

INTRODUCTION

In the USA colorectal cancer is the third most common cancer in males and females. In the year 2000, there were an estimated 130,200 cases diagnosed, including 93,800 cases of colon cancer and 36,400 cases of rectal cancer. Between 1992 and 1996, incidence rates declined approximately 2% per year, a decline thought to be due, in part, to improved screening and treatment of polyps before their progression to invasive cancers. However, colorectal cancer still accounts for 11% of cancer deaths. Estimated for the year 2000 show 56,300 deaths: 47,700 from colon cancer, 8,600 from rectal cancer (*Dennis et al., 2004*).

It has been well established that mutations in a group of genes known as the "mismatch repair genes" increases a patient's susceptibility to cancer. Germ line mutations in these genes constitute the genetic basis of Lynch syndrome, a cancer predisposition syndrome characterized by early onset of cancer in several body parts, including the gastrointestinal tract, and particularly the colon (*Peltomaki, 2000*). Mutations in these genes result in microsatellite instability (MSI), which is defined as the accumulation of mono- or dinucleotide repeats in the DNA. Patients with colon cancer and MSI frequently have tumors involving the right colon and are characterized by a better prognosis (*Watanabe et al., 2001*).

As many as 25% of patients with colorectal cancer have a family history of the disease, which suggests the involvement of a genetic factor. Such inherited colon cancers can be divided into two main types: the well-studied but rare familial adenomatous polyposis (FAP) syndrome, which accounts for approximately 1% of cases of colon cancer annually, and the increasingly well-characterized, more common hereditary nonpolyposis colorectal cancer (HNPCC), which accounts for 5% to 10% of cases (*Farnell et al., 2000*).

Colorectal cancer has been notoriously difficult to treat successfully. Despite numerous attempts at modifying existing therapeutic regimens or designing new ones, survival rates for nonresectable tumors remain low. Progress in endoscopic techniques and interest in early detection of gastrointestinal cancers has enabled the early diagnosis of colorectal cancer. Surgical resection achieves high cure rates for early colorectal cancers, but increasing evidence suggests that endoscopic therapy may be a useful alternative in cases of localized early colorectal cancer. Endoscopic therapy for early colorectal cancer is more advantageous than the conventional operative treatment, in that it is a relatively non-invasive and less costly method. However, endoscopic treatment is completely ineffective in lesions with lymph node or distant metastasis. Therefore, the indications of endoscopic resection for the purpose of curative treatment of early colorectal cancer should be considered carefully (*Kawamura et al., 1999 and Igarashi et al., 2000*).

Because of the potential for cure of early-stage disease, the definition of populations at risk and screening of asymptomatic patients are important considerations. Controlled clinical trials have demonstrated that the multidisciplinary approach to the treatment of localized colorectal cancer has improved the morbidity and mortality of this disease. The first step in determining an appropriate colorectal cancer screening strategy is assessment of an individual's colorectal cancer risk. Although 75% of cases of colorectal cancer occur in individuals who may be considered average risk (*Winawer et al., 2001*).

Laparoscopic colectomies were initially described by Jacobs and associates in 1991 (*Jacobs et al., 1991*). Since this initial report, there has been widespread application of laparoscopic techniques for the treatment of colorectal disease. (*Chapman et al., 2001*).

Treatment of colorectal cancers by laparoscopic resection remains controversial. Prospective and retrospective trials have suggested the safety and feasibility of laparoscopic colectomies. Reported advantages include decreased postoperative pain, decreased ileus, shorter hospital stay, and earlier return to normal activity. There have been concerns; however, regarding potential complications, such as the reported high incidence of trocar site recurrences, the ability to perform an adequate oncologic resection, and the impact on long-term patient survival (*Young-Fadok, et al., 2000*).

AIM OF THE WORK

The aim of this study is to explain the recent trends in management of carcinoma of the colon.

EMBRYOLOGY & ANATOMY OF THE COLON

Embryology:

The primitive gut begins to form during the fourth week of gestation. For descriptive purposes, it is divided into the foregut, midgut, and hindgut. Midgut derivatives include the entire small intestine distal to the ampulla of Vater, the cecum and appendix, the ascending colon, and the right half to two-thirds of the transverse colon. These structures receive their blood supply from branches of the superior mesenteric artery (SMA). At the beginning of the sixth week of gestation, the midgut loop undergoes a physiologic umbilical herniation and migrates into the extraembryonic coelom. During the next 4 weeks the midgut loop elongates considerably and undergoes a series of counter clockwise rotations around the axis of the superior mesenteric artery. During the tenth week of gestation, the midgut structures return into the abdomen, having undergone a total counter clockwise rotation of 270 degrees. During the final months of gestation, the cecum grows down into the right iliac fossa. The hindgut structures include the left one-third to one-half of the transverse colon, the descending colon, the sigmoid colon, the rectum, and the superior portion of the anal canal. Branches of the inferior mesenteric artery

supply all these hindgut structures (*John, 2006*).

Anatomy of the colon:

☉ **Introduction:**

The large intestine extends from the distal end of the ileum to the anus, and is 1.5m long, although there is considerable variation in its length. Its caliber is greatest near the cecum, and gradually diminishes to the level of mid rectum. It enlarges in the lower third of the rectum to form the rectal ampulla above the anal canal (*Jeremiah et al., 2005*).

The large intestine is divided into five segments. From proximal to distal, these segments are: right colon, transverse colon, left colon, sigmoid colon, and rectum (*Rolandelli and Roslyn, 2001*).

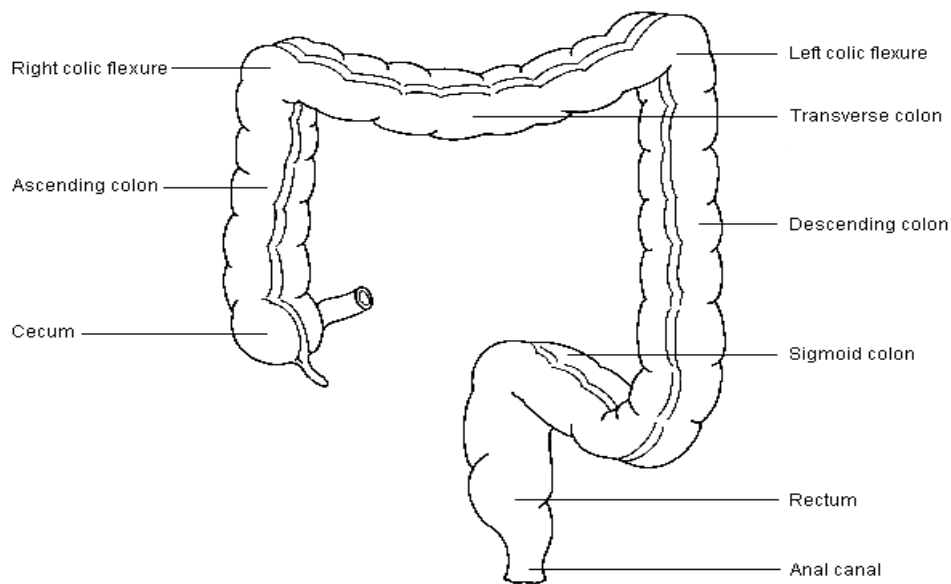


Figure (1): The various anatomic components of the colon. (*John F. Sweeney, 2006*).

Throughout its length there is an alternating pattern of fixed and mobile components:

- The cecum and the transverse and sigmoid colons possess considerable mobility.
- The ascending and descending colons are fixed to the posterior wall.

(Richard, 2001).

The large intestine differs from the small intestine in:

- It has a greater caliber.
- It is for the most part more fixed in position.
- Its longitudinal muscle, though a complete layer, is concentrated into three longitudinal bands, teniae coli.
- In all but the distal sigmoid colon and rectum; small adipose projections (appendices epiploicae) are scattered over the free surface of the whole colon (they tend to be absent from the cecum, vermiform appendix and rectum).
- Moreover, the colonic wall is puckered into sacculations (haustrations), which may, in part, be due to the presence of the teniae coli, and which may be demonstrated on plain radiographs as incomplete septations arising from the bowel wall.

The function of the large intestine is chiefly absorption of fluid and solutes (*Jeremiah et al., 2005*).

The locations of the teniae are useful landmarks and specific in relation to the position of the colon itself. The posterior teniae (teniae omental) is found on the posterolateral

border of the ascending and descending colon and the anterior border of the transverse colon; the anterior teniae (teniae libera) is visible on the exposed, or antimesenteric border of the cecum and the ascending, descending, and sigmoid colons, but is located on the inferior surface of the transverse colon and covered by the attachment of the greater omentum; the lateral teniae (teniae mesocolica) is located on the posteromedial side of the cecum and the ascending, descending, and sigmoid colons, and on the posterior border of the transverse colon at the attachment of the transverse mesocolon. Between the teniae coli the colon is sacculated, forming the haustra coli (**Richard, 2001**).

☉ **Cecum and appendix:**

The cecum is the saccular commencement of the colon. It is located in the right iliac fossa where it lies on the iliacus muscle cranial to the lateral half of the inguinal ligament. At times it may cross the pelvic brim to lie in the true pelvis. Anteriorly, it is usually in contact with the anterior abdominal wall. Superiorly, it is continuous with the ascending colon, at some point along its medial border the ileum enters it at the ileocecal ostium (**Richard, 2001**).

The ileocecal valve is a sphincter located at the junction of the terminal ileum and the cecum. This sphincter is the result of fusion of an upper and lower lip of circular muscle fibers from ileum and cecum, and it serves as a sphincter that prevents the reflux of luminal contents from the cecum back

into the terminal ileum (*Rolandelli and Roslyn, 2001*).

The cecum has no mesentery even though it is referred to as being an intraperitoneal structure in most individuals since it has a considerable amount of mobility (*Richard, 2001*).

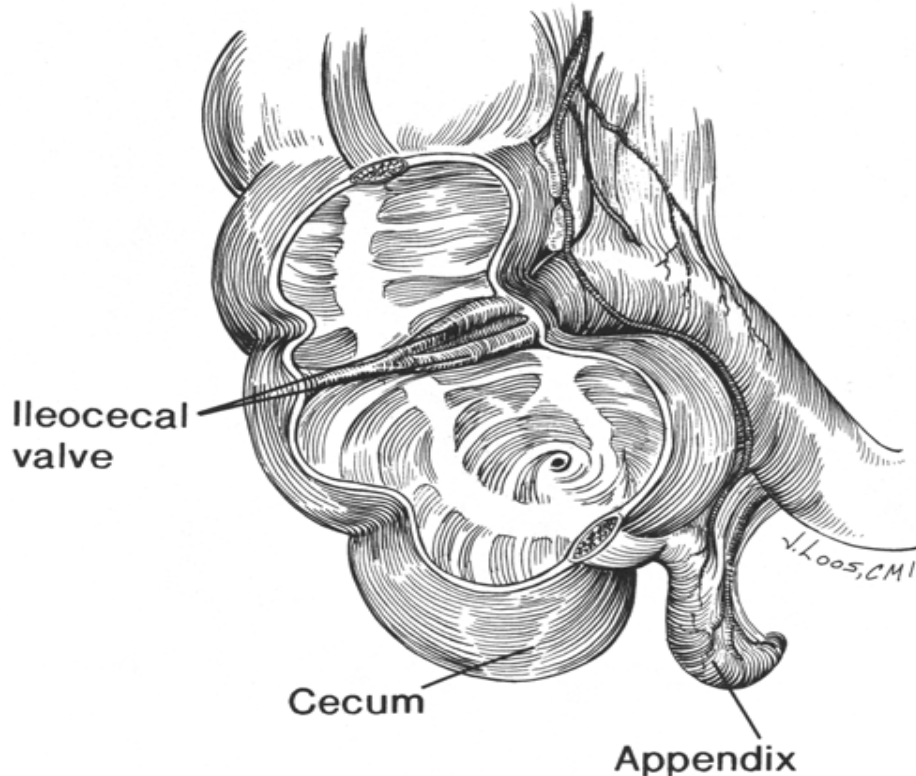


Figure (2): The cecum is opened to see the ileocecal valve (*Richard L. Drake, 2001*).

The vermiform (worm-shaped) appendix is a blind-ending tube varying in length (commonly about 6-9 cm), which opens into the posteromedial wall of the cecum 2 cm below the ileocaecal valve. On the surface of the abdomen this point (McBurney's) lies one-third of the way up the oblique line that joins the right anterior superior iliac spine to the umbilicus.

While the position of its base is constant in relation to the Cecum, the appendix itself may lie in a variety of positions. The most common, as found at operation, is the retrocaecal position, with the pelvic position next in order of frequency; but recent scanning studies suggest that a retroileal site is the most common in the absence of disease. The three teniae of the cecum merge into a complete longitudinal muscle layer for the appendix. The submucosa contains many lymphoid masses and the lumen is thereby irregularly narrowed. This lumen is wider in the young child and may be obliterated in old age. The appendix has its own short mesentery, the mesoappendix, which is a triangular fold of peritoneum from the left (inferior) layer of the mesentery of the terminal ileum. A small fold of peritoneum extends from the terminal ileum to the front of the mesoappendix. This is the ileocaecal fold (or bloodless fold of Treves, although it sometimes contains blood vessels) and the space between it and the mesoappendix is the inferior ileocaecal recess. Another fold lies in front of the terminal ileum, between the base of the mesentery and the anterior wall of the cecum. This fold is raised up by the contained anterior caecal artery and is called the vascular fold of the cecum. The space behind it is the superior ileocaecal recess (fossa of Luschka) (*Ghummy, 2005*).

⊙ **Ascending colon**

The ascending colon is about 13 cm long and lies in the right lower quadrant. It extends upward from the cecum to the

inferior surface of the right lobe of the liver and anterior to the right kidney, where it turns to the left, forming the right colic (hepatic) flexure. The peritoneum covers the front and the sides of the ascending colon, binding it to the posterior abdominal wall (**Richard, 2005**).

The upper part is covered interiorly by coils of small intestine, but the lower part may come into direct contact with the anterior abdominal wall. Posteriorly, the ascending colon is related to the lower pole of the right kidney and lies on the iliacus muscle and the aponeurotic origin of the transversus abdominis muscle. The kidney and branches of the lumbar plexus separate the ascending colon from the quadratus lumborum muscle. The iliohypogastric and the ilioinguinal nerves cross behind it (**Richard, 2005**).

Lateral to the ascending colon is the right paracolic sulcus, or gutter. This depression is formed by a reflection of the peritoneum after it crosses the ascending colon and before it continues onto the posterior abdominal wall. Material can move along this gutter from the appendix to the hepatorenal recess or from the liver into the pelvis, and surgeons can incise the peritoneum along this border to mobilize the ascending colon. The blood vessels and lymphatics lie in the retroperitoneal connective tissue in this area which, during development, composed the mesentery of the ascending colon. Thus, by mobilizing the ascending colon on this lateral avascular border and raising it towards the midline, the

retroperitoneal connective tissue containing the blood vessels and lymphatics can be lifted intact (*Richard, 2001*).

☉ **Mobilization of the fixed part of the right colon:**

The right colon comprises the cecum and the ascending colon and the hepatic flexure. It is largely a retroperitoneal structure and bound down to the right posterior abdominal wall and associated structures, the peritoneal reflection from the right colon onto the posterior abdominal wall is marked by line of **Toldt**, incision of the peritoneum along this line is a preliminary step in mobilizations (*Pemberton, 2000*).

☉ **Transverse colon:**

The transverse colon is 50 cm long, and extends from the hepatic flexure in the right lumbar region across into the left hypochondrial region, where it curves posteroinferiorly below the spleen as the splenic flexure. It is highly variable in length and position, as may be confirmed by radiological assessment, but it often describes an inverted arch, with its concavity directed posteriorly and superiorly. The posterior surface at the hepatic flexure is devoid of peritoneum, and is attached by loose connective tissue to the front of the descending part of the duodenum and the head of the pancreas. The transverse colon from here to the splenic flexure is almost completely invested by peritoneum, and is suspended from the anterior border of the body of the pancreas by the transverse mesocolon. The latter is attached from the inferior part of the right kidney, across the second part of the duodenum and

pancreas, to the inferior pole of the left kidney. The transverse colon hangs down between the flexures to a variable extent, and sometimes reaches the pelvis. Above it, are the liver and gall bladder, the greater curvature of the stomach and the body of the spleen. The transverse colon is usually attached to the greater curvature of the stomach by, the gastrocolic ligament, which is in continuity with the greater omentum, lying anteriorly and extending inferiorly. Behind and below the transverse colon lie the descending part of the duodenum, the head of the pancreas, the upper end of the small bowel mesentery, the duodenojejunal flexure and loops of the jejunum and ileum (*Jeremiah, 2005*).

The left colic (or splenic) flexure is higher and situated more posteriorly than the right colic flexure. It is usually in contact with the spleen, the greater curvature of the stomach, the tail of the pancreas, and the anterior surface of the left kidney. In addition, the splenic flexure is suspended from the diaphragm by the left phrenicocolic ligament, a distinct peritoneal fold. And it lies more superiorly, and posteriorly than the right hepatic flexure (*Richard, 2001*).

☉ **Descending colon:**

The descending colon is 25 cm long. It descends through, the left hypochondrial and lumbar regions, initially following the lateral border of the lower pole of the left kidney and then descending in the angle between psoas major and quadratus

lumborum to the iliac crest. It then curves inferomedially, lying anterior to iliacus and psoas major, to become the sigmoid colon at the inlet of the lesser pelvis. It is a retroperitoneal structure covered interiorly and on both sides by peritoneum. Its posterior surface is separated by loose connective tissue from the anterior perirenal fascia inferolateral to the left kidney, the aponeurosis of transversus abdominis, quadratus lumborum, iliacus and psoas major. The subcostal vessels and nerves, iliohypogastric and ilioinguinal nerves, fourth lumbar artery (usually); the lateral femoral cutaneous, femoral and genitofemoral nerves, the gonadal vessels, and the external iliac artery all pass behind the descending colon. Loops of jejunum lie anteriorly: if the anterior abdominal wall is relaxed, the most inferior part of the descending colon may be directly palpated transabdominally. The descending colon is smaller in caliber, more deeply placed, and more frequently covered posteriorly by peritoneum, than the ascending colon (*Jeremiah, 2005*).

⊙ **Sigmoid colon:**

At or below the crest of the ilium, the colon acquires a mesentery and becomes the sigmoid colon. This section of the colon continues inferiorly until it loses its mesentery, usually anterior to 3rd sacral vertebra (S3), and becomes the rectum. This junction is indicated by a slight constriction caused by a functional rectosigmoid sphincter that controls the passage of the contents of the colon into the rectum. Throughout its length the sigmoid colon is suspended by a mesentery, the sigmoid

mesocolon, whose inverted V-shaped attachment is to the pelvic brim and the posterior wall of the pelvis. The sigmoid colon is quite variable in length and is separated from the bladder in the male and the uterus in the female by coils of the small intestine (*Richard, 2001*).

⊙ **Mobilization of the fixed part of the left Colon:**

The splenic flexure lies at a higher level than the hepatic flexure, generally tucked well up under the left costal margin and closely related to the spleen. The splenic flexure is attached to the diaphragm posterolaterally by the phrenocolic ligament, which appears as a band of thickened peritoneum running below the lower pole of the spleen. The splenic flexure may be free of the spleen or attached by adhesions to the splenic capsule. Before mobilization these adhesions should be freed with cautery to avoid a capsular tear working with fingers simultaneously, medially from the lesser sac and laterally under phrenocolic ligament, the attachments of the splenic flexure are defined. Although there are no named vessels in this attachment, it is often wise to divide between clamps (*Pemberton, 2000*).

⊙ **Vascular supply:**

The vascular supply to the colon is through the superior mesenteric artery (SMA), (the second ventral branch of the abdominal aorta), and the inferior mesenteric artery (IMA), (the third ventral branch of the abdominal aorta) (*Richard, 2001*).