A DISSERTATION ON

"A PROSPECTIVE STUDY ON MANAGEMENT OF ACUTE INTESTINAL OBSTRUCTION AND ITS OUTCOME IN ADULTS IN GOVT .RAJAJI HOSPITAL, MADURAI"

M.S. GENERAL SURGERY

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CERTIFICATE

This is to certify that this Dissertation titled "**A PROSPECTIVE STUDY ON MANAGEMENT OF ACUTE INTESTINAL OBSTRUCTION AND ITS OUTCOME IN ADULTS IN GOVT RAJAJI HOSPITAL, MADURAI"** submitted by **Dr.M.DURAI RAJ KUMAR** to the faculty of general surgery, The Tamilnadu Dr.M.G.R Medical University, Chennai in partial fulfilment of the requirement for the award of MS Degree (Branch I) General Surgery, is a bonafide research work carried out by him under our direct supervision and guidance from May 2018 to May 2019.

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This is to certify that this dissertation entitled "**A PROSPECTIVE STUDY ON MANAGEMENT OF ACUTE INTESTINAL OBSTRUCTION AND ITS OUTCOME IN ADULTS IN GOVT RAJAJI HOSPITAL, MADURAI"** is the bonafide work of **Dr. M. DURAI RAJ KUMAR** in partial fulfilment of the university regulations of the Tamil Nadu Dr.M.G.R.Medical University, Chennai, for **M.S.General Surgery Branch I** examination to be held in May 2020.

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DECLARATION BY THE CANDIDATE

I **Dr. M. DURAI RAJ KUMAR**, hereby solemnly declare that this Dissertation entitled "**A PROSPECTIVE STUDY ON MANAGEMENT OF ACUTE INTESTINAL OBSTRUCTION AND ITS OUTCOME IN ADULTS IN GOVT .RAJAJI HOSPITAL, MADURAI"** is a bonafide and genuine research work carried out by me. This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the regulations for the award of M.S Degree (Branch I) in General Surgery.

Place: Madurai
Date: -10-2019 (**Dr. M. DURAI RAJ KUMAR**)

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Date: -10-2019 (**Dr. M.DURAI RAJ KUMAR**)

CONTENTS

INTESTINAL OBSTRUCTION

INTRODUCTION[1]

The diagnosis and treatment of acute intestinal obstruction can be one of the most challenging tasks facing the general or specialist surgeon. Both differential diagnosis and timing of intervention may differ significantly, depending upon whether or not the patient has had previous abdominal surgery as compared with patients who have not had prior abdominal surgery. Generally speaking, postoperative adhesions have long been the most common cause for small bowel obstruction; however, this may change with the increasing use of minimally invasive surgery and the presumed lesser incidence of observed postoperative adhesions associated with minimal access surgery. Along with this minimally invasive surgery, we are also seeing an increase in the incidence of small bowel obstruction due to internal hernias. The old adage “the sun never sets on a small bowel

Obstruction” may still apply, but only in select cases, those with signs of closed loop obstruction or vascular compromise. Discerning this, and differentiating those patients, in whom watchful waiting is in order and those in whom prompt intervention is required, is the surgeon’s priority

AIMS AND OBJECTIVES

Aim:

To study the Outcome of patients diagnosed with Acute Intestinal Obstruction, managed surgically in Govt.Rajaji Hospital, Madurai.

Objectives of the study:

- To Estimate the burden of Acute Intestinal Obstruction in GRH, Madurai.
- To Surgically manage the patients of Acute Intestinal Obstruction depending on the Etiology
- To Analyze the outcomes and the Complications associated with the same

REVIEW OF LITERATURE

HISTORY

Sushruta -6th Century B.C. -Wrote oldest known descriptions of bowel surgery. Described using a cautery over the swelling of strangulated hernias. Used the mandibles of black ants to clamp the edges of bowel wounds together. [2]

Hippocrates (460-370 B.C.) – Argued against surgical treatment of the abdomen.

Provided a detailed description of intestinal obstruction: **"In ileus, the belly becomes hard, there are no motions; the whole abdomen is painful, there are fever and thirst and sometimes the patient is so tormented that he vomits bile.** [2]

Le Peyronie 1723

Removed gangrenous bowel from a man with intestinal obstruction. Brought two loops out into the wound to serve as an artificial anus.

Placing traction on a suture placed in the mesentery between the two loops to quickly heal the fistula. [2]

Meckel 1781-1833 -Described diverticulum iliei verum, also known as Meckel's diverticulum. [2]

Treves 1899

After winning the 1883 Jacksonian Prize of the Royal College of Surgeons for a thesis regarding the benefits of operative management of intestinal obstruction, he wrote in 1899: **"It is less dangerous to leap from the Clifton Suspension Bridge than to suffer from acute intestinal obstruction and decline operation."** His work stimulated a movement toward the modern era of surgical management of intestinal obstruction. [2]

In 1884, he published a detailed discussion of the etiologies (including adhesions) and surgical management of mechanical intestinal obstruction.

Treves also distinguished mechanical from nonmechanical (i.e., paralytic) causes of intestinal distention, classifying the latter causes under the term ileus.

From 1880 to 1925, proximal intestinal decompression was recognized to provide relief from the symptoms of mechanical obstruction or ileus. [7], [8]

In **1933, Wangensteen and Paine** reported the efficacy of gastrointestinal intubation in relieving symptoms of intestinal distention caused by intestinal obstruction or from the ileus that resulted from laparotomy

Subsequently, Wangensteen and Rea provided experimental evidence that the source of gaseous distention in cases of obstruction or ileus was swallowed air. [5], [6]

The value of intravenous fluid resuscitation in experimental models of intestinal obstruction was recognized as early as 1912

By 1920, plain abdominal radiographs were used in diagnosis of intestinal obstruction

Thus, the principles of early diagnosis, rapid intravenous fluid resuscitation, gastrointestinal decompression, and early operation to avoid intestinal gangrene and peritonitis, were established well before the advent of antibiotic therapy, invasive hemodynamic monitoring, and
Parenteral nutrition.

These early developments were most important in reducing morbidity and mortality of mechanical intestinal obstruction and ileus.

EMBRYOLOGY [2]

The distal foregut and the proximal midgut are responsible for the genesis of the three parts of the small bowel (duodenum, jejunum, and ileum).

The approximate junction of the distal foregut and proximal midgut lies just distal to the ampulla of Vater in the adult.

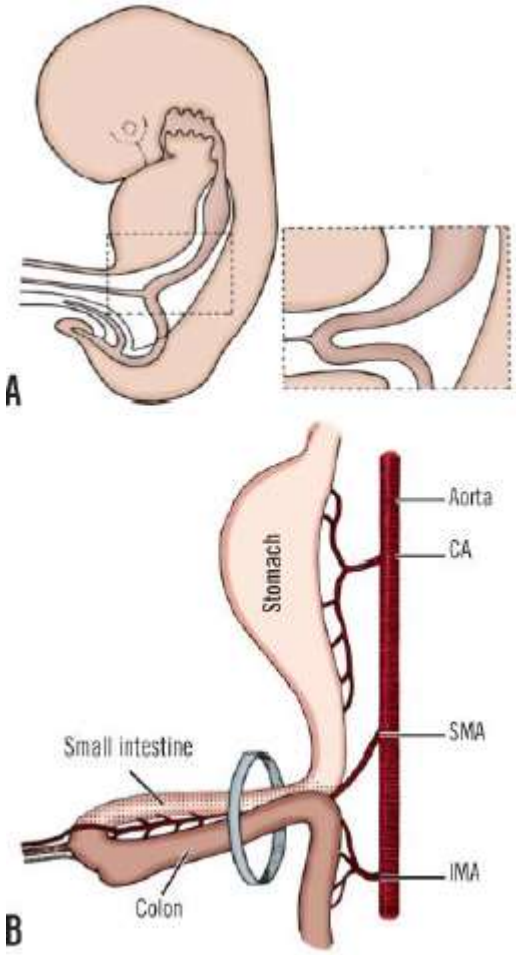
The demarcation of the small bowel into three parts takes place by the start of the third week of embryonic life

The position of the duodenum posterior to the superior mesenteric artery is the result of the normal development and rotation of the embryonic gut.

Early in the second month of gestation, the intestines, which elongate faster than the abdominal cavity expands, push a loop out into the umbilical cord.

This is the "midgut" of the embryologist, not the "midgut" of the surgeon.

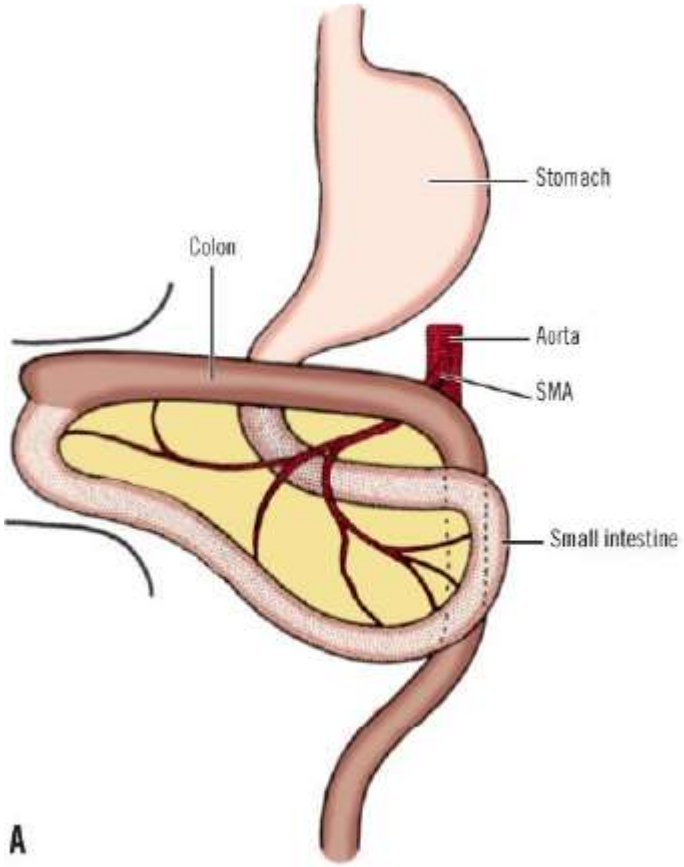
The herniated segment extends from approximately the distal one-third of the duodenum through the proximal one-third of the transverse colon. It is supplied by branches of the superior mesenteric artery. The axis of this herniation is the superior mesenteric artery. This artery, together with the celiac axis and the inferior mesenteric artery, is a remnant of the arterial side of the primitive vitelline circulation to the yolk sac. Originally paired and segmentally arranged, the pairs of arteries fuse, and their number is reduced to three by the sixth week of development. At this stage, the superior mesenteric artery continues past the intestine to supply the vitelline stalk, which occasionally persists as Meckel's Diverticulum.

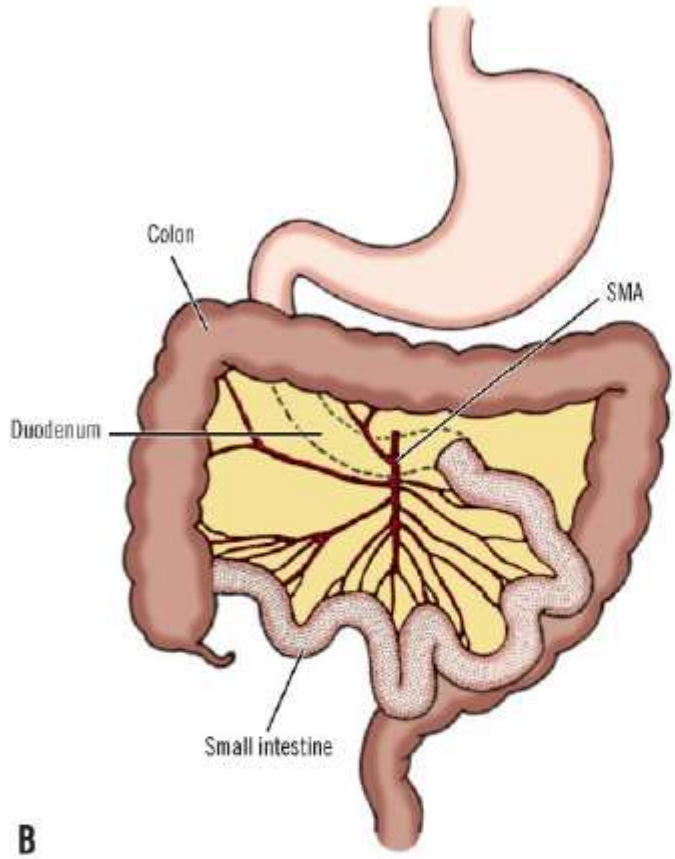


[2]

Rotation of the intestinal loop counter clockwise through 90° brings the future duodenum and proximal small intestine to the right of the future colon. The axis of this rotation is the superior mesenteric artery. The intestines continue to elongate in the umbilical cord. In the tenth week, they rather suddenly return to the abdomen. The cranial limb of the intestinal loop returns first, so that the duodenum passes behind the superior mesenteric artery.

The caudal limb, which will form the distal ileum and the entire colon, returns later, bringing the transverse colon in front of the artery and the duodenum by a further 180° counter clockwise rotation





A. Return of the intestines to the abdomen in the tenth week. The prearterial *(stippled)* limb returns first, passing behind the superior mesenteric artery. B. Final position of the intestines attained shortly after birth. *SMA*, superior mesenteric artery. (Modified from Gray SW, Akin JT Jr, Milsap JH Jr, Skandalakis JE. Vascular compression of the duodenum. (Part 1). Contemp Surg 9:37, 1976; with permission.)

[2]

The first part of the duodenum retains both dorsal and ventral mesentery. However, during the rotation, the duodenal loop is fixed in the retroperitoneal space. Therefore, the dorsal mesentery of the rest of the duodenum disappears.

The "disappearing" dorsal duodenal mesentery remains as an avascular plane of loose connective tissue (the fascia of Treitz). It is not related to the ligament of Treitz.

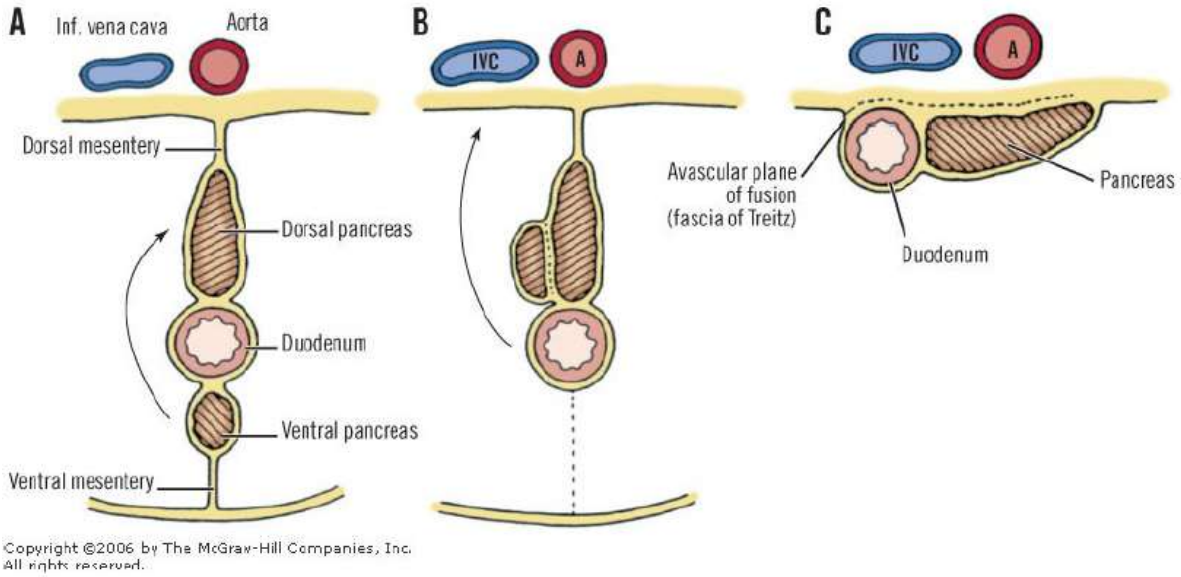


Diagram of the rotation of pancreas and duodenum. **A.** Primitive relation of dorsal and ventral pancreatic primordia. **B.** Disappearance of ventral mesentery and rotation of ventral pancreas. **C.** Final retroperitoneal position of duodenum and pancreas. The plane of fusion of the mesoduodenum is the avascular fascia of Treitz. (Modified from Gray SW, Colborn GL, Pemberton

[2]

During the third and fourth weeks, the embryo grows rapidly, but the yolk sac and open midgut do not. By the fifth week, the foregut is as large as the opening of the midgut, which may then be called the *yolk stalk,* the *vitelline duct,* or the *omphalomesenteric duct.* At this time, a ventral swelling of the midgut just caudal to the yolk stalk marks the site of the cecum, and hence, the boundary between the small and large intestine.

Elongation of the midgut, especially of the portion between the yolk stalk and the duodenum, proceeds faster than elongation of the whole body of the embryo.

The result of this growth differential is a series of movements that ends with the adult position of the intestines in the abdomen. These movements occur in three well-defined stages that will be described only briefly here

Stage 1: Herniation

The midportion of the growing intestine buckles ventrally and protrudes into the coelom of the body stalk in the fifth week. The apex of the protrusion is marked by the yolk stalk. Its axis is marked by the superior mesenteric artery, which represents part of the primitive blood supply to the yolk sac. This loop of intestine undergoes a counter clockwise twist of 90°, so that the "prearterial" (cranial) limb lies to the right of the post arterial (caudal) limb. The caudal limb remains nearly straight, while the cranial limb grows rapidly and is thrown into coils.

Stage 2: Return (Reduction)

The intestines return to the abdomen rather suddenly during the tenth week. The cranial limb enters first, to the right of the superior mesenteric artery. The caudal loop enters later: the left colon first; the transverse colon in front of the superior mesenteric artery; and lastly, the cecum with the terminal ileum.

Stage 3: Fixation

From the fourth month until well after birth, the growth of the colon is completed. The mesenteries of the ascending and descending portions become obliterated by fusion with the peritoneum of the body wall. The transverse mesocolon fuses with the posterior leaf of the omental bursa.

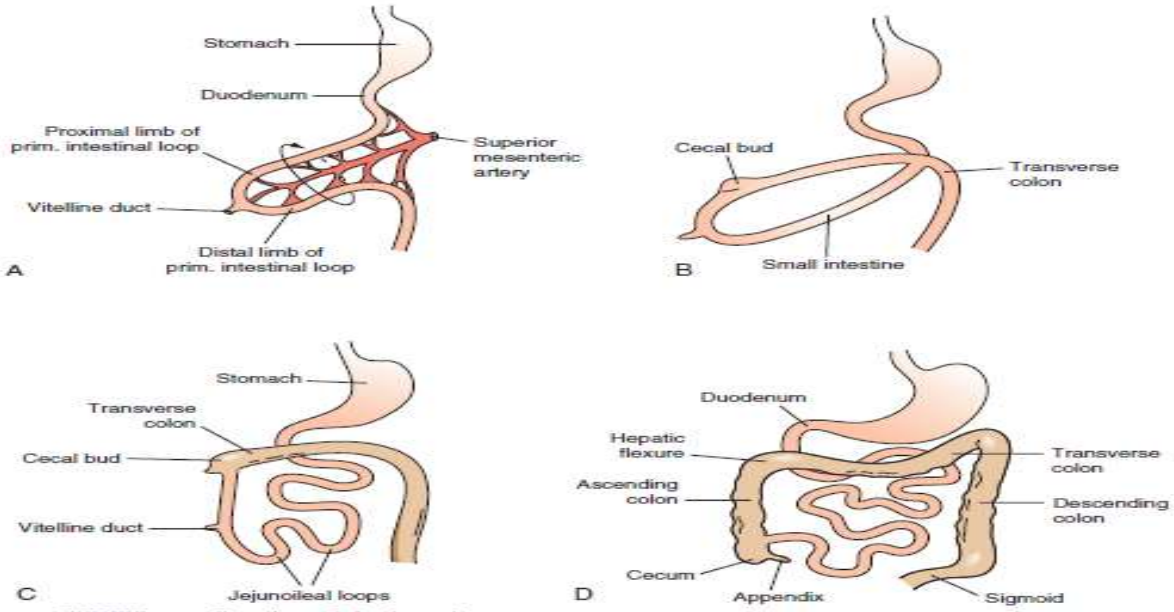


FIGURE 49-1 Rotation of the Intestine. A, The intestine after a 90-degree rotation around the axis of the superior mesenteric artery, the proximal loop on the right and the distal loop on the left. **B,** The intestinal loop after a further 180-degree rotation. The transverse colon passes in front of the duodenum. **C,** Position of the intestinal loops after reentry into the abdominal cavity. Note the elongation of the small intestine, with formation of the small intestine loops. **D,** Final position of the intestines after descent of the cecum into the right iliac fossa. (From Podolsky DK, Babyatshy MW: Growth and development of the gastrointestinal tract. In Yamada T, editor: *Textbook of gastroenterology* (vol 2). Philadelphia, 1995, JB Lippincott.)

[2]

ANATOMY

DUODENUM [3]

- The duodenum comprises the **first portion of the small intestine** and plays an important role in connecting the foregut organs to the midgut.

- It anatomically **begins at the duodenal bulb which is immediately distal to the pylorus and terminates at the ligament of Treitz,** where it joins the jejunum.

- The duodenum is approximately 20 to 30 cm in length and is divided into four distinct areas.

- **The first portion of the duodenum** is approximately 5 cm in length and is referred to as the bulb or cap. This area is directly attached to the pylorus and extends laterally and cephalad. It serves as an attachment for the hepatoduodenal ligament and traverses over the common bile duct, portal vein, pancreatic head, and the gastro duodenal artery. The mucosal surface of the duodenal bulb is smooth until its junction with the second portion of the duodenum, where the concentric Kerckring folds begin.
- This portion of the duodenum is prone to ulceration, with approximately 90% of duodenal ulcers occurring here. Unfortunately, due to its anatomic positioning, these ulcers may erode into the gastro duodenal artery which lies directly posterior, causing potentially life-threatening bleeding.

- **The second portion of the duodenum** (descending duodenum) extends from the origin of the Kerckring folds to the beginning of the transverse duodenum and travels over the right renal vasculature,

the medial aspect of Gerota fascia, the inferior vena cava, and to the right of the L1 and L2 vertebra. It is approximately 10 cm in length and 3 to 5 cm in diameter.

- **The Kerckring folds** (plicae circulares) are concentric mucosal folds which are 1 to 2 mm thick and 2 to 4 mm high and are separated by 2 to 4 mm of smooth, flat mucosa.
- This portion of the duodenum serves as an entry point for pancreatic and biliary secretions into the gastrointestinal tract. This is typically through the major papilla (ampulla of Vater), which is a valvular structure arising in the midportion of the descending duodenum, approximately 7 to 10 cm from the pylorus. Through this point, the confluence of the common bile duct and the main pancreatic duct (duct of Wirsung) join the duodenum. The valvular function of the papilla is regulated through the muscular sphincter of Oddi. The minor pancreatic duct (duct of Santorini) enters through the minor papilla proximal to the ampulla of Vater in 50% to 60% of patients and endoscopically appears as a 1- to 3-mm polypoid structure.
- **The second portion of the duodenum** is important surgically as it represents the entry of the duodenum into the retroperitoneum.

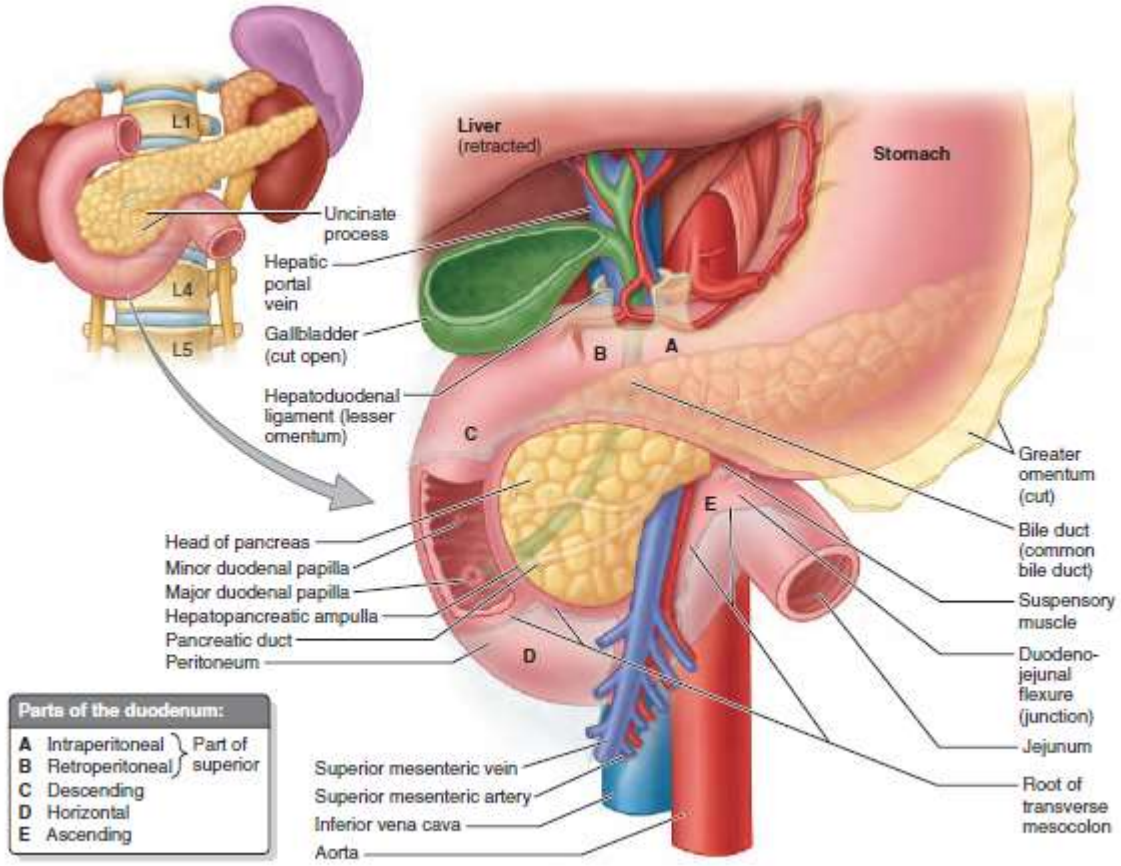


FIGURE 2.45. Relationships of duodenum. The duodenum pursues a C-shaped course around the head of the pancreas.

[3]

- Surgical evaluation of this part of the duodenum requires Mobilization from its posterior and lateral attachments, described as a Kocher manoeuvre. This allows for further evaluation of the duodenum, pancreatic head, and bile duct.

- **The third (transverse) and fourth (ascending)** portions of the duodenum complete the duodenal sweep.

- **The third portion of the duodenum** is about 10 cm in length and courses transversely from right to left, crossing the midline anterior to the spine, aorta, and inferior vena cava. This portion is closely Attached to the uncinate process of the pancreas. The superior mesenteric artery (SMA) and vein (SMV) course anterior to the third portion of the duodenum to provide blood supply to the gut.

- The transition between the third and fourth portions of the duodenum is marked by the passage of the SMA in front of the duodenum. The SMA forms an acute angle as it originates from the aorta. An abnormally narrow angle can result in obstruction of the duodenum at this location (SMA syndrome).

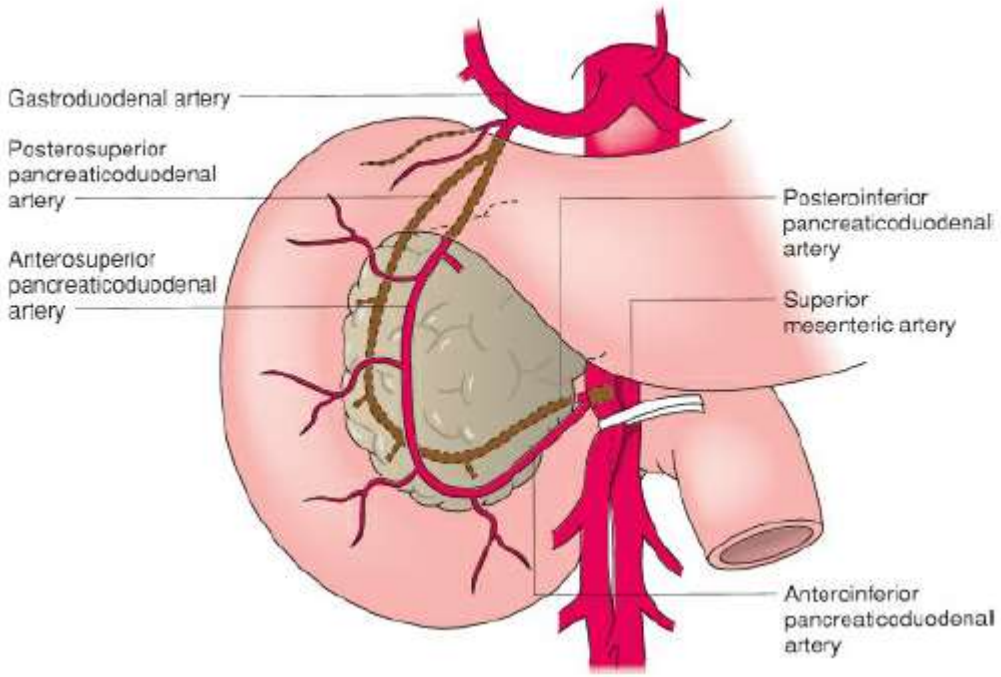
- **The fourth portion of the duodenum** is approximately 5-cm long and courses upward and obliquely to reach the ligament of Treitz, marking the end of the duodenum and the return of the small bowel to the peritoneal cavity.

TABLE 2.8. RELATIONSHIPS OF DUODENUM

Part of Duodenum	Anterior	Posterior	Medial	Superior	Inferior	Vertebral Level
Superior (1st part) **(A & B)**	Peritoneum Gallbladder Quadrate lobe of liver	Bile duct Gastroduodenal artery Hepatic portal vein IVC	Pylorus	Neck of gallbladder	Neck of pancreas	Anterolateral to L1 vertebra
Descending (2nd part) **(C)**	Transverse colon Transverse mesocolon Coils of small intestine	Hilum of right kidney Renal vessels Ureter Psoas major	Head of pancreas Pancreatic duct Bile duct	Superior part of duodenum	Inferior part of duodenum	Right of L2–L3 vertebrae
Inferior (horizontal) (3rd part) **(D)**	SMA SMV Coils of small intestine	Right psoas major IVC Aorta Right ureter		Head and uncinate Process of pancreas Superior mesenteric vessels	Coils of small intestine (ilium)	Anterior to L3 vertebra
Ascending (4th part) **(E)**	Beginning of root of mesentery Coils of jejunum	Left psoas major Left margin of aorta	SMA, SMV, uncinate process of pancreas	Body of pancreas	Coils of jejunum	Left of L3 vertebra

IVC, inferior vena cava; *SMA*, superior mesenteric artery; *SMV*, superior mesenteric vein.

[4]



Arterial blood supply to the duodenum. [3]

- Following its embryologic origins, the vascular supply to the duodenum arises from branches of the celiac trunk for the foregut portion, whereas the distal (midgut origin) duodenum is supplied By branches of the SMA

Venous drainage includes a series of pancreatic duodenal veins which Drain into the SMV–portal vein system. Lymphatic drainage follows the vascular supply with drainage to the pancreatic duodenal nodes. From here, lymph drains superiorly to the hepatic nodes or inferiorly to the superior mesenteric nodes.

JEJUNUM AND ILEUM [3]

- Distal to the ligament of Treitz, the jejunum and ileum form the remainder of the small intestine.
- The boundary between the two is arbitrarily determined such that 40% of the intraperitoneal small intestine comprises jejunum and 60% comprises ileum.

- This portion of the bowel is suspended within the peritoneal cavity by a thin, broad-based mesentery that is attached to the posterior abdominal wall.
- The jejunum and ileum are freely mobile within the peritoneal cavity.
- The jejunum is the widest portion of the small intestine, whose calibre progressively decreases as it approaches the ileocecal valve.
- The mucosa of the jejunum has a thick lining and is characterized by prominent plicae circulares that become shorter and less frequent in the ileum.
- The total length of jejunum and ileum varies, but is usually between 5 and 7 m in length.
- The small intestine terminates in the right lower quadrant at the ileocecal valve.
- The ileocecal valve exhibits motor characteristics separate from the terminal ileum and colon, postulated to prevent reflux of fecal material from the colon into the small intestine.

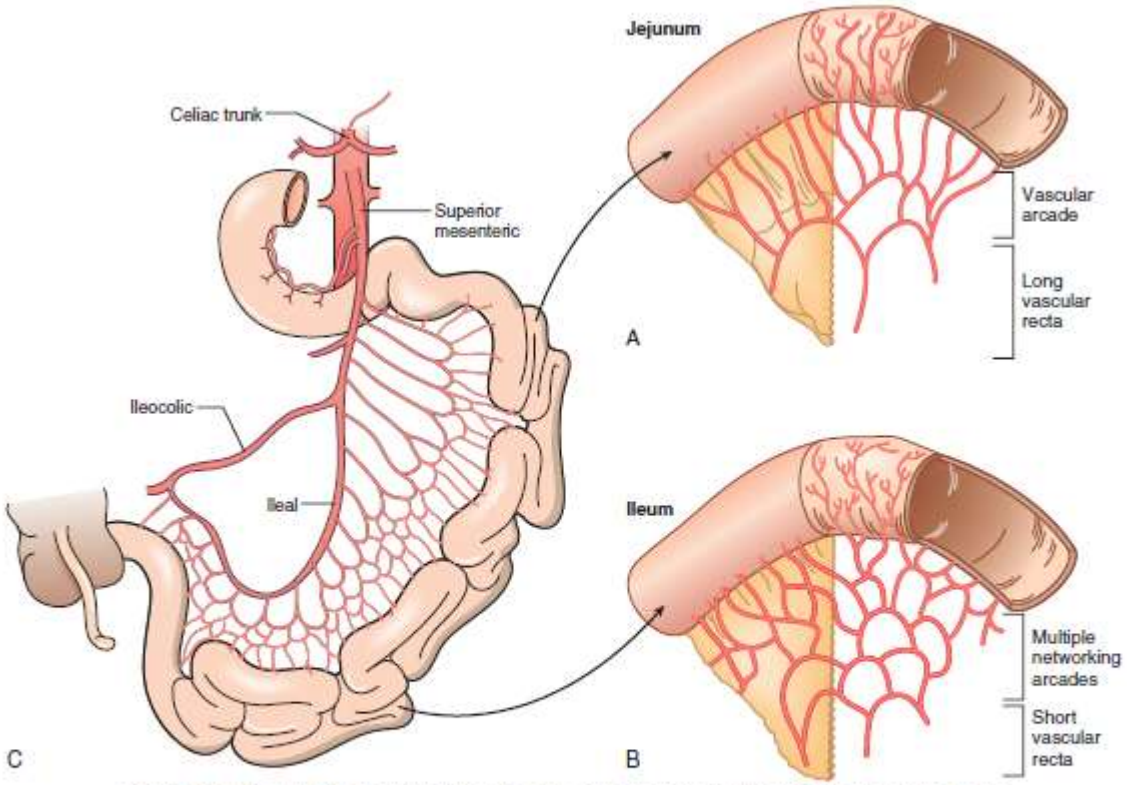


FIGURE 49-2 **A,** The jejunal mesenteric vessels form only one or two arcades with long vasa recta. **B,** The mesenteric vessels of the ileum form multiple vascular arcades with short vasa recta. **C,** The superior mesenteric artery, which courses anterior to the third portion of the duodenum, provides blood supply to the jejunoileum and distal duodenum. The celiac artery supplies the proximal duodenum. (Adapted from Keljo DJ, Gariepy CE: Anatomy, histology, embryology, and developmental anomalies of the small and large intestine. In Feldman M, Scharschmidt BF, Sleisenger MH, editors: *Sleisenger and Fordtran's gastrointestinal and liver disease: Pathology, diagnosis, management,* Philadelphia, 2002, WB Saunders, p 1646; illustration courtesy Matt Hazzard, University of Kentucky Medical Center, Lexington, KY.)

[4]

- The arterial blood supply of the jejunum and the ileum arises from the SMA.
- The main vascular branches form arcades within the mesentery. The vasa recta, intestinal arterial branches, enter into the intestinal wall without anastamosing. The vasa recta of the jejunum are straight and long, in contrast to the vasa recta of the ileum, which are shorter with greater arborisation

- The venous and lymphatic drainage follow the arterial supply. The main venous outflow is through the SMV which, along with the splenic vein, becomes the portal vein.

COLON [4]

The colon begins in the right lower quadrant of the abdomen as the cecum. The ileum enters the colon at the posteromedial aspect at the ileocecal valve.

Characteristics unique to the colon are

(a) taeniae coli

(b) haustra

(c) appendices epiploicae, located on the antimesenteric surface of the colon.

- There are three taeniae (anterior, posterior medial, and posterior lateral), which are condensations of the outer longitudinal muscle layer in the colon.
- They are named according to their attachments: taenia mesocolica (attached to the mesocolon), taenia omentalis (attached to the greater omentum), taenia libera (no attachments).

- The taeniae originate at the base of the appendix, course along the length of the colon, and then converge at the recto sigmoid junction.
- On average, the colon is 150 cm long.
- The taenia is one-sixth shorter than the colon and is believed to be responsible for pockets of the colon wall called sacculations or haustra.
- The epiploicae appendices are fat appendages seen on the colonic serosa.
- The colon begins in the right lower quadrant with the cecum.
- The cecum extends approximately 6 to 8 cm below the ileocecal valve (where the terminal ileum enters the posteromedial aspect of the cecum)
- The angulation between the ileum and cecum via the superior and inferior ileocecal ligaments is important in maintaining competence against reflux at the ileocecal junction.
- The cecum is the widest portion of the colon (7.5 to 8.5 cm in diameter), has the thinnest wall, and is entirely enveloped by peritoneum.
- The appendix originates from the lowest portion of the cecum and can be readily identified by following the converging taeniae. In

85% to 95% of people, the appendix lies posterior to the cecum, lateral and in line to the terminal ileum, but the position can vary, with the most frequent variants being retrocecal (toward the psoas muscle), pelvic, and retroileal.

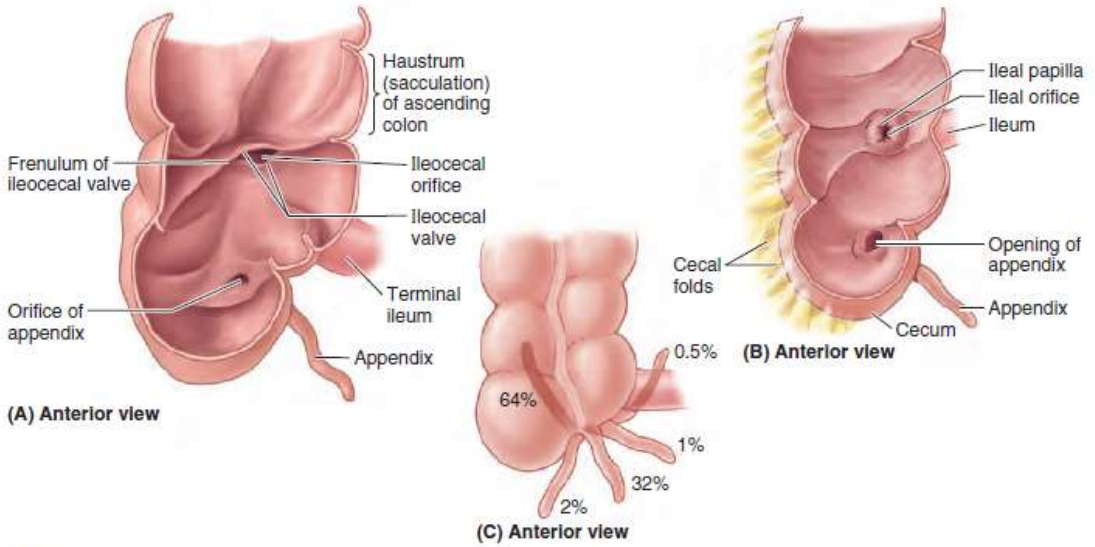


FIGURE 2.53. Terminal ileum, cecum, and appendix. A. The cecum was filled with air until dry and then opened. Observe the ileocecal valve and ileal orifice. The frenulum is a fold (more evident in cadavers) that runs from the ileocecal valve along the wall at the junction of the cecum and ascending colc **B.** The interior of the cecum showing the endoscopic (living) appearance of the ileocecal valve. **C.** The approximate incidences of various locations of the appendix, based on an analysis of 10,000 cases, are shown.

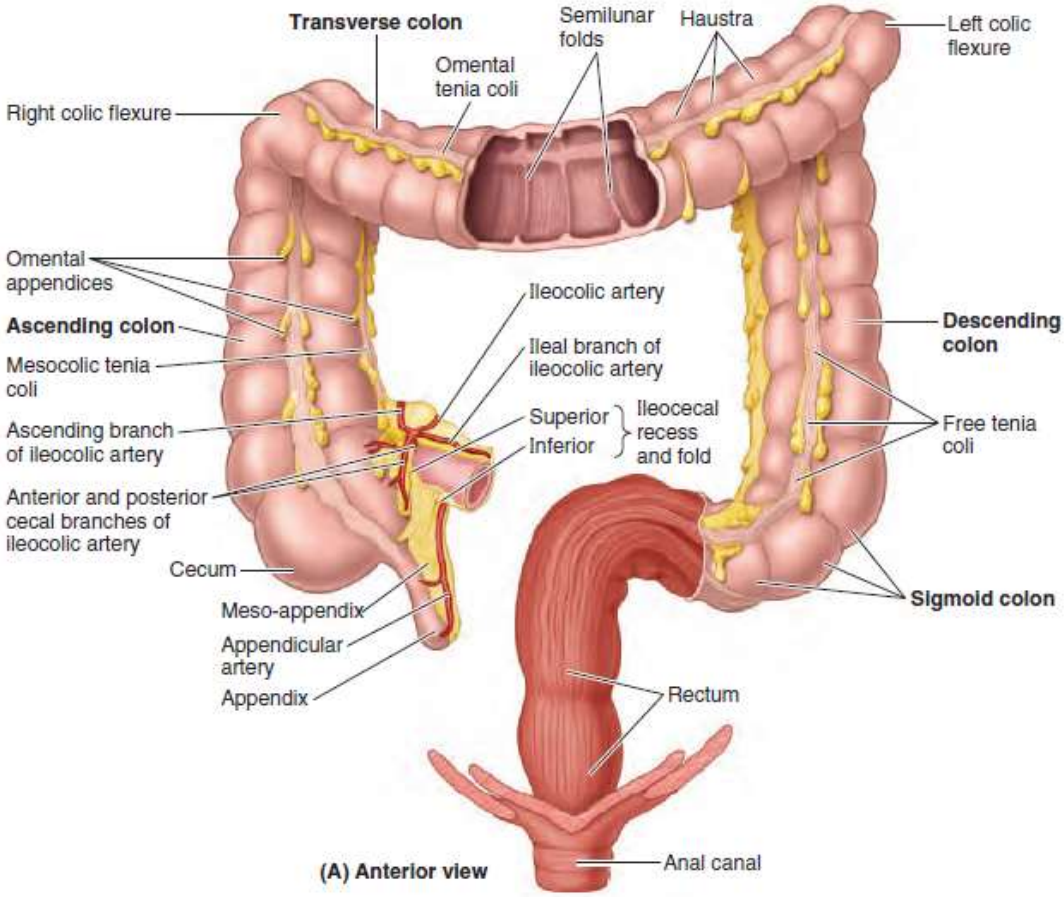
[4]

- During colonoscopy, visualization of the appendiceal orifice and ileocecal valve is the landmarks required in a complete colonic examination.

ASCENDING COLON

- From the cecum, the right colon ascends to the hepatic flexure (approximately 15 cm)

- The hepatic flexure is anterior to the inferior pole of the right kidney and overlies the second portion of the duodenum.
- The hepatic flexure is marked by medial, anterior, and downward angulation of the colon.
- When the right colon and mesentery are mobilized during a colectomy, care must be taken to avoid injury to the underlying duodenum. Only the anterior surface of the right colon is invested with peritoneum; laterally, the white line of Toldt marks the extent of the peritoneal covering and serves as an important landmark during surgical mobilization of the colon.



[4]

TRANSVERSE COLON

- The transverse colon stretches from the hepatic flexure to the splenic flexure and is the longest segment of colon (between 30 cm and 60 cm).
- The transverse colon is suspended by the transverse mesocolon and is completely intraperitoneal.
- It is the most mobile portion of the colon and may descend to the level of the iliac crests or deep into the pelvis. The greater omentum

descends from the greater curve of the stomach in front of the transverse colon and then ascends to attach to the transverse colon on its anterosuperior edge.

- To mobilize the transverse colon or enter the lesser sac, the fusion plane of the omentum to the transverse colon must be dissected. The splenic flexure is situated high in the left upper quadrant, more cephalad than the hepatic flexure, and lies anterior to the mid-left kidney and abuts the lower pole of the spleen.
- There are attachments from the colon to the diaphragm at the level of the 10th and 11th ribs and spleen (phrenocolic and splenocolic ligaments), and these must be carefully divided during mobilization of the splenic flexure to avoid splenic injury.
- The descending colon is approximately 25 cm long and courses from the splenic flexure to its junction with the sigmoid colon at the pelvic brim.
- It lies anterior to the left kidney and, like the right colon; the anterior, lateral, and medial portions of the descending colon are covered by peritoneum.

SIGMOID COLON

- The sigmoid colon extends from the pelvic brim to the sacral promontory, where it continues as the rectum and generally measures 15 to 50 cm in length. It is completely invested by peritoneum. The recto sigmoid junction is marked by the convergence of the colonic taenia. The sigmoid colon is extremely mobile and has a generous mesentery that extends along the pelvic brim from the iliac fossa across the sacroiliac joint to the second or third sacral segment.
- Because of its mobile mesentery, the sigmoid colon can twist and cause an obstruction, termed sigmoid volvulus.
- The left ureter runs in the intersigmoid fossa, which is at the base of the mesosigmoid.
- When a high ligation of the inferior mesenteric artery is performed during a cancer operation or the sigmoid colon is being mobilized along the white line of Toldt, the left ureter should be identified to avoid inadvertent injury.
- Preoperative placement of urinary stents can be useful for locating the ureter intraoperatively in complex pelvic surgery

RECTUM

- The rectum is 12 cm long and is continuous with the sigmoid colon at S3.
- The human rectum follows the posterior concavity of the sacrum and shows three lateral curves or flexures that are most prominent when the viscus distended.
- upper and lower curves convex to the right and a middle curve convex to the left the lowest part is slightly dilate as the rectal ampulla.

- It ends 2-3 cm in front and below the tip of the coccyx, turning abruptly downwards and backwards through levator ani muscle to become the anal canal 4 cm from the anal verge.

Blood Supply to Intestines [4]

TABLE 2.10. ARTERIAL SUPPLY TO INTESTINES

Artery	Origin	Course	Distribution
Superior mesenteric	Abdominal aorta	Runs in root of mesentery to ileocecal junction	Part of gastrointestinal tract derived from midgut
Intestinal (jejunal and ileal) (n =15–18)	Superior mesenteric artery	Passes between two layers of mesentery	Jejunum and ileum
Middle colic		Ascends retroperitoneally and passes between layers of transverse mesocolon	Transverse colon
Right colic		Passes retroperitoneally to reach ascending colon	Ascending colon
Ileocolic	Terminal branch of superior mesenteric artery	Runs along root of mesentery and divides into ileal and colic branches	Ileum, cecum, and ascending colon
Appendicular	Ileocolic artery	Passes between layers of meso-appendix	Appendix
Inferior mesenteric	Abdominal aorta	Descends retroperitoneally to left of abdominal aorta	Supplies part of gastrointestinal tract derived from hindgut
Left colic	Inferior mesenteric artery	Passes retroperitoneally toward left to descending colon	Descending colon
Sigmoid (n = 3–4)		Passes retroperitoneally toward left to descending colon	Descending and sigmoid colon
Superior rectal	Terminal branch of inferior mesenteric artery	Descends retroperitoneally to rectum	Proximal part of rectum
Middle rectal	Internal iliac artery	Passes retroperitoneally to rectum	Midpart of rectum
Inferior rectal	Internal pudendal artery	Crosses ischioanal fossa to reach rectum	Distal part of rectum and anal canal

Arterial Supply [4]

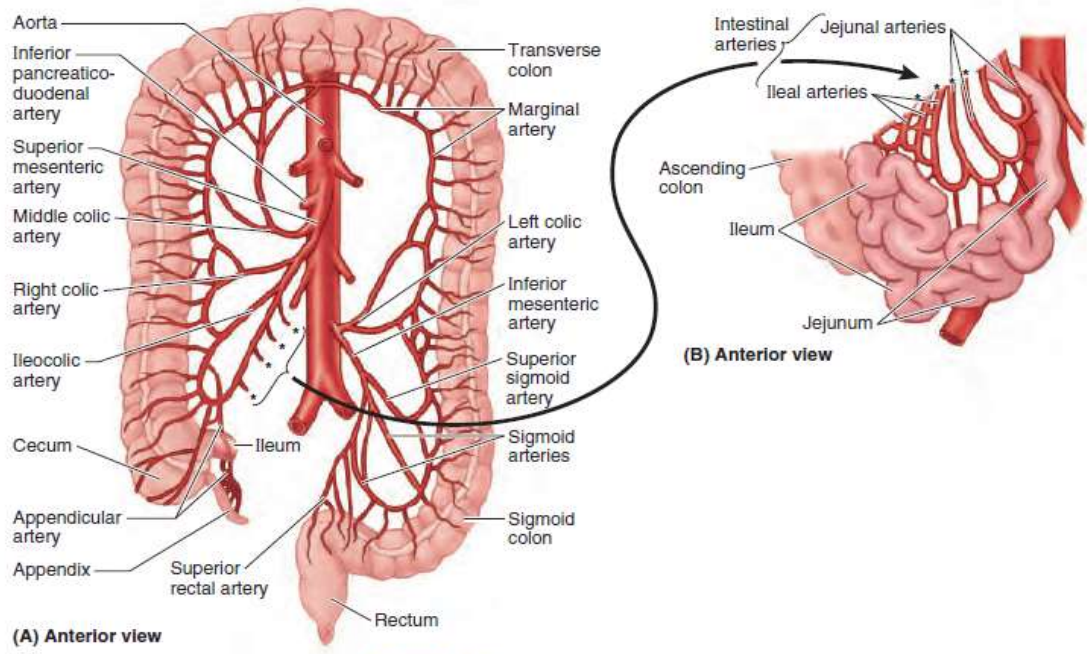
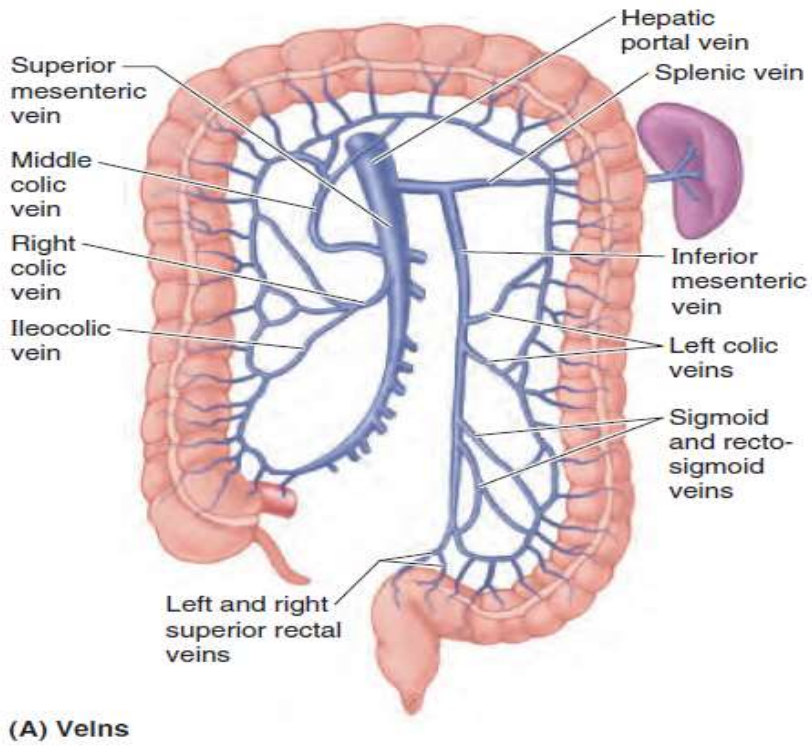
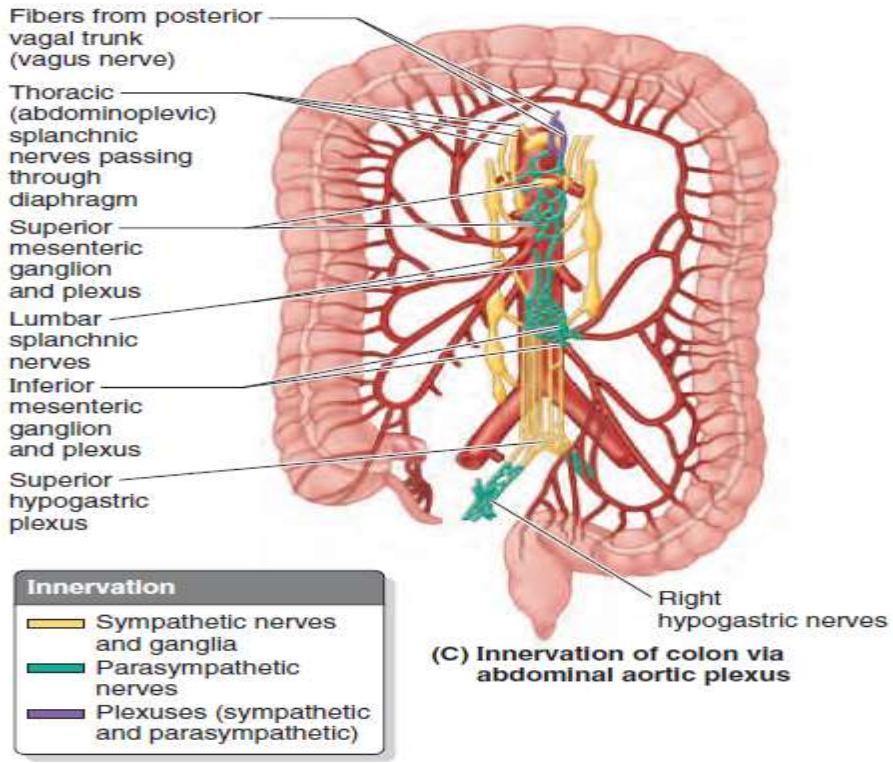


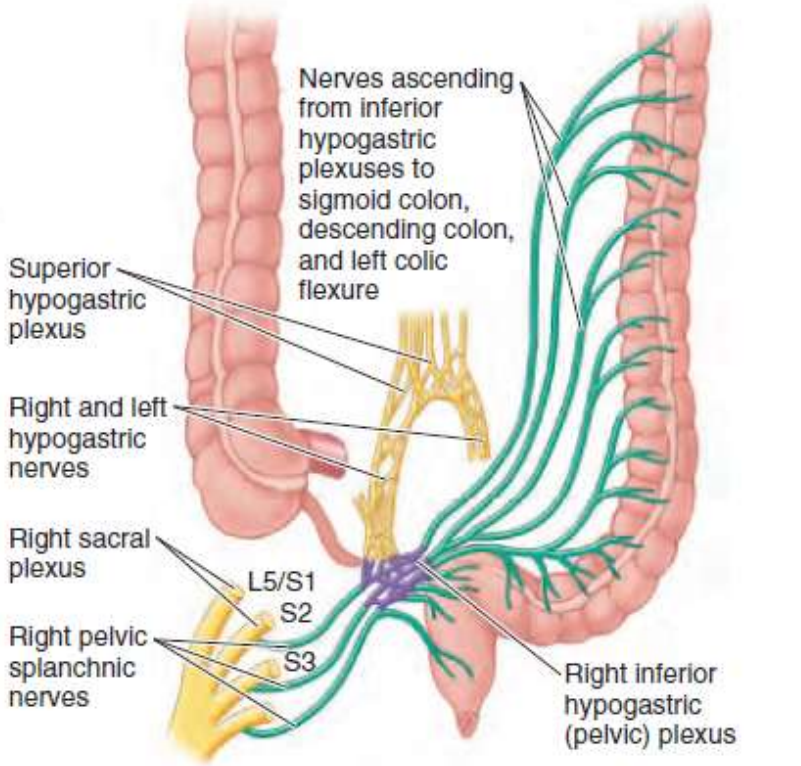
FIGURE 2.54. Arterial supply to intestines.

Venous Supply [4]



Nerve Supply to the Colon [4]





(D) Parasympathetic nerves to descending and sigmoid colon

Lymphatic Supply [4]

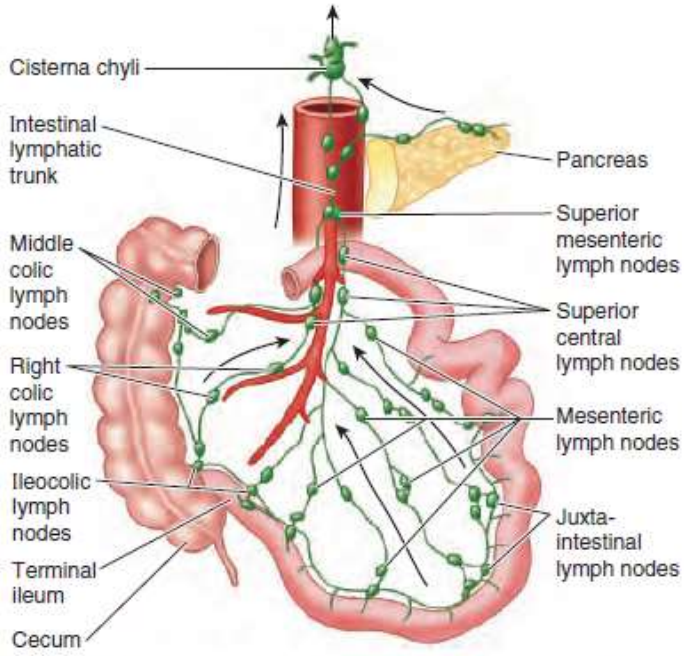


FIGURE 2.50. Mesenteric lymph nodes. The superior nodes form a system in which the central nodes, at the root of the superior mesenteric artery, receive lymph from the mesenteric, ileocolic, right colic, and middle colic nodes, which in turn receive lymph from juxta-intestinal lymph nodes. The juxta-intestinal nodes adjacent to the intestines are most abundant. Fewer occur along the arteries.

ACUTE INTESTINAL OBSTRUCTION

Introduction

Descriptions of patients with small bowel obstruction (SBO) date back to the earliest medical literature. It was not until the 19th century and the advent of anaesthesia and antisepsis, however, that surgery became a recognized and effective treatment. At the same time, physiologic studies of fluid shifts, electrolyte imbalances, intravenous resuscitation, and antibiotics allowed even safer surgical approaches to patients with obstruction.

Moreover, these admissions are costly both in terms of time and money: the average length of stay for patients requiring operative exploration is 6 days following a laparoscopic lysis of adhesions and 11 days following a laparoscopic bowel resection, with heavy hospital charges.

At the time of initial evaluation for SBO, it is critical to determine whether a true mechanical obstruction or pseudo obstruction (dysmotility/ileus) is the cause of symptoms; this distinction will guide all subsequent treatment.

Clinical judgment must also be employed to determine illness severity, resuscitation requirements, and the urgency of operative intervention.

Patients may present acutely, or with a chronic and relapsing problem with symptoms ranging from modest discomfort to critical illness and shock. [9]

CLASSIFICATION AND TERMINATION [10]

SBO can be classified by mechanism. The patient's symptoms and presenting signs may be caused by a functional obstruction from dysmotility, or a true mechanical obstruction.

Mechanical SBO may be further classified **as partial or complete obstruction.**

The aetiology of mechanical SBO divided into three main categories: **extrinsic, intrinsic/intramural, and intraluminal.**

Table 49-1 Classification of Adult Mechanical Intestinal Obstructions

Intraluminal	Intramural	Extrinsic
Foreign bodies	Congenital (rare in adult)	Adhesions
Barium inspissation (colon)	Atresia, stricture or stenosis	Congenital: Ladds or Meckel bands
Bezoar	Web	Postoperative
	Intestinal duplication	Postinflammatory (after pelvic inflammatory disease)
	Meckel diverticulum	
Inspissated feces	Inflammatory Process	Hernias
Gallstone ileus	Crohn disease	Abdominal wall
Meconium (cystic fibrosis)	Diverticulitis	Internal
	Stricture from ischemia	Volvulus
	Radiation enteritis or stricture	External mass effect
	Medication-induced (NSAIDs, KCl tablets)	Abscess
Parasites (ascaris, diphyllobothrium)	Neoplasms	Annular pancreas
Enterolith	Primary intestinal or colon (malignant or benign)	Pancreatic pseudocyst
Intussusception	Secondary (metastasis or carcinomatosis)	Carcinomatosis
		Endometriosis
Polypoid and exophytic lesions	Trauma (e.g., intramural hematoma)	Pregnancy

The term **mechanical obstruction** means that luminal contents cannot pass through the gut tube because the lumen is blocked. This obstruction is in contrast with neurogenic or functional obstructions in which luminal contents are prevented from passing because of disturbances in gut motility that prevents coordinated peristalsis from one region of the gut to the next. This latter form of obstruction is commonly referred to as **ileus** in the small intestine and **pseudo-obstruction** in the large intestine.

In **simple obstruction**, the intestinal lumen is partially or completely occluded without compromise of intestinal blood flow. Simple obstructions may be complete, meaning that the lumen is totally occluded or incomplete, meaning that the lumen is narrowed but permitting distal passage of some fluid and air.

In **strangulation obstruction**, blood flow to the obstructed segment is compromised and tissue necrosis and gangrene are imminent.

Strangulation usually implies that the obstruction is complete, but some forms of partial obstruction can also be complicated by strangulation.

The various forms of mechanical intestinal obstruction can be classified according to different but overlapping schemes. Most commonly, obstruction is classified according to aetiology.

Adhesions are the most common cause of intestinal obstruction, accounting for more than half of all cases. In order to highlight the path physiology, presentation, and natural history, however,
it is useful to classify obstruction according to the location of the obstructing lesion. **Proximal or "high" obstructions** involve the pylorus, duodenum, and proximal jejunum. **Intermediate levels** of obstruction
involve the intestine from the mid jejunum to the midileum. **Distal levels** of obstruction arise in the distal ileum, ileocecal valve, and proximal colon whereas the most distant or "low" obstructions would
arise in regions beyond the transverse colon.

clinical symptoms and signs of obstruction (pain, vomiting, abdominal distention, and gas pattern on abdominal radiographs) vary with
the level of obstruction.

It is also important to distinguish between **open-loop** and **closed-loop** obstructions. An **open-loop obstruction** occurs when intestinal flow is blocked but proximal decompression is possible through

vomiting. A **closed-loop obstruction** occurs when inflow to the loop of bowel and outflow from the loop are both blocked.

This obstruction permits gas and secretions to accumulate in the loop without a means of decompression, proximally or distally. Examples of closed-loop obstructions are torsion of a loop of small intestine around an adhesive band, incarceration of the bowel in a hernia, volvulus of the cecum or colon, or development of an obstructing carcinoma of the colon with a competent ileocecal valve.

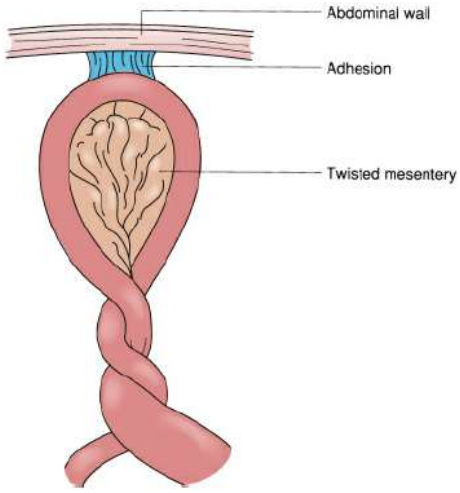


Figure 49-2. Schematic illustration of a closed-loop obstruction. The small intestine twists around its mesentery, compromising inflow and outflow of luminal contents from the loop. Also, the vascular supply to the loop may be compromised due to the twisting of the mesentery. The risk of strangulation is high.

The primary symptoms of a closed-loop obstruction of the small intestine are sudden, severe midabdominal pain and vomiting whereas symptoms of the large intestine are pain and sudden abdominal distention. This pain often occurs before associated findings of localized abdominal tenderness or involuntary guarding. When signs of peritoneal irritation or frank peritonitis develop, there is a high level of suspicion that the viability of the bowel is compromised.

Table 49-2 Symptoms and Signs of Bowel Obstruction

Symptom/ Sign	Proximal Small Bowel (Open Loop)	Distal Small Bowel (Open Loop)	Small Bowel (Closed Loop)	Colon/Rectum
Pain	Intermittent, intense, colicky, often relieved by vomiting	Intermittent to constant	Progressive, intermittent to constant; rapidly worsens	Continuous
Vomiting	Large volumes, bilious and frequent	Low volume and frequency, progressively feculent with time	May be prominent (reflex)	Intermittent, not prominent; when present, feculent
Tenderness	Epigastric or periumbilical; quite mild, unless strangulation is present	Diffuse and progressive	Diffuse, progressive	Diffuse
Distension	Absent	Moderate to marked	Often absent	Marked
Obstipation	May not be present	Present	May not be present	Present

Adapted from Schuffler MD, Sinanan MN. Intestinal obstruction and pseudo-obstruction. In: Sleisenger MH, Fordtran JS, eds. *Gastrointestinal Disease*. 5th ed. Philadelphia, PA: WB Saunders, Co.; 1993;898–916.

Pathophysiology of Intestinal Obstruction

Local Effects of Bowel Obstruction

When a loop of bowel becomes obstructed, intestinal gas and fluid accumulate. Stasis of luminal content favors bacterial overgrowth, alters intestinal fluid transport properties and motility, and causes

variations in intestinal perfusion and lymph flow. Luminal contents and volume, bacterial proliferation, and alterations in motility and perfusion work in concert to determine the rate at which symptoms and complications develop. Each of these factors merits discussion in some detail.

Intestinal Gas.

Approximately 80% of the gas seen on plain abdominal radiographs is attributable to swallowed air. Approximately 70% of the gas in the obstructed gut is inert nitrogen. Oxygen accounts for 10% to 12%, CO2 for 6% to 9%, hydrogen 1%, methane 1%, and hydrogen disulfide 1% to 10%. In the setting of acute pain and anxiety, patients with intestinal obstruction may swallow excessive amounts of air. Passage of such swallowed air distally is prevented by nasogastric suction.

Intestinal Flora. An important contribution to normal digestive function comes from its bacterial population. In patients with normal gastric acid secretion, the chyme entering the duodenum is sterile.

The small numbers of bacteria that are found in stomach and proximal intestine are aerobic, gram-positive species found in the oropharynx. Distally, in the ileum and colon, gram-negative aerobes are present and anaerobic organisms predominate.

Total bacterial counts in normal feces reach organisms per gram of fecal matter.

Control of the bacterial populations depends on intact motor activity of the intestines and the interactions of all species present. This ecology can be disturbed by antibiotic therapy or by surgical reconstructions that result in stasis within intestinal segments.

Intestinal bacteria serve several functions, including metabolism of fecal sterols, releasing the small chain fatty acids that are an important food source for colonocytes; metabolism of fecal bile acids, fat-soluble vitamins (e.g., vitamin K) and vitamin B12; and breakdown of complex carbohydrates and organic matter, leading to formation of CO2, H2, and CH4 gases.

Intestinal Fluid.

Classical experimental studies established that fluid accumulates intraluminally with open- or closed-loop small intestinal obstruction.

Factors contributing to the accumulation of fluid include intraluminal distention and pressure, release of prosecretory and antiabsorptive hormones and paracrine substances, changes in mesenteric circulation, and elaboration and luminal release of bacterial toxins.

Experimental studies and clinical investigation demonstrated that elevation of luminal pressures above 20 cm H2O inhibits absorption and stimulates secretion of salt and water into the lumen proximal to an obstruction.

In closed-loop obstructions, luminal pressures may exceed 50 cm H2O and may account for a substantial proportion of luminal fluid accumulation.

In simple, open-loop obstructions, distention of the lumen by gas rarely leads to luminal pressures higher than 8 to 12 cmH2O.

The release of endocrine/paracrine substances remains relatively uncharacterized in states of mechanical bowel obstruction. vasoactive intestinal polypeptide (VIP) may be released from the sub mucosal and myenteric plexuses within the gut wall, promoting epithelial secretion and inhibiting absorption.

Intestinal Blood Flow.

Micro vascular responses to intestinal obstruction may also play an important role in determining the hydrostatic gradients for fluid transfer across the mucosa into the lumen. In response to heightened luminal pressure, total blood flow to the bowel wall may initially increase.

The breakdown of epithelial barrier structures and enzymatic breakdown of stagnant intestinal contents leads to increased osmolarity of luminal contents.

In addition to secretory stimulation and absorptive

inhibition of the mucosa, the simultaneous changes in hydrostatic and osmotic pressures on the blood and lumen sides of the mucosa favor flow of extracellular fluid into the lumen. Perfusion is then

compromised as luminal pressures increase, bacteria invade, and inflammation leads to oedema within the bowel wall.

Intestinal Motility

Obstruction of the intestinal lumen does not simply block distal passage of luminal contents. The accumulation of fluid and gas in the obstructed lumen also elicit changes in myoelectrical function of the gut, proximal and distal to the obstructed segment. In response to this distention, the

obstructed segment itself may dilate, a process known as receptive relaxation.

Such changes ensure that, despite accumulation of air and fluid, intraluminal pressures do not amplify easily to the point of compromising blood flow to the intestinal mucosa.

At sites proximal and distal to the obstruction, changes in myoelectrical activity are time dependent. Initially, there may be intense periods of activity and peristalsis. Subsequently, myoelectrical activity is diminished and the interdigestive migrating myoelectrical complex pattern is replaced by ineffectual and seemingly disorganized clusters of contractions.

Complications and Systemic Effects of Bowel Obstruction

Closed-Loop Obstructions. The complications of closed-loop obstructions evolve rapidly. The reasons for this rapid evolution are best understood by considering the simplest and most common form of closed-loop obstruction, appendicitis.

When a fecalith obstructs the blind-ended appendix, secretion of mucus and enhanced peristalsis represent the initial attempt to clear the blockage. Intense crampy abdominal pain focused at the umbilicus results. Nausea and vomiting are not uncommon as a result of luminal obstruction

but as a reflexive response to hyper peristalsis and stretching of the mesentery. Over the next 8 to 18 hours, continued secretion of mucus to high intraluminal pressures, stasis, bacterial overgrowth, mucosal disruption, and elevation of luminal pressures convert intermittent cramps to constant and worsening pain. When luminal pressure exceeds mural venous pressure and then capillary perfusion pressures, inflammatory cells are recruited from surrounding peritoneal structures. This sequence of events leads to intense inflammation, release of exudates in the area of the appendix and the first localization of pain from the umbilicus to the area of peritoneum lying nearest the inflamed appendix. Peritoneal findings (localized tenderness, involuntary guarding, rebound, or referred tenderness) and fevers appear. Subsequently, 20 to 24 hours into the illness, the blood supply of the appendix is compromised. Gangrene and perforation follow and, if not contained by surrounding structures, free perforation leads to a rigid abdomen. Toxins from necrotic tissue and bacterial overgrowth are released into the systemic circulation and shock ensures.

Torsion of a loop of small intestine around an adhesive band or inside a hernia leads to a similar pattern of events. Torsions of the large bowel are usually accompanied by massive distention of the loop by air

and feces, but the compromise of intestinal wall perfusion and evolution into peritonitis, systemic toxicity, and shock are similar.

Open-Loop Obstructions. Complications in open-loop obstructions do not necessarily evolve as rapidly as in closed-loop obstructions. Not uncommonly, an open-loop obstruction located in the proximal jejunum can be decompressed by the patient's ability to vomit.

Proximal obstruction is characterized by vomiting and loss of gastric, pancreatic, and biliary secretions, with resulting electrolyte disturbances.

These disturbances include dehydration, metabolic alkalosis, hypochloremia, hypokalemia, and usually hyponatremia.

In contrast, obstructions of the distal ileum may lead only to a slowly progressing distention of the small intestine, with accommodation by intestinal myoelectrical function and minor alterations in fluid and electrolyte balances.

Open-loop obstructions located in the midgut are often complicated by events similar to those seen in closed-loop obstructions or combinations of events seen in high and low obstructions.

Patients with distal jejunal obstruction tend to present with a combination of complications resulting from loss of intestinal contents from vomiting, as well as distention and compromise of intestinal wall perfusion.

In simple or uncomplicated obstruction, the intestinal lumen is partially or completely occluded without compromise of intestinal blood flow. Simple obstructions may be complete, meaning that the lumen is totally occluded or incomplete, meaning that the lumen is narrowed but permitting distal passage of some fluid and air.

In strangulation obstruction, blood flow to the obstructed segment is compromised and tissue necrosis and gangrene are imminent.

Clinical Presentation and Differential Diagnosis

The four key symptoms associated with acute mechanical bowel obstruction include

- abdominal pain
- vomiting
- distention
- obstipation

When bowel obstruction is the most likely diagnosis, “abdominal pain out of proportion to physical findings” represents a surgical emergency.

Colon obstruction is usually accompanied by varying levels of pain with massive abdominal distention and obstipation.

As noted earlier, the signs and symptoms of acute but simple small intestinal obstructions are related to the level of the obstruction and the closed- or open-loop nature of the obstruction.

Strangulation obstruction is accompanied by symptoms and signs suggesting peritonitis, large fluid shifts, or systemic toxicity.

These symptoms and signs include abdominal tenderness or involuntary guarding localized to the area of the strangulated loop of bowel, decreasing urine output, fever, and tachycardia.

Different laboratory tests have been advocated for early detection of strangulated intestine. Metabolic (i.e., lactic) acidosis and increases in serum amylase, inorganic phosphate, hexosaminidase, intestinal fatty acid–binding protein and serum D-lactate levels have all been associated with intestinal ischemia.

Radiographs and Imaging

Plain Films

The role of plain abdominal radiographs and imaging studies is

- To confirm the diagnosis of bowel obstruction
- Locate the site of obstruction
- Identify lesion responsible for the obstruction.
- Accumulation of air and fluid proximal clearance of fluid and air distal to the point of obstruction.
- Dilated loops of small intestine are defined as those greater than 3 cm in diameter.
- Free air represents perforation of a viscus and mandates immediate operation.
- Dilated loops of small bowel on the flat plate
- Multiple air–fluid levels located at different levels on the upright film or lateral decubitus film
- Plain radiographs can miss SBO in patients without air–fluid levels because of fluid-filled distended loops.

Figure 49-5. Plain upright abdominal film of a patient with sigmoid volvulus. The dilated centrally located sigmoid loop is seen (*arrowheads*). The proximal colon is dilated and gas filled. T, transverse colon; D, descending colon.

- In complete obstruction of the small intestine, the colon loops and rectum do not contain air.
- If there is air in the colon, the obstruction may be complete, but early, or it may be incomplete.
- In the colon, tight closed-loop obstructions (i.e., volvulus of the cecum, transverse colon, or sigmoid colon) are accompanied by distention of the obstructed segment
- The proximal colon is considered dilated when it reaches 8 to 10 cm
- The sigmoid colon is considered dilated at 4 to 5 cm.
- In contrast, obstruction by carcinoma or diverticulitis presents with massive distention of the entire colon from the point of obstruction to the ileocecal valve.

Figure 49-4. Plain upright abdominal film of a patient with small-intestinal obstruction. Note the air–fluid levels in the stomach and multiple dilated loops of small intestine (*black arrows*), and absence of air in the colon or rectum.

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CONTRAST STUDIES

Contrast studies (i.e., small bowel follow through, enteroclysis, and contrast enema) may provide specific localization of the point of obstruction and the nature of the underlying lesion.

however, contrast studies are unnecessary and may be contraindicated in the classic setting of abdominal pain, nausea, vomiting, and a plain film indicating multiple air–fluid levels in the small intestine and colonic collapse, where the diagnosis of acute obstruction can be made clinically.

Contrast agents used

- **Water Insoluble suspension – Barium**
- **Water soluble – Gastrograffin/Hypaque**

Barium studies – given as Oral/NG tube for small bowel studies and as enema for colonic studies

- **Advantage –** provide the clearest anatomy
- **Disadvantage** – contraindicated if suspicious of gangrene/perforation as barium may leak and cause intense peritonitis

Gastrograffin/Hypaque -sodium amidotrizoate/meglumine amidotrizoate oral solution or diatrizoate sodium/diatrizoate meglumine

Advantage

- hyper osmotic(1900mosm/l)

- Can be both diagnostic/therapeutic
- Sensitivity -96%
- Permits mobilization of fluid into the bowel lumen which decreases oedema of the intestinal wall and increases the pressure gradient across obstructive site resulting in resolution of obstruction

Imaging Modalities

- **Computed tomography (CT**)

  It need not be routinely performed unless history, physical examination, and plain films are not conclusive for SBO diagnosis; it is increasingly the "go-to" study for confirmation.

- Sensitivity – 96% ,Specificity – 93%,Negative predictive value – 99%

Advantages

- using radiographic contrast, the obstructing segment may be localized and characterized as complete or incomplete.

- the nature of the obstructing lesion, especially if it is malignant, can be established.

- Additional abdominal pathology (e.g., metastases, ascites, parenchymal liver abnormalities) may be identified.

- Anatomic information obtained from the CT can be used in operative planning.

Findings on CTscan suggesting strangulation /obstruction include

- beak-like narrowing
- mesenteric oedema
- vascular engorgement
- moderate to severe intestinal wall thickening (greater than 2 mm)
- high attenuation of bowel wall on unenhanced CT scans

- low or reduced attenuation of bowel wall on intravenous contrast CT scans, and presence of intramural air (pneumatosis) or portal venous gas.

ULTRASONOGRAPHY

- It has limited use in diagnosing SBO as visualization can be obscured by the intraluminal air. Presence of significant amounts of peritoneal fluid and of an akinetic and dilated loop of bowel were strongly associated with the presence of strangulation

MAGNETIC RESONANCE IMAGING (MRI)

- Magnetic resonance imaging (MRI) has the sensitivity comparable to CT scan in diagnosing obstruction but limitations include lack of availability after hours, poor definition of mass lesion, and poor visualization of colonic obstructions. The use of MRI should be limited to patients who have contraindications to CT or are allergic to contrast material
- It should be emphasized that when the clinical picture suggests strangulation, unnecessary imaging studies should not delay resuscitation or expeditious movement to the operating room.

- Such studies will not necessarily be helpful when clinical criteria and basic abdominal radiographs have indicated the presence of a simple and complete obstruction.
- By itself, this diagnosis mandates urgent exploration and the information sought should be weighed against the risk of delay in going to the operating room
- In patients with equivocal findings or uncertain clinical diagnosis, CT can be highly useful in confirming the diagnosis, localizing the site and detecting the cause of intestinal obstruction and strangulation.

General Considerations in Management of the Patient with Bowel Obstruction

- Patients with obstruction of the large bowel present with abdominal pain, distention, and obstipation. Vomiting and electrolyte imbalances are sometimes prominent, though usually delayed.

- The presentation of SBO depends on level of obstruction, open- or closed-loop nature, and interval since onset of symptoms. Symptoms

and signs of pain, vomiting, obstipation, and distention are present in variable degrees.

In the settings described previously, the following questions must be addressed as expeditiously as possible:

1. Is the abdominal pain disproportionate to the physical findings and laboratory studies?

2. How rapidly the symptoms and signs are evolving: minutes, hours, or less acutely?

3. Does the patient suffer from dehydration, electrolyte imbalance and acid–base disturbance?

4. Is the obstruction complete or incomplete?

5. Is there a possibility of strangulation?

- Clinical data and basic laboratory studies will provide reliable information to answer the first three questions.
- Abdominal radiographs and imaging studies are frequently used to provide additional information to help answer these latter questions, as well as providing information to identify the obstructing lesion

- The initial management of all patients with suspected bowel obstruction includes designating the patient “NPO” and starting

intravenous fluids comprised of isotonic Ringers or normal saline solutions.

- In rapidly evolving cases or patients with significant dehydration, an indwelling urinary catheter should be placed to monitor urine output.

- Invasive hemodynamic monitoring (e.g., a Swan–Ganz catheter) may be necessary to monitor the response to fluid resuscitation in patients with severe cardiac, pulmonary, or renal insufficiency.

- Nasogastric decompression is indicated in most cases. The nasogastric tube, typically a 16- or 18-Fr sump tube, serves to prevent distal passage of swallowed air and minimizes the discomfort of refluxing intestinal content

Intravenous Antibiotics

- Administer antibiotics perioperatively, in order to reduce wound infection and abdominal sepsis rates in patients undergoing operation to relieve intestinal obstruction, simple or strangulated

- Studies indicate that bacteria counts raise and can translocate across the intestinal mucosa, passing into lymph channels in a case of intestinal obstruction
- A second generation cephalosporin or a combination of a first-generation cephalosporin and metronidazole is a rational practice for perioperative coverage in both simple and strangulation obstruction

It should be emphasized that once a diagnosis of complete obstruction is made, simple or strangulated, the operation should proceed without undue delay

It is reasonable to commit the patient to a period of observation when the diagnosis is uncertain (i.e., there is a possibility of a nonsurgical diagnosis or that the obstruction is not complete).

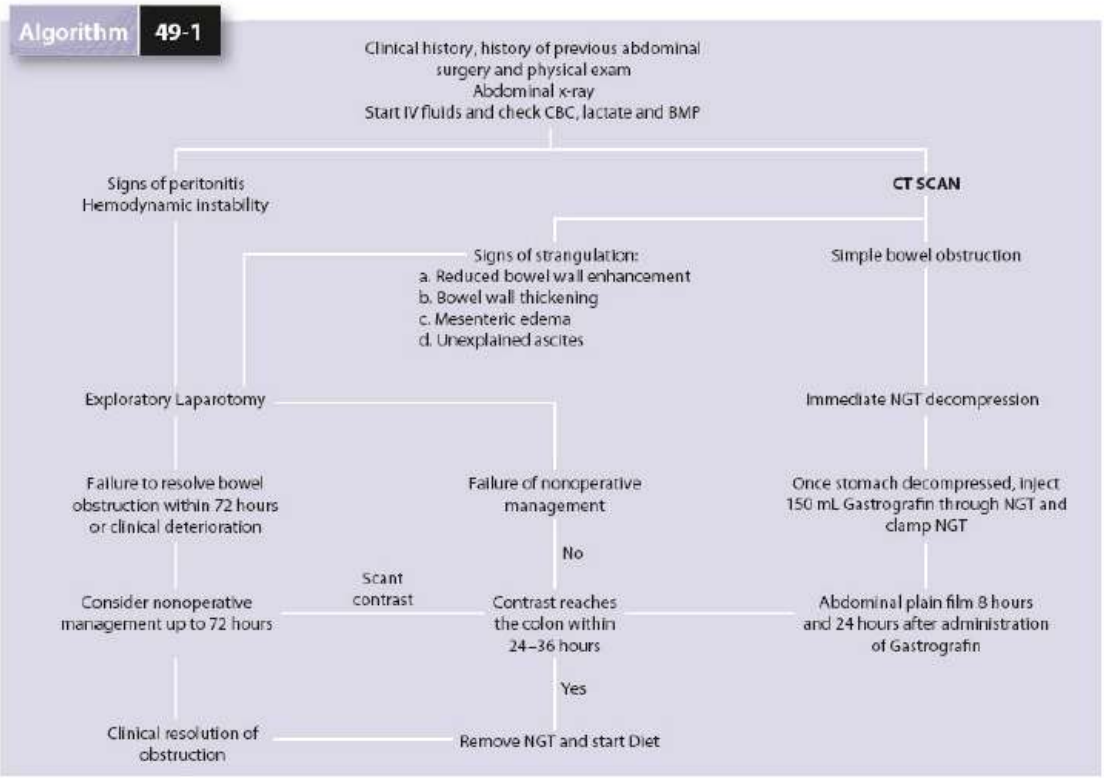
A practical point is that obstruction occurring in a patient without a previous history of laparotomy is not likely to be caused by peritoneal adhesions. This is known as **de novo obstruction** and whatever the underlying cause will not usually resolve without operation.

Specific Types of Bowel Obstruction

CHRONIC ADHESIONS

- ✓ Peritoneal adhesions account for more than half of SBO cases
- ✓ Lower abdominal procedures such as appendectomy, hysterectomy, colectomy, and abdominoperineal resection are common precursors

- ✓ Up to 80% episodes of SBO due to adhesions may resolve nonoperatively
- ✓ An index episode and three recurrences indicate a likelihood of over 80% that there will be more recurrences
- ✓ Surgical management of an acute episode appears to reduce subsequent recurrence rates from 15% to 6%
- ✓ No studies have been able to establish whether the immediate benefit of laparotomy outweighs the overall benefit of expectant management and operation only for serial recurrences
- ✓ Ultimately, patients who present with signs and symptoms of bowel obstruction are managed according to the CT findings and clinical course



Algorithm 49-1. Algorithm for the management of adhesive small bowel obstruction.

Mechanism of adhesion formation

- Histologic examination of chronic adhesions reveals foreign body reaction, usually to talc, starch, lint, intestinal content, or suture
- In early studies, inflammatory cells, including mast cells were implicated in the process that produces adhesions.

Strategy to prevent Adhesion

- Meticulous attention to hemostasis and surgical technique
- Avoidance of excessive tissue dissection
- Careful search and removal of any extraneous material
- Use of laparoscopic approaches, when feasible.

Hyaluronic acid–carboxymethylcellulose membrane (Seprafilm, Genzyme, Cambridge, MA) –

- This compound mechanically prevents adhesion formation by physically separating adjoining tissues. Absorbed by the body in 7 days and thus is present only during the phase of fibrosis, and not as a persistent foreign body.
- **Icodextrin 4%** solution before abdominal closure
- Use of **hydrogen adhesion barrier spray**
- Use of **lyophilized human peritoneal membrane**

EARLY POSTOPERATIVE ADHESIONS

- ✓ Obstruction in the immediate period following abdominal surgery is uncommon but may occur in up to 1% of patients in the 4 weeks following laparotomy
- ✓ Adhesions are responsible for approximately 90% of such cases and hernias for approximately 7%
- ✓ Majority of such cases may be treated as partial intestinal obstruction.
- ✓ Nasogastric suction and intravenous fluids will help resolve symptoms within a few days

- ✓ When the clinical course does not demand earlier intervention, a nonoperative approach may be tried for 10 to 14 days and will resolve the obstruction in over 75% of such cases
- ✓ Patients with acutely evolving symptoms and signs represent complete obstruction and should be managed as such as the mortality may be as high as 15% due to delays in recognition and operative intervention.

HERNIAS

- ✓ Hernias of all types are second only to adhesions as the most frequent causes of obstruction.

- ✓ External hernias such as inguinal or femoral hernias may present with the symptoms of obstruction and will not be diagnosed unless sought

- ✓ Femoral hernias are particularly prone to incarceration and bowel necrosis due to the small size of the hernia

- ✓ Inlet hernias such as umbilical, incisional, paracolostomy or lumbar hernias are obvious.

- ✓ Internal hernias are usually diagnosed at laparotomy for obstruction and they include obturator hernias,paraduodenal hernias and hernias through the foramen of Winslow or mesenteries

- ✓ When hernia has been identified as the cause of the obstruction, the patient is quickly resuscitated, given antibiotics, and taken to the operating room.

- ✓ The hernia is then reduced and the viability of the bowel assessed. If viable, the bowel is left alone; if not, it is resected and hernia defect is then repaired.

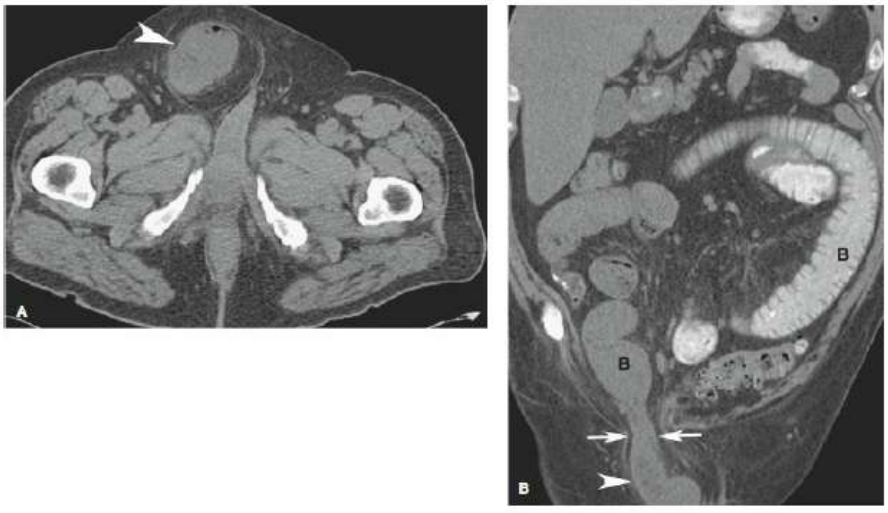


Figure 49-7. Computed tomography images of an inguinal hernia. Axial (A) and coronal (B) CT scan images showing incarcerated right inguinal hernia with air- and fluid-filled loop of small bowel (*arrowhead*) in the right inguinal canal (*arrow*) causing small bowel obstruction with dilated loops of proximal small bowel (B).

GALL STONE ILEUS

- ✓ Due to intense inflammation surrounding a gallstone, a fistula may develop between the biliary tree and the small or large intestine.
- ✓ Fistulae develop between the gallbladder fundus and duodenum.
- ✓ Stone greater than 2.5 cm in diameter can lodge in the narrowest portion of the terminal ileum, which is just proximal to the ileocecal valve.
- ✓ A Rare complication accounting for less than 6 in 1,000 cases of cholelithiasis and less than 3% of cases of intestinal obstruction.
- ✓ The classic findings on plain radiographs include those of intestinal obstruction, a stone lying outside the right upper quadrant, and air in the biliary tree
- ✓ Treatment includes removal of the stone and resection of the obstructed segment only if there is evidence of tissue necrosis.
- ✓ Risk of a recurrent gallstone ileus is about 5% to 10%. And recurrences usually occur within 30 days of the initial episode and are usually due to stones in the small intestine that were missed at the original operation.

- ✓ The consensus is that cholecystectomy should not be performed at the initial operation for gallstone ileus, except in highly selected patients.
- ✓ A careful search of the entire intestine should be performed to exclude the possibility of additional large stones which can occur in up to 25% of patients.

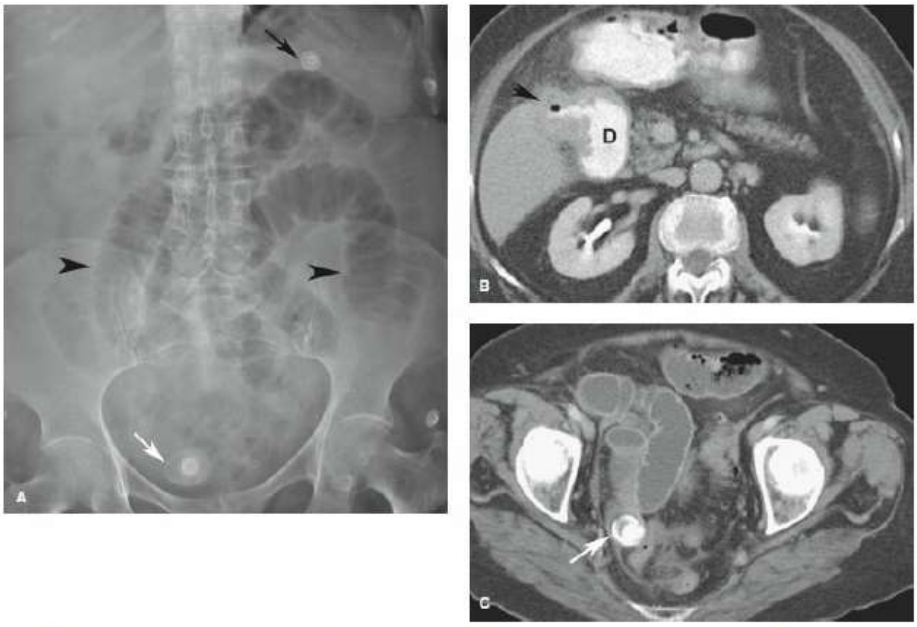


Figure 49-10. A: Plain radiograph of a patient with gallstone ileus, showing obstructed loops of small intestine (*black arrow*) in the abdomen and a gallstone (*white arrow*) in the pelvis (gallstone was initially misinterpreted as an EKG lead [*black arrow*]). B: Computed tomography (CT) scan showing a cholecystoduodenal fistula (*black arrow*) with air in the biliary tree (D, duodenum). C: CT scan showing gallstone (*white arrow*) in the distal ileum and fecalization of luminal content adjacent to the stone.

Intussusception

- ✓ An intussusception occurs when one segment of bowel

telescopes into an adjacent segment, resulting in obstruction and ischemic injury to the intussuscepting segment and the obstruction may become complete, particularly if tissue inflammation and necrosis occur.

- ✓ 5% of intussusceptions occur in adults
- ✓ 90% are associated with pathologic processes(65% - malignancy)

Four types of intussusception are recognized

- ✓ Enteric(ileo- ileal)
- ✓ Ileocolic
- ✓ Ileocecal
- ✓ Colonic(colo- colic)

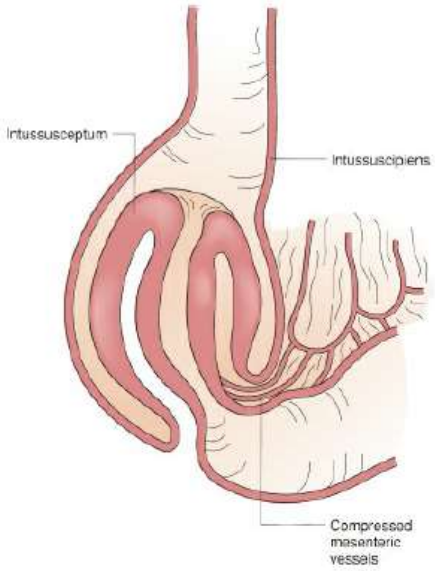


Figure 49-11. Anatomy of intussusception. The intussusceptum is the segment of bowel that invaginates into the intussuscipiens.

Radiography

- ✓ Plain X-ray may show multiple air fluid levels. Barium enema in ileo-colic type shows typical ‘pincer shaped’ or ‘coiled spring’ deformity or ‘pinch fork’ sign.

- ✓ Reduction by hydrostatic pressure, which is the standard of care in pediatric cases, is not usually attempted in adults.

- ✓ Clear indications for operation
    - ➢ long length and wide diameter of the intussusceptions
    - ➢ presence of a lead point
    - ➢ evidence of bowel obstruction.

CROHN'S DISEASE

Crohn disease is characterized by transmural inflammation that can involve any part of the gastrointestinal tract, from mouth to anus,

Medical treatment regimens should be tailored to the severity of inflammation (mild, moderate, or severe) and the location of the active lesions.There is no surgery that will cure crohn's disease.

- ➢ Indication for surgery include - obstruction , fistula , perforation and resistance/failure of medical therapy
- ➢ Crohn's disease can present with Obstruction due to fibrotic stricture. In such cases, It is important to examine the entire length of the small bowel to ensure that all obstructive strictures

are identified to avoid recurrence of symptomatic obstruction due to missed segments of disease.

- Crohn-affected bowel may not be dilated proximal to the obstruction but can be complicated by a small perforation which may not show up as free air on plain film
- A CT scan is likely to be the most sensitive imaging modality for obtaining evidence that differentiates conditions that require immediate surgery (closed-loop obstruction and micro perforation) from simple obstruction that would otherwise be observed
- The number, length, and location of strictures should inform surgical decision making when considering stricturoplasty versus resection.
- All patients with high-grade small bowel obstruction should undergo decompression and resuscitation prior to surgical intervention.

Malignant Obstruction

- Obstruction can complicate malignancies of the small and large bowel.
- 10% to 28% of patients with colorectal cancer and 20% to 50% of patients with ovarian cancer will present with a malignant bowel obstruction
- Patients who previously has undergone surgery for malignancy can now return with evidence of bowel obstruction (adhesions in colorectal cancer primary)
- Gastric and pancreatic carcinomas often present with or are subsequently complicated by peritoneal carcinomatosis and the subsequent obstruction is most likely due to malignancy.
- Patients with advanced malignant obstruction in the absence of a solitary or correctable obstructing lesion are generally managed Palliatively
- Patients are managed without a nasogastric tube if possible and encouraged to eat as soon as obstructive symptoms resolve using a low-fibre diet.
    - Antiemetic and opioids via continuous subcutaneous infusions are used to manage nausea, vomiting, and colic, respectively.

- Octreotide, a somatostatin analog, is used in palliation of refractory malignant intestinal obstruction by improving intestinal mucosal absorption, improving motility, reducing gastrointestinal hormone levels and intestinal secretions, and having a direct antineoplastic effect on the obstructing tumour

Approach to management of Malignant Obstruction

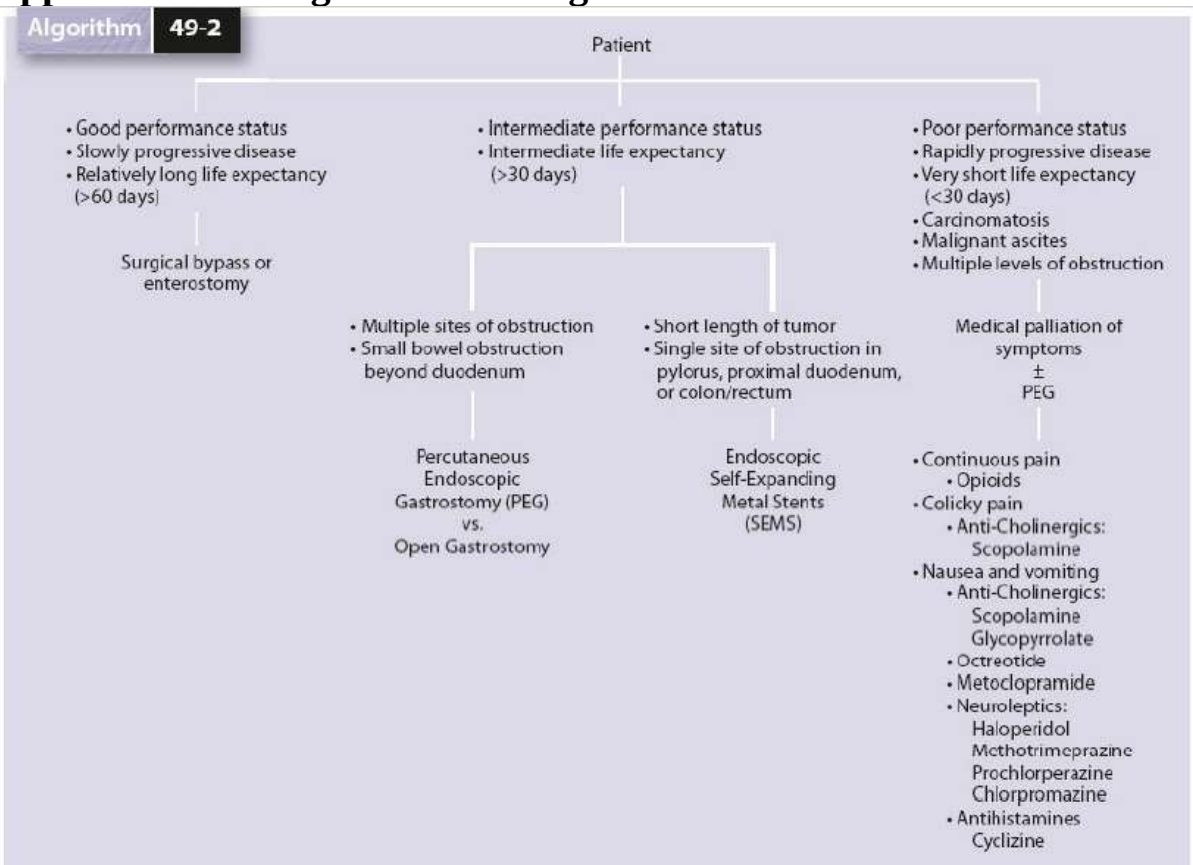


Table 49-4 Results of Management of Malignant Obstructions: Risk Factors for Unfavorable Outcome

Advanced age (biologic/physiologic)
Poor performance status
Advanced stage of primary malignancy (previous treatments, availability of anticancer options, life expectancy)
Malnutrition/cachexia
Comorbidities
Absence of psychosocial support
Ascites (>100 cc associated with poor outcome)
Single vs. multiple sites of obstruction
Carcinomatosis (believed to respond poorly to surgical intervention)

Volvulus

- Volvulus indicates that a loop of bowel is twisted more than 180 degrees about the axis of its mesentery
- Volvulus has been reported for the cecum, transverse colon, splenic flexure, and sigmoid colon.
- A special variant of volvulus, complicating a condition known as Chilaiditi syndrome, can occur when redundant loops of the transverse colon slip between the liver and diaphragm and then undergo torsion.
- the most common radiographic feature is the “bent inner tube” appearance of the sigmoid, which is located in the upper abdomen.

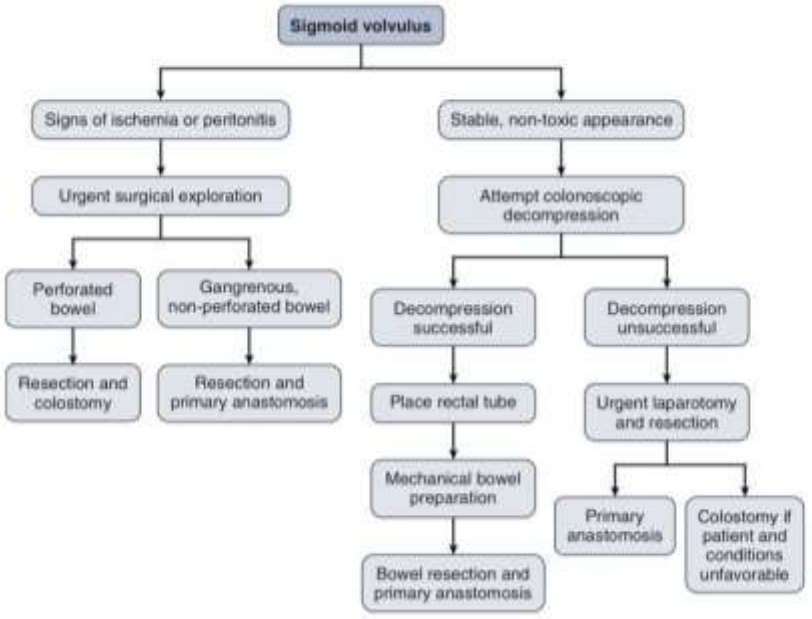


FIGURE 2 Suggested algorithm for management of sigmoid volvulus.

[11]

Role of Laparoscopy in Small Bowel Obstruction [10]

- Laparoscopic management of bowel obstruction provides many potential benefits including quicker recovery of bowel function, shorter hospital stay, less postoperative pain, reduced recovery time and early return to full activity, and fewer postoperative complications including a decreased incidence of wound infection and pneumonia
- clinical and experimental studies in animal models, laparoscopy is associated with decrease in incidence, extent and severity of intra-abdominal adhesions compared with open surgery

- laparoscopic management of bowel obstruction may result in a decreased lifetime risk for recurrent bowel obstruction
- Studies of laparoscopic lysis of adhesions for SBO indicate that it is feasible, with acceptable operative times, length of hospital stay, as well as conversion and complication rates
- Laparoscopy is discouraged when the surgeon is uncomfortable with the technique or in patients presenting with peritonitis, hemodynamic instability, severe co morbid conditions, complete and distal obstruction, or when contraindications to pneumoperitoneum exist
- Open exploratory laparotomy is the gold standard in treating unresolved SBO, but laparoscopic management should be considered in select group of patients.

MATERIAL AND METHODS

- All the cases diagnosed as Acute intestinal obstruction and admitted to various surgical wards in GOVT RAJAJI HOSPITAL, MADURAI from May 2018 to May 2019, and managed surgically have been studied.

- Patients belonged to the age groups ranging from 12 years to 65 years; paediatric age group is excluded from this study.

- The criteria for selection of cases were based on clinical history, physical findings, radiological and haematological investigations.

- Patients who were having sub acute intestinal obstruction treated conservatively were excluded from the study, and only those cases of acute intestinal obstruction which were managed surgically were studied to establish the pathology of intestinal obstruction with an aim to know the operative findings and outcome of acute intestinal obstruction.

- After the admission of the patient, clinical data were recorded as per Proforma.
- The diagnosis mainly based on clinical examination and haematological and radiological examinations.

Methods

a. Clinical study

b. Investigations

c. Treatment

a. History taking

b. Physical examination

c. Laboratory examination

d. Radiological examination – Plain X-ray erect abdomen

e. Ultrasound examination in selected cases

f. Surgical treatment and results

g. Follow-up

a. History taking

A complete history was obtained from the patient and the

complaints entered in the proforma in a chronological order. Each complaint in the history of presenting illness was documented in detailed enquiry.

b. physical examination

(i) General physical examination – evidence of dehydration and the severity of it was looked into it and vital parameters were recorded.

(ii) Local examination – Abdominal examination was done under standard headings inspection, palpation, percussion and palpation. Per rectal examination was done and findings were noted.

(iii) Systemic examination – All other systems were examined carefully to rule out associated anomalies and to assess the fitness for surgery.

c. Laboratory examination

(i) Haemoglobin

(ii) TC & DC

(iii) BT and CT

(iv) Blood grouping and Rh typing

(v) Urine for albumin and microscopy

d. Radiological examination

Erect abdomen X-ray done in all cases, barium enema and ultrasound examination in selected cases.

SURGICAL MANAGEMENT

- Immediately after the admission along with above procedure resuscitation with IV fluids especially ringer lactate and normal saline infusion started till the hydration and urine output become normal.

- Nasogastric decompression with Ryles tube carried out and antibiotic prophylaxis given.

All clinical parameters (like pulse rate, BP, RR, urine output, urine output, abdominal girth, bowel sounds and tenderness and guarding) was done.

- Patients who showed reduction in abdominal distension and improvement in general condition especially in individuals with postoperative adhesions conservative management was confined (by extending the supportive treatment) for next 24 hours, those who showed improvement by moving bowels, reduction in pain/tenderness

were decided for conservative treatment, such individuals are excluded from this study.

- Patients with signs and symptoms of acute Intestinal obstruction

was managed appropriate surgical procedure after resuscitation.

- Surgery adopted and criteria for deciding the procedure were noted, e.g. release of band or adhesion, resection and anastamosis for gangrenous bowel and release and repair for strangulated obstruction. Stoma was kept for suspected cases with poor factors for a healthy anastomosis

- Histopathological examination of the

specimen of resection/biopsy was done whenever necessary.

- The postoperative period was monitored carefully.

- Postoperatively Ryle's tube aspiration, intravenous fluids and antibiotics were administered. Any complications noted and treated accordingly

.

- Postoperative follow up after the discharge of patients was done in majority of the patients upto 3 months.

- The results are tabulated stressing on following points

Probable causative factors, operative findings and operative procedure adopted and complications if any

.

Statistical Methods:

- Data will be entered in Microsoft Excel and analyzed using SPSS software latest version
- Data on continuous scale will be represented as MEAN and STANDARD DEVIATION
- Categorical datas will be represented as NUMBERS and PERCENTAGES
- Results will be represented as Graphs and Tables

DATA ANALYSIS AND RESULTS

The outcome of acute intestinal obstruction in adult age group, who were managed surgically, was studied from the cases admitted in Department of Surgery of GOVERNMENT RAJAJI HOSPITAL, MADURAI from MAY 2018 to MAY 2019.

75 cases of intestinal obstruction managed surgically have been studied.

During the period of 1 year, the total number of cases admitted in surgery opd/Casualty requiring emergency Surgery was 954, among which 75 cases were diagnosed as Acute Intestinal Obstruction due to various Aetiology requiring emergency Surgery.

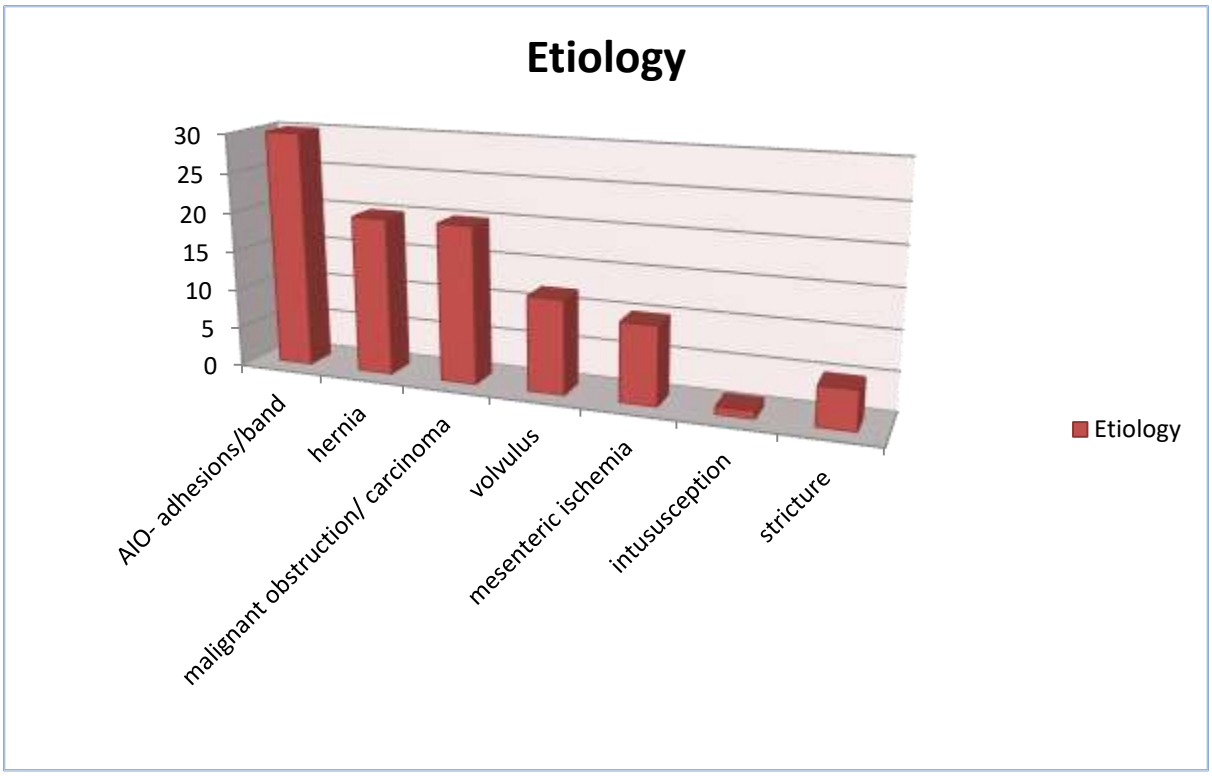
Thus Acute Intestinal Obstruction forms 7% of the cases admitted in Surgery block requiring Emergency Surgery

Incidence of Different Aetiology

The incidence of different etiologies of Intestinal Obstruction in the present series is

Tab.1 Various Aetiology of intestinal Obstruction in our study

Causes	No of cases	Percentage
Adhesions/band	23	30%
Hernia	15	20%
Malignant Obstruction	15	20%
Volvulus	9	12%
Mesenteric Ischemia	8	10%
Intususception	1	1%
Stricture	4	5%



The most common Aetiology of Intestinal Obstruction in our study was Post operative Adhesions/bands followed by Hernia, Malignant obstruction , Volvulus, Mesenteric Ischemia , Stricture , Intususception in descending order

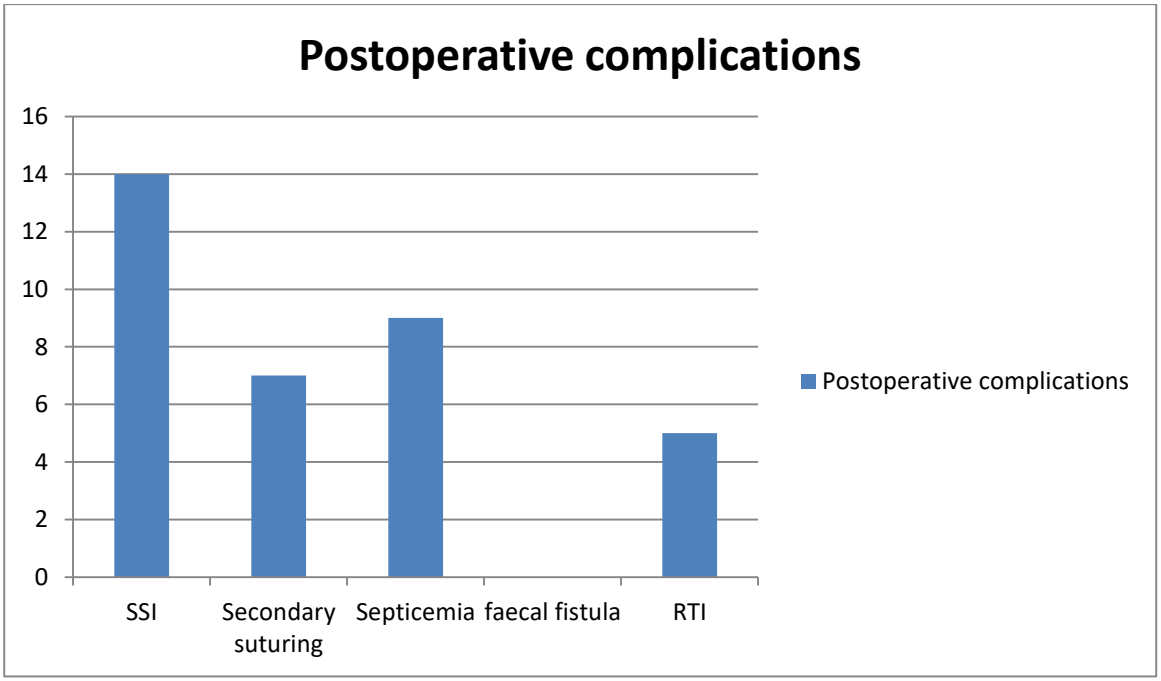
Tab 2. Surgical Management

Management	No of Cases
Adhesiolysis	21
Resection and Anastomosis	17
Hernia repair	9
Hartmann's procedure	7
Hemicolectomy	4
Ileostomy	6
Colostomy	6
Jejunostomy	2
Reduction	1

In our Series of 75 cases of acute intestinal Obstruction due to various Aetiology, appropriate surgical management was done as listed in the table. Few cases required more than one procedure. Among the procedure done, Adhesiolysis was the most common followed by resection of the diseased segment. Certain cases required a radical procedure like hemicolectomy.

Tab 3. Postoperative Complications

Postoperative complication	No of cases
Surgical site infection	13
Secondary suturing	7
Septicaemia	9
RTI	5

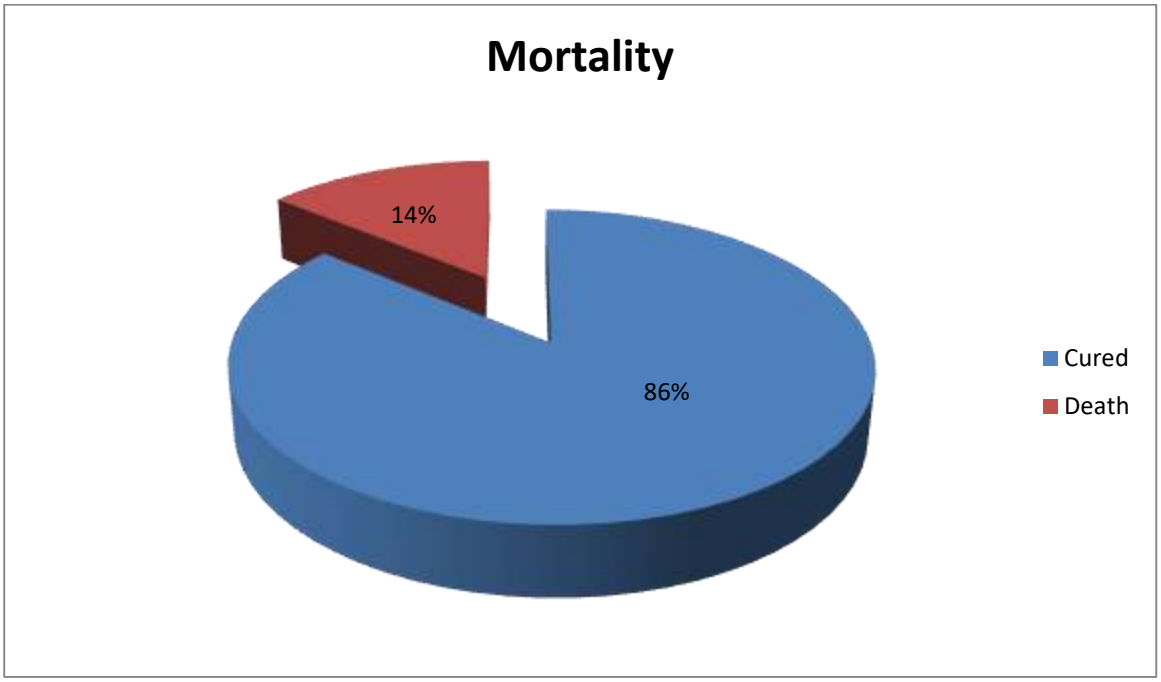


In the present series, a lot of cases were associated with surgical site infections (14), with 7 cases among them requiring secondary suturing. 9 cases developed Septicaemia related to the cause of the obstruction as a result of the ongoing pathology. 5 cases had associated RTI which was

secondary to the associated COPD or prolonged elective post op ventilation.

Tab 4. Mortality in our study

	No of cases	Percentage
Cured	64	85%
Death	11	14%



S.No	Case	Age/sex	Diagnosis	Procedure	Cause of Death
1	Case no 7	60/f	CA colon /Malignant Obstruction	Hartmann's procedure	Septicaemia
2	Case no 12	38/M	Mesenteric Ischemia	Resection and Anastomosis	Septicaemia
3	Case no 16	48/M	Mesenteric Ischemia	Resection and Anastomosis	Septicaemia
4	Case no 24	40/M	Acute Intestinal Obstruction with bowel Gangrene/?Mesenteric Ischemia	Jejunostomy	Septicaemia
5	Case No 42	57/M	Ca colon/Malignant Obstruction	Loop Ileostomy	Septicaemia
6	Case no 44	56/M	Acute Mesenteric Ischemia	Resection and Anastomosis	Septicaemia
7	Case no 54	55/M	Acute Mesenteric Ischemia	Ileostomy	Septicemia, COPD
8	Case no 56	65/F	Acute Mesenteric Ischemia	Jejunostomy	Septicaemia
9	Case no 58	39/M	Acute Mesenteric Ischemia	Resection and Anastomosis	Septicaemia
10	Case no 66	47/M	Acute Intestinal Obstruction /Ileal gangrene	Ileostomy	Septicaemia
11	Case no 69	52/M	Sigmoid volvulus	Hartmann's Procedure	Septicaemia , COPD

In Our case Series, among the 75 cases of intestinal obstruction operated, 11 patients expired during the post operative period. Without a doubt, Acute Mesenteric Ischemia was the deadliest of all the causes and most of the patients of Mesenteric Ischemia got expired in the post operative period due to the delay in the diagnosis and the intra abdominal sepsis related to the aetiology.

Follow up:

Tab 5. Follow up Status

Follow up	2 weeks	1 months	3 months
Wound Infection	4	Nil	Nil
Septicaemia	Nil	Nil	Nil
Enterocutaneous Fistula	Nil	Nil	Nil
Prolonged Ileus	Nil	Nil	Nil
Fever	2	Nil	Nil
Respiratory Infection	2	Nil	Nil
Death	Nil	Nil	Nil

DISCUSSION

Acute intestinal obstruction continues to be the most common Surgical emergency. In our study a total number of 954 patients, who were? Admitted in the surgical casualty department from June 2018 to June 2019 required emergency surgical management. Among these 75 cases were diagnosed as Acute Intestinal Obstruction and operated. These were the patients who were included in the study

Disease burden

In our study, the Incidence of Acute intestinal Obstruction was 7% of the total surgical cases operated as emergency during the period of study. In Souvik Adhikari et al. series incidence was 9.87% of total surgical cases

Tab 6. Comparison of Aetiology with other Studies

Causes	Souvik Adhikari[12]	Jahangir[13]	Arshad M Malik[14]	Present Study
Adhesions	16%	49%	41%	30%
Hernia	36%	34%	19%	20%
Volvulus	6%	5%	4%	12%

Malignancy	17%	3%	2%	20%
Mesenteric ischemia	9%	2%	10%	10%
Intussusceptions	2%	6%	-	1%

In Souvik Adhikari et al, the commonest aetiology was Hernia (16%). In our study the commonest aetiology was post operative adhesions (30%) which were comparable with Arshad malik et al. (41%) and Jahangir et al (49%)

The commonest cause in our study was found to be postoperative adhesions followed by obstructed/ strangulated hernia, malignancy, Volvulus, Mesenteric ischemia, stricture and intussusception.

Though Hernia is the commonest cause of intestinal obstruction in developing countries. In our study, post operative adhesions remained the commonest cause

Firstly, the reason being the poor socioeconomic status of the people included in the study and their affordability to minimal access surgery.

Second, most of the previous surgeries were done during the open era.

And finally due to the development of Health care in the state, most of the people with hernias are diagnosed at an early stage (before obstruction/strangulation) and operated on elective basis

Mortality

Tab 7. Mortality rate in other studies

Studies	Year	No .of Cases Studied	Mortality
Souvik Adhikary[12]	2005	367	7.35%
Jahangir[13]	2001	100	7%
Arshad M Malik[14]	2009	229	3%
Present Study	2019	75	14%

In our study, the mortality was found to be 14% i.e. 11 out of 75 cases

Among the 11 cases, 9 of them were Acute Mesenteric Ischemia and the remaining were malignant obstruction and sigmoid volvulus

On comparing the Mortality with other studies, our percentage was on higher side

These are attributable to various factors like delayed presentation to the hospital, delay in diagnosis.

Acute Mesenteric Ischemia is increasingly becoming a common cause as in our study, the low suspicion and the delay in diagnosis, significantly increases the risk of septicaemia and poor prognosis

One case was due to malignant obstruction, who due to the tumour itself presented severely emaciated, with Liver metastasis

The other case was a delayed presentation (>72hours) to the hospital, which was a case of sigmoid volvulus, readily seen on X-ray. But the septicaemia was severe that resulted in the death of the patient.

Tab 8. Outcome of the treatment in relation to the Duration of symptoms

Duration of symptoms	Total no of cases	Death
1-2 days	48	1
3-4 days	18	1
>5days	9	9

CONCLUSION

- Acute Intestinal Obstruction, is a common surgical problem , that accounts for a large percentage of Surgical admission for acute abdomen

- One of the difficult task as a General Surgeon is in deciding when to operate on a patient with Intestinal Obstruction

- Adhesions still remain as one of the commonest cause for Acute intestinal Obstruction

- Though X ray is a valuable modality, Computed Tomography is increasingly becoming the “go-to” imaging modality to identify the source of obstruction.

- Open Exploratory Laparotomy is the gold standard in treating unresolved intestinal obstruction, but laparoscopy should be considered in a selective group.

KEY TO MASTER CHART

M - Male

F - Female

CA - Carcinoma

R & A - Resection and Anastomosis

C - Cured

SSI - Surgical Site Infection

SS - Secondary suturing

RTI - Respiratory tract Infection

AIO – Acute Intestinal Obstruction

MASTER CHART

S No	NAME	AGE	SEX	DIAGNOSIS	TREATMENT	CURED	COMPLICATIONS	DEATH
1	KUPUTHAI	65	F	SIGMOID VOLVULUS	R & A	C		
2	RAMU	60	M	OBSTRUCTED UMBLICAL HERNIA	R & A , ANATOMICAL REPAIR	C		
3	RIYAS	64	M	SIGMOID VOLVULUS	R & A	C	SSI	
4	PERUMALAMMAL	55	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS, R&A	C		
5	MUNIYAMMAL	58	F	OBSTRUCTED INCISIONAL HERNIA	R & A,	C		
6	ANNAPOORANI	60	F	CA COLON /MALIGNANT OBSTRUCTION	HARTMANN' S PROCEDURE		SEPTICEMIA	DEATH
7	SANTHANA MARY	65	F	OBSTRUCTED INCISIONAL HERNIA	R & A	C	SSI	
8	PANDI	57	M	OBSTRUCTED INGUINAL HERNIA	R &A, HERNIORAPPHY	C		
9	ALAGARSAMY	45	M	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
10	ESWARAN	45	M	AIO/MESENTERIC ISCHEMIA	R, END COLOSTOMY	C	RTI, SSI	
11	BALASUBRAMANIAN	40	M	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS, R&A	C		
12	SHIVA	38	M	MESENTERIC ISCHEMIA	R&A		SEPTICEMIA	DEATH
13	DEIVANAI	60	F	CA COLON /MALIGNANT OBSTRUCTION	HEMICOLECTOMY	C	SSI	
14	PUSHPAM	63	F	OBSTRUCTED UMBLICAL HERNIA	R &A, ANATOMICAL REPAIR	C		
15	MUTHURAM	60	M	SIGMOID VOLVULUS	HARTMANN' S PROCEDURE	C	SS,RTI	
16	RAMU	48	M	MESENTERIC ISCHEMIA	R&A		SEPTICEMIA	DEATH
17	MAYALAGU	65	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
18	PITCHAIMANI	45	M	CA COLON /MALIGNANT OBSTRUCTION	HEMICOLECTOMY	C	SS, RTI	
19	TAMILSELVI	47	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
20	SELVI	52	F	CA COLON /MALIGNANT OBSTRUCTION	ILEOSTOMY	C		
21	SARASWATHI	57	F	INCARCERATED INCISIONAL HERNIA	R&A	C	SSI	
22	SELVAMANI	35	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
23	VASANTHI	49	M	SIGMOID VOLVULUS	HARTMANN' S PROCEDURE	C	SSI	
24	DHANDAYUTHAPANI	40	M	ACUTE INTESTINAL OBSTRUCTION	JEJUNOSTOMY		SEPTICEMIA	DEATH
25	PANDI	38	M	AIO/STRICTURE DESCENDING COLON	R & A	C		
26	KUMAR	55	M	CA SIGMOID COLON/MALIGNANT OBSTRUCTION	HARTMANN' S PROCEDURE	C	SSI,SS	
27	SELVAM	53	M	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
28	KARUPAYEE	65	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
29	SEENIMUTHU	65	M	OBSTRUCTED INGUINAL HERNIA	R & A , HERNIORRAPHY	C		
30	VIKRAM	16	M	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
31	JEGADEESAN	31	M	AIO/ILEAL STRICTURE	R&A	C		
32	DEVI	28	F	SIGMOID VOLVULUS	R & A	C		
33	PANDI KUMAR	43	M	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
34	RAVI	45	M	CA COLON /MALIGNANT OBSTRUCTION	DIVERSION COLOSTOMY	C		
35	ANDAL	48	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
36	KALIAMMAL	55	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
37	RAJA	40	M	AIO/ILEAL STRICTURE	R& A	C		
38	KARUPASAMY	64	M	SIGMOID VOLVULUS	HARTMANN' S PROCEDURE	C	SSI	
39	MEENATCHI	60	F	OBSTRUCTED INGUINAL HERNIA	R & A, HERNIORRAPHY	C		

40	JEYARANI	36	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
41	KUMARANGI	65	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
42	ELANGOVAN	57	M	CA COLON /MALIGNANT OBSTRUCTION	LOOP ILEOSTOMY		SEPTICEMIA	DEATH
43	CHINNAMMAL	40	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS, R&A	C		
44	RAJENDIRAN	56	M	AIO/MESENTERICA ISCHEMIA	R&A		SEPTICEMIA	DEATH
45	SUDHAKAR	22	M	INTUSUSSCEPTION	REDUCTION	C		
46	KALAVATHI	57	F	INCARCERATED UMBLICAL HERNIA	R&A ,ANATOMICAL REPAIR	C		
47	*SHANKAR	58	M	CA RECTUM/MALIGNANT OBSTRUCTION	DIVERSION COLOSTOMY	C	SSI,SS	
48	SYED MEERAN	65	M	CA COLON /MALIGNANT OBSTRUCTION	DIVERSION COLOSTOMY	C		
49	AMMASI	60	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
50	JEYAKUMAR	38	M	AIO/ SMV THROMBOSIS	R&A, LOOP ILEOSTOMY	C		
51	AMUTHA	52	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS, R&A	C	SS	
52	KUMARAVALLI	54	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
53	PONNUTHAI	55	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS	C		
54	CHINNAYE	55	F	MESENTERIC ISCHEMIA	ILEOSTOMY		RTI, Septicaemia	DEATH
55	ARUMUGAM	60	M	CA COLON /MALIGNANT OBSTRUCTION	HARTMANN' S PROCEDURE	C		
56	SIRUMANI	65	F	MESENTERIC ISCHEMIA	JEJUNOSTOMY		SEPTICEMIA,RTI	DEATH
57	ADAIKAN	56	M	CA ANAL CANAL /MALIGNANT OBSTRUCTION	DIVERSION COLOSTOMY	C		
58	KALIRAJAN	39	M	MESENTERIC ISCHEMIA	R&A		SEPTICEMIA	DEATH
59	SELVAM	54	M	OBSTRUCTED INGUINAL HERNIA	R& A , HERNIORAPPHY	C		
60	KOODALINGAM	51	M	CA COLON /MALIGNANT OBSTRUCTION	HEMICOLECTOMY	C	SSI,SS	
61	CHITRA	47	F	CA COLON /MALIGNANT OBSTRUCTION	HEMICOLECTOMY	C	SSI	
62	RAKKU	65	F	OBSTRUCTED FEMORAL HERNIA	R& A, HERNIORAPPHY	C		
63	MAGESWARI	65	F	CA RECTUM/MALIGNANT OBSTRUCTION	DIVERSION COLOSTOMY	C	SSI ,SS	
64	RANGASAMY	62	M	OBSTRUCTED INGUINAL HERNIA	R & A, HERNIORRAPHY	C		
65	ALAMELU	65	F	OBSTRUCTED FEMORAL HERNIA	R& A, HERNIORAPPHY	C		
66	VINCENT	47	M	AIO/ILEAL GANGRENE	R & A, ILEOSTOMY		SSI	DEATH
67	SUBBIAH	55	M	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS, R&A	C		
68	MANOHARAN	36	M	CA COLON /MALIGNANT OBSTRUCTION	R, ILEOSTOMY	C		
69	MUNIRAJ	52	M	SIGMOID VOLVULUS	HARTMANN' S PROCEDURE		SEPTICEMIA,RTI	DEATH
70	KADHAR MEERAN	65	M	OBSTRUCTED INGUINAL HERNIA	R&A , HERNIORRAPHY	C		
71	BABU	55	M	OBSTRUCTED INGUINAL HERNIA	R& A, HERNIORAPPHY	C		
72	KANNAN	55	M	AIO/ILEAL STRICTURE	R& A	C		
73	LAKSHMI	38	F	SIGMOID VOLVULUS	R&A	C	SSI	
74	PASUPATHI	65	F	SIGMOID VOLVULUS	R&A	C		
75	MARIYAMMAL	56	F	ACUTE INTESTINAL OBSTRUCTION	ADHESIOLYSIS, R&A	C		

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PROFORMA (ANNEXURE I)

Name: - I. P. No

Age: - Unit

Sex: - D.O.A

Occupation : - D.O.D

Address :-

Phone no :

DIAGNOSIS:

PRESENTING COMPLAINTS

- site of abdominal pain
- duration of abdominal pain
- abdominal distension
- constipation
- vomiting
- duration of fever
- history of trauma
- history of smoking and alcohol abuse
- history of any drug intake
- co existing co morbidities
- Treatment history of peptic ulcer

General Examination:

- 1. General survey
- 2. Body build and nourishment
- 3. Appearance
- 4. Attitude: Restless/ Quiet
- 5. Dehydration: Mild/ Moderate/ Severe/ Nil
- 6. Anemia/ Jaundice/ Clubbing/ Cyanosis/Lymphadenopathy/ Pedal edema
- 7 Pulse
- 8. Temperature
- 9. Respiratory rate
- 10. Blood pressure

Examination of Abdomen

- 1. INSPECTION
- 2. PALPATION
- 3. PERCUSSION
- 4. AUSCULTATION
- SYSTEMIC EXAMINATION - CVS/ RS/CNS
- Per rectal examination

CONSENT (ANNEXURE II)

I_____ Hosp No _____ in my full senses hereby give my complete for _____ or any other procedure deemed fit which is a diagnostic/therapeutic/procedure/biopsy/transfusion/operation to be performed on me/my/son/daughter/ward _____ age_____ under any anaesthesia deemed fit. The nature and risks involved in the procedure have been explained to me in my own language to my satisfaction. For academic and scientific purpose, the operation/procedure be Television or photographed, or used for statistical measurements.

Date:

Signature/Thumb Impression/ of Patient/Guardian

Name:

Designation:

Guardian

Relationship

Full Address

(ANNEXURE – III)

URKUND

Urkund Analysis Result

Analysed Document:	pliag - int obstruction - thesis.docx (D57179362)
Submitted:	10/17/2019 2:48:00 PM
Submitted By:	mdurai07@gmail.com
Significance:	6 %

(ANNEXURE – IV)

MADURAI MEDICAL COLLEGE
MADURAI, TAMILNADU, INDIA -625 020
(Affiliated to The Tamilnadu Dr.MGR Medical University, Chennai, Tamil Nadu)

Prof Dr V Nagaraajan MD MNAMS DM (Neuro) DSc.,(Neurosciences) DSc (Hons)
Professor Emeritus in Neurosciences, Tamil Nadu Govt Dr MGR Medical University
Chairman, IEC

Dr.K.Raadhika, MD.,
Member Secretary,
Asso.Professor of Pharmacology, Madurai Medical College, Madurai.

Members

1. Dr.C.Anitha Mohan, MD, Asso.Professor of Physiology & Vice Principal, Madurai Medical College

2. Dr.P.Raja, MCh., Urology, Medical Superintendent Govt. Rajaji Hospital, Madurai

3.Dr.R.Balajinathan MD., (General Medicine) Professor & HOD of Medicine, Madurai Medical & Govt. Rajaji Hospital, College, Madurai.

4.Dr.P.Amutha, MS., (General Surgery) Professor & H.O.D Madurai Medical College & Govt. Rajaji Hospital, Madurai.

5.Dr.N.Sharmila thilagavathi, MD., Professor of Pathology, Madurai Medical College, Madurai

6.Mrs.Mercy Immaculate Rubalatha, M.A., B.Ed., Social worker, Gandhi Nagar, Madurai

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8.Thiru.P.K.M.Cheillah, B.A., Businessman,21, Jawahar Street, Gandhi Nagar, Madurai.

ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.M.Durai Raj kumar
Designation	:	PG in M.S., General Surgery
Course of Study	:	2017- 2020
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	A prospective study on management of acute intestinal obstruction and its outcome in adults in Govt Rajaji Hospital, Madurai
Ethical Committee as on	:	25.04.2019

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary Chairman Dean / Convenor

Prof Dr V Nagaraajan
M.D., MNAMS, D.M., Dsc.(Neuro), Dsc (Hon)
CHAIRMAN
IEC - Madurai Medical College
Madurai

DEAN
Madurai Medical College
Madurai-20

03 MAY 2019

CERTIFICATE (ANNEXURE –V)

This is to certify that this dissertation work titled **A PROSPECTIVE STUDY ON MANAGEMENT OF ACUTE INTESTINAL OBSTRUCTION AND ITS OUTCOME IN ADULTS IN GOVT. RAJAJI HOSPITAL, MADURAI** of the candidate **DURAI RAJ KUMAR.M** with Reg.No **221711103** for the award of Master Degree in the branch of General Surgery. I have personally verified the urkund.com website for plagiarism check. I found that the uploaded thesis file contains all from introduction to conclusion pages and results shows **SIX (6%)** percentage plagiarism in the dissertation

Guide and Supervisor Sign and seal