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OBJECTIVES:

To provide a review of the etiology, risk factors, treatment, and nursing care of patients with colorectal cancer.

DATA SOURCES:

Review articles, screening guidelines, and textbook chapters.

CONCLUSIONS:

Although colorectal cancer remains a major health threat in the United States, advances made over the last 10 years in prevention, diagnosis, and treatment have changed the management and care of patients with this disease. The key to survival of colorectal cancer is screening and early detection.

IMPLICATIONS FOR NURSING PRACTICE:

Regardless of the multimodalities of treatment used, the nurse's role as educator, caregiver, supporter, and advocate requires an ongoing commitment to remain knowledgeable of and current in advances made in the prevention, detection, and treatment of colorectal cancer.

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COLORECTAL CANCER

DELORES A. SADDLER AND CONI ELLIS

AMERICANS have an average lifetime risk of approximately 6% of developing colorectal cancer.[1,2] Estimated new cases in 1998 for cancer of the colon, rectum, and anus are 95,600, 36,000 and 3,300, respectively, totaling 134,900 new cases.[3] Cancer of the colon and rectum is the third leading cause of cancer death for men and women aged 35 to 75 years and the second leading cause of death from cancer overall. The estimated number of colorectal cancer deaths for 1998 is 57,000. Between 1990 and 1994, however, mortality rates for men decreased by approximately 1.9% per year for men with colorectal cancer and by approximately 1.5% for women.[3] The 5-year survival rate for colorectal cancer has increased to greater than 90% in the early, localized stage, and to greater than 60% after spread to adjacent organs or nodes.[4]

The overall incidence of colorectal cancer is similar in men and women. Rectal cancer is seen more frequently in men than in women, as is anal cancer. Although the risk of colorectal cancer begins at the age of 40 years and increases with advancing age, its mean presentation is in patients 60 to 65 years of age. Rates also vary by race and ethnicity. Because these factors are strongly correlated with socioeconomic status, some differences in incidence and mortality rates found among racial and ethnic groups may be the result of differences in socioeconomic status.[3] In blacks, the incidence of colon cancer has increased by 30% since 1973 and is now higher than in whites.[4] The incidence of colorectal cancer is higher in industrialized regions than in Asia, Africa, and South America. The highest rates are found in North America and Australia.

Colon cancer includes more than half of all cancers of the large bowel. Seventy percent of colon cancers occur in the right side of the proximal colon. Rectal cancer arises below the peritoneal reflection or less than 12 cm from the anal verge. Anal cancers account for fewer than 4% of all lower gastrointestinal cancers.[4] Etiology, risk factors, and treatment for colon, rectal, and anal cancers differ and are discussed separately in the literature. Educational programs designed to increase awareness of the effects of colorectal cancer and the value of screening and surveillance for the disease are under way. An estimated 30,000 lives could be saved each year if the general public, primary care clinicians, and

managed care organizations were aware of and used methods of early detection and treatment.[1,2] New approaches to multimodal therapy are continuously being investigated to improve survival.

Etiology and Risk Factors

The specific causes of colon cancer are not known, but environment, nutrition, and genetic factors, as well as pre-existing diseases, are often associated with the disease. Persons who emigrate from low-risk parts of the world, such as the Asian countries, assume the colon cancer risk of their adopted country if they follow diets rich in fat (especially saturated animal fat) and cholesterol, which have been linked to an increased risk of colorectal tumors.[4]

Dietary fat causes endogenous production of secondary bile acids and neutral steroids. This increases bacterial degradation and excretion of these acids and steroids, promoting colonic carcinogenesis. Diets rich in fiber or bran and yellow and green vegetables are believed to have a protective effect. Calcium salts and calcium-rich foods, because they decrease colon-cell turnover and reduce the cancer-promoting effects of bile and fatty acids, are also said to have a protective role.[1]

Several genetic premalignant polyposis syndromes are associated with a high risk of colorectal cancer.[5] Persons with a positive family history of polyposis syndromes are at high risk. Familial adenomatous polyposis (FAP) coli is an inherited autosomal dominant trait that results in the development of polyps throughout the colon and rectum in late adolescence. These persons have a 100% risk of developing colorectal cancer.[6] Hereditary nonpolyposis colorectal cancer (HNPCC) is an autosomal dominantly inherited condition characterized by the occurrence of colorectal cancer at an average age of 45 years.[7] Affected individuals often have one or a few adenomatous polyps, but not polyposis. Hereditary nonpolyposis colon cancer occurs as type A (Lynch type I) and type B (Lynch type II). Type A is familial, site-specific, nonpolyposis colon cancer. Type B is nonpolyposis colon cancer in association with other forms of cancer, such as breast, endometrial, gastric, and ovarian. Once cancer occurs in an individual at risk for HNPCC, a subtotal colectomy should be performed. Prophylactic hysterectomy and/or bilateral salpingo-oophorectomy should be considered for women with HNPCC diagnosed as having cancer or who are genetic carriers.

A variant of FAP consists of multiple flat adenomas that have an increased risk of becoming cancerous.[8,9] Several other genetic premalignant polyposis syndromes are associated with a high risk of colorectal cancer. Hamartomatous polyposis syndromes include Peutz-Jeghers, juvenile polyposis, Cowden's disease (multiple hamartoma syndrome), and neurofibromatosis; types of adenomatous polyposis are FAP, Gardner's syndrome, and Turcot's syndrome.[4]

Hereditary nonpolyposis colorectal cancer arises from mutations in any one of four genes that participate in mismatch repair, the repair of defective DNA strands. When mismatch repair is not functioning, mutations occur in one or more genes that are important to the control of cell growth.[10] The genes that are mutated in colon cancer syndromes have been identified and reproduced and are now available for testing in humans. Although the genes involved in common familial risk have not been identified, colon cancer is known to be caused by an accumulation of genetic mutations by which, initially, a colonic epithelial cell acquires the characteristics of an adenoma, which eventually acquires the characteristics of invasive cancer. With or without familial risk, colon cancers seem to develop from a similar set of genes and a similar progression of accumulated mutations.[10] The commonly mutated genes in colon cancer are the adenomatous polyposis coli gene (responsible for FAP), the K-*ras* oncogene, the p53 gene, the deleted in colon cancer gene, and DNA mismatched pair genes (HNPCC). Relatives of persons with large-bowel malignancy have an increased risk of colorectal cancer. First-degree relatives of two persons with colon cancer have a risk factor two to three times that of the population at large, or if their colon cancer is diagnosed at 50 years or younger. Detecting and removing polyps significantly reduces the incidence of colorectal cancer.[2]

Adenomatous polyps identified as villous and tubovillous adenomas are more likely to undergo malignant transformation than are tubular adenomas. The probability that cancer will be present in an adenoma is approximately 5%. A polyp smaller than 1 cm can transform into invasive cancer in approximately 10 years.[1,2]

Patients with a history of colorectal cancer are at increased risk of developing a second primary

colon cancer or other malignancy, particularly at the site at which an anastomotic connection was made by previous surgery. Patients with ulcerative colitis are at risk of developing colorectal cancer, the degree of risk depending on the extent of colitis, the development of mucosal dysplasia, and the duration of symptoms. Colorectal cancer risk is also higher than normal in patients with Crohn's disease, although less than in the above-mentioned patients.

Prevention and Detection

Approximately 75% of all new cases of colorectal cancer occur in people who have no known predisposing factors and are therefore considered to be at average risk. Fifteen percent to 20% of members of the high-risk population have a family history of colon cancer but no genetic predisposition. Hereditary nonpolyposis colon cancer accounts for 4% to 7% and FAP accounts for approximately 1% of cases.[1,2] The screening options available now are based on whether persons are at average or high risk (Fig 1).

There are two methods of prevention: primary and secondary. Primary prevention consists of, among other things, eating a healthy diet, including lots of fruits and vegetables, and increasing the amount of total fiber in the diet with regular consumption of wheat bran. Vegetables and fruits contain important antioxidants such as beta-carotene, vitamin C, vitamin E, selenium, and calcium. Vitamin D is a micronutrient with antipromotive activity. The consumption of calcium is inversely correlated with colonic cancer incidence.[11,12] Calcium is believed to bind cytotoxic bile acids in the intestines, making them insoluble and less harmful to the epithelium of the colon. Nonsteroidal anti-inflammatory drugs are also used as preventives. Diets high in fat and red meat are the strongest dietary determinants of colon cancer risk. Fermenting carbohydrates in the colon undergo a series of activities that are likely to influence carcinogenesis. Maintenance of

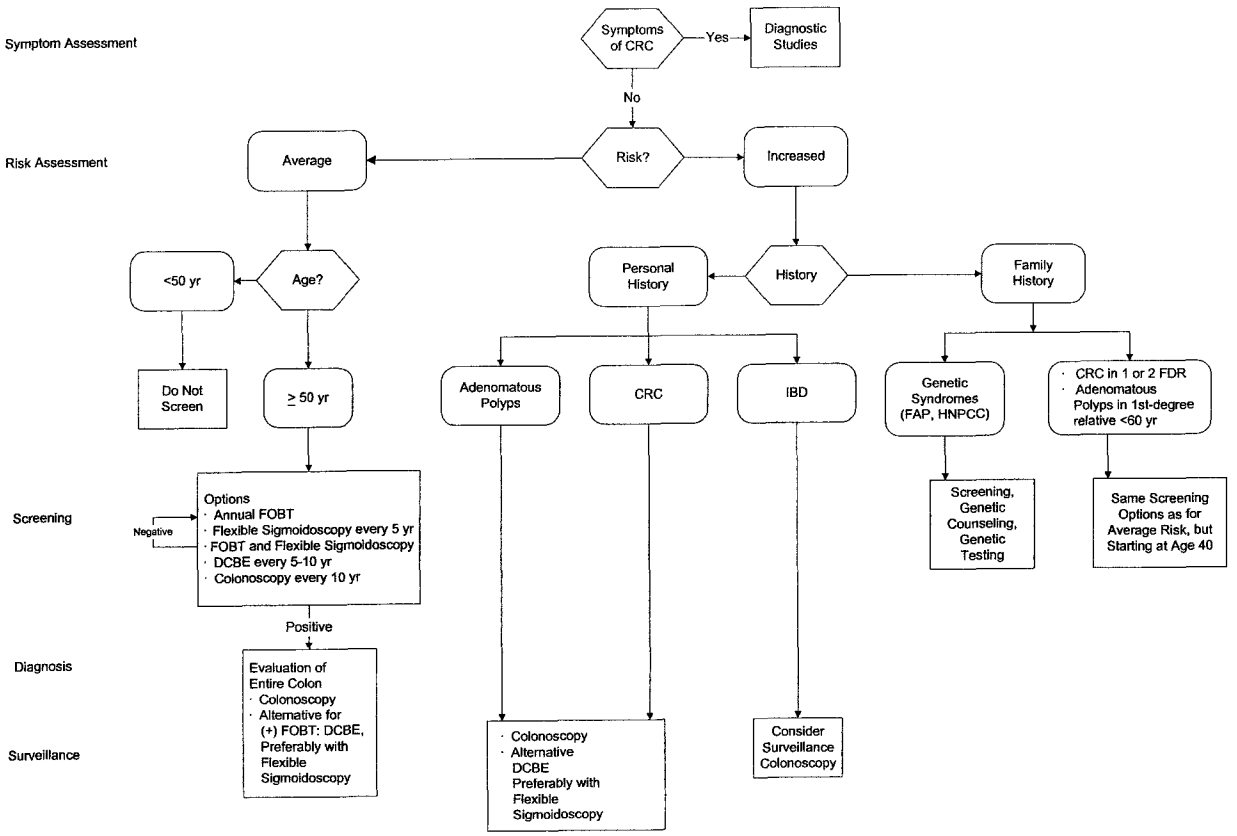

FIGURE 1. Algorithm summarizing screening options. CRC, colorectal cancer; FOBT, fecal occult blood test; DCBE, double contrast barium enema; IBC, irritable bowel disease. (Reprinted with permission.[2])

a normal body weight so that total caloric intake does not exceed energy requirements helps prevent weight gain and decreases the risk of colorectal cancer.[1,2] Increasing the amount of exercise helps to decrease body fat. Chemoprevention, the secondary method, is an effort to block the action of carcinogens on cells before the appearance of cancer.

End points for evaluating these preventive measures include evaluating changes in adenomatous polyps, alterations in mucosal proliferation, and colorectal cancer incidence.[4] The nonsteroidal anti-inflammatory drugs inhibit colorectal carcinogenesis by redirecting endogenous prostaglandin production. Sulindac, for example, has been found to induce regression of large bowel polyps in patients with FAP.[13] Studies have demonstrated a reduction in the incidence of colorectal cancer with regular long-term use of aspirin.[14] Reductions in smoking and alcohol consumption are other means of reducing the incidence of colorectal cancer. Postmenopausal women should be on a hormonal replacement regimen. Although many of these preventive measures are still under investigation, they are milestones in the identification of preventive measures and the possible causes of colorectal cancer.

Screening for colorectal cancer identifies individuals who are more likely to have colorectal cancers or adenomatous polyps. By educating the general public, primary caregivers, and managed care companies about the symptoms and signs of colorectal cancer, and the importance of surveillance and follow-up, approximately 30,000 lives could be saved each year.[2]

Colorectal cancer is difficult to diagnose because patients are asymptomatic early in the disease process. Clinical manifestations depend on the location of the tumor. Most patients have lymph node involvement or metastatic spread at the time of presentation. Table 1 describes clinical signs and symptoms that may be present in patients with colorectal cancer.[4,12,15,16]

TABLE 1. *Clinical Signs and Symptoms That May Be Present in Patients With Colorectal Cancer*

Stage/Location	Signs and Symptoms
Early cancer	Asymptomatic; vague abdominal pain and flatulence, minor changes in bowel movements with or without rectal bleeding
Left-side cancer	Constipation alternating with diarrhea; abdominal pain; obstructive symptoms, ie, nausea/vomiting
Right-side cancer	Vague abdominal aching discomfort, anemia, weakness, weight loss, right-side abdominal mass
Rectal cancer	Changes in bowel movement, rectal fullness, urgency, bleeding, tenesmus, jaundice, malaise, occult blood, pelvic pain
Anal cancer	Bleeding, discharge, rectal mass, tenderness on palpation, pain on defecation

Diagnosis and Staging

Next to surgical resection, staging is the most important prognostic tool. The most widely used clinical and pathologic staging system is the modified Astler-Coller Dukes system, based on the depth of the tumor invasion into and through the intestinal wall, the number of regional lymph nodes involved, and the presence or absence of distant metastasis. The TNM staging system has been modified to correlate with the Dukes system for staging colorectal tumors (Table 2).[4] Other factors to consider are age at diagnosis, presurgical level of carcinoembryonic antigen, duration of symptoms, site of disease, histologic characteristics of tumor, perineural invasion, presence of perforation or obstruction, venous or lymphatic invasion, and ploidy. Five-year survival rates of 50% to 100%, 30% to 80%, and 11% to 58% have been reported for, respectively, grades 1, 2, and 3 tumors.

The diagnostic work-up includes a clinical evaluation of specific complaints, evaluation of family risk factors, palpation for masses, a digital rectal examination, fecal occult blood test, and a vaginal examination to assess for fistulas. Other studies include an endoscopic ultrasound for evaluation of rectal tumors, colonoscopy preoperatively to assess for polyps or other tumors, barium enema if needed, a complete blood cell count to check for anemia, liver function tests, carcinoembryonic antigen level, CA 19-9 (a colon-specific protein) level, chest x-ray, and computed tomography (CT) scans of the abdomen and pelvis. Three

TABLE 2. TNM Staging of Colorectal Cancer

TNM Stage	Primary Tumor	Lymph Node Metastasis	Distant Metastasis	Modified Astler-Coller
Stage 0	Tis	N0	M0	
Stage I	T1	N0	M0	A
	T2	N0	M0	B1
Stage II	T3	N0	M0	B2
	T4	N0	M0	B3
Stage IIIA	Any T	N1	M0	C
Stage IIIB	Any T	N2, N3	M0	
Stage IV	Any T	Any N	M1	

Abbreviations: Tis, carcinoma in situ; T1, tumor invades submucosa; T2, tumor invades muscularis propria; T3, tumor invades through the muscularis propria into the subserosa or into nonperitoneal pericolic or perirectal tissues; T4, tumor perforates the visceral peritoneum or directly invades other organs or structures; N0, no regional lymph node metastasis; N1, metastases in one to three pericolic or perirectal lymph nodes; N2, metastases in four or more pericolic or perirectal lymph nodes; N3, metastases in any lymph node along the course of a named vascular trunk; M0, no distant metastasis; M1, distant metastasis.
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to six samples should be obtained for the fecal occult blood test. Before the samples are collected, the health care team should give a thorough explanation of the diet. Patients should be notified of the results of the tests and a colonoscopy should be scheduled if one or more samples are positive.

Magnetic resonance imaging is a sensitive method for detecting lesions smaller than 2 cm in diameter, especially those in the rectum and pelvis. This procedure and other diagnostic methods for colorectal cancer, such as spiral CT scan and radioimmunoscintgraphy, are used for staging.

Computed tomography portography is used preoperatively to detect small focal hepatic metastases 1 to 2 cm in diameter. The technique allows the clinician to see more tumors than can be seen on conventional CT scan with contrast medium. Computed tomography portography is based on the principle that hepatic metastasis derives its blood supply from the hepatic artery, while mainly the portal circulation supplies normal hepatocytes. For CT portography, contrast medium is infused through an angiography catheter introduced into the superior mesenteric or splenic artery. Seconds after injection of the contrast medium, dynamic-sequence CT scanning of the liver begins. As the dye passes into the portal vein, the normal hepatic parenchyma infused by the portal blood circulation is enhanced with contrast, while the hepatic metastasis, which receives no blood via the portal route, is visualized as a dark spot that indicates a solid lesion. For all CT portography imaging, a rounded, well-delineated space-occupying lesion indicates a metastatic lesion. This testing precedes any planned liver resection or cryosurgery.[17]

Physiology of the Colon and Rectum

The colon consists of four layers: the mucosa, the submucosa, the muscularis, and the serosa. The mucosa and submucosa are divided by the muscularis. Cellular reproduction in the colon takes place in the crypts of Lieberkühn located in the mucosal layer. New cells, constantly produced in the crypts, mature, migrate out of the crypt to the surface, and shed. Exposure to carcinogens affects the reproducing cells in the crypt: in the presence of transformed cells or repeated exposure to carcinogens, the crypts may become populated with DNA repair cells that are prone to errors and the formation of early adenomatous lesions.[18]

The colon's main physiologic functions are absorption of water and electrolytes, fermentation of carbohydrates, and absorption of the main products of the process. Fiber, which enters the colon unaltered, is subjected to fermentation by colonic flora that yields short-chain fatty acids and gases. Fermented or not, fiber components have major effects on colon physiology. They increase the bulk of colonic content, which shortens transit time, reduces water absorption, and increases fecal bulk. Absorption of fat depends on the presence of conjugated bile acids. Primary bile acids are synthesized and secreted by the liver into the small intestine and reabsorbed in the terminal ileum. From there they are transported to the liver and excreted again; this is the so-called enterohepatic circulation. Approximately 3% of bile acids escape reabsorption and enter the colon. Low stool weight and low water content seem to be related to colon cancer incidence. Solid material remains in the colon up to 24 hours before ascending to the transverse colon. Under normal physiologic conditions, the descending colon is

considered to be only a conduit between the transverse and sigmoid colon. The rectum is usually empty except shortly before defecation. The assumption is that high fat consumption stimulates bile acid output, resulting in higher concentrations of conjugated bile acids in colonic contents. In the colon, bile acids are deconjugated and the primary bile acids are dehydroxylated to form secondary bile acids. Increased risk is associated with consumption of animal fat; olive oil and fish oil may decrease risk.[19]

Radiation Therapy for Colorectal Cancer

Tumor cells are well-oxygenated when treated preoperatively by radiation because the blood supply to the tumor has not been surgically manipulated. Well-oxygenated cells are believed to have increased radiosensitivity; tumor cell killing by radiotherapy may be increased.[20] Despite these advantages, preoperative radiation has not affected overall rates of survival, distant recurrence, or cure rates.[21,22] However, locoregional tumor control rates have improved. Randomized studies have shown a significant decrease in local recurrence rates when preoperative doses of radiotherapy were higher than 34.5 Gy.[21,23-26] Additionally, one study[21] reported a 91% sphincter preservation rate for patients with T3 and T4 lesions treated with 45 Gy of preoperative radiotherapy. Local control and overall survival have been at acceptable levels with this approach, and 10% of patients in this series achieved a complete pathologic response.[20]

The advantages for the use of postoperative radiotherapy are (1) adequate pathology data are available to evaluate the extent of disease, (2) patients who will not benefit from therapy are not treated, and (3) surgical treatment is not delayed. Despite the performance of several large prospective trials, rates of survival, local pelvic control, and extrapelvic recurrence have not been improved consistently by radiation doses of 45 to 50 Gy.[21-23] In addition, a large study comparing preoperative with postoperative radiotherapy found a significant decrease in local recurrence in the group who received preoperative therapy.[27] Table 3 describes the uses of radiation therapy for patients with colorectal cancer. Table 4 lists the toxic effects of colorectal radiation therapy.

TABLE 3. Radiation Therapy for Colorectal Cancer

Preoperatively
To reduce tumor size and increase potential for sphincter preservation
To decrease risk of local failure and distant metastasis from cells shed at operation
To decrease risk of late radiation enteritis since small bowel can be excluded from radiation field
Postoperatively
For patients considered at high risk of local recurrence
When distant metastases are still a potential problem
Combined preoperative and postoperative radiation ("sandwich technique")
To decrease potential of tumor dissemination
To decrease potential of complication resulting from repopulation by residual cells if postoperative therapy is delayed by slow wound healing
Palliative radiation
To treat symptoms of advanced cancer
To control pain and bleeding

Surgical Options for Colon Cancer

Surgery is the primary form of treatment for colon cancer. Its goal is to eliminate the disease in the colon, the draining of nodal basins, and contiguous organs. The tumor location, blood supply, and lymph node patterns in the involved region will define the extent of resection. The various surgical options as well as their indications and major morbidities are briefly discussed below and are illustrated in Fig 2.[28]

Right Hemicolectomy

Right hemicolectomy involves removal of the distal 5 to 8 cm of the ileum, right colon, hepatic flexure, and transverse colon just proximal to the middle colic artery. This procedure is indicated for

TABLE 4. Side Effects of Colorectal Radiation Therapy

Side Effects	Signs and Symptoms
Proctosigmoiditis	Bleeding, tenesmus, and pain
Increased bowel motility	Cramping, loose watery stools
Chronic radiation enteritis	Ulcerations
Colon fibrosis and stricture	Large bowel obstruction
Nausea and vomiting	

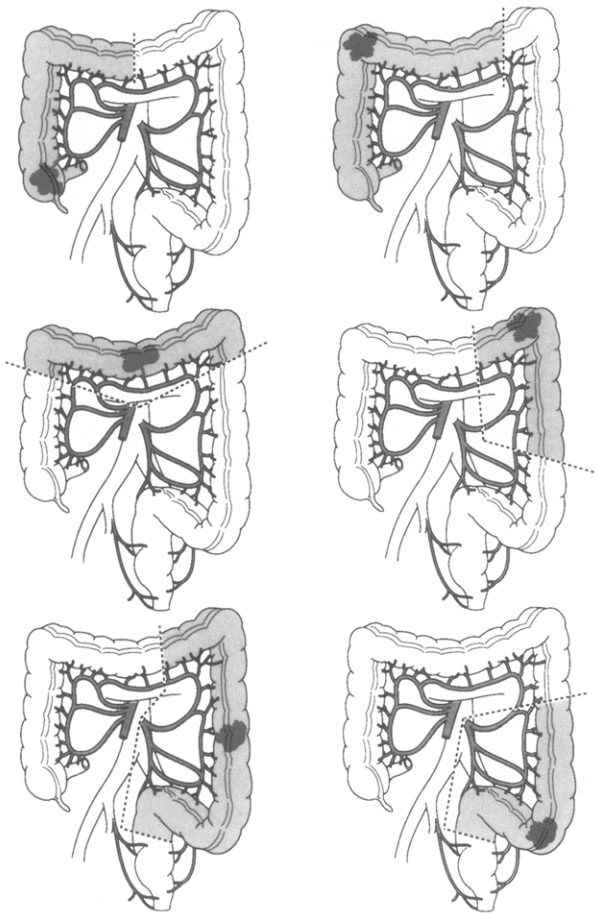

FIGURE 2. Segmental resections for cancers of the colon and upper third of the rectum: (top left) right hemicolectomy, (top right) extended right hemicolectomy, (middle left) transverse colectomy, (middle right) left hemicolectomy, (bottom left) extended left hemicolectomy, and (bottom right) low anterior resection. (Reprinted with permission.[28])

cecal, ascending colonic, and hepatic flexure lesions. Major morbidities include ureteral injury, duodenal injury, and bile acid deficiency (the last rarely seen and only with extensive resection of the terminal ileum).

Right Radical Hemicolectomy

Right radical hemicolectomy involves the removal of the transverse colon (including resection of the middle colonic artery at its origin) in addition to structures removed in the right hemicolectomy. Indications for this procedure are hepatic flexure or transverse colon lesions. Morbidities include anastomotic dehiscence and diarrhea in addition to the complications associated with right hemicolectomy.

Transverse Colectomy

Transverse colectomy is the segmental resection of the transverse colon. This procedure is indicated for midtransverse colon lesions. The major morbidity is anastomotic dehiscence. At M.D. Anderson Cancer Center, this procedure is rarely performed because of the difficulty of achieving a tension-free anastomosis with adequate blood supply (as the marginal artery of Drummond is sacrificed). Surgeons prefer to perform an extended right radical hemicolectomy with an ileosigmoid anastomosis.

Left Hemicolectomy

A left hemicolectomy includes the removal of the transverse colon distal to the right branch of the middle colic artery and the descending colon up to, but not including, the rectum, plus division and ligation of the inferior mesenteric artery. Indications for this procedure are left colon lesions. Anastomotic dehiscence is the major morbidity.

Low Anterior Resection

Low anterior resection involves the removal of the descending colon distal to the splenic flexure, the sigmoid colon, the upper two thirds of the rectum, and ligation of the inferior mesenteric artery and inferior mesenteric vein either at the origin or just distal to the origin of the left colic artery. This procedure is indicated for sigmoid and proximal rectal lesions. Morbidities include anastomotic dehiscence and bowel ischemia (secondary to inadequate flow through the marginal artery of Drummond).

Subtotal Colectomy

Subtotal colectomy is the removal of the right, transverse, descending, and sigmoid colon with ileorectal anastomosis. This procedure is indicated for multiple synchronous colonic tumors and distal transverse colon lesions in a patient with a clotted inferior mesenteric artery. Morbidities include diarrhea, perineal excoriation, and anastomotic dehiscence.[29]

These surgical procedures may produce many different results. Patients who have an abdominoperineal resection (APR) will have a permanent sigmoid or descending colostomy, whereas, with a low anterior resection (LAR) or subtotal colectomy, the patient may have a temporary ileostomy. All these patients benefit from having a certified wound, ostomy, continence nurse (otherwise known as an enterostomal therapist or ET

nurse) beginning with the first clinic visit at which they are told the diagnosis, through the preoperative and postoperative phases.

Surgical Management of Rectal Cancer

The successful management of rectal cancer has five goals: cure, local control, restoration of intestinal continuity, preservation of anorectal sphincter function, and preservations of the patient's sexual and urinary function. Because of the anatomic constraints of the bony pelvis, it may be difficult at times to achieve adequate sphincter, sexual, and urinary function without compromising cure and local control.[30]

Local control is an extremely important aspect of treatment. Up to 25% of patients dying of rectal cancer will have local recurrence only, while another 50% will have local recurrence in addition to distant disease.[20] Patients with local recurrence after treatment for rectal cancer are rarely salvaged by additional surgery. Many of these patients suffer greatly from bone and nerve pain, hemorrhage, pelvic sepsis, and bowel and urinary obstruction.[29,30]

Abdominoperineal Resection

The radical surgical approach and standard treatment of patients with rectal cancer has been the APR, which involves transabdominal resection of the rectum and mesorectum from the level of the inferior mesenteric vessels to the levator muscles, in combination with transperineal excision of the anus and distal rectum.[29] An APR is currently indicated for distal-third rectal tumors within 3 cm of the anal verge, tumors involving the anal sphincter musculature, tumors of the rectovaginal septum, patients with poor continence preoperatively, and patients with diarrheal disorders.[29]

Sphincter-Preserving Surgery

In recent years, the use of adjuvant therapy, the introduction of circular stapling devices, and the demonstrated adequacy of 2-cm distal margins have allowed safe use of sphincter-preserving surgery for resection of mid- and some distal-rectal cancers.[29] Low anterior resection, as described earlier, is a procedure in which the dissection and anastomosis are performed below the peritoneal reflection.

A coloanal anastomosis preserves the sphincter mechanism in patients with low-lying rectal tumors whose negative distal margin of resection is up to but does not include the anal-sphincter musculature. The operative dissection is similar to that of LAR and APR, with transection of the distal margin at the level of the levator ani muscles within the abdomen. Through a perineal approach, the remaining anal mucosa is stripped and an anastomosis is made between the colon and the anus to restore intestinal continuity.[29,31] Surgeons at M.D. Anderson Cancer Center hand sew the anastomosis at this level. To provide adequate bowel length and a tension-free anastomosis, the splenic flexure of the colon is completely mobilized. The vascular supply of the left colon is then based on the middle colic artery. The surgeons then perform a protective diverting ileostomy in all patients undergoing coloanal anastomosis.

Contraindications for an LAR or coloanal anastomosis include tumors involving the anal-sphincter musculature, tumors involving the rectovaginal septum, patients with poor continence preoperatively, patients with diarrheal disorders, and unfavorable anatomic constraints (ie, obesity, narrow pelvis).Table 5 identifies some of the complications of colorectal surgery.

Postoperative Nursing Care

Because in today's environment the delivery of care has been accelerated, we have come to an era of critical pathways to ensure that we bring the

TABLE 5. Complications of Colorectal Surgery

Complications	Signs and Symptoms
Anastomotic leak	Intra-abdominal pain; pelvic abscess; peritonitis
Intra-abdominal abscess	Recurring or persistent fever postoperatively; leukocytosis, no abdominal pain
Staphylococcal enteritis	Diarrhea; prostration; sepsis
Large-bowel obstruction	Constipation; abdominal pain; nausea/vomiting; abdominal distention
Injury to the genitourinary tract	Leakage of urine through the incision; oliguria; anuria
Sexual dysfunction	Impotence
Ostomy complications	Peristomal skin and stoma

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patient to the desired outcome in a timely fashion. Table 6 provides an example of the treatment and nursing management for a patient who has had an LAR or an APR. The role of a certified wound, ostomy, continence nurse is emphasized.

Adjuvant Therapy of Colon Cancer

The most commonly used chemotherapeutic agent for the adjuvant treatment of colorectal cancer is 5-fluorouracil (5-FU). The addition of other drugs is being studied to increase the effectiveness of 5-FU and to develop new drugs that function in the same manner. The current combination regimens of systemic adjuvant chemotherapy for colorectal cancer uses 5-FU plus levamisole or leucovorin. Prolonged disease-free survival and overall survival have been seen in patients with stage III colon cancer treated with 5-FU + levamisole[33] and 5-FU + leucovorin.[34]

Since micrometastatic hepatic disease derives its vascular supply from the portal vein, direct delivery of chemotherapeutic agents by this route is being investigated. This treatment has not shown a survival benefit and is not yet recommended except in clinical trials.[4] Future trials should determine whether administration of chemotherapy in the perioperative setting has a positive influence on survival.

Adjuvant Therapy of Rectal Cancer

Rectal cancer patients receive radiation therapy and chemotherapy in sequence, as well as concomitantly, as adjuvant therapy. The combination of radiation therapy and 5-FU seems to be as

TABLE 6. Critical Pathway Nursing for Low Anterior or Abdominoperineal Resection

Restaging/ Preoperative Visit (5 wk Postradiotherapy)	Same-Day Admission	Postoperative Day	
CWOCN meets with patient to focus on patient support, relieving anxiety by providing information; after assessing patient's knowledge, nurse reviews A&P of GI system, surgery to be performed, impact on abilities for activities of daily living, sexuality concerns, and proposed ostomy equipment; the nurse closes the session by assessing the patient's abdomen and marking the location for stoma placement Staff nurse will review preoperative bowel preparation, pain management, ambulation, and turn, cough, and deep breathing needed postoperatively	Staff nurses will manage IV lines, medications, NPO/nasogastric tube, incentive spirometer, Foley catheter, patient-controlled analgesia, TCDB, compression boots, and ambulation; will assess the patient's stoma, pain, need for information CWOCN continues with patient teaching while reassuring the patient about his or her perceived losses (ie, body image, control, love, social status); assesses the peristomal skin and stoma, its function, presence of bridge/rod; focuses on application of pouch, emptying, and cleansing pouch; offers a United Ostomy visitor for additional support	Day 1	When patient resumes bowel sounds and is passing flatus, NGT will be pulled and the patient will be put on a clear liquid diet; the surgeon removes the original dressing; assesses the incision; other treatments continue, patient teaching progresses from pouch application, to cleansing of pouch, to using the clamp
		Day 2	Diet advanced as tolerated; treatments the same
		Day 3	Discontinue IV heplock; discontinue patient-controlled analgesia, give oral analgesics; increase ambulation, continue with respiratory support
		Day 4	Discontinue Foley catheter; continue other treatments
		Day 5	Discontinue heplock, oral analgesics, diet as tolerated, ambulation ad libitum, TCDB; assess incision, stoma; finalize discharge instructions

Outcome criteria/discharge criteria: patient has ambulatory status, adequate oral intake (>1,500 mL/d), evidence of bowel function (no abdominal distention, bowel sounds present, passing flatus/feces from stoma), pain controlled, afebrile, and self-care in most activities of daily living, including wound and ostomy care.
Abbreviations: CWOCN, certified wound, ostomy, and continence nurse; A&P, anatomy & physiology; GI, gastrointestinal; IV, intravenous; NPO, nothing by mouth; TCDB, turn cough, deep breath; NGT, nasogastric tube; PCA, patient controlled analgesia.
Adapted from The University of Texas M. D. Anderson Cancer Center and Physicians Network Critical Pathway.

effective with or without another agent. The general belief is that radiation therapy with a chemosensitizer, in addition to adjuvant therapy with 5-FU, should be the standard treatment for stage II and III disease. Adjuvant and neoadjuvant chemotherapy and radiation therapy are increasingly important in the treatment of lower gastrointestinal cancers.[35] In patients with small rectal cancers, the tumors may be totally ablated with endoscopic laser therapy. Because local regional tumor spread cannot be detected with certainty by current techniques, endoscopic curative treatment is limited to patients with at least relative surgical contraindications. Laser therapy is of greatest value in cases of bleeding exophytic lesions, less so for intramural disease, and of no value for treating extraluminal extrinsic compressive disease.[36] Laser therapy, therefore, is only valuable as palliation and not for cure.

Advanced Colorectal Cancer

The liver is the most common site of metastasis from colorectal cancer. The spread of the tumor is primarily due to the anatomy of the portal vein system; metastasis reaches the liver by portal vein circulation, lymphatic spread, hepatic arterial circulation, and direct invasion.[37] Although improved surgical techniques have increased the numbers of successfully resected tumors, most tumors in the liver are unresectable because multiple segments of the liver are involved. Surgical resection of a solitary liver lesion leads to a 5-year survival rate of 20%.[38] Hepatic cryosurgery is a new technique that has been shown to be effective in controlling hepatic malignancies that are unresectable by traditional surgery. Systemic chemotherapy with 5-FU and leucovorin have demonstrated minimal benefit. Hepatic arterial infusion of various agents, alone or as an adjuvant to liver resection, is being investigated.

Proved effective against 5-FU–resistant colorectal cancers in clinical trials, irinotecan (CPT-11) is a topoisomerase I inhibitor synthesized from *Camptotheca acuminata,* a tree native to China, and has been approved by the Food and Drug Administration for use in the treatment of metastatic colorectal cancer. Response rates of 20% to 30% have been reported in patients refractory to 5-FU.[39] The main toxic effects of irinotecan are diarrhea and neutropenia. Intensive treatment with loperamide is important in managing the diarrhea. Clinical evaluations of the combination of 5-FU with leucovorin and irinotecan in colorectal cancer are pending.

Intrahepatic floxuridine has followed the introduction of implantable infusion pumps. The pumps are designed to allow chemotherapeutic drugs to be delivered at higher concentrations directly into the hepatic artery. Compared with intravenous administration of floxuridine, a survival advantage has not been conclusively demonstrated for this method. In addition, it is costly and associated with gastroduodenal mucosal ulceration, hepatitis, and sclerosing cholangitis.

New agents under development for colorectal cancer include thymidylate synthase inhibitors and oral fluorinated pyrimidines. Raltitrexed is a potent, selective inhibitor of thymidylate synthase when given every 15 minutes over 21 days; it is being compared with 5-FU/leucovorin in phase III trials. Oral fluorinated pyrimidines include two currently undergoing phase III testing in the United States and Europe: UFT, a combination of uracil and tegafur administered together with leucovorin, and capecitabine. Both drugs are metabolized to 5-FU. Their mechanism of action is similar to that of 5-FU/leucovorin. The advantages are the convenience of oral administration and a favorable toxicity profile.[4]

Oral 5-FU plus an enzyme inactivator of dihydropyrimidine dehydrogenase is another drug combination under investigation. Small oral doses of 5-FU combined with the inactivator of dihydropyrimidine dehydrogenase (776C85) have been demonstrated to produce consistent, therapeutic levels of 5-FU in patients' plasma. The drug is currently being studied in phase II/III trials.[4]

Oxaliplatin is a diaminocyclohexane platinum compound used in clinical trials in this country and in Europe. When used alone or with 5-FU, its demonstrated activity in pretreated 5-FU–resistant colorectal cancer shows 10% and 45% response rates, respectively. In patients with untreated metastatic colon cancer, response rates of 27% have been reported.[4]

Conclusion

Survival statistics indicate that early detection of colorectal cancer is the key to survival. As patient advocates, nurses should become familiar with the information and resources available

concerning the latest therapies for patients with colorectal cancer. Care should focus on patient and family education, provision of emotional support, and reinforcement of the treatment information concerning postoperative care and follow-up. Nurses may not have all the answers, but they can guide their patients to the resources they need to battle their disease.

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