with primary or secondary immune system disorders (Norris, 2019) require care from nurses who are knowledgeable about the pathophysiology, diagnostic procedures, and interventions that are used in the management of these disorders.

PRIMARY IMMUNE DEFICIENCIES DISEASES

The majority of **primary immune deficiency diseases (PIDDs)**, rare inherited disorders that impair the immune system, are commonly diagnosed in infancy, with a male-to-female ratio of 5 to 1. However, some PIDDs are not diagnosed until adolescence or early adulthood, when the gender distribution equalizes. Diagnosis at this stage frequently is confounded by frequent use of antibiotics that mask symptoms. Adults may present with clinical episodes of infectious diseases beyond the scope of normal immunocompetence, such as infections that are unusually persistent, recurrent, or resistant to treatment and that involve unexpected dissemination of disease or atypical pathogens. These rare inherited disorders not only lead to frequent infections, but also to increased risk of autoimmune disorders and malignancy (Hajjar, Guffey, Minard, et al., 2017).

Pathophysiology

There are more than 200 forms of PIDDs affecting about 500,000 people in the United States, and more than 270 different genes are associated with PIDDs. These rare genetic diseases prevent the body from developing normal immune responses resulting in a complex group of disorders with a wide array of clinical presentations. Many present during the first year of life and may be chronic, debilitating, and costly (National Institute of Allergy and Infectious Diseases [NIAID], 2019a).

Clinical Manifestations

Major signs and symptoms include multiple infections despite aggressive treatment, infections with unusual or opportunistic organisms, failure to thrive or poor growth, and a significant family history. Fatigue was reported in 18% of patients with PIDDs (Hajjar et al., 2017). See [Table 32-1](#) for select PIDDs along with some of their clinical manifestations.

Assessment and Diagnostic Findings

There is often a considerable delay between the onset of symptoms and time of diagnosis of PIDDs. Because of the genetic origins of PIDDs, family history should be carefully assessed but epidemiologic data about specific infectious agents should be considered as well.

Laboratory tests are used to identify antibody deficiencies, cellular (T-cell) defects, neutrophil disorders, and complement deficiencies. A complete blood cell count with manual differential should always be analyzed first. Lymphopenia may indicate an immunologic abnormality; serum Ig levels (IgG, IgM, and IgA) and antibody responses to vaccines should be assessed to detect a humoral immune defect. Age-matched normal ranges need to be used since antibody levels change as the person ages (Fischbach & Fischbach, 2018).

Prevention

Live vaccines are contraindicated in patients with antibody deficiency disorders. The patient is incapable of generating antibodies and the live substance in the vaccine can cause disease. Family planning should be addressed in terms of future pregnancies; in some situations, prenatal in utero testing of a fetus can determine whether the infant will be affected.

Medical Management

A pattern of unusually frequent, opportunistic, or severe infections generates the possibility of a PIDD and initial testing or referral to an immunologist. Patients with neutropenia are at increased risk for developing severe infections despite substantial advances in supportive care. Epidemiologic shifts occur periodically and must be detected early because they influence prophylactic, empiric, and specific strategies for medical management. Attention to infection control practices is important, especially with the emergence of multidrug-resistant organisms.

Hematopoietic stem cell transplantation (HSCT) is a curative modality. The stem cells may be from embryos or adults. Toxicity and reduced efficacy are

frequent limitations of HSCT. See [Chapter 12](#) for further discussion of HSCT.

Another therapy involves the use of cells as vehicles for the delivery of genes or gene products. Gene therapy has had many adverse effects; the first studies in human participants revealed numerous toxicities with this therapy. Rapidly emerging new technologies allowing precise DNA targeting may prove to be a useful approach (McDermott & Murphy, 2019).

Pharmacologic Therapy

Pharmacologic treatment depends upon the type and severity of presenting infection and the particular PIDD diagnosis. Prophylactic drug treatment prevents some bacterial and fungal infections but it must be used with caution because it has been implicated in the emergence of resistant organisms. The choices for empiric therapy include combination regimens and monotherapy. Specific choices depend on local factors (epidemiology, susceptibility/resistance patterns, availability, cost). Patients with antibody deficiencies receive regular Ig replacement therapy including both IV immunoglobulin (IVIG) and subcutaneous immunoglobulin (SCIG) to provide functional antibodies (Stonebraker, Hajjar, & Orange, 2018; Vitiello, Emmi, Silvestri, et al., 2019).

TABLE 32-1 Select Primary Immune Deficiency Disorders (PIDDs)

Disorder	Characteristics
Autoimmune lymphoproliferative syndrome (ALPS)	Unusually high numbers of lymphocytes accumulate in the lymph nodes, liver, and spleen leading to enlargement of those organs. Causes numerous autoimmune problems including low levels of red blood cells, platelets, and neutrophils that can increase the risk of infection and hemorrhage.
Autoimmune polyglandular syndrome type 1 (APS-1) (also called autoimmune polyendocrinopathy-candidiasis-ectodermal dystrophy [APECED])	Causes a diverse range of symptoms, including autoimmunity against different types of organs and increased susceptibility to candidiasis, a fungal infection caused by <i>Candida</i> yeast.
CARD9 Deficiency and other syndromes of susceptibility to candidiasis	Results in susceptibility to fungal infections such as candidiasis; particularly susceptible to <i>Candida</i> infections of the central nervous system.
Chronic granulomatous disease (CGD)	May be caused by mutations in one of five different genes. Phagocytes are unable to kill certain bacteria and fungi resulting in increased susceptibility to infections.
Common variable immunodeficiency (CVID)	Caused by a variety of genetic abnormalities resulting in defective ability of immune cells to produce normal amounts of antibodies, resulting in frequent bacterial or viral infections of the upper airway, sinuses, and lungs.
Congenital neutropenia syndromes	Characterized by low levels of neutrophils from birth.
CTLA4 Deficiency	Autoimmunity, low levels of antibodies, and excessive numbers of lymphocytes which infiltrate the gut, lungs, bone marrow, central nervous system, kidneys resulting in recurrent infections.
DOCK8 Deficiency	Lower-than-normal numbers of immune cells, which have a diminished capacity to move through dense tissues like the skin leading to recurrent viral infections of the skin and respiratory system.
GATA2 Deficiency	Characterized by immune deficiency, lung disease, problems of the vascular and lymphatic systems, and myelodysplastic syndrome (a condition characterized by ineffective blood cell production).
Glycosylation disorders with immune deficiency	Defects in glycosylation, which refers to the attachment of sugars to proteins; can disrupt the immune system resulting in immune deficiency.
Hyper-immunoglobulin E syndrome (HIES)	A rare primary immune deficiency disease characterized by eczema, recurrent staphylococcal skin abscesses, recurrent lung infections, eosinophilia (a high number of eosinophils in the blood) and high serum levels of IgE.

Hyper-immunoglobulin M (hyper-IgM) syndromes	Immune system fails to produce normal IgA, IgG, and IgE antibodies but can produce normal or elevated IgM.
Interferon gamma, interleukin 12, interleukin 23 deficiencies	Interferon gamma, interleukin 12, and interleukin 23 are key signals that raise alert against bacteria and other infectious microbes; deficiencies result in susceptibility to infections caused by bacteria and viruses.
Leukocyte adhesion deficiency (LAD)	Phagocytes are unable to move to the site of an infection resulting in an inability to fight pathogens resulting in recurrent, life-threatening infections and poor wound healing.
PI3 Kinase disease	Genetic mutations overactivate an important immune signaling pathway causing a change reaction that disrupts the infection-fighting B and T cells resulting in a weakened immune system and frequent bacterial and viral infections.
PLCG2-associated antibody deficiency and immune dysregulation (PLAID)	Rare disorder with allergic response to cold (cold urticaria) as the most distinct symptom.
Severe combined immune deficiency (SCID)	Group of rare, life-threatening disorders caused by mutations in different genes involved in development and function of T and B cells; infants appear healthy at birth but are highly susceptible to severe infections.
Warts, hypogammaglobulinemia, infections, and myelokathexis syndrome (WHIMS)	Low levels of white blood cells, especially neutrophils, which predispose to frequent infections and persistent warts.
Wiskott–Aldrich syndrome (WAS)	Problems with B and T cells and platelets resulting in prolonged episodes of bleeding, recurrent bacterial and fungal infections and increased risk of cancers and autoimmune diseases.

Adapted from National Institute of Allergy and Infectious Diseases (NIAID). (2019a). Types of primary immune deficiency disorders. Retrieved on 10/13/2019 at: www.niaid.nih.gov/diseases-conditions/types-pidds



For the procedural guidelines for managing immunoglobulin therapy, go to thepoint.lww.com/Brunner15e.

Nursing Management

Many patients with PIDDs have comorbid autoimmune disorders, such as thyroid disease, rheumatoid arthritis, cytopenias, and inflammatory bowel disease. Many patients require immunosuppression to ensure engraftment of depleted bone marrow during transplantation procedures. For this reason, nursing care must be meticulous. Appropriate hand hygiene and infection prevention precautions are essential. See [Chapter 66](#), [Chart 66-1](#) for hand hygiene methods and [Chart 66-2](#) for a summary of infection prevention precautions. Institutional policies and procedures related to infection prevention care must be followed scrupulously until definitive evidence demonstrates that precautions are unnecessary. Continual monitoring of the patient's condition is critical, so early signs of impending infection may be detected and treated before they seriously compromise the patient's status.

Patients and caregivers in the home are taught how to administer medications, including regular Ig replacement therapy, if prescribed. Instruction is provided to the patient and family about how to administer the therapy at home (see [Chart 32-1](#)). Nurses provide ongoing education and support for the patient and family.

ACQUIRED IMMUNE DEFICIENCY

Immune deficiency can be acquired due to medical treatment such as chemotherapy (see [Chapter 12](#)) or infection from pathogens such as human immune deficiency virus (HIV). Advances have been made in treating HIV infection and acquired immune deficiency syndrome (AIDS); however, AIDS remains a critical public health issue in communities across the United States and around the world. Prevention, early detection, and ongoing treatment remain important aspects of care for people living with HIV infection or AIDS, who are sometimes referred to as persons living with HIV/AIDS (PLWHA). The American Nurses Association (ANA) has issued a position statement in support of efforts to end HIV (ANA, 2019).

HIV Infection and AIDS

Since the disease now known as AIDS was first identified in 1981, remarkable progress has been made in improving the length and quality of life for PLWHA. During the first decade, progress was associated with the recognition and treatment, including prophylactic medications, of **opportunistic infections** (illnesses caused by various organisms, some of which usually do not cause disease in people with normal immune systems). The second decade witnessed progress in the development of highly active antiretroviral drug therapies (HAART) as well as continuing progress in the treatment of opportunistic infections. The third decade has focused on issues of preventing

new infections, adherence to antiretroviral therapy (ART), development of second-generation combination medications that affect different stages of the viral life cycle, and continued need for an effective vaccine. The HIV antibody test, an **enzyme immunoassay** (EIA; or a variant of this test called *enzyme-linked immunosorbent assay* [ELISA]), became available in 1984, allowing early diagnosis of the infection before the onset of symptoms. Since then, HIV infection has been best managed as a chronic disease, most appropriately in an outpatient care setting, whereas AIDS may involve acute conditions that require hospitalization.

Chart 32-1



HOME CARE CHECKLIST

Home Administration of Ig Replacement Therapy

At the completion of education, the patient and/or caregiver will be able to:

- State the impact of immune deficiency on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State what types of changes are needed (if any) to maintain a clean home environment and prevent infection.
- State how to contact the primary provider, the team of home care professionals overseeing care, and intravenous supply vendor.
- State how to obtain medical supplies and carry out dressing changes, IV access site care, and other prescribed regimens.
- Identify the benefits and expected outcome of regular Ig replacement therapy.
- State rationale for prophylactic use of acetaminophen and diphenhydramine before treatment begins.
- State the rationale for prehydration on the day before infusion.
- Demonstrate how to prepare regular Ig replacement therapy.
- Demonstrate how to administer regular Ig replacement therapy.
- Demonstrate how to clean and maintain IV equipment, as applicable.
- Identify side effects and adverse effects of regular Ig replacement therapy.
- Demonstrate how to monitor for adverse effects of regular Ig replacement therapy.
- Describe to whom, how, and when to report adverse effects of regular Ig replacement therapy.
- Describe appropriate actions to take for adverse effects.
- Verbalize understanding of emergency measures for anaphylactic shock.
- Describe time periods associated with possible development of adverse reactions

ADLs, activities of daily living; IADLs, instrumental activities of daily living.

Epidemiology

Since the first cases of AIDS were reported in the United States in 1981, surveillance case definitions for HIV infection and AIDS have undergone several revisions (in 1985, 1987, 1993, 2008, and 2014) in response to diagnostic advances. Criteria for a confirmed case of HIV infection can be met by either laboratory evidence or clinical evidence but laboratory evidence, usually obtained through blood tests, is preferred over clinical evidence (e.g.,

patient signs and symptoms). A case of HIV infection can be classified in one of five HIV infection stages (0, 1, 2, 3, or unknown). Stage 0 indicates early HIV infection, inferred from laboratory testing; stages 1, 2, and 3 are based on the CD4⁺ T-lymphocyte count; while cases with no information on CD4⁺ T-lymphocyte count or percentage are classified as stage unknown (Centers for Disease Control and Prevention [CDC], 2014). See [Table 32-2](#) for further explanation of stages.

In July 2015, the Obama White House released the *National HIV/AIDS Strategy for the United States: Updated to 2020*. This document has four Strategic Goals, which include reducing new infections; increasing access to care and improving health outcomes for people living with HIV; reducing HIV-related health disparities and health inequities; and achieving a more coordinated national response to the HIV epidemic. The strategy is being updated by the Office of HIV/AIDS and Infectious Disease Policy (OHAIDP, 2019).

According to the CDC, 1,006,691 persons aged 13 years and older are living with HIV infection, and an additional 534,515 are living with AIDS in the United States (CDC, 2019a). From 2012 through 2016, the rate of diagnoses of HIV infection decreased, although the annual number of diagnoses remained stable as survival rates have increased. The incidence was 11.8 per 100,000 persons for 2017, with men accounting for 81% of all HIV infection diagnoses (CDC, 2019a). The incidence per 100,000 persons was 41.1 for Blacks/African Americans, followed by 16.1 for Hispanics/Latinos, and 12.6 for persons of multiple races. The highest rate (32.9%) was for persons aged 25 to 29 years, followed by 28.7% for persons aged 20 to 24 years. Male-to-male sexual contact (MSM) (70%, including 3% male-to-male sexual contact and injection drug use) and those attributed to heterosexual contact (24%) accounted for approximately 94% of diagnosed HIV infections. Rates within the United States were 16.1% in the South, 10.6% in the Northeast, 9.4% in the West, and 7.4% in the Midwest (CDC, 2019a). Data about infections in transgender persons were not collected.

TABLE 32-2 HIV Infection Stages 1, 2, and 3 Based on Age-Specific Laboratory Data

Stage	Age on Date of CD4 ⁺ T-lymphocyte Test					
	<1 yr		1–5 yrs		≥6 yrs	
	Cells/IL	%	Cells/IL	%	Cells/IL	%
1	≥1500	≥34	≥1000	≥30	≥500	≥26
2	750–1499	26–33	500–999	22–29	200–499	14–25
3	<750	<26	<500	<22	<200	<14

If a stage-3-defining opportunistic illness has been diagnosed, then the stage is 3 regardless of CD4⁺ T-lymphocyte test.

Stage-3-Defining Opportunistic Illnesses in HIV Infection

- Bacterial infections, multiple or recurrent (only among children aged less than 6 yrs)
- Candidiasis of bronchi, trachea, or lungs
- Candidiasis of esophagus
- Cervical cancer, invasive (only in persons aged 6 yrs or older)
- Coccidioidomycosis, disseminated or extrapulmonary
- Cryptococcosis, extrapulmonary
- Cryptosporidiosis, chronic intestinal (>1 mo duration)
- Cytomegalovirus disease (other than liver, spleen, or nodes), onset at age >1 mo
- Cytomegalovirus retinitis (with loss of vision)
- Encephalopathy attributed to HIV
- Herpes simplex: chronic ulcers (>1 mo duration) or bronchitis, pneumonitis, or esophagitis (onset at age >1 mo)
- Histoplasmosis, disseminated or extrapulmonary
- I sporiasis, chronic intestinal (>1 mo duration)
- Kaposi sarcoma
- Lymphoma, Burkitt (or equivalent term)
- Lymphoma, immunoblastic (or equivalent term)
- Lymphoma, primary, of brain
- Mycobacterium avium complex or Mycobacterium kansasii, disseminated or extrapulmonary
- Mycobacterium tuberculosis of any site, pulmonary (only in persons aged 6 yrs or older) disseminated, or extrapulmonary
- Mycobacterium, other species or unidentified species, disseminated or extrapulmonary
- Pneumocystis jirovecii (previously known as "Pneumocystis carinii") pneumonia (PCP)
- Pneumonia, recurrent (only in persons aged 6 yrs or older)
- Progressive multifocal leukoencephalopathy
- Salmonella septicemia (recurrent)
- Toxoplasmosis of brain, onset at age >1 mo
- Wasting syndrome attributed to HIV

Adapted from Centers for Disease Control and Prevention (CDC). (2014). Revised surveillance case definition for HIV infection—United States, 2014. MMWR. Recommendations and Reports: Morbidity and Mortality Weekly Report. Recommendations and Reports, 63(RR-03), 1–10.

According to the World Health Organization ([WHO], 2019), HIV through its morbid consequence of AIDS has claimed more than 32 million lives globally; in 2018, 770,000 people died from HIV-related causes. Globally, there were approximately 37.9 million people living with HIV at the end of 2018, with 1.7 million people becoming newly infected in 2018. The WHO African Region is the most affected region, with 25.7 million people living with HIV in 2018. The African region also accounts for almost two thirds of the global total of new HIV infections. Between 2000 and 2018, new HIV infections fell by 37%, and HIV-related deaths fell by 45% with 13.6 million lives saved due to patients receiving effective medication during that same period. The decrease in infection rates and deaths was the result of many efforts by national HIV programs supported by a range of funding partners (WHO, 2019).

HIV Transmission

Inflammation and breaks in the skin or mucosa result in the increased probability that an HIV exposure will lead to infection. Human immune deficiency virus type 1 (**HIV-1**) is transmitted in body fluids (blood, seminal fluid, vaginal secretions, amniotic fluid, and breast milk) that contain infected

cells. Higher amounts of HIV and infected cells in the body fluid are associated with the probability that the exposure will result in infection. Mother-to-child transmission of HIV-1 may occur in utero, at the time of delivery, or through breast-feeding, but most perinatal infections are thought to occur during labor and delivery. HIV is not transmitted through casual contact (see [Chart 32-2](#)).

Blood and blood products can transmit HIV to recipients. However, the risk associated with transfusions has been virtually eliminated as a result of voluntary self-deferral, completion of a detailed health history, extensive testing, heat treatment of clotting factor concentrates, and more effective virus inactivation methods. Donated blood is tested for antibodies to HIV-1, human immune deficiency virus-2 (retrovirus identified in 1986 in patients with AIDS in Western Africa), and the p24 antigen; since 1999, additional testing has been performed.

Chart 32-2 RISK FACTORS

Risks Associated with HIV Infection

- Sharing infected injection drug use equipment
- Having sexual relations with infected persons (both genders)
- Infants born to mothers with HIV infection or who are breast-fed by HIV-infected mothers
- People who received organ transplants, HIV-infected blood, or blood products (especially between 1978 and 1985)

Adapted from Centers for Disease Control and Prevention (CDC). (2019a). HIV Surveillance Report, 2017. Retrieved on 10/21/2019 at: www.cdc.gov/hiv/library/reports/hiv-surveillance.html

Gerontologic Considerations

According to the CDC, 91,127 persons between the ages of 60 and 64 years are living with HIV infection, and an additional 58,526 persons are living with AIDS. For persons aged 65 years and older, 82,046 persons are living with HIV infection and an additional 52,995 persons are living with AIDS (CDC, 2019a). Many were diagnosed with HIV in their younger years and are benefitting from effective treatment. However, thousands of older adults unknowingly become infected with HIV every year. Older adults are less likely than younger people to get tested. Thirty-five percent of persons aged 50 and older were diagnosed with late-stage infection or AIDS, indicating a failure to screen for HIV in this population. Although this rate is an improvement from 2011, when it was 42%, it is still high. Late diagnosis has an adverse impact on

treatment effectiveness (CDC, 2019b). Medicare will pay for annual screenings up until age 65 but there needs to be a special indication (such as unprotected sex with an infected partner) for a person to be screened after that age in order for Medicare to cover the cost of testing (CDC, 2019a).

Persons are often embarrassed to share that they have engaged in activities associated with HIV infection and so might not disclose this to the health care provider. The provider needs to use other cues, such as the presence of other sexually transmitted diseases, and recommend HIV testing. Providers may bring societal age and gender biases to their perceptions of patient sexuality, which influence their communication about sexual health with older adult patients. For some providers, assumptions that discussions about sexual health will offend their patients, as well as their own discomfort with talking to older adults about sex, inhibit them from assessing risk factors that may indicate a need for HIV and STI testing.

Comorbidities in older persons with HIV/AIDS include type 2 diabetes; non-AIDS cancerous diseases; cardiovascular disease; osteoporosis; and depression (Guaraldi, Malagoli, Calcagno, et al., 2018). Due to the high number of chronic illnesses, polypharmacy along with drug interactions becomes a major clinical challenge. Loneliness has been defined as the distress that exists between actual and desired relationships and is different from the concept of aloneness or living alone. Loneliness was studied in older persons living with HIV/AIDS by Greene and colleagues, who found that 58% of the participants reported at least some loneliness. Compared to participants who did not report feeling lonely, those who did report loneliness were more likely to smoke, misuse alcohol, or use illicit drugs. They also reported less physical social supports in place, depressive symptoms, poor or fair health-related quality of life, and functional impairment in one or more Instrumental Activities of Daily Living (IADLs) (Greene, Hessol, Perissinotto, et al., 2018). Loneliness has been associated with increased mortality in the general population.

Prevention of HIV Infection

Nurses need to participate in efforts to prevent HIV infection by educating patients how to eliminate or reduce risks associated with HIV infection and AIDS, particularly in younger adults, in order to promote a healthy lifestyle and longevity (Eliopoulos, 2018). HIV is transmitted through the exchange of some infected body fluids (see [Chart 32-2](#)). While behavioral interventions such as encouraging the use of condoms are highly effective in reducing the transmission of HIV, the HIV-negative person must be motivated and have the freedom to choose to use the method. In some situations, however, that freedom is absent. For example, a lack of freedom exists in a discordant couple (those in which only one partner has HIV) if the husband refuses to use

condoms and the wife's cultural and religious beliefs require sexual activity with her husband. In this context, **pre-exposure prophylaxis (PrEP)** might be appropriate. PrEP involves taking one pill containing two HIV medications (tenofovir disoproxil fumarate 300 mg and emtricitabine 200 mg) daily in order to avoid the risk of sexual HIV acquisition in adults and adolescents age 12 and older. HIV status should be checked every 3 months to be sure that the person has not become infected. The ultimate goal of PrEP is to reduce the acquisition of HIV infection with its resulting morbidity, mortality, and cost to individuals and society (CDC, 2019c). For persons taking ART as prescribed and achieving and maintaining viral suppression, there is no risk of transmitting HIV through sex. Since PrEP does not prevent other sexually transmitted infections, which are increasing (CDC, 2019d), it is important for couples to also use a condom.



Preventive Education

Prevention of HIV infection is achieved through: (a) behavioral interventions have been effective in reducing the risk of acquiring or transmitting HIV by ensuring that people have the information, motivation, and skills necessary to reduce their risk; (b) HIV testing, because most people change behaviors to protect their partners if they know they are infected with HIV; and (c) linkage to treatment and care, which enables individuals with HIV to live longer, healthier lives and reduce their risk of transmitting HIV (CDC, 2019e). The CDC (2019f), through the HIV/AIDS Prevention Research Synthesis Project, provides information about evidence-based behavioral interventions that can be used in a number of settings with targeted populations. Strategies to protect against infection are outlined in [Chart 32-3](#). Other than abstinence, consistent and correct use of condoms (see [Chart 32-4](#)) is the only effective method to decrease the risk of sexual transmission of HIV infection. When latex male condoms are used consistently and correctly during vaginal or anal intercourse, they are highly effective in preventing the sexual transmission of HIV. Nonlatex condoms made of natural materials such as lambskin are available for people with latex allergy but will not protect against HIV infection. A male condom should be used for oral contact with the penis, and a dental dam (a flat piece of latex used by dentists to isolate a tooth for treatment) or an altered condom can be used for oral contact with the vagina or rectum. Voluntary medical male circumcision (VMMC) is 50% to 60% effective at preventing the acquisition of HIV infection. This is a key intervention in generalized epidemic settings with high HIV prevalence and low male circumcision rates (Ensor, Davies, Rai, et al., 2019).

Chart 32-3 HEALTH PROMOTION

Protecting Against HIV Infection

All patients should be advised to:

- Abstain from exchanging sexual fluids (semen and vaginal fluid).
- Reduce the number of sexual partners to one.
- Always use latex condoms. If the patient is allergic to latex, nonlatex condoms should be used; however, they will not protect against HIV infection.
- Not reuse condoms.
- Avoid using cervical caps or diaphragms without using a condom as well.
- Always use dental dams for oral–genital or anal stimulation.
- Avoid anal intercourse, because this practice may injure tissues; if not possible, use lubricant—there are water and silicone-based products designed for anal sex.
- Avoid manual–anal intercourse (“fisting”).
- Avoid sharing needles, razors, toothbrushes, sex toys, or blood-contaminated articles.
- Consider PrEP if regularly engage in high-risk behaviors.
- Use needle-exchange programs (as appropriate) and do not share drug-using equipment.

Patients who are HIV seropositive should also be advised to:

- Take ART regularly to achieve viral suppression.
- Inform previous, present, and prospective sexual and drug-using partners of their HIV-positive status. If the patient is concerned for their safety, advise the patient that many states have established mechanisms through the public health department in which professionals are available to notify exposed contacts.
- Avoid having unprotected sex with another HIV-seropositive person. Cross-infection with that person's HIV can increase the severity of infection.
- Not donate blood, plasma, body organs, or sperm.

ART, antiretroviral therapy; PrEP, pre-exposure prophylaxis.

Chart 32-4 PATIENT EDUCATION

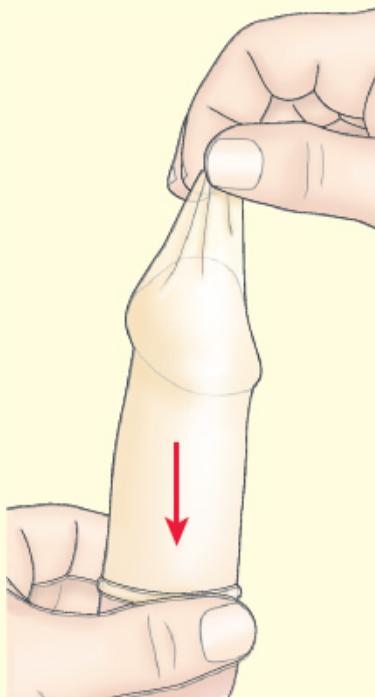
The Correct Way to Use a Male Condom

The nurse instructs the patient to:

- Put on a new condom before any kind of sex.
- Hold the condom by the tip to squeeze out the air.



- Unroll the condom all the way over the erect penis.



- Have sex.
- Hold the condom so it cannot come off the penis.
- Pull out.

- Use a new condom if you want to have sex again or if you want to have sex in a different place (e.g., in the anus and then in the vagina).

Note: Keep condoms cool and dry. Never use skin lotions, baby oil, petroleum jelly, or cold cream as lubricants; the oil in these products will cause the latex condom to break. Products made with water (such as K-Y jelly or glycerin) are safer to use.

The polyurethane female condom, which is an effective contraceptive, provides a physical barrier that prevents exposure to genital secretions containing HIV, such as semen and vaginal fluid, and is inserted by the woman (see [Chapter 50](#)). Other safe and effective woman-controlled methods such as microbicides remain elusive although clinical trials continue globally. Microbicides are gels, films, or suppositories that can kill or neutralize viruses and bacteria; vaginal and rectal microbicides are being researched to see if they can prevent sexual transmission of HIV.

Total abstinence from addictive drugs might not be a realistic short-term goal. The harm reduction framework uses practical strategies and ideas aimed at reducing negative consequences associated with drug use. It is also a movement for social justice built on a belief in, and respect for, the rights of people who use drugs. Using a harm reduction framework, the nurse works with people who inject drugs to assist them to increase their healthy behaviors. Sharing of drug-use equipment is a high-risk behavior for a number of bloodborne infections and should be avoided. Syringe services programs are also referred to as syringe exchange programs and needle exchange programs. Although the services they provide may vary, they are community-based programs that provide access to sterile needles and syringes, facilitate safe disposal of used syringes, and provide and link to other important services and programs (CDC, 2019g). Therefore, under the harm reduction framework, participation in needle exchange programs is encouraged since they do not promote increased drug use; on the contrary, they have been found to decrease the incidence of bloodborne infections in people who inject drugs. Nurses should refer patients to accessible needle exchange programs whenever available.

Related Reproductive Education

Because HIV infection in women often occurs during the childbearing years, family planning issues need to be addressed. Attempts to achieve pregnancy by couples in which only one partner has HIV (known as discordant couples) expose the unaffected partner to the virus. Efforts at artificial insemination using processed semen from an HIV-infected partner continue. Studies are needed, because HIV has been found in the spermatozoa of patients with HIV infection, and it is possible that HIV can replicate in the male germ cell. Women considering pregnancy need to have accurate information about the

risks of transmitting HIV infection to themselves, their partner, and their future children, and about the benefits of taking ART to reduce perinatal HIV transmission. Results from the Evidence for Contraceptive Options and HIV Outcomes (ECHO) Trial Consortium found that, among African women seeking effective contraception and living in areas of high HIV incidence, there was no substantial difference in HIV risk among the methods evaluated (DMPA-IM, a copper IUD, and a levonorgestrel implant) and all methods were safe and highly effective (ECHO Trial Consortium, 2019). HIV-infected women in resource-rich settings should be instructed not to breast-feed their infants, because HIV is transmitted through breast milk.

Prevention in Lesbian, Gay, Bisexual, Transgender, and Queer Persons

Many health care providers are insufficiently prepared to meet the unique health needs of those who identify as lesbian, gay, bisexual, transgender, and queer (LGBTQ). Young gay men, in particular, are at higher risk for contracting HIV (CDC, 2019a). At the same time, these sexual and gender minorities experience significant challenges due to family rejection, lack of social support, stigma, isolation, minority stress, as well as abuse and harassment. Nurses need to be culturally competent in order to be effective in educating these unique populations about prevention methods (see [Chapter 54](#)).

Reducing the Risk of Transmission to Health Care Providers

Prevention of HIV and AIDS among health care providers involves the use of standard precautions to prevent exposure, using post-exposure prophylaxis when exposure does occur and hopefully, in the near future, the use of a vaccine.

Standard Precautions

Implementation of appropriate hand hygiene measures (see [Chapter 66, Chart 66-1](#) for further information on hand hygiene methods) remains the most effective measure to prevent transmission of organisms. To reduce the risk of exposure of health care workers to HIV, the CDC developed standard precautions (see [Chart 32-5](#); also see [Chapter 66, Chart 66-2](#)) which are designed to reduce the risk of transmission of pathogens. Standard precautions are used when working with all patients in all health care settings, regardless of their diagnosis or presumed infectious status.

Post-Exposure Prophylaxis for Health Care Providers

Post-exposure prophylaxis (PEP) includes taking antiretroviral medicines as soon as possible, but no more than 72 hours (3 days) after possible HIV exposure; three drugs are prescribed. Health care workers who are exposed to a needle stick involving HIV-infected blood in a health care setting have a 0.3% risk of becoming HIV infected; the risk of infection due to occupational exposure is very low (CDC, 2018a).

Occupational exposure is an urgent medical concern and should be managed immediately after possible exposure—the sooner the better; every hour counts (CDC, 2018a). The CDC suggests that these post-exposure guidelines be followed after occupational and other exposures such as sexual assault. [Chart 32-6](#) provides the strategies and emphasizes the need for quick action. See the Resources section for the phone number for the Health Resources and Service Administration (HRSA) Post-exposure Prophylaxis Hotline which is answered by a health care provider.

Chart 32-5

Recommendations for Standard Precautions

- **Hand hygiene:** Use after touching blood, body fluids, secretions, excretions, or contaminated items; immediately after removing gloves; and between patient contacts.
- **Personal protective equipment:**
 - *Gloves:* Use for touching blood, body fluids, secretions, excretions, and contaminated items, and for touching mucous membranes and nonintact skin.
 - *Gown:* Use during procedures and patient care activities when contact of clothing/exposed skin with blood or body fluids, secretions, and excretions is anticipated.
 - *Mask, eye protection (goggles), face shield¹:* Use during procedures and patient care activities likely to generate splashes or sprays of blood, body fluids, and secretions, especially suctioning or endotracheal intubation.
- **Soiled patient care equipment:** Handle in a manner that prevents transfer of microorganisms to others and to the environment; wear gloves if visibly contaminated; and perform hand hygiene.
- **Environmental control:** Develop procedures for routine care, cleaning, and disinfection of environmental surfaces, especially frequently touched surfaces in patient care areas.
- **Textiles and laundry:** Handle in a manner that prevents transfer of microorganisms to others and to the environment.
- **Needles and other sharps:** Do not recap, bend, break, or hand manipulate used needles; if recapping is required, use a one-handed scoop technique only; use safety features when available; and place used sharps in a puncture-resistant container.
- **Patient resuscitation:** Use mouthpiece, resuscitation bag, and other ventilation devices to prevent contact with mouth and oral secretions.
- **Patient placement:** Prioritize for single-patient room if patient is at increased risk for transmission, is likely to contaminate the environment, does not maintain appropriate hygiene, or is at increased risk for acquiring infection or developing adverse outcome following infection.
- **Respiratory hygiene/cough etiquette** (source containment of infectious respiratory secretions in symptomatic patients, beginning at initial point of encounter, such as triage and reception areas in emergency departments and provider offices): Instruct symptomatic people to cover mouth and nose when sneezing or coughing, use tissues and dispose in no-touch receptacle, observe hand hygiene after soiling of hands with respiratory secretions, and wear surgical mask if tolerated.

¹During aerosol-generating procedures on patients with suspected or proven infections transmitted by respiratory aerosols (e.g., severe acute

respiratory syndrome), wear a fit-tested N95 or higher respirator in addition to gloves, gown, and face/eye protection.

Adapted from Centers for Disease Control and Prevention (CDC). (2018a).

Updated U.S. Public Health Service guidelines for the management of occupational exposures to HIV and recommendations for postexposure prophylaxis. Retrieved on 10/27/2019 at: stacks.cdc.gov/view/cdc/20711

Vaccination

In the face of a global pandemic, the search for an effective vaccine against the HIV remains an urgent priority. The first U.S. government-sponsored phase I trial of an HIV vaccine was launched in 1987 and intensive efforts worldwide using a variety of strategies continue (NIAID, 2019b).

Chart 32-6

Post-HIV Exposure Prophylaxis for Health Care Providers

If you sustain an occupational exposure to HIV, take the following actions immediately:

- Alert your supervisor and initiate the occupational exposure reporting system used in the setting.
- Determine the HIV status of the exposure source (i.e., patient) when possible to guide appropriate use of HIV post-exposure prophylaxis (PEP). Use rapid-testing if the HIV status of the patient is unknown. Check laws in your state as you proceed to determine HIV status of the source patient.

Get counseling at the time of exposure and at follow-up appointments:

- Exposed health care providers (HCP) are advised to use precautions (e.g., use of barrier contraception and avoidance of blood or tissue donations, pregnancy, and, if possible, breast-feeding) to prevent secondary transmission, especially during the first 6 to 12 weeks after exposure.
- For exposures for which PEP is prescribed, HCPs are informed regarding the following:
 - Possible drug toxicities (e.g., rash and hypersensitivity reactions that could imitate acute HIV seroconversion and the need for monitoring)
 - Possible drug interactions
 - The need for adherence to PEP regimens

Undergo early reevaluation after exposure:

- Regardless of whether an HCP is taking PEP, reevaluation of exposed HCP within 72 hours after exposure is strongly recommended, as additional information about the exposure or the source patient may be available.

Follow up with HIV testing and appointments. At a minimum, this follow-up should include:

- HIV testing at baseline and at 6 weeks, 12 weeks, and 6 months after exposure; alternatively, if the HCP is certain that a fourth-generation combination HIV p24 antigen–HIV antibody test is being utilized, then HIV testing could be performed at baseline, 6 weeks after exposure, and 4 months after exposure
- Complete blood counts and renal and hepatic function tests (at baseline and 2 weeks after exposure; further testing may be indicated if abnormalities are detected)

Note: All HIV testing results should preferably be given to the exposed HCP during face-to-face appointments.

Adapted from Centers for Disease Control and Prevention (CDC). (2018a). Updated U.S. Public Health Service guidelines for the management of

Pathophysiology

Because HIV is an infectious disease, it is important to understand how HIV-1 integrates itself into a person's immune system and how the immune response plays a pivotal role in the course of HIV disease. This knowledge is also essential for understanding medication therapy and vaccine development. Viruses are intracellular parasites. HIV is in the subfamily of lentiviruses and is a **retrovirus** because it carries its genetic material in the form of ribonucleic acid (RNA) rather than deoxyribonucleic acid (DNA) (Norris, 2019).

Physiology/Pathophysiology

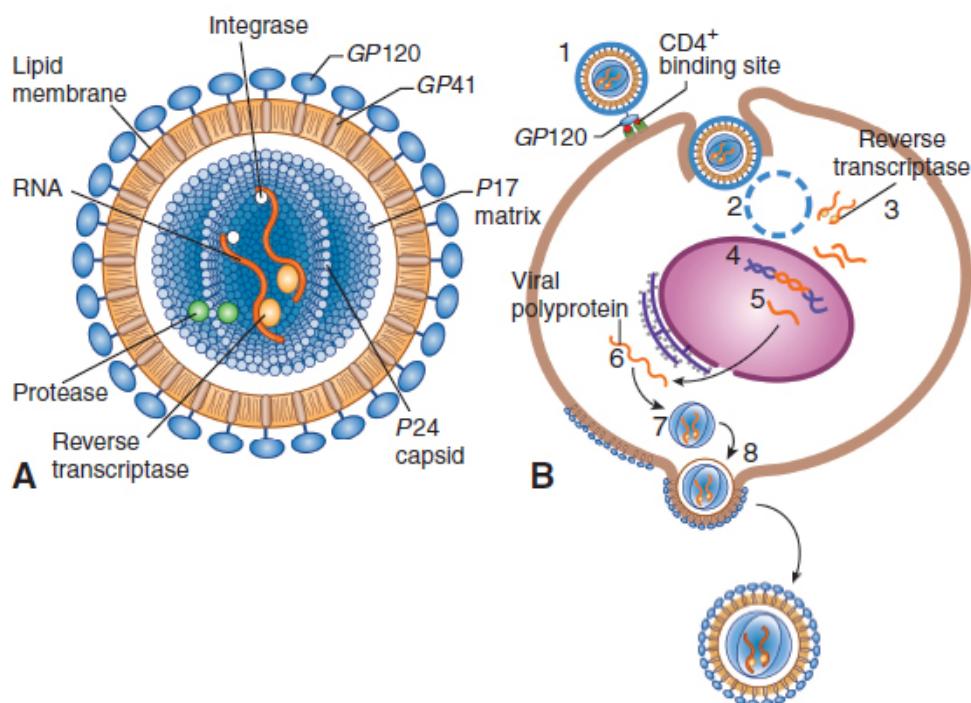


Figure 32-1 • Structure of HIV-1 virus. The virus surrounded by a lipid envelope. Reprinted with permission from Norris, T. L., & Lalchandani, R. (2019). *Porth's pathophysiology: Concepts of altered health states* (10th ed., Fig. 12.7 (left side)). Philadelphia, PA: Wolters Kluwer.

Two genetically different but closely related forms of HIV (HIV-1 and HIV-2) have been identified. Globally, it has been estimated that one to two million individuals have HIV-2, including people with HIV-1/HIV-2 dual infection.

The course of illness is slower when infection is caused by HIV-2, which seems to be more common in Western Africa compared to HIV-1, which is more common in other regions of the globe (Panel, 2019). Blood tests may be used to screen for both forms of HIV. As shown in [Figure 32-1](#), HIV consists of a viral core, containing viral RNA, which is surrounded by an envelope consisting of protruding glycoproteins.

All viruses target specific cells. HIV targets cells with CD4⁺ receptors, which are expressed on the surface of T lymphocytes, monocytes, dendritic cells, and brain microglia. Mature T cells (T lymphocytes) are composed of two major subpopulations that are defined by cell surface receptors of CD4⁺ or CD8⁺. Approximately two thirds of peripheral blood T cells are CD4⁺, and approximately one third is CD8⁺. Most people have about 700 to 1000 CD4⁺ cells/mm³, but a level as low as 500 cells/mm³ can be considered within normal limits.

The HIV life cycle is complex. [Figure 32-2](#) illustrates the seven stages of the life cycle and the various classes of antiretroviral medications that target each specific stage (HIV Information, 2020; Norris, 2019).

In resting (nondividing) CD4⁺ cells, HIV survives in a latent state as an integrated **provirus** that produces few or no viral particles. These resting CD4⁺ T cells can be stimulated to produce new particles if something activates them, such as another infection. When a resting T cell that harbors this integrated DNA (also known as provirus) becomes activated against HIV or other microbes, the cell begins to produce new copies of both RNA and viral proteins. Consequently, whenever the infected CD4⁺ cell is activated, HIV replication and budding occur, which can destroy the host cell. Newly formed HIV released into the blood can infect other CD4⁺ cells.

HIV-1 mutates quickly, at a relatively constant rate, with about 1% of the virus's genetic material changing annually. HIV-1 exhibits substantial genetic diversity, and several different genotypes of HIV-1 exist throughout the world. There is a major group (group M), which consists of subtypes A through L, and a more diverse collection of outliers, which has been referred to as groups N and O. Subtype B HIV-1 viruses predominate in the Western world; this genetic variation is one of the major reasons why effective vaccine development has been such a challenge.

Stages of HIV Infection

There are five stages of HIV infection based on clinical history, physical examination, laboratory evidence (CDC, 2014), signs and symptoms, and associated infections and malignancies. See [Table 32-2](#).

The period from infection with HIV to the development of HIV-specific antibodies is known as **primary infection or acute HIV infection** (previously known as the window period) and is part of stage 0 (CDC, 2014). Acute HIV

infection is the interval between the appearance of detectable HIV RNA and the first detection of antibodies. Initially, persons test negative on the HIV antibody blood test, although they are not only infected, but also highly contagious because their viral loads are very high. About 40% to 80% of patients develop clinical symptoms of a nonspecific viral illness (e.g., fever, fatigue, or rash) lasting 1 to 2 weeks. After 2 to 3 weeks, antibodies to the glycoproteins of the HIV envelope can be detected in the sera of people infected with HIV, but most of these antibodies lack the ability to totally control the virus. By the time neutralizing antibodies can be detected, HIV-1 is firmly established in the host.

Primary or acute infection is characterized by high levels of viral replication, widespread dissemination of HIV throughout the body, and destruction of CD4⁺ T cells, which leads to dramatic drops in CD4⁺ T-cell counts (normally 500 to 1500 cells/mm³ of blood). The host responds to the HIV infection through a CD4⁺ T-cell response that causes other immune cells, such as CD8⁺ lymphocytes, to increase their killing of infected, virus-producing cells. The body produces antibody molecules in an effort to contain the free HIV particles (outside cells) and assist in their removal. During this stage, the virus is widely disseminated in lymphoid tissue, and a **latent reservoir** within resting memory CD4⁺ T cells is created.

During stage 1, the amount of virus in the body after the initial immune response subsides results in a **viral set point** which reflects an equilibrium between HIV levels and the immune response. Untreated, this set point can last for years and is inversely correlated with disease prognosis. The higher the viral set point, the poorer the prognosis. After the viral set point is reached, a chronic stage persists in which the immune system cannot eliminate the virus despite its best efforts. This set point varies greatly from patient to patient and dictates the subsequent rate of disease progression; on average, 8 to 10 years can pass before a major HIV-related complication develops. In this prolonged, chronic stage (stage 1), patients feel well and have few, if any, symptoms, which is why this stage had been referred to as asymptomatic. Apparent good health continues because CD4⁺ T-cell levels remain high enough to preserve immune defensive responses, but, over time, the number of CD4⁺ T cells decrease.

The HIV Life Cycle

HIV medicines in seven drug classes stop (stop) HIV at different stages in the HIV life cycle.

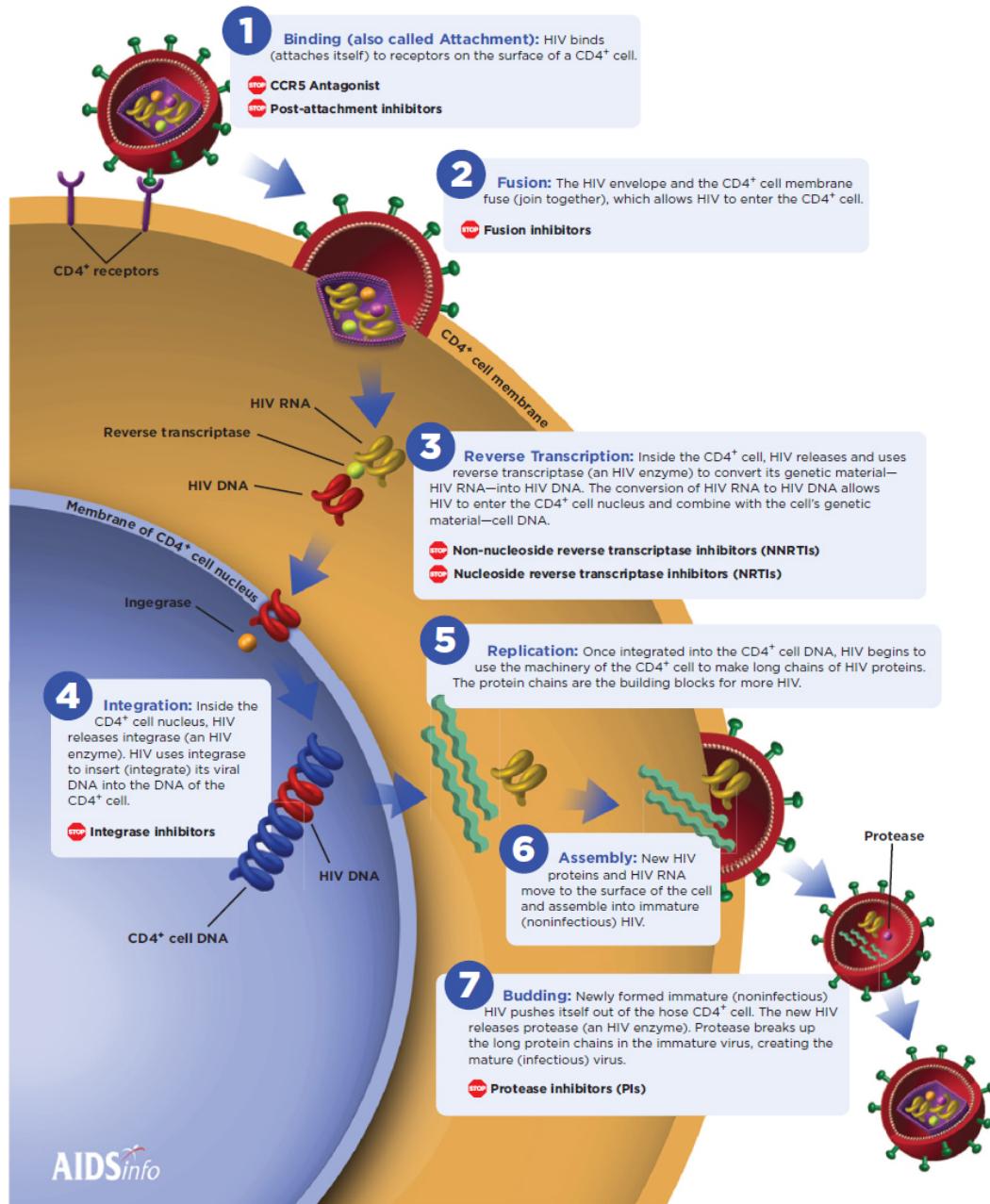


Figure 32-2 • The HIV life cycle. Adapted from HIV Information, 2020.

Stage 2 occurs when CD4⁺ T-lymphocyte cells decrease to between 200 and 499 cells/mm³ and had previously been referred to as the symptomatic stage. Stage 3 is diagnosed when the count drops below 200 cells/mm³ and the person has AIDS. HIV disease progression is classified from less to more severe; once a case is classified into a surveillance severity stage, it cannot be

reclassified into a less severe stage even if the CD4⁺ T lymphocytes increase, which often occurs when a person receives ART. A stage 3, AIDS, diagnosis has implications for services (e.g., disability benefits, housing, and food stamps), because these programs are often linked to living with severe immune dysfunction.

Assessment and Diagnostic Findings in HIV Infection

During the first stage of HIV infection, the patient may have no symptoms or generalized signs and symptoms such as fatigue or skin rash. Patients who are in later stages of HIV infection may have a variety of symptoms related to their immunosuppressed state. The staging system requires laboratory evidence of HIV infection in order to diagnose HIV or AIDS (see [Table 32-2](#)).

HIV Tests

Several tests are used to diagnose HIV infection, and others are used to determine the stage and severity of the infection. A serologic testing algorithm for recent HIV seroconversion (STARHS) analyzes HIV-positive blood samples to determine whether an HIV infection is recent or has been ongoing. There are three types of HIV diagnostic tests: antibody tests, antigen/antibody tests, and nucleic acid (RNA) tests. Antibody tests detect antibodies, not HIV itself, while antigen and RNA tests directly detect HIV. The CDC recommends tests for HIV antigens and HIV nucleic acid because studies from high-risk populations found that antibody testing alone might miss a considerable percentage of HIV infections detectable by virologic tests, especially during stage 0.

Blood tests can detect HIV infection sooner after exposure compared to oral fluid tests because the level of antibody in blood is higher than it is in oral fluid. Likewise, antigen/antibody and RNA tests detect infection in blood before antibody tests. Some newer antigen/antibody laboratory tests can sometimes find HIV as soon as 3 weeks after exposure to the virus.

Follow-up testing is performed if the initial test result is positive to ensure a correct diagnosis. These tests include:

- antibody *differentiation tests*, which distinguishes HIV-1 from antibodies
- *HIV-1 nucleic acid tests*, which looks for the virus RNA directly

Table 32-3 identifies common blood tests used for screening.

Since negative perceptions and judgments associated with being HIV infected continue to persist, stigma remains one of the biggest social challenges (Glynn, Llabre, Lee, et al., 2019). When the result of the HIV antibody test is received, it is carefully explained to the patient in private (see [Chart 32-7](#)). All test results are confidential. Education and counseling about

the test result and about preventing transmission are essential. The patient's psychological response to a positive test result may include feelings of panic, depression, and hopelessness. The social and interpersonal consequences of a positive test result can be devastating. The patient may lose their sexual partner, housing, and their job because of disclosure. They may be subjected to physical abuse and, although illegal, experience discrimination in employment as well as social ostracism. For these reasons and others, patients who test positive may need ongoing counseling as well as referrals for social, financial, medical, and psychological support services. The HIV Care Continuum (CDC, 2019h) starts when patients receive their positive HIV blood test results. They must be connected to health care services to evaluate their stage of HIV infection and start treatment.

TABLE 32-3 HIV Screening Blood Tests

Laboratory Test	Indications
HIV-1/HIV-2 immunoassay	Tests for both HIV-1 and HIV-2 antibodies
HIV-1-HIV-2 antigen/antibody combination immunoassay	Tests for both virus (antigen) and antibody for both HIV-1 and HIV-2
HIV-1 differentiation assay	Differentiates HIV-1 from HIV-2
HIV-1 nucleic acid amplification test	Tests directly for virus
HIV-1 p24 antigen	Tests directly for virus

HIV, human immune deficiency virus.

Adapted from Centers for Disease Control and Prevention. (2018b). Quick reference guide: Recommended laboratory HIV testing algorithm for serum or plasma specimens. Retrieved on 10/28/2019 at: stacks.cdc.gov/view/cdc/50872

Chart 32-7

HIV Test Results: Implications for Patients

Interpretation of Positive Antibody Test Results

- Antibodies to HIV are present in the blood (the patient has been infected with the virus, and the body has produced antibodies).
- HIV is active in the body, and the patient can transmit the virus to others.
- Despite HIV infection, the patient does not necessarily have AIDS.
- The patient is not immune to HIV (the antibodies do not indicate immunity).

Interpretation of Negative Test Results

- Antibodies to HIV are not present in the blood at this time, which can mean that the patient has not been infected with HIV or, if infected, the body has not yet produced antibodies (stage 0).
- The patient should continue taking precautions. The test result does not mean that the patient is immune to the virus, nor does it mean the patient is not infected; it just means that the body may not have produced antibodies yet. If viral test used, a negative result is more consistent with the conclusion that the patient is uninfected.

AIDS, acquired immune deficiency syndrome; HIV, human immune deficiency virus.

Adapted from Centers for Disease Control and Prevention (CDC). (2018b). Quick reference guide: Recommended laboratory HIV testing algorithm for serum or plasma specimens. Retrieved on 10/28/2019: stacks.cdc.gov/view/cdc/50872

Patients whose test results are seronegative may develop a false sense of security, possibly resulting in continued high-risk behaviors or feelings that they are immune to the virus. These patients may need ongoing counseling to help modify high-risk behaviors and to encourage returns for repeated testing. Other patients may experience anxiety regarding the uncertainty of their status. In addition to screening for HIV, patients should be screened for other bloodborne coinfections such as hepatitis; other STIs such as syphilis; and other infections associated with T-cell immunity such as tuberculosis (TB). The rates of coinfection of hepatitis C and HIV are approximately 25% (Starbird, Hong, Sulkowski, et al., 2020).

Staging

Two surrogate markers are used routinely to assess immune function and level of HIV viremia: CD4⁺ T-cell count (CD4⁺ count) and plasma HIV RNA (viral load). CD4⁺ count should be measured in all patients at entry into care. **Viral**

load tests use target amplification methods to quantify HIV RNA or DNA levels in the plasma. Target amplification methods include reverse transcriptase–**polymerase chain reaction** (RT-PCR) and nucleic acid sequence–based amplification (NAT). A widely used viral load test measures plasma HIV RNA levels. Currently, these tests are used to track viral load and response to treatment of HIV infection. RT-PCR is also used to detect HIV in high-risk seronegative people before antibodies are measurable, to confirm a positive EIA result, and to screen neonates. HIV culture or quantitative plasma culture and plasma viremia are additional tests that measure viral burden, but they are used infrequently. Viral load is a better predictor of the risk of HIV disease progression than the CD4⁺ count. The lower the viral load, the longer the time to AIDS diagnosis and the longer the survival time.

Treatment of HIV Infection

The U.S. Department of Health and Human Services Panel on Antiretroviral Guidelines for Adults and Adolescents (Panel) (2019) is composed of HIV specialists from across the country who regularly meet to review the latest scientific evidence. The CD4⁺ count serves as the major laboratory indicator of immune function and prophylaxis for opportunistic infections, and is the strongest predictor of subsequent disease progression and survival (Panel, 2019). New drugs offer strategies based on the interaction between the life cycle of HIV and the host response, improvements in potency and activity even against multidrug-resistant viruses, dosing convenience, and tolerability. Goals of ART treatment include: (1) reduce HIV-associated morbidity and prolong the duration and quality of survival, (2) restore and preserve immunologic function, (3) maximally and durably suppress plasma HIV viral load, and (4) prevent HIV transmission (Panel, 2019). HIV suppression with ART may also decrease inflammation and immune activation thought to contribute to higher rates of cardiovascular and other end-organ damage. ART is recommended for all HIV-infected patients regardless of their viral load or CD4⁺ count (Panel, 2019). Optimal viral suppression is defined generally as a viral load persistently below the level of detection (HIV RNA less than 20 to 75 copies/mL, depending on the assay used). Providers, in partnership with patients, make treatment decisions based on a number of factors, including whether the patient has already taken ART or is ART-naive and the willingness of the patient to adhere to the lifelong treatment regimen.

Achieving viral suppression requires the use of combination ART regimens that generally include three active drugs from two or more drug classes (Panel, 2019). Viral load suppression to below limits of assay detection usually occurs within the first 12 to 24 weeks of taking ART. Predictors of virologic reduction include: (1) low baseline viremia; (2) high potency of the ART regimen; (3)

tolerability of the regimen; (4) convenience of the regimen; and (5) excellent adherence to the regimen.

More than 30 antiretroviral (ARV) drugs in seven classes are Food and Drug Administration (FDA)-approved for treatment of HIV infection. These seven classes target different stages of the HIV/host interaction. Examples include the nucleoside/nucleotide reverse transcriptase inhibitors (NRTIs) (stage 3), non-nucleoside reverse transcriptase inhibitors (NNRTIs) (stage 3), protease inhibitors (PIs) (stage 7), integrase strand transfer inhibitors (INSTIs) (stage 4), a fusion inhibitor (stage 2), a CCR5 antagonist (stage 1), and a CD4⁺ post-attachment inhibitor (stage 1) (see Fig. 32-2). In addition, some drugs are used as pharmacokinetic enhancers (or boosters) to improve the effectiveness of other ARV drugs (Panel, 2019). The Panel (2019) provides clear directions regarding which medications should be prescribed for both ART-naïve and experienced patients.

The CDC estimates that HIV has not yet been diagnosed in about 13% of the people living with HIV in the United States. After receiving an HIV diagnosis, about 75% of individuals are linked to care within 30 days, but only 57% of persons who receive an HIV diagnosis are retained in HIV care. It is estimated that only 55% of persons with diagnosed HIV are virally suppressed because of poor linkage to care and retention in care (Panel, 2019). Viral loads are often not suppressed because the patient is not adhering to the treatment plan. Psychosocial barriers such as depression and other mental illnesses, neurocognitive impairment, low health literacy, low levels of social support, stressful life events, high levels of alcohol consumption and active substance use, homelessness, poverty, nondisclosure of HIV serostatus, denial, stigma, and inconsistent access to medications affect adherence to ART. Failure to adopt practices that facilitate adherence, such as linking medication taking to daily activities or using a medication reminder system or a pill organizer, is also associated with treatment failure (Panel, 2019). Simplifying treatment regimens and decreasing the number of medications that must be taken each day also helps to increase patients' adherence to therapy. Although antiretroviral regimens have become less complex, side effects create barriers to adherence and inadequate dosing can lead to viral resistance. It is difficult to predict patients' adherence to medication regimens, but a positive relationship between the patient and health care provider is associated with better adherence (Hill, Golin, Pack, et al., 2020).

Chart 32-8 summarizes strategies that health care providers can encourage to promote treatment regimen adherence. Many real-time monitoring systems, such as electronic pill boxes, are under investigation as well as pharmacologic monitoring of blood and hair (Hill et al., 2020). Real-time monitoring has the potential to enhance ART intervention programs by providing objective information about medication taking behaviors. Every health care encounter

should be used as an opportunity to briefly review the treatment regimen, identify any new issues, and reinforce successful behaviors.

Laboratory tests evaluate whether ART is effective for a specific patient. An adequate CD4⁺ response for most patients on ART is an increase in CD4⁺ count in the range of 50 to 150 mm³ per year, generally with an accelerated response in the first 3 months (Panel, 2019). Viral load should be measured at baseline and on a regular basis thereafter because viral load is the most important indicator of response to ART.

Adverse effects associated with all HIV treatment regimens include hepatotoxicity, nephrotoxicity, and osteopenia, along with increased risk of cardiovascular disease and myocardial infarction (see [Table 32-4](#)). Many of the antiretroviral agents may cause fat redistribution syndrome and metabolic alterations such as dyslipidemia and insulin resistance, which put the patient at risk for early-onset heart disease and diabetes. The fat redistribution syndrome (lipodystrophy) consists of lipoatrophy (localized subcutaneous fat loss in the face, arms, legs, and buttocks) and lipohypertrophy (central visceral fat [lipomata] accumulation in the abdomen, although possibly in the breasts, dorsocervical region [buffalo hump], and within the muscle and liver). Facial wasting, characterized as a sinking of the cheeks, eyes, and temples caused by the loss of fat tissue under the skin, may be treated by injectable fillers such as poly-L-lactic acid (see [Fig. 32-3](#)). These changes can disturb the body image of people living with HIV/AIDS and may be a reason that they decline or stop ART.

Chart 32-8

Promoting Adherence to ART

Strategies	Examples
<p>Use a multidisciplinary team approach.</p> <p>Provide an accessible, trustworthy health care team.</p>	<ul style="list-style-type: none"> Nonjudgmental providers, nurses, social workers, pharmacists, and medication managers.
<p>Strengthen early linkage to care and retention in care.</p>	<ul style="list-style-type: none"> Encourage health care team participation in linkage to and retention in care.
<p>Assess patient readiness to start ART.</p>	<ul style="list-style-type: none"> Address specific concerns such as drug interactions between ART and hormones for transgender patients.
<p>Evaluate patient's knowledge about HIV disease, prevention and treatment and, on the basis of the assessment, provide HIV-related information.</p>	<ul style="list-style-type: none"> Consider the patient's current knowledge base, provide information about HIV, including the natural history of the disease, HIV viral load and CD4+ count and expected clinical outcomes according to these parameters, and therapeutic and prevention consequences of nonadherence.
<p>Identify facilitators, potential barriers to adherence, and necessary medication management skills before starting ART medication.</p>	<ul style="list-style-type: none"> Assess patient's cognitive competence and any impairment. Assess behavioral and psychosocial challenges including depression, mental illnesses, levels of social support, high levels of alcohol consumption and active substance use, nondisclosure of HIV serostatus and stigma. Identify and address language and literacy barriers. Assess beliefs, perceptions, and expectations about taking ART (e.g., impact on health, side effects, disclosure issues, consequences of nonadherence). Ask about medication taking skills and foreseeable challenges with adherence (e.g., past difficulty keeping appointments, adverse effects from previous medications, issues managing other chronic medications, need for medication reminders and organizers). Assess structural issues including unstable housing, lack of income, unpredictable daily schedule, lack of prescription drug coverage, lack of continuous access to medications.
<p>Provide needed resources.</p>	<ul style="list-style-type: none"> Provide or refer for mental health and/or substance abuse treatment.

	<ul style="list-style-type: none"> Provide resources to obtain prescription drug coverage, stable housing, social support, and income and food security. Encourage use of valid Internet sites for health-related information.
Involve the patient in ARV regimen selection.	<ul style="list-style-type: none"> Review regimen potency, potential side effects, dosing frequency, pill burden, storage requirements, food requirements, and consequences of nonadherence. Assess daily activities and tailor regimen to predictable and routine daily events. Use single tablet fixed-dose combination formulation. Assess if cost/co-payment for drugs can affect access to medications and adherence.
Assess adherence at every clinic visit.	<ul style="list-style-type: none"> Monitor viral load as a strong biologic measure of adherence. Use a simple behavioral rating scale. Employ a structured format that normalizes or assumes less-than-perfect adherence and minimizes socially desirable or “white coat adherence” responses. Ensure that other members of the health care team also assess adherence.
Use positive reinforcement to foster adherence success.	<ul style="list-style-type: none"> Inform patients of low or nondetectable levels of HIV viral load and increases in CD4⁺ cell counts. When needed, consider providing incentives and rewards for achieving high levels of adherence and treatment success.
Identify the type of and reasons for nonadherence.	<ul style="list-style-type: none"> Failure to fill the prescription(s). Failure to understand dosing instructions. Complexity of regimen (e.g., pill burden, size, dosing schedule, food requirements). Pill aversion. Pill fatigue. Adverse effects. Inadequate understanding of drug resistance and its relationship to adherence. Cost-related issues. Depression, drug and alcohol use, homelessness, poverty.

<p>Select from among available effective treatment adherence interventions.</p> <p>Systematically monitor retention in care.</p> <p>On the basis of any problems identified through systematic monitoring, consider options to enhance retention in care given resources available.</p>	<ul style="list-style-type: none"> • Stigma. • Nondisclosure. • Other potential barriers. <ul style="list-style-type: none"> • Use evidence-based interventions to promote adherence. • Use adherence-related tools to complement education and counseling interventions (e.g., pill boxes, dose planners, reminder devices). • Use community resources to support adherence (e.g., visiting nurses, community workers, family, peer advocates). • Use patient prescription assistance programs. • Use motivational interviews. • Record and follow up on missed visits. <ul style="list-style-type: none"> • Provide outreach for those patients who drop out of care. • Use peer or paraprofessional treatment navigators. • Employ incentives to encourage clinic attendance or recognize positive clinical outcomes resulting from good adherence. • Arrange for directly observed therapy (if feasible).
<p>Adapted from Panel on Antiretroviral Guidelines for Adults and Adolescents (Panel). (2019). Guidelines for the use of antiretroviral agents in HIV-1-infected adults and adolescents. Department of Health and Human Services. Strategies to Improve Linkage to Care, Retention in Care, Adherence to Appointments, and Adherence to Antiretroviral Therapy (pp. 236–237).</p>	

TABLE 32-4

Select Antiretroviral Agents

Generic Name (Abbreviation) and Single-Tablet Combination Names (Italic)	Food Interactions	Adverse Effects
Nucleoside Reverse Transcriptase Inhibitors (NRTIs)		
Abacavir (ABC) <i>Trizivir</i> (ABC/ZDV/3TC) <i>Epzicom</i> (ABC/3TC) (ABC/3TC/DTG)	Take without regard to meals	Hypersensitivity reaction, which can be fatal; symptoms may include fever, rash, nausea, vomiting, diarrhea, abdominal pain, malaise or fatigue, loss of appetite, and respiratory symptoms such as sore throat, cough, shortness of breath.
Didanosine (ddl)	Take half hour before or 2 h after meals	Pancreatitis, peripheral neuropathy, retinal changes, nausea, diarrhea, lactic acidosis with fatty degeneration of the liver, insulin resistance/diabetes.
Emtricitabine (FTC) <i>Atripla</i> (FTC/EFV/TDF) <i>Biktarvy</i> (BIC/TAF/FTC) <i>Complera</i> (RPV/TDF/FTC) <i>Descovy</i> (TAF/FTC) <i>Genvoya</i> (EVG/c/TAF/FTC) <i>Odefsey</i> (RPV/TAF/FTC) <i>Syntuzza</i> (DRV/c/TAF) <i>Stribild</i> (FTC/EVG/c/TDF) <i>Truvada</i> (FTC/TDF)	Take without regard to meals	Minimal toxicity; hyperpigmentation/skin discoloration. Severe acute exacerbation of hepatitis may occur in patients with HBV/HIV coinfection who discontinue FTC.
Lamivudine (3TC) <i>Cimduo</i> (TDF/3TC) <i>Combivir</i> (ZDV/3TC) <i>Epzicom</i> (ABC/3TC) <i>Temixys</i> (TDF/3TC) <i>Trizivir</i> (ABC/ZDV/3TC) <i>Delstrigo</i> (DOR/TDF/3TC)	Take without regard to meals	Minimal toxicity. Severe acute exacerbation of hepatitis may occur in patients with HBV/HIV coinfection who discontinue 3TC.

<i>Dovato</i> (DTG/3TC) <i>Symfi/Symfi LO</i> (EFV/TDF/3TC)		
<i>Triumeq</i> (DTG/ABC/3TC)		
Stavudine (d4T)	Take without regard to meals	Peripheral neuropathy; lipoatrophy; pancreatitis; lactic acidosis/severe hepatomegaly with hepatic steatosis (this is a rare, but potentially life-threatening, toxicity); hyperlipidemia; insulin resistance/diabetes mellitus; rapidly progressive ascending neuromuscular weakness (rare)
Tenofovir alafenamide (TAF) <i>Biktarvy</i> (BIC/TAF/FTC) <i>Descovy</i> (TAF/FTC) <i>Genvoya</i> (EVG/c/TAF/FTC) <i>Odefsey</i> (RPV/TAF/FTC) <i>Syntuzia</i> (DRV/c/TAF/FTC)	Take without regard to meals	Renal insufficiency, Fanconi syndrome, and proximal renal tubulopathy are less likely to occur with TAF than with TDF. Osteomalacia and decrease in bone mineral density are less likely to occur with TAF than with TDF. Severe acute exacerbation of hepatitis may occur in patients with HBV/HIV coinfection who discontinue TAF. Diarrhea, nausea, headache
Tenofovir disoproxil fumarate (TDF) <i>Atripla</i> (EFV/TDF/FTC) <i>Cimduo</i> (TDF/3TC) <i>Complera</i> (RPV/TDF/FTC) <i>Delstrigo</i> (DOR/TDF/3TC) <i>Stribild</i> (EVG/c/TDF/FTC) <i>Symfi/Symfi Lo</i> (EFV/TDF/3TC) <i>Temixys</i> (TDF/3TC) <i>Truvada</i> (TDF/FTC)	Take without regard to meals	Renal insufficiency, Fanconi syndrome, proximal renal tubulopathy, osteomalacia, decrease in bone mineral density. Severe acute exacerbation of hepatitis may occur in patients with HBV/HIV coinfection who discontinue TDF. Asthenia, headache, diarrhea, nausea, vomiting, flatulence
Zidovudine (AZT or ZDV) <i>Combivir</i> (3TC/AZT) <i>Trizivir</i> (ABC/3TC/AZT)	Take without regard to meals	Bone marrow suppression; macrocytic anemia or neutropenia; nausea, vomiting, headache, insomnia, asthenia, nail pigmentation; lactic acidosis/severe hepatomegaly with hepatic steatosis (this is a rare, but potentially life-threatening, toxicity). Hyperlipidemia; insulin resistance/diabetes mellitus; lipoatrophy; myopathy
Non-Nucleoside Reverse Transcriptase Inhibitors		
Doravirine (DOR)	Take without meals	Nausea, dizziness, abnormal dreams

<i>Delstrigo</i> (DOR/TDF/3TC)	regard to meals	
Efavirenz (EFV) <i>Atripla</i> (EFV/TDF/FTC)	Take on empty stomach at bedtime	Rash; neuropsychiatric symptoms; serum transaminase elevations; hyperlipidemia; QT interval prolongation
<i>Symfi/Symfi Lo</i> (EFV/TDF/3TC)		Use of Efv may lead to false-positive results with some cannabinoid and benzodiazepine screening assays.
Etravirine (ETR)	Take following a meal	Rash, including Stevens–Johnson syndrome; HSRs, characterized by rash, constitutional findings, and sometimes organ dysfunction (including hepatic failure), have been reported; nausea.
Nevirapine (NVP)	Take without regard to meals	Rash (reported in approximately 50% of cases), including Stevens–Johnson syndrome; symptomatic hepatitis, including fatal hepatic necrosis; symptomatic hepatitis occurs at a significantly higher frequency in ARV-naive female patients with pre-NVP CD4 ⁺ counts >250 cells/mm ³ and in ARV-naive male patients with pre-NVP CD4 ⁺ counts >400 cells/mm ³ . NVP should not be initiated in these patients unless the benefit clearly outweighs the risk.
Rilpivirine (RPV) <i>Complera</i> (RPV/TDF/FTC) <i>Juluca</i> (DTG/RPV) <i>Odefsey</i> (RPV/TAF/FTC)	Take with a meal	Rash, depression, insomnia, headache, hepatotoxicity, QT interval prolongation
Protease Inhibitors		
Atazanavir (ATV) <i>Evotaz</i> (ATV/c)	Take with food	Indirect hyperbilirubinemia; prolonged PR interval (some patients experience asymptomatic first-degree AV block); EKG changes; hyperglycemia; fat maldistribution; possible increased bleeding episodes in patients with hemophilia; Cholelithiasis; nephrolithiasis; renal insufficiency; serum transaminase elevations; hyperlipidemia (especially with RTV boosting); rash; hyperglycemia; fat maldistribution
Darunavir (DRV) <i>Prezcobix</i> (DRV/c)	Take with food	Rash: DRV has a higher allergy risk, Stevens–Johnson syndrome, toxic epidermal necrolysis, acute generalized exanthematous pustulosis, and erythema multiforme have been reported. Hepatotoxicity; diarrhea; nausea; headache; hyperlipidemia; serum

		transaminase elevation; hyperglycemia; fat maldistribution. An increase in serum creatinine may occur when DRV is administered with COBI.
Fosamprenavir (FPV)	Tablets may be taken with or without food	Rash has been reported in 12–19% of patients on FPV. FPV has an increased allergy risk. Diarrhea, nausea, vomiting, headache, hyperlipidemia, serum transaminase elevation, hyperglycemia, fat maldistribution, possible increase in the frequency of bleeding episodes in patients with hemophilia, nephrolithiasis.
Indinavir (IDV)	For upboosted IDV: Should be taken 1 h before or 2 h after meals; may take with skim milk or low-fat meal For RTV-boosted IDV: Can be taken with or without food Drink at least 48 oz of water daily	Nephrolithiasis, GI intolerance, nausea, hepatitis, indirect hyperbilirubinemia, hyperlipidemia, headache, asthenia, blurred vision, dizziness, rash, metallic taste, thrombocytopenia, alopecia, hemolytic anemia, hyperglycemia, fat maldistribution, possible increased bleeding episodes in patients with hemophilia.
Lopinavir + ritonavir (LPV/r)	Tablet: take without regard to meals Oral solution: take with food; contains 42% alcohol	GI intolerance, nausea, vomiting, diarrhea, asthenia, pancreatitis, hyperlipidemia (especially hypertriglyceridemia), elevated serum transaminase, hyperglycemia, insulin resistance/diabetes mellitus, fat maldistribution, possible increased bleeding episodes in patients with hemophilia, EKG changes.
Nelfinavir (NFV)	Dissolve tablets in a small amount of water, mix admixture well, and consume immediately. Take with food	Diarrhea, hyperlipidemia, hyperglycemia, fat maldistribution, possible increased bleeding episodes in patients with hemophilia, serum transaminase elevation.
Ritonavir (RTV)	Take with food	GI intolerance, nausea, vomiting, diarrhea, paresthesias (circumoral and extremities), hyperlipidemia (especially

		hypertriglyceridemia), hepatitis, asthenia, taste perversion, hyperglycemia, fat maldistribution, possible increased bleeding in patients with hemophilia.
Saquinavir (SQV)	Take with meals or within 2 h after meal	GI intolerance, nausea, diarrhea, abdominal pain and dyspepsia, headache, hyperlipidemia, elevated transaminase enzymes, hyperglycemia, fat maldistribution, possible increased bleeding episodes in patients with hemophilia, EKG changes.
Tipranavir (TPV)	Take with food	Hepatotoxicity; clinical hepatitis (including hepatic decompensation and hepatitis-associated fatalities) has been reported. Skin rash. Rare cases of fatal and nonfatal intracranial hemorrhages have been reported. Hyperlipidemia, hyperglycemia, fat maldistribution. Possible increase in the frequency of bleeding episodes in patients with hemophilia.

Integrase Strand Transfer Inhibitors

Dolutegravir (DTG) <i>Dovato</i> (DTG/3TC) <i>Juluca</i> (DTG/RPV) <i>Triumeq</i> (DTG/ABC/3TC)	Take without regard to meals	Insomnia, headache, depression and suicidal ideation (rare; usually occurs in patients with preexisting psychiatric conditions). Weight gain, hepatotoxicity. Preliminary data suggest an increased rate of neural tube defects in infants born to mothers who were taking DTG at the time of conception. Hypersensitivity reaction, including rash, constitutional symptoms, and organ dysfunction (including liver injury), have been reported.
Elvitegravir (EVG) <i>Stribild</i> (EVG/c/FTC/TDF)	Take with food	Nausea, diarrhea, depression and suicidal ideation (rare; usually occurs in patients with preexisting psychiatric conditions).
Raltegravir (RAL)	Take without regard to meals	Rash including Stevens–Johnson, hypersensitivity reaction and toxic epidermal necrolysis. Nausea, headache, diarrhea, pyrexia, CPK elevation, muscle weakness, rhabdomyolysis, insomnia. Depression and suicidal ideation (rare; usually occurs in patients with preexisting psychiatric conditions).

Fusion Inhibitor

Enfuvirtide (T-20)	Injected	Local injection site reactions (e.g., pain, erythema, induration, nodules and cysts, pruritus, ecchymosis) in almost 100% of patients. Increased incidence of bacterial
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pneumonia. Hypersensitivity reaction can occur and rechallenge is not recommended.

CCR5 Antagonist

Maraviroc (MVC)	Take without regard to meals; requires CCR5 tropism blood test before starting	Abdominal pain, cough, fever, dizziness, headache, orthostatic hypotension, nausea, bladder irritation; possible liver problems and cardiac events; an increased risk for some infections; a slight increase in cholesterol levels; orthostatic hypotension, especially in patients with severe renal insufficiency.
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CD4 Post-Attachment Inhibitor

Ibalizumab (IBA)	IV administration	Diarrhea, dizziness, nausea, rash
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Adapted from Panel on Antiretroviral Guidelines for Adults and Adolescents (Panel). (2019). Guidelines for the use of antiretroviral agents in HIV-1-infected adults and adolescents. Department of Health and Human Services. Retrieved on 10/28/18 at: aidsinfo.nih.gov/contentfiles/lvguidelines/adultadolescentgl.pdf

ART Drug Resistance

Drug resistance is the ability of pathogens to withstand the effects of medications that should be toxic to them. There are two major components of ART resistance: (1) transmission of drug-resistant HIV at the time of initial infection and (2) selective drug resistance in patients who are receiving nonsuppressing regimens. Genotypic and phenotypic resistance assays are used to assess viral strains and inform selection of treatment strategies. Genotypic assays detect drug-resistant mutations present in relevant viral genes while phenotypic assays measure the ability of a virus to grow in different concentrations of ART drugs. Resistance testing in persons who are chronically infected is recommended at the time of entry into HIV care. Although no definitive prospective data exist to support the choice of one type of resistance testing over another, genotypic testing is generally preferred because of lower cost, more rapid turnaround time, the assay's ability to detect mixtures of wild-type and resistant virus, and the relative ease of interpreting test results. If therapy is deferred, repeat testing soon before initiation of ART should be considered because the patient may have acquired drug-resistant virus (i.e., superinfection) (Panel, 2019).

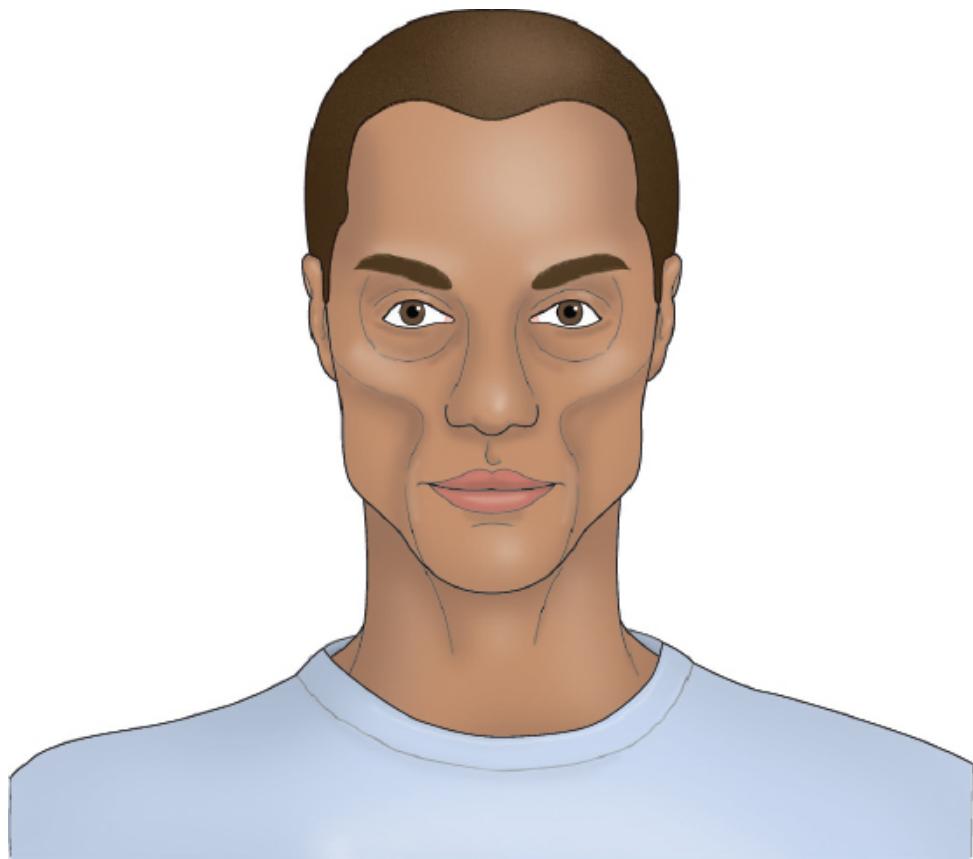


Figure 32-3 • Facial lipoatrophy.

Immune Reconstitution Inflammatory Syndrome

Immune reconstitution inflammatory syndrome (IRIS) results from rapid restoration of organism-specific immune responses to infections that cause either the deterioration of a treated infection or new presentation of a subclinical infection. This syndrome typically occurs during the initial months after beginning ART and is associated with a wide spectrum of organisms, most commonly mycobacteria, herpes viruses, and deep fungal infections. IRIS is characterized by fever, respiratory and/or abdominal symptoms, and worsening of the clinical manifestations of an opportunistic infection or the appearance of new manifestations. IRIS is treated with anti-inflammatory medications such as cortisone. The nurse should be alert to the possibility of IRIS, especially in the 3-month period after treatment with ART is initiated, because this syndrome is associated with significant morbidity and patients often require hospital admission.

Paradoxical tuberculosis-associated immune reconstitution inflammatory syndrome (TB-IRIS) is a serious complication that arises during successful ART in patients with HIV-TB co-infection who are receiving TB treatment. In the majority of patients, TB-IRIS occurs within the first few weeks of ART but can occur much later. Patients with HIV-TB co-infection with low CD4⁺

counts who start ART are at high risk of developing TB-IRIS. Although the immunopathogenesis of TB-IRIS is still not completely understood, the explosive restoration of T-cell function is believed to play a distinct role (Narendran, Oliveira-de-Souza, Vinhaes, et al., 2019).

Clinical Manifestations

Patients with HIV/AIDS experience a number of symptoms related to the disease, side effects of treatment, and other comorbidities, including pancreatitis, hepatitis, and cardiometabolic abnormalities. The clinical manifestations of HIV/AIDS are widespread and may involve virtually any organ system. Patients in stage 3 or AIDS (see [Table 32-2](#)) are severely immune depressed and can develop opportunistic infections. Nurses need to understand the causes, signs and symptoms, and interventions, including self-management strategies that can enhance the quality of life for patients throughout the different stages of the illness. Symptom assessment tools can be used to assess patients' symptom intensity and severity. PLWHA use a variety of self-management strategies to minimize common symptoms.

Respiratory Manifestations

Shortness of breath, dyspnea (labored breathing), cough, chest pain, and fever are associated with various opportunistic infections, such as those caused by *Pneumocystis jirovecii*, *Mycobacterium avium-intracellulare*, cytomegalovirus (CMV), and *Legionella* species.

Pneumocystis Pneumonia

Pneumocystis pneumonia (PCP) is caused by *P. jirovecii* (formerly *P. carinii*) (Panel on Opportunistic Infections in Adults and Adolescents with HIV [OI-Panel], 2019) and is associated with CD4⁺ T-lymphocyte (CD4⁺) cell counts less than 200 cells/mm³. The most common manifestations of PCP are subacute onset of progressive dyspnea, fever, nonproductive cough, and chest discomfort that worsens within days to weeks. In mild cases, pulmonary examination usually is normal at rest. With exertion, tachypnea, tachycardia, and diffuse dry (cellophane) rales may be auscultated. Fever is apparent in most cases and may be the predominant symptom. Hypoxemia is the most characteristic laboratory abnormality, along with elevated lactate dehydrogenase levels. Because clinical presentation, blood tests, and chest X-rays are not pathognomonic for PCP, and because the organism cannot be cultivated routinely, histopathologic or cytopathologic demonstration of organisms in tissue, bronchoalveolar lavage fluid, or induced sputum samples is required for a definitive diagnosis (OI-Panel, 2019).

Mycobacterium Avium Complex

***Mycobacterium avium* complex (MAC)** disease is a common opportunistic infection that typically occurs in patients with CD4⁺ T-lymphocyte (CD4⁺) cell counts less than 50 cells/mm³. MAC is caused by infection with different types of mycobacterium: *Mycobacterium avium*, *Mycobacterium intracellulare*, or *Mycobacterium kansasii*. Early symptoms may be minimal and can precede detectable mycobacteremia by several weeks and include fever, night sweats, weight loss, fatigue, diarrhea, and abdominal pain. A confirmed diagnosis of disseminated MAC disease is based on compatible clinical signs and symptoms coupled with the isolation of MAC from cultures of blood, lymph node, bone marrow, or other normally sterile tissue or body fluids (OI-Panel, 2019).

Tuberculosis

The estimated annual risk of reactivation with TB among those with untreated HIV infection and latent TB infection is 3% to 16% and approximates the lifetime risk for individuals without HIV infection who have latent TB infection. TB disease can occur at any CD4⁺ T-lymphocyte (CD4⁺ cell) count, although the risk increases with progressive immune deficiency. Testing for latent TB at the time of HIV diagnosis should be routine, regardless of an individual's risk of TB exposure. Individuals with negative diagnostic tests for latent TB who have stage 3 HIV infection should be retested once their CD4⁺ count increases due to ART. Screening for symptoms (asking for cough of *any* duration) coupled with chest radiography is recommended to exclude TB disease in a patient with a positive skin test or interferon-gamma release assays. Latent TB in a person with HIV infection is treated with isoniazid, supplemented with pyridoxine to prevent peripheral neuropathy, for 9 months since it has proven efficacy, good tolerability, and infrequent severe toxicity (OI-Panel, 2019).

TB disease can develop in the lungs as well as in extrapulmonary sites such as the central nervous system (CNS), bone, pericardium, stomach, peritoneum, and scrotum and initial diagnostic testing is directed at the anatomic site of symptoms or signs, such as the lungs, lymph nodes, and cerebrospinal fluid. TB in individuals with advanced immune deficiency can be rapidly progressive and fatal if treatment is delayed and such patients often have smear-negative sputum specimens. Therefore, after collection of available specimens for culture and molecular diagnostic tests, empiric treatment for TB is warranted in patients with clinical and radiographic presentation suggestive of HIV-related TB. Treatment of suspected TB in individuals with HIV infection is the same as for those who are HIV uninfected and should include an initial four-drug combination of isoniazid, rifampin, pyrazinamide, and ethambutol (OI-Panel, 2019).

Gastrointestinal Manifestations

The gastrointestinal manifestations of HIV infection and AIDS include loss of appetite, nausea, vomiting, oral and esophageal candidiasis, and chronic diarrhea. Gastrointestinal symptoms may be related to the direct inflammatory effect of HIV on the cells lining the intestines. Some of the enteric pathogens that occur most frequently, identified by stool cultures or intestinal biopsy, are *Cryptosporidium muris*, *Salmonella* species, *Isospora belli*, *Giardia lamblia*, cytomegalovirus (CMV), *Clostridium difficile*, and *M. avium-intracellulare*. In patients with AIDS, the effects of diarrhea can be devastating in terms of profound weight loss (more than 10% of body weight), fluid and electrolyte imbalances, perianal skin excoriation, weakness, and inability to perform the usual activities of daily living.

Candidiasis

Oropharyngeal and esophageal **candidiasis** (fungal infections) are common in patients with HIV infection. Oropharyngeal candidiasis is characterized by painless, creamy white, plaque-like lesions that can occur on the buccal surface, hard or soft palate, oropharyngeal mucosa, or tongue surface. Lesions can be easily scraped off with a tongue depressor or other instrument which is in contrast to lesions associated with oral hairy leukoplakia. In women with early-stage HIV infection, *Candida* vulvovaginitis usually presents the same as in women without HIV infection, with white adherent vaginal discharge associated with mucosal burning and itching of mild-to-moderate severity and sporadic recurrences (OI-Panel, 2019).

HIV Wasting Syndrome

Wasting syndrome is the involuntary loss of more than 10% of one's body weight while having experienced diarrhea or weakness and fever for more than 30 days. Wasting refers to the loss of muscle mass, although part of the weight loss may also be due to loss of fat.

Oncologic Manifestations

Those with HIV/AIDS are at greater risk of developing certain cancers. These include **Kaposi sarcoma** (KS), lymphoma, and invasive cervical cancer. KS and lymphomas are discussed next. Cervical carcinoma is described later in the Gynecologic Manifestations section.

Kaposi Sarcoma

KS is caused by human herpesvirus-8 (HHV-8); affects eight times more men than women; and may spread through sexual contact. It involves the epithelial layer of blood and lymphatic vessels. AIDS-related KS exhibits a variable and aggressive course, ranging from localized cutaneous lesions to disseminated disease involving multiple organ systems. Cutaneous signs may be the first

manifestation of HIV; they can appear anywhere on the body and are usually brownish pink to deep purple. They may be flat or raised and surrounded by ecchymosis (hemorrhagic patches) and edema (see Fig. 32-4). Rapid development of lesions involving large areas of skin is associated with extensive disfigurement and significant body image issues. The location and size of some lesions can lead to venous stasis, lymphedema, and pain. Ulcerative lesions disrupt skin integrity and increase discomfort and susceptibility to infection. The most common sites of visceral involvement are the lymph nodes, gastrointestinal tract, and lungs. Involvement of internal organs may eventually lead to organ failure, hemorrhage, infection, and death.



Figure 32-4 • Lesions of the AIDS-related Kaposi sarcoma. Whereas some patients may have lesions that remain flat, others experience extensively disseminated, raised lesions with edema. Reprinted with permission from DeVita, V. T., Jr., Hellman, S., & Rosenberg, S. (Eds.). (1993). *AIDS: Etiology, diagnosis, treatment, and prevention* (4th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Diagnosis of KS is confirmed by biopsy of suspected lesions. Prognosis depends on the extent of the tumor, the presence of other symptoms of HIV infection, and the CD4⁺ count. Death may result from tumor progression, but more often it results from other complications of HIV infection.

AIDS-Related Lymphomas

AIDS-related lymphomas include both Hodgkin lymphoma and non-Hodgkin lymphoma. Non-Hodgkin lymphoma is more common. AIDS-related

lymphoma is usually aggressive; there are three main types: diffuse large B-cell; B-cell immunoblastic; and small noncleaved cell lymphoma. Symptoms include weight loss, night sweats, and fever. The complete blood count might be abnormal and a biopsy will confirm the diagnosis.

Neurologic Manifestations

HIV-related brain changes have profound effects on cognition, including motor function, executive function, attention, visual memory, and visuospatial function. Neurologic dysfunction results from direct effects of HIV on nervous system tissue, opportunistic infections, primary or metastatic neoplasm, cerebrovascular changes, metabolic encephalopathies, or complications secondary to therapy. Immune system response to HIV infection in the CNS includes inflammation, atrophy, demyelination, degeneration, and necrosis.

Subcortical Neurodegenerative Disease

Approximately 20% of those living with HIV infection are at risk for developing a subcortical neurodegenerative disease known as HIV-associated neurocognitive disorder (HAND) (Cummins, Waters, Aggar, et al., 2019). The signs of HAND can be subtle and include changes in language, memory, problem solving and slowing of psychomotor skills (Cummins et al., 2019). Early identification is important as HAND can be treated by changing ART medications.

Peripheral Neuropathy

Peripheral neuropathy is a common neurologic symptom at any stage of HIV infection. It may be a side effect of some ART drugs, and may occur in a variety of patterns, with distal sensory polyneuropathy or distal symmetric polyneuropathy the most frequently occurring type. It can lead to significant pain of feet and hands and functional impairment. Patients use a variety of physical and psychological self-management strategies to minimize this symptom.

HIV Encephalopathy

HIV encephalopathy was formerly referred to as AIDS dementia complex (see [Chart 32-9](#)). It is a clinical syndrome that is characterized by a progressive decline in cognitive, behavioral, and motor functions as a direct result of HIV infection. HIV has been found in the brain and cerebrospinal fluid (CSF) of patients with HIV encephalopathy. The brain cells infected by HIV are predominantly the CD4⁺ cells of monocyte–macrophage lineage. HIV infection is thought to trigger the release of toxins or lymphokines that result in cellular dysfunction, inflammation, or interference with neurotransmitter function rather than cellular damage.

Chart 32-9

Care of the Patient with HIV Encephalopathy

Chronic Confusion

- Assess mental status and neurologic functioning.
- Monitor for medication interactions, infections, electrolyte imbalance, and depression.
- Frequently orient the patient to time, place, person, reality, and the environment.
- Use simple explanations.
- Instruct the patient to perform tasks in incremental steps.
- Provide memory aids (clocks and calendars).
- Provide memory aids for medication administration.
- Post activity schedule.
- Give positive feedback for appropriate behavior.
- Educate caretakers about orienting patient to time, place, person, reality, and the environment.
- Encourage the patient to designate a responsible person to assume power of attorney.

Disturbed Sensation

- Assess sensory impairment.
- Decrease number of stimuli in the patient's environment.
- Correct inaccurate perceptions.
- Provide reassurance and safety if the patient displays fear.
- Provide a secure and stable environment.
- Educate caregivers about recognizing inaccurate sensory perceptions.
- Provide caregivers techniques to correct inaccurate perceptions.
- Instruct the patient and caregivers to report any changes in the patient's vision to the patient's health care provider.

Risk for Injury

- Assess the patient's level of anxiety, confusion, or disorientation.
- Assess the patient for delusions or hallucinations.
- Remove potentially dangerous objects from the patient's environment.
- Structure the environment for safety (ensure adequate lighting, avoid clutter, provide bed rails if needed).
- Supervise smoking.
- Do not let the patient drive a car if confusion is present.
- Instruct the patient and caregiver in home safety.
- Provide assistance as needed for ambulation and in getting in and out of bed.
- Pad headboard and side rails if the patient has seizures.

Self-Care Deficits

- Encourage activities of daily living within the patient's level of ability.
- Encourage independence, but assist if the patient cannot perform an activity.
- Demonstrate any activity that the patient is having difficulty accomplishing.
- Monitor food and fluid intake.
- Weigh patient weekly.
- Encourage the patient to eat, and offer nutritious meals, snacks, and adequate fluids.
- If patient is incontinent, establish a routine toileting schedule.
- Educate caregivers about meeting the patient's self-care needs.

Signs and symptoms may be subtle and difficult to distinguish from fatigue, depression, or the adverse effects of treatment for infections and malignancies. Early manifestations include memory deficits, headache, difficulty concentrating, progressive confusion, psychomotor slowing, apathy, and ataxia. Later stages include global cognitive impairments, delay in verbal responses, a vacant stare, spastic paraparesis, hyperreflexia, psychosis, hallucinations, tremor, incontinence, seizures, mutism, and death.

Confirming the diagnosis of HIV encephalopathy can be difficult. Extensive neurologic evaluation includes a computed tomography scan, which may indicate diffuse cerebral atrophy and ventricular enlargement. Other tests that may detect abnormalities include magnetic resonance imaging, analysis of CSF through lumbar puncture, and brain biopsy.

Cryptococcus Neoformans

A fungal infection *Cryptococcus neoformans* is another common opportunistic infection among patients with AIDS, and it causes neurologic disease. Cryptococcal meningitis is characterized by symptoms such as fever, headache, malaise, stiff neck, nausea, vomiting, mental status changes, and seizures. Diagnosis is confirmed by CSF analysis.

Progressive Multifocal Leukoencephalopathy

Progressive multifocal leukoencephalopathy (PML) is a demyelinating CNS disorder that affects the oligodendroglia. Clinical manifestations often begin with mental confusion and rapidly progress to include blindness, aphasia, muscle weakness, paresis (partial or complete paralysis), and death. ART has greatly reduced the mortality associated with this disorder.

Other Neurologic Disorders

Other infections involving the nervous system include *Toxoplasma gondii*, CMV, and *Mycobacterium tuberculosis* infections.

Depressive Manifestations

Depression and apathy are neuropsychiatric complications of HIV infection. Estimates suggest that the prevalence of current depression is between 12% and 60% in persons with HIV/AIDS (Lu, Hsiao, Sheng, et al., 2018). Similarly, apathy, which refers to reduced, self-initiated, cognitive, emotional, and behavioral activity, is also commonly reported among those living with a diagnosis of HIV. Alcohol and cocaine use—both current and former—have been associated with depression and apathy in this population, and depression has been associated with less adherence with ART medications (Lu et al., 2018).

Integumentary Manifestations

Cutaneous manifestations are associated with HIV infection and the accompanying opportunistic infections and malignancies. KS (described earlier) and opportunistic infections such as herpes zoster and herpes simplex are associated with painful vesicles that disrupt skin integrity. Molluscum contagiosum is a viral infection characterized by deforming plaque formation. Seborrheic dermatitis is associated with an indurated, diffuse, scaly rash involving the scalp and face. Patients with AIDS may also exhibit a generalized folliculitis associated with dry, flaking skin or atopic dermatitis, such as eczema or psoriasis. Many patients treated with the antibacterial agent trimethoprim–sulfamethoxazole develop a drug-related rash that is pruritic with pinkish-red macules and papules (Panel, 2019). Patients with any of these rashes experience discomfort, have body image changes, and are at increased risk for infection from disrupted skin integrity.

Gynecologic Manifestations

Persistent, recurrent vaginal candidiasis may be the first sign of HIV infection in women. Past or present genital ulcers are a risk factor for the transmission of HIV infection. Women with HIV infection are more susceptible to genital ulcers and venereal warts and have increased rates of incidence and recurrence of these conditions. Ulcerative STIs such as chancroid, syphilis, and herpes are more severe in women with HIV infection. Human papillomavirus (HPV) causes venereal warts and is a risk factor for cervical intraepithelial neoplasia, a cellular change that is frequently a precursor to cervical cancer. Women who are HIV seropositive and have cervical carcinoma present at a more advanced stage of disease and have more persistent and recurrent disease and a shorter interval to recurrence and death than women without HIV infection.

Women with HIV are at increased risk for pelvic inflammatory disease, a reportable infection, and the associated inflammation may increase HIV transmission to the uninfected sexual partner. Moreover, women with HIV infection appear to have a higher incidence of menstrual abnormalities,

including amenorrhea or bleeding between periods, than do women without HIV infection.

Medical Management

Treatment of Opportunistic Infections and Coinfection with Hepatitis C

Guidelines for the treatment of opportunistic infections should be consulted for the most current recommendations (OI-Panel, 2019). Although ART is highly effective in keeping the CD4⁺ cell count high, opportunistic infections continue to cause considerable morbidity and mortality for three main reasons: (1) many patients are unaware of their HIV infection and present with an opportunistic infection as the initial indicator of their disease, (2) some patients are aware of their HIV infection but do not take antiretroviral agents because of psychosocial or economic factors, and (3) others receive prescriptions for antiretroviral medications but fail to attain adequate virologic and immunologic response as a result of issues related to adherence, pharmacokinetics, or unexplained biologic factors.

Coinfection with hepatitis C requires careful medical management for the PLWHA. Hepatitis C is curable with an 8- to 12-week drug therapy in most patients. However, the medications for the treatment of hepatitis C interact with many ART medications and therefore need to be carefully managed to avoid interactions and maximize adherence (OI-Panel, 2019; Starbird et al., 2020).

Pneumocystis Pneumonia

Persons in stage 3 HIV infection should receive chemoprophylaxis to prevent PCP with trimethoprim-sulfamethoxazole if they have CD4⁺ counts less than 200 cells/mm³ or a history of oropharyngeal candidiasis (OI-Panel, 2019). Once the CD4⁺ count improves, prophylaxis can be discontinued. When a person is diagnosed with PCP, trimethoprim-sulfamethoxazole is the treatment of choice, lowering the dose if there is abnormal renal function. Adjunctive corticosteroids are indicated as early as possible, preferentially within 72 hours after starting specific PCP therapy. Treatment duration is usually 21 days; rates of adverse reaction to trimethoprim-sulfamethoxazole are high including rash (30% to 55%) (including Stevens-Johnson syndrome), fever (30% to 40%), leukopenia (30% to 40%), hepatitis (20%), thrombocytopenia (15%), azotemia (1% to 5%), and hyperkalemia. Because long-term survival is possible for patients in whom ART is effective, individuals with AIDS and severe PCP should be given the option to choose mechanical ventilation and critical care management if their functional status is such that it would be appropriate, just

as with patients without HIV infection. Paradoxical IRIS has been reported following PCP and starting ART (OI-Panel, 2019).

Mycobacterium Avium Complex

Initial treatment of MAC disease should consist of two or more antimycobacterial drugs to prevent or delay the emergence of resistance. Clarithromycin is the preferred first agent; however, azithromycin can be substituted for clarithromycin when drug interactions or intolerance to clarithromycin preclude its use. Ethambutol is the recommended second drug (OI-Panel, 2019).

Cryptococcal Meningitis

Cryptococcosis among patients with HIV infection most commonly occurs as a subacute meningitis or meningoencephalitis with fever, malaise, and headache. Treating cryptococcosis consists of three phases: induction, consolidation, and maintenance therapy. The preferred induction treatment for cryptococcal meningitis and other forms of extrapulmonary cryptococcosis is the IV lipid formulation of amphotericin B in combination with fluconazole. Serious potential adverse effects of amphotericin B include anaphylaxis, kidney and hepatic impairment, electrolyte imbalances, anemia, fever, and severe chills. After at least 2 weeks of successful induction therapy—defined as substantial clinical improvement and a negative CSF culture after repeat lumbar puncture—amphotericin B and flucytosine can be discontinued. Follow-up or consolidation therapy is then initiated with oral fluconazole daily which should continue for at least 8 weeks (OI-Panel, 2019).

Cytomegalovirus Retinitis

Retinitis caused by CMV is a leading cause of blindness in patients with AIDS. Oral valganciclovir, IV ganciclovir, IV ganciclovir followed by oral valganciclovir, IV foscarnet, IV cidofovir, and a ganciclovir intraocular implant coupled with valganciclovir are all effective treatments for CMV retinitis (OI-Panel, 2019). All of these drugs have significant toxicities (bone marrow suppression, neutropenia, hepatitis, renal toxicity, seizures, etc.) and are used with caution.

Antidiarrheal Therapy

Although many forms of diarrhea respond to treatment, it is not unusual for this condition to recur and become a chronic problem for the patient with HIV infection. Therapy with octreotide acetate, a synthetic analogue of somatostatin, has been shown to effectively manage chronic severe diarrhea. High concentrations of somatostatin receptors have been found in the gastrointestinal tract and in other tissues. Somatostatin inhibits many

physiologic functions, including gastrointestinal motility and intestinal secretion of water and electrolytes.

Chemotherapy

Kaposi Sarcoma

KS can be treated with local therapy, radiation therapy, chemotherapy, and biologic therapy depending upon the location of the lesions.

Lymphoma

There is no standard treatment for AIDS-related peripheral or systemic lymphoma. The treatment plan is adjusted for each patient and usually includes one or more of combination chemotherapy, high-dose chemotherapy and stem cell transplant.

Antidepressant Therapy

Treatment for depression in people with HIV infection involves cognitive behavioral therapy integrated with pharmacotherapy (Lu et al., 2018). If depressive symptoms are severe and of sufficient duration, treatment with antidepressants may be initiated. Antidepressants such as imipramine, desipramine, and fluoxetine may be used, because these medications also alleviate the fatigue and lethargy that are associated with depression. A psychostimulant such as methylphenidate may be used in low doses in patients with neuropsychiatric impairment. Electroconvulsive therapy may be an option for patients with severe depression who do not respond to pharmacologic interventions.

Nutrition Therapy

Alterations in lipid metabolism are associated with HIV infection and ART. Malnutrition increases the risk of infection and the incidence of opportunistic infections. Nutrition therapy should be part of the overall management plan and should be tailored to meet the nutritional needs of the patient, whether by oral diet, enteral tube feedings, or parenteral nutritional support, if needed. As with all patients, a healthy diet is essential for the patient with HIV infection. For all patients with AIDS who experience unexplained weight loss, calorie counts and weight monitoring should be obtained to evaluate nutritional status and initiate appropriate therapy. The goal is to maintain the ideal weight and, when necessary, to increase weight.

Appetite stimulants have been successfully used in patients with AIDS-related anorexia. Megestrol acetate, a synthetic oral progesterone preparation, promotes significant weight gain and inhibits cytokine IL-1 synthesis. In patients with HIV infection, it increases body weight primarily by increasing

body fat stores. Dronabinol, which is a synthetic tetrahydrocannabinol, the active ingredient in marijuana, has been used to relieve nausea and vomiting associated with cancer chemotherapy. After beginning dronabinol therapy, almost all patients with HIV infection experience a modest weight gain. The effects on body composition are unknown.

Oral supplements may be used when the diet is deficient in calories and protein. Ideally, oral supplements should be lactose free (people with HIV infection may be intolerant to lactose), high in calories and easily digestible protein, low in fat with the fat easily digestible, palatable, inexpensive, and tolerated without causing diarrhea. Nutritional supplements have been developed specifically for people with HIV infection and AIDS. Parenteral nutrition is the final option because of its prohibitive cost and associated risks, including possible infection.

Complementary, Alternative, and Integrative Health Therapies

People with HIV infection, along with many Americans, report the use of complementary, alternative, and integrative therapies. Combined with traditional therapies, these may improve the patient's overall well-being. However, there can be adverse drug-drug interactions between certain therapies (e.g., St. John's wort) and some ART.

Although there is insufficient research on the effects of complementary, alternative, and integrative therapies, a growing body of literature reports benefits for modalities involving nutrition, exercise, psychosocial treatment, and Chinese medicine. See [Chapter 4](#) for further information on complementary, alternative, and integrative therapies.

Many patients who use these therapies do not report their use to their health care providers. To obtain a complete health history, the nurse should ask about the patient's use of complementary, alternative, and integrative therapies. Patients may need to be encouraged to report their use of these therapies to their primary provider. Problems may arise, for example, when patients are using complementary, alternative, and integrative therapies while participating in clinical drug trials; alternative therapies can have significant adverse side effects, making it difficult to assess the effects of the medications in the clinical trial. The nurse needs to become familiar with the potential adverse side effects of these therapies; if it is suspected that the therapy is causing side effects, the nurse needs to discuss this with the patient, the alternative therapy provider, and the primary provider. The nurse needs to view complementary, alternative, and integrative therapies with an open mind and try to understand the importance of this treatment to the patient. This approach will improve communication with the patient and reduce conflict.

Supportive Care

Patients who are weak and debilitated as a result of chronic illness associated with HIV infection and AIDS typically require many kinds of supportive care. Nutritional support may be as simple as providing assistance in obtaining or preparing meals. For patients with more advanced nutritional impairment resulting from decreased intake, wasting syndrome, or gastrointestinal malabsorption associated with diarrhea, parenteral feedings may be required. Imbalances that result from nausea, vomiting, and profuse diarrhea often necessitate IV fluid and electrolyte replacement.

Management of skin breakdown associated with KS, perianal skin excoriation, or immobility entails thorough and meticulous skin care that involves regular turning, cleansing, and applications of medicated ointments and dressings. To combat pain associated with skin breakdown, abdominal cramping, peripheral neuropathy, or KS, the nurse administers analgesic agents at regular intervals around the clock. Patients with a history of drug abuse will need to have tailored approaches for pain management. Relaxation and guided imagery may help reduce pain and anxiety.

Pulmonary symptoms, such as dyspnea and shortness of breath, may be related to opportunistic infections, KS, or fatigue. For patients with these symptoms, oxygen therapy, relaxation training, and energy conservation techniques may be effective. Patients with severe respiratory dysfunction may require mechanical ventilation. Before mechanical ventilation is instituted, the procedure is explained to the patient and the caregiver.

As noted previously, with the advent of ART, there are generally more positive outcomes than could be achieved years ago, and a patient mechanically ventilated has a reasonable likelihood of survival. However, if the patient decides to forego mechanical ventilation, those wishes must be followed. Ideally, the patient has prepared an advance directive identifying preferences for treatments and end-of-life care, including hospice care. If the patient has not identified preferences in advance, treatment options are described so that the patient can make informed decisions and have those wishes respected.

Nurses should anticipate that patients as well as family and friends will need support and time to share concerns. In some family systems, more than one person might be living with HIV/AIDS.

NURSING PROCESS

The Patient with HIV Infection



The nursing care of patients with HIV infection is complicated by many emotional, social, and ethical issues. The plan of care for the patient with AIDS (see [Chart 32-10](#)) is individualized to meet the needs of the patient. Care includes many of the interventions and concerns cited in the Supportive Care section.

Assessment

Nursing assessment includes identification of potential risk factors, including a history of risky sexual practices or IV/injection drug use. The patient's physical status and psychological status are assessed.

Nutritional status is assessed by obtaining a dietary history and identifying factors that may interfere with oral intake, such as anorexia, nausea, vomiting, oral pain, or difficulty swallowing. In addition, the patient's ability to purchase, prepare, and store food safely is assessed. Weight history (i.e., changes over time), anthropometric measurements, and blood urea nitrogen (BUN), serum protein, albumin, and transferrin levels provide objective measurements of nutritional status.

Chart 32-10



PLAN OF NURSING CARE

Care of the Patient with AIDS

NURSING DIAGNOSIS: Diarrhoea associated with enteric pathogens or HIV infection

GOAL: Resumption of usual bowel habits

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess patient's normal bowel habits. 2. Assess for diarrhea: frequent, loose stools; abdominal pain or cramping, volume of liquid stools, and exacerbating and alleviating factors. 3. Obtain stool cultures, and administer antimicrobial therapy as prescribed. 4. Initiate measures to reduce hyperactivity of bowel. <ol style="list-style-type: none"> a. Maintain food and fluid restrictions as prescribed. Suggest BRAT diet (<i>bananas, rice, applesauce, tea, and toast</i>). b. Discourage smoking and use of electronic nicotine delivery systems (ENDS) including e-cigarettes, e-pens, e-pipes, e-hookah, and e-cigars. c. Avoid bowel irritants such as fatty or fried foods, raw vegetables, and nuts. Offer small, frequent meals. 	<ol style="list-style-type: none"> 1. Provides baseline for evaluation. 2. Detects changes in status, quantifies loss of fluid, and provides basis for nursing measures. 3. Identifies pathogenic organism; therapy targets specific organism. 4. Promotes bowel rest, which may decrease acute episodes. <ol style="list-style-type: none"> a. Reduces stimulation of bowel. b. Eliminates nicotine, which acts as bowel stimulant. c. Prevents stimulation of bowel and abdominal 	<ul style="list-style-type: none"> • Exhibits return to normal bowel patterns • Reports decreasing episodes of diarrhea and abdominal cramping • Identifies and avoids foods that irritate the gastrointestinal tract • Takes appropriate therapy as prescribed • Exhibits normal stool cultures • Maintains adequate fluid intake • Maintains body weight and reports no additional weight loss • States rationale for avoiding smoking • Enrolls in program to stop smoking and using ENDS • Uses medication as prescribed

5. Administer anticholinergic antispasmodics and opioids or other medications as prescribed.	distention and promotes adequate nutrition.	• Maintains adequate fluid status
6. Maintain fluid intake of at least 3 L/day unless contraindicated.	5. Decreases intestinal spasms and motility. 6. Prevents hypovolemia.	• Exhibits normal skin turgor, moist mucous membranes, adequate urine output, and absence of excessive thirst

NURSING DIAGNOSIS: Risk for infection associated with immune deficiency

GOAL: Absence of infection

Nursing Interventions	Rationale	Expected Outcomes
<p>1. Monitor for infection: fever, chills, and diaphoresis; cough; shortness of breath; oral pain or painful swallowing; creamy-white patches in oral cavity; urinary frequency, urgency, or dysuria; redness, swelling, or drainage from wounds; vesicular lesions on face, lips, or perianal area.</p> <p>2. Educate patient or caregiver about need to report possible infection.</p> <p>3. Monitor white blood cell (WBC) count and differential.</p> <p>4. Obtain cultures of wound drainage, skin lesions, urine, stool, sputum, mouth, and blood as prescribed. Administer</p>	<p>1. Allows for early detection of infection, essential for prompt initiation of treatment. Repeated and prolonged infections contribute to patient's debilitation.</p> <p>2. Allows early detection of infection.</p> <p>3. Elevated WBC count possibly associated with infection.</p> <p>4. Assists in determining offending organism to</p>	<ul style="list-style-type: none"> Identifies reportable signs and symptoms of infection Reports signs and symptoms of infection if present Exhibits and reports absence of fever, chills, and diaphoresis Exhibits normal (clear) breath sounds without adventitious breath sounds Maintains weight Reports adequate energy level without excessive fatigue

	antimicrobial therapy as prescribed.	initiate appropriate treatment.	<ul style="list-style-type: none"> Reports absence of shortness of breath and cough
5.	Instruct patient in ways to prevent infection. <ol style="list-style-type: none"> Clean kitchen and bathroom surfaces with disinfectants. Clean hands thoroughly after exposure to body fluids. Avoid exposure to others' body fluids or sharing eating utensils. Turn, cough, and deep breathe, especially when activity is decreased. Maintain cleanliness of perianal area. Avoid handling pet excreta or cleaning litter boxes, birdcages, or aquariums. Cook meat and eggs thoroughly. 	5. Minimizes exposure to infection and transmission of HIV infection to others. 6. Prevents hospital-acquired infections.	<ul style="list-style-type: none"> Exhibits pink, moist oral mucous membranes without fissures or lesions Takes appropriate therapy as prescribed Experiences no infection States rationale for strategies to avoid infection Modifies activities to reduce exposure to infection or infectious persons Practices "safer sex" Avoids sharing eating utensils and toothbrush Exhibits normal body temperature Uses recommended techniques to maintain cleanliness of skin, skin lesions, and perianal area Has others handle pet
6.	Maintain aseptic technique when performing invasive procedures such as venipunctures, bladder catheterizations, and injections.		

- excreta and cleanup
- Uses recommended cooking techniques

NURSING DIAGNOSIS: Impaired airway clearance associated with *Pneumocystis pneumonia*, increased bronchial secretions, and decreased ability to cough associated with weakness and fatigue

GOAL: Improved airway clearance

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess and report signs and symptoms of altered respiratory status, tachypnea, the use of accessory muscles, cough, color and amount of sputum, abnormal breath sounds, dusky or cyanotic skin color, restlessness, confusion, or somnolence. 2. Obtain sputum sample for culture as prescribed. Administer antimicrobial therapy as prescribed. 3. Provide pulmonary care (cough, deep breathing, postural drainage, and vibration) every 2 to 4 hours. 4. Assist patient in attaining semi- or high Fowler position. 5. Encourage adequate rest periods. 	<ol style="list-style-type: none"> 1. Indicates abnormal respiratory function. 2. Aids in identification of pathogenic organisms. 3. Prevents stasis of secretions and promotes airway clearance. 4. Facilitates breathing and airway clearance. 5. Maximizes energy expenditure and prevents excessive fatigue. 6. Facilitates expectoration of secretions; prevents stasis of secretions. 	<ul style="list-style-type: none"> • Maintains normal airway clearance: <ul style="list-style-type: none"> • Respiratory rate <20 breaths/min • Unlabored breathing without the use of accessory muscles and flaring nares (nostrils) • Skin color pink (without cyanosis) • Alert and aware of surroundings • Arterial blood gas values normal • Normal breath sounds without adventitious

- | | | |
|---|--|--|
| <p>6. Initiate measures to decrease viscosity of secretions.</p> <ul style="list-style-type: none"> a. Maintain fluid intake of at least 3 L/day unless contraindicated. b. Humidify inspired air as prescribed. c. Consult with primary provider concerning the use of mucolytic agents delivered through nebulizer or intermittent positive pressure breathing treatment. <p>7. Perform tracheal suctioning as needed.</p> <p>8. Administer oxygen therapy as prescribed.</p> <p>9. Assist with endotracheal intubation; maintain ventilator settings as prescribed.</p> | <p>7. Removes secretions if patient is unable to do so.</p> <p>8. Increases availability of oxygen.</p> <p>9. Maintains ventilation.</p> | <p>breath sounds</p> <ul style="list-style-type: none"> • Begins appropriate therapy • Takes medication as prescribed • Reports improved breathing • Maintains clear airway • Coughs and takes deep breaths every 2 to 4 hours as recommended • Demonstrates appropriate positions and practices postural drainage every 2 to 4 hours • Reports reduced breathing difficulty when in semi- or high Fowler position • Practices energy-conserving strategies and alternates rest with activity • Demonstrates reduction in thickness (viscosity) of pulmonary secretions |
|---|--|--|

- Reports increased ease in coughing up sputum
- Uses humidified air or oxygen as prescribed and indicated
- Indicates need for assistance with removal of pulmonary secretions
- Understands need for and cooperates with endotracheal intubation and the use of a mechanical ventilator
- Verbalizes concerns about respiratory difficulty, intubation, and mechanical ventilation

NURSING DIAGNOSIS: Impaired nutritional intake associated with decreased oral intake

GOAL: Intake of nutrients sufficient to meet metabolic needs

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess nutritional status with height, weight, age; blood urea nitrogen, serum protein, albumin, transferrin, hemoglobin, and hematocrit levels; and cutaneous anergy. 2. Obtain dietary history, including likes and dislikes and food intolerances. 3. Assess factors that interfere with oral intake. 4. Consult with dietitian to determine patient's nutritional needs. 5. Reduce factors limiting oral intake. <ol style="list-style-type: none"> a. Encourage patient to rest before meals b. Plan meals so that they do 	<ol style="list-style-type: none"> 1. Provides objective measurement of nutritional status. 2. Defines need for nutritional education; helps individualize interventions. 3. Provides basis and directions for interventions. 4. Facilitates meal planning. 5. Addresses factors limiting intake. <ol style="list-style-type: none"> a. Minimizes fatigue, which can decrease appetite. b. Decreases noxious stimuli. 	<ul style="list-style-type: none"> • Identifies factors limiting oral intake, and uses resources to promote adequate dietary intake • Reports increased appetite • States understanding of nutritional needs • Identifies ways to reduce factors that limit oral intake • Rests before meals • Eats in pleasant, odor-free environment • Arranges meals to coincide with visitors' visits • Reports increased dietary intake • Uses oral hygiene before meals • Takes analgesic agents before

	<p>not occur immediately after painful or unpleasant procedures.</p> <p>c. Encourage patient to eat meals with visitors or others when possible.</p> <p>d. Encourage patient to prepare simple meals or to obtain assistance with meal preparation if possible.</p> <p>e. Serve small, frequent meals: 6/day.</p> <p>f. Limit fluids 1 hour before meals and with meals.</p>	<p>c. Limits social isolation.</p> <p>d. Limits energy expenditure.</p> <p>e. Prevents overwhelming patient.</p> <p>f. Reduces satiety.</p> <p>6. Provides additional proteins and calories.</p> <p>7. Provides nutritional support if patient is unable to take sufficient amounts by mouth.</p> <p>8. Increases availability of resources and nutrition.</p>	<p>meals as prescribed</p> <ul style="list-style-type: none"> Identifies ways to increase protein and caloric intake Identifies foods high in protein and calories Consumes foods high in protein and calories Reports decreased rate of weight loss Maintains adequate caloric intake States rationale for enteral or parenteral nutrition if needed Demonstrates skill in preparing alternate sources of nutrition
6.	Instruct patient in ways to supplement nutrition: consume protein-rich foods (meat, poultry, fish) and carbohydrates (pasta, fruit, breads).		
7.	Consult with primary provider and dietitian about alternative feeding (enteral or parenteral nutrition).		
8.	Consult with social worker or		

community liaison about financial assistance if patient cannot afford food.

NURSING DIAGNOSIS: Lack of knowledge associated with means of preventing HIV transmission

GOAL: Increased knowledge concerning means of preventing disease transmission

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Instruct patient, family, and friends about routes of transmission of HIV.2. Instruct patient, family, and friends about means of preventing transmission of HIV.<ol style="list-style-type: none">a. Avoid sexual contact with multiple partners, and use precautions if sexual partner's HIV status is not certain.b. Use condoms during sexual intercourse (vaginal, anal, oral-genital); avoid mouth contact with the penis, vagina, or rectum; avoid	<ol style="list-style-type: none">1. Knowledge about disease transmission can help prevent spread of disease; may also alleviate fears.2. Reduces transmission risk.<ol style="list-style-type: none">a. The risk of infection increases with the number of sexual partners, male or female, and sexual contact with those who engage in high-risk behaviors.b. Risk of HIV transmission is reduced.c. Many sex workers are infected with HIV through sexual contact with multiple partners or	<ul style="list-style-type: none">• Patient, family, and friends state means of transmission• Reports and demonstrates practices to reduce exposure of others to HIV• Demonstrates knowledge of safer sexual practices• Identifies means of preventing disease transmission• States that sexual partners are informed about patient's positive HIV status in blood• Avoids IV/injection drug use and sharing of drug

- sexual practices that can cause cuts or tears in the lining of the rectum, vagina, or penis.
- c. Avoid sex with sex workers and others at high risk.
 - d. Do not use IV/injection drugs; if addicted and unable or unwilling to change behavior, use clean needles and syringes.
 - e. Women who may have been exposed to HIV through sexual or drug practices should consult with a primary provider before becoming pregnant; consider the use of antiretroviral agents if pregnant.
 - f. Consider using PrEP
- IV/injection drug use.
- d. Clean needles and syringes are the only way to prevent HIV transmission for those who continue to use drugs. Taking precautions is important for those who are antibody positive to prevent transmitting HIV.
 - e. HIV can be transmitted from mother to child in utero; the use of antiretroviral agents during pregnancy significantly reduces perinatal transmission of HIV.
 - f. Taking ART before engaging in high-risk activity seems to protect against infection.
- equipment with others
- Understands risks and benefits associated with PrEP

NURSING DIAGNOSIS: Social isolation associated with stigma of the disease, withdrawal of support systems, isolation procedures, and fear of infecting others

GOAL: Decreased sense of social isolation

Nursing Interventions	Rationale	Expected Outcomes
<p>1. Assess patient's usual patterns of social interaction.</p> <p>2. Observe for behaviors indicative of social isolation, such as decreased interaction with others, hostility, noncompliance, sad affect, and stated feelings of rejection or loneliness.</p> <p>3. Provide instruction concerning modes of transmission of HIV.</p> <p>4. Assist patient to identify and explore resources for support and positive mechanisms for coping (e.g., contact with family, friends, AIDS task force).</p> <p>5. Allow time to be with patient other than for medications and procedures.</p> <p>6. Encourage participation in diversional activities such as reading,</p>	<p>1. Establishes basis for individualized interventions.</p> <p>2. Promotes early detection of social isolation, which may be manifested in several ways.</p> <p>3. Provides accurate information, corrects misconceptions, and alleviates anxiety.</p> <p>4. Enables mobilization of resources and supports.</p> <p>5. Promotes feelings of self-worth and provides social interaction.</p> <p>6. Provides distraction.</p>	<ul style="list-style-type: none"> • Shares with others the need for valued social interaction • Demonstrates interest in events, activities, and communication • Verbalizes feelings and reactions to diagnosis, prognosis, and life changes • Identifies modes of transmission of HIV • States ways of preventing transmission of HIV to others while maintaining contact with valued friends and relatives • Reveals HIV/AIDS diagnosis to others when appropriate • Identifies resources (i.e., family, friends, and support groups) • Uses resources

television, or
handcrafts.

when
appropriate

- Accepts offers of assistance and support
- Reports decreased sense of isolation
- Maintains contacts with those of importance to them
- Develops or continues hobbies that effectively serve as diversion or distraction

Collaborative Problems: Opportunistic infections; impaired breathing; wasting syndrome and fluid and electrolyte imbalances; adverse reaction to medications

Goal: Absence of complications

Nursing Interventions	Rationale	Expected Outcomes
Opportunistic Infections		
1. Monitor vital signs including temperature. 2. Obtain laboratory specimens, and monitor test results. 3. Instruct the patient and caregiver about signs and symptoms of infection and the need to report them early.	1. Changes in vital signs such as increases in pulse rate, respirations, blood pressure, and temperature may indicate infection. 2. Smears and cultures can identify causative agents such as bacteria, fungi, and protozoa, and sensitivity studies can identify antibiotics or other medications effective against the causative agent. 3. Early recognition of symptoms facilitates prompt treatment and avoids extra complications.	<ul style="list-style-type: none"> • Exhibits stable vital signs • Experiences control of infection • Identifies signs and symptoms correctly and experiences no complications • Identifies signs and symptoms that are reportable to the primary provider • Takes medications as prescribed
Impaired Breathing		
1. Monitor respiratory rate and pattern. 2. Auscultate the chest for breath sounds and	1. Rapid shallow breathing, diminished breath sounds, and shortness of breath may indicate respiratory failure resulting in hypoxia. 2. Crackles and wheezes may indicate fluid in the	<ul style="list-style-type: none"> • Maintains stable respiratory rate and pattern within the normal limits

	abnormal lung sounds.	lungs, which disrupts respiratory function and alters the blood's oxygen-carrying capacity.	<ul style="list-style-type: none"> Exhibits no adventitious lung sounds; normal breath sounds
3.	Monitor pulse rate, blood pressure, and oxygen saturation levels.	<p>Changes in pulse rate, blood pressure, and oxygen levels may indicate the development of respiratory or cardiac failure.</p>	<ul style="list-style-type: none"> Has stable pulse rate and blood pressure within normal limits, and exhibits no evidence of hypoxia Oxygen saturation levels within acceptable range

Wasting Syndrome and Fluid and Electrolyte Disturbances

1.	Monitor weight and laboratory values for nutritional status.	1. Weight loss, malnutrition, and anemia are common in HIV infection and increase risk of superinfection.	<ul style="list-style-type: none"> Maintains stable weight
2.	Monitor intake and output and laboratory values for fluid and electrolyte imbalance (potassium, sodium, calcium, phosphorus, magnesium, and zinc).	<p>2. Chronic diarrhea, inadequate oral intake, vomiting, and profuse sweating deplete electrolytes. Small intestine inflammation may impair the absorption of fluids and electrolytes.</p>	<ul style="list-style-type: none"> Eats a nutritious diet Attains and maintains hemoglobin, hematocrit, and ferritin levels within normal limits
3.	Monitor for and report	<p>3. Fluid loss results in decreased circulating volume leading to tachycardia, dry skin and mucous membranes, poor skin turgor, elevated urine specific gravity, and</p>	<ul style="list-style-type: none"> Sustains fluid and electrolyte balance within normal limits Exhibits no signs and symptoms of dehydration

signs and symptoms of dehydration.

thirst. Early detection allows early treatment.

Reactions to Medications

1. Monitor for medication interactions.
 2. Monitor for and promptly report side effects from antiretroviral agents.
 3. Instruct the patient and caregiver in the medication regimen.
1. People with HIV infection receive many medications for HIV and for disease complications. Early detection of medication interactions is necessary to prevent complications.
 2. Side effects from antiretroviral agents can be life-threatening. Serious side effects include anemia, pancreatitis, peripheral neuropathy, mental confusion, and persistent nausea and vomiting. Corrective measures need to be instituted.
 3. Knowledge of the medication purpose, correct administration, side effects, and strategies to manage or prevent side effects promotes safety and greater compliance with treatment.
- Experiences no serious side effects or complications from medications
 - Correctly describes medication regimen and complies with therapy, including adaptations in eating routines and type of food used with prescribed medications

The patient's level of knowledge about HIV infection, modes of disease transmission, and adherence to ART are evaluated. In addition, the level of knowledge of family (biologic and family of choice) and friends is assessed. The patient's psychological reaction to the diagnosis of HIV infection is important to explore. Reactions vary among patients and may include denial, anger, fear, shame, withdrawal from social interactions, and depressive symptoms (Lu et al., 2018). It is often helpful to gain an

understanding of how the patient has dealt with illness and major life stresses in the past. The patient's resources for support are also identified.

Diagnosis

NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include the following:

- Impaired nutritional intake associated with decreased oral intake
- Social isolation associated with stigma of HIV infection, withdrawal of support systems, isolation procedures, and fear of infecting others
- Grief associated with changes in lifestyle and roles
- Lack of knowledge associated with HIV infection, means of preventing HIV transmission, ART, and self-management strategies

COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Possible complications may include the following:

- Adverse effects of medications
- Development of HAND
- Body image changes

Planning and Goals

Goals for the patient may include improved nutritional status, increased socialization, expression of grief, increased knowledge regarding disease prevention and self-care, and absence of complications.

Nursing Interventions

IMPROVING NUTRITIONAL STATUS

Nutritional status is assessed by monitoring weight; dietary intake; and serum albumin, BUN, protein, and transferrin levels. The patient is also assessed for factors that interfere with oral intake, such as anorexia and lactose intolerance. Based on the results of assessment, the nurse can implement specific measures to facilitate oral intake. The dietitian is consulted to determine the patient's nutritional requirements.

The patient is encouraged to eat foods that are easy to swallow and to avoid spicy or sticky food items and foods that are excessively hot or cold. Oral hygiene before and after meals is encouraged. The patient who is underweight is instructed about ways to enhance the nutritional value of meals. Adding eggs, butter, or fortified milk (milk to which powdered skim milk has been added to increase the caloric content) to gravies, soups, or milkshakes can provide additional calories and protein. High-calorie,

nutritional foods such as puddings, powders, milkshakes, and nutritional supplements may also be useful.

DECREASING THE SENSE OF ISOLATION

People with HIV are at risk for double stigmatization. They have what society refers to as a “dreaded disease,” and they may have a lifestyle that differs from what is considered acceptable by many people. The diagnosis might prompt disclosure about hidden lifestyles or behaviors to family, friends, coworkers, and health care providers. As a result, people with HIV infection may be overwhelmed with emotions such as anxiety, guilt, shame, and fear. They also may be faced with multiple losses, such as loss of financial security, normal roles and functions, self-esteem, privacy, ability to control bodily functions, ability to interact meaningfully with the environment, and sexual functioning as well as rejection by sexual partners, family, and friends. Some patients may harbor feelings of guilt because of their lifestyle or because they may have infected others in current or previous relationships. Other patients may feel anger toward sexual partners who transmitted the virus to them. Infection control measures used in the hospital or at home may further contribute to the patient’s emotional isolation. Any or all of these stressors may cause the patient to withdraw both physically and emotionally from social contact.

Nurses are in a key position to provide an atmosphere of acceptance and understanding for people with HIV infection and their social networks. The patient’s usual level of social interaction is assessed as early as possible to provide a baseline for monitoring changes in behaviors that suggest social isolation (e.g., decreased interaction with staff or family, hostility, nonadherence). Patients are encouraged to express feelings of isolation and loneliness, with the assurance that these feelings are not unique or abnormal.

Providing information about how to protect themselves and others may help patients avoid social isolation. Patients, family, and friends must be reassured that HIV is not spread through casual contact. Education of ancillary personnel, nurses, and physicians helps reduce factors that might contribute to patients’ feelings of isolation.

COPING WITH GRIEF

The nurse can help the patient verbalize feelings and explore and identify resources for support and mechanisms for coping, especially when the patient is grieving anticipated losses. The patient is encouraged to maintain contact with family, friends, and coworkers and to use local or national support groups and hotlines. If possible, losses are identified and addressed. The patient is encouraged to continue usual activities whenever possible. Consultations with mental health counselors are useful for many patients and their families.

IMPROVING KNOWLEDGE OF HIV

The patient and family are educated about HIV infection, means of preventing HIV transmission, ART, and appropriate self-care measures. Information about the purpose of the medications, their correct administration, side effects, and strategies to manage or prevent side effects is provided.

MONITORING AND MANAGING POTENTIAL COMPLICATIONS

Side Effects of Medications. Adverse effects are of concern in patients who receive numerous medications for HIV infection. Many medications can cause severe toxic effects. Patients and their caregivers need to know which signs and symptoms of side/toxic effects should be reported immediately to their primary care provider (see [Table 32-4](#)).

In addition to medications used to treat HIV infection, other medications that may be required include opioids, tricyclic antidepressants, and NSAIDs for pain relief; medications for treatment of opportunistic and coinfections; antihistamines (diphenhydramine for relief of pruritus; acetaminophen or aspirin for management of fever; and antiemetic agents for control of nausea and vomiting). Concurrent use of these medications can cause many drug interactions, resulting in hepatic and hematologic abnormalities. Therefore, careful monitoring of laboratory test results is essential.

During each contact with the patient, the nurse not only asks about side effects, but also about how well the patient is adhering to the medication regimen. To promote adherence, the nurse should assist the patient to organize and plan the medication schedule. Individualized adherence plans should consider housing and social support issues, in addition to health indicators including possible drug-drug interactions. Self-reported adherence measures can distinguish clinically meaningful patterns of medication-taking behaviors; therefore, nurses should assess if patients can describe how they are taking their ART.

Research suggests that the development of effective self-management strategies leads to increased ART adherence (Schreiner, Perazzo, Currie, et al., 2019), PLWHA at risk for high treatment burden and subsequent nonadherence are those with multiple comorbidities and low social support (Schreiner et al., 2019). See the Nursing Research Profile in [Chart 32-11](#).

Monitoring for HAND. Each contact with the patient is also an opportunity to assess for the presence of HAND. A baseline assessment and then an annual assessment for signs and symptoms are recommended (Cummins et al., 2019). Nurses also need to educate caregivers about signs and symptoms of this subtle disorder.

Body Image Changes. Body image changes often occur in patients with HIV and are an important collaborative problem. The nurse helps the patient verbalize feelings and explore and identify resources for support and mechanisms for coping with body image changes. Consultations with

mental health counselors may be indicated for patients adjusting to body image changes.

Chart 32-11



NURSING RESEARCH PROFILE

Treatment Burden in People Living with HIV

Schreiner, N., Perazzo, J., Currie, J., et al. (2019). A descriptive, cross-sectional study examining treatment burden in people living with HIV. *Applied Nursing Research*, 46, 31–36.

Purpose

Successful HIV treatment associated with advances in ART has resulted in HIV being classified as a chronic condition. The purposes of this study were to: (1) describe persons living with HIV/AIDS (PLWHA) experiencing high levels of treatment burden who are at high risk for self-management nonadherence, and (2) test the relationship between known antecedent correlates (the number of chronic conditions, social capital, and age) of self-management and treatment burden in community dwelling sample of PLWHA while controlling for socio-demographics. As an indicator of social support, the relationship between social capital and treatment burden was tested.

Design

This was a descriptive, correlational, cross-sectional secondary analysis of a larger, multi-site study that examined physical activity patterns of PLWHA. Participants were ≥ 18 years of age and had confirmed HIV (HIV+ ELISA with confirmatory PCR or Western blot). An additional inclusion criterion was diagnosis of two or more chronic conditions identified in the patient's medical record. The Treatment Burden Questionnaire-13 (TBQ-13) was used to measure participant treatment burden. The TBQ-13 is a psychometrically tested instrument containing 13 items inquiring about burden associated with self-management tasks such as medication administration, self-monitoring of chronic conditions, or changes in diet. The TQB-13 asks the respondent to rank the level of burden for each question with responses ranging from 0-No Burden, to 10-Very High Burden, with summed scores ranging from 0 to 130 (higher scores indicating greater treatment burden). The Social Capital Measurement Tool was used to measure social resources.

Findings

Participants were on average 50 years of age; most were African American, male, Medicaid insured, and had a high school diploma. There was a mean of 3.63 ($SD = 1.76$) chronic conditions and the mean treatment burden score was 22.84 ($SD = 24.57$). Based on established cut-off points for low, medium, and high treatment burden, the sample experienced low levels of treatment burden, though there was wide variation in treatment burden scores. Approximately 60 PLWHA (58%) reported low treatment burden, 27 (26%) moderate treatment burden, and 16 (16%) high treatment burden. The number of comorbidities was

positively associated with treatment burden, and social resources were negatively correlated with treatment burden.

Nursing Implications

Nurses need to be aware that there are potential benefits of treatment burden screening that may help to improve self-management adherence. These results can help inform nurses how to improve the self-management adherence in PLWHA who are affected by treatment burden in the clinical setting.

PROMOTING, HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE



Educating Patients About Self-Care. Patients, families, and friends are educated about the routes of transmission of HIV. The nurse discusses precautions the patient can use to avoid transmitting HIV sexually (see Charts 32-2 and 32-3) or through sharing of body fluids, especially blood. Patients and their families or caregivers must receive instructions about how to prevent disease transmission, including hand hygiene techniques and methods for safely handling and disposing of items soiled with body fluids. Clear guidelines about avoiding and controlling infection, keeping regular health care appointments, symptom management, nutrition, rest, and exercise are necessary (see [Chart 32-12](#)). The importance of personal and environmental hygiene is emphasized. Caregivers are taught hand hygiene and appropriate infection prevention precautions (see [Chapter 66](#), Charts 66-1 and 66-2). Kitchen and bathroom surfaces should be cleaned regularly with disinfectants to prevent growth of fungi and bacteria. Patients with pets are encouraged to have another person clean areas soiled by animals, such as birdcages and litter boxes. If this is not possible, patients should use gloves to clean the area and then wash their hands afterward. Patients are advised to avoid exposure to others who are sick or who have been recently vaccinated, especially with live vaccine. The importance of avoiding smoking, excessive alcohol, and over-the-counter and street drugs is emphasized. Patients who are HIV positive or who inject drugs are instructed not to donate blood. IV/injection drug users who are unwilling to stop using drugs are advised to avoid sharing drug equipment with others.

Caregivers in the home are taught how to administer medications. The medication regimens used for patients with HIV infection can be complex and expensive. Patients receiving combination therapies for the treatment of HIV infection require careful education about the importance of taking medications as prescribed and explanations and assistance in fitting the medication regimen into their lives (see [Chart 32-8](#)). If the patient requires enteral or parenteral nutrition, instruction is provided to the patient and family about how to administer nutritional therapies at home. Nurses provide ongoing education and support for the patient and family.

Continuing and Transitional Care. Many people with HIV remain in their community and continue their usual daily activities, whereas others can no longer work or maintain their independence. Families or caregivers may need assistance in providing supportive care. Many community-based organizations provide a variety of services for people living with HIV infection; nurses can help identify these services.

Home, community-based, transitional, and hospice nurses are in an excellent position to provide the support and guidance that is so often needed in the home setting. Home health nurses are key to the safe and effective administration of parenteral antibiotics, chemotherapy, and nutrition in the home.

During home visits, the nurse assesses the patient's physical and emotional status and home environment. The patient's adherence to the therapeutic regimen is assessed, and strategies are suggested to assist with adherence. The patient is assessed for progression of disease and for adverse side effects of medications. Previous education is reinforced, and the importance of keeping follow-up appointments is stressed.

Chart 32-12



HOME CARE CHECKLIST

Infection Prevention for the Patient with Immune Deficiency

At the completion of education, the patient and/or caregiver will be able to:

- State the impact of immune deficiency on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State changes in lifestyle (e.g., hygiene, activity) necessary to decrease risk for infection.
 - Maintain good hand hygiene technique before eating, after using the bathroom, and before and after performing health care procedures.
 - Maintain total body hygiene and foot care to prevent bacterial and fungal diseases.
 - Maintain skin integrity, using cream and emollients to prevent or manage dry, chafed, or cracked skin.
 - Maintain good oral hygiene and dental checkups.
 - Avoid people with infections, recent vaccinations, and crowds.
 - Perform deep breathing; use incentive spirometer every 4 hours while awake if mobility is restricted.
 - Provide adequate lubrication with gentle vaginal manipulation during sexual intercourse; avoid anal intercourse.
- State changes in home environment necessary to decrease risk for infection.
 - Avoid cleaning birdcages and litter boxes; consider avoiding garden work (soil) and fresh flowers in stagnant water.
 - Identify the rationale for frequent cleaning of kitchen and bathroom surfaces with disinfectant.
- Verbalize understanding of ways to maintain a nutritious diet and adequate calories and necessary changes to decrease risk of infection.
- State the reason for avoiding the eating of raw fruits and vegetables, cooking all foods thoroughly, and immediately refrigerating all leftover food.
- Identify rationale and benefits of avoiding alcohol, tobacco, and unprescribed medications.
- State the name, dose, side effects, frequency, and schedule for all medications.
- Verbalize ways to cope with stress successfully, plans for regular exercise, and rationale for obtaining adequate rest.
- Identify signs and symptoms of infection to report to the primary provider, such as fever; chills; wet or dry cough; breathing problems; white patches in the mouth; swollen glands; nausea; vomiting; persistent abdominal pain; persistent diarrhea; problems

with urination or changes in the character of the urine; red, swollen, or draining wounds; sores or lesions on the body; persistent vaginal discharge with or without itching; and severe fatigue.

- Demonstrate how to monitor for signs of infection.
- Describe to whom, how, and when to report signs of infection.
- Describe appropriate actions to take should infection occur.

ADLs, activities of daily living; IADLs, instrumental activities of daily living.

Complex wound care or respiratory care may be required in the home. Patients and families are often unable to meet these skilled care needs without assistance. Nurses may refer patients to community programs that offer a range of services for patients, friends, and families, including help with housekeeping, hygiene, and meals; transportation and shopping; individual and group therapy; support for caregivers; telephone networks for the homebound; and legal and financial assistance. These services are typically provided by both professionals and nonprofessional volunteers. A social worker may be consulted to identify sources of financial support, if needed.

Home health and hospice nurses are increasingly called on to provide physical and emotional support to patients and families as patients with AIDS enter the terminal stages of disease. This support takes on special meaning when people with AIDS lose friends and when family members fear the disease or feel anger concerning the patient's lifestyle. The nurse encourages the patient and family to discuss end-of-life decisions and to ensure that care is consistent with those decisions, all comfort measures are employed, and the patient is treated with dignity at all times.

Evaluation

Expected patient outcomes may include:

1. Maintains adequate nutritional status
2. Experiences decreased sense of social isolation
3. Progresses through grieving process
4. Reports increased understanding of HIV infection, prevention of HIV transmission, and ART, and participates in self-management strategies as possible
5. Remains free of complications

Emotional and Ethical Concerns

Nurses in all settings are called on to provide care for patients with HIV infection. In doing so, they encounter not only the physical challenges of this epidemic but also emotional and ethical concerns. The concerns raised by health care professionals involve issues such as fear of infection, responsibility for giving care, values clarification, confidentiality, developmental stages of patients and caregivers, and poor prognostic outcomes.

Many patients with HIV infection have engaged in “stigmatized” behaviors. Because these behaviors challenge some traditional religious and moral values, nurses may feel reluctant to care for these patients. In addition, health care providers may still have fear and anxiety about disease transmission despite education concerning infection control and the low incidence of transmission to health care providers. Nurses are encouraged to examine their personal beliefs and to use the process of values clarification to approach controversial issues (see [Chapter 1, Chart 1-10](#)). The ANA’s Code of Ethics for Nurses (ANA, 2015) provides guidance including the first provision which states, “The nurse practices with compassion and respect for the inherent dignity, worth, and unique attributes of every person” (p. 1).

Nurses are responsible for protecting the patient’s right to privacy by safeguarding confidential information. Inadvertent disclosure of confidential patient information may result in personal, financial, and emotional hardships for the patient. The controversy surrounding confidentiality concerns the circumstances in which information may be disclosed to others (see [Chart 32-13](#)). Health care team members need accurate patient information to conduct assessment, planning, implementation, and evaluation of patient care. Failure to disclose HIV status could compromise the quality of patient care. Sexual partners of patients infected with HIV should know about the potential for infection and the need to engage in safer sex practices, as well as the possible need for testing and health care. Nurses are advised to discuss concerns about confidentiality with nurse administrators and to consult professional nursing organizations such as the Association of Nurses in AIDS Care and legal experts in their state to identify the most appropriate course of action.

Chart 32-13 ETHICAL DILEMMA

What if Maintaining Confidentiality Results in Harm?

Case Scenario

You are a nurse employed on a *per diem* basis for a home health agency. A nursing professor whom you know has received funding to conduct a mixed methods research study on men in your community who are experiencing homelessness and living with being human immune deficiency virus (HIV) positive. She has funds to pay nurses with community health experience to gather data for her study, and you were solicited and received education to serve as one of her nurse data collectors. You are assigned to conduct interviews at a homeless shelter for veterans. Eligible participants must sign an informed consent prior to being interviewed; this informed consent was approved by an institutional review board (IRB) at the nursing professor's university and was also approved by the governance board of the homeless shelter for veterans. One day while you are conducting an interview with T.M., a 28-year-old man with a history of male prostitution and substance use disorder (SUD), he suddenly begins to cry softly and says to you "I am sorry.... I just feel life is no longer worth living. I need to end things and stop going on like this." After further validating the meaning of his words, you learn that T.M. has made a plan to commit suicide. The informed consent that T.M. has signed contains assurances that his name and any information that he shares will not be divulged to anyone outside of the research team. You are conflicted about whether or not you may share his suicidal intentions with anyone.

Discussion

It is standard practice that participants who enroll in research studies are guaranteed confidentiality of their identities. However, T.M. is a particularly vulnerable research participant because he is homeless and HIV positive. Interviewing a participant in a research study who is vulnerable can cause emotional distress that might cause harm. Ideally, this contingency should have been identified by the nursing professor and the IRB, and there should have been additional information in the informed consent that identified a behavioral health professional who could be contacted should participants experience distress. Furthermore, the clause in the informed consent that assured T.M. of the confidential nature of the interview should have specified that this confidentiality would need to be breached if it was determined that participant safety was compromised.

Analysis

- Describe the ethical principles that are in conflict in this case (see [Chapter 1, Chart 1-7](#)). Can the principle of nonmaleficence be considered the preeminent principle that guides your decision regarding next steps?

- Assume that the informed consent does not make any of the specifications previously noted to protect vulnerable participants. Would you feel obligated to relay these findings to the nursing professor who serves as the principal investigator of the research team? Would you feel obligated to relay these findings to the social worker at the homeless shelter? Now assume that the provisions to protect T.M. were indeed specified in the informed consent. Would that change whether or not you would notify either the nursing professor or the social worker?
- What resources might be mobilized to be of assistance to you as you navigate your role as a research assistant? What are your legal and ethical obligations? Do you have the right or the responsibility to contact the nursing professor's IRB?

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Resources

See [Chapter 1, Chart 1-10](#), for Steps of an Ethical Analysis and Ethics Resources.

Education and provision of up-to-date information help to alleviate apprehension and prepare nurses to deliver safe, high-quality patient care. Interdisciplinary meetings allow participants to support one another and provide comprehensive patient care. Staff support groups give nurses an opportunity to solve problems and explore values and feelings about caring for patients with AIDS and their families; they also provide a forum for grieving. Other sources of support include nursing administrators, peers, and spiritual advisors.

CRITICAL THINKING EXERCISES

1  ebp Your patient is a 33-year-old transgender woman who has been diagnosed as HIV infected, stage 2. The patient tells you that they do not want to take ART because it is interfering with their self-managed hormone therapy. What is the evidence about the drug/drug interaction between ART and transitional hormone therapy? What strategies could you identify to promote adherence to ART regimen? What are the consequences of discontinuing ART?

2  pq A 55-year-old male who uses injection drugs and is HIV infected comes to the clinic where you work. He tells you that he has been experiencing night sweats, late afternoon fevers, cough, and weight loss. Identify this patient's health priorities and state the next steps you will use to meet his identified needs and other health promotion needs.

3  jpc Your patient, a heterosexual woman who is HIV infected, tells you that she wants to have unprotected sexual relations with her male partner who is HIV negative. What preventive measures should you educate this patient about? What additional members of the health care team should be included in the care of this patient?

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*Asterisk indicates nursing research.

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Resources

- AIDS Community Research Initiative of America (ACRIA), www.acria.org
AIDS Education and Training Centers (AETCs) Program (regional, national, and international training opportunities), www.aidsetc.org
AIDSinfoglossary 9th edition (2018),
clinicalinfo.hiv.gov/themes/custom/aidsinfo/documents/glossaryhivrelatedterms_english.pdf
AIDS vaccines, <http://www.avac.org/prevention-option/aids-vaccines>
Antiretroviral medication information websites: www.saf.org; hivinsite.ucsf.edu/; www.amfAR.org; www.natap.org; www.thebody.com
Centers for Disease Control and Prevention, HIV/AIDS Prevention Research Synthesis Project, <http://www.cdc.gov/hiv/dhap/prb/prs/>
Gay Men's Health Crisis Network (GMHC), www.gmhc.org
Harm Reduction Coalition, <http://harmreduction.org/about-us/>

Health Resources and Service Administration (HRSA), National Clinician's Post-exposure Prophylaxis Hotline (health care providers only), 1-888-448-4911
Health Resources and Service Administration (HRSA), National HIV Telephone Consultation Service, 1-800-933-3413
International AIDS Vaccine Initiative (AVI), www.iavi.org
International Partnership for Microbicides, www.ipmglobal.org
National Institutes of Health, HIV/AIDS Treatment, Prevention, and Research, www.aidsinfo.nih.gov
Office of Minority Health Resource Center, www.minorityhealth.hhs.gov/omh/browse.aspx?lvl=4&lvlid=21
POZ, Health, Life, & HIV. Published by Smart+Strong, 500 Fifth Avenue, Suite 320, New York, NY 10110, www.poz.com/
Prevention Access Campaign, www.preventionaccess.org

33 Assessment and Management of Patients with Allergic Disorders

LEARNING OUTCOMES

On completion of this chapter, the learner will be able to:

1. Describe the physiologic events involved with allergic reactions and types of hypersensitivity.
2. Use appropriate parameters for assessment of the status of patients with allergic disorders.
3. Identify the pathophysiology, clinical manifestations, and management of patients with allergic disorders.
4. Specify measures to prevent and manage anaphylaxis.
5. Use the nursing process as a framework for care of the patient with allergic rhinitis.

NURSING CONCEPTS

Cellular Regulation
Immunity
Infection

GLOSSARY

allergen: substance that causes manifestations of allergy

allergy: inappropriate and often harmful immune system response to substances that are normally harmless

anaphylaxis: rapid clinical response to an immediate immunologic reaction between a specific antigen and antibody

angioedema: condition characterized by urticaria and diffuse swelling of the deeper layers of the skin (*synonym:* angioneurotic edema)

antibody: protein substance developed by B cells in response to and interacting with a specific antigen

antigen: substance that the body identifies as a foreign invader; antigens induce the production of antibodies

antihistamine: medication that opposes the action of histamine

atopic dermatitis: type I hypersensitivity involving inflammation of the skin evidenced by itching, redness, and a variety of skin lesions

atopic march: a progression of allergic disease beginning with atopic dermatitis continuing to IgE-mediated food allergy, asthma, and allergic rhinitis

atopy: term often used to describe immunoglobulin E-mediated diseases (i.e., atopic dermatitis, asthma, and allergic rhinitis) with a genetic component

B cells: lymphocyte cells that are stimulated to produce antibodies

bradykinin: a substance that stimulates nerve fibers and causes pain

eosinophil: granular leukocyte

erythema: diffuse redness of the skin

hapten: incomplete antigen

histamine: substance in the body that causes increased gastric secretion, dilation of capillaries, and constriction of the bronchial smooth muscle

hypersensitivity: abnormal heightened reaction to a stimulus of any kind

immunoglobulins: a family of closely related proteins capable of acting as antibodies

leukotrienes: a group of chemical mediators that initiate the inflammatory response

mast cells: connective tissue cells that contain heparin and histamine in their granules

plasma cells: upon stimulation by antigen, B lymphocytes differentiate into plasma cells that secrete antibodies

prostaglandins: unsaturated fatty acids that have a wide assortment of biologic activity

serotonin: chemical mediator that acts as a potent vasoconstrictor and bronchoconstrictor

T cells: lymphocyte cells that participate in cellular immunity and assist in humoral (B-cell) immunity

urticaria: a round, reddened skin elevation or hives

Allergic disorders are common and are encountered by nurses in every setting, from the community to the intensive care unit. Expert management of patients with allergic disorders is integral to nursing regardless of the practice setting. This chapter covers general allergic assessment and management of various allergic disorders including anaphylaxis.

The human body is bombarded by a host of potential invaders—allergens as well as microbial organisms—that constantly threaten its defenses. These invaders are termed **antigens**. After penetrating the body's defenses, these antigens, if allowed to continue unimpeded, disrupt the body's enzyme systems and destroy its vital tissues. To protect against these antigens, the body is equipped with an elaborate defense system.

The epithelial cells that coat the skin and make up the lining of the respiratory, gastrointestinal, and genitourinary tracts provide the first line of defense against microbial invaders. The structure and continuity of these surfaces and their resistance to penetration are initial deterrents to antigens (Marshall, Warrington, Watson, et al., 2018).

One of the most effective defense mechanisms is the body's capacity to equip itself rapidly with antibodies specifically designed to combat each new invader—namely, specific protein antigens. Antibodies react with antigens in a variety of ways: (1) by coating the antigens' surfaces, (2) by neutralizing the antigens, and (3) by precipitating the antigens out of solution if they are dissolved. The antibodies prepare the antigens so that the phagocytic cells of the blood and the tissues can eliminate them. However, although this system is normally protective, in some cases the body produces inappropriate or exaggerated responses to specific antigens, and the result is an allergic or hypersensitivity disorder.

Physiologic Overview

An allergic reaction is a manifestation of tissue injury resulting from interaction between an antigen and an **antibody** (a protein substance developed by B cells in response to and interacting with a specific antigen) (Marshall et al., 2018). **Allergy** is an inappropriate and often harmful response of the immune system to normally harmless substances, called **allergens** (e.g., dust, weeds, pollen, dander). Chemical mediators released in allergic reactions may produce symptoms that range from mild to life-threatening.

In allergic reactions, the body encounters allergens that are types of antigens, usually proteins that the body's defenses recognize as foreign, and a series of events occurs in an attempt to render the invaders harmless, destroy them, and remove them from the body. There is a generalized white blood cell response to the entrance of antigens into the body. Specific types of white blood cells, called B lymphocytes (also called B cells), are specifically triggered by the presence of antigen. B lymphocytes differentiate into **plasma cells**, which then secrete antibodies that attack the antigen.

Specific antibodies develop in response to specific antigens. Antibodies combine with antigens in a special way, which has been likened to a lock and key. Antigens (the keys) fit only certain antibodies (the locks). Hence, the term *specificity* refers to the specific reaction of an antibody to an antigen. There are many variations and complexities in these patterns. In addition, antibodies have memory for the specific antigen, so that upon future exposure, the lock and key reaction occurs again (Actor, 2019).

Function of Immunoglobulins

Antibodies that are formed by plasma cells in response to an immunogenic stimulus constitute a group of serum proteins called **immunoglobulins**. Grouped into five classes (IgG, IgA, IgM, IgD, and IgE), immunoglobulins can be found in the lymph nodes, tonsils, appendix, and Peyer patches of the intestinal tract or circulating in the blood and lymph. These antibodies are capable of binding with a wide variety of antigens. Immunoglobulins of the IgE class are involved in allergic disorders and some parasitic infections. IgE-producing cells are located in the respiratory and intestinal mucosa. Two or more IgE molecules bind together to an allergen and trigger **mast cells** or basophils to release chemical mediators, such as histamine, serotonin, kinins, slow-reacting substances of anaphylaxis, and the neutrophil factor, which produces allergic skin reactions, asthma, and hay fever. **Atopy** refers to IgE-mediated diseases, such as allergic rhinitis, that have a genetic component (Actor, 2019). See [Chapter 31](#) for more information on immunoglobulins.

Role of B Cells

B cells, or B lymphocytes, are programmed to produce one specific antibody. On encountering a specific antigen, B cells generate plasma cells, at the site of antibody production. The plasma cells secrete antibodies for the purpose of destroying and removing the antigens. B cells participate in humoral immunity (also called antibody-mediated immunity), which is one kind of adaptive immunity (Marshall et al., 2018).

Role of T Cells

There are different types of **T cells**, or T lymphocytes, that participate in cellular immunity (also called cell-mediated immunity), a type of adaptive immunity. T helper cells are specific types of T lymphocytes that assist B cells in the immune response. T cells secrete substances that direct the flow of cell activity and stimulate macrophages. Macrophages present the antigens to the T cells and initiate the immune response. They also digest antigens and assist in removing cells and other debris (Marshall et al., 2018).

Function of Antigens

Antigens are divided into two groups: complete protein antigens and low-molecular-weight substances. Complete protein antigens, such as animal dander, pollen, and horse serum, stimulate a complete humoral response. A humoral response is another name for a B lymphocyte–mediated response. See Chapter 31 for a discussion of humoral immunity. Low-molecular-weight substances, such as medications, function as **haptens** (incomplete antigens), binding to tissue or serum proteins to produce a carrier complex that initiates an antibody response. In an allergic reaction, the production of antibodies requires active communication between cells. When the allergen is absorbed through the respiratory tract, gastrointestinal tract, or skin, allergen sensitization occurs. In the humoral response, B cells mature into allergen-specific secreting plasma cells that synthesize and secrete antigen-specific antibodies (Marshall et al., 2018).

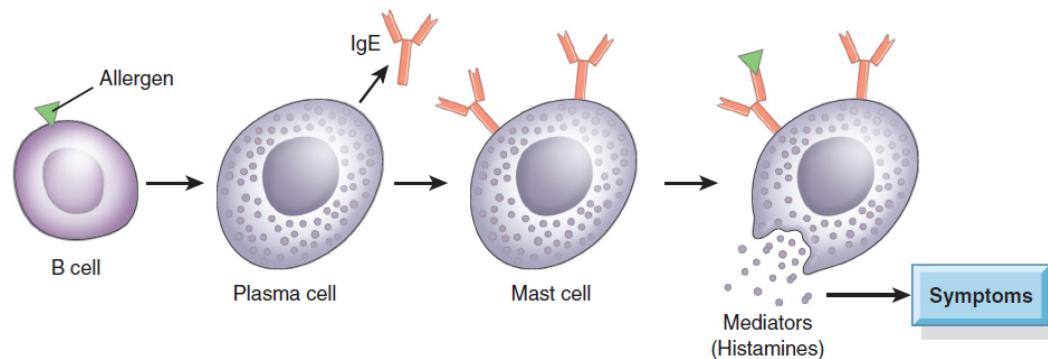


Figure 33-1 • Antigen stimulates activation of a B cell (B lymphocyte), which then transforms into a plasma cell that secretes immunoglobulins. Immunoglobulins stimulate mast cells to release histamine and other inflammatory mediators.

Function of Chemical Mediators

Mast cells, which are located in the skin, respiratory tract, and gastrointestinal tract, play a major role in IgE-mediated immediate hypersensitivity. When mast cells are stimulated by antigens, powerful chemical mediators, such as

histamine, are released, causing a sequence of physiologic events that result in symptoms of immediate hypersensitivity (see Fig. 33-1). There are two types of chemical mediators: primary and secondary. Primary mediators are preformed and are found in mast cells or basophils. Secondary mediators are inactive precursors that are formed or released in response to primary mediators. Table 33-1 summarizes the actions of primary and secondary chemical mediators (Castells, 2017).

Primary Mediators

Histamine, eosinophil chemotactic factor of anaphylaxis, platelet-activating factor, and prostaglandins are primary chemical mediators in allergic responses.

Histamine

Histamine, which is released by mast cells, plays an important role in the immune response. It is the first chemical mediator to be released in immune and inflammatory responses. It is synthesized and stored in high concentrations in body tissues exposed to environmental substances. Histamine's effects peak 5 to 10 minutes after antigen contact and include the following: erythema; localized edema in the form of wheals; pruritus; contraction of bronchial smooth muscle, resulting in wheezing and bronchospasm; dilation of small venules and constriction of larger vessels; and increased secretion of gastric and mucosal cells, resulting in diarrhea. Histamine action results from stimulation of histamine-1 (H_1) and histamine-2 (H_2) receptors. H_1 receptors are found predominantly on bronchiolar and vascular smooth muscle cells; H_2 receptors are found on gastric parietal cells (Castells, 2017).

Certain medications are categorized by their action at these receptors. Diphenhydramine is an example of an **antihistamine**, a medication that displays an affinity for H_1 receptors. Cimetidine targets H_2 receptors to inhibit gastric secretions in peptic ulcer disease.

TABLE 33-1 Chemical Mediators of Hypersensitivity

Mediators	Action
Primary Mediators	
Preformed and Found in Mast Cells or Basophils	
Histamine (preformed in mast cells)	Vasodilation Smooth muscle contraction, increased vascular permeability, increased mucus secretions
Eosinophil chemotactic factor of anaphylaxis (preformed in mast cells)	Attracts eosinophils
Platelet-activating factor (requires synthesis by mast cells, neutrophils, and macrophages)	Smooth muscle contraction Incites platelets to aggregate and release serotonin and histamine
Prostaglandins (chemically derived from arachidonic acid; require synthesis by cells)	D and F series → bronchoconstriction E series → bronchodilation D, E, and F series → vasodilation
Basophil kallikrein (preformed in mast cells)	Frees bradykinin, which causes bronchoconstriction, vasodilation, and nerve stimulation
Secondary Mediators	
Inactive Precursors Formed or Released in Response to Primary Mediators	
Bradykinin (derived from precursor kininogen)	Smooth muscle contraction, increased vascular permeability, stimulates pain receptors, increased mucus production
Serotonin (preformed in platelets)	Smooth muscle contraction, increased vascular permeability
Heparin (preformed in mast cells)	Anticoagulant
Leukotrienes (derived from arachidonic acid and activated by mast cell degranulation) C, D, and E or slow-reacting substance of anaphylaxis	Smooth muscle contraction, increased vascular permeability

Adapted from Norris, T. (2019). *Porth's pathophysiology: Concepts of altered health states* (10th ed.). Philadelphia, PA: Wolters Kluwer.

Eosinophil Chemotactic Factor of Anaphylaxis

Eosinophil chemotactic factor of anaphylaxis affects the movement of **eosinophils** (granular leukocytes) to the site of allergens. It is preformed in the mast cells and is released from disrupted mast cells.

Platelet-Activating Factor

Platelet-activating factor is responsible for initiating platelet aggregation and leukocyte infiltration at sites of immediate hypersensitivity reactions. It also causes vasodilation, bronchoconstriction, and increased vascular permeability (Castells, 2017).

Prostaglandins

Prostaglandins produce smooth muscle contraction as well as vasodilation and increased capillary permeability. They sensitize pain receptors and increase the pain associated with inflammation. In addition, prostaglandins induce inflammation and enhance the effects of mediators of inflammatory response. Local manifestations include erythema, heat, and edema (Castells, 2017).

Secondary Mediators

Leukotrienes, bradykinin, and serotonin are all secondary chemical mediators.

Leukotrienes

Leukotrienes are chemical mediators that initiate the inflammatory response. Many manifestations of inflammation can be attributed in part to leukotrienes. In addition, leukotrienes cause smooth muscle contraction, bronchial constriction, mucus secretion in the airways, and the typical wheal-and-flare reactions of the skin. Compared with histamine, leukotrienes are 100 to 1000 times more potent in causing bronchospasm (Castells, 2017).

Bradykinin

Bradykinin is a substance that has the ability to cause increased vascular permeability, vasodilation, hypotension, and contraction of many types of smooth muscle, such as the bronchi. Increased permeability of the capillaries results in edema. Bradykinin stimulates nerve cell fibers and produces pain.

Serotonin

Serotonin is a chemical mediator that acts as a potent vasoconstrictor and causes contraction of bronchial smooth muscle.

Hypersensitivity

Although the immune system defends the host against infections and foreign antigens, immune responses can themselves cause tissue injury and disease.

Hypersensitivity is an excessive or aberrant immune response to any type of stimulus (Actor, 2019). It usually does not occur with the first exposure to an allergen. Rather, the reaction follows a re-exposure after sensitization, or buildup of antibodies, in a predisposed person. Injurious or pathologic immune

reactions are classed as hypersensitivity reactions. To promote understanding of the immunopathogenesis of disease, hypersensitivity reactions have been classified into four specific types of reactions (see Fig. 33-2).

Anaphylactic (Type I) Hypersensitivity

The most severe hypersensitivity reaction is **anaphylaxis**. An unanticipated severe allergic reaction that is rapid in onset, anaphylaxis is characterized by edema in many tissues, including the larynx, and is often accompanied by hypotension, bronchospasm, and cardiovascular collapse in severe cases. Anaphylaxis is a severe type I hypersensitivity reaction, which is an immediate reaction beginning within minutes of exposure to an antigen. Primary chemical mediators are responsible for the symptoms of type I hypersensitivity because of their effects on the skin, lungs, and gastrointestinal tract. If chemical mediators continue to be released, a delayed reaction may occur and may last for up to 24 hours (Marshall et al., 2018).

Clinical symptoms are determined by the amount of the allergen, the amount of mediator released, the sensitivity of the target organ, and the route of allergen entry. Type I hypersensitivity reactions may include both local and systemic anaphylaxis (Actor, 2019).

Cytotoxic (Type II) Hypersensitivity

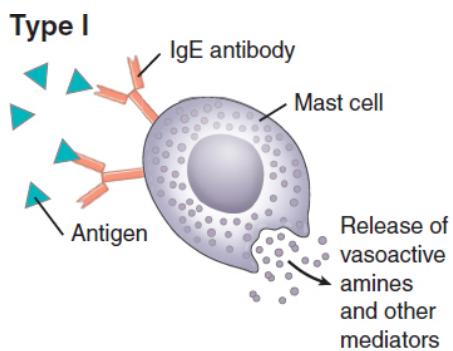
Type II, or cytotoxic hypersensitivity occurs when antibodies are directed against antigens on cells or basement membranes of tissues. This reaction can lead to cell lysis and tissue damage. Type II hypersensitivity reactions are associated with several disorders. The best example is a hemolytic transfusion reaction. For example, if a person with type A blood is mistakenly given type B blood, anti-B antibodies are triggered in the recipient that attack the infused type B blood cells and cause hemolysis (Actor, 2019).

Immune Complex (Type III) Hypersensitivity

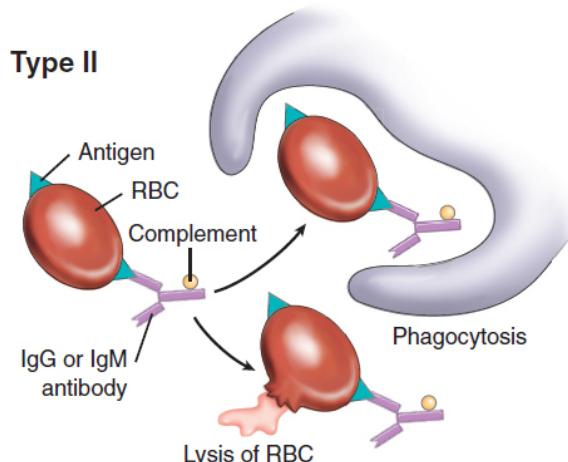
Type III, or immune complex hypersensitivity, is a damaging inflammatory reaction caused by the insoluble immune complexes formed by antigens that bind to antibodies. These complexes are too large to be cleared from the circulation by phagocytic action. The immune complexes are deposited in tissues or vascular endothelium and trigger inflammation at different sites throughout the body. An example of this kind of hypersensitivity reaction occurs in rheumatoid arthritis. An unknown antigen triggers antibody formation, which then forms immune complexes that are deposited in the joints. Many autoimmune disorders are type III hypersensitivity reactions. In autoimmune reactions, such as systemic lupus erythematosus, patients form autoantibodies that form immune complexes that deposit in the lungs, skin, and kidney (Actor, 2019).

Delayed (Type IV) Hypersensitivity

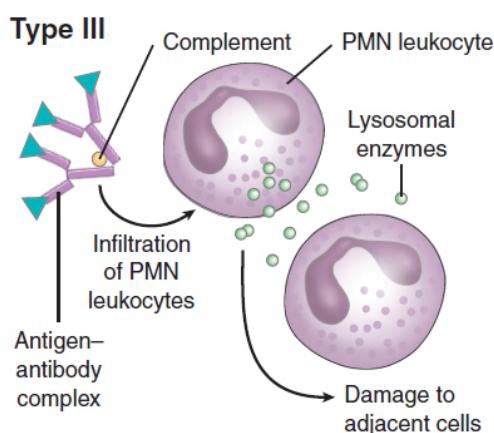
Type IV, or delayed hypersensitivity, is a T cell-mediated immune reaction after exposure to an antigen. This immune reaction typically occurs 24 to 48 hours after exposure to an antigen. The prototypical type IV hypersensitivity reaction occurs in response to the subcutaneous injection of purified protein derivative (PPD) antigen from *Mycobacterium tuberculosis*. Patients who have had previous exposure or have tuberculosis (TB) infection will demonstrate a reaction of erythema and induration due to sensitized T cells (Actor, 2019).



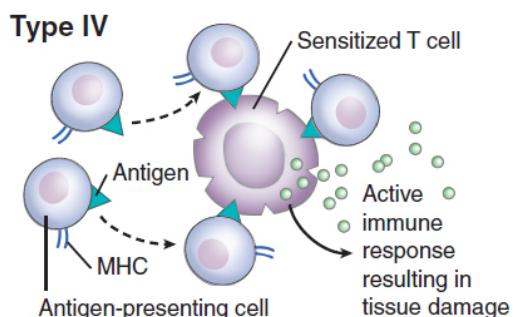
Type I. An anaphylactic reaction is characterized by vasodilation, increased capillary permeability, smooth muscle contraction, and eosinophilia. Systemic reactions may involve laryngeal stridor, angioedema, hypotension, and bronchial, GI, or uterine spasm; local reactions are characterized by hives. Examples of type I reactions include extrinsic asthma, allergic rhinitis, systemic anaphylaxis, and reactions to insect stings.



Type II. A cytotoxic reaction, which involves binding either the IgG or IgM antibody to a cell-bound antigen, may lead to eventual cell and tissue damage. The reaction is the result of mistaken identity when the system identifies a normal constituent of the body as foreign and activates the complement cascade. Examples of type II reactions are myasthenia gravis, Goodpasture syndrome, pernicious anemia, hemolytic disease of the newborn, transfusion reaction, and thrombocytopenia.



Type III. An immune complex reaction is marked by acute inflammation resulting from formation and deposition of immune complexes. The joints and kidneys are particularly susceptible to this kind of reaction, which is associated with systemic lupus erythematosus, serum sickness, nephritis, and rheumatoid arthritis. Some signs and symptoms include urticaria, joint pain, fever, rash, and adenopathy (swollen glands).



Type IV. A delayed, or cellular, reaction occurs 1 to 3 days after exposure to an antigen. The reaction, which results in tissue damage, involves activity by lymphokines, macrophages, and lysozymes. Erythema and itching are common; a few examples include contact dermatitis, graft-versus-host disease, Hashimoto's thyroiditis, and sarcoidosis.

Figure 33-2 • Four types of hypersensitivity reactions. GI, gastrointestinal; Ig, immunoglobulin; PMN, polymorphonuclear;

RBC, red blood cell.

Assessment

A comprehensive allergy history and a thorough physical examination provide useful data for the diagnosis and management of allergic disorders. An allergy assessment form is useful for obtaining and organizing pertinent information (see [Chart 33-1](#)).

The degree of difficulty and discomfort experienced by the patient because of allergic symptoms and the degree of improvement in those symptoms with and without treatment are assessed and documented. The relationship of symptoms to exposure to possible allergens is noted.

Chart 33-1 ASSESSMENT

Allergy Assessment Form

Name _____ Age _____ Sex _____ Date _____

I. Chief complaint: _____

II. Present illness: _____

III. Collateral allergic symptoms:

Eyes:	Pruritus _____	Burning _____	Lacrimation _____
	Swelling _____	Injection _____	Discharge _____
Ears:	Pruritus _____	Fullness _____	Popping _____
	Frequent infections _____		
Nose:	Sneezing _____	Rhinorrhea _____	Obstruction _____
	Pruritus _____	Mouth breathing _____	
	Purulent discharge _____		
Throat:	Soreness _____	Postnasal discharge _____	
	Palatal pruritus _____	Mucus in the morning _____	
Chest:	Cough _____	Pain _____	Wheezing _____
	Sputum _____	Dyspnea _____	
	Color _____	Rest _____	
	Amount _____	Exertion _____	
Skin:	Dermatitis _____	Eczema _____	Urticaria _____

IV. Family allergies: _____

V. Previous allergic treatment or testing:

Prior skin testing: _____

Medications:

Antihistamines	Improved _____	Unimproved _____
Bronchodilators	Improved _____	Unimproved _____
Nose drops	Improved _____	Unimproved _____
Hyposensitization	Improved _____	Unimproved _____
Duration _____		
Antigens _____		
Reactions _____		
Antibiotics	Improved _____	Unimproved _____
Corticosteroids	Improved _____	Unimproved _____

VI. Physical agents and habits: _____

Bothered by:

Tobacco for _____ years	Alcohol _____	Air-conditioning _____
Cigarettes _____ packs/day	Heat _____	Muggy weather _____
Cigars _____ per day	Cold _____	Weather changes _____
Pipes _____ per day	Perfumes _____	Chemicals _____
Never smoked _____	Paints _____	Hair spray _____
Bothered by smoke _____	Insecticides _____	Newspapers _____
	Cosmetics _____	Latex _____

VII. When symptoms occur:

Time and circumstances of 1st episode: _____

Prior health: _____

Course of illness over decades: progressing _____ regressing _____

Time of year: _____ Exact dates: _____

Perennial _____
Seasonal _____
Seasonally exacerbated _____

Monthly variations (menses, occupation): _____

Time of week (weekends vs. weekdays): _____

Time of day or night: _____

After insect stings: _____

VIII. Where symptoms occur:

Living where at onset: _____

Living where since onset: _____

Effect of vacation or major geographic change: _____

Symptoms better indoors or outdoors: _____

Effect of school or work: _____

Effect of staying elsewhere nearby: _____

Effect of hospitalization: _____

Effect of specific environments: _____

Do symptoms occur around:

old leaves _____ hay _____ lakeside _____ barns _____
summer homes _____ damp basement _____ dry attic _____
lawn mowing _____ animals _____ other _____

Do symptoms occur after eating:							
cheese	<input type="checkbox"/>	mushrooms	<input type="checkbox"/>	beer	<input type="checkbox"/>	melons	<input type="checkbox"/>
bananas	<input type="checkbox"/>	fish	<input type="checkbox"/>	nuts	<input type="checkbox"/>	citrus fruits	<input type="checkbox"/>
other foods (list) _____							
Home: city <input type="checkbox"/> rural <input type="checkbox"/>							
house <input type="checkbox"/> age <input type="checkbox"/>							
apartment <input type="checkbox"/> basement <input type="checkbox"/> damp <input type="checkbox"/> dry <input type="checkbox"/>							
heating system <input type="checkbox"/>							
vacuum cleaner system <input type="checkbox"/> use of HEPA filter <input type="checkbox"/>							
pets (how long) <input type="checkbox"/> dog <input type="checkbox"/> cat <input type="checkbox"/> other <input type="checkbox"/>							
<i>Bedroom:</i>			Type	Age	<i>Living room:</i>	Type	Age
Pillow	<input type="checkbox"/>	<input type="checkbox"/>	Rug	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Mattress	<input type="checkbox"/>	<input type="checkbox"/>	Matting	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Blankets	<input type="checkbox"/>	<input type="checkbox"/>	Furniture	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Quilts	<input type="checkbox"/>						
Furniture	<input type="checkbox"/>						
Anywhere in home symptoms are worse: _____							
IX. What does patient think makes symptoms worse? _____							
X. Under what circumstances is patient free of symptoms? _____							
XI. Summary and additional comments: _____							

Diagnostic Evaluation

Diagnostic evaluation of the patient with allergic disorders commonly includes blood tests, smears of body secretions, skin tests, and the serum-specific IgE test (formerly known as radioallergosorbent test [RAST]). Results of laboratory blood studies provide supportive data for various diagnostic possibilities; however, they are not the major criteria for the diagnosis of allergic disease (Kowal & DuBuske, 2017).

Complete Blood Count with Differential

The white blood cell (WBC) count is usually within normal limits except when infection and inflammation are present along with an allergic disorder. Eosinophils, which are granular leukocytes, normally make up 2% to 5% of the total number of WBCs. They can be found in blood, sputum, and nasal secretions. A level greater than 5% to 10% is considered abnormal and may be found in patients with allergic disorders (Kowal & DuBuske, 2017).

Eosinophil Count

An actual count of eosinophils can be obtained from blood samples or smears of secretions. During symptomatic episodes, smears obtained from nasal secretions and sputum of patients with allergies usually reveal an increase in eosinophils, indicating an active allergic response (Kowal & DuBuske, 2017).

Total Serum Immunoglobulin E Levels

High total serum IgE levels support the diagnosis of allergic disease. In the majority of cases, the antibody typically responsible for an allergic reaction

belongs to the IgE isotype. Patients with this disorder are said to have an IgE-mediated allergic disease (Kowal & DuBuske, 2017).

Skin Tests

Skin testing entails the intradermal injection or superficial application (epicutaneous) of solutions at several sites. Depending on the suspected cause of allergic signs and symptoms, many different solutions may be applied at separate sites. These solutions contain individual antigens representing an assortment of allergens most likely to be implicated in the patient's disease. Positive (wheal-and-flare) reactions are clinically significant when correlated with the history, physical findings, and results of other laboratory tests. Skin testing is considered the most accurate confirmation of allergy (Kowal & DuBuske, 2016).

The results of skin tests complement the data obtained from the history. They indicate which of several antigens are most likely to provoke symptoms and indicate the intensity of the patient's sensitization. The dosage of the antigen (allergen) injected is also important. Most patients are hypersensitive to more than one allergen. Under testing conditions, they may not react (although they usually do) to the specific allergens that induce their attacks.

When there is doubt about the validity of the skin tests, a serum-specific IgE test or a provocative challenge test may be performed. If a skin test is indicated, there is a reasonable suspicion that a specific allergen is producing symptoms in a patient with allergies. However, several precautionary steps must be observed before skin testing with allergens is performed:

- Testing is not performed during periods of bronchospasm.
- Epicutaneous tests (scratch or prick tests) are performed before other testing methods, in an effort to minimize the risk of systemic reaction.
- Emergency equipment must be readily available to treat anaphylaxis.

Types of Skin Tests

The methods of skin testing include prick skin tests, scratch tests, and intradermal skin testing. After negative prick or scratch tests, intradermal skin testing is performed with allergens that are suggested by the patient's history to be problematic. The back is the most suitable area of the body for skin testing because it permits the performance of many tests. A multitest applicator with multiple test heads is commercially available for simultaneous administration of antigens by multiple punctures at different sites. Research suggests that use of a multi-prong applicator for allergy testing decreases patient discomfort and application time (Pestotnik & Krueger, 2018). A negative response on a skin test cannot be interpreted as an absence of sensitivity to an allergen. Such a

response may occur with insufficient sensitivity of the test or with the use of an inappropriate allergen in testing. Therefore, it is essential to observe the patient undergoing skin testing for an allergic reaction even if the previous response was negative (Kowal & DuBuske, 2016).

Interpretation of Skin Test Results

Familiarity with and consistent use of a grading system are essential. The grading system used should be identified on a skin test record for later interpretation. A positive reaction, evidenced by the appearance of an urticarial wheal (round, reddened skin elevation) (see Fig. 33-3), localized **erythema** (diffuse redness) in the area of inoculation or contact, or pseudopodia (irregular projection at the end of a wheal) with associated erythema is considered indicative of sensitivity to the corresponding antigen. False-positive results may occur because of improper preparation or administration of allergen solutions (Kowal & DuBuske, 2016).



Quality and Safety Nursing Alert

Corticosteroids and antihistamines, including over-the-counter allergy medications, suppress skin test reactivity and should be stopped 48 to 96 hours before testing, depending on the duration of their activity. False-positive results may occur because of improper preparation or administration of allergen solutions.

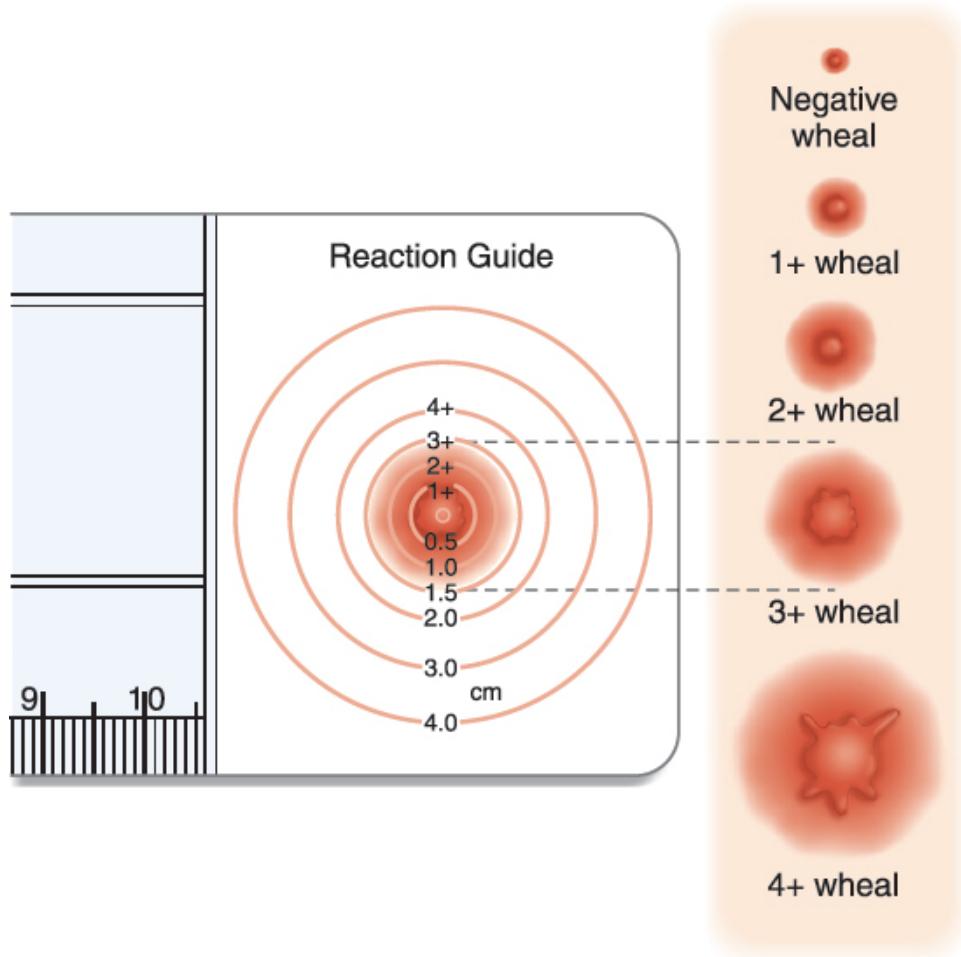


Figure 33-3 • Interpretation of reactions: Negative = wheal soft with minimal erythema; 1+ = wheal present (5 to 8 mm) with associated erythema; 2+ = wheal (7 to 10 mm) with associated erythema; 3+ = wheal (9 to 15 mm), slight pseudopodia possible with associated erythema; 4+ = wheal (12 mm+) with pseudopodia and diffuse erythema.

Positive or negative skin tests are interpreted in the context of a patient's history, clinical signs, and exposure to allergens. The following guidelines are used for the interpretation of skin test results:

- Skin tests are used most frequently with the diagnosis of allergic rhinitis.
- Negative test results are helpful in ruling out food allergy.
- Positive skin tests correlate highly with food allergy.

The use of skin tests to diagnose immediate hypersensitivity to medications is limited, because metabolites of medications, not the medications themselves, are usually responsible for causing hypersensitivity.

Provocative Testing

Provocative testing involves the direct administration of the suspected allergen to the sensitive tissue, such as the conjunctiva, nasal or bronchial mucosa, or gastrointestinal tract (by ingestion of the allergen), with observation of target organ response. This type of testing is helpful in identifying clinically significant allergens in patients who have a large number of positive tests. Major disadvantages of this type of testing are the limitation of one antigen per session and the risk of producing severe symptoms, particularly bronchospasm, in patients with asthma (Kowal & DuBuske, 2016).

Serum-Specific IgE Test

The serum-specific IgE test, formerly known as RAST, is an automated test performed on blood samples by a pathology laboratory. As the name suggests, it detects free antigen-specific IgE in serum as opposed to antigen-specific IgE bound to mast cells in the skin. The advantages of this test over other tests include decreased risk of systemic reaction, stability of antigens, and lack of dependence on skin reactivity modified by medications. The major disadvantages include limited allergen selection and reduced sensitivity compared with intradermal skin tests, lack of immediate results, and higher cost (Kowal & DuBuske, 2017).

ALLERGIC DISORDERS

There are two types of IgE-mediated allergic reactions: atopic and nonatopic disorders. Although the underlying immunologic reactions of the two types of disorders are the same, the predisposing factors and manifestations are different. Atopy is defined as the genetic predisposition to mount an IgE response to inhaled or ingested innocuous proteins. Atopic diseases consist of asthma, allergic rhinitis, and atopic dermatitis. All share a common pathogenesis, mediated by IgE, and are frequently present together in the same individual and in families. The nonatopic disorders lack the genetic component and organ specificity of the atopic disorders. Latex allergy (see later discussion) can present as an IgE-mediated anaphylaxis, type I reaction, or a type IV hypersensitivity referred to as contact dermatitis (Stokes & Casale, 2019).

Unfolding Patient Stories: Jennifer Hoffman • Part 2



Recall from [Chapter 20](#) Jennifer Hoffman, who came to the clinic with continued symptoms of asthma. The nurse confirmed that she was following the asthma action plan; however, Jennifer now returns to the clinic with worsening symptoms. What assessment findings would indicate to the nurse that Jennifer is having a hypersensitivity reaction to an allergen(s)? What questions can assist the nurse in identifying the causative agent(s)? What interventions should be implemented when allergen(s) are identified?

Care for Jennifer and other patients in a realistic virtual environment: **vSim for Nursing** (thepoint.lww.com/vSimMedicalSurgical). Practice documenting these patients' care in DocuCare (thepoint.lww.com/DocuCareEHR).

Anaphylaxis

Anaphylaxis is a clinical response to an immediate (type I hypersensitivity) immunologic reaction between a specific antigen and an antibody. The reaction results from a rapid release of IgE-mediated chemicals, which can induce a severe, life-threatening reaction (Actor, 2019).

Pathophysiology

Anaphylaxis is a type I IgE allergic reaction to an antigen, a foreign substance that has entered the body. It is caused by the cross-links of an allergen with allergen-specific IgE antibodies found on the surface membrane of mast cells and basophils, leading to cellular degranulation. The subsequent release of histamine and other bioactive mediators causes activation of platelets, eosinophils, and neutrophils. Histamine, prostaglandins, and inflammatory leukotrienes are potent vasoactive mediators that are implicated in the vascular permeability changes, flushing, **urticaria** (hives), angioedema, hypotension, and bronchoconstriction that characterize anaphylaxis. Smooth muscle spasm, bronchospasm, mucosal edema and inflammation, and increased capillary permeability result. Symptoms of anaphylaxis are sudden in onset and progress in severity over minutes to hours (Kemp, 2018).

Closely resembling anaphylaxis is an anaphylactoid reaction, which is caused by the release of mast cell and basophil mediators triggered by non-immunoglobulin E (IgE)-mediated events. This nonallergenic anaphylaxis reaction may occur with medications, food, exercise, or cytotoxic antibody transfusions. The reaction may be local or systemic. Local reactions usually

involve urticaria and angioedema at the site of the antigen exposure. Although possibly severe, nonallergenic anaphylaxis reactions are rarely fatal. Systemic reactions occur within about 30 minutes after exposure and involve cardiovascular, respiratory, gastrointestinal, and integumentary organ systems. For the most part, the treatment of nonallergenic anaphylaxis reaction is identical to that of anaphylaxis (Campbell & Kelso, 2018).

Common causes of anaphylaxis are listed in [Chart 33-2](#). Antibiotics and radiocontrast agents cause the most serious anaphylactic reactions. Penicillin is the most common medication to cause anaphylaxis. Approximately 10% of the population report having an allergy to penicillin. However, less than 5% have clinically significant IgE or T cell-mediated penicillin allergy (Shenoy, Macy, Rowe, et al., 2019).

Chart 33-2

Common Causes of Anaphylaxis

Foods

Peanuts, tree nuts (e.g., walnuts, pecans, cashews, almonds), shellfish (e.g., shrimp, lobster, crab), fish, milk, eggs, soy, wheat

Medications

Antibiotics, especially penicillin and sulfa antibiotics, allopurinol, radiocontrast agents, anesthetic agents (lidocaine, procaine), vaccines, hormones (insulin, vasopressin, adrenocorticotropic hormone), aspirin, nonsteroidal anti-inflammatory drugs

Other Pharmaceutical/Biologic Agents

Animal serums (tetanus antitoxin, snake venom antitoxin, rabies antitoxin), antigens used in skin testing

Insect Stings

Bees, wasps, hornets, yellow jackets, ants (including fire ants)

Latex

Medical and nonmedical products containing latex

Clinical Manifestations

Anaphylactic reactions produce a clinical syndrome that affects multiple organ systems. Reactions may be categorized as mild, moderate, or severe. The time from the exposure to the antigen to the onset of symptoms is a good indicator of the severity of the reaction—the faster the onset, the more severe the

reaction. The severity of previous reactions does not determine the severity of subsequent reactions, which could be the same or more or less severe. The severity depends on the degree of allergy and the dose of allergen exposure (Actor, 2019).

Mild systemic reactions consist of peripheral tingling and a sensation of warmth, possibly accompanied by a sensation of fullness in the mouth and throat. Nasal congestion, periorbital swelling, pruritus, sneezing, and tearing of the eyes can also be expected. The onset of symptoms begins within the first 2 hours after the exposure.

Moderate systemic reactions may include flushing, warmth, anxiety, and itching in addition to any of the milder symptoms. More serious reactions include bronchospasm and edema of the airways or larynx with dyspnea, cough, and wheezing. The onset of symptoms is the same as for a mild reaction (Kemp, 2018).

Severe systemic reactions have an abrupt onset with the same signs and symptoms described previously. These symptoms progress rapidly to bronchospasm, laryngeal edema, severe dyspnea, cyanosis, and hypotension. Dysphagia (difficulty swallowing), abdominal cramping, vomiting, diarrhea, and seizures can also occur. Cardiac arrest and coma may follow. Severe reactions are also referred to as anaphylactic shock (Marshall et al., 2018) (see Chapter 11).

Prevention

Strict avoidance of potential allergens is an important preventive measure for the patient at risk for anaphylaxis. Those at risk for anaphylaxis from insect stings should avoid areas populated by insects and should use appropriate clothing, insect repellent, and caution to avoid further stings.

If avoidance of exposure to allergens is impossible, an autoinjector system for epinephrine will be prescribed. The patient should be instructed to carry and administer epinephrine to prevent an anaphylactic reaction in the event of exposure to the allergen. People who are sensitive to insect bites and stings, those who have experienced food or medication reactions, and those who have experienced idiopathic or exercise-induced anaphylactic reactions should always carry an emergency kit that contains epinephrine. Autoinjection devices are commercially available for first aid and deliver premeasured doses of epinephrine (Comerford & Durkin, 2020). The autoinjector system requires no preparation, and the self-administration technique is not complicated. The patient must be given an opportunity to demonstrate the correct technique for use; a training device can be used for educating about the correct technique. Verbal and written information about the emergency kit, as well as strategies to avoid exposure to threatening allergens, must also be provided (Campbell & Kelso, 2018).

Screening for allergies before a medication is prescribed or first administered is an important preventive measure. A careful history of any sensitivity to suspected antigens must be obtained before administering any medication, particularly in parenteral form, because this route is associated with the most severe anaphylaxis. Nurses caring for patients in any setting (hospital, home, outpatient diagnostic testing sites, long-term care facilities) must assess patients' risks for anaphylactic reactions. Patients are asked about previous exposure to contrast agents used for diagnostic tests and any allergic reactions, as well as reactions to any medications, foods, insect stings, and latex. People who are predisposed to anaphylaxis should wear medical identification such as a bracelet or necklace, which identifies allergies to medications, food, and other substances.

People who are allergic to insect venom may require venom immunotherapy, which is used as a control measure and not a cure. The most common serious allergic reactions to insect stings are from the Hymenoptera family, which includes bees, ants, wasps, and yellow jackets (Warrell, 2019). Venom immunotherapy is an effective treatment for people with systemic reactions to an insect sting. It reduces the systemic reaction, reduces the risk of future large local reactions, and improves quality of life (Larsen, Broge, & Jacobi, 2016).

Patients with diabetes who are allergic to insulin and those who are allergic to penicillin may require desensitization. Desensitization is based on controlled anaphylaxis, with a gradual release of mediators. Patients who undergo desensitization are cautioned to avoid lapses in therapy, because this may lead to the reappearance of the allergic reaction when the use of the medication is resumed (Castells & Solensky, 2017).

Medical Management

Management depends on the severity of the reaction. Initially, respiratory and cardiovascular functions are evaluated. If the patient is in cardiac arrest, cardiopulmonary resuscitation (CPR) is instituted (Campbell & Kelso, 2018). Supplemental oxygen is provided during CPR or if the patient is cyanotic, dyspneic, or wheezing. Epinephrine, in a 1:1000 dilution, is given subcutaneously in the upper extremity or thigh and may be followed by a continuous intravenous infusion. Most adverse events associated with administration of epinephrine (i.e., adrenaline) occur when the dose is excessive or is given intravenously. Patients at risk for adverse effects include older patients and those with hypertension, arteriopathies, or known ischemic heart disease.

Antihistamines and corticosteroids should not be given in place of epinephrine. However, they may also be given as adjunct therapy (Campbell & Kelso, 2018).

Intravenous fluids (e.g., normal saline solution), volume expanders, and vasopressor agents are given to maintain blood pressure and normal hemodynamic status. In patients with episodes of bronchospasm or a history of bronchial asthma or chronic obstructive pulmonary disease, aminophylline and corticosteroids may also be given to improve airway patency and function. See [Chapter 11](#) for management of anaphylactic shock.

Patients who have experienced anaphylactic reactions and received epinephrine should be transported to the local emergency department (ED) for observation and monitoring because of the risk for a “rebound” or delayed reaction 4 to 8 hours after the initial allergic reaction. However, the observation time should be individualized based on the severity of the anaphylaxis. Longer periods of observation should be considered for patients who ingested the allergen, required more than one dose of epinephrine, had hypotension or pharyngeal edema, or have a history of asthma (Lieberman, 2018).

Nursing Management

If a patient is experiencing an allergic response, the nurse assesses the patient for signs and symptoms of anaphylaxis. Airway, breathing pattern, and vital signs are assessed. The patient is observed for signs of increasing edema and respiratory distress. Prompt notification of the rapid response team, the provider, or both is required. Rapid initiation of emergency measures (e.g., intubation, administration of emergency medications, insertion of intravenous lines, fluid administration, and oxygen administration) is important to reduce the severity of the reaction and to restore cardiovascular function. The nurse documents the interventions used and the patient’s vital signs and response to treatment (Campbell & Kelso, 2018).

The patient who has recovered from anaphylaxis needs to be educated about what occurred, how to avoid future exposure to antigens, and how to administer emergency medications to treat anaphylaxis. The nurse assesses the health literacy level of the patient and determines the best method of providing discharge instructions (Wilkin, 2020). See the Nursing Research Profile in [Chart 33-3](#).

Patients who have experienced an anaphylactic reaction should receive a prescription for an autoinjectable epinephrine device. The nurse educates the patient and family in their use and has the patient and family return demonstrate correct administration (Moore, Kemp, & Kemp, 2015) (see [Chart 33-4](#)).

Chart 33-3



NURSING RESEARCH PROFILE

Using Video Discharge Instructions

Wilkin, Z. L. (2020). Effects of video discharge instructions on patient understanding. *Advanced Emergency Nursing Journal*, 42(1), 71–78.

Purpose

Patients have difficulty understanding and retaining education at the time of discharge, particularly if instructions are given only in the form of printed materials. The purpose of this study was to evaluate the effects of using video discharge instructions on patient understanding of the education provided upon discharge from an emergency department (ED).

Design

This was a prospective, randomized, controlled trial that used a convenience sample of patients admitted to the ED. Participants were randomized to receive either standard discharge procedures, or standard discharge procedures plus video discharge instructions. Ten minutes after receiving one of the education methods, participants were tested with a 5-question multiple choice test.

Findings

Participants included 60 adults with a mean age of 37 years, and with a medical diagnosis of either upper respiratory infection, pharyngitis, or gastroenteritis. Thirty participants received the video discharge instructions, while the other 30 received the standard discharge procedure. There was a significant difference in knowledge level, with those receiving the video instructions having a higher level of knowledge (4.53 vs. 4, $p = 0.009$) on the multiple choice test.

Nursing Implications

This study provides evidence that video discharge instructions have the potential to increase the knowledge level of patients receiving discharge education. Nurses who work in busy settings, such as EDs, should consider developing supplemental materials to augment standard written discharge instructions to help increase patients' understanding and retention of important information.

Chart 33-4 PATIENT EDUCATION

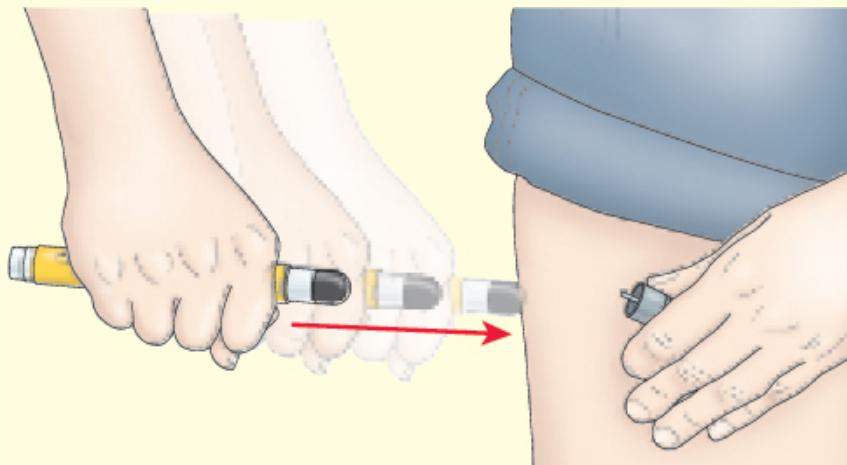
Self-Administration of Epinephrine

The nurse instructs the patient to:

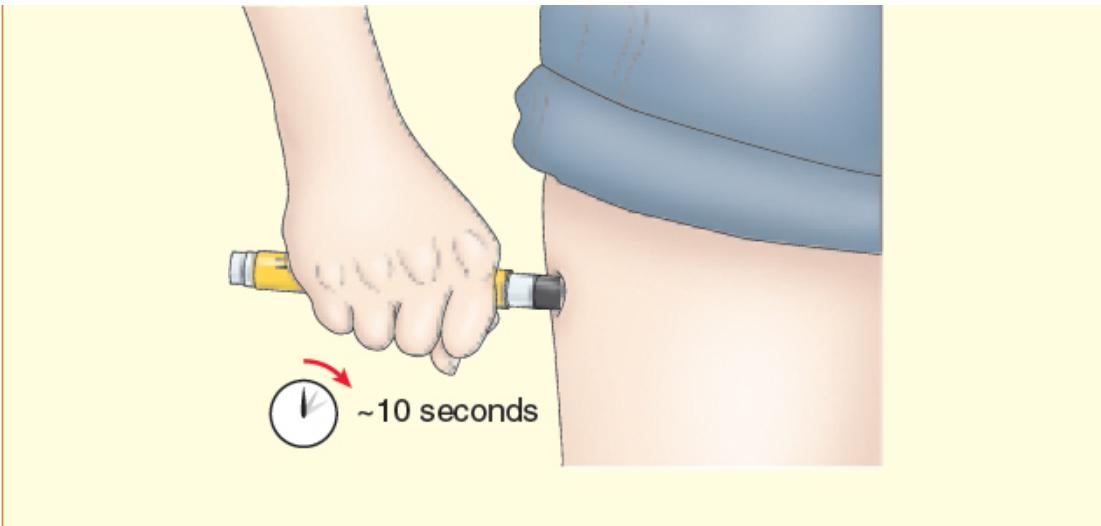
1. After removing the autoinjector from its carrying tube, grasp the unit with the orange tip (injecting end) pointing downward. Form a fist around the unit with the orange tip down; with your other hand, remove the blue safety release cap.



2. Hold the black tip near outer thigh. Swing and jab firmly into the outer thigh until a click is heard with the device perpendicular (90-degree angle) to the thigh. Do NOT inject into buttocks.



3. Hold firmly against the thigh for approximately 10 seconds. Remove the unit from the thigh, and gently massage the injection area for 10 seconds. Call 911 and seek immediate medical attention. Carefully place the used autoinjector unit, needle-end first, into the device storage tube without bending the needle. Screw on the storage tube completely, and take it with you to the hospital emergency room.



Allergic Rhinitis

Allergic rhinitis (hay fever, seasonal allergic rhinitis) is the most common form of respiratory allergy, which is mediated by an immediate (type I hypersensitivity) immunologic reaction. In the United States approximately 20 million adults have allergic rhinitis (CDC, 2017). Early diagnosis and adequate treatment are essential to reduce complications and relieve symptoms.

Because allergic rhinitis is induced by airborne pollens or molds, it is characterized by the following seasonal occurrences (deShazo & Kemp, 2018a):

- Early spring—tree pollen
- Early summer—grass pollen
- Early fall—weed pollen (ragweed)

Each year, attacks begin and end at about the same time. Airborne mold spores require warm, damp weather. Although there is no rigid seasonal pattern, these spores appear in early spring, are rampant during the summer, then taper off and disappear by the first frost in areas that experience dramatic seasonal temperature variation. In temperate areas that do not experience freezing temperatures, these allergens, especially mold, can persist throughout the year.

Pathophysiology

Allergic rhinitis is caused by an allergen-specific IgE-mediated immunologic response. Sensitization most commonly begins by inhalation of antigen. IgE antibodies are stimulated and bind to mast cells in the respiratory mucosa, basophils in the peripheral blood, and eosinophils in the nasal and respiratory mucosa. Eosinophilia in the tissues is the key characteristic of allergic rhinitis.

When IgE antibodies bind to mast cells there is histamine release. Histamine is the major mediator of allergic reactions in the nasal mucosa. Inflammation, tissue edema, vasodilation, and increased capillary permeability occur (deShazo & Kemp, 2017).

Clinical Manifestations

Symptoms include sneezing, rhinorrhea, nasal itching, conjunctivitis, and nasal obstruction. Postnasal drip, cough, itching of the eyes, and fatigue are often present. If symptoms are severe, allergic rhinitis may interfere with sleep, leisure, school, work, and overall quality of life. Allergic rhinitis is commonly associated with chronic sinusitis, atopic dermatitis (eczema), and asthma. Infraorbital edema and dilation of peripheral vessels (due to histamine) can cause darkening under the eyes, sometimes referred to as “allergic shiners.” A horizontal nasal crease can develop from constant rubbing of the nose; commonly referred to as the “allergic salute.” The nasal mucosa can exhibit a grayish hue. Clear rhinorrhea, tonsillar hyperplasia, and postnasal rhinorrhea may be visible on examination of the throat. Tympanic membranes can be retracted or serous fluid may accumulate in the middle ear. Some patients can suffer oral allergy syndrome (OAS) with itching and irritation of the hard and soft palate. Allergic rhinitis and sinusitis can trigger migraine headache in some patients (deShazo & Kemp, 2018a).

Assessment and Diagnostic Findings

Diagnosis of seasonal allergic rhinitis is mainly based on history and physical examination. Blood or laboratory testing is usually unnecessary. Diagnostic testing can be done with immediate hypersensitivity skin testing (skin prick testing). In sensitive patients, testing with select diagnostic solutions of tree, grass, or weed pollen, mold, house dust mites, and animal allergens results in a wheal-and-flare reaction at the skin test site within 20 minutes. IgE immunoassay can provide similar information to skin prick test. Nasal cytology can be performed to differentiate rhinitis due to allergy from that due to infection, although it is relatively nonspecific and insensitive (deShazo & Kemp, 2018a).

Medical Management

The goal of therapy is to provide relief from symptoms. Therapy may include one or all of the following interventions: avoidance therapy, pharmacologic therapy, and immunotherapy. Verbal instructions must be reinforced by written information. Knowledge of general concepts regarding assessment and therapy

in allergic diseases is important so that the patient can learn to manage certain conditions as well as prevent severe reactions and illnesses.

Avoidance Therapy

In avoidance therapy, every attempt is made to remove the allergens that act as precipitating factors. Simple measures and environmental controls are often effective in decreasing symptoms. Examples include the use of air conditioners, air cleaners, humidifiers, and dehumidifiers; removal of dust-catching furnishings, carpets, and window coverings; removal of pets from the home or bedroom; the use of pillow and mattress covers that are impermeable to dust mites; and a smoke-free environment (Platts-Mills, 2019). Additional measures include changing clothing when coming in from outside, showering to wash allergens from hair and skin, and using an over-the-counter nasal irrigation device or saline nasal spray to reduce allergens in the nasal passages. Because multiple allergens are often implicated, multiple measures to avoid exposure to allergens are often necessary. High-efficiency particulate air (HEPA) purifiers and vacuum cleaner filters may also be used to reduce allergens in the environment (Platts-Mills, 2019). Multiple avoidance strategies tailored to a person's risk factors can reduce the severity of symptoms, the number of work or school days missed because of symptoms, and the number of unscheduled health care visits for treatment. In many cases, it is impossible to avoid exposure to all environmental allergens, so pharmacologic therapy or immunotherapy is needed.

Pharmacologic Therapy

Many antihistamines, corticosteroid nasal sprays, adrenergic agents, mast cell stabilizers, nasal decongestant sprays, and corticosteroids are pharmacologic agents used in allergic rhinitis.

Antihistamines

First-generation antihistamines, which are readily absorbed, are most effective when administered orally at the first occurrence of symptoms because they prevent the development of new symptoms. Diphenhydramine is a common first-generation antihistamine available in over-the-counter (OTC) medications. Others include chlorpheniramine and hydroxyzine. First-generation antihistamines bind to histamine 1 receptors and can effectively relieve symptoms of hay fever, vasomotor rhinitis, urticaria (hives), and mild asthma. However, first-generation antihistamines cross the blood–brain barrier and cause significant sedation which impairs cognitive function and psychomotor performance. First-generation antihistamines can also have anticholinergic effects such as dry mouth, blurry vision, urinary hesitancy, and confusion. Due to their anticholinergic effects, first-generation antihistamines

should be avoided in older adults (Fick, Semla, Steinman, et al., 2019). First-generation antihistamines have a limited role in the treatment of allergy due to their adverse effects (deShazo & Kemp, 2018b).

Second-generation (nonsedating H₁-receptor antagonists) are antihistamines that do not cross the blood–brain barrier to the same extent as first-generation antihistamines. They mainly bind to peripheral rather than central nervous system H₁ receptors, causing less sedation. Examples of these OTC medications are loratadine, cetirizine, and fexofenadine (Sanchez-Borges & Ansotegui, 2019). Contraindications, major side effects, nursing implications, and patient education for select H₁ antihistamines can be found in [Table 33-2](#).

If H₁ antihistamines are not completely effective, H₂ antihistamines such as famotidine, which blocks the H₂ receptors found in the stomach, vascular smooth muscle, and elsewhere, can be added to the drug regimen. It is given twice a day with the same total dose as for gastroesophageal reflux. H₂ antihistamines do not relieve urticaria on their own, but can augment the effect of H₁ antihistamines (Randall & Hawkins, 2018).

Antihistamines may also be combined with decongestants to reduce the nasal congestion associated with allergies. Most combination products are available as OTC medications; examples are loratadine/pseudoephedrine and cetirizine/pseudoephedrine. Decongestants can cause an increase in blood pressure; therefore, patients with a history of hypertension should be cautioned about long-term use of any medication that contains decongestants (Randall & Hawkins, 2018). Decongestants are contraindicated in patients receiving monoamine oxidase inhibitor therapy and should be used with caution in patients with closed-angle glaucoma, cardiovascular or cerebrovascular disease, hyperthyroidism, or bladder neck obstruction.

Antihistamine nasal sprays azelastine and olopatadine are available by prescription. They not only reduce inflammation and decrease nasal congestion, but also have rapid onset of action and can be used “on demand” (deShazo & Kemp, 2018b).

Corticosteroid Nasal Spray

Corticosteroid nasal sprays are recognized as the most effective pharmacotherapy for allergic rhinitis. These anti-inflammatory agents work directly on the nasal mucosa but take a few hours to work. The patient should understand that maximal therapeutic effectiveness of these agents can take 1 to 2 weeks. They are recommended as the best single therapy for patients with mild to moderate or moderate to severe symptoms (Berger & Melizer, 2015).

Corticosteroid nasal sprays are derived from hydrocortisone and are divided into first-generation and second-generation agents. Beclomethasone, flunisolide, triamcinolone, and budesonide are first-generation agents, whereas

fluticasone, mometasone, and ciclesonide are second-generation agents (deShazo & Kemp, 2018b).

Corticosteroid nasal spray therapy should begin with the maximal dose for patient age. Once patient symptoms are adequately controlled, the dose can be reduced at 1-week intervals to the lowest effective dose that controls symptoms. Patients with severe symptoms usually need daily use of the nasal spray. Some patients may find relief of symptoms with use of the nasal spray every other day or as needed. Newer second-generation corticosteroid spray preparations act rapidly (within 3 to 12 hours), and as-needed use appears to be effective. This kind of treatment may be adequate in patients with episodic symptoms (deShazo & Kemp, 2018b).

Adverse effects of corticosteroid nasal sprays are mild and include drying of the nasal mucosa and burning and itching sensations caused by the vehicle used to administer the medication. Beclomethasone, budesonide, flunisolide, and triamcinolone are deactivated rapidly after absorption, so they do not achieve significant blood levels. Inhaled corticosteroids do not affect the immune system to the same degree as systemic corticosteroids (i.e., oral corticosteroids). Because corticosteroids are inhaled into the upper respiratory tract, tuberculosis or untreated bacterial infections of the lungs may become apparent and progress. Whenever possible, patients with tuberculosis or other bacterial infections of the lungs should avoid corticosteroid nasal sprays (deShazo & Kemp, 2018b).

The combination of an antihistamine and a corticosteroid nasal spray may be helpful for patients who do not obtain sufficient relief with one agent. A combination spray containing azelastine and fluticasone is available (deShazo & Kemp, 2018b).

Adrenergic Agents

Alpha-adrenergic agonist (also called sympathomimetic) medications, such as pseudoephedrine, can be used as decongestants in allergies. These agents activate alpha-adrenergic receptor sites on the smooth muscle causing vasoconstriction of the nasal mucosal blood vessels, reducing local blood flow, fluid exudation, and mucosal edema. Oral alpha-adrenergic medications have a systemic effect and are not recommended as first-line treatment of allergic rhinitis (Laccourreye, et al., 2015). Oral alpha-adrenergic agents are also available in combination with antihistamines (e.g., diphenhydramine and pseudoephedrine fexofenadine and pseudoephedrine). Alpha-adrenergic topical nasal spray (e.g., oxymetazoline) is also available. Alpha-adrenergic agonist ophthalmic drops, such as tetrahydrozoline, are commonly used for allergic conjunctivitis as they vasoconstrict the blood vessels of the eye.

There are many potential side effects of oral alpha-adrenergic agonists. These include hypertension, arrhythmias, palpitations, central nervous system stimulation, irritability, tremor, and tachyphylaxis. Oral adrenergic agents are

not first-line agents in allergy and have potential for cardiovascular and neurological adverse effects (deShazo & Kemp, 2018b). The topical preparations (i.e., drops and sprays) of alpha-adrenergic agonists cause fewer side effects than oral medications; however, the use of drops and sprays should be limited to a few days to avoid rebound congestion, also referred to as rhinitis medicamentosa.

The Combat Methamphetamine Epidemic Act of 2005 banned OTC sales of medications that contain the ingredients pseudoephedrine, ephedrine, or phenylpropanolamine as these can be used to make methamphetamine, a highly addictive stimulant. Laws to enforce this act vary state by state. There is a legal age requirement to purchase these drugs and pharmacists must limit and keep records of the amount purchased. Some states require a prescription (U.S. Food and Drug Administration [FDA], 2017).

TABLE 33-2



Select H₁ Antihistamines

H ₁ Antihistamine	Contraindications	Major Side Effects	Nursing Implications and Patient Education
First-Generation H₁ Antihistamines (Sedating)			
Diphenhydramine	Allergy to any antihistamines Third trimester of pregnancy Lactation Use cautiously with narrow-angle glaucoma, asthma, stenosing peptic ulcer, benign prostatic hyperplasia (BPH) or bladder neck obstruction, first and second trimester of pregnancy, older patients, hypertension	Drowsiness, confusion, dizziness, dry mouth, nausea, vomiting, photosensitivity, urinary retention	Administer with food if gastrointestinal (GI) upset occurs. Caution patients to avoid alcohol, driving, or engaging in any hazardous activities until central nervous system (CNS) response to medication is stabilized. Suggest sucking on sugarless lozenges or ice chips for relief of dry mouth. Encourage the use of sunscreen and hat while outdoors. Assess for urinary retention; monitor urinary output.
Chlorpheniramine	Allergy to any antihistamines Third trimester of pregnancy Lactation Use cautiously with narrow-angle glaucoma, asthma, stenosing peptic ulcer, BPH or bladder neck obstruction, first and second trimesters of pregnancy, older patients, hypertension	Drowsiness, sedation, and dizziness, although less than other sedating agents; confusion, dry mouth, nausea, vomiting, urinary retention, epigastric distress, thickening of bronchial secretions	Caution patients to avoid alcohol, driving, or engaging in any hazardous activities until CNS response to medication is stabilized. Suggest sucking on sugarless lozenges or ice chips for relief of dry mouth. Recommend the use of a humidifier.
Hydroxyzine	Allergy to hydroxyzine or cetirizine, pregnancy, lactation, hypertension	Drowsiness; dry mouth; involuntary motor activity, including	Caution patients to avoid alcohol, driving, or engaging in any hazardous activities until CNS response to medication is stabilized. Suggest sucking

		tremor and seizures	on sugarless lozenges or ice chips for relief of dry mouth. Instruct patients to report tremors.
Second-Generation H₁ Antihistamines (Nonsedating)			
Cetirizine	Allergy to any antihistamines Narrow-angle glaucoma Asthma Stenosing peptic ulcer BPH or bladder neck obstruction Lactation Hypertension	Dry nasal mucosa, thickening of bronchial secretions	Can be taken without regard to meals. Instruct patients to use caution if driving or performing tasks that require alertness. Recommend the use of a humidifier.
Desloratadine	Allergy to loratadine Lactation Use cautiously with renal or hepatic impairment, pregnancy, hypertension	Somnolence, nervousness, dizziness, fatigue, dry mouth	Can be taken without regard to meals. Suggest sucking on sugarless lozenges or ice chips for relief of dry mouth. Recommend the use of a humidifier.
Loratadine	Allergy to any antihistamines Narrow-angle glaucoma Asthma Stenosing peptic ulcer BPH or bladder neck obstruction Hypertension	Headache, nervousness, dizziness, depression, edema, increased appetite	Instruct patients to take on empty stomach (1 h before or 2 h after meals or food). Instruct patients to avoid alcohol and to use caution if driving or performing tasks that require alertness. Suggest sucking on sugarless lozenges or ice chips for relief of dry mouth. Recommend the use of a humidifier.
Fexofenadine	Allergy to any antihistamines Pregnancy Lactation Use cautiously with hepatic or renal impairment, older patients, hypertension	Fatigue, drowsiness, GI upset	Should not be given within 15 min of ingestion of antacids. Instruct patients to use caution if driving or performing tasks that require alertness. Recommend the use of a humidifier.
Levocetirizine	Hypersensitivity to	Drowsiness, GI	Can be taken without regard

any antihistamines	disturbance, headache	to meals. Instruct patients to use caution if driving or performing tasks that require alertness.
End-stage kidney disease		
Hemodialysis		
Use cautiously with pregnancy, lactation, older patients		Recommend the use of a humidifier.

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer.

Mast Cell Stabilizers

Intranasal cromolyn sodium is a spray that acts by stabilizing the mast cell membrane, thus reducing the release of histamine and other mediators of the allergic response. In addition, it inhibits macrophages, eosinophils, monocytes, and platelets involved in the immune response. Cromolyn interrupts the physiologic response to nasal antigens, and it is used prophylactically (before the exposure to allergens) to prevent the onset of symptoms and to treat symptoms once they occur. It is also used therapeutically in chronic allergic rhinitis. This spray is as effective as antihistamines but is less effective than corticosteroid nasal sprays in the treatment of seasonal allergic rhinitis. It is best to use this agent 30 minutes prior to exposure to allergen. Frequent use is necessary to obtain an effect (1 to 2 nasal sprays 4 times per day). The patient must be informed that the beneficial effects of the medication may take a week or longer to manifest. The medication is of no benefit in the treatment of nonallergic rhinitis. Adverse effects (e.g., sneezing, local stinging and burning sensations) are usually mild (deShazo & Kemp, 2018b).

Nasal Decongestant Sprays

Nasal decongestant sprays include phenylephrine, oxymetazoline, and naphazoline. These agents vasoconstrict blood vessels in the nasal mucosa by blocking alpha-adrenergic receptors. Nasal decongestant sprays are not recommended as monotherapy. After using these agents for 3 to 7 days, reduced sensitivity of alpha-adrenergic receptors develops which can cause worsening nasal congestion. This effect of worsening nasal congestion often causes patients to overuse these agents with less and less therapeutic effect. This is termed rhinitis medicamentosa and can lead to increased need for the agent and eventual dependency (Wahid & Shermetaro, 2019).

The combination of a topical nasal decongestant and topical corticosteroid may effectively treat symptoms without causing rhinitis medicamentosa. Nasal decongestants are helpful when used just before air travel in patients who have difficulties with middle ear or sinus equilibration with flying or in patients who have problems with altitude changes (deShazo & Kemp, 2018b).

Corticosteroids

Oral and parenteral corticosteroids can be used when conventional therapy has failed and symptoms are severe and of short duration. They can control symptoms of allergic reactions such as hay fever, medication-induced allergies, and allergic reactions to insect stings. However, corticosteroids have a delayed onset of action and cannot be used for immediate relief of allergic symptoms. The agents are not effective as singular treatment for severe allergic reactions such as anaphylaxis (deShazo & Kemp, 2018b). Oral corticosteroids in the lowest dose to control symptoms for the shortest period of time may be prescribed if other agents fail. However, corticosteroids are usually avoided because of their side effects.



Quality and Safety Nursing Alert

Patients who receive high-dose or long-term corticosteroid therapy must be cautioned not to stop taking the medication suddenly. Doses are tapered when discontinuing this medication to avoid adrenal insufficiency.

The patient should be cautioned about side effects, which include fluid retention, weight gain, hypertension, gastric irritation, glucose intolerance, osteoporosis, immunosuppression, and adrenal suppression. Further discussion of corticosteroids is provided in [Chapter 45, Table 45-3](#).

Leukotriene Receptor Antagonists

Leukotrienes are inflammatory mediators that cause bronchospasm, vascular permeability, and activation of leukocytes. They are three to four times more potent than histamine in perpetuating inflammation in the upper respiratory tract (Castells, 2017). Leukotriene receptor antagonists (LTRAs), such as zafirlukast and montelukast, block the synthesis or action of leukotrienes and prevent the signs and symptoms associated with asthma (see [Table 33-3](#)). LTRAs are also used to counteract allergic rhinitis.

Montelukast efficacy has been compared to a second-generation antihistamine agent. In combination with a second-generation antihistamine such as loratadine, montelukast has shown to be more effective than montelukast alone. Adverse effects of montelukast include neuropsychiatric changes such as anxiety, depression, and insomnia. This agent may be not appropriate for patients with preexisting mood disorders (Badri & Takov, 2019).

Leukotriene receptor antagonists are for long-term use, and patients should be advised to take their medication daily. LTRAs are not effective as “rescue” medications. Patients take appropriate “rescue” medications for symptom

exacerbation but continue to take the LTRA on a daily basis. Studies report that using an LTRA in conjunction with an inhaled corticosteroid is effective for mild persistent asthma (Chauchan, Jeyaraman, Singh Mann, et al., 2017).

Allergen Immunotherapy

Allergen immunotherapy (AIT) is primarily used to treat IgE-mediated diseases by injections of allergen extracts. Immunotherapy, also referred to as allergy vaccine therapy, involves the administration of gradually increasing quantities of specific allergens to the patient until a dose is reached that is effective in reducing disease severity from natural exposure. This is an effective treatment for 80% to 90% of certain allergens such as grass and pollen. This type of therapy provides an adjunct to symptomatic pharmacologic therapy and can be used when avoidance of allergens is not possible. Specific immunotherapy has been used in the treatment of allergic disorders for many years. Goals of immunotherapy include reducing the level of circulating IgE, increasing the level of blocking antibody IgG, and reducing mediator cell sensitivity. Immunotherapy has been most effective for ragweed pollen, grass, tree pollen, cat dander, and house dust mite allergens (Akdis, 2018). Indications and contraindications for immunotherapy are presented in [Chart 33-5](#).

TABLE 33-3  Leukotriene Receptor Antagonists

Leukotriene Receptor Antagonist	Available Formulations	Frequency of Dosing
Zafirlukast	Tablets: 10 mg; 20 mg	Taken twice a day
Montelukast	Tablets: 10 mg Chewable tablets: 4 mg; 5 mg Granules: 4 mg/packet	Taken once a day in PM
Zileuton	Tablets: 600 mg extended release	Taken twice a day within 1 h after morning and evening meals

Chart 33-5

IMMUNOTHERAPY: Indications and Contraindications

Indications

- Allergic rhinitis, conjunctivitis, or allergic asthma
- History of a systemic reaction to Hymenoptera and specific immunoglobulin E antibodies to Hymenoptera venom
- Desire to avoid the long-term use, potential adverse effects, or costs of medications
- Lack of control of symptoms by avoidance measures or the use of medications

Contraindications

- The use of beta-blocker or angiotensin-converting enzyme inhibitor therapy, which may mask early signs of anaphylaxis
- Presence of significant pulmonary or cardiac disease or organ failure
- Inability of the patient to recognize or report signs and symptoms of a systemic reaction
- Nonadherence of the patient to other therapeutic regimens and nonlikelihood that the patient will adhere to the immunization schedule (often weekly for an indefinite period)
- Inability to monitor the patient for at least 30 minutes after administration of immunotherapy
- Absence of equipment or adequate personnel to respond to allergic reaction if one occurs

Correlation of a positive skin test with a positive allergy history is an indication for immunotherapy if the allergen cannot be avoided. The benefit of immunotherapy has been fairly well established in instances of allergic rhinitis and bronchial asthma that are clearly due to sensitivity to one of the common pollens, molds, or household dust. Unlike antiallergy medication, allergen immunotherapy has the potential to alter the allergic disease course after 3 to 5 years of therapy. Because it may prevent the progression or development of asthma or multiple or additional allergies, it is also considered to be a potential preventive measure. The patient must understand what to expect and the importance of continuing therapy for several years before immunotherapy is accomplished. When skin tests are performed, the results are correlated with symptoms; treatment is based on the patient's needs rather than on the results of skin tests (Klimek, Pfaar, Bousquet, et al., 2017).

There are three methods of immunotherapy: subcutaneous immunotherapy (SCIT), sublingual immunotherapy (SLIT), and epicutaneous immunotherapy (EPIT).

Subcutaneous Immunotherapy

The most common method of treatment is SCIT, which consists of the serial injection of one or more antigens that are selected in each particular case on the basis of skin testing. This method provides a simple and efficient technique for targeting IgE antibodies to specific antigens. Specific treatment consists of injecting extracts of the allergens that cause symptoms in a particular patient. Injections begin with very small amounts and are gradually increased, usually at weekly intervals, until a maximum tolerated dose is attained. Although severe systemic reactions are rare, the risk of systemic and potentially fatal anaphylaxis exists. It tends to occur most frequently at the induction or “up-dosing” phase. Therefore, the patient must be monitored after administration of immunotherapy. Because of the risk of anaphylaxis, injections should not be given by a lay person or by the patient. The patient must remain in the office or clinic for at least 30 minutes after the injection and is observed for possible systemic symptoms. If a large, local swelling develops at the injection site, the next dose should not be increased, because this may be a warning sign of a possible systemic reaction (James & Bernstein, 2017).

Maintenance booster injections are given at 2- to 4-week intervals, frequently for a period of several years, before the maximum benefit is achieved, although some patients will note early improvement in their symptoms. Long-term benefit seems to be related to the cumulative dose of vaccine given over time (Nelson, 2018).



Quality and Safety Nursing Alert

Because the injection of an allergen may induce systemic reactions, such injections are given only in a setting where epinephrine is immediately available (i.e., primary provider's office, clinic).

Sublingual Immunotherapy

SLIT has been reported to have a 30% to 40% reduction in reactions and rescue medication usage in seasonal allergic rhinitis. Administration of SLIT includes a buildup phase that is followed by a treatment plan of three times per week with a rapid dissolving tablet or liquid containing allergen extracts. Recent studies show comparable efficacy of SLIT with SCIT (Chaabani, Mansi, Tripple, et al., 2019; Durham & Penagos, 2016). Systemic side effects are rare but have been reported in patients who also report systemic reactions with SCIT. Side effects include irritation, minor swelling or itching inside the mouth, and stomach upset and nausea.

Epicutaneous Immunotherapy

EPIT is an investigational alternative allergen immunotherapy with delivery of the allergen to the epidermis. Because the epidermis is less vascular, it is theorized that there is reduced risk of systemic allergic side effect. Epicutaneous allergen patches are applied to the upper inner arm or interscapular region of the back. Doses are lower than those used in SCIT or SLIT. The allergen is soluble and absorbed into the skin. Adverse reactions include localized erythema, pruritus, and urticaria at the site of allergen patch. Studies of EPIT have shown modest results compared to placebo, and there has been a higher rate of treatment-related mild to moderate anaphylactic reactions. Long-term studies of EPIT are currently in progress for peanut allergies (Nowak-Wegrzyn, 2019).

Immunotherapy should not be initiated during pregnancy; for patients who have been receiving immunotherapy before pregnancy, the dosage should not be increased during pregnancy.

Therapeutic failure is evident when a patient does not experience a decrease of symptoms within 12 to 24 months, fails to develop increased tolerance to known allergens, and cannot decrease the use of medications to reduce symptoms. Potential causes of treatment failure include misdiagnosis of allergies, inadequate doses of allergen, newly developed allergies, and inadequate environmental controls (Akdis, 2018).

NURSING PROCESS

The Patient with Allergic Rhinitis

Assessment

The examination and history of the patient reveal sneezing, often in paroxysms; thin and watery nasal discharge; itching eyes and nose; lacrimation; and occasionally headache. The health history includes a personal or family history of allergy. The allergy assessment identifies the nature of antigens, seasonal changes in symptoms, and medication history. The nurse also obtains subjective data about how the patient feels just before symptoms become obvious, such as the occurrence of pruritus, breathing problems, and tingling sensations. In addition to these symptoms, hoarseness, wheezing, hives, rash, erythema, and edema are noted. Any relationship between emotional problems or stress and the triggering of allergy symptoms is assessed (Kakli & Riley, 2016).

Diagnosis

NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses may include:

- Impaired breathing associated with allergic reaction
- Lack of knowledge about allergy and the recommended modifications in lifestyle and self-care practices
- Difficulty coping with chronicity of condition and need for environmental modifications

COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Anaphylaxis
- Impaired breathing
- Nonadherence to the therapeutic regimen

Planning and Goals

The goals for the patient may include restoration of a breathing pattern that provides adequate ventilation, increased knowledge about the causes and control of allergic symptoms, improved coping with alterations and modifications, and absence of complications.

Nursing Interventions

IMPROVING BREATHING PATTERN

The patient is instructed and assisted to modify the environment to reduce the severity of allergic symptoms or to prevent their occurrence. The patient is also instructed to reduce exposure to people with upper respiratory tract

infections. Adherence to medication schedules and other treatment regimens is encouraged and reinforced.

PROMOTING UNDERSTANDING OF ALLERGY AND ALLERGY CONTROL

Instruction includes strategies to minimize exposure to allergens and explanation about desensitization procedures and correct use of medications. The nurse informs and reminds the patient of the importance of keeping appointments for desensitization procedures, because dosages are usually adjusted on a weekly basis, and missed appointments may interfere with the dosage adjustment (Pitsios & Dietis, 2019).

Patients need to understand the difference between rescue medications for allergy exacerbation and seasonal flares (e.g., antihistamines) and medications used for allergy control throughout the year (e.g., inhaled corticosteroids, leukotriene modifiers). Patients also need to understand that medications for allergy exacerbation and seasonal flares should be used only when the allergy is apparent. Continued use of these medications when not required can result in tolerance; consequently, the medications will be ineffective when needed (Pitsios & Dietis, 2019; Scadding, 2017).

COPING WITH A CHRONIC DISORDER

Although allergic reactions are infrequently life-threatening, they require vigilance to avoid allergens and modification of the lifestyle or environment to prevent recurrence of symptoms. Allergic symptoms are often present year-round and create discomfort and inconvenience for the patient. Although patients may not feel ill during allergy seasons, they often do not feel well, either. The need to be alert for possible allergens in the environment may be tiresome, placing a burden on the patient's ability to lead a normal life. Stress related to these difficulties may in turn increase the frequency or severity of symptoms. To assist the patient in adjusting to these modifications, the nurse must have an appreciation of the difficulties encountered by the patient. The patient is encouraged to verbalize feelings and concerns in a supportive environment and to identify strategies to deal with them effectively (Scadding, 2017).

MONITORING AND MANAGING POTENTIAL COMPLICATIONS

Anaphylaxis and Impaired Breathing. Respiratory and cardiovascular functioning can be significantly altered during allergic reactions by the reaction itself or by the medications used to treat reactions. Anaphylaxis is an acute, systemic reaction that causes vasodilation and bronchiole constriction which can lead to hypotensive shock and asphyxiation. The respiratory status is evaluated by monitoring the respiratory rate and pattern and by assessing for breathing difficulties or abnormal lung sounds. The pulse rate and rhythm and blood pressure are monitored to assess cardiovascular status regularly or any time the patient reports symptoms such as itching or difficulty breathing. In the event of signs and symptoms

suggestive of anaphylaxis, emergency medications and equipment must be available for immediate use. People with severe allergic reactions may be advised to carry an autoinjectable epinephrine device (Song & Lieberman, 2019). See [Chapter 11](#) for treatment of anaphylactic shock.

Nonadherence to the Therapeutic Regimen. Knowledge about the treatment regimen does not ensure adherence. Having the patient identify potential barriers and explore acceptable solutions for effective management of the condition (e.g., installing tile floors rather than carpet, not gardening in the spring) can increase adherence to the treatment regimen (Pitsios & Dietis, 2019).

PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE



Educating Patients About Self-Care. The patient is instructed about strategies to minimize exposure to allergens, the actions and adverse effects of medications, and the correct use of medications. The patient should know the name, dose, frequency, actions, and side effects of all medications taken.

Instruction about strategies to control allergic symptoms is based on the needs of the patient as determined by the results of tests, the severity of symptoms, and the motivation of the patient and family to deal with the condition (Pitsios & Dietis, 2019). Suggestions for patients who are sensitive to dust and mold in the home are given in [Chart 33-6](#).

If the patient is to undergo allergen immunotherapy (AIT), the nurse reinforces the primary provider's explanation regarding the purpose and procedure. Instructions are given regarding the series of injections, which usually are given initially every week and then at 2- to 4-week intervals. These instructions include remaining in the primary provider's office or the clinic for at least 30 minutes after the injection, so that emergency treatment can be given if the patient has a reaction; avoiding rubbing or scratching the injection site; and continuing with the series for the period of time required. In addition, the patient and family are instructed about emergency treatment of severe allergic symptoms (James & Bernstein, 2017).

Because antihistamines may produce drowsiness, the patient is cautioned about this and other side effects applicable to the medication. Operating machinery, driving a car, and performing activities that require intense concentration should be postponed. The patient is also informed about the dangers of drinking alcohol when taking antihistamines, because they tend to exaggerate the effects of alcohol.

The patient must be aware of the effects caused by overuse of the sympathomimetic agents in nose drops or sprays, because rhinitis medicamentosa may result. After topical application of the medication, a rebound period occurs in which the nasal mucous membranes become more edematous and congested than they were before the medication was used.

Such a reaction encourages the use of more medication, and a cyclic pattern results. The topical agent must be discontinued immediately and completely to correct this problem (Wahid & Shermetaro, 2019).

Continuing and Transitional Care. Follow-up telephone calls to the patient are often reassuring to the patient and family and provide an opportunity for the nurse to answer any questions. The patient is reminded to keep follow-up appointments and informed about the importance of continuing with treatment. The importance of participating in health promotion activities and health screening is also emphasized.

Evaluation

Expected patient outcomes may include:

1. Exhibits a breathing pattern that provides adequate ventilation
 - a. Demonstrates lungs clear on auscultation
 - b. Exhibits absence of adventitious breath sounds (crackles, rhonchi, wheezing)
 - c. Has a normal respiratory rate and pattern
 - d. Reports no complaints of respiratory distress (shortness of breath, difficulty on inspiration or expiration)

Chart 33-6



HOME CARE CHECKLIST

Allergy Management

At the completion of education, the patient and/or caregiver will be able to:

- State the impact of environmental allergens (e.g., dust, molds, perfumes, foods) on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State changes in home environment necessary to minimize exposure to allergens.
 - Removing drapes, curtains, and venetian blinds and replacing them with pull shades; covering the mattress with a hypoallergenic cover that can be zipped; and removing rugs and replacing them with wood flooring or linoleum.
 - Reducing dust in the house as a whole by using steam or hot water for heating and using high-efficiency particulate air (HEPA) purifiers or air-conditioning.
 - Washing the floor and dusting and vacuuming daily, using clean filters, wearing a mask whenever cleaning is being done.
 - Replacing stuffed furniture with wood pieces that can easily be dusted.
 - Avoiding the use of tufted bedspreads, stuffed toys, and feather pillows and replacing them with washable cotton material.
 - Avoiding the use of any clothing that causes itching.
- Verbalize ways to reduce exposure to pollens or molds by identifying seasons of the year when pollen counts are high; wearing a mask at times of increased exposure (windy days and when grass is being cut); and avoiding contact with weeds, dry leaves, and freshly cut grass.
- State rationale for seeking air-conditioned areas at the height of the allergy season.
- State rationale for avoiding sprays and perfumes.
- State rationale for the use of hypoallergenic cosmetics.
- State rationale for taking prescribed medications as prescribed.
- Identify specific foods that may cause allergic symptoms and develop a list of foods to avoid (e.g., fish, nuts, eggs, chocolate).
- Verbalize ways to cope with stress successfully, plans for regular exercise, and rationale for obtaining adequate rest.
- State how to reach primary provider with questions or complications.
- State time and date of follow-up appointments, testing.
- Identify the need for health promotion, disease prevention, and screening activities.

ADLs, activities of daily living; IADLs, instrumental activities of daily living.

2. Demonstrates knowledge about allergy and strategies to control symptoms
 - a. Identifies causative allergens, if known
 - b. States methods of avoiding allergens and controlling indoor and outdoor precipitating factors
 - c. Removes from the environment items that retain dust
 - d. Wears a dampened mask if dust or mold may be a problem
 - e. Avoids smoke-filled rooms and dust-filled or freshly sprayed areas
 - f. Uses air-conditioning for a major part of the day when allergens are high
 - g. Takes antihistamines as prescribed; participates in allergen immunotherapy program, if applicable
 - h. Describes name, purpose, side effects, and method of administration of prescribed medications
 - i. Identifies when to seek immediate medical attention for severe allergic responses
 - j. Describes activities that are possible, including ways to participate in activities without activating the allergies
3. Adapts to the inconveniences of an allergy
 - a. Relates the emotional aspects of the allergic response
 - b. Demonstrates the use of measures to cope positively with allergy
4. Demonstrates absence of complications
 - a. Exhibits vital signs within normal limits
 - b. Reports no symptoms or episodes of anaphylaxis (urticaria, itching, peripheral tingling, fullness in the mouth and throat, flushing, difficulty swallowing, coughing, wheezing, or difficulty breathing)
 - c. Demonstrates correct procedure to self-administer emergency medications to treat severe allergic reaction
 - d. Correctly states medication names, dose and frequency of administration, and medication actions
 - e. Correctly identifies side effects and untoward signs and symptoms to report to primary provider
 - f. Discusses acceptable lifestyle changes and solutions for identified potential barriers to adherence to treatment and medication regimen

Contact Dermatitis

Contact dermatitis is an inflammatory reaction of the skin due to contact with an exogenous substance. There are two basic types of contact dermatitis: irritant contact dermatitis and allergic contact dermatitis (see [Table 33-4](#)). Irritant contact dermatitis is an inflammatory response of the skin to direct chemical damage that releases mediators predominantly from epidermal cells. Allergic contact dermatitis is a delayed (type 4) hypersensitivity reaction to exogenous contact antigens that involves activation of T cells (Litchman, Nair, & Atwater, 2019). Eighty percent of contact dermatitis is irritant type and 20% is allergic type (Fornacier & Noor, 2018). Most cases are caused by excessive exposure to or additive effects of irritants (e.g., soaps, detergents, metals, organic solvents, cosmetics). Skin sensitivity may develop after brief or prolonged periods of exposure, and the clinical picture may appear hours or weeks after the sensitized skin has been exposed.

Clinical Manifestations

Symptoms of acute contact dermatitis include itching, burning, erythema, skin lesions (vesicles and bullae), and oozing (Goldner & Fransway, 2018). The reaction is limited to the site of contact. Chronic contact dermatitis symptoms can include scaling, lichenification, thickening of the skin, and pigmentary changes. Secondary invasion by bacteria may develop in skin that is abraded by rubbing or scratching. Usually, there are no systemic symptoms unless the eruption is widespread.

Assessment and Diagnostic Findings

Determining allergens responsible requires a history, physical examination, and patch testing.

Assessment includes the date of onset and any identifiable relationship to work environment and skin care products. The location of the lesions, distribution of the dermatitis, absence of other etiologies, and the history of exposure aid in determining the condition (Goldner & Fransway, 2018). Patch testing and environmental history of exposure to contact allergens are required to verify the diagnosis. Patch testing is the standard test for identification of culprit allergens in persons with allergic contact dermatitis. The patch test most commonly used is the thin-layer rapid use epicutaneous (TRUE) test. An extended screening panel (North American screening series) has increased sensitivity. Researchers reported an extended screening panel identified an additional 10.8% of patients with positive tests that were negative to TRUE test allergens (Sundquist, Lang, & Pasha, 2019).

TABLE 33-4 Types, Testing, and Treatment of Contact Dermatitis

Type	Etiology	Clinical Presentation	Diagnostic Testing	Treatment
Allergic	Results from contact of skin and allergenic substance; has a sensitization period of 10–14 days	Vasodilation and perivascular infiltrates on the dermis Intracellular edema Usually seen on dorsal aspects of hand	Patch testing (contraindicated in acute, widespread dermatitis)	Avoidance of offending material Aluminum acetate (Burrow Solution, Dombro Powder) or cool water compress Systemic corticosteroids (prednisone) for 7–10 days Topical corticosteroids for mild cases Oral antihistamines to relieve pruritus
Irritant	Results from contact with a substance that chemically or physically damages the skin on a nonimmunologic basis; occurs after first exposure to irritant or repeated exposures to milder irritants over an extended time	Dryness lasting days to months Vesication, fissures, cracks Hands and lower arms most common areas	Clinical picture Appropriate negative patch tests	Identification and removal of source of irritation Application of hydrophilic cream or petroleum to soothe and protect Topical corticosteroids and compresses for weeping lesions Antibiotics for infection and oral antihistamines for pruritus

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer.

Atopic Dermatitis

Atopic dermatitis (commonly called eczema) is a chronic, inflammatory allergic skin disorder that is triggered by environmental factors in individuals who are genetically susceptible. This disorder affects 7% of adults in the United States (Weston & Howe, 2019). More than half of patients are also affected by asthma, allergic rhinitis, and food allergies.

Atopic dermatitis is a type I immediate hypersensitivity disorder involving IgE antibodies that causes dry, pruritic, hypersensitive skin. It often begins with small red pruritic papules that stimulate intense itching, leaving erythematous, excoriated areas of skin. This often triggers an “itch-scratch cycle” where rubbing or scratching the skin causes further irritation, redness, and skin breakdown. Skin thickening from chronic scratching (lichenification) and fissuring may develop over time. In many patients, lesions in different stages may be present at the same time (Weston & Howe, 2019). Atopic dermatitis in adults often occurs at the hands, wrists, elbows, knees, ankles, face, and neck.

Atopic dermatitis is due to a “leaky” skin barrier that allows water to leave the skin dried out and hypersensitive. Exposure of the hypersensitive skin to soaps, detergent, house dust mites, pollen, animal dander, and some bacteria break down the skin barrier (Weston & Howe, 2019).

Defective filaggrin (FLG) genes at chromosome 1q21.3 are common in persons with the disorder. Filaggrin is a protein produced by keratinocytes in the skin that is encoded by the FLG gene (Løset, Brown, Saunes, et al., 2019). Currently, there is ongoing investigation into the etiology of atopic dermatitis, including other genes involved in processing of filaggrin.

Nurses should be aware that atopic dermatitis is often linked to a process called the **atopic march** that refers to the natural history of allergic diseases as they begin in infancy and through childhood. Atopic march begins with atopic dermatitis and progresses to IgE-mediated food allergy, asthma, and allergic

rhinitis. It is the result of interactions between susceptibility genes, the environment, defective function of the skin barrier, and immunologic responses (AAAAI, 2019a; Hill & Spergel, 2018).

The diagnosis of atopic dermatitis is based on the health history, morphology and distribution of skin lesions, and associated clinical signs. Laboratory testing, patch testing, and skin biopsy may be necessary if there is a need to rule out other skin conditions.

Medical Management

Treatment of patients with atopic dermatitis involves avoidance of irritative agents, use of anti-inflammatory topical agents, and moisturization of the skin. Patients should avoid potential triggers of atopic dermatitis, which include excessive bathing without subsequent moisturization, low humidity environments, animal dander, dust mites, xerosis (dry skin), overheating of skin, and exposure to solvents and detergents. Individuals are commonly hypersensitive to fragrances, perfumes, and contact allergens such as nickel.

Topical corticosteroids are the mainstay of treatment of atopic dermatitis. If mild OTC corticosteroids are not adequate, more potent corticosteroids such as fluocinoline 0.025%, triamcinolone, 0.1%, or betamethasone, 0.05% are prescribed. These agents should not be used on the face as they can cause skin atrophy. Topical calcineurin inhibitors, which are nonsteroidal immunomodulating agents (e.g., tacrolimus, pimecrolimus), are best for the facial area. Treatment of severe flare-ups of chronic disease can be treated with a short course of systemic corticosteroids (Weston & Howe, 2019).

Dupilumab is an interleukin (IL)-4 and IL-13 receptor-alpha antagonist that was approved by the FDA for the treatment of patients aged 12 years and older with moderate to severe atopic dermatitis not adequately controlled with topical prescription therapies (Spergel & Lio, 2019).

Colonization with *Staphylococcus aureus* occurs more frequently in individuals with atopic dermatitis than in the general population, and *S. aureus* is a common cause of secondary infection in these patients. The presence of purulence or honey-colored crusts suggests *S. aureus* infection. Antibiotic treatment is needed to eradicate infection (Spergel & Lio, 2019).

Nursing Management

Patients who experience atopic dermatitis and their families require assistance and support from the nurse to cope with the disorder. The symptoms are often disturbing to the patient and disruptive to the family. The appearance of the skin may affect the patient's self-esteem and their willingness to interact with others. Instructions and counseling about strategies to incorporate preventive measures and treatments into the lifestyle of the family may be helpful.

Skin hydration is a key component of treatment as atopic skin is low in moisture. Thick cream moisturizers and emollients that contain glycerol or urea should be used as these will keep the skin hydrated. A hydrating bath with mild soap followed by immediate emollient application is recommended, or a shower of short duration (Spergel & Lio, 2019).

Itching can be decreased by wearing cotton fabrics, washing clothes with a mild detergent, and humidifying dry heat in winter. Antihistamines such as diphenhydramine may be used as treatment, but since they are sedating, the patient can be advised that it is best to use them at bedtime.

The patient and family need to be aware of signs of secondary infection and of the need to seek treatment if infection occurs. The nurse also educates the patient and family about the side effects of medications used in treatment.

Drug Hypersensitivity

Drug hypersensitivity is the leading cause of fatal anaphylaxis, comprising 43% of deaths from anaphylaxis. All routes of administration are potentially fatal, but drugs given parenterally incur the greatest risk. Cutaneous rashes are among the most common reactions to medications and occur in approximately 2% to 3% of hospitalized patients (Habif, 2016).

A drug hypersensitivity reaction is defined by the time of appearance, possible mode of action (mechanism of immune stimulation), and resulting pathophysiology. According to the World Allergy Organization, immunologic drug reactions can be divided into immediate reactions (i.e., onset within 1 hour of exposure) and delayed reactions (onset after 1 hour), based on the timing of the appearance of symptoms (Tanno, Torres, Castells, et al., 2018).

IgE-mediated immediate type 1 hypersensitivity reactions to a drug occur within 1 hour of administration of the agent. Delayed hypersensitivity reactions occur after 1 hour; most occur after 6 hours or days of treatment. These reactions can also occur after the course of medication is finished. These reactions may be caused by several different mechanisms, but they are not IgE mediated. Types II, III, and IV immunologic reactions are all considered delayed reactions (Pichler, 2019).

A disorder known as drug rash with eosinophilia and systemic symptoms (DRESS) can occur after weeks of continuous treatment. Also known as “drug-induced hypersensitivity syndrome” (DiHS), it is characterized by fever, rash, and multiorgan involvement, and may or may not be associated with eosinophilia and lymphocytosis. Hepatitis and myocarditis can be part of DRESS. These reactions can persist for weeks to months, even after the medication is stopped (Mockenhaupt, 2019). Anticonvulsant agents (e.g., lamotrigine, phenytoin, phenobarbital) and allopurinol are the most frequently reported causes of DRESS (Mockenhaupt, 2019).

Anaphylaxis is the most severe presentation of an IgE-mediated drug reaction. Medications administered IV may cause symptoms in seconds to minutes, while the same drug administered orally may cause symptoms in 3 to 30 minutes if taken on an empty stomach, and in 10 to 60 minutes if taken with food. The agents that most commonly exhibit this type of reaction include the following (Pichler, 2019):

- Beta-lactam drugs (e.g., penicillins and cephalosporins)
- Neuromuscular blocking agents (e.g., pancuronium)
- Quinolones (e.g., ciprofloxacin)
- Platinum-containing chemotherapeutic agents (e.g., carboplatin)
- Foreign proteins such as monoclonal antibodies (e.g., rituximab)

Type II cytotoxic reactions involve antibody-mediated cell destruction. Type II reactions may arise when drugs bind to surfaces of certain cell types and act as antigens. Clinical manifestations include hemolytic anemia, thrombocytopenia, or neutropenia, since these are the cell types that are most often affected. The drugs most commonly implicated in hemolytic anemia are cephalosporins, penicillins, nonsteroidal anti-inflammatory drugs (NSAIDs), and quinine and quinidine. Drugs implicated in thrombocytopenia include heparin, abciximab, quinine and quinidine, sulfonamides, vancomycin, gold compounds, beta-lactam antibiotics, carbamazepine, and NSAIDs. Severe neutropenia due to type II drug reactions presents days to weeks after beginning the medication. Clinical manifestations include symptoms of infection, such as fever, stomatitis, pharyngitis, pneumonia, or sepsis. Propylthiouracil, amodiaquine, and flecainide can cause these reactions (Pichler, 2019).

Type III reactions are mediated by antigen–antibody complexes that deposit on basement membranes and usually present as serum sickness, vasculitis, or drug fever. Signs and symptoms take 1 or more weeks to develop after drug exposure, since significant quantities of antibody are needed to generate symptoms related to antigen–antibody complexes. These are uncommon reactions but are seen with antitoxins for rabies, botulism, and venoms, tetanus, hepatitis, and diphtheria vaccines (Pichler, 2019).

Type IV reactions involve activated T cells, which take time to develop. Type IV reactions usually take at least 48 to 72 hours and sometimes days to weeks to develop following exposure to the drug. Type IV reactions can vary from a nonurticular, maculopapular rash (drug fever) to Stevens–Johnson syndrome and toxic epidermal necrolysis (see [Chapter 56](#)), or drug rash with eosinophilia and systemic symptoms, or DRESS/DiHS. Type IV reactions can appear after weeks of drug treatment (Pichler, 2019).

Medications are the most common agent responsible for approximately 90% of all drug rashes. Commonly prescribed medications (e.g., antibiotics, sulfonamides) are implicated in most cases (Samel & Chu, 2019).

Drug fever, which usually causes fever and rash, is associated with azathioprine, sulfasalazine, minocycline, trimethoprim-sulfamethoxazole, sirolimus, and tacrolimus (McDonald & Sexton, 2019). Contact dermatitis skin reactions can result from topical anesthetics such as benzocaine, topical antibiotics such as neomycin or bacitracin, and topical corticosteroids.

Symmetrical drug-related intertriginous and flexural exanthem (SDRIFE), formerly called baboon syndrome, is a distinctive drug eruption that typically develops within a few hours to days of drug exposure and presents with demarcated, V-shaped erythema in the gluteal/perianal or inguinal/perigenital areas, often with involvement of at least one other flexural area, such as the axillae, elbows, or knees. Aminopenicillins are a common trigger of SDRIFE (Bircher, 2018).

Acute generalized exanthematous pustulosis (AGEP) is a rare type of reaction characterized by superficial pustules, usually appearing within 24 hours after the administration of the culprit drug. Antimicrobial drugs (amoxicillin), antimalarials, and calcium channel blockers are the most frequently reported triggers of AGEP (Cho & Chu, 2017).

Stevens–Johnson syndrome and toxic epidermal necrolysis are severe reactions commonly triggered by medications. The disorder can evolve into extensive epidermal necrosis and become life-threatening. Mucous membranes are affected in over 90% of patients, usually at two or more distinct sites (ocular, oral, and genital). In some patients, an exanthematous eruption can be the heralding sign of Stevens–Johnson syndrome and toxic epidermal necrolysis. Fever, often exceeding 39°C (102.2°F), and influenzalike symptoms precede development of mucocutaneous lesions and erythematous macules with purpuric centers that evolve into blisters and bullae. Photophobia, conjunctival itching or burning, and pain on swallowing may be due to mucosal involvement. Malaise, myalgia, and arthralgia are present in most patients. The following agents are most commonly implicated in Stevens–Johnson syndrome and toxic epidermal necrolysis (High, 2019):

- Allopurinol
- Aromatic antiepileptic drugs and lamotrigine
- Antibacterial sulfonamides (including sulfasalazine)
- Nevirapine
- Oxicam NSAIDs

Some conventional and targeted anticancer drugs have been associated with these syndromes including thalidomide, capecitabine, afatinib, vemurafenib, tamoxifen, and immune checkpoint inhibitors ipilimumab, pembrolizumab, and nivolumab. Radiation treatment in combination with antiepileptic drugs (e.g., phenytoin, phenobarbital, carbamazepine) can trigger Stevens–Johnson syndrome and toxic epidermal necrolysis (High, 2019).

Pharmacogenetic studies suggest that there is a genetic predisposition to drug allergy. In some populations, individuals with HLA-B*1502, HLA-B*5801, or HLA-B*5701 have an increased risk of developing Stevens–Johnson syndrome and toxic epidermal necrolysis to aromatic anticonvulsants such as carbamazepine, allopurinol, cotrimoxazole, and abacavir, respectively (Bircher, 2018).

Pseudoallergic Drug Reactions

Pseudoallergic drug reactions are adverse drug reactions with signs and symptoms that mimic immunologic drug allergies, but no immunologic mechanisms are occurring. They are referred to as nonimmunologic hypersensitivity reactions or anaphylactoid reactions. It is unclear how certain drugs elicit pseudoallergic reactions. However, degranulation of mast cells occurs. Some affected patients have underlying dermographism which indicates an “instability” of their mast cells. In dermographism, when pressure is applied to the skin, the skin reddens for a prolonged time in the same pattern as the pressure was applied. The following drugs may cause a pseudoallergic drug reaction (Pichler, 2019):

- Radiocontrast agents
- Opioids (e.g., morphine and meperidine)
- NSAIDs (e.g., ibuprofen) and aspirin
- Vancomycin
- Local anesthetic agents (e.g., lidocaine, benzocaine)
- Chemotherapeutic agents (e.g., platinum-based drugs)

The patient should be educated about avoidance and provided with a written list of the generic and brand names of the causative agents to avoid in the future (Pichler, 2019).

Urticaria and Angioedema

Urticaria (hives) is a type I hypersensitive allergic reaction of the skin that is characterized by the sudden appearance of intensely pruritic pink or red discrete papules that progress to wheals of variable size. Urticular lesions coalesce and evolve into large erythematous plaques (AAAAI, 2019b). This is a common condition with up to 20% of people having at least one episode of hives during their lifetime. Urticaria is most commonly instigated by infections, allergic reactions to food, insect stings, and medications (Asero, Tedeschi, Marzano, et al., 2017).

Acute urticaria evolves over a time span of minutes to hours and disappears by 24 hours; lesions can be in different stages over this time. Urticaria is

considered acute if it has been present for less than 6 weeks. However, if urticaria occurs frequently and reoccurs daily for longer than 6 weeks, the condition is called chronic urticaria. In urticaria, mast cells and basophils within the skin are activated and release histamine and inflammatory mediators that cause vasodilation (AAAAI, 2019b).

Common causes of urticaria include allergic reactions to medications or contact allergens, foods, insect stings and bites; reactions to medications that cause nonallergic mast cell activation (e.g., opioids); latex allergy; transfusions; and NSAIDs. Physical urticarial syndromes are forms of chronic urticaria that are triggered by specific physical and environmental factors, such as cold exposure, sudden changes in body temperature, pressure or vibration against the skin, exercise, exposure to sunlight, or other stimuli (Asero, 2017). Serum sickness, a type III hypersensitivity reaction, commonly due to medication, can also cause urticaria. Serum sickness classically causes rash, fever, and polyarthralgias or polyarthritis, which begin 1 to 2 weeks after the first exposure to the responsible agent and resolve within a few weeks of discontinuation (Wener, 2018).

The diagnosis of urticaria can usually be made by health history and physical examination. Laboratory testing is usually not necessary.

Management of the condition includes eliminating the causative agent; avoiding the use of NSAIDs; and minimizing potential aggravators, including heat, stress, alcohol, and tight clothes. Treatment with second-generation H₁ antihistamine agents (e.g., cetirizine, fexofenadine, loratadine) is the mainstay of treatment. These agents are better tolerated as they have less sedating effects than first-generation H₁ antihistamines (e.g., diphenhydramine, chlorpheniramine, hydroxyzine). Doses of second-generation antihistamines may be increased as high as four times the standard dose (Khan, 2019a). Oral corticosteroids given in a decreasing dose schedule may be prescribed to relieve severe symptoms for a few days. About 50% of cases of chronic spontaneous urticaria will respond to treatment with antihistamines, as discussed above. For those patients who do not improve with antihistamines, 65% might respond to prescribed omalizumab. Omalizumab, a monoclonal antibody that acts against IgE antibodies, is injected under the skin every 2 to 4 weeks by a primary provider. Therapeutic effects can be seen within 3 to 6 months (AAAAI, 2019b; Stokes & Casale, 2018).

Angioedema is an allergic reaction that involves the infiltration of fluid in subcutaneous tissue and mucous membranes resulting in diffuse swelling. It is manifested by nonpruritic, brawny, widespread, nonpitting edema. Urticaria and angioedema often occur together (AAAAI, 2019b; Habif, 2016).

The regions most often involved in angioedema are the lips, eyelids, cheeks, hands, feet, genitalia, and tongue; the mucous membranes of the larynx, bronchi, and gastrointestinal tract may also be affected, particularly in the hereditary type (see discussion in the following section). On occasion, this

reaction covers the entire back or large area of the body. Swellings may appear suddenly, in a few seconds or minutes, or slowly over 1 or 2 hours. It usually resolves within 24 hours. Angioedema is usually a benign and transient condition, although it can be life-threatening when severe angioedema of the larynx, upper airway, or tongue results in airway obstruction (Zuraw, 2019).

Two types of angioedema can be distinguished: mast cell-mediated, also called histaminergic angioedema, and bradykinin-mediated angioedema. Allergic reactions to foods, latex, certain drugs, or insect stings are common examples of mast cell-mediated angioedema. Histamine is the main inflammatory mediator and signs and symptoms include urticaria, flushing, generalized pruritus, bronchospasm, throat tightness, and/or hypotension. Patients may be experiencing anaphylaxis and should be treated immediately with epinephrine. Mast cell-mediated angioedema usually begins within minutes of exposure to the allergen, builds over a few hours, and resolves in 24 to 48 hours (Zuraw, 2019).

Alternatively, bradykinin-induced angioedema does not involve histamine and is not associated with urticaria, bronchospasm, or other symptoms of allergic reactions. Bradykinin is a potent vasodilator which also increases vasopermeability (Cicardi & Zuraw, 2018a). The fluid infiltration of the tissues usually develops over 24 to 36 hours and resolves within 2 to 4 days. The relationship between the trigger and the onset of symptoms is often not apparent. Angiotensin-converting enzyme inhibitors (e.g., captopril) are common causes of bradykinin-induced angioedema; swelling may appear within a week of starting the medication or after years of use (Zuraw, 2019).

Second-generation H₁ antihistamines and corticosteroids are the mainstay of treatment for mast cell-mediated angioedema. If angioedema is part of an anaphylactic reaction, intramuscular epinephrine is an important part of treatment (Guyer & Banerji, 2019).

Treatment of bradykinin-induced angioedema involves avoidance of drug, icatibant, C1 inhibitor concentrate, ecallantide, and possibly administration of fresh-frozen plasma. Antihistamines are ineffective (Cicardi & Zuraw, 2018b). Icatibant is a synthetic bradykinin beta-2-receptor antagonist. C1 inhibitor concentrate and exallantide inhibit kallikrein which is a protease involved in bradykinin production. Fresh-frozen plasma contains angiotensin-converting enzyme, and the administration of plasma is thought to degrade high levels of bradykinin (Guyer & Banerji, 2019).

Hereditary Angioedema

Hereditary angioedema (HAE) is a rare, potentially life-threatening, autosomal dominant genetic disorder. It is a bradykinin-mediated type of angioedema that is due to a lack of C1 inhibitor activity, a specific protein that takes part in

kinin generation. Kinins are inflammatory mediators; bradykinin is one of these. C1INH usually plays a role in limiting bradykinin production, so when C1INH is deficient or dysfunctional, bradykinin production is relatively unchecked (AAAAI, 2019c).

There are two different types of HAE; HAE type I is due to C1 inhibitor (C1INH) deficiency, and type II is caused by C1INH dysfunction (Cicardi & Zuraw, 2018a).

Clinical Manifestations

HAE is commonly categorized as laryngeal, gastrointestinal, or cutaneous. Swelling of the skin is usually diffuse, nonpruritic, and not accompanied by urticaria. Attacks of swelling in patients with HAE generally involve the extremities, abdomen, genitourinary tract, face, oropharynx, or larynx and follow a stereotypical pattern in which the swelling worsens over 24 hours, peaks, and then slowly resolves over the following 48 hours. Gastrointestinal edema may cause abdominal pain severe enough to be incapacitating. Typically, attacks last 2 to 4 days and resolve without intervention; however, attacks can occasionally affect the subcutaneous and submucosal tissues in the region of the upper airway and can be associated with respiratory obstruction and asphyxiation (Cicardi & Zuraw, 2018a).

Medical Management

Attacks usually subside within 2 to 4 days, but during this time the patient should be observed carefully for signs of laryngeal obstruction, which may necessitate tracheostomy as a lifesaving measure. Epinephrine, antihistamines, and corticosteroids are commonly administered in an attempt to relieve HAE; however, these are often ineffective. A trial of high doses of second-generation H₁ antihistamines for 1 month may be effective in some patients. If this proves unsuccessful, C1INH concentrate, derived from human plasma, recombinant human C1INH (rhC1INH, conestat alfa), icatibant, a synthetic bradykinin beta-2-receptor antagonist and ecallantide, a recombinant plasma kallikrein inhibitor are agents that are available (Cicardi & Zuraw, 2018b). Nurses need to be prepared to use different emergency approaches in the treatment of HAE and to have lifesaving equipment readily available.

Cold Urticaria

Cold urticaria is a type of physical urticaria. Physical urticarias are inducible urticarias that are stimulated by environmental triggers. Cold urticaria is the development of wheals (hives) or angioedema due to exposure to cold. Mast

cells release histamine and inflammatory mediators are stimulated in response to skin contact with cold objects, cold fluids, or cold air. It is an IgE-mediated atopic immune reaction. Cold urticaria most commonly affects young adults as a self-limited disorder that occurs over a period of 5 to 6 years.

Clinical Manifestations

The patient feels a burning or pruritic sensation and the skin is erythematous due to activation of sensory nerves and vasodilation of arterioles. Most commonly it is a localized reaction in areas exposed to cold. However, extensive cold contact, such as swimming in cold water, may result in systemic reactions, ranging from generalized urticaria to anaphylaxis, with symptoms involving the respiratory, gastrointestinal, and/or cardiovascular systems. Oropharyngeal angioedema, which can cause suffocation and severe hypotension, has been observed in some persons with the disorder (Maurer, 2019).

Medical Management

Cold urticaria is diagnosed by cold stimulation testing. An ice cube within a thin plastic bag of cold water is applied to the volar aspect of the forearm for 1 to 5 minutes. A positive test results in development of urticaria at the site. The test is considered positive if the test site shows a palpable and clearly visible wheal-and-flare skin reaction upon rewarming. All patients with any form of cold urticaria should carry an autoinjectable epinephrine device for emergency use because hives can progress to anaphylaxis. Pretreatment with an antihistamine prior to predictable cold exposure is recommended, because clinical experience suggests that antihistamine pretreatment can prevent skin reactions and systemic reactions (Maurer, 2019). Patients with cold urticaria refractory to antihistamines can be treated with immunomodulator agents, such as omalizumab, certain antibiotics, leukotriene receptor antagonists, or cold desensitization therapy (Khan, 2019b).

Nursing Management

Prevention involves avoidance of cold stimuli. Patient education is needed about what environmental conditions can stimulate a reaction. For instance, patients should be instructed to expose a small section of the body to the water of a swimming pool prior to submerging the body in water. A wet suit can be used during swimming. Patients should understand that cold foods and beverages can stimulate oropharyngeal angioedema or anaphylaxis and should be avoided. Patients anticipating surgery may develop a reaction to the cold air within operating rooms and are instructed to alert surgical personnel that they

have cold urticaria and should be kept warm during any procedures. Cold intravenous solutions can also provoke a reaction (Singleton & Halverstam, 2016).

Food Allergy

Food allergy is an adverse reaction to certain foods due to immunologic mechanisms. Food allergies are categorized as either IgE-mediated or non-IgE-mediated allergies. IgE-mediated food allergy, a type I hypersensitivity reaction, occurs in about 5% of adults. IgE-mediated food allergy is more common and better understood than non-IgE-mediated food allergy (Commins, 2019). Recent studies show that within the population there are many with the misconception that they have a food allergy (19%) compared to persons with true immune-mediated food allergy (10%) (Gupta, Warren, & Smith, 2019).

Almost any food can cause an IgE-mediated allergic reaction. Allergic reactions can range from cutaneous urticaria to anaphylaxis. Fish and shellfish (e.g., lobster, shrimp, crab, clams, fin fish) as well as peanuts and tree nuts (e.g., cashew, walnut) are the two food groups that cause the majority of adult food allergies (Gupta et al., 2019). Other common foods causing allergy include cow's milk, eggs, soy, and wheat. Allergens are proteins and there is a danger that cross-reactivity can occur in foods containing similar proteins (AAAAI, 2018).

OAS, or pollen-food allergy syndrome (PFAS or PFS), is the most common form of IgE-mediated food allergy in adults. OAS often occurs in persons who are also allergic to pollen that causes seasonal allergic rhinitis. Raw fruits and vegetables often cause OAS but cooked fruit and vegetables do not (Burks, 2019).

Non-IgE-mediated food allergies present as more subacute and/or chronic symptoms that are typically isolated to the gastrointestinal tract and/or skin.

Clinical Manifestations

Acute urticaria and angioedema (swelling of the mouth, face, lips, tongue, and throat) are the most common clinical manifestations of food allergy. The reaction develops within minutes to hours after eating the offending food. The clinical symptoms may include wheezing, cough, laryngeal edema, and gastrointestinal symptoms (abdominal pain, nausea, cramps, vomiting, and diarrhea). Tree nuts and peanuts can cause the most severe allergic reactions evolving into anaphylaxis.

Assessment and Diagnostic Findings

Diagnosis of food allergy requires clinical history, physical examination, trial elimination diets, diet diaries, skin prick testing (SPT), and allergen-specific serum IgE immunoassay. A patch test is used if non-IgE-mediated food allergy is suspected. However, the standard is clinician-supervised oral food challenges to confirm or rule out the diagnosis. SPT is used to identify the source of symptoms and assists in identifying specific foods as causative agents. The patient is injected with a minute quantity of food antigen under the skin. Then the patient and primary provider wait to observe if hives and erythema arise within the 15 minute period. If the allergen triggers mast cells to release histamine, a localized wheal will be raised in the skin. A positive skin prick test for IgE-mediated food allergy is diagnosed when a wheal of 3 mm in diameter is raised within 15 minutes. A different kind of test, referred to as a 48-hour atopy patch test, is used for non-IgE-mediated food allergy. This is a topical application of food allergen that is applied to the small surface area of skin. This is a delayed reaction that requires 48 hours of observation (Andreae & Schreffler, 2019).

Medical Management

Therapy for food hypersensitivity includes avoidance of the food responsible for the hypersensitivity. Pharmacologic therapy is necessary for patients who cannot avoid exposure to offending foods and for patients with multiple food sensitivities not responsive to avoidance measures. Medication therapy involves the use of H₁ blockers, antihistamines, adrenergic agents, corticosteroids, and cromolyn sodium. All patients with food allergies, especially seafood and nuts, should have an autoinjectable epinephrine device prescribed. Another essential aspect of management is educating patients and family members about how to recognize and manage the early stages of an acute anaphylactic reaction.

Oral immunotherapy (OIT), or tolerance induction, is an increasingly used treatment. The patient ingests very small amounts of the allergen in increasing dosages over several months. OIT is not a curative treatment. The goal of OIT is to increase the threshold that triggers a reaction. OIT can be used to achieve desensitization or sustained unresponsiveness to a food allergen (AAAAI, 2018).

Initially, OIT has to be administered in a clinical setting equipped for treatment of potential anaphylaxis. Patients are generally started on a very small daily dose of the food (e.g., 3 to 6 mg of food protein) and advanced periodically (usually every 2 weeks) to a maintenance dose (e.g., 300 mg or, depending on the food and goals, 1 to 2 g of food protein daily) over several months (Nowak-Wegrzyn, 2019).

Peanut, egg, and milk OIT have been shown to desensitize approximately 60% to 80% of patients studied. OIT has not cured food allergy in these

individuals; it has raised the patient's tolerance of the offending food. Most children outgrow their allergies to cow's milk, egg, soy and wheat, even if they have a history of a severe reaction. However, peanut, tree nuts, fish, and shellfish allergies tend to persist through adulthood (AAAAI, 2018).

Chart 33-7



HOME CARE CHECKLIST

Managing Food Allergies

At the completion of education, the patient and/or caregiver will be able to:

- State the impact of food allergies on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- Verbalize understanding of the need to maintain an allergen-free diet.
- Demonstrate reading of food labels to identify hidden allergens in food.
- Identify ways to manage an allergen-free diet when eating away from home.
- State the need to wear medical identification bracelet or necklace.
- List symptoms of food allergy.
- Demonstrate emergency administration of epinephrine.
- State the importance of replacing epinephrine when outdated.
- State the importance of prompt treatment of allergic reactions and health care follow-up.
- State how to reach primary provider with questions or complications.
- State time and date of follow-up appointments, testing.
- Verbalize ways to cope with stress successfully, plans for regular exercise, and rationale for obtaining adequate rest.
- Identify the need for health promotion, disease prevention, and screening activities.

ADLs, activities of daily living; IADLs, instrumental activities of daily living.

Nursing Management

In addition to participating in management of the allergic reaction, the nurse focuses on preventing future exposure of the patient to the food allergen. The nurse should educate the patient about the signs of anaphylaxis and devise an action plan with the patient. If a severe allergic or anaphylactic reaction to food allergens has occurred, the nurse must instruct the patient and family about avoidance strategies to prevent its recurrence (see Chart 33-7). All patient allergies, including food allergies, should be noted on patient medical

records, as dietary restrictions would be necessary in the case of hospitalization. Also, there may be risk of cross-reactivity with some medications containing similar substances. The patient also needs to understand how to select and specify preparation of restaurant food (Sicherer, 2019).

Latex Allergy

Latex allergy—the allergic reaction to the proteins in the saplike fluid of a rubber tree—has been implicated in rhinitis, conjunctivitis, contact dermatitis, urticaria, asthma, and anaphylaxis. The prevalence is estimated at 4% to 7% of the population, but this has been steadily declining because of the use of nonpowdered latex and latex-free gloves (Hamilton, 2017a).

The sap of the rubber tree (*Hevea brasiliensis*) contains 250 different polypeptides that can react with IgE. The polypeptides and proteins in natural rubber latex (Hevea proteins) or the various chemicals that are used in the manufacturing process are thought to be the source of the allergic reactions.

Those at risk include health care workers, patients who have undergone multiple surgeries, people working in factories that manufacture latex products, and patients with spina bifida. Patients with spina bifida are at risk because they have had multiple surgeries, multiple urinary catheterization procedures, and other treatments involving use of latex products (Hamilton, 2017a). Because food handlers, hairdressers, automobile mechanics, and police may wear latex gloves and use latex products, they are also at risk for latex allergy. Increasingly, however, nonlatex gloves are being used in various occupational settings. Risk factors include occupational exposure to latex and atopic tendency in the affected person. Patients are at risk for anaphylactic reactions as a result of contact with latex during medical treatments, particularly surgical procedures (AAAAI, 2019d).

Persons with latex allergy are also prone to cross-reactions to pollen and some fruits; such as kiwis, bananas, pineapples, mangoes, passion fruit, avocados, and chestnuts (Hamilton, 2017a).

Routes of exposure to latex products can be cutaneous, percutaneous, mucosal, parenteral, or aerosol. The most frequent source of exposure is cutaneous, which usually involves the wearing of natural latex gloves. The powder used to facilitate putting on latex gloves can become a carrier of latex proteins from the gloves; when the gloves are put on or removed, the particles become airborne and can be inhaled or settle on skin, mucous membranes, or clothing (AAAAI, 2019d). Mucosal exposure can occur from the use of latex condoms, catheters, airways, and nipples. Parenteral exposure can occur from intravenous lines or hemodialysis equipment. In addition to latex-derived medical devices, many household items also contain latex. Examples of

medical and household items containing latex and a list of alternative products are found in [Table 33-5](#). It is estimated that more than 40,000 medical devices and nonmedical products contain latex (AAAAI, 2019d).

Clinical Manifestations

Many different types of reactions to latex are possible (see [Table 33-6](#)). Irritant contact dermatitis, a nonimmunologic response, may be caused by mechanical skin irritation or an alkaline pH associated with latex gloves. Common symptoms of irritant dermatitis include erythema and pruritus.

Delayed hypersensitivity to latex, a type IV reaction mediated by T cells, is localized to the area of exposure and is characterized by symptoms of contact dermatitis. These include vesicular skin lesions, papules, pruritus, edema, erythema, and crusting and thickening of the skin. These symptoms usually appear on the back of the hands 1 to 4 days postcontact. It is the most common allergic reaction to latex. Although usually not life-threatening, delayed hypersensitivity reactions often require major changes in the patient's home and work environment to avoid further exposure (AAAAI, 2019d).

TABLE 33-5 Select Products Containing Natural Rubber Latex and Latex-Free Alternatives

Products Containing Latex	Examples of Latex-Safe Alternatives ^a
Hospital Environment	
Ace bandage (brown)	Ace bandage, white all cotton
Adhesive bandages, Band-Aid dressing, Telfa	Cotton pads and plastic or silk tape, Active Strips (3M), DuoDERM
Anesthesia equipment	Neoprene anesthesia kit (King)
Blood pressure cuff, tubing, and bladder	Clean Cuff, single-use nylon or vinyl blood pressure cuffs or wrap with stockinette or apply over clothing
Catheters	All-silicone or vinyl catheters
Catheter leg bag straps	Velcro straps
Crutch axillary pads and hand grips, tips	Cover with cloth, tape
ECG pads	Baxter, Red Dot 3M ECG pads
Elastic compression stockings	Kendall SCD stockings with stockinette
Gloves	Derma Prene, neoprene, polymer, or vinyl gloves
IV catheters	Jelco, Deseret IV catheters
IV rubber injection ports	Cover Y-sites and ports; do not puncture. Use three-way stopcocks on plastic tubing
Levin tube	Salem sump tube
Medication vials	Remove rubber stopper
Penrose drains	Jackson-Pratt, Zimmer Hemovac drains
Prepackaged enema kits	Therevac, Fleet Ready-to-Use
Pulse oximeters	Noninoximeters
Resuscitation bags	Laerdal, Puritan Bennett, <i>certain</i> Ambu
Stethoscope tubing	PVC tubing; cover with latex-free stockinette
Suction tubing	PVC (Davol, Laerdal)
Syringes—single use (Monoject, BD)	Terumo syringes, Abbott PCA Abboject
Tapes	Dermicel, Micropore
Theraband	New Thera-band Exercisers, plastic tubing
Thermometer probes	Diatek probe covers
Tourniquets	X-Tourn straps (Avcor)
Home Environment	
Balloons	Mylar balloons
Condoms, diaphragms	Polyurethane products, Durex Avanti and Reality products (female condom)
Diapers, incontinence pads	Huggies, Always, <i>some</i> Attends

Feminine hygiene pads	Kimberly-Clark products
Wheelchair cushions	ROHO cushions, Sof Care bed/chair cushions

^aConfirmation is essential to verify that all items are latex free before using, especially if risk of latex allergy is present.

ECG, electrocardiogram; IV, intravenous; PVC, polyvinyl chloride.

Adapted from Centers for Disease Control and Prevention (CDC). (2014). Latex allergy: A prevention guide. Retrieved on 12/2/2019 at: www.cdc.gov/niosh/docs/98-113/default.html; Mayo Foundation for Medical Education and Research. (2019).

Diseases and conditions: Latex allergy. Retrieved on 12/2/2019 at:

www.mayoclinic.org/diseases-conditions/latex-allergy/basics/definition/CON-20024233

Latex allergy can also be an immediate hypersensitivity, type I allergic reaction, mediated by IgE. Localized itching, erythema, or local urticaria within 10 to 15 minutes after exposure to latex are often the initial symptoms (Hamilton, 2017a). Symptoms commonly include rhinitis, conjunctivitis, and nasal congestion. An asthma attack can be triggered. Severe anaphylaxis can occur, which includes generalized urticaria, angioedema, bronchospasm, and hypotension minutes after dermal or mucosal exposure to latex.

Assessment and Diagnostic Findings

The diagnosis of latex allergy is based on the history, physical examination, and diagnostic test results. Sensitization is detected by skin testing; serum-specific IgE, EIA, or ELISA; or the level of Hevea latex-specific IgE antibody in the serum. Testing for the chemicals found in the rubber production that makes latex is performed using the patch test. Skin patch testing is the preferred method for patients with contact allergies. The TRUE test and other skin tests should be performed only by primary providers who have expertise in their administration and interpretation and who have the necessary equipment available to treat local or systemic allergic reactions to the reagent (Hamilton, 2017a).

Medical Management

The best prevention strategy for latex allergy is the avoidance of latex-based products. A new, natural derivative from the desert plant guayule is now being used as a replacement for latex in many products. The predominant nonsterile, nonlatex examination gloves used in medical institutions today are made of nitrile, neoprene, vinyl, or synthetic polyisoprene rubber that is extracted from oil. Nonlatex condoms are available for contraception but these do not prevent transmission of HIV or other sexually transmitted infections (STIs). Persons allergic to latex should be cautioned to not blow up latex balloons or be in

enclosed spaces where these are used, as these balloons are still used as decorations (AAAAI, 2019d).

Patients at risk for an anaphylactic reaction to latex should be instructed to carry auto-injectable epinephrine in case of a reaction (Hamilton, 2017b).

Patients should report their allergy prior to any medical, dental, gynecologic, or surgical procedure and request a latex-safe environment (Hamilton, 2017b).

TABLE 33-6 Types of Reactions to Latex

Type of Reaction	Cause	Signs/Symptoms	Treatment
Irritant contact dermatitis	<p>Damage to skin because of irritation and loss of epidermoid skin layer; not an allergic reaction. Can be caused by excessive use of soaps and cleansers, repeated handwashing, inadequate hand drying, mechanical irritation (e.g., sweating, rubbing inside powdered gloves), exposure to chemicals added during the manufacturing of gloves, and alkaline pH of powdered gloves.</p> <p>Reaction may occur with first exposure, is usually benign, and is not life-threatening.</p>	<p><i>Acute:</i> Redness, edema, burning, discomfort, itching</p> <p><i>Chronic:</i> Dry, thickened, cracked skin</p>	<p>Referral for diagnostic testing</p> <p>Avoidance of exposure to irritant</p> <p>Thorough washing and drying of hands</p> <p>Use of powder-free gloves with more frequent changes of gloves</p> <p>Changing glove types</p> <p>Use of water- or silicone-based moisturizing creams, lotions, or topical barrier agents</p> <p>Avoidance of oil- or petroleum-based skin agents with latex products, because they cause breakdown of the latex product</p>
Allergic contact dermatitis	<p>Delayed hypersensitivity (type IV) reaction. Usually affects only area in contact with latex; reaction is usually to chemical additives used in the manufacturing process rather than to latex itself. Cause of reaction is T-cell-mediated sensitization to additives of latex.</p> <p>Reaction is not life-threatening and is far more common than a type I reaction.</p>	<p>Pruritus, erythema, swelling, crusty thickened skin, blisters, other skin lesions</p>	<p>Referral for diagnosis (patch tests) and treatment</p> <p>Thorough washing and drying of hands</p> <p>Use of water- or silicone-based moisturizing creams,</p>

	<p>Slow onset; occurs 18–24 h after exposure. Resolves within 3–4 days after exposure. More severe reactions may occur with subsequent exposures.</p>	<p>lotions, or topical barrier agents Avoidance of oil- or petroleum-based products unless they are latex compatible Avoidance of identified causative agent, because continued exposure to latex products in presence of breaks in skin may contribute to latex protein sensitization</p>
Latex allergy	<p>Type I IgE-mediated immediate hypersensitivity to plant proteins in natural rubber latex. In sensitized people, antilatex IgE antibody stimulates mast cell proliferation and basophil histamine release. Exposure can be through contact with the skin, mucous membranes, or internal tissues, or through inhalation of traces of powder from latex gloves.</p> <p>Severe reactions usually occur shortly after parenteral or mucous membrane exposure. People with any type I reaction to latex are at high risk for anaphylaxis. Local swelling, redness, edema, itching, and systemic reactions, including anaphylaxis, occur within minutes after exposure.</p>	<p>Rhinitis, flushing, conjunctivitis, urticaria, laryngeal edema, bronchospasm, asthma, severe vasodilation angioedema, anaphylaxis, cardiovascular collapse, death</p> <p>Immediate treatment of reaction with epinephrine, fluids, vasopressors, and corticosteroids, and airway and ventilator support, with close monitoring for recurrence for the next 12–14 h</p> <p>Prompt referral for diagnostic evaluation</p> <p>Treatment and diagnostic evaluation in latex-free environment</p> <p>Assessment of all patients for symptoms of latex allergy</p>

Educating patients and family members about the disorder and the importance of preventing future reactions by avoiding latex (e.g., wearing medical identification, carrying and autoinjector of epinephrine)

IgE, immunoglobulin E.

Adapted from American Academy of Allergy, Asthma, and Immunology (AAAAI). (2019d). Latex Allergy. Retrieved on 7/7/2019 at: www.aaaai.org/conditions-and-treatments/library/allergy-library/latex-allergy

Nursing Management

The nurse can assume a pivotal role in the management of latex allergies in both patients and staff. All patients should be asked about latex allergy. Every time an invasive procedure must be performed, the nurse should consider the possibility of latex allergies. Nurses working in operating rooms, intensive care units, short procedure units, and EDs need to pay particular attention to latex allergy (Hamilton, 2017b). See [Chapter 14, Figure 14-2](#), for a sample latex allergy assessment form.

Although the type I reaction is the most significant of the reactions to latex, care must be taken in the presence of irritant contact dermatitis and delayed hypersensitivity reaction to avoid further exposure of the person to latex. Patients with latex allergy are advised to notify their health care providers and to wear medical identification. Patients must become knowledgeable about what products contain latex and what products are safe, nonlatex alternatives. They must also become knowledgeable about signs and symptoms of latex allergy and emergency treatment and self-injection of epinephrine in case of allergic reaction (Hamilton, 2017b).

CRITICAL THINKING EXERCISES

1 ipc You are the nurse working in an outpatient walk-in clinic. A 28-year-old patient is newly diagnosed with allergic rhinitis. What nursing and interprofessional assessments are indicated? What interventions, including patient education, will you implement? What interprofessional referrals would be appropriate?

2 ebp A 44-year-old presents to the emergency department (ED) complaining of dyspnea after eating at a new restaurant. The patient states that this has happened two other times in the past and seems to be worse each time. What is the evidence for management of this patient? Describe the strength of the evidence and criteria used to assess its strength.

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*Asterisk indicates nursing research.

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Resources

American Academy of Allergy, Asthma, and Immunology (AAAAI),

www.aaaai.org

Asthma and Allergy Foundation of America (AAFA), www.aafa.org

Asthma and Allergy Foundation of America. (2021). Find a local support group.

Retrieved from: <https://www.aafa.org/aafa-affiliated-asthma-allergy-support-groups/>

Food Allergy Research Education (FARE), www.foodallergy.org

Mayo Foundation for Medical Education and Research, www.mayoclinic.org

National Institute of Allergy and Infectious Diseases, www.niaid.nih.gov

Occupational Safety and Health Administration (OSHA), www.osha.gov

34 Assessment and Management of Patients with Inflammatory Rheumatic Disorders

LEARNING OUTCOMES

On completion of this chapter, the learner will be able to:

- 1.** Explain the pathophysiology of inflammatory rheumatic diseases and describe the assessment and diagnostic findings seen in patients with these disorders.
- 2.** Use the nursing process as a framework for care of the patient with an inflammatory rheumatic disorder.
- 3.** Devise an education plan for the patient with newly diagnosed inflammatory rheumatic disease.
- 4.** Identify modifications in interventions to accommodate changes in patients' functional ability that may occur with disease progression.

NURSING CONCEPTS

Assessment
Cellular Regulation
Comfort
Immunity
Infection
Inflammation

GLOSSARY

- arthritis:** inflammation of a joint
- cytokines:** cell signaling proteins that are vital to regulation of hematopoiesis, apoptosis, and immune responses
- exacerbation:** period when disease symptoms occur or increase
- pannus:** proliferation of newly formed synovial tissue infiltrated with inflammatory cells
- remission:** period when disease symptoms are reduced or absent
- rheumatic diseases:** numerous disorders affecting skeletal muscles, bones, cartilage, ligaments, tendons, and joints
- rheumatoid arthritis:** a systemic autoimmune disease with symmetric arthritic manifestations and multiple extra-articular features
- subchondral bone:** bony plate that supports the articular cartilage
- tophi:** accumulation of crystalline deposits in articular surfaces, bones, soft tissue, and cartilage

The **rheumatic diseases** encompass autoimmune, degenerative, inflammatory, and systemic conditions that affect the joints, muscles, and soft tissues of the body. Rheumatic diseases most commonly manifest the clinical features of **arthritis** (inflammation of a joint) and pain. There are more than 100 types of rheumatic diseases. The problems caused by rheumatic diseases include limitations in mobility and activities of daily living, pain, fatigue, altered self-image, and sleep disturbances, as well as systemic effects that can lead to organ failure and death. An understanding of inflammatory rheumatic diseases and their effects on a patient's function and well-being is essential to developing an appropriate plan of nursing care.

Rheumatic Diseases

Rheumatic disease processes affect males and females of all ages and ethnic groups. Some disorders are more likely to occur at a particular time of life or

to affect one gender more often than the other. In general, women are two to nine times more commonly affected by rheumatic diseases than men (Norris, 2019). Arthritis and other rheumatic diseases and the physical limitations that occur with them are becoming more prominent and a larger public health issue, which can be attributed to the increased number of older adults in the United States.

The onset of these conditions may be acute or insidious, with a course possibly marked by periods of **remission** (a period when disease symptoms are reduced or absent) and **exacerbation** (a period when symptoms occur or increase). Treatment can be simple, aimed at localized relief, or it can be complex, directed toward relief of systemic effects. Permanent changes and disability may result from these disorders.

Nurses need to understand the classification of rheumatic diseases. One system is to classify disease as either monoarticular (affecting a single joint) or polyarticular (affecting multiple joints). Another system is to classify the disease as either inflammatory or noninflammatory. This chapter focuses on inflammatory rheumatic diseases, while noninflammatory rheumatic diseases (i.e., osteoarthritis) are covered in [Chapter 36](#). Conditions that may secondarily affect the musculoskeletal structure are also considered in disease classification.

Pathophysiology

Each of the inflammatory rheumatic diseases exhibits unique pathophysiologic features. Three distinct characteristics of pathophysiology include inflammation, autoimmunity, and degeneration.

Inflammation

Inflammation is a complex physiologic process mediated by the immune system that occurs in response to harmful stimuli like damaged cells or antigens, which may include pathogens (e.g., viruses, bacteria). Inflammation is meant to protect the body from insult by removing the triggering antigen or event. In response to a triggering episode, the antigen stimulus activates the body's immune system to form antibodies like monocytes and T lymphocytes (also referred to as T cells). Next, the immunoglobulin antibodies form immune complexes with antigens. Phagocytosis of the immune complexes is initiated, generating an inflammatory reaction (joint effusion, pain, and edema) (see [Fig. 34-1](#)). Phagocytosis produces chemicals such as leukotrienes and prostaglandins. Leukotrienes contribute to the inflammatory process by attracting other white blood cells to the area. Prostaglandins act as modifiers to inflammation. In some cases, they increase inflammation; in other cases, they decrease it. Leukotrienes and prostaglandins produce enzymes such as collagenase that break down collagen, which is a vital part of a normal joint.

The release of these enzymes in the joint causes edema and proliferation of synovial membrane. In patients with chronic inflammation, the immune response can deviate from normal. Instead of resolution of swelling and joint pain once the triggering event has subsided, **pannus** (proliferation of newly formed synovial tissue infiltrated with inflammatory cells) formation occurs. Destruction of the joint's cartilage and erosion of bone soon follow (Norris, 2019).

The immunologic inflammatory process begins when antigens are presented to T lymphocytes, leading to a proliferation of T and B cells. B cells (also referred to as plasma cells) are a source of antibody-forming cells. In response to specific antigens, plasma cells produce and release antibodies. Antibodies combine with corresponding antigens to form pairs, or immune complexes. The immune complexes build up and are deposited in synovial tissue or other organs in the body, triggering the inflammatory reaction that can ultimately damage the involved tissue (Norris, 2019).

Autoimmunity

A hallmark of inflammatory rheumatic diseases is autoimmunity, where the body mistakenly recognizes its own tissue as a foreign antigen. Autoimmunity leads to destruction of tissue via the same inflammatory process as discussed earlier, along with chronic and long-standing pain. Although focused in the joints, inflammation and autoimmunity also involve other areas. The blood vessels (vasculitis and arteritis), lungs, heart, and kidneys may be affected by the autoimmunity and inflammation. See [Chapter 32](#) for more information on autoimmune disease. A large group of genes, called *human leukocyte antigen* (HLA) genes, has been linked to the immune response and the development of multiple rheumatic diseases (Norris, 2019).

Physiology/Pathophysiology

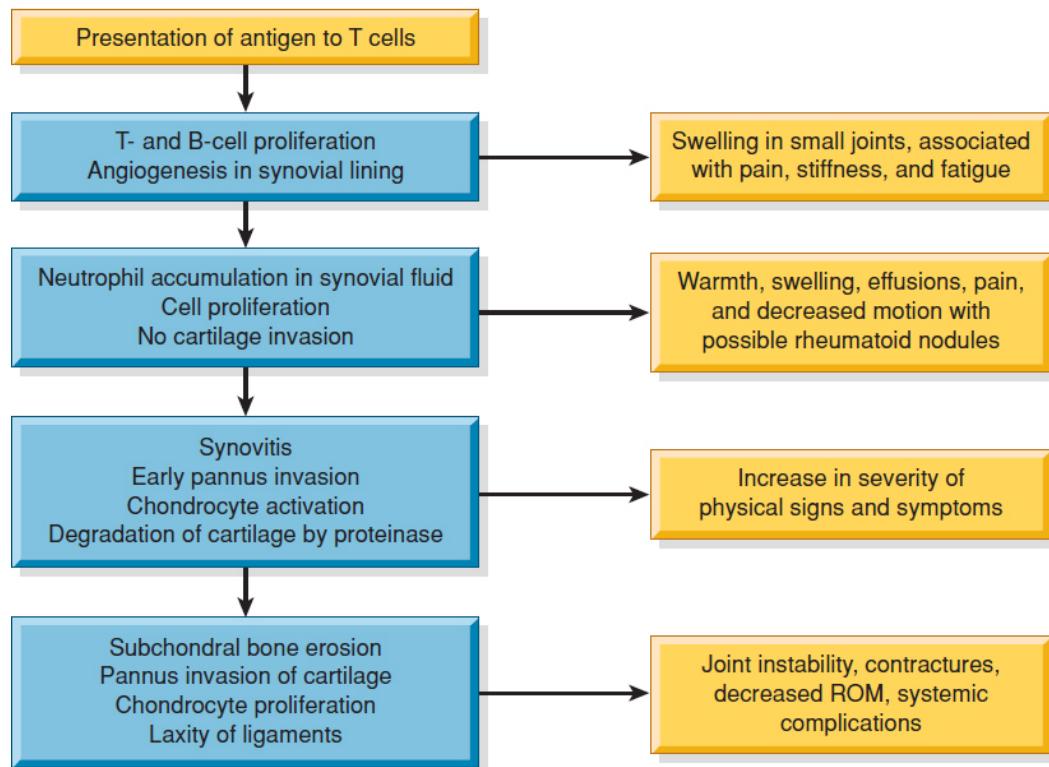


Figure 34-1 • Pathophysiology and associated physical signs of rheumatoid arthritis. ROM, range of motion.

Degeneration

In degenerative rheumatic diseases, inflammation also occurs, but as a secondary process. Although the cause of degeneration of the articular cartilage is poorly understood, the process is known to be metabolically active and therefore is more accurately called *degradation*. One theory of degradation is that genetic or hormonal influences, mechanical factors, and prior joint damage cause cartilage failure. Degradation of cartilage ensues, and increased mechanical stress on bone ends causes stiffening of bone tissue. Another theory is that bone stiffening occurs and results in increased mechanical stress on cartilage, which in turn initiates the processes of degradation. See [Chapter 35](#) for more information on the structure and function of the articular system.

Clinical Manifestations

The most common symptom in the rheumatic diseases is pain. Other common symptoms include joint swelling, limited movement, stiffness, weakness, and fatigue.

Assessment and Diagnostic Findings

Assessment begins with a general health history, which includes the onset of symptoms and how they evolved, family history, past health history, and any other contributing factors. Because many of the rheumatic diseases are chronic conditions, the health history should also include information about the patient's perception of the problem, previous treatments and their effectiveness, the patient's support systems, and the patient's current knowledge base and the source of that information. A complete health history is followed by a complete physical assessment (see [Chapter 4](#)).

Assessment for rheumatic diseases combines the physical examination with a functional assessment (Eliopoulos, 2021). Inspection of the patient's general appearance occurs during the initial contact. Gait, posture, and general musculoskeletal size and structure are observed. Gross deformities and abnormalities in movement are noted. The symmetry, size, and contour of other connective tissues, such as the skin and adipose tissue, are also noted and recorded (Weber & Kelley, 2019). [Chart 34-1](#) outlines the important areas for consideration during the physical assessment. The functional assessment is a combination of history (what the patient reports that they can and cannot do) and examination (observation of activities, in which the patient demonstrates what they can and cannot do, such as dressing and getting in and out of a chair). Observation also includes the adaptations and adjustments the patient may have made (sometimes without awareness)—for example, with shoulder or elbow involvement, the person may bend over to reach a fork rather than raising the fork to the mouth.

Laboratory Studies

In [Table 34-1](#) common laboratory studies are listed with their corresponding normal ranges and significance. Many of the tests require special laboratory techniques and may not be performed in every health care facility. The primary provider determines which tests are necessary based on symptoms, stage of disease, cost, and likely benefit. In some instances, tests are used to monitor the course of the disease.

Other Diagnostic Studies

Imaging studies commonly used for patients with rheumatic diseases include x-ray studies, computed tomography (CT) scans, magnetic resonance imaging (MRI) scans, and arthrography. See [Chapter 35](#) for further information about these and other diagnostic studies.

Medical Management

The chronic nature of rheumatic conditions requires cooperation between the patient and the provider. Patient education regarding the disease process and treatment options is imperative to enable patients to make informed decisions regarding their care. Management of rheumatic diseases is based on a shared decision process between the provider and patient that takes into account the patient's values, preferences, and comorbidities (Singh, Saag, Bridges, et al., 2016).

Pharmacologic Therapy

Medications are used with the rheumatic diseases to manage symptoms, to control inflammation, and, in some instances, to modify the disease. Useful medications include the salicylates, nonsteroidal anti-inflammatory drugs (NSAIDs), and both biologic and nonbiologic disease-modifying antirheumatic drugs (DMARDs). As their name suggests, DMARDs have the ability to suppress the autoimmune response; alter disease progression; and stop or decrease further tissue damage on the joints, cartilage, and organs. DMARDs have been found to halt the progression of bone loss and destruction and can induce remission (Singh et al., 2016). Nonbiologic DMARDs are thought to reduce proinflammatory **cytokines** (cell signaling proteins vital to regulation of hematopoiesis, apoptosis, and immune responses) and increase anti-inflammatory cytokines. Biologic DMARDs, in contrast, have been specifically engineered to target a certain cell or molecule within the immune system to treat the specific rheumatic condition. Specific biologic DMARDs target tumor necrosis factor alpha (TNF- α), B cells, T cells, interleukin 1 (IL-1), and interleukin 6 (IL-6). [Table 34-2](#) reviews select medications.

Controlling the inflammation related to the disease process helps manage pain, but this is often a delayed response. Nonopioid medications are often used for pain management, especially early in the treatment program, until other measures can be instituted.

Nonpharmacologic Pain Management

Nonpharmacologic methods of pain management are important. Heat applications are helpful in relieving pain, stiffness, and muscle spasm (Kapale, Vardharajulu, & Warude, 2017). Superficial heat may be applied in the form of warm tub baths or showers and warm moist compresses. Paraffin baths (dips), which offer concentrated heat, are helpful to patients with wrist and small-joint involvement. Maximum benefit is achieved within 20 minutes after application. More frequent use for shorter lengths of time is most beneficial. Therapeutic exercises can be carried out more comfortably and effectively after heat has been applied (Kapale et al., 2017).

Chart 34-1



ASSESSMENT

Assessing for Rheumatic Disorders

In addition to the head-to-toe assessment or systems review, the following are important areas of consideration to be noted when performing the complete physical assessment of a patient with a known or suspected rheumatic disorder.

Manifestation	Significance
Skin (inquire and inspect)	
Rash, lesions	Associated with systemic lupus erythematosus (SLE) vasculitis, adverse effect of medication
Increased bruising	Associated with several rheumatic diseases and adverse effect of medication
Erythema	Sign of inflammation
Thinning	Adverse effect of medication
Warmth	Sign of inflammation
Photosensitivity	Associated with SLE, dermatomyositis, adverse effect of medication
Hair (inquire and inspect)	
Alopecia or thinning	Associated with rheumatic diseases or adverse effect of medication
Eye (inquire and inspect)	
Dryness, grittiness	Associated with Sjögren's syndrome (commonly occurring with rheumatoid arthritis [RA] and SLE)
Decreased acuity or blindness	Associated with temporal arteritis, medication complications
Cataracts	Adverse effect of medication
Decreased peripheral vision	Adverse effect of medication
Conjunctivitis, uveitis	Associated with ankylosing spondylitis and Reiter's syndrome
Ear (inquire)	
Tinnitus	Adverse effect of medication
Decreased acuity	Adverse effect of medication
Mouth (inquire and inspect)	
Buccal, sublingual lesions	Associated with vasculitis, dermatomyositis, adverse effect of medication
Altered sense of taste	Adverse effect of medication
Dryness	Associated with Sjögren's syndrome
Dysphagia	Associated with myositis
Difficulty chewing	Associated with decreased range of motion of jaw
Chest (inspect and inquire)	
Pleuritic pain	Associated with RA and SLE
Decreased chest expansion	Associated with ankylosing spondylitis
Activity intolerance (dyspnea)	Associated with pulmonary hypertension in scleroderma
Cardiovascular system (inquire, inspect, palpate)	

Blanching of fingers on exposure to cold	Associated with Raynaud's phenomenon
Peripheral pulses	Deficit may indicate vascular involvement or edema associated with medication effect or rheumatic diseases, especially SLE or scleroderma
Abdomen (inquire and palpate)	
Altered bowel habits	Associated with scleroderma, spondylosis, ulcerative colitis, decreased physical mobility, medication effect
Nausea, vomiting, bloating, and pain	Adverse effect of medication
Weight change (measure)	Associated with RA (decreased), adverse effect of medication (increased or decreased)
Genitalia (inquire and inspect)	
Dryness, itching	Associated with Sjögren's syndrome
Abnormal menses	Adverse effect of medication
Altered sexual performance	Fear of pain (or of pain caused by partner) and limitation of motion may affect sexual mobility
Hygiene	Poor hygiene may be related to limitations in activities of daily living
Urethritis, dysuria	Associated with ankylosing spondylitis and Reiter's syndrome
Lesions	Associated with vasculitis
Neurologic (inquire and inspect)	
Paresthesias of extremities; abnormal reflex pattern	Nerve compressions (e.g., carpal tunnel syndrome, spinal stenosis)
Headaches	Associated with temporal arteritis, adverse effect of medication
Musculoskeletal (inspect and palpate)	
Joint redness, warmth, swelling, tenderness, deformity—location of first joint involved, pattern of progression, symmetry, acute vs. chronic nature	Signs of inflammation
Joint range of motion	Decreased range of motion may indicate severity or progression of disease
Surrounding tissue findings	
Muscle atrophy, subcutaneous nodules, popliteal cyst	Extra-articular manifestations
Muscle strength (grip)	Muscle strength decreases with increased disease activity

Adapted from Weber, J. R., & Kelley, J. H. (2019). *Health assessment in nursing* (6th ed.). Philadelphia, PA: Wolters Kluwer.

TABLE 34-1 Common Blood Studies for Rheumatic Diseases

Test	Normal Value	Significance
Serum		
Creatinine		
Metabolic waste excreted through the kidneys	<i>Men:</i> 0.6–1.2 mg/dL (71–106 mmol/L) <i>Women:</i> 0.4–1.0 mg/dL (36–90 mmol/L)	Increase may indicate kidney damage in SLE, scleroderma, and polyarteritis.
Erythrocyte Count		
Measures circulating erythrocytes	<i>Men:</i> 4,200,000–5,400,000/mm ³ (4.2–5.4 × 10 ¹² /L) <i>Women:</i> 3,600,000–5,000,000/mm ³ (3.6–5.0 × 10 ¹² /L)	Decrease can be seen in RA, SLE.
Erythrocyte Sedimentation Rate (ESR)		
Measures the rate at which RBCs settle out of unclotted blood in 1 hour	Westergren: Men under 50 yr: <15 mm/h Men over 50 yr: <20 mm/h Women under 50 yr: <20 mm/h Women over 50 yr: <30 mm/h	Increase is usually seen in inflammatory connective tissue diseases. An increase indicates rising inflammation, resulting in clustering of RBCs, which makes them heavier than normal. The higher the ESR, the greater the inflammatory activity.
Hematocrit		
Measures the size, capacity, and number of cells present in blood	<i>Men:</i> 42–52% <i>Women:</i> 36–48%	Decrease can be seen in chronic inflammation (anemia of chronic disease); also, blood loss through GI bleed.
White Blood Cell Count		
Measures circulating leukocytes	4,500–11,000 cells/mm ³	Decrease may be seen in SLE.
Uric Acid		
Measures level of uric acid in serum	<i>Men:</i> 3.4–7 mg/dL (202–416 µmol/L) <i>Women:</i> 2.4–6 mg/dL (143–357 µmol/L)	Increase is seen with gout. During acute flare, levels may be normal. After flare has subsided, levels will be elevated in gout.

Serum Immunology

Antinuclear Antibody (ANA)

Measures antibodies that react with a variety of nuclear antigens
If antibodies are present, further testing determines the type of ANA circulating in the blood (anti-DNA, anti-RNP).

Negative
Healthy adults may also have a positive ANA.

Positive test may be associated with SLE, RA, scleroderma, Raynaud's disease, Sjögren's syndrome, necrotizing arteritis.
The higher the titer, the greater the inflammation.
The pattern of immunofluorescence (speckled, homogeneous, or nucleolar) helps determine the diagnosis.

Anti-DNA, DNA Binding

Titer measurement of antibody to double-stranded DNA

Negative

High titer is seen in SLE; increases in titer may indicate an increase in disease activity.

C-Reactive Protein (CRP)

Shows presence of abnormal glycoprotein due to inflammatory process

<1 mg/dL (<10 mg/L)

A positive reading indicates active inflammation.

Immunoglobulin Electrophoresis

Measures the values of immunoglobulins

IgA: 60–400 mg/dL (600–4000 mg/L)

Increased levels are found in people who have autoimmune disorders.

IgG: 700–1,500 mg/dL (7–15 g/L)
IgM: 60–300 mg/dL (600–3000 mg/L)

Rheumatoid Factor (RF)

Determines the presence of abnormal antibodies seen in connective tissue disease

Negative

Positive titer >1:80
Present in 80% of those with RA
Positive RF may also suggest SLE, Sjögren's syndrome, or mixed connective tissue disease. The higher the titer (number at right of colon), the greater the inflammation.

Tissue Typing

HLA-B27 Antigen

Measures presence of HLA antigens, which are used for tissue recognition

Negative

Found in 80–90% of those with ankylosing spondylitis and Reiter's syndrome

DNA, deoxyribonucleic acid; GI, gastrointestinal; HLA, human leukocyte antigen; IgA, immunoglobulin A; IgG, immunoglobulin G; IgM, immunoglobulin M; RA, rheumatoid arthritis; RBCs, red blood cells; RNP, ribonucleoprotein; SLE, system lupus erythematosus.

Adapted from Fischbach, F. T., & Fischbach, M. A. (2018). *A manual of laboratory and diagnostic tests* (10th ed.). Philadelphia, PA: Wolters Kluwer.

TABLE 34-2

Select Medications Used in Rheumatic Diseases

Medication	Action, Use, and Indication	Nursing Considerations
Salicylates		
<i>Acetylated:</i> aspirin <i>Nonacetylated:</i> choline trilisalicylate, salsalate, sodium salicylate	<i>Action:</i> Anti-inflammatory, analgesic, antipyretic Acetylated salicylates are platelet aggregation inhibitors	Administer with food, milk, antacids or large glass of water to reduce GI effects. Assess for tinnitus, gastric intolerance, GI bleeding, and purpura. Administer enteric coated or extended release whole, do not crush.
Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)		
diclofenac, diflunisal, etodolac, ibuprofen, ketoprofen, meloxicam, nabumetone, naproxen, piroxicam, sulindac <i>COX-2 enzyme blockers:</i> celecoxib	<i>Action:</i> Anti-inflammatory, analgesic, antipyretic, platelet aggregation inhibitor Anti-inflammatory effect occurs 2–4 wk after initiation All NSAIDs are useful for short-term treatment of acute gout attack NSAIDs are an alternative to salicylates for first-line therapy in several rheumatic diseases <i>Action:</i> Inhibit only COX-2 enzymes, which are produced during inflammation, and spare COX-1 enzymes, which can be protective to the stomach	Administer NSAIDs with food. Monitor for GI, CNS, cardiovascular, renal, hematologic, and dermatologic adverse effects. Avoid salicylates; use acetaminophen for additional analgesia. Watch for possible confusion in older adults. Monitoring is the same as for other NSAIDs. Increased risk of cardiovascular events, including myocardial infarction and stroke. Appropriate for older adults and patients who are at high risk for gastric ulcers.
Disease-Modifying Antirheumatic Drugs (DMARDs)		
<i>Antimalarials:</i> hydroxychloroquine, chloroquine	<i>Action:</i> Anti-inflammatory, inhibit lysosomal enzymes Slow acting; onset may take 2–4 mo. May be used in conjunction with other DMARD therapy. Useful in RA and SLE.	May be administered concurrently with NSAIDs. Assess for visual changes, GI upset, skin rash, headaches, photosensitivity, bleaching of hair. Emphasize need for ophthalmologic

examinations (every 6–12 mo).

Janus Kinase (JAK) inhibitors

tofacitinib, baricitinib *Action:* Enters the cell and binds to the active JAK site, inhibits autophosphorylation and JAK activation that inhibits cytokine production. May be used in combination with methotrexate or other nonbiologic DMARDs. May also be used as monotherapy.

Administer twice a day (immediate release) or once daily (extended release). Do not administer with biologic DMARDs or potent immunosuppressants. Test for latent TB before initiation of therapy. Monitor liver enzymes routinely.

sulfasalazine

Action: Anti-inflammatory, reduces lymphocyte response, inhibits angiogenesis
Useful in RA, seronegative spondyloarthropathies

Administer concurrently with NSAIDs.
Do not use in patients with allergy to sulfa medications or salicylates.

Emphasize adequate fluid intake.
Assess for GI upset, skin rash, headache, liver abnormalities, anemia.

Immunosuppressives:

methotrexate,
azathioprine,
cyclophosphamide

Action: Nonbiologic immune suppression, affect DNA synthesis and other cellular effects
Have teratogenic potential; azathioprine and cyclophosphamide reserved for more aggressive or unresponsive disease
Methotrexate is generally the first-line agent for RA treatment; also useful in SLE. Methotrexate may be given orally or by intramuscular or subcutaneous injection

Assess for bone marrow suppression, GI ulcerations, skin rashes, alopecia, bladder toxicity, increased infections.

Monitor CBC, liver enzymes, creatinine at 6 wk after initiation, then every 2–3 mo or accordingly.

Advise patient of contraceptive measures because of teratogenicity.

cyclosporine

Action: Nonbiologic immune suppression by inhibiting T lymphocytes
Used for severe, progressive RA, unresponsive to other DMARDs
Used in combination with methotrexate

Assess slow dose titration upward until response noted or toxicity occurs.

Assess for toxic effects, such as bleeding gums,

		fluid retention, hair growth, tremors. Monitor blood pressure and renal function (creatinine) every 2 wk until stable.
Immunomodulators		
<i>Pyrimidine synthesis inhibitor:</i> leflunomide	<i>Action:</i> Nonbiologic with antiproliferative and anti-inflammatory effects; used in moderate to severe RA May be used alone or in combination with other DMARDs	Long half-life; requires loading dose followed by daily administration. Assess for diarrhea, hair loss, skin rash, mouth sores. Monitor liver function tests. Contraindicated in pregnancy and breastfeeding. Given orally.
<i>TNF-blocking agents:</i> adalimumab, certolizumabpegol, etanercept, infliximab, golimumab	<i>Action:</i> Biologic response modifier that binds to TNF, a cytokine involved in inflammatory and immune responses. Used in moderate to severe RA. Can be used alone or with methotrexate or other nonbiologic DMARDs. Adalimumab is given by subcutaneous injection every 2 wk, but may be used every week if efficacy not reached. Certolizumabpegol is given by subcutaneous injection every 2 wk. Etanercept is given by subcutaneous injection weekly. Infliximab is given intravenously over 2 h or more. Medication must be refrigerated. Golimumab is given by subcutaneous injection once a month. Golimumab SQ is a second alternative that is administered intravenously every 8 wk after initial 2 loading doses	Patient should be tested for tuberculosis before beginning this medication. Educate patient about subcutaneous self-injection. Monitor for injection site reactions. Educate patient about increased risk for infection and to withhold medication if fever occurs. Notify provider if any illness or infection occurs and medication is held.
<i>T-cell costimulation modulator:</i> abatacept	<i>Action:</i> Blocks one of the pathways needed to fully activate T cells, decreasing inflammatory and immunologic responses. Used in moderate to severe RA	Administered IV initially, then transitions to subcutaneous dosage once weekly. Educate patient about

	<p>unresponsive to TNF inhibitors. Used with methotrexate or DMARDs other than TNF inhibitors or anakinra.</p>	<p>subcutaneous self-injections given daily. Monitor for injection site reactions. Educate patient about increased risk of infection and to withhold medication if fever occurs. IV infusions are given every 4 wk over 30-min infusion.</p>
<i>B-cell production blocker:</i> rituximab	<p><i>Action:</i> Binds to B-lymphocyte CD20 surface antigens. Used in refractory RA in patients with inadequate response to TNF antagonist. Given with methotrexate.</p>	<p>Rituximab is given as two 1000-mg doses via IV infusion separated by 2 wk. Given on wk 0 and 2, and then subsequent doses are infused 24 wk later or based on clinical diagnosis (commonly given every 6 mo). Premedicate with acetaminophen, antihistamine, and methylprednisolone 30 min prior to infusion of rituximab. Educate patient about increased risk of infection.</p>
<i>Human IL-1 receptor antagonist:</i> anakinra	<p><i>Action:</i> Blocks IL-1 receptors, decreasing inflammatory and immunologic responses. Used in moderate to severe RA. Can be used alone or with methotrexate or DMARDs other than TNF-blocking agents</p>	<p>Given daily by subcutaneous injection. Educate patient about subcutaneous self-injections given daily. Medication must be refrigerated. Monitor for injection site reactions. Educate patient about increased risk of infection and to withhold medication if fever occurs.</p>
<i>Human IL-6 receptor antagonist:</i> tocilizumab	<p><i>Action:</i> Binds to and inhibits IL-6 receptors, decreasing inflammatory and immunologic responses. Can be used alone or with methotrexate or in combination with other nonbiologic DMARDs</p>	<p>Administered IV every 4 wk. Educate patient about increased risk of infection.</p>

Corticosteroids

prednisone,
prednisolone,
hydrocortisone

Action: Anti-inflammatory. Used for shortest duration and at lowest dose possible to minimize adverse effects.

Useful for unremitting RA, SLE, polymyalgia rheumatica, myositis, arteritis

Fast acting; onset in days

Intra-articular injections useful for joints unresponsive to NSAIDs

Assess for toxicity:
Cataracts, GI irritation, hyperglycemia, hypertension, fractures, avascular necrosis, hirsutism, psychosis.

Joints most amenable to injections include ankles, knees, hips, shoulders, and hands.

Repeated injections can cause joint damage.
Use caution in patients diagnosed with diabetes, due to effects causing elevation in blood sugar.

CBC, complete blood count; CNS, central nervous system; COX, cyclooxygenase; GI, gastrointestinal; IL-1, interleukin 1; IL-6, interleukin 6; IV, intravenous; RA, rheumatoid arthritis; SLE, systemic lupus erythematosus; TNF, tumor necrosis factor.

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer; Mogul, A., Corsi, K., & McAuliffe, L. (2019). Baricitinic: The second FDA approved JAK inhibitor for the treatment of rheumatoid arthritis. *Annals of Pharmacotherapy*, 53(9), 947–953.

Devices such as braces, splints, and assistive devices for ambulation (e.g., canes, crutches, walkers) ease pain by limiting movement or stress from putting weight on painful joints. Acutely inflamed joints can be rested by applying splints to limit motion. Splints also support the joint to relieve spasm. Canes and crutches can relieve stress from inflamed and painful weight-bearing joints while promoting safe ambulation. Cervical collars may be used to support the weight of the head and limit cervical motion. A metatarsal bar or special pads may be put into the patient's shoes if foot pain or deformity is present. A combination of methods may be required, because different methods often work better at different times.

Exercise and Activity

The ongoing nature of most rheumatic diseases makes it important to maintain and, when possible, improve joint mobility and overall functional status. Appropriate programs of exercise have been shown to decrease pain and improve function (Eliopoulos, 2021). Changes in gait as well as joint limitations commonly require referral for rehabilitation therapy. An individualized exercise program is crucial to improve movement. [Table 34-3](#)

summarizes the exercises appropriate for patients with rheumatic diseases. Physical and occupational therapy programs and interventions are beneficial in improving physical activity and maintaining range of motion. Such interventions may include stretching exercises, muscle conditioning, aerobic exercise, massage, acupuncture, and chiropractic and osteopathic manipulation. Other strategies for decreasing pain include muscle relaxation techniques, imagery, self-hypnosis, and distraction. A mild analgesic agent may be suggested before exercise to improve pain during exercise. A weight reduction program may be recommended to relieve stress on painful joints for patients who are overweight. Any patient who experiences acute or prolonged pain associated with exercise should report the symptoms to their primary provider for evaluation.

TABLE 34-3 Exercise to Promote Mobility

Type of Exercise	Purpose	Recommended Performance	Precautions
Range of motion	Maintain flexibility and joint motion	Active or active/self-assisted at least daily	Reduce the number of repetitions when inflammation is present
Isometric exercise	Improve muscle tone, static endurance, and strength; prepare for dynamic and weight-bearing exercises	Perform at 70% of maximal voluntary contraction daily	Monitor blood pressure; isometric exercises may increase blood pressure and decrease blood flow to muscles
Dynamic exercise	Maintain or increase dynamic strength and endurance; increase muscle power; enhance synovial blood flow; promote strength of bone and cartilage	Start with repetitions against gravity and add progressive resistance; perform 2–3 days per week	May increase biomechanical stress on unstable or misaligned joints
Aerobic exercise	Improve cardiovascular fitness and endurance	Perform 3–5 days per week for 20–30 min of moderate-intensity exercise	Progress slowly as activity tolerance and fitness improve
Pool exercise	Water supports or resists movement; warm water may provide muscle relaxation	Provide buoyant medium for performance of dynamic or aerobic exercise	Heated swimming pool; deep water to minimize joint compression; nonslip footwear for safety and comfort. Receive appropriate education in a program designed for people with arthritis

Adapted from Kapale, P., Vardharajulu, G., & Warude, T. (2017). Effect of free exercise and rheumatoid arthritis. *Indian Journal of Physiotherapy and Occupational Therapy*, 11(3), 62–65.

The major challenge for the patient and the health care provider is the need to adjust all aspects of treatment according to the activity of the disease. Especially for the patient with an active diffuse connective tissue disease, such as rheumatoid arthritis (RA) or systemic lupus erythematosus (SLE), activity levels may vary from day to day and even within a single day.

Sleep

Short-term use of low-dose antidepressant medications, such as amitriptyline, may be prescribed to reestablish adequate sleep patterns and improve pain management (Comerford & Durkin, 2020). Patients need restful sleep so that they can cope with pain, minimize physical fatigue, and deal with the changes related to having a chronic disease. In patients with acute disease, sleep time is frequently reduced and fragmented by prolonged awakenings. Stiffness, depression, and medications may also compromise the quality of sleep and increase daytime fatigue. A sleep-inducing routine, medication, and comfort measures may help improve the quality of sleep.

Education about sleep hygiene strategies may help promote restorative sleep. These strategies include establishing a set time to sleep and a regular wake-up time, creating a quiet sleep environment with a comfortable room temperature, avoiding factors that interfere with sleep (e.g., the use of alcohol and caffeine), using relaxation exercises, and getting out of bed and engaging in another activity (e.g., reading) if unable to sleep.

Nursing Management

Much of the care of patients with arthritis involves self-management; thus, using a standardized assessment of self-management behaviors will help plan effective on-going care and treatment targets (Oh, Han, Kim, et al., 2018). The Nursing Research Profile in [Chart 34-2](#) describes one such tool. [Chart 34-3](#) details the nursing diagnoses, interventions, and expected outcomes for the patient with a rheumatic disorder.



Gerontologic Considerations

The various rheumatic disease conditions in the older adult pose unique challenges. These challenges relate to disability, cognitive changes, comorbid conditions, and diagnosis. Musculoskeletal problems are the most frequently reported conditions in older adults (Eliopoulos, 2021) and will be seen more frequently by health professionals in the coming years along with associated disability, especially among frail older adults.

Comorbid conditions pose a unique challenge in diagnosing rheumatic disease in older adults because they have the potential to mask or alter presenting symptoms. The frequency, pattern of onset, clinical features, severity, and effects on function of the rheumatic disease in older patients needs to be assessed. One study reported that functional disability was correlated with disease state and thus must be addressed in planning care (Omma, Celik, Bes, et al., 2018). Additional medical conditions may take precedence over the rheumatic disease, causing it to become a secondary diagnosis and concern. Decreased vision and altered balance, often present in older adults, may be problematic if rheumatic disease in the lower extremities

affects locomotion. The combination of decreased hearing and visual acuity, memory loss, and depression contributes to failure to follow the treatment regimen in older adult patients as well (Eliopoulos, 2021). Special techniques for promoting patient safety, self-management, and strategies such as memory aids for medications may be necessary.

Chart 34-2 NURSING RESEARCH PROFILE

Assessing Self-Management in Patients with Arthritis

Oh, H. S., Han, S. Y., Kim, S. H., et al. (2018). Development and validity testing of an arthritis self-management assessment tool. *Orthopaedic Nursing*, 37(1), 24–35.

Purpose

Self-management is central to arthritis treatment; yet, no instrument existed to measure arthritis self-management ability. Therefore, the purpose of this research was to develop and test the reliability and validity of a comprehensive tool to assess self-management in patients with arthritis.

Design

A nonexperimental correlational design was used for this study. Items for inclusion on the Arthritis Self-Management Assessment Tool (ASMAT) were generated using a chronic illness management model. Content validity of the initial 42 items were reviewed by a panel of experts and decreased to 32 items. The tool was then tested with 150 patients with arthritis in an outpatient setting. Factor analysis was used to test construct validity.

Findings

The mean age of participants was 52 years and approximately 60% were male. There were 32 items generated and tested in the final version of the ASMAT. The 32 items were validated and the scale was found to have 3 subscales: that of medical management tasks (10 items), behavioral management tasks (13 items), and psycho-emotional management tasks (9 items). Confirmatory factor analysis showed construct validity for the 32 item tool. Cronbach α levels showed the overall toll and the 3 subscales to be reliable.

Nursing Implications

Nurses are in a key position to assist patients with arthritis to self-manage their care. Performing an ASMAT helps evaluate the patient's self-management abilities and the effectiveness of self-management interventions. Early identification of barriers to adoption of self-management strategies can lead to improved symptom management, independence, and improved quality of life.

Behavioral clues such as gait patterns, guarding, and joint flexion may aid the nurse in assessing the patient's pain when cognitive impairment is present. Older adults, especially men, may also neglect to communicate their pain unless elicited by the provider. Pain, in general, in this population that is not treated or undertreated may impact the quality of life for these patients, which can exacerbate all other medical conditions.

Chart 34-3



PLAN OF NURSING CARE

Care of the Patient with a Rheumatic Disorder

Nursing Diagnosis: Acute and chronic pain associated with inflammation and increased disease activity, tissue damage, fatigue, or lowered tolerance level

Goal: Improvement in comfort level; incorporation of pain management techniques into daily life

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Provide variety of comfort measures: <ol style="list-style-type: none"> a. Application of heat or cold b. Massage, position changes, rest c. Foam mattress, supportive pillow, splints d. Relaxation techniques, diversional activities 2. Administer anti-inflammatory, analgesic, and slow-acting antirheumatic medications as prescribed. 3. Individualize medication schedule to meet patient's need for pain management. 4. Encourage verbalization of feelings about pain and chronicity of disease. 5. Assess for subjective changes in pain. 	<ol style="list-style-type: none"> 1. Pain may respond to nonpharmacologic interventions, such as exercise, relaxation, and thermal modalities. 2. Pain of rheumatic disease responds to monotherapy or combination medication regimens. 3. Previous pain experiences and management strategies may be different from those needed for persistent pain. 4. Verbalization promotes coping. 5. The impact of pain on an individual's life often leads to misconceptions about pain and pain management techniques. The individual's description of pain is a more reliable indicator than objective measurements 	<ul style="list-style-type: none"> • Identifies factors that exacerbate or influence pain response • Identifies and uses pain management strategies • Verbalizes decrease in pain • Reports signs and symptoms of side effects in timely manner to prevent additional problems • Verbalizes that pain is characteristic of rheumatic disease • Establishes realistic pain relief goals • Identifies changes in quality or intensity of pain

such as change in vital signs, body movement, and facial expression.

NURSING DIAGNOSIS: Fatigue associated with increased disease activity, pain, inadequate sleep/rest, deconditioning, inadequate nutrition, emotional stress, anxiety, and depressive symptoms

Goal: Incorporates as part of daily activities strategies necessary to modify fatigue

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Provide education about fatigue. <ol style="list-style-type: none"> a. Describe relationship of disease activity to fatigue. b. Describe comfort measures while providing them. c. Develop and encourage a sleep routine (warm bath and relaxation techniques that promote sleep). d. Explain importance of rest for relieving systematic, articular, and emotional stress. e. Explain how to use energy conservation techniques (pacing, delegating, setting priorities). f. Identify physical and emotional factors that can cause fatigue. 	<ol style="list-style-type: none"> 1. The patient's understanding of fatigue will affect their actions. <ol style="list-style-type: none"> a. The amount of fatigue is directly related to the activity of the disease. b. Relief of discomfort can relieve fatigue. c. Effective bedtime routine promotes restorative sleep. d. Different kinds of rest are needed to relieve fatigue and are based on patient's need and response. e. A variety of measures can be used to conserve energy. 	<ul style="list-style-type: none"> • Self-evaluates and monitors fatigue pattern • Verbalizes the relationship of fatigue to disease activity • Uses comfort measures as appropriate • Practices effective sleep hygiene and routine • Makes use of various assistive devices (splints, canes) and strategies (bed rest, relaxation techniques) to ease different kinds of fatigue • Incorporates time management

<p>2. Facilitate development of appropriate activity/rest schedule.</p> <p>3. Encourage adherence to the treatment program.</p> <p>4. Refer to and encourage a conditioning program.</p> <p>5. Encourage adequate nutrition, including source of iron from food and supplements.</p>	<p>f. Awareness of the various causes of fatigue provides the basis for measures to modify the fatigue.</p> <p>2. Alternating rest and activity conserves energy while allowing most productivity.</p> <p>3. Overall control of disease activity can decrease the amount of fatigue.</p> <p>4. Deconditioning resulting from lack of mobility, understanding, and disease activity contributes to fatigue.</p> <p>5. A nutritious diet can help counteract fatigue.</p>	<p>strategies in daily activities</p> <ul style="list-style-type: none"> • Uses appropriate measures to prevent physical and emotional fatigue • Has an established plan to ensure well-paced, therapeutic activity schedule • Adheres to therapeutic program • Follows a planned conditioning program • Consumes a nutritious diet consisting of the five major groups and recommended daily allowance of vitamins and minerals
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Nursing Diagnosis: Impaired mobility associated with decreased range of motion, muscle weakness, pain on movement, limited endurance, lack of or improper use of ambulatory devices

Goal: Attains and maintains optimal functional mobility

Nursing Interventions	Rationale	Expected Outcomes
1. Encourage verbalization	1. Mobility is not necessarily	• Identifies factors that

<p>regarding limitations in mobility.</p> <p>2. Assess need for occupational or physical therapy consultation.</p> <ul style="list-style-type: none"> a. Emphasize range of motion of affected joints. b. Promote the use of assistive ambulatory devices. c. Explain the use of safe footwear. d. Use individual appropriate positioning/posture. <p>3. Assist to identify environmental barriers.</p> <p>4. Encourage independence in mobility and assist as needed.</p> <ul style="list-style-type: none"> a. Allow ample time for activity. b. Provide rest period after activity. c. Reinforce principles of pacing and work simplification. <p>5. Initiate referral to community health agency.</p>	<p>related to deformity. Pain, stiffness, and fatigue may temporarily limit mobility. The degree of mobility is not synonymous with the degree of independence. Decreased mobility may influence a person's self-concept and lead to social isolation.</p> <p>2. Therapeutic exercises, proper footwear, and assistive equipment may improve mobility. Correct posture and positioning are necessary for maintaining optimal mobility.</p> <p>3. Furniture and architectural adaptations may enhance mobility.</p> <p>4. Changes in mobility may lead to a decrease in personal safety.</p> <p>5. The degree of mobility may be slow to improve or may not improve with intervention.</p>	<p>interfere with mobility</p> <ul style="list-style-type: none"> • Describes and uses measures to prevent loss of motion • Identifies environmental (home, school, work, community) barriers to optimal mobility • Uses appropriate techniques, assistive equipment, or both to aid mobility • Identifies community resources available to assist in managing decreased mobility
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Nursing Diagnosis: Able to perform self care associated with contractures, fatigue, or loss of motion

Goal: Performs self-care activities independently or with the use of resources

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assist patient to identify self-care deficits and factors that interfere with ability to perform self-care activities. 2. Develop a plan based on the patient's perceptions and priorities on how to establish and achieve goals to meet self-care needs, incorporating energy conservation, and work simplification concepts. <ol style="list-style-type: none"> a. Provide appropriate assistive devices. b. Reinforce correct and safe use of assistive devices. c. Allow patient to control timing of self-care activities. d. Explore with the patient different ways to perform difficult tasks or ways to enlist the help of someone else. 3. Consult with community health care agencies when individuals have attained a maximum level of self-care yet still have some 	<ol style="list-style-type: none"> 1. The ability to perform self-care activities is influenced by the disease activity and the accompanying pain, stiffness, fatigue, muscle weakness, loss of motion, and depression. 2. Assistive devices may enhance self-care abilities. Effective planning for changes must include the patient, who must accept and adopt the plan. 3. Individuals differ in ability and willingness to perform self-care activities. Changes in ability to care for self may lead to a decrease in personal safety. 	<ul style="list-style-type: none"> • Identifies factors that interfere with the ability to perform self-care activities • Identifies alternative methods for meeting self-care needs • Uses alternative methods for meeting self-care needs • Identifies and uses other health care resources for meeting self-care needs

deficits, especially regarding safety.

Nursing Diagnosis: Disturbed body image associated with physical and psychological changes and dependency imposed by chronic illness

Goal: Adapts to physical and psychological changes imposed by the rheumatic disease

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Help patient identify elements of control over disease symptoms and treatment.2. Encourage patient's verbalization of feelings, perceptions, and fears.<ol style="list-style-type: none">a. Help to assess present situation and identify problems.	<ol style="list-style-type: none">1. The individual's self-concept may be altered by the disease or its treatment.2. The individual's coping strategies reflect the strength of their self-concept.	<ul style="list-style-type: none">• Verbalizes an awareness that changes taking place in self-concept are normal responses to rheumatic disease and other chronic illnesses• Identifies strategies to cope with altered self-concept

Nursing Diagnosis: Difficulty coping associated with actual or perceived lifestyle or role changes

Goal: Use of effective coping behaviors for dealing with actual or perceived limitations and role changes

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Identify areas of life affected by disease. Answer questions and dispel possible myths. <ol style="list-style-type: none"> a. Assist to identify past coping mechanisms. b. Assist to identify effective coping mechanisms. 2. Develop plan for managing symptoms and enlisting support of family and friends to promote daily function. 	<ol style="list-style-type: none"> 1. The effects of disease may be more or less manageable once identified and explored reasonably. 2. By taking action and involving others appropriately, patient develops or draws on coping skills and community support. 	<ul style="list-style-type: none"> • Names functions and roles affected and not affected by disease process • Describes therapeutic regimen and states actions to take to improve, change, or accept a particular situation, function, or role

Collaborative Problems: Complications secondary to effects of medications

Goal: Absence or resolution of complications

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Perform periodic clinical assessment and laboratory evaluation. 	<ol style="list-style-type: none"> 1. Skillful assessment helps detect early symptoms of side effects of medications. 	<ul style="list-style-type: none"> • Adheres to monitoring procedures and experiences minimal side effects

Provide education about correct self-administration, potential side effects, and importance of monitoring.	2. The patient needs accurate information about medications and potential side effects to avoid or manage them.	• Takes medication as prescribed and lists potential side effects
3. Counsel regarding methods to reduce side effects and manage symptoms.	3. Appropriate identification and early intervention may minimize complications.	• Identifies strategies to reduce or manage side effects
4. Administer medications in modified doses as prescribed if complications occur.	4. Modifications may help minimize side effects or other complications.	• Reports that side effects or complications have subsided

Pharmacologic therapy (including analgesic agents), exercise, postural assistance, modification of activities of daily living, and psychological support are useful components of a self-management program for the older adult (Oh et al., 2018).

Identifying the effects of the rheumatic disease on the patient's lifestyle, independence, and psychological status is important and can improve the quality of life for older adults. Depressed mood is routinely found in those suffering from chronic joint disease. The body image and self-esteem of the older adult with rheumatic disease, combined with underlying depression, may interfere with the use of assistive devices such as canes. The use of adaptive equipment such as long-handled reachers or tongs may be viewed by the older adult as evidence of aging rather than as a means of increasing independence.

Because most rheumatic diseases involve pain, especially with joints, some older adults may consider their symptoms as inevitable consequences of aging. In fact, many older adults expect and accept the immobility and self-care problems related to the rheumatic diseases and do not seek help, thinking that nothing can be done.

The older adult usually has a lifelong pattern of dealing with the stresses of daily life. Depending on the success of that pattern, the older adult can often maintain a positive attitude and self-esteem when faced with a rheumatic disease, especially if support is available. Previous stress management strategies are assessed. If these strategies have been effective, the patient is

encouraged and supported in their use. If they were ineffective, the nurse assists the patient in identifying alternative strategies, encourages the use of new strategies, and assesses their effectiveness.

Pharmacologic treatment of rheumatic disease in older patients is more difficult than in younger patients. If therapeutic medications have an effect on the senses (hearing, cognition), this effect is intensified in the older adult. The cumulative effect of medications, in general, is accentuated because of the physiologic changes of aging. For example, decreased renal function in the older adult alters the metabolism of certain medications, such as NSAIDs. Older adults are more prone to side effects associated with the use of multiple-drug therapy.

Partly because of the more frequent contact of older adults with health care providers for a variety of health issues, overtreatment or inappropriate treatment is possible. Complaints of pain may be met with a prescription for analgesic agents rather than instructions for rest, the use of an assistive device, and local comfort measures such as heat or cold. Acetaminophen may be appropriate and worth trying before other medications that pose a greater chance of side effects. NSAIDs can be used; however, long-term use of NSAIDs can increase the risks of peptic ulcers, hemorrhage, and cardiovascular toxicity (Comerford & Durkin, 2020).

Intra-articular corticosteroid injections, with their usually rapid relief of symptoms, may be requested by the patient who is unaware of the consequences of too-frequent use of this treatment. In addition, exercise programs may not be instituted or may be ineffective because the patient expects results to occur quickly or fails to appreciate the effectiveness of a program of exercise. In fact, strength training is encouraged in the older adult with chronic diseases.

Diffuse Connective Tissue Diseases

Diffuse connective tissue disease refers to a group of systemic disorders that are chronic in nature and are characterized by diffuse inflammation and degeneration in the connective tissues. These disorders share similar clinical features and may affect some of the same organs. The characteristic clinical course is one of exacerbations and remissions. Although diffuse connective tissue diseases have unknown causes, they are thought to be the result of immunologic abnormalities. They include RA, SLE, Sjögren's syndrome, scleroderma, polymyositis, polymyalgia rheumatica (PMR), and giant cell arteritis (GCA).

Rheumatoid Arthritis

Rheumatoid arthritis is an autoimmune disease of unknown origin that affects 1% to 2% of the population worldwide, with females having a three times greater incidence than males. It may occur at any age but the onset commonly occurs between the third and sixth decade of life. The incidence of RA increases after the sixth decade of life (Norris, 2019). RA that occurs after the age of 65 is referred to as elderly onset RA (Norris, 2019). Additional risks that have been identified include family history, environmental influences such as diet or geographic location, nulliparity, as well as the modifiable factors of smoking and obesity (Mogul, Corsi, & McAuliffe, 2019).

Pathophysiology

The exact mechanism of action for the etiology of RA is unknown. Evidence points to a genetic predisposition and the development of immunologically mediated joint inflammation (Eliopoulos, 2021; Norris, 2019). An autoimmune reaction (see Fig. 34-1) occurs in the synovial tissue. RA synovium breaks down collagen, causing edema, proliferation of the synovial membrane, and ultimately pannus formation. Pannus destroys cartilage and erodes the bone. The consequence is the loss of articular surfaces and joint motion. Muscle fibers undergo degenerative changes. Tendon and ligament elasticity and contractile power are lost.

The RA inflammatory process has also been implicated in other disease processes (i.e., arteriosclerosis). It is hypothesized that the RA disease process somehow interferes with the production of high-density lipoprotein cholesterol, which is the form of cholesterol responsible for decreasing cellular lipids and, therefore, is considered antiatherosclerotic.

The nervous system is also affected by the RA inflammatory process. The synovial inflammation can compress the adjacent nerve, causing neuropathies and paresthesias. Axonal degeneration and neuronal demyelination are also possible due to the infiltration of polymorphonuclear leukocytes, eosinophils, and mononuclear cells, causing necrotizing or occlusive vasculitis (Norris, 2019).

Clinical Manifestations

The American College of Rheumatology and the European League Against Rheumatism have collaborated and established criteria for classifying RA. These criteria are based on a point system where a total score of 6 or greater is required for the diagnosis of RA. The scoring system is based on joint involvement (number of joints affected), serology (low positive or high positive rheumatoid factor [RF] or anti-citrullinated peptide antibody [ACPA]), abnormal results of the acute phase reactants (erythrocyte sedimentation rate [ESR] or C-reactive protein [CRP]), and duration of symptoms greater than 6

weeks. Patients diagnosed with RA who are excluded from these diagnostic criteria include: (1) patients who have one joint with synovitis that is not related to any other clinical disease and who also score at least 6 to 10 points on the scale, and (2) patients diagnosed with bony erosions on x-ray (Aletaha, Neogi, Silman, et al., 2010; Molano-Gonzalez, Olivares-Matinez, Anaya, et al., 2019).

The initial clinical manifestations of RA include symmetric joint pain and morning joint stiffness lasting longer than 1 hour. Over the course of the disease, clinical manifestations of RA vary, usually reflecting the stage and severity of the disease. Symmetric joint pain, swelling, warmth, erythema, and lack of function are classic symptoms. Palpation of the joints reveals spongy or boggy tissue. Often, fluid can be aspirated from the inflamed joint. Characteristically, the pattern of joint involvement begins in the small joints of the hands, wrists, and feet (Omma et al., 2018). As the disease progresses, the knees, shoulders, hips, elbows, ankles, cervical spine, and temporomandibular joints may be affected. The onset of symptoms is usually acute. Symptoms are usually bilateral and symmetric.

In the early stages of disease, even before the presentation of bony changes, limitation in function can occur when there is active inflammation in the joints. Joints that are hot, swollen, and painful are not easily moved. The patient tends to guard or protect these joints by immobilizing them. Immobilization for extended periods can lead to contractures, creating soft tissue deformity.

Deformities of the hands (e.g., ulnar deviation and swan neck deformity) and feet are common in RA (see [Chapter 35](#), [Fig. 35-6](#)). The deformity may be caused by misalignment resulting from swelling, progressive joint destruction, or the subluxation (partial dislocation) that occurs when one bone slips over another and eliminates the joint space. Deformities of RA differ from those seen with osteoarthritis (OA), such as Heberden's and Bouchard's nodes (see [Chapter 36](#)).

RA is a systemic disease with multiple extra-articular features. Most common are fever, weight loss, fatigue, anemia, lymph node enlargement, and Raynaud's phenomenon (cold- and stress-induced vasospasm causing episodes of digital blanching or cyanosis). Rheumatoid nodules are common in patients with more advanced RA. These nodules are usually nontender and movable in the subcutaneous tissue. They usually appear over bony prominences such as the elbow, are varied in size, and can disappear spontaneously or progress to ulceration (Weber & Kelley, 2019; Young, 2019). Nodules occur only in people who have rheumatoid factor. Other extra-articular features include arteritis, neuropathy, pericarditis, splenomegaly, and Sjögren's syndrome (dry eyes and dry mucous membranes).

Assessment and Diagnostic Findings

Several assessment findings are associated with RA: rheumatoid nodules, joint inflammation detected on palpation, and certain laboratory findings. The history and physical examination focus on manifestations, such as bilateral and symmetric stiffness, tenderness, swelling, and temperature changes in the joints (Weber & Kelley, 2019). The patient is also assessed for extra-articular changes; these often include weight loss, sensory changes, lymph node enlargement, and fatigue. Symptoms and examination findings are often recorded using a disease activity score, a variety of which are in use, to evaluate disease activity, help guide treatment decisions, and monitor treatment efficacy (Mahmood, van Tuyl, Schoonmade, et al., 2019).

Rheumatoid factor is present in many patients with RA, but its presence alone is not diagnostic of RA, and its absence does not rule out the diagnosis. Antibodies to cyclic citrullinated peptide (anti-CCP) have a specificity of approximately 95% at detecting RA (Norris, 2019). The ESR and CRP tend to be significantly elevated in the acute phases of RA and are therefore useful in monitoring active disease and disease progression. The complete blood count (CBC) should be assessed to establish a baseline count especially prior to starting medications (Fischbach & Fischbach, 2018). Patients may exhibit anemia, and platelets may be elevated due to the inflammatory process. A tuberculin (TB) skin test should be done prior to the initiation of certain medications to rule out tuberculosis. In the event the patient has latent TB and has never been treated, the infection can be reactivated. The patient should also be assessed for hepatitis B and hepatitis C, which could impact treatment strategies if positive. If the client tests positive for hepatitis, the infection should be treated prior to starting medication. Liver and kidney monitoring are recommended for most DMARD therapy because it can cause elevation of the liver enzymes and can also affect kidney function.

X-ray, ultrasound, or both of the hands, wrists, and feet can be useful in establishing a baseline for joint evaluation, and assessing the joints for erosions and synovitis. Joint damage may occur within the first 6 to 12 months of diagnosis and should be followed as indicated. Plain x-ray is the most common radiographic study used to track disease progression as it is inexpensive, reliable, and reproducible (Mahmood et al., 2019). MRI can also be useful to detect small erosions that may not be visible on x-ray or ultrasound.

Medical Management

The goal of treatment at all phases of the RA disease process is to decrease joint pain and swelling, achieve clinical remission, decrease the likelihood of joint deformity, and minimize disability. Initial treatment delays have been implicated in greater long-term joint deformity. Aggressive and early treatment

regimens are warranted. The use of a targeted pharmacologic treatment strategy is recommended to decrease RA disease activity (Singh et al., 2016).

Early Rheumatoid Arthritis

Once the diagnosis of RA is made, treatment should begin with either a nonbiologic or biologic DMARD. The goal of using DMARD therapy is preventing inflammation and joint damage.

Recommended treatment guidelines include beginning with the nonbiologic DMARDs (methotrexate is the preferred agent but leflunomide, sulfasalazine, or hydroxychloroquine are also used) biologics, or tofacitinib within 3 months of disease onset. Care should be used with each of these medications by performing routine blood testing for liver and kidney function, along with monitoring the CBC for anemia. Dosage may need to be modified for patients with renal impairment (Comerford & Durkin, 2020).

Another treatment approach for RA is the use of biologic DMARDs. These agents have been specifically engineered to target the most prominent proinflammatory mediators in RA—TNF- α , B cells, T cells, IL-1, and IL-6 (see Table 34-2). Biologic DMARDs are the first targeted therapy for RA. Clinical evidence suggests that biologic DMARDs work more quickly and show a greater delay in radiologic disease progression when compared to nonbiologic DMARDs. The biologic DMARDs are more expensive and have fewer years of usage with the RA population. Therefore, they tend to be reserved for patients with persistent moderate to severe RA who have not responded adequately to synthetic DMARDs (Singh et al., 2016).

After initiating treatment with a DMARD, patients generally report a beneficial effect within 6 weeks and tolerate the medication relatively well. However, some patients may take longer to see improvement. Corticosteroids are recommended as a “bridge” in the early treatment but are not recommended for long-term therapy due to side effects (Singh et al., 2016).

A newer class of drugs, the Janus Kinase (JAK) inhibitors, bind to the active JAK enzyme sites, inhibiting autophosphorylation and thus inhibiting cytokine production and decreasing the immune response (Mogul et al., 2019). JAK inhibitors are used in combination with methotrexate or other nonbiologic DMARDs. They may also be used as monotherapy (Mogul et al., 2019; Singh et al., 2016).

NSAIDs and specifically the cyclo-oxygenase 2 (COX-2) enzyme blockers are used for pain and inflammation relief. NSAIDs, such as ibuprofen and naproxen, are commonly prescribed because of their low cost and analgesic properties. They must be used with caution, however, in long-term chronic diseases because of the possibility of gastric ulcers. Several COX-2 enzyme blockers have been approved for treatment of RA. Cyclo-oxygenase is an enzyme that is involved in the inflammatory process. COX-2 medications block the enzyme involved in inflammation (COX-2) while leaving intact the

enzyme involved in protecting the stomach lining (COX-1). As a result, COX-2 enzyme blockers are less likely to cause gastric irritation and ulceration than other NSAIDs; however, they are associated with increased risk of cardiovascular disease and must be used with caution (Comerford & Durkin, 2020). The nurse should be aware that NSAIDs do not prevent erosions or alter disease progression and, consequently, are medications useful only for symptom relief (Singh et al., 2016).

Additional analgesia may be prescribed for periods of extreme pain. Opioid analgesic agents are avoided because of the potential for continuing need for pain relief. Nonpharmacologic pain management techniques (e.g., relaxation techniques, heat and cold applications) are taught.

Established Rheumatoid Arthritis

In patients with established RA, a formal program with occupational and physical therapy is prescribed to educate the patient about principles of pacing activities, work simplification, range of motion, and muscle-strengthening exercises. The patient is encouraged to participate actively in the management program. The medication program is reevaluated periodically, and appropriate changes are made if disease progression is occurring despite pharmacologic treatment. Additional agents may be added to enhance the disease-modifying effect of methotrexate. Combination therapy using one nonbiologic DMARD and one biologic DMARD is common (Singh et al., 2016).

For more established RA, reconstructive surgery and corticosteroids are often used. Reconstructive surgery is indicated when pain cannot be relieved by conservative measures and the threat of loss of independence is eminent. Surgical procedures include synovectomy (excision of the synovial membrane), tenorrhaphy (suturing of a tendon), arthrodesis (surgical fusion of the joint), surgical repair, and replacement of the joint. Surgery is not performed during exacerbations.

Systemic corticosteroids are used when the patient has unremitting inflammation and pain or needs a “bridging” medication while waiting for the slower DMARDs (e.g., methotrexate) to begin taking effect. Low-dose corticosteroid therapy is prescribed for the shortest time necessary to minimize side effects (Singh et al., 2016). Single large joints that are severely inflamed and fail to respond promptly to the measures outlined previously may be treated by local injection of a corticosteroid.

Topical analgesic agents such as capsaicin and methylsalicylate are often prescribed. Topical diclofenac sodium gel may help with joint pain in the hands and knees (Comerford & Durkin, 2020).

For most patients with RA, the emotional and possible financial burden of the disease can lead to depressive symptoms and sleep deprivation. The patient may require the short-term use of low-dose antidepressant medications, such as amitriptyline, paroxetine, or sertraline, to reestablish an adequate sleep

pattern and manage depressive symptoms. Patients may benefit from referrals for talk therapy or group support.



Obesity Considerations

The prevalence of obesity is increasing in the general population and in patients with RA. One large study reported that the prevalence of obesity, diagnosed with a body mass index (BMI) of greater than or equal to 30 kg/m², was approximately 20% at diagnosis and increased each year (Nikiphorou, Norton, Young, et al., 2018). Furthermore, the presence of obesity adversely affected disease progression, function, and quality of life in patients with RA followed for 10 to 25 years (Nikiphorou et al., 2018). Management of obesity in the patient with RA is essential.

Certain medications (i.e., oral corticosteroids) used in RA treatment stimulate the appetite and, when combined with decreased activity, may lead to weight gain. In fact, one study reported that approximately half the increase in BMI within the first year of diagnosis with RA could be attributed to steroid use (Nikiphorou et al., 2018).

A dietitian can counsel the patient about better food choices. Food selection should include the five major groups (grains, vegetables, fruits, dairy, and protein), with emphasis on foods high in vitamins, protein, and iron for tissue building and repair. Patients who are overweight or have obesity need to be counseled about eating a healthy, calorie-restricted diet.

Nursing Management

Nursing care of the patient with RA follows the basic plan of care presented earlier (see [Chart 34-3](#)). The most common issues for the patient with RA include pain, sleep disturbance, fatigue, altered mood, and limited mobility. The patient with newly diagnosed RA needs information about the disease to make daily self-management decisions and cope with having a chronic disease. Consultation with a dietitian for assessment and assistance with appropriate food choices may be helpful.

Monitoring and Managing Potential Complications

Patients commonly have comorbid conditions such as cardiovascular disease that can lead to complications. It has been estimated that the primary cause of death for up to 40% of patients diagnosed with RA is cardiovascular disease. The cause of cardiovascular disease in these patients is thought to be due to elevated lipid values, chronic inflammation, dysfunction of the endothelium, and/or abnormal homocysteine levels (Norris, 2019).

Medications used for treating RA may cause serious and adverse effects. The primary provider bases the prescribed medication regimen on clinical findings and past medical history, and then, with the help of the nurse, monitors for side effects using periodic clinical assessments and laboratory testing. The nurse, who can be available for consultation between visits with the primary provider, works to help the patient recognize and deal with these side effects (see [Table 34-2](#)). Medication may need to be stopped or the dose reduced. If the patient experiences an increase in symptoms while the complication is being resolved or a new medication is being initiated, the nurse's counseling regarding symptom management may relieve potential anxiety and distress.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

Patient education is an essential aspect in nursing care of the patient with RA to enable the patient to maintain as much independence as possible, to take medications accurately and safely, and to use adaptive devices correctly (Oh et al., 2018). Patient education focuses on self-management related to the disorder, the therapeutic regimen prescribed to treat it, and the potential side effects of medications. Patients undergoing surgery need education as well. The nurse works with the patient and family on strategies to maintain independence, function, and safety in the home (see [Chart 34-4](#)).

The patient and family are encouraged to verbalize their concerns and ask questions. Because RA commonly affects young women, major concerns may be related to the effects of the disease on childbearing potential, caring for family, or work responsibilities. The patient with a chronic illness may seek a “cure” or have questions about alternative therapies. Research indicates that acupuncture is safe and may be beneficial for patients with RA (Chou & Chu, 2018). There is not enough evidence of the effectiveness of other complementary, alternative, and integrative health therapies, and more rigorous research is needed (Chou & Chu, 2018; Katz-Talmor, Katz, Porta-Katz, et al., 2018).

The nurse educates patients using topical analgesic agents to apply sparingly, avoid areas of open skin, and avoid contact with eyes and mucous membranes. Patients should also wash their hands carefully after application and assess for local skin irritation. Pain, fatigue, and depressive symptoms can interfere with the patient’s ability to learn and should be addressed before the education is initiated. Various educational strategies may then be used, depending on the patient’s previous knowledge base, interest level, degree of comfort, social or cultural influences, and readiness to learn. The nurse

educates the patient about basic disease management and necessary adaptations in lifestyle. Some types of aerobic exercise and strength training should be discussed (Kapale et al., 2017). Because suppression of inflammation and autoimmune responses require the use of anti-inflammatory, disease-modifying antirheumatic, and immunosuppressive agents, the patient is taught about prescribed medications, including type, dosage, rationale, potential side effects, self-administration, and required monitoring procedures. If hospitalized, the patient is encouraged to practice new self-management skills with support from caregivers and significant others. The nurse then reinforces disease management skills during each patient contact. Barriers are assessed, and measures are taken to promote adherence to medications and the treatment program.

Chart 34-4



HOME CARE CHECKLIST

The Patient with Rheumatoid Arthritis

At the completion of education, the patient and/or caregiver will be able to:

State the impact of rheumatoid arthritis on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.

- State changes in lifestyle (e.g., diet, activity, rest) necessary to maintain health.
- State the name, dose, side effects, frequency, and schedule for all medications.
- Demonstrate accurate and safe self-administration of medications.
- Describe and demonstrate the use of pain management techniques.
- Demonstrate ability to perform ADLs independently or with assistive devices/adaptive equipment.
- Verbalize ways to cope with stress successfully, plans for regular, safe exercise, and rationale for obtaining adequate rest. Demonstrate a relaxation technique.
- Verbalize a dietary plan that includes maintaining or losing weight while maximizing vitamins, protein, and iron for tissue building and repair
- State how to reach primary provider with questions or complications
- State time and date of follow-up appointments, testing
- Identify the need for health promotion, disease prevention, and screening activities
- Identify community resources for peer and caregiver/family information and support:
 - Identify sources of support (e.g., friends, relatives, faith community)
 - Identify the contact details for support services for patients and their caregivers/families

ADLs, activities of daily living; IADLs, instrumental activities of daily living.

Continuing and Transitional Care

Depending on the severity of the disease and the patient's resources and supports, referral for home care may be warranted. For example, the patient who is an older adult or frail, has RA that limits function significantly, and lives alone may need referral for home care.

The impact of RA on everyday life is not always evident when the patient is seen in the hospital or in an ambulatory care setting. The increased frequency with which nurses see patients in the home provides opportunities for recognizing problems and implementing interventions aimed at improving the quality of life of patients with RA.

During home visits, the nurse has the opportunity to assess the home environment and its adequacy for patient safety and management of the disorder. Adherence to the treatment program can be more easily monitored in the home setting, where physical and social barriers to adherence are more readily identified. For example, a patient who also has diabetes and requires insulin may be unable to fill the syringe accurately or unable to administer the insulin because of impaired joint mobility. Appropriate adaptive equipment needed for increased independence is often identified more readily when the nurse sees how the patient functions in the home. Any barriers to adherence are identified, and appropriate referrals are made.

For patients at risk for impaired skin integrity, the home health nurse can closely monitor skin status and also educate, provide, or supervise the patient and family in preventive skin care measures. The nurse also assesses the patient's need for assistance in the home and supervises home health aides, who may meet many of the needs of the patient with RA. Referrals to physical and occupational therapists may be made as problems are identified and limitations increase. A home health nurse may visit the home to make sure that the patient can function as independently as possible despite mobility problems and can safely manage treatments, including pharmacotherapy. The patient and family should be informed about support services such as Meals on Wheels and local Arthritis Foundation chapters.

Because many of the medications used to suppress inflammation are injectable, the nurse may administer the medication to the patient or educate about self-injection. These frequent contacts allow the nurse to reinforce other disease management techniques.

The nurse also assesses the patient's physical and psychological status, adequacy of symptom management, and adherence to the management plan. Patients should know which type of rheumatic disease they have, not just that they have "arthritis" or "arthritis of the knee." The importance of attending follow-up appointments is emphasized to the patient and family, and they should be reminded about the importance of participating in other health promotion activities and health screening.

Systemic Lupus Erythematosus



SLE is an inflammatory, autoimmune disorder that affects nearly every organ in the body. The overall incidence of SLE is estimated to be 1.8 to 7.6 per 100,000 people (Centers for Disease Control and Prevention [CDC], 2018). It occurs 4 to 12 times more frequently in women than in men and occurs more often in African Americans, Hispanics/Latino Americans, Asians, and American Indians/Alaska Natives than among White Americans (CDC, 2018). In addition to SLE, other forms of adult lupus exist, including subacute

cutaneous or discoid lupus erythematosus, and drug-induced lupus (Aringer, Costenbader, Daikh, et al., 2019).

Pathophysiology

While the exact cause is not known, SLE starts with the body's immune system inaccurately recognizing one or more components of the cell's nucleus as foreign, seeing it as an antigen. The immune system starts to develop antibodies to the nuclear antigen. In particular, B cells begin to overproduce antibodies with the help of multiple cytokines such as B-lymphocyte stimulator (BLyS), which is overexpressed in SLE. The antibodies and antigens form antigen–antibody complexes and have the propensity to get trapped in the capillaries of visceral structures. The antibodies also act to destroy host cells. It is thought that those two mechanisms are responsible for the majority of the clinical manifestations of this disease process. The immunoregulatory disturbance is thought to be brought about by some combination of four distinct factors: genetic, immunologic, hormonal, and environmental (Norris, 2019).

Research into the genetic origins of SLE has thus far revealed that multiple genes are likely implicated in the development of SLE (Norris, 2019). The large majority of SLE cases, however, remain sporadic and unrelated to family medical history.

Given the high number of women with SLE compared with men, it is hypothesized that female sex hormones (estrogen) play a role in the predisposition to SLE. Estrogen may contribute to the body's response of overreacting to the body's own tissues.

Although genetics and hormones likely play a role in the predisposition of SLE, it is hypothesized that exogenous or environmental triggers are also implicated in the onset of the disease process. These triggers may include cigarette smoke, ultraviolet rays, exposure from sunlight and fluorescent light bulbs, medications (hydralazine, minocycline, or procainamide), viral infections, emotional stress, stress on the body (e.g., surgery, pregnancy), and silica dust exposure in the occupational setting (Norris, 2019).

Clinical Manifestations

SLE is an autoimmune, systemic disease that can affect any body system ([Fig. 34-2](#)). The disease process involves chronic states where symptoms are minimal or absent and acute flares where symptoms and lab results are elevated. Symptoms most often include fever, fatigue, skin rashes, as well as joint pain and swelling (Aringer et al., 2019; CDC, 2018). The mucocutaneous, musculoskeletal, renal, nervous, cardiovascular, and respiratory systems are

most commonly involved. Less commonly affected are the gastrointestinal tract and liver as well as the ocular system.

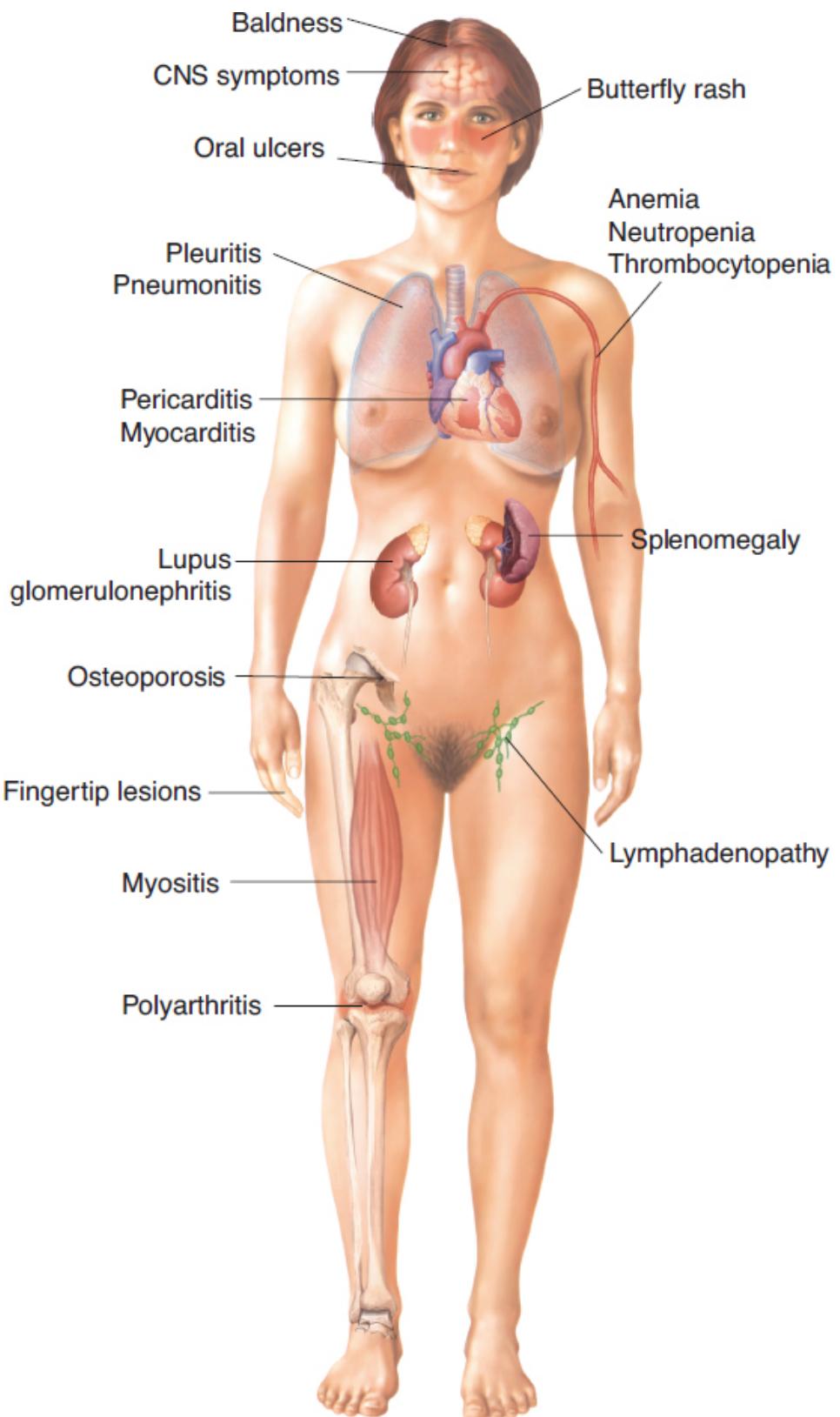


Figure 34-2 • Clinical manifestations of SLE. Reprinted with permission from Norris, T. L. (2019). *Porth's pathophysiology*:

Concepts of altered health states (10th ed., Fig. 50.5). Philadelphia, PA: Wolters Kluwer.

Some type of cutaneous system manifestation is experienced in up to 85% of patients with SLE (Norris, 2019). Several skin manifestations may occur in patients with SLE, including subacute cutaneous lupus erythematosus, which involves papulosquamous or annular polycyclic lesions, and a discoid rash, which is a chronic rash with erythematous papules or plaques and scaling and can cause scarring and pigmentation changes (Aringer et al., 2019). In some cases, the only skin involvement may be a discoid rash. In some patients with SLE, the initial skin involvement is the precursor to more systemic involvement. The lesions often worsen during **exacerbations** (flares) of the systemic disease and possibly are provoked by sunlight or artificial ultraviolet light (Norris, 2019). Oral ulcers, which may accompany skin lesions, may involve the buccal mucosa or the hard palate, occur in crops, and are often associated with exacerbations. Other cutaneous manifestations include splinter hemorrhages, alopecia, and Raynaud's phenomenon.

Joint pain and swelling occur in more than 90% of patients with SLE (Norris, 2019). Joint swelling, tenderness, and pain on movement are also common. Frequently, these are accompanied by morning stiffness.

The cardiac system is also commonly affected in SLE. Pericarditis is the most common cardiac manifestation (Norris, 2019). Patients may present with substernal chest pain that is aggravated by movement or inspiration. Symptoms can be acute and severe or last for weeks at a time. Other cardiac symptoms may involve myocarditis, hypertension, cardiac arrhythmias, and valvular incompetence.

Nephritis as a result of SLE, also referred to as lupus nephritis, occurs due to a buildup of antibodies and immune complexes that cause damage to the nephrons. Serum creatinine levels and urinalysis are used in screening for renal involvement. Early detection allows for prompt treatment so that renal damage can be prevented. Renal involvement may lead to hypertension, which also requires careful monitoring and management (see [Chapter 27](#)).

Central nervous system involvement is widespread, encompassing the entire range of neurologic disease. The varied and frequent neuropsychiatric presentations of SLE are now widely recognized and include psychosis, cognitive impairment, seizures, peripheral and cranial neuropathies, transverse myelitis, and strokes. These are generally demonstrated by subtle changes in behavior patterns or cognitive ability.

Assessment and Diagnostic Findings

Diagnosis of SLE is based on a complete history, physical examination, and blood tests. In addition to the general assessment performed for any patient

with a rheumatic disease, assessment for known or suspected SLE has special features. The skin is inspected for erythematous rashes. Cutaneous erythematous plaques with an adherent scale may be observed on the scalp, face, or neck. Areas of hyperpigmentation or depigmentation may be noted, depending on the phase and type of disease. The patient should be questioned about skin changes (because these may be transitory) and specifically about sensitivity to sunlight or artificial ultraviolet light. The scalp should be inspected for alopecia and the mouth and throat for ulcerations reflecting gastrointestinal involvement.

Cardiovascular assessment includes auscultation for pericardial friction rub, possibly associated with myocarditis and accompanying pleural effusions. The pleural effusions and infiltrations, which reflect respiratory insufficiency, are demonstrated by abnormal lung sounds. Papular, erythematous, and purpuric lesions developing on the fingertips, elbows, toes, and extensor surfaces of the forearms or lateral sides of the hand that may become necrotic suggest vascular involvement.

Joint swelling, tenderness, warmth, pain on movement, stiffness, and edema may be detected on physical examination. The joint involvement is often symmetric and similar to that found in RA.

The neurologic assessment is directed at identifying and describing any central nervous system changes. The patient and family members are asked about any behavioral changes, including manifestations of neurosis or psychosis. Signs of depression are noted, as are reports of seizures, chorea, or other central nervous system manifestations.

The antinuclear antibody (ANA) is positive in more than 95% of patients with SLE, indicating exceptional specificity (Aringer et al., 2019). Other laboratory tests include anti-DNA (antibody that develops against the patient's own DNA), anti-ds DNA (antibody against DNA that is highly specific to SLE, which helps differentiate it from drug-induced lupus), and anti-Sm (antibody against Sm, which is a specific protein found in the nucleus). Other blood work includes the CBC, which may reveal anemia, thrombocytopenia, leukocytosis, or leukopenia.

Medical Management

SLE can be life-threatening, but advances in its treatment have led to improved survival and reduced morbidity. Acute disease requires interventions directed at controlling increased disease activity or exacerbations that can involve any organ system. Disease activity is a composite of clinical and laboratory features that reflect active inflammation secondary to SLE. Management of the more chronic condition involves periodic monitoring and recognition of meaningful clinical changes requiring adjustments in therapy.

The goals of treatment include preventing progressive loss of organ function, reducing the likelihood of acute disease, minimizing disease-related disability, and preventing complications from therapy. Management of SLE involves regular monitoring to assess disease activity and therapeutic effectiveness.

Pharmacologic Therapy

The mainstay of SLE treatment is based on pain management and nonspecific immunosuppression. Therapy includes monoclonal antibodies, corticosteroids, antimalarial agents, NSAIDs, and immunosuppressive agents. Each of these medications has potentially serious side effects, including organ damage.

Belimumab is a monoclonal antibody that specifically recognizes and binds to BLyS. BLyS acts to stimulate B cells to produce antibodies against the body's own nuclei, which is an integral part of the disease process in SLE. Belimumab acts to render BLyS inactive, preventing it from binding to B-cell surfaces and stimulating B-cell activity. This action then halts the production of unnecessary antibodies and decreases disease activity in SLE. Live vaccines are contraindicated for 30 days before taking this medication (Comerford & Durkin, 2020). Rituximab is an additional monoclonal antibody used in the treatment of SLE for its immune modulating effects (MacIsaac, Siddiqui, Jamula, et al., 2018).

Corticosteroids are another medication used topically for cutaneous manifestations, in low oral doses for minor disease activity, and in high doses for major disease activity. Intravenous (IV) administration of corticosteroids is an alternative to traditional high-dose oral administration. One of the most important risk factors associated with the use of corticosteroids in SLE is osteoporosis and fractures (Comerford & Durkin, 2020).

An antimalarial medication, hydroxychloroquine, is effective for managing cutaneous, musculoskeletal, and mild systemic features of SLE (Comerford & Durkin, 2020). The NSAIDs used for minor clinical manifestations are often used in conjunction with corticosteroids in an effort to minimize corticosteroid requirements.

Immunosuppressive agents (alkylating agents and purine analogues) are used because of their effect on overall immune function. These medications are generally reserved for patients who have serious forms of SLE that have not responded to conservative therapies. Examples include cyclophosphamide, azathioprine, mycophenolic acid, and methotrexate, which are contraindicated in pregnancy and have been used most frequently in SLE nephritis (Comerford & Durkin, 2020).

Nursing Management

Nursing care of the patient with SLE is based on the fundamental plan presented earlier in the chapter (see [Chart 34-3](#)). The most common nursing diagnoses include fatigue, impaired skin integrity, body image disturbance, and lack of knowledge for self-management decisions. The disease or its treatment may produce dramatic changes in appearance and considerable distress for the patient. The changes and the unpredictable course of SLE necessitate expert assessment skills and nursing care with sensitivity to the psychological reactions of the patient. The patient may benefit from participation in support groups, which can provide disease information, daily management tips, and social support. Because sun and ultraviolet light exposure can increase disease activity or cause an exacerbation, patients should be instructed to avoid exposure or to protect themselves with sunscreen and appropriate clothing.

Because of the increased risk of involvement of multiple organ systems, patients should understand the need for routine periodic screenings as well as health promotion activities. A dietary consultation may be indicated to ensure that the patient is knowledgeable about dietary recommendations, given the increased risk of cardiovascular disease, including hypertension and atherosclerosis.

In the healthy population, smoking and using electronic nicotine delivery systems (ENDS) including e-cigarettes, e-pens, e-pipes, e-hookah, and e-cigars clearly poses health risks. Smoking increases the incidence of respiratory infections, lung cancer, risk of coronary artery disease; increases blood pressure, which can worsen kidney function; inhibits liver function (which can also inhibit treatment medications from working appropriately, such as hydroxychloroquine); increases the risk for skin diseases; and increases the risk for osteoporosis. The health risks of ENDS use is under investigation. Patients diagnosed with SLE are at even higher risk of developing lung cancer and other rare cancers. Therefore, smoking cessation programs should be offered to all patients who report smoking habits (Montes, Mocarzel, Lanzieri, et al., 2016).

The nurse educates the patient about the importance of continuing prescribed medications and addresses the changes and potential side effects that are likely to occur with their use. The patient is reminded of the importance of monitoring because of the increased risk of systemic involvement, including renal and cardiovascular effects.

Because of the immunosuppression associated with systemic corticosteroid usage, the nurse must watch for signs and symptoms of infection, especially with patients who are acutely ill.

The nurse should also screen the patient for osteoporosis, because long-term use of corticosteroids increases the incidence of osteoporosis. Patients should have a bone mineral density test performed at diagnosis and prior to beginning steroid use to determine a baseline status and then every 2 years thereafter. Educating the patient regarding calcium and vitamin D supplementation daily

is encouraged, along with the benefits of weight-bearing activities to support bone health.

Primary Sjögren's Syndrome

Primary Sjögren's syndrome is a rare systemic autoimmune disease that predominantly affects middle-aged women (Cornec, Devauchelle-Pensec, Mariette, et al., 2017).

Sjögren's syndrome often manifests in conjunction with other autoimmune diseases, most commonly autoimmune thyroid disease, RA, and SLE (MacIsaac et al., 2018; Molano-Gonzalez et al., 2019).

Pathophysiology

Primary Sjögren's is a multisystem disease characterized by lymphocytic infiltration leading to failure of the lacrimal and salivary glands (Cui, Li, Yin, et al., 2018).

Clinical Manifestations

The most common symptoms involve keratoconjunctivitis sicca (dry eyes), xerostomia (dry mouth), pain, and fatigue (Cornec et al., 2017; Cui et al., 2018). Some patients will complain that their eyes feel "gritty," as if there is sand present. The patient's eyes will exhibit increased redness and lack of tearing. The mouth will have dry and sticky mucous membranes. The reduced saliva production may lead to difficulty swallowing.

Sjögren's syndrome can also exhibit symptoms in many other organ systems. Lesions may ulcerate and can be painful. Optic neuritis, trigeminal neuralgia, and sensory neuropathy may be present, with symptoms such as burning pain in the extremities, numbness, vertigo, arthralgia, and/or myalgia. Raynaud's phenomenon, which involves blood vessel spasms leading to decreased circulation to the toes, fingers, nose, and ears, may be reported. Patients experience symptoms of pain, fatigue, depression, and anxiety (Cui et al., 2018). Sleep disturbances are frequently reported and may be related to pain, fatigue, and depressive symptoms (Hackett, Gotts, Ellis, et al., 2017).

Assessment and Diagnostic Findings

The classification criteria for diagnosis of Sjögren's syndrome identify six distinct indices (Klippenstein, Stone, Crofford, et al., 2008):

- Ocular symptoms such as chronic dry eye (Keratoconjunctivitis Sicca).

- Positive ocular tests (evaluating tear production, corneal, and conjunctival damage). Ocular testing may include Schmerer tear test or Rose Bengal tests.
- Oral symptoms, dry mouth.
- Histopathology evaluation (of salivary glands). This will help differentiate the cause of dry mouth from other causes, such as infection, malignancy, stones, and sarcoidosis.
- Salivary gland involvement.

Laboratory tests include autoantibodies to ribonucleoprotein particles (Ro[SS-A] and/or La[SS-B]), which act as antigens in this disease process. Other laboratory indices are also useful in diagnosing Sjögren's syndrome. Rheumatoid factor is present in 50% to 70% of patients. ANA, circulating DNA (cDNA), anti-CCP, ACPA, and anticentromere antibody (ACA) are all potentially present in Sjögren's syndrome and in some cases may act as markers for disease activity (Molano-Gonzalez et al., 2019).

For vasculitis manifestations, a skin biopsy can yield useful information such as leukocytoclastic vasculitis found on histologic examination. If neurologic symptoms are present, nerve conduction studies, MRI, electroencephalograms, and cerebrospinal fluid testing may be used to aid in diagnosis and treatment planning.

Medical Management

There is no cure for Sjögren's syndrome, and treatment is aimed at symptom management and improving quality of life (Cornec et al., 2017; Cui et al., 2018; Hackett et al., 2017). Artificial tears, drops such as pilocarpine, and ocular ointments such as topical cyclosporine are used for dry eyes. Tears normally drain through the lacrimal puncta to the nose, which can render artificial tears ineffective. Therefore, punctum plugs may be a useful management tool. One systematic review reported that pilocarpine is highly effective in decreasing symptoms of dry mouth by increasing salivary flow (Hamad, Lodi, Porter, et al., 2018). Biotene oral rinse may also be useful for some patients. Additional suggestions include eating small frequent meals; omitting spicy, salty, and irritating food; and avoiding smoking (including ENDS), excessive alcohol use, and drugs with anticholinergic side effects. There is some evidence for the effectiveness of rituximab and interferone (Cornec et al., 2017; Hamad et al., 2018; MacIsaac et al., 2018). Patients need to be screened for depression and sleep disturbances, then appropriate referrals made when these comorbid conditions are present (Cui et al., 2018; Hackett et al., 2017).

Nursing Management

Nursing care is based on the fundamental plan of nursing care presented earlier (see [Chart 34-3](#)). The primary issues for the patient with Sjögren's syndrome are pain, fatigue, and inadequate knowledge of self-management techniques. The nurse is in the unique position to educate and reinforce the treatment regimen with the patient, especially involving the ocular treatments to avoid eye infections secondary to the dry eyes. The high prevalence of pain, fatigue, and depressive symptoms may interfere with the patient's ability to learn and engage in self-management techniques and should be addressed (Cui et al., 2018).

Scleroderma

Scleroderma is a rare autoimmune disease affecting the connective tissue of the skin, blood vessel walls, and internal organs. There are two general types: localized (affecting only the cutaneous system) or diffuse (routinely referred to as systemic sclerosis and affecting multiple organ systems). Similar to other autoimmune diseases, women are affected four times more than men, and the onset occurs typically between the ages of 25 and 50 years (Norris, 2019). Scleroderma has a variable course with remissions and exacerbations.

Pathophysiology

The pathogenesis is poorly understood. Scleroderma commonly begins with skin involvement. Mononuclear cells cluster on the skin and stimulate lymphokines to stimulate procollagen. Insoluble collagen is formed and accumulates excessively in the tissues (Norris, 2019). Initially, the inflammatory response causes edema, with a resulting taut, smooth, and shiny skin appearance. The skin then undergoes fibrotic changes, leading to loss of elasticity and movement. Eventually, the tissue degenerates and becomes nonfunctional. This chain of events, from inflammation to degeneration, also occurs in blood vessels, major organs, and body systems (Norris, 2019).

Clinical Manifestations

The skin and subcutaneous tissues become increasingly hard and rigid due to excess collagen and cannot be pinched up from the underlying structures. Wrinkles and lines are obliterated. The skin is dry because sweat secretion over the involved region is suppressed. The extremities stiffen and lose mobility. The condition spreads slowly; for years, these changes may remain localized in the hands and the feet. The face appears masklike, immobile, and expressionless, and the mouth becomes rigid; referred to as "stone facies" (Norris, 2019).

The changes within the body, although not visible directly, are vastly more important than the visible changes. The esophagus hardens, interfering with swallowing. The lungs become scarred, impeding respiration. Digestive disturbances occur because of sclerosing (hardening) of the intestinal mucosa. Vascular involvement of the kidneys leads to malignant hypertension and renal insufficiency. Cardiac disorders include pericarditis, heart block, and myocardial fibrosis (Norris, 2019).

The patient may manifest a variety of symptoms referred to as the CREST syndrome. *CREST* stands for *calcinosis* (calcium deposits in the tissues), *Raynaud's phenomenon*, *esophageal dysmobility*, *sclerodactyly* (scleroderma of the digits), and *telangiectasia* (capillary dilation that forms a vascular lesion) (Norris, 2019).

Assessment and Diagnostic Findings

Assessment focuses on the sclerotic changes in the skin, contractures in the fingers, and color changes or lesions in the fingertips. Assessment of systemic involvement requires a systems review with special attention to gastrointestinal, pulmonary, renal, and cardiac symptoms. Limitations in mobility and self-care activities should be assessed, along with the impact the disease has had (or will have) on body image.

There is no one conclusive diagnostic test used to diagnose scleroderma. Generally, the patient is diagnosed with the CREST type of scleroderma if they have four of the five symptoms in the syndrome (Norris, 2019).

Medical Management

Treatment of scleroderma is mainly symptomatic and supportive. No medication regimen is effective in modifying the disease process in scleroderma, but various medications are used to treat organ system involvement. The use of angiotensin-converting enzyme inhibitors when there is kidney involvement has led to a substantial decrease in mortality from hypertensive kidney disease (Norris, 2019).

All patients require counseling, during which realistic individual goals may be determined. Support measures include strategies to decrease pain and limit disability. A moderate exercise program is encouraged to prevent joint contractures. Patients are advised to avoid extreme temperatures and to use lotion to minimize skin dryness.

Nursing Management

Nursing care of the patient with scleroderma is based on the fundamental plan of nursing care presented earlier (see [Chart 34-3](#)). The primary nursing

diagnoses are impaired skin integrity; self care deficits; impaired nutritional status; and disturbed body image. The patient with advanced disease may also have impaired gas exchange, impaired cardiac output, impaired swallowing, and constipation.

Providing meticulous skin care and preventing the effects of Raynaud's phenomenon are major nursing challenges. See [Chapter 26](#) for further discussion of Raynaud's phenomenon.

Polymyositis

Polymyositis is a group of diseases that are termed *idiopathic inflammatory myopathies* (Klippel et al., 2008). They are rare chronic conditions, with an incidence estimated at 2 cases per 10,000 adults per year. Polymyositis is most commonly seen in women versus men (2:1) and usually seen between 40 and 50 years of age (Miller & Vleugels, 2019).

Pathophysiology

Polymyositis is classified as autoimmune because autoantibodies are present. However, these antibodies do not cause damage to muscle cells, indicating only an indirect role in tissue damage. The pathogenesis is multifactorial, including cellular and humoral immune mechanisms (Norris, 2019).

Clinical Manifestations

The onset may be very slow and insidious, with symptoms gradually worsening over weeks to months. Proximal muscle weakness is typically the first symptom. Muscle weakness is usually symmetric and diffuse. Common complaints include having difficulty rising from a chair, climbing steps, or holding up the head. Myalgia and muscle tenderness occur in 25% to 50% of patients. Dermatomyositis, a related condition, is most commonly identified by an erythematous smooth or scaly lesion found over the joint surface, which often occurs prior to symptoms of weakness in 50% to 60% of patients (Miller & Vleugels, 2019).

Assessment and Diagnostic Findings

A complete history and physical examination help exclude other muscle-related disorders. As with other diffuse connective tissue disorders, no single test confirms polymyositis. An electromyogram is performed to rule out degenerative muscle disease. A muscle biopsy may reveal inflammatory infiltrate in the tissue. Serum studies indicate increased muscle enzyme activity.

Medical Management

Corticosteroid therapy is the mainstay of medical management (Norris, 2019). IV immune globulin, plasmapheresis, lymphapheresis, and total-body irradiation have been used if there is no response to corticosteroids. The goal is to control inflammation and prevent long-term damage to muscles, joints, and internal organs (Norris, 2019). The antimalarial agent hydroxychloroquine may be effective for skin rashes. Physical therapy is initiated slowly, with range-of-motion exercises to maintain joint mobility, followed by gradual strengthening exercises (Klippen et al., 2008).

Nursing Management

Nursing care is based on the fundamental plan of nursing care presented earlier (see [Chart 34-3](#)). The primary nursing diagnoses for the patient with polymyositis are impaired mobility, fatigue, self care deficit, and lack of knowledge of self-management techniques.

Patients with polymyositis may have symptoms similar to those of other inflammatory diseases. However, proximal muscle weakness is characteristic, making activities such as combing the hair, reaching overhead, and using stairs difficult. Therefore, the use of assistive devices may be recommended, and referral to occupational or physical therapy may be warranted.

Polymyalgia Rheumatica and Giant Cell Arteritis

PMR involves stiffness of muscles and pain in the neck, shoulder, and pelvic girdle. GCA is a form of vasculitis affecting the medium-sized and large arteries of the body (Klippen et al., 2008). GCA is also sometimes referred to as temporal arteritis (Hill, Black, Nossent, et al., 2017). PMR and GCA represent a spectrum of one disease. Both primarily affect individuals older than 50 years and are associated with the same HLA haplotype genetic markers. PMR and GCA occur predominately in Caucasians and often in first-degree relatives. PMR has an annual incidence rate of 52 cases per 100,000 people older than 50 years. GCA varies by geographic location and has the highest incidence in Scandinavian countries. PMR is two to three times more common than GCA (Klippen et al., 2008).

Pathophysiology

The underlying mechanism of action involved with PMR and GCA is unknown. It is clear, however, that the immune system is abnormally activated in both disease processes with increases in circulating monocytes that produce IL-1 and IL-6. These circulating monocytes make the endothelial linings of

blood vessels more vulnerable to vasculitis (Klippel et al., 2008). Immunoglobulin deposits in the walls of inflamed temporal arteries suggest that an autoimmune process is at work.

Clinical Manifestations

PMR is characterized by severe proximal muscle discomfort with mild joint swelling. Severe aching in the neck, shoulder, and pelvic muscles is common. Stiffness is noticeable most often in the morning and after periods of inactivity. This stiffness can become so severe that patients struggle putting on a coat or combing their hair. Systemic features include low-grade fever, weight loss, malaise, anorexia, and depression. Because PMR usually occurs in people 50 years and older, it may be confused with, or dismissed as, an inevitable consequence of aging.

GCA may cause headaches, changes in vision, and jaw claudication. These symptoms should be evaluated immediately because of the potential for blindness and stroke if left untreated (Hill et al., 2017). PMR and GCA have a self-limited course, lasting several months to several years (Klippel et al., 2008).

Assessment and Diagnostic Findings

Assessment focuses on musculoskeletal tenderness, weakness, and decreased function. Careful attention should be directed toward assessing the head (for changes in vision, headaches, and jaw claudication). An MRI scan may be used in the assessment of extra-articular synovitis in patients with PMR, regardless of symptoms.

Often, diagnosis is difficult because of the lack of specificity of tests. A markedly high ESR is a screening test but is not definitive. The CRP level and platelet count also provide valuable data. In fact, simultaneous elevation in the ESR and CRP has a sensitivity of 98.6% and a specificity of 75.7% in making the diagnosis of GCA when coupled with clinical findings (Seetharaman, 2019). Diagnosis of both GCA and PMR is more likely to be made by eliminating other potential diagnoses. The dramatic and immediate response to treatment with corticosteroids is considered by some to be diagnostic.

In the case of GCA, biopsy of the temporal artery is the definitive diagnostic tool (Seetharaman, 2019). High-resolution MRI is an alternative or adjunct to the traditional temporal artery biopsy.

Medical Management

The treatment for patients with PMR (without GCA) is moderate doses of corticosteroids. Longer durations of corticosteroid treatment are required with

patients who have higher baseline inflammatory markers. Gradual tapering of the corticosteroid treatment should be monitored. NSAIDs are sometimes given in mild disease. The treatment for patients with GCA is rapid initiation of and strict adherence to a regimen of corticosteroids. This is essential to avoid the complication of blindness (Hill et al., 2017). Aspirin is an adjunctive treatment may help reduce the risk of visual loss.

Nursing Management

Nursing care of the patient with PMR is based on the fundamental plan of nursing care presented earlier (see [Chart 34-3](#)). The most common nursing diagnoses are pain and lack of knowledge of medications.

A management concern is that the patient will take the prescribed medication, frequently corticosteroids, until symptoms improve and then discontinue the medication. The decision to discontinue the medication should be based on clinical and laboratory findings and the prescription. Nursing implications are related to helping the patient prevent and monitor adverse effects of medications (e.g., infections, diabetes, gastrointestinal problems, and depression) and adjust to those side effects that cannot be prevented (e.g., increased appetite and altered body image).



Quality and Safety Nursing Alert

The nurse must emphasize to the patient the need for continued adherence to the prescribed medication regimen to avoid complications of GCA, such as blindness and stroke.

The loss of bone mass with corticosteroid use increases the risk of osteoporosis in this already at-risk population. Interventions to promote bone health, such as adequate dietary calcium and vitamin D, measurement of bone mineral density, weight-bearing exercise, smoking and ENDS cessation, and reduction of alcohol consumption if indicated, should be emphasized.

Spondyloarthropathies

The spondyloarthropathies are another category of systemic inflammatory disorders of the skeleton. The spondyloarthropathies include ankylosing spondylitis (AS), reactive arthritis (formerly known as Reiter's syndrome), and psoriatic arthritis. Spondyloarthritis is also associated with inflammatory bowel diseases such as Crohn's disease (regional enteritis) and ulcerative colitis (Norris, 2019).

These rheumatic diseases share several clinical features. The inflammation tends to occur peripherally at the sites of attachment—at tendons, joint capsules, and ligaments. Periosteal inflammation may be present. Many patients have arthritis of the sacroiliac joints. The onset tends to occur during young adulthood, with the disease affecting men more often than women. There is a strong tendency for these conditions to occur in families. Frequently, the HLA-B27 genetic marker is found. In addition, more than one of these conditions can be found simultaneously in the same person or another family member (Norris, 2019).

As with other inflammatory conditions, patients with spondyloarthropathies have an increased risk for cardiovascular disease. These findings may be related to a state of chronic systemic inflammation and an increase in traditional cardiac risk factors, such as lack of exercise due to increased pain (Norris, 2019).

Ankylosing Spondylitis

AS is a chronic inflammatory disease of the spine. It is more prevalent in males than in females and is usually diagnosed in the second or third decade of life. The disease is also more severe in males, and significant systemic involvement is likely (Norris, 2019).

AS affects the cartilaginous joints of the spine and surrounding tissues, making them rigid, decreasing mobility, and leading to kyphosis (a stooped position). This kyphosis can, in turn, lead to decreased stability and balance. Back pain is the characteristic feature. The back pain can be so severe that it may mask symptoms of a cervical fracture, which can lead to neurologic problems if left untreated. Occasionally, the large synovial joints, such as the hips, knees, or shoulders, may be involved (Norris, 2019).

AS also exhibits systemic effects as the heart and lungs become constricted in the chest cavity (Norris, 2019). Another potential complication of AS is the risk of osteoporosis, which appears to be related to the inflammatory process as well as bone turnover and low vitamin D levels. Other complications involve atrioventricular conduction defects, aortic insufficiency, and pulmonary fibrosis. As the disease progresses, ankylosis (i.e., fixation or immobility) of the entire spine may occur, leading to respiratory compromise and further complications.

Reactive Arthritis (Reiter's Syndrome)

The disease process involved in reactive arthritis is called *reactive* because the arthritis occurs after an infection, primarily gastrointestinal or genitourinary (Norris, 2019). It mostly affects young adult males and is characterized

primarily by urethritis, arthritis, and conjunctivitis. Dermatitis and ulcerations of the mouth and penis may also be present. Low back pain is common.

Psoriatic Arthritis

Psoriatic arthritis is an inflammatory arthritis associated with the skin disease psoriasis. Approximately 7% of people with psoriasis develop psoriatic arthritis (Norris, 2019). Psoriasis is the most common autoimmune disease in the United States, affecting 2% to 3% of the population. Psoriatic arthritis onset occurs between 30 and 50 years of age and affects equal numbers of men and women (Dewing, 2015). See [Chapter 56](#) for more information on psoriasis.

Psoriatic arthritis is characterized by synovitis, polyarthritis, and spondylitis. Inflammatory back pain is a common symptom, which is differentiated from other back pain by symptoms of back pain presenting at a young age, pain improving with activity, and pain occurring at night. Radiographic evidence of asymmetrical sacroilitis or spondylitis may also assist with diagnosing psoriatic arthritis (Dewing, 2015). Other sites of pain commonly seen in these patients are the Achilles tendon, plantar fascia, or tibial tuberosity areas. Pain in these areas is common from the inflammation that occurs at the entheses, where tendons and ligaments attach to the bone.

Medical Management

Medical management of spondyloarthropathies focuses on treating pain and maintaining mobility by suppressing inflammation. For the patient with AS, good body positioning and posture are essential so that if ankylosis does occur, the patient is in the most functional position. Maintaining range of motion with regular exercise and a muscle-strengthening program is especially important and has been linked with higher quality of life for patients.

Pharmacologic Management

NSAIDs are the first-line therapy for treating all spondylarthropathies. All chronic conditions (cardiac, renal, and gastrointestinal) should be taken into consideration when prescribing long-term NSAIDs. Methotrexate, sulfasalazine, and leflunomide may also be used; these drugs may help with skin and peripheral joint disease but may not prevent spinal changes. Corticosteroid injections may be used for periodic flares; however, oral and long-term use of steroids is not recommended due to the possibility of psoriatic skin flare when discontinuing use.

Disease remission is now the target for psoriatic arthritis (Mease & Coates, 2018). Anti-TNF medications that have been used effectively include etanercept, infliximab, adalimumab, golimumab, and certolizumabpegol.

Additional agents include apremilast, which is a PDE4 inhibitor, and ustekinumab, an anti-IL12/anti-IL23 agent (Mease & Coates, 2018).

Surgical Management

With advanced AS and subsequent debilitating kyphosis, an osteotomy of the spine can be done. One study showed that an average correction of 45 degrees in the cervical spine was obtained and that quality of life also improved. Surgical management may also include total joint replacement (see [Chapter 36](#)).

Nursing Management

Major nursing interventions in the spondyloarthropathies are related to symptom management and maintenance of optimal functioning. Affected patients are primarily young men. Their major concerns are often related to prognosis and job modification, especially among those who perform physical work. Patients may also express concerns about leisure and recreational activities. Focusing on physical activity and staying active and maintaining good posture will help to prevent chronic changes that may lead to deformities. It is important to address psychological changes, such as depression and emotional stress, that can occur with the diagnosis and chronic nature of the disease. If symptoms are present, the primary provider should assess for emotional stress and treat as appropriate.

Metabolic and Endocrine Diseases Associated with Rheumatic Disorders

Metabolic and endocrine diseases may be associated with rheumatic disorders. These include biochemical abnormalities (amyloidosis and scurvy), endocrine diseases (diabetes and acromegaly), immune deficiency diseases (human immune deficiency virus infection, acquired immune deficiency syndrome), and some inherited disorders (hypermobility syndromes). However, the most common conditions are the crystal-induced arthropathies, in which crystals such as monosodium urate (gout) or calcium pyrophosphate (calcium pyrophosphate dihydrate disease or pseudogout) are deposited within joints and other tissues (Norris, 2019).

Gout

Gout is the most common form of inflammatory arthritis. More than 8.3 million Americans self-report the diagnosis of gout (CDC, 2019). The prevalence is reported to be about 3.9% and appears to be on the rise. Men are

three to four times more likely to be diagnosed with gout than women. The incidence of gout increases with age, body mass index, alcohol consumption, hypertension, and diuretic use (CDC, 2019). Evidence links the consumption of fructose-rich beverages with the risk of gout for both men and women (CDC, 2019). Patients with gout have an increased risk of cardiovascular disease. Comorbid conditions such as hypertension, dyslipidemia, diabetes, osteoarthritis, kidney disease, and depression may be present in patients with gout (Lin, Zhang, & Ma, 2018; Norris, 2019).

Pathophysiology

Gout is caused by hyperuricemia (increased serum uric acid). Uric acid is a by-product of purine metabolism; purines are basic chemical compounds found in high concentrations in meat products. Urate levels are affected by diet, medications, overproduction in the body, and inadequate excretion by the kidneys. Hyperuricemia (serum concentration greater than 6.8 mg/dL) can, but does not always, cause urate crystal deposition. However, as uric acid levels increase, the risk becomes greater. The initial cause for the gout attack occurs when macrophages in the joint space phagocytize urate crystals. Through a series of immunologic steps, interleukin-1 β is secreted, increasing the inflammation. This process is exacerbated by the presence of free fatty acids. Both alcohol and consumption of a large meal, especially with red meat, can lead to increases in free fatty acid concentrations; they also are implicated as triggers to acute gout attacks (Norris, 2019).

With repeated attacks, accumulations of sodium urate crystals, called tophi, are deposited in peripheral areas of the body, such as the great toe, the hands, and the ear. Renal uratolithiasis (kidney stones), with chronic kidney disease secondary to urate deposition, may develop.

Primary hyperuricemia may be caused by severe dieting or starvation, excessive intake of foods that are high in purines (shellfish, organ meats), or heredity. In secondary hyperuricemia, gout is a clinical feature secondary to any of a number of genetic or acquired processes, including conditions in which there is an increase in cell turnover (leukemia, multiple myeloma, some types of anemias, psoriasis) and an increase in cell breakdown. Altered renal tubular function, either as a major action or as an unintended side effect of certain pharmacologic agents (e.g., diuretics such as thiazides and furosemide), low-dose salicylates, or ethanol can contribute to uric acid underexcretion (Klippel et al., 2008). The finding of urate crystals in the synovial fluid of asymptomatic joints suggests that factors other than crystals may be related to the inflammatory reaction. Recovered monosodium urate crystals are coated with immunoglobulins that are mainly IgG. IgG enhances crystal phagocytosis, thereby demonstrating immunologic activity (Klippel et al., 2008).

Clinical Manifestations

Manifestations of the gout syndrome include acute gouty arthritis (recurrent attacks of severe articular and periarticular inflammation), **tophi** (crystalline deposits accumulating in articular tissue, osseous tissue, soft tissue, and cartilage), gouty nephropathy (renal impairment), and uric acid urinary calculi. Four stages of gout can be identified: asymptomatic hyperuricemia, acute gouty arthritis, intercritical gout, and chronic tophaceous gout (Neoai, Jansen, Dalbeth, et al., 2015). The subsequent development of gout is directly related to the duration and magnitude of the hyperuricemia. Therefore, the commitment to lifelong pharmacologic treatment of hyperuricemia is deferred until there is an initial attack of gout.

Acute arthritis is the most common early clinical manifestation. The metatarsophalangeal joint of the big toe is a commonly affected joint. The tarsal area, ankle, or knee may also be affected. Less commonly, the wrists, fingers, and elbows may be affected. Trauma, alcohol ingestion, dieting, medications, surgical stress, or illness may trigger the acute attack. The abrupt onset often occurs at night, awakening the patient with severe pain, redness, swelling, and warmth of the affected joint. Early attacks tend to subside spontaneously over 3 to 10 days without treatment. The attack is followed by a symptom-free period (the intercritical stage) until the next attack, which may not come for months or years. However, with time, attacks tend to occur more frequently, involve more joints, and last longer (Becker & Gaffo, 2019).

Tophi (seen in chronic tophaceous gout) are generally associated with more frequent and severe inflammatory episodes. Higher serum concentrations of uric acid are also associated with more extensive tophus formation. Tophi most commonly occur in the synovium, olecranon bursa, **subchondral bone** (bony plate that supports the articular cartilage), infrapatellar and Achilles tendons, and subcutaneous tissue on the extensor surface of the forearms and overlying joints. They have also been found in the aortic walls, heart valves, nasal and ear cartilage, eyelids, cornea, and sclera. Joint enlargement may cause a loss of joint motion. Uric acid deposits may cause renal stones and kidney damage.

Medical Management

Given that the incidence of gout increases with age, its management can be complicated by other medical conditions, medications, and age-related changes. A definitive diagnosis of gouty arthritis is established by polarized light microscopy of the synovial fluid of the involved joint. Uric acid crystals are seen within the polymorphonuclear leukocytes in the fluid during a disease flare up (CDC, 2019).

Acute attacks are managed with colchicine (oral or parenteral), an NSAID such as indomethacin, or a corticosteroid. Management of hyperuricemia,

tophi, joint destruction, and renal disorders is usually initiated after the acute inflammatory process has subsided. Once the acute attack has subsided, uric acid lowering therapy should be considered. Xanthine oxidase inhibitors, such as allopurinol and febuxostat, are the agents of choice. Given the role of IL-1 in the pathogenesis of gout, some experts suggest that there may be a role for anakinra, an IL-1 receptor antagonist in the management of acute gout (Becker & Perez-Ruiz, 2019).

Management between gout attacks needs to include lifestyle changes such as avoiding purine-rich foods, weight loss, decreasing alcohol consumption, and avoiding certain medications. Uricosuric agents, such as probenecid, may be indicated in patients with frequent acute attacks. Uricosuric medications correct hyperuricemia and dissolve deposited urate. Corticosteroids may also be used in patients who have no response to other therapy. In patients with refractory chronic gout who are not controlled with the regimens mentioned earlier, pegloticase, a newer agent, has been shown to be effective in lowering uric acid levels (Becker & Perez-Ruiz, 2019). Specific treatment is based on the serum uric acid level, 24-hour urinary uric acid excretion, and renal function (see Table 34-4).

TABLE 34-4 Common Medications Used to Treat Gout

Medication	Actions and Use	Nursing Implications
colchicine	Lowers the deposition of uric acid and interferes with leukocyte infiltration, thus reducing inflammation; does not alter serum or urine levels of uric acid; used in acute and chronic management	<i>Acute management:</i> Administer when attack begins; dosage increased until pain is relieved or diarrhea develops, then stop medication <i>Chronic management:</i> Causes gastrointestinal upset in most patients.
probenecid	Uricosuric agent; inhibits renal reabsorption of urates and increases the urinary excretion of uric acid; prevents tophi formation	Be alert for nausea and rash.
allopurinol, febuxostat	Xanthine oxidase inhibitors; interrupt the breakdown of purines before uric acid is formed; inhibit xanthinoxidase because uric acid formation is blocked	Monitor for side effects, including bone marrow depression, nausea, vomiting, diarrhea, abdominal pain, or rash. Avoid starting medication or increasing dose if active flare present.

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer.

Nursing Management

Research indicates that providers overestimate patient knowledge of gout and that patients prefer the use of both written and verbal materials (Abhishek & Doherty, 2018). Therefore, the nurse takes every opportunity to educate and reinforce knowledge of gout verbally and in writing. Severe dietary restriction is not necessary; however, the nurse encourages the patient to restrict consumption of foods high in purines, especially organ meats, and to limit alcohol intake. Maintenance of normal body weight should be encouraged. In an acute episode of gouty arthritis, pain management with prescribed medications is essential, along with avoidance of factors that increase pain and inflammation, such as trauma, stress, and alcohol. Medication adherence is critical but poor among patients prescribed urate lowering therapies (Scheepers, van Onna, Stehouwer, et al., 2018). The nurse reinforces the importance of taking prescribed medications. Between acute episodes, the patient feels well and may abandon medications and preventive behaviors, which may result in an acute attack. Acute attacks are most effectively treated if therapy begins early.

Fibromyalgia

Fibromyalgia is a chronic pain syndrome that involves chronic fatigue, generalized muscle aching, stiffness, sleep disturbances, and functional impairment. It is estimated to affect more than 5 million Americans, representing 2% to 5% of the general population, with women affected more than men. Between 25% and 65% of patients with fibromyalgia have other rheumatic conditions such as RA, SLE, and AS (CDC, 2017).

Pathophysiology

The amplified pain experienced by patients with fibromyalgia is thought to be neurogenic in origin. The central nervous system's ascending and descending pathways that regulate and moderate pain processing function abnormally, causing amplification of pain signals. Some describe this as if the "volume control setting" for pain were abnormally high. Therefore, stimulation that may not normally elicit pain, such as touch, may do so. In addition, there are a number of predisposing factors to pain, including anxiety, depression, physical trauma, emotional stress, sleep disorder, and viral infection (CDC, 2017; Melin, Svensson, & Thulesius, 2018).

Assessment and Diagnostic Findings

Since fibromyalgia is a diffuse syndrome, standard diagnostic testing is often not useful except to rule out other conditions that may be causing the pain.

One study reported that two items, pain upon pinching the Achilles tendon using 4 kg of pressure for 4 seconds and an affirmative answer to the statement “I have a persistent deep aching all over my body,” were the most useful in recognizing fibromyalgia (Jones, Aebischer, St John, et al., 2017). Positive responses to these screening items need to be followed by a comprehensive examination for confirmation of a diagnosis (Jones et al., 2017).

Medical Management

Treatment consists of attention to the specific symptoms reported by the patient. NSAIDs may be used to treat the diffuse muscle aching and stiffness. Tricyclic antidepressants such as amitriptyline and nortriptyline as well as sleep hygiene measures are used to improve or restore normal sleep patterns (CDC, 2017). Muscle relaxants such as cyclobenzaprine may also be used to help with relaxation and pain. Cognitive behavioral therapy is also useful in improving sleep and attentional dysfunction. In addition, serotonin norepinephrine reuptake inhibitors, such as duloxetine, venlafaxine, and milnacipran; selective serotonin reuptake inhibitors, including fluoxetine, paroxetine, and sertraline; as well as anticonvulsants such as gabapentin and pregabalin may be effective. Individualized programs of exercise are used to decrease muscle weakness and discomfort and to improve the general deconditioning that occurs in affected patients. There has been some promising research in complementary, alternative, and integrative health therapies, such as acupuncture (Kim, Kim, Lee, et al., 2019).

Nursing Management

Typically, patients with fibromyalgia have endured their symptoms for a long period of time. They may feel as if their symptoms have not been taken seriously. Nurses need to pay special attention to supporting these patients and providing encouragement as they begin their program of therapy. Patient support groups may be helpful (Melin et al., 2018). Careful listening to patients’ descriptions of their concerns and symptoms is essential to help them make the changes that are necessary to improve their quality of life.

Miscellaneous Disorders

The last category in the classification of the rheumatic diseases is aptly labeled miscellaneous disorders because it contains a mix of disorders that are frequently associated with arthritis and other conditions. These include the direct consequences of trauma (including internal derangement and loose bodies of joints), pancreatic disease (related to avascular necrosis or

osteonecrosis), sarcoidosis (a multisystem disorder particularly of the lymph nodes and lungs), and palindromic rheumatism (an uncommon variety of recurring and acute arthritis and periarthritis that in some may progress to RA but is characterized by symptom-free periods of days to months). Other conditions include villonodular synovitis, chronic active hepatitis, and drug-related rheumatic syndromes. The nursing interventions related to these varied conditions are specific to the multisystemic problems experienced by the patient. However, the musculoskeletal components should not be neglected or overlooked. Further information about these rare disorders can be found in specialty references.

CRITICAL THINKING EXERCISES

1 pq Your patient, a 52-year-old male with RA, is being discharged from the medical surgical unit where you work. He smokes 1 pack of cigarettes a day and drinks alcohol on occasion. He is 6'0" and weighs 240 lb. Identify the priorities, approach, and techniques you would use to provide discharge education to this patient. What lifestyle modifications are a priority for this patient?

2 ipc A 28-year-old patient who was recently diagnosed with SLE comes to the clinic where you work for a follow-up appointment. She tells you that she is planning to become pregnant soon. What additional members of the health care team should be included in the care of this patient given her childbearing plans?

3 ebp You are the nurse taking care of a woman newly diagnosed with fibromyalgia. What is the evidence for symptom management for this patient? What criteria would you use to assess the strength of the evidence? What is the evidence for complementary therapy options to improve her symptoms?

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*Asterisk indicates nursing research.

**Double asterisk indicates classic reference.

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Resources

- American College of Rheumatology and Association of Rheumatology Health Professionals, www.rheumatology.org
- American Fibromyalgia Syndrome Association (AFSA), www.afsafund.org
- Arthritis Foundation, www.arthritis.org
- Centers for Disease Control and Prevention, www.cdc.gov
- Lupus Foundation of America, www.lupus.org
- National Institute of Arthritis and Musculoskeletal and Skin Diseases, National Institutes of Health, www.niams.nih.gov

Scleroderma Foundation, www.scleroderma.org

Sjögren's Syndrome Foundation, www.sjogrens.org

Spondylitis Association of America, www.spondylitis.org