Odontogenic Infections: General Principles

20

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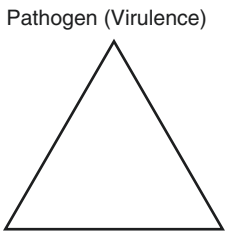
20.1 Introduction

Relationships like symbiosis, commensalism, ectoparasitism, and endoparasitism exist between various species in the biological world. An infection is a classic example of either ectoparasitism or endoparasitism. All infections occurring in the human body are usually of microbial origin and odontogenic infections are no exception.

This relationship is based on discordance in the delicate balance between the host defense mechanism and pathogenicity or the virulence of the infecting microbes depending upon their potentially harmful biomolecules viz. exotoxin, endotoxin, enzymes, and others that disrupt the host defense. Essentially, the environment also seems to play an important role in the overall pathogenic behavior of the microorganism. This interrelationship can be expressed by a simple triangle.



In short, when a low host resistance, pathogen-friendly environment, and a pathogen of high virulence are seen in states of physical being, we refer to as Infections

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20.2 Host Defense Mechanisms

As a generalization, host defense mechanisms may be studied as

1. Local.
2. Systemic

- Local Mechanisms:
  1. Oral Mucosa:

     The oral mucosa forms the first line of defense against the pathogens with an epithelialization rate of 4–5 days wherein the entire oral mucous membrane is replaced by newer more equipped cells take its place. The cells that are infected or otherwise worn out are replaced. As both the skin and oral mucosa are keratinized structures, water and along with it the microbes cannot naturally percolate into the deeper tissues. The oral mucosa is water resistant and relatively dry and does not form a congenial niche for microbial adherence and growth.
  2. Natural spillways:

     Human dentition has evolved to chew the food we eat. The surface anatomy of the occlusal surfaces is such that with a fibrous diet, the food automatically spills off the occlusal table into the labio-lingual vestibule. The interdental contacts are also evolved and provide natural spillways for masticated food.
  3. Dental Histology:

     The microscopic structures of our dentition reveal the hardest substance of the human body (the dental enamel) to cap its relatively softer structures (dentin and pulp). Thus, even if microbes are able to hold onto the enamel layer, the sheer density and mineralization of this layer are sufficient enough to handle most of the microbial attacks.

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4. Saliva:
   By virtue of evolution, the human race has been privileged with 3 pairs of major salivary glands and numerous minor salivary glands. Their secretions, on the one hand, keep the oral environment moist to avoid inadvertent trauma and, on the other hand, help transform the chewed food into a bolus to be swallowed. Salivary secretion continues throughout the awake state, so even if one is not eating, the salivary secretions flush out the food debris deposited on the hard and soft surfaces of the mouth, in conjunction with the movements of the muscular elements of oral region such as lips, cheek, tongue, etc. Other physiological properties of the human saliva such as pH (6.5–8.5) and the presence of immunoglobulins (IgG and IgA) makes saliva a biological bacteriostatic fluid.
5. Oral sub mucosal immune system (OSMIS):
   A large number of immunocompetent cells, such as the mononuclear leukocytes, T cells, and B cells, are present between the oral mucous membrane and are responsible for the production of antibodies as well as offering a direct cellular immune response to the host and oral environment. The cell concentrations of IgG, IgA, and IgM may remarkably reach 85, 72, and 75%, respectively, with respect to plasma concentration, in the gingival sulcular fluid. However, generally only IgA levels are high in the oral mucosa, while the concentrations of IgG may rise dramatically during infectious states. Also, the true ponderance of mast cells in the submucosal layer and the presence of IgE explain the immediate hypersensitivity (type I) reaction. The role of these immunoglobulins is to neutralize the microbial toxins and maintain hemostasis in the oral environment.
6. Dentinal reparative mechanisms:
   If the bacterial attack breaks the enamel barrier, the toxins reach the dentinal tubules and through them irritate the pulp. This may or may not be symptomatic but pulpal reaction is evident with the formation of reparative dentin to seal off the breach as part of the defense mechanism. When this occurs, we know it as arrested caries and the dentin so formed is the affected dentin. If due measures are not taken at this stage, an advanced carious lesion may result.
7. Waldayer's Ring:
   The OSMIS, lingual tonsil, palatine tonsil, all other lymphoid structures in the naso-oropharyngeal region together make a ring-like primary defense mechanism against microbes in the oro-digestive tract and are constitutionally known as the Waldayer's ring.

- *Systemic Mechanisms*: Broadly speaking, the systemic host immunity may be classified as.
  1. Cellular.
  2. Humoral.

Both these immune systems may be either innate (natural) or acquired (adaptive). Majority of pathogens are dealt with by the innate component of the host systemic immunity. However, when the innate response is overwhelmed, the adaptive or active immunity comes into play.

Components of Innate Immunity:

- Physical barriers/skin/mucosa.
- Neutrophils.
- Macrophages.
- Dendritic cells.
- Natural killer cells.
- Lymphoid cells.
- Complement system.

Components of Acquired Immunity:

- B-lymphocytes.
- Dendritic cells.
- T-lymphocytes.
  - Helper.
  - Cytotoxic.
  - Regulatory.

Innate immunity is basically a receptor-pattern recognition mechanism, where about 100 intracellular and extracellular receptors (e.g., toll-like receptors, NOD-like receptors, KIG like receptors, etc.) recognize over 1000 different cell injury-related patterns generated from bacterial, viral, or fungal inflictions [1]. These receptors generate a response mediated by cytokines and antiviral interferon and stimulate the most proactive immune response.

Acquired immunity is of two types, viz. humoral immunity, mediated by soluble proteins known as antibodies (produced by B lymphocytes) and cell-mediated immunity effected by T lymphocytes (T cells). While antibodies provide protection against extracellular pathogens in blood, mucosa, and tissues, T lymphocytes work to defend against intracellular microbes to kill ingested microbes by production of soluble proteins mediators, the cytokines (produced by helper T cells).

20.3 The Infectious Microbes

The concept of an infectious agent was established by Robert Koch. Koch's postulates became the standard for defining infectious agents, but they do not apply to uncultivable

organisms (e.g. M. leprae) or members of normal humoral flora (e.g. *E. coli*).

Koch's postulates:
1. The same organism must be present in every case of that disease but not in any healthy individual.
2. The organism must be isolated from the diseased individual and cultured.
3. The isolate must cause the disease, when inoculated in a healthy individual.
4. The organisms must be re-isolated from inoculated diseased individual.

Odontogenic infections arise within or around the dentition, initiating from simple dental caries, periodontal diseases, and pulpitis, and may spread way beyond their loco-regional origin, invading deeper structures of the face, oral cavity, head and neck, and even mediastinum or the vertebral column.

These infections are principally bacterial in nature Tables 20.1 and 20.2. These bacteria are a part of normal oral flora found in the dental plaque, mucosal surfaces, and the gingival sulcus. These primarily are aerobic gram positive cocci, anaerobic gram positive cocci, and gram negative rods [3].

Table 20.1 Types of bacteria in periapical abscess [4]

Types of bacterial infections	Percentage
Anaerobic only	50%
Mixed anaerobsic and aerobic	44%
Anaerobic only	6%

Table 20.2 Common bacterial species in periapical abscess

Endodontic	Periodontal [2]
1. Anaerobic gram negative bacteria (a) Treponema (b) Dialister (c) Porphyromonas	(a) Aggregatibacter actinomycetemcomitans (b) Treponema denticola (c) Campylobacter rectus (d) Prevotella intermedia (e) Tannerella forsythia (f) Porphyromonas gingivalis
2. Facultative gram negative bacteria (a) Neisseria (b) Capnocytophaga (c) Haemophilus	
3. Anaerobic gram positive bacteria (a) Actinomycetes (b) Peptostreptococcus	
4. Facultative gram positive (a) Enterococcus (b) Streptococcus	

20.4 Pathways of Odontogenic Infection

Odontogenic infections have two major origins (Fig. 20.1).

(a) Periodontal- due to bacterial inoculations into underlying tissues via deep periodontal pockets and
(b) Periapical-, more common, and occur subsequent to pulpal necrosis, reaching the periapical structures (Fig. 20.2).

Once the periodontal or periapical tissues get inoculated with bacteria, the infection may spread equally in all directions but mostly follows the path of least resistance. It travels through the cancellous bone to reach the cortical plate. If the cortical plate is thin, infection easily perforates it to enter the surrounding soft tissue. Periapical enzymes that help the bacteria in doing so include collagenase, hyaluronidase, and streptokinase, which dissolve through the organic matrix of the bone, while the acids produced by the bacteria eliminate the mineral content.

At this stage, if an intervention such as an endodontic or periodontal procedure or dental extraction is done, the further spread may be arrested or even abolished with judicious antibiotics. Antimicrobials alone may not cure the condition as the focus of infection from necrotic pulp or periapical tissues still remains and may cause recurrence of the infection, if the therapy is stopped [5].

When left untreated, the infection continues to spread depending principally on the thickness of bone and the type of muscle attachment. For example, if maxillary anterior tooth gets involved in the periapical region and the inclination of the root is such that the apex is closer to the labial cortical plate, the soft tissue reaction would present as swelling on the labial side (Fig. 20.3a) and if the apex is closer to the palatal cortical plate, swelling would be palatal. (Fig. 20.3b). Similarly, in case of carious involvement of maxillary first molar: if the apices of the buccal roots lie above the attachment of the buccinator, swelling is likely to occur facially (Fig. 20.4a) and if apices are below, a vestibular swelling is more likely to occur (Figs. 20.4b and 20.5).

Apart from these factors, the angulation of root apex is also important. For example, in periapical abscess with respect to maxillary lateral incisor, swelling is likely to occur on the hard palate rather than labial vestibule as its root apex is slightly palato-distally curved. (Fig. 20.6).

Muscle attachment is another influencing factor. Supposing a mandibular second molar gets apically infected and the apices lie above the external oblique ridge, infection following the path of least resistance and assisted with gravity, may manifest as swelling in a more anterior region of the jaw, buccally than the relative position of the first mandibular molar. Similarly, in the mandibular posterior region, the lingual cortex is closer to the root apices and relatively thinner

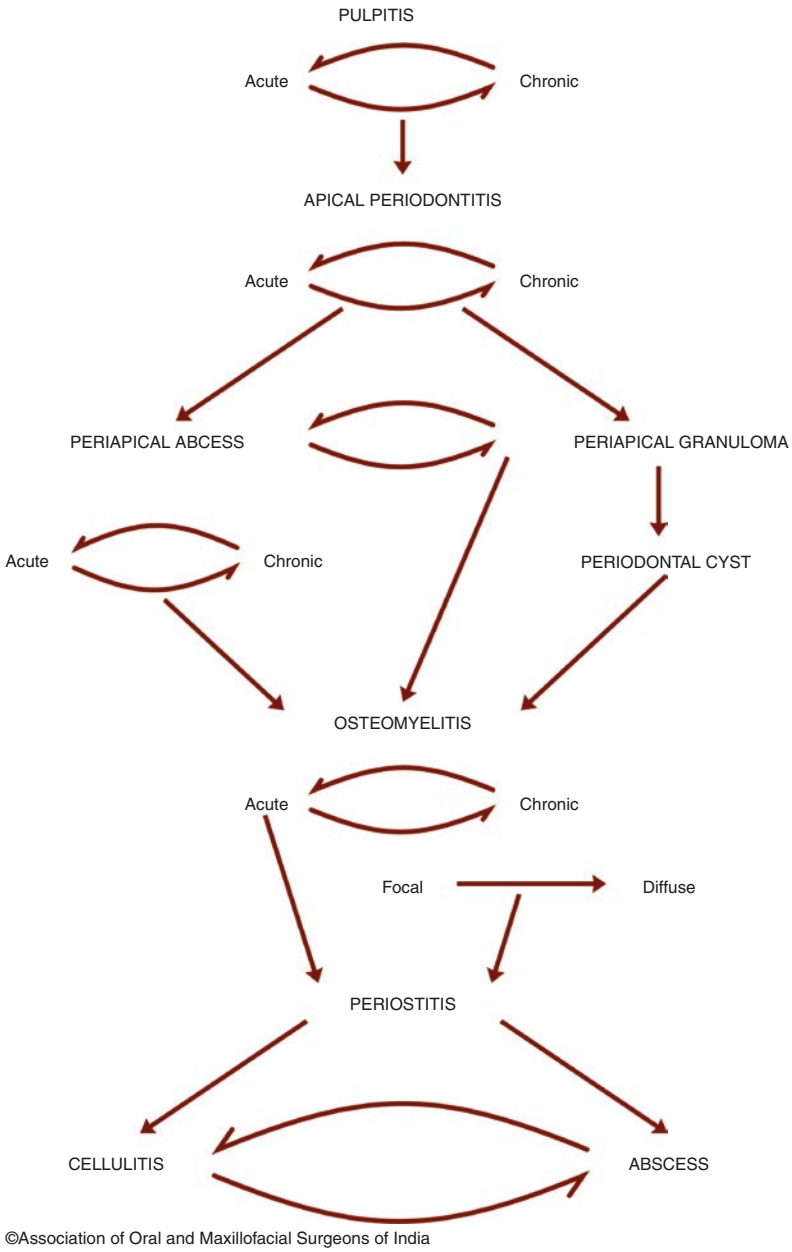

Fig. 20.1 Natural progression of odontogenic infection

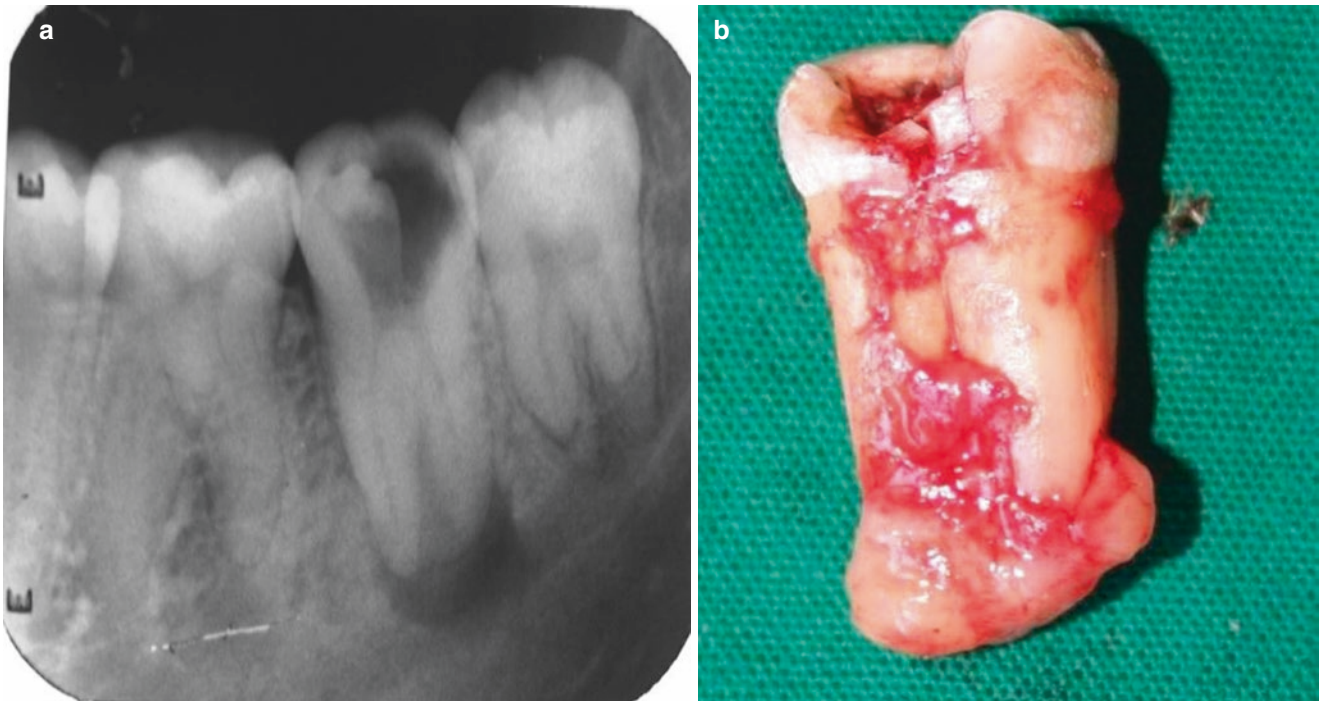

Fig. 20.2 (**a**, **b**) shows a lower left second molar with a periapical radiolucency. On extraction, the periapical granuloma can be seen attached to the root. If this tooth was not extracted, the infection would have progressed in any of the pathways as shown in Fig. 20.1

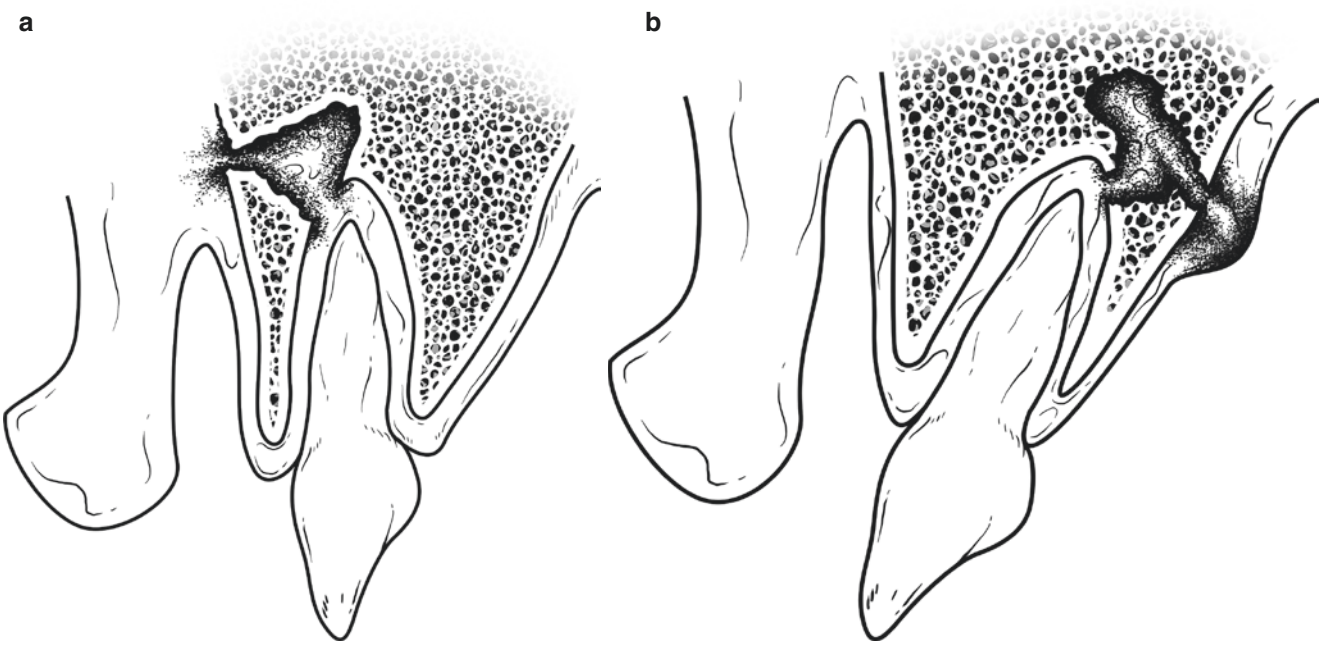

Fig. 20.3 (**a**, **b**) Infection following the path of least resistance in case of maxillary incisors based on angulation of root

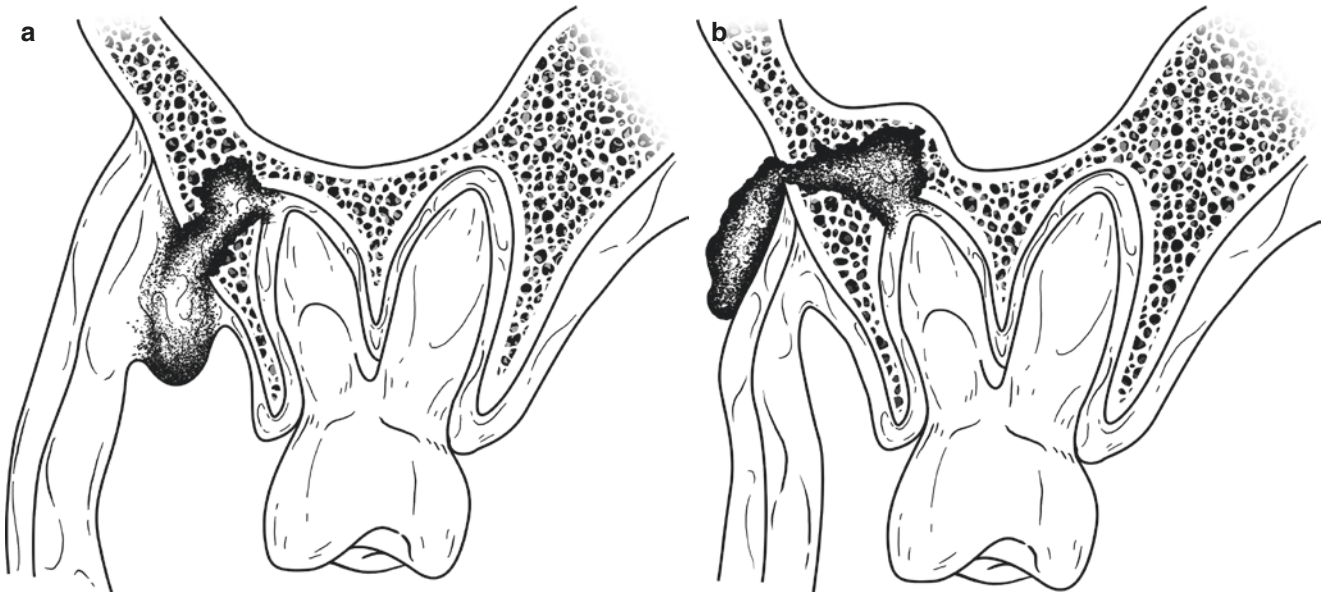

Fig. 20.4 (**a**, **b**) Effect of buccinator muscle attachment on the site of appearance of swelling in case of maxillary molars. In (**a**) the infection has localised to the vestibule while in (**b**) the infection has spread to the buccal space, as the root apex is situated above the attachment of the buccinator muscle

Fig. 20.5 Shows the clinical picture of a vestibular abscess arising from an infected upper right first molar

Fig. 20.6 Palatal abscess arising from upper incisors

as compared to the buccal cortical plate. If this gets perforated above the mylohyoid line, a sublingual swelling occurs and if below it, a submandibular swelling occurs.

The commonest manifestation of odontogenic infections is either a palatal or a vestibular abscess. When untreated, it may lead to an intraoral or extraoral drainage, in due course establishing a sinus tract. The treatment involves removing the cause (endodontically or via exodontia) and management of the sinus tract. In certain cases, the sinus tract resolves by itself, following the treatment of the offending cause (Fig. 20.7).

20.5 Pathways of Spread of Periapical Infections

An odontogenic infection follows the path of least resistance. A periapical infection may perforate the nearest or the weakest cortex and travel along the soft tissue, initially as cellulitis and eventually resulting in abscess formation. This abscess may drain spontaneously, extraorally or intraorally and may involve one or more anatomically potential spaces. When this happens, it is known as a space infection.

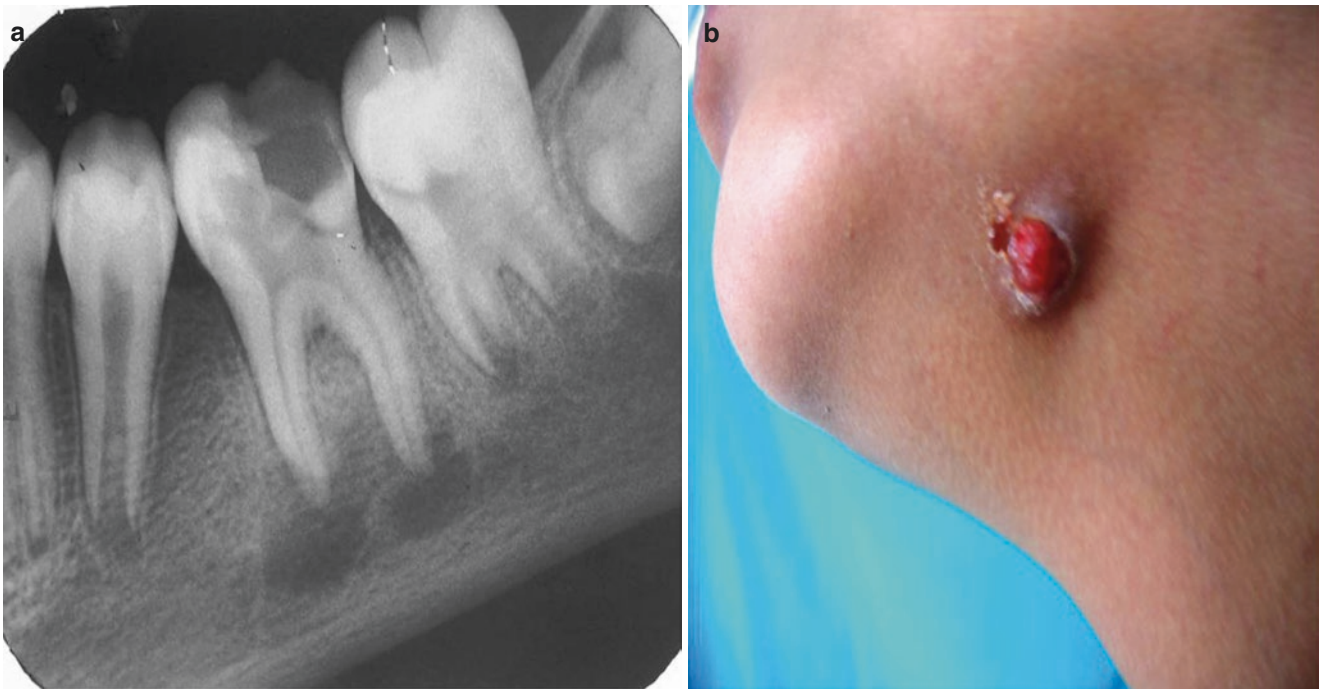

Fig. 20.7 (**a**, **b**) Shows the periapical lesion in lower left first molar in a 14-year-old patient causing extraoral sinus at the left lower border mandible region

Following are frequently affected anatomic spaces, (Fig. 20.8):

1. With any tooth.
   - Subcutaneous.
   - Vestibular.
   - Buccal.
2. With any offending maxillary tooth.
   - Buccal.
   - Maxillary along with other para nasal sinuses.
   - Infraorbital.
   - Infratemporal.
   - Temporal.
3. With any offending mandibular tooth.
   - Medullary space of mandibular body.
   - Submandibular.
   - Sublingual.
   - Submental.
   - Masticator.
     - *Submasseteric*.
     - *Pterygomandibular*.
     - *Superficial temporal*.
     - *Deep temporal*.
4. Deep fascial spaces.
   - Lateral pharyngeal.
   - Retropharyngeal.
   - Pretracheal.
   - Danger (Alar space).
   - Prevertebral.

Various spaces involved in an infection differ in their relative severity depending upon the proximity of the vital anatomic structures in their vicinity. This may be seen as follows.

20.6 Various Space Infections and Their Relative Severity

MILD—Vital structures and airway may be mildly threatened

- Subperiosteal.
- Infraorbital.
- Buccal.
- Vestibular.
- Osteomyelitis of the mandible.

MODERATE—Airway may be compromised

- Pterygomandibular.
- Superficial temporal.
- Submandibular.
- Sublingual.
- Submental.

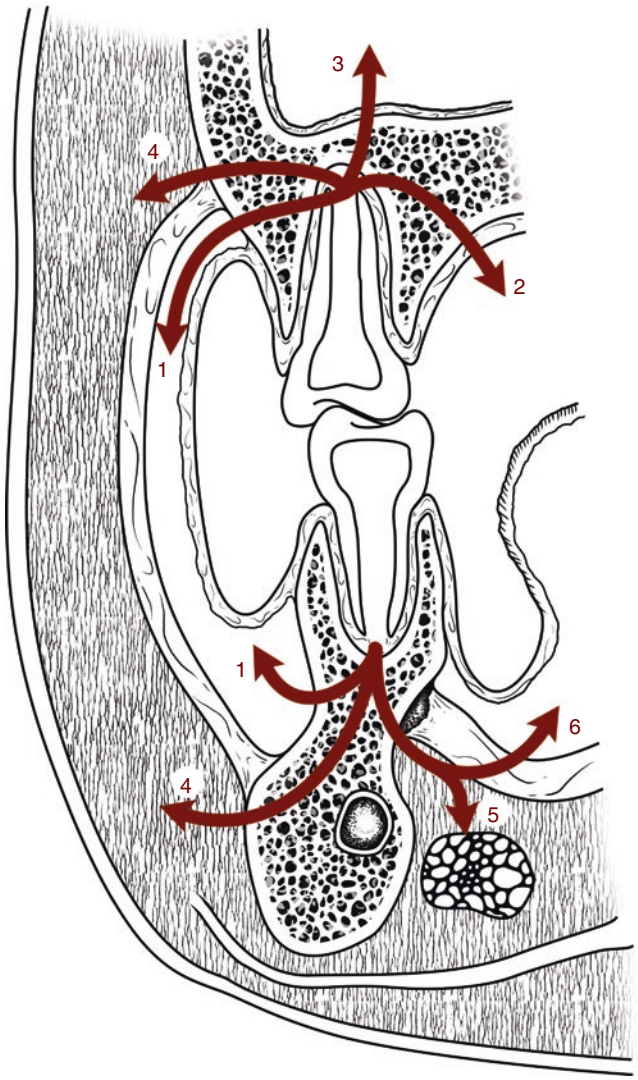

Fig. 20.8 Pathways of spread of periapical infection. (1) Vestibular space. (2) Palatal abscess. (3) Maxillary sinus/odontogenic sinusitis. (4) Buccal space. (5) Submandibular space. (6) Sublingual space

- Submasseteric.
- Infratemporal.
- Deep temporal.
- Masticator.

HIGH—Vital structures or airway under direct threat

- Lateral pharyngeal.
- Retropharyngeal.
- Danger space (Alar space).
- Pretracheal.
- Cavernous sinus thrombosis.
- Intracranial infections (brain abscess).
- Mediastinal.
- Prevertebral.

20.7 Basic Therapeutic Principles

1. Delineation of cause and severity.
2. Assessment of host's immune response.
3. Role of specialist.
4. Surgical intervention.
5. Pharmacotherapeutics.
6. Follow-ups.

1. *Delineation of cause and severity:* As a standard protocol, a proper history should be recorded in chronological order stating the patients' chief complaints in his/her own words.

   Once the chief complaint is noted, history of present illness is determined with an estimation of how long back the patient was absolutely symptom free. Usually, infections have a short history of onset as compared to tumors in their acute state [6].

   Next thing to be noted is any change in the patients' state of physical well-being since the symptoms first appeared, i.e. if they have been the same, improved, or worsened with time. Since the response to an infection is essentially an inflammatory one, classic sign of inflammation are always looked for (tumor, rubor, dolor, color, and loss of function). Principal presenting feature is pain, so the nature of pain, site, and diurnal variation should be noted. A sharp, piercing, and lancinating type of pain is generated by Aδ nerve fibers that usually awake the patient at night (in accordance with the hydrodynamic theory of pulpal pain by Brannstrom [7]); however, a dull, aching, and gnawing kind of pain arises due to stimulation of slowly firing "c" type nerve fibers.

   Swelling, redness, and rise of temperature should be inquired for and inspected even during history taking if these are apparent. Loss of function may manifest in the form of dyspnea, dysphagia, difficulty in chewing, or trismus [8]. A feeling of general malaise usually indicates a physical or physiological response against moderate to severe infection [9].

   At this point, an enquiry into any previous treatments by another dentist, specialist, and/or self-treatment should be made and recorded. After carefully listening to the chief complaints and history of present illness, a comprehensive medical history should be recorded. The most efficient means of doing so is either by personal structured interview or by means of a questionnaire along with a discussion with the patient for any positive findings.

History taking is followed by a physical examination that begins with the recording of patients' vitals as temperature, pulse rate, respiratory rate, and blood pressure. Patients with infections usually have elevated body temperature, and increased pulse and respiratory rates [10]. If the pain is significant, patient's blood pressure may be elevated. However, hypotension is seen in septicemic states. Since odontogenic infections are inflammatory states, a partial or complete upper airway compromise may occur owing to an extension of the current infection to the deep facial spaces of the neck [11]. The patients may present with fatigue, fever, and malaise or so-called toxic appearance (Table 20.3) [12].

Palpation of swellings should be done to know the tenderness and consistency of the swelling. Consistency of a swelling may vary from normal, firm, fleshy (dough like) to indurated (consistency of a taught muscle/wooden like/brawny hard), or fluctuant (feel of a fluid-filled balloon). Fluctuation almost always indicates presence of pus in the center.

Intraoral examination gives a fair amount of idea as to the cause of the infection, i.e. the offending tooth. A careful look may reveal a grossly carious tooth/teeth, periodontal diseases, and/or a fractured tooth.

Investigative phase begins with a radiograph; intraoral periapical (IOPA) or orthopantomogram (OPG) depending on the symptomatology and clinical examination. CT scan may be required in severe cases to assess the pathway of the spread of infection [13].

If any surgical intervention is required, a routine blood assessment is always helpful in deciding the type of procedure to be followed.

A decision has to be made as to the stage of the infection, i.e. inoculation, cellulitis, or abscess stage. Usual attributes are as follows.

2. *Assessment of Immune response:* General medical history is the guide to detect any interfering systemic conditions that may compromise the host defense mechanism and may worsen patient's condition rapidly. Conditions of compromised host defense mechanism are as under:

Table 20.3 Stages of infection

Stage of infection	Clinical features
Inoculation	Edema/Inflammation
Cellulitis	Acute pain
	Firm to borderline swelling/induration
	Diffuse borders
	All cardinal features of inflammation
Abscess	Localized pain (acute abscess)
	Fever (more often than not)
	Fluctuant swelling

(a) Diabetes Mellitus (type I and II): When uncontrolled, it results in an overall decrease in the immunity owing to decreased phagocytosis, chemotaxis, and generalized decrease in leukocyte function. However, in patients with moderate to severe infection, an increased random blood sugar cannot be a diagnostic criteria for underlying diabetes. Since these are high-stress states, the body gluconeogenic mechanisms are upregulated to meet the increased BMR. So, a more comprehensive analysis like HBA_1C is performed to know the long-term glycemic status.

(b) Leukocytic upsurge is expected in infection. The baseline leukocyte/neutrophil count serves as a marker to track the response of the patient to medical and surgical intervention in a case of severe odontogenic infection.

(c) Immuno-deficient states: HIV infection principally affects the T lymphocytes, meaning that the humoral immune system is still intact. Odontogenic infections are generally bacterial and usually well defended by the patients' immune system, but in immunocompromised states, the basic host defense mechanism no longer functions. When the disease advances to involve even the B-cells, then, even simple infections may become serious life-threatening issues in such patients. It is hence important to know that such immunocompromised HIV-positive individuals need more intensive therapy than a normally infected individual.

(d) Another example of systemic immuno-deficient states may be prolonged corticosteroid therapy. Here the inflammatory cascade itself gets modulated, and affects the immune system as a whole, resulting in decreased production of the cell adhesive molecules, deficient leukocyte chemotaxis, and decreased cytokines production (IL-1, TN α, IL-6, etc.). No apoptotic activity results in generalized lymphocytopenia along with IL-2 mediated cyto-proliferative responses. When used long enough or in high doses, delayed-type hypersensitivity (type IV) response is inhibited along with decreased antibody formation. Such patients soon develop oral thrush and other superimposed bacterial or viral infections of the oral cavity, worsening of an existing odontogenic infection being no exception.

(e) End Stage Renal Diseases (ESRD): End stage renal diseases affect almost all aspects of the innate and acquired immunity, and many such patients will be on maintenance hemodialysis. ESRD patients having odontogenic infections also may have to be managed

comprehensively where adequate support from the allied speciality is available.

(f) Drug-induced immuno-incompetence: While blood counts less than 1000 cells/ml are typically seen in patients on anti-cancer chemotherapy, patients who undergo organ transplants have to be on immune-suppressive therapy (IST). Drugs like corticosteroids and cyclosporins have decreased B and T lymphocyte function and consequently deficient immunoglobulin production. As such, patients are likely to have exaggerated manifestation for even mild odontogenic infections.

(g) Alcoholism and/or Hepatic insufficiency: Alcoholics are usually malnourished and have pending hepatic damage that leads to poor qualitative and quantitative immunoglobulin supply.

Above discussion reinforces that while assessing a patient, a through medical history as well as a history of habits provides vital clues for the action to be adopted for his/her management.

3. *Role of Specialist:* Role for patient care should shift from a general dentist to a maxillofacial surgeon. Antibiotics should be started if indicated as in Table 20.4. When the situation demands, either a hospital admission or immediate surgical care under regional or general anesthesia is required. The following criteria may be adopted for making this vital discussion in patient's interest:
   (a) Failed earlier management.
   (b) Medically compromised patients.
   (c) Toxic appearance patients.
   (d) Febrile patient: temperature greater than 101 °F.
   (e) Signs of dehydration.
   (f) Dyspnea.
   (g) Difficulty swallowing (dysphagia).
   (h) Moderate to severe trismus (with mouth-opening between 10 and 20 mm).
   (i) Need for general anesthesia.
   (j) Airway compromise.
   (k) Signs of ascending or descending facial infections (cavernous sinus thrombosis/deep neck infections/mediastinitis).

Table 20.4 Indications for antimicrobial therapy

1. Trismus (mouth opening between 10 and 20 mm).
2. Cellulitis (infection may not have drained completely).
3. Lymphadenopathy (lymph nodes may harbor residual infection).
4. Temperature > 101 °F (bacteremia).
5. Osteomyelitis.
6. Severe soft tissue involvement (e.g. necrotizing fasciitis).
7. Swelling extends beyond alveolar anatomy.
8. Systemically compromised patient.

4. *Surgical Intervention:* Surgical drainage and removal of the course of infection is the mainstay of surgical management of odontogenic infection [14]. This may be as simple as endodontic therapy to wide incision in the submandibular/neck region that may require mediastinal exploration. The removal of the cause is the primary goal and drainage of the accumulated pus and necrotic debris is the secondary goal.

Incision and drainage principally includes insertion of a drain in the abscess cavity to prevent premature closure of the mucosal/skin incision. Purpose of this procedure is to achieve adequate drainage, if it is not possible by endodontic means (especially in mandibular teeth) (refer Chap. 21 for details on incision and drainage).

Pus culture and Sensitivity Tests: This must be done before the incision and drainage procedure if possible. The site is prepared with antiseptics and a thick needle is used to aspirate pus, may be in multiple directions, under negative pressure of syringe, and taking due care to prevent relevant anatomic structures. In case of complex space infections involving the digastric/carotid triangle, ultrasound-guided aspiration must be done. This culture when obtained from sites of cellulitis (Ludwig's Angina) usually contains serosanguinous fluid but harbors sufficient bacteria for culture. This aspirate is inoculated into both anaerobic and aerobic cultures (sterile tubes having sterile swabs and bacterial transport medium) with viable shelf life [15].

20.8 Antimicrobial Therapy

Once the culture and sensitivity reports are available, specific antimicrobial therapy may be installed but meanwhile empirical antibiotics should be started immediately [16]. The choice of antibiotic should be carefully thought off to prevent unnecessary disturbance of the essential microflora of the gut and development of cross resistance (Also refer Chap. 10 of this book).

Cases of moderate to severe infection generally need postoperative antibiotics, where the host defense cannot be solely relied upon to fight the residual infection. Since the antibiotic sensitivity of usual causative organisms of odontogenic infections is fairly well known and consistent, an empirical therapy may include clindamycin, metronidazole, and penicillin. Metronidazole being narrow spectrum covers only obligate anaerobic population and the rest may be covered by other drugs dealing with facultative microbes.

Odontogenic infections are complex entities, both, on the basis of the plethora of flora associated and mechanism of their spread to the adjacent as well as distant anatomic

Following principles should be additionally borne in mind before prescribing antibiotics [17]:

1. Use of narrowest possible spectrum- prevents bacterial resistance.
2. Use of antimicrobial with least toxicity and side effects- prevents vital organ damage.
3. Use of bactericidal rather than bacteriostatic drug- prevents residual infection.
4. Weigh cost to benefit ratio- prevents over expenditure.
5. Use only in prescribed dose and duration- restricts side effects and maintains efficacy.
6. Use the appropriate route of administration- restricts side effects and maintains efficacy.

sites. Fortunately, culture and sensitivity behaviors of the causative microbes are fairly consistent and well understood. As such timely management should alleviate most of the dangerous outcomes but still these infections remain one of the leading cause of maxillofacial morbidity and at times mortality.

20.9 Conclusion

Odontogenic infection can be severe enough and become life threatening, hence should be dealt with very carefully. Proper evaluation of the signs and symptoms guide the clinician to make the diagnosis. Radiographic assessment may suggest the involvement of one or more teeth. Surgical intervention includes drainage of abscess, and pus be sent for culture and sensitivity to help choose the right antibiotic regime.

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