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Харківський національний медичний університет

Abdominal Surgery.
Contents module 1

ACUTE INTESTINAL OBSTRUCTION

Guidelines for students and interns

Абдомінальна хірургія
Змістовний модуль 1

ГОСТРА КИШКОВА НЕПРОХІДНІСТЬ

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Intestinal obstruction exists when prevents the normal flow of intestinal contents through the intestinal tract.

1. CLASSIFICATION AND DEFINITIONS

Types:

Mechanical obstruction:

- Obturation/obstruction;
- Strangulation ileus.
- Mixed (intussusception).

Functional obstruction:

- Spastic ileus;
- Paralytic ileus, including ileus as a result of mesenteric thrombosis.

Stages of acute intestinal obstruction:

- Stage of acute disturbance of intestinal evacuation and peristalsis (12–16 hours from the beginning of the disease).
- Stage of hemodynamic disorders of the bowel wall and its mesentery (16–36 hours from the beginning of the disease).
- Stage of peritonitis (more than 36 hours from the beginning of the disease).

Mechanical obstruction. An intraluminal obstruction or a mural obstruction from pressure on the intestinal walls occurs. Examples are volvulus of the intestine, intussusception, polypoid tumors and neoplasms, stenosis, strictures, adhesions, hernias, etc (*Fig. 1*).

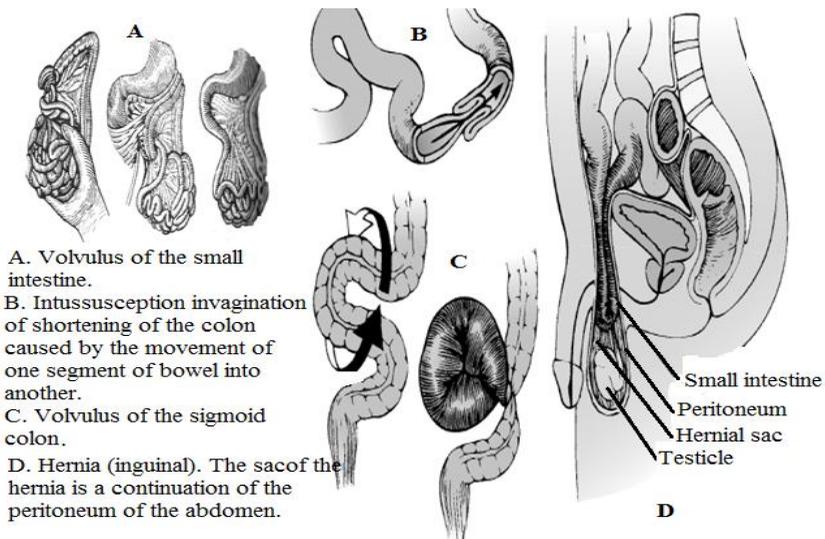


Figure 1. Four causes of the intestinal obstruction

Functional obstruction. The intestinal musculature cannot propel the contents along the bowel. Examples are peritonitis and others irritations of intestine, pancreatitis, amyloidosis, muscular dystrophy, endocrine disorders such as diabetes mellitus, or neurologic disorders, impact of some poisons.

Conclusions. The obstruction can be partial or complete. Its severity depends on the region of bowel affected, the degree to which the lumen is occluded, and especially the degree to which the vascular supply to the bowel wall is disturbed.

Intestinal obstruction may be functional due to disruption of the intestinal wall or articular nerve dysfunction, or mechanical, due to a mechanical barrier. Obstruction may occur with obstruction of the small intestine (SBO) or large intestinal obstruction (LBO). Most intestinal obstructions occur in the small intestine. Adhesion is the most common cause of small intestinal obstruction accompanied by hernias and neoplasms. Other causes include intussusception, curvature (bowel twisting), and paralytic ileus. About 15 % of intestinal obstructions are found in the large intestine. Most of them are in the left half of the large intestine.

Acute functional dilatation of the colon is referred to as “colonic pseudo-obstruction”. Acute functional small bowel dilatation is referred to as “adynamic or paralytic ileus”. Small intestinal pseudo-obstruction describes a clinical syndrome characterized by manifestations of mechanical bowel obstruction in the absence of an obstructive lesion. A multitude of conditions cause functional bowel obstruction. Mechanical SBO may be due to a luminal, mural, or extra-mural mechanical barrier. Mechanical SBO may be proximal (high SBO) or distal (low SBO), closed loop or open-ended obstruction. In closed loop obstruction the lumen of the bowel is occluded at two points, thus, preventing prograde and retrograde movement of bowel contents. In open-ended obstruction a one-point obstruction interferes with the prograde propulsion of bowel contents.

Bowel obstruction may be partial or complete, simple or complicated. Partial obstruction allows some liquid contents and gas to pass through the point of obstruction, whereas complete obstruction impedes passage of all bowel contents. Unlike simple obstruction, complicated obstruction indicates compromise of the circulation to a segment of bowel with resultant ischemia, infarction, and perforation.

Intussusception is a unique type of obstruction that results from invagination of a segment of bowel into another. It may occur anywhere along the gastrointestinal tract distal to the gastric cardia. Intussusception may occur in a downward direction or may be retrograde. The exact mechanism of colic and enterocolic intussusception is not known but an organic lesion, diseased segment of bowel, or an adjacent area of normal bowel may serve as a lead

point in initiating the process. Accordingly, intussusception is classified into idiopathic, postoperative, and intussusception due to an organic lesion. In adults, a neoplasm is the lead point in 80–90% of cases. A Meckel's diverticulum (MD) may invaginate into the ileum and sometimes, thence, into the colon. Volvulus is axial twist of the gastrointestinal tract around its mesentery resulting in partial or complete luminal obstruction (closed loop) of the bowel and a variable degree of arterial or venous obstruction. Volvulus commonly occurs in the colon and may affect the stomach or SB. Volvulus occurs when the small bowel twists around a MD that is attached by a fibrous cord to the umbilicus, or when a closed loop obstruction twists along its long axis. Gallstone ileus is a mechanical bowel obstruction caused by migration of gallstones from the biliary system through a biliary-enteric fistula with impaction within lumen of the bowel. Littre's hernia is incarcerated MD in an external hernia.

2. PATHOPHYSIOLOGIC CHANGES

Acute small bowel obstruction (SBO) results in local as well as systemic physiologic and pathologic derangements. Significant partial or complete obstruction is associated with increased incidence of migrating clustered contractions (MCC) proximal to the site of obstruction. Such contractions are associated with abdominal cramps. With partial obstruction MCC propel intraluminal contents and allow them to pass distal to the point of obstruction. With complete unrelieved obstruction, bowel contents fail to pass distally, with resultant progressive accumulation of intraluminal fluids and distention of the proximal bowel. This eventually initiates retrograde giant contractions (RGC) in the small bowel (SB) as the first phase of vomiting. In adynamic ileus migratory motor complexes (MMC) (contractions initiated in the stomach and proximal SB occur almost simultaneously and propagate distally to clear the intestine of secretions and debris) and fed contractions (intermittent and irregular contractions that provide mixing and slow distal propulsion) are inhibited.

As intraluminal pressure in the bowel proximal to the obstruction increases, venous flow in the bowel wall and adjacent mesentery decreases, and ceases if pressure reaches systolic pressure. Blood flow to the mucosa decreases, followed by capillary rupture and hemorrhagic infiltration. A twist of the mesentery or direct pressure on the mesenteric vessels results in venous and/or arterial occlusion. Intestinal epithelium is very vulnerable to anoxia and is the first to suffer necrosis. Perforation may occur as a result of ischemic or pressure necrosis. Pressure necrosis may occur at site where a tight band adhesion passes across a loop of bowel, or where an impacted gall stone or fecoloma produces stercoral ulceration and subsequent perforation. In simple

obstruction the bowel proximal to the obstruction appears heavy, edematous, and even cyanosed. In advanced cases serosal tears appear at the antimesenteric border of the bowel.

Acute SBO results in volume depletion and electrolyte disturbances. Intestinal contents are cut off from the absorptive surface of the colon. Further loss of volume occurs as bowel contents stagnate in the dilated loops of obstructed bowel, lost through vomiting, or sequestered in the bowel wall or peritoneal cavity. Water loss is accompanied by electrolyte loss, and depending upon the level of obstruction specific electrolyte concentration changes. As intraluminal pressure increases, absorption of water and sodium decreases and luminal secretion of water, sodium, and potassium increases. In addition there is edema of the bowel wall and leakage of proteins. With strangulation, protein and electrolyte rich exudate accumulate in the peritoneal cavity, and with infarction sequestration of blood in bowel wall occurs. The peritoneal fluid exudate changes from plasma-like clear fluid, to bloody, then foul dark exudate. There is also change in the ecology of bacterial population with increase fecal type of bacterial colonies in the bowel proximal to the obstruction and altered proximal-to-distal gradient change in bacterial flora. Bacterial breakdown of stagnant bowel contents results in formation of "feculent fluid". With strangulation physiologic changes are complicated by blood loss in the infarcted bowel, death of tissues, gut translocation of bacteria and toxins, and the final insult of perforation.

3. CLINICAL FEATURES

The initial symptom is usually crampy pain that is wavelike and colicky. The patient may pass blood and mucus, but no fecal matter and no flatus. Vomiting occurs. If the obstruction is complete, the peristaltic waves initially become extremely vigorous and eventually assume a reverse direction, with the intestinal contents propelled toward the mouth instead of toward the rectum. If obstruction is in the ileum, fecal vomiting takes place. The abdomen becomes distended. The lower the obstruction is in the GI tract, the more marked the abdominal distention. If the obstruction continues uncorrected, hypovolemic shock occurs from dehydration and loss of plasma volume.

Complains:

- Pain: suddenly arising; paroxysmal.
- Nausea, vomiting.
- Abdominal distension is typical for mechanical and dynamic intestinal obstruction.
- Constipation, gas formation and absence of gas evolution.

Objective signs of disease:

- Inspection: hemodynamic parameters; state of nutrition, behavior, skin color, and turgor and warmth of the skin.
- Tongue is initially wet, with development of dehydration it becomes dry.
- Abdomen is asymmetric, distended.
- Palpation of the anterior abdominal wall: characteristics and localization of pain, symptoms corresponding pathognomonically to intestinal obstruction are evaluated.
- Percussion of the anterior abdominal wall: tympanitis across the whole abdominal wall.
- Auscultation: during attacks of colic, the sounds become loud, high-pitched and metallic.

Pathognomonic signs:

- **Wahl's sign:** asymmetry of the abdomen, high tympanic sound over the distended bowel.
- **Sklyarov's sign:** splashing sounds in the small or large intestine during balloting palpation of anterior abdominal wall.
- **Kywul's sign:** metallic sound over bloated intestinal loop during percussion of plesimer.
- **Schlange's sign:** visible peristals.
- **Spasokukotsky's sign:** during auscultation "sound of falling drop" above the stretched intestinal loops.
- **Tsege Manteuffel's sign:** when setting the siphon enema all entered the liquid (up to 500 ml) quickly leaves the intestine does not contain impurities of feces and gases). Positive at sigmoid volvulus.

4. DIAGNOSIS

Radiologic studies

X-ray. Plain radiograph of the abdomen is the most valuable initial diagnostic test in acute intestinal obstruction. This imaging method gives diagnostic information of in 50–60 % of cases and provides enough information needed for clinical decision making (**Kloiber's cups**).

The **Kloiber's cups** (air-fluid level) is the main sign of mechanical obstruction of the small intestine. In the small intestine they are low and wide, located closer to the spine. In the colon they are located in a circumferential direction of the abdominal cavity, their number is lesser, the cups are higher, but not wider, and on the gas background mucosal folds are seen in the loops of intestine (**Kerckring's folds**). In dynamic obstruction horizontal fluid levels are located in the loops of small intestine as well as large one.

In 20–30 % the radiographic findings are equivocal, and in 10–20 % are normal (*Fig. 2, 3*). The typical air fluid levels seen in the dilated bowel

proximal to the obstruction may be absent in high acute intestinal obstruction, closed loop obstruction or late obstruction (*Fig. 4*). Low grade obstruction is difficult to assess with plain radiograph of the abdomen.



Figure 2. X-ray abdominal: there are multiple intestinal air fluid level in erect film of abdomen (Kloiber's cups)



Figure 3. X-ray: ileus involving small and large intestine

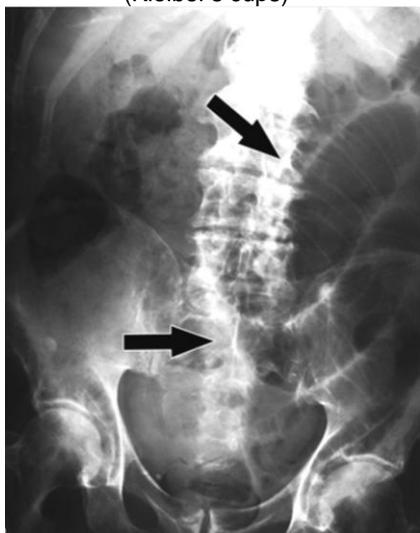
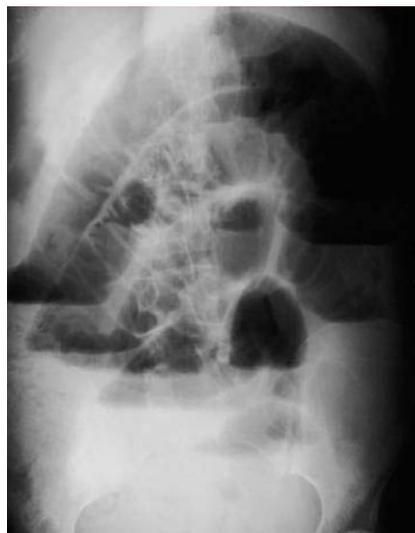


Figure 4. Small bowel obstruction: multiple dilated loops of small bowel with air/fluid levels present at different heights



Intraluminal contrast studies (small bowel follow-through, enteroclysis, barium enema) are utilized in certain clinical situations (Fig. 5). Small bowel follow – through (SBFT) is indicated when: 1) clinical presentation of bowel obstruction is confusing; 2) plain radiograph of the abdomen is non-diagnostic, and 3) response to nonoperative management is inadequate, and more diagnostic accuracy is needed to aid in decision making i.e. to continue with nonoperative treatment or resort to surgical intervention.

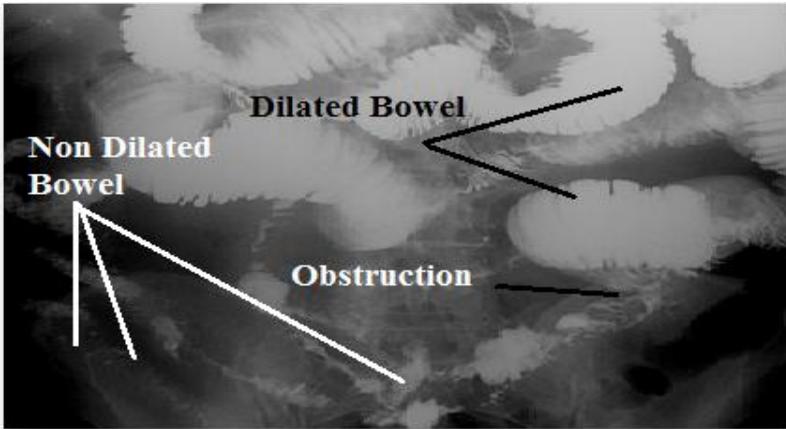


Figure 5. Partial small bowel obstruction: proximal loops are dilated and distal loops are collapsed indicating an obstruction

4.1. DIAGNOSIS

The diagnosis of majority of cases of bowel obstruction can be made based on clinical presentation and initial plain radiograph of the abdomen.

Abdominal x-ray studies show abnormal quantities of gas, fluid, or both in the bowel (Fig. 2, 3).

Laboratory studies:

- complete blood count;
- serum electrolytes and amylase determination;
- arterial blood gas analysis;
- urine test.

Clinical features:

Past surgical and medical history may shed light on etiology of SBO. In the absence of prior surgery and any apparent cause, or in presence of clinically confusing clinical picture, intussusception, MD, gallstone ileus, and neoplasms are suspects. The four cardinal symptoms of bowel obstruction are pain, vomiting, obstipation/absolute constipation, and distention. Obstipation, change in bowel habits, complete constipation, and abdominal distention are the predominant symptoms in LBO. Vomiting occurs late in the course of the disease. On the other hand, pain, vomiting, and distention are commonly seen

in SBO. The pain is colicky in nature and becomes dull, late in the course of SBO. Vomiting is a pronounced symptom in high SBO. The vomitus is bilious or semi-indigested food in high SBO, and feculent in low SBO. Obstipation and constipation are present to a variable degree. "Tumbling SBO" describes intermittent symptoms of obstruction seen in patients with gallstone ileus. These episodes correspond to stone impaction, subsequent release, and reobstruction. Biliary symptoms are present before the onset of obstruction in 20–56 % of cases. Intermittent partial bowel obstructive symptoms are also suggestive of intussusception.

The presence of strangulation/gangrene in SBO cannot always be reliably excluded or confirmed even in the hands of the most experienced clinician. Four classical findings are often used as indicators of strangulation: tachycardia, localized abdominal tenderness or pain, leucocytosis, and fever. The absence of these four signs indicates simple obstruction, the development of any of the indicators raises the index of suspicion of strangulation, and the presence of multiple clinical parameters is correct in 70 % of SBO with strangulation.

Radiologic studies:

X-ray. Abdominal x-ray studies show abnormal quantities of gas, fluid, or both in the bowel (*Fig. 2, 3*).

Ultrasonography (US) is a valuable diagnostic tool in the evaluation of acute abdomen. It is useful in the diagnosis of gallstone ileus, intussusception, pelvic disease, and gallbladder disease, and can aid in the exclusion of SBO. In gallstone ileus, US reveals diseased gallbladder (GB), gas in the GB or bile ducts or both, and fluid filled bowels that can be followed to the stone in the intestine. The presence of stones in the GB will modify the planned operative procedure in the treatment of gallstone ileus. In intussusception, US reveals the diagnostic "target sign", a mass with sonolucent periphery (due to edematous bowel) and a strongly hyperechoic center (from compressed center of intussusception). Paralytic ileus is differentiated from mechanical SBO by the presence of peristaltic movement that is easily observed by US.

With US for mechanical obstruction, dilated loops of small bowel with air/fluid levels present at different heights (*Fig. 6*).

Computed tomography (CT scan) is emerging as a valuable tool in the management of bowel obstruction (*Fig. 7*).

It confirms the diagnosis, differentiates between mechanical and functional obstruction, provides information about cause and site of obstruction, and helps differentiate between simple and complicated SBO. Furthermore, CT scan can diagnose other disease states. Hence CT scan helps in decision making for early surgical intervention, and prevents delay in treatment. CT scan may give false positive results and may be difficult to interpret when colonic abnormalities cause predominantly SB dilatation. CT scan is unable to identify location and cause of obstruction accurately in 18 % of cases. Furthermore, CT scan cannot predict who will benefit from conservative treatment in cases of partial SBO. In these situations SBFT or enteroclysis are more helpful.



Figure 6. Ultrasonography: dilated loops of small bowel with air/fluid levels present at different heights



Figure 7. CT scan: multiple dilated loops of small bowel with air/fluid levels present at different heights

Differential diagnosis with: perforated peptic ulcer; acute pancreatitis; acute appendicitis complicated by peritonitis; hepatic abscess; renal colic.

4.2. SURGICAL TREATMENT OF SBO

Time of operation:

- For strangulation and closed-loop obstruction the operation is required as soon as possible.
- Partial SBO: nonoperative management for 24–48 hrs; if no improvement, small bowel follow – through or CT to increase accuracy of diagnosis; laparotomy if radiologic evidence of high grade obstruction, or if clinical, laboratory, or radiologic evidence of strangulation.

The Procedure of Operation:

- Procedures not requiring opening of bowel.
- Enterotomy for removal of obturation obstruction.
- Resection of the obstructing lesion or strangulated bowel with primary anastomosis (*Fig. 8*).
- Gastrointestinal decompression.
- Bypass anastomosis around an obstruction.
- Formation of a cutaneous stoma proximal to the obstruction.

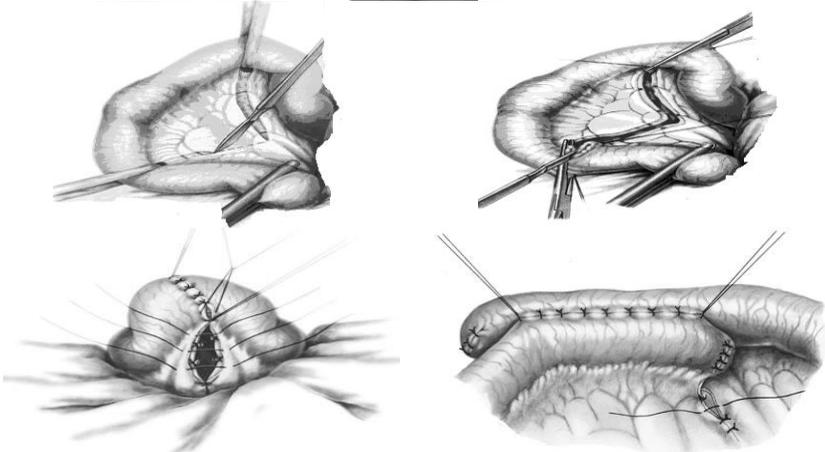
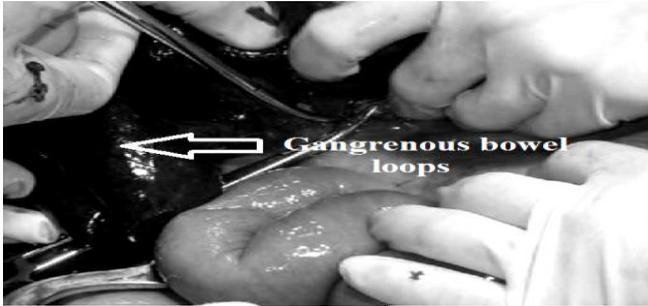


Figure 8. Resection of the obstructing lesion or strangulated bowel with primary anastomosis

Postoperative Care: the principles are: fluid and electrolyte management, antibiotics and gastrointestinal decompression.

4.3. ADHESIVE SBO

With nonoperative treatment, complete SBO resolves far less frequently than partial SBO, 15–36 % vs. 55–75 %. Surgical intervention is indicated when strangulation is suspected to develop during nonoperative treatment, or when conservative treatment fails. The appearance of the bowel before and after release of adhesion is compared. Vascular compromise is recognized by bluish discoloration of intestinal wall, loss of arterial pulsation, subserosal and mesenteric hemorrhage, and lack of peristalsis (*Fig. 9*). If the bowel loop pinks up, resection is avoided, otherwise resection is indicated.

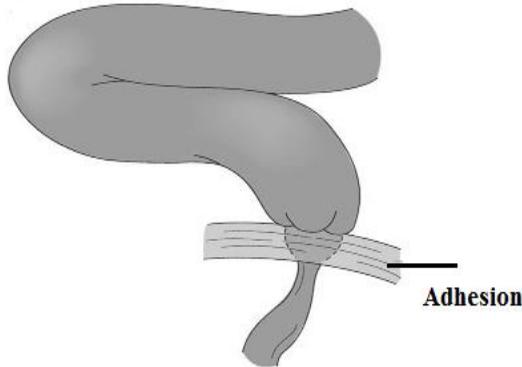


Figure 9. Schematic illustration:
simple obstruction is most often due to adhesion

To prevent subsequent adhesion formation various mechanical and chemical methods have been employed. Mechanical methods include plication (small bowel and mesenteric), and stenting with long intestinal tubes. In addition to failure to prevent re-obstruction, plication is time consuming and tedious, and carries the risk of injury to the bowel or mesenteric vessels. Similarly, long intestinal tubes, in addition to difficulty in positioning distal to ligament of Treitz, are not without complications, and long term results are not adequately evaluated. Although high dose steroids with or without promethazine, antihistamines, and dextran-70 proved to reduce adhesion formation in animals, the potential for disastrous complications prevented their use in humans. A variety of other chemicals have been used to prevent adhesions with mixed results and associated significant complications. Sodium hyaluronate based bioabsorbable membrane has been shown to reduce adhesion formation in human, but its effect on intestinal obstruction is yet to be determined.

4.4. GALLSTONE ILEUS

Obstruction of the intestinal tract by gallstones, commonly known as “gallstone ileus”, is a unique clinical entity, not particularly uncommon but so infrequently encountered that the average surgeon rarely observes more than a few in his professional lifetime. Bartholin is credited with observing and describing the first case of gallstone ileus in a patient examined at autopsy in 1654. Courvoisier focused attention on the entity in 1890 by recording 131 cases in the first published paper on the subject. Gallstone Ileus is an infrequent cause of mechanical bowel obstruction. It is caused by an impaction of a gallstone in the terminal ileum by passing through a biliary-enteric fistula (often from duodenum) (*Fig. 10*).

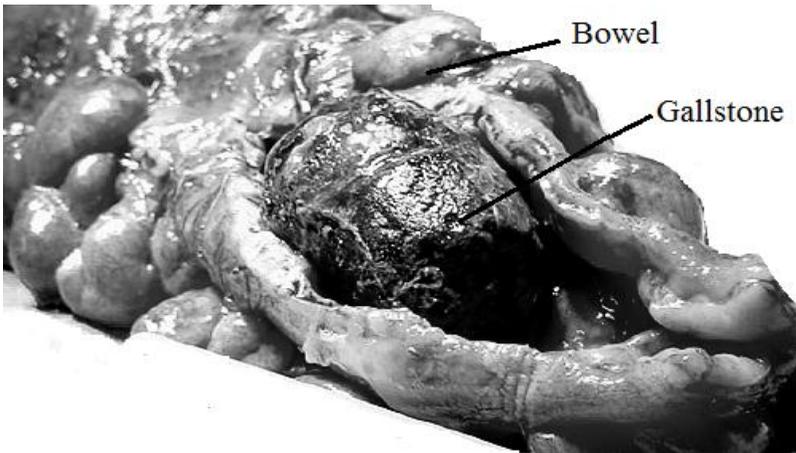


Figure 10. Gallstone Ileus

It occurs more frequently in women with average age of 70 years. The diagnosis of gallstone ileus is often difficult to make. Time from onset of symptoms to surgical intervention is often long, and correct diagnosis is made preoperative only in 13–60 % of cases. In the small bowel, the site of obstruction is usually the distal ileum, and multiple stones are present in 3–15 %.

The clinical symptoms of gallstone ileus are by no means diagnostic. A typical history of cholecystic disease is obtained in less than half of the patients, probably due to the difficulty in obtaining an accurate story from an elderly seriously ill patient. The gradual onset of colicky abdominal pain, distension, nausea and vomiting, and the recurrent nature of the attacks are characteristic of partial or intermittent intestinal obstruction from any cause. Symptoms lack consistency and may even simulate early cholecystitis, pancreatitis or early diverticulitis. There is usually no suggestion of an acute inflammatory episode coincident with fistula formation and passage of the stone, but this may be over

shadowed by obstructive symptoms. One facet of the history if carefully evaluated may be important, namely the progressive nature of the symptoms and shifting of the primary site of pain. Bearing in mind the mechanism of propulsion of the obstructing stone, it produces what one might call a “tumbling” obstruction, the episodes of cramping pain and nausea corresponding to the halting passage of the stone along the alimentary tract.

Physical findings are inconsistent, Tenderness, distension, tympany, and hyperpeperistalsis may be present in variable degree, but no more pronounced than one would expect from any other type of obstruction or, in fact, other nonsurgical conditions. The tenderness in the gallbladder area which might be expected from the acute inflammation and edema associated with the fistula formation is usually absent or masked by abdominal distension. Dehydration is usually present, insidious in its development. Palpation of a large gallstone through the abdominal wall or on rectal examination has been reported but there is nothing to indicate the nature of the mass and to the absence of other confirmatory, findings, is of little significance.

X-ray demonstration of distended loops of bowel, an opaque shadow suggestive of gallstone, a changing obstructive level and air in the biliary tree is almost positive confirmatory evidence.

Abdominal radiography showed a hyperdense lesion with calcified margins in the right upper quadrant (*Fig. 11, panel 1, arrow*). The upper gastrointestinal series, which was performed with the use of barium contrast material, showed a fistulous communication between the gallbladder and the duodenum, with multiple filling defects in the jejunum (*Fig. 11, panel 2, arrows*). Cholecystoduodenal fistulas can occur through erosion of gallstones (typically, more than 2.5 cm in diameter) into the intestinal lumen.

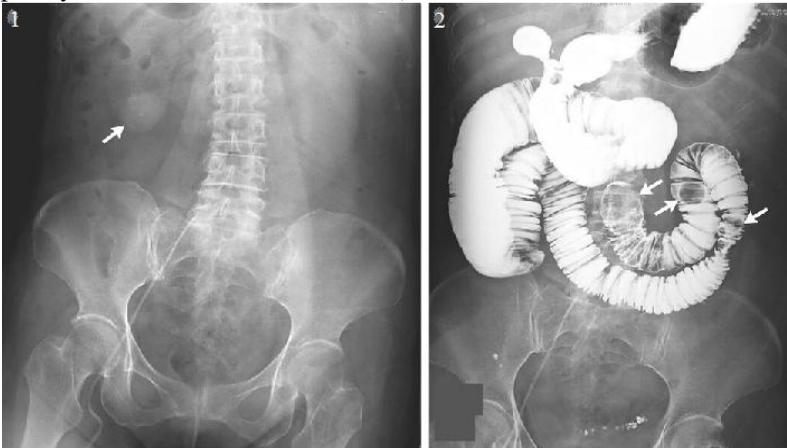


Figure 11. Abdominal radiography

If some but not all of these are present, one is justified to entertaining a high index of suspicion and instituting therapy on a presumptive diagnosis. Since successful treatment of the disease is so dependent upon early diagnosis it is imperative that these factors be evaluated as early as possible in its course. Fortunately our concept of diagnosis has, during recent years, assumed a positive attitude in contrast to the pessimistic outlook voiced by earlier investigators.

CT of the abdomen showed a Rigler's triad (*Fig. 12*):

1. Pneumobilia.
2. SBO.
3. Impacted gallstone-usually in the terminal ileum at ileocaecal valve.

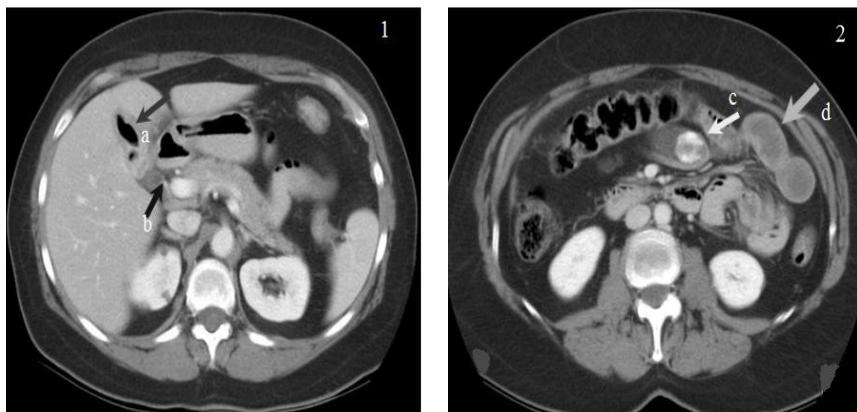


Figure 12. CT scan:

- panel 1 – CT of the abdomen show air in the gallbladder (arrow – a) and air in the common bile duct (arrow - b) representing pneumobilia;
- panel 2 – the gallstone in the small bowel lumen (arrow – c) and dilated and fluid-filled loops of small bowel from small bowel obstruction (arrow – d)

Treatment consists of relief of the obstruction at the earliest optimal time. Inasmuch as these patients are usually elderly, seriously ill, victims of concomitant disease such as obesity, diabetes, and myocardial disease, and are dehydrated and electrolytic O₂ depleted: it is of paramount importance to restore the patient to optimal condition before proceeding with definitive operation. This includes restoration of electrolyte balance, hydration, and decompression with nasogastric suction so long as one does not rely too long on intubation. Exploration and removal of the abstracting lesion should be carried out as soon as the patient's condition permits. Attempts to move or crush the stone are not advisable since this entails: the risk of injury to an already devitalized bowel. A longitudinal incision with removal of the stone

and transverse closure is usually sufficient. If the bowel appears injured beyond the point of spontaneous recovery, resection of the segment should be done or in extremely critical cases, exteriorization of the diseased portion. The options are enterolithotomy, cholecystectomy, and fistula division, with or without common bile duct exploration (one-stage procedure), with definitive repair performed at a second operation (two-stage procedure). The treatment of choice is the enterolithotomy which consists in localize and extract the gallstone. Often the cholecystectomy is contraindicated by comorbidities and the general state of the patient.

4.5. MALIGNANT SBO

This refers to obstruction occurring after treatment of a primary malignancy. Obstruction is due to benign causes (adhesion, radiation enteritis, internal hernia) occurs in 18–38 % of cases (*Fig. 13*).

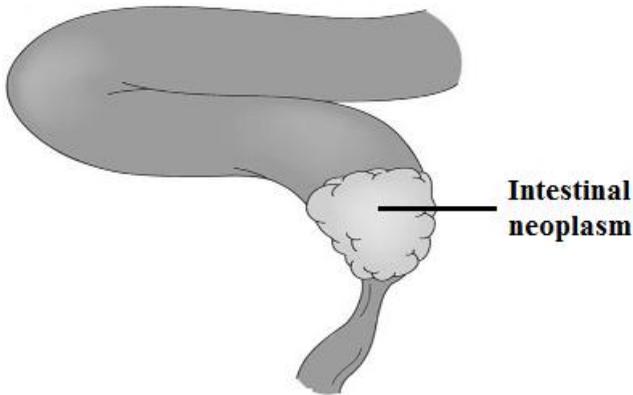


Figure 13. Schematic illustration:
simple obstruction is most often due to neoplasm

Ten percent to 30 % of patients will have relief of obstruction with nonoperative management alone, and about 40 % will eventually require surgery. Resolution with nasogastric decompression occurs in 68 % of cases and within 3 days. About 35–80 % of patients will obtain relief of symptoms with surgery depending on nature of obstruction. Patients presenting in shock, with carcinomatosis, ascites, or palpable mass, have 54 % to 100% mortality. Hence, patients with known cancers should be treated as any other patient presenting with SBO, and final decision making regarding surgical intervention must be individualized. Early surgical intervention is indicated in patients with no known recurrence or long interval to the development of SBO. In patients with carcinomatosis, ascites, or palpable masses, more prolonged course of

nonoperative treatment is justifiable. Surgical intervention is indicated if nasogastric decompression fails or if re-obstruction develops after removal of nasogastric tube. Selection of surgical procedure, resection, bypass, gastrostomy, or tube jejunostomy is based on extent of the disease. Used selectively, percutaneous gastrostomy can improve quality of life.

4.6. INTUSSUSCEPTION

In adults 85–90 % of intussusceptions are associated with a discrete, pathologic process leading the intussusception, and neoplasms account for majority of cases (*Fig. 14*). Malignant lesions are being recognized with increasing frequency. A recently recognized subtype is postoperative intussusception. The point of origin of the intussusception is the small bowel and more specifically, the jejunum, particularly proximal jejunum, and dense desmoplastic inflammatory reaction within the mesentery may be the underlying mechanism precipitating the intussusception.

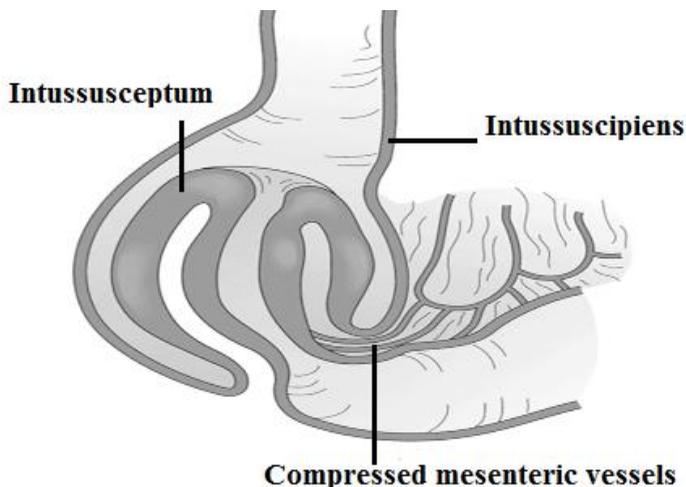


Figure 14. Schematic illustration:
simple obstruction is most often due to intussusception

Treatment of intussusception in adults is surgical without attempts at hydrostatic reduction. Optimal surgical procedure depends on the anatomic location, presence of a lead point, and local factors, such as edema, inflammation, and ischemia of involved bowel. While resection is the treatment of colic and enterocolic intussusception, the choice in enteric type i.e. attempt at operative reduction vs. resection without attempt at reduction, depends on presence of underlying lesion, chances that the lesion is malignant, and viability of involved bowel.

4.7. EARLY POST-OPERATIVE OBSTRUCTION

This is defined as SBO within 30 days after celiotomy. In this clinical situation bowel activity may not return (prolonged ileus) or there is initial temporary return of bowel function (*Fig. 15*).

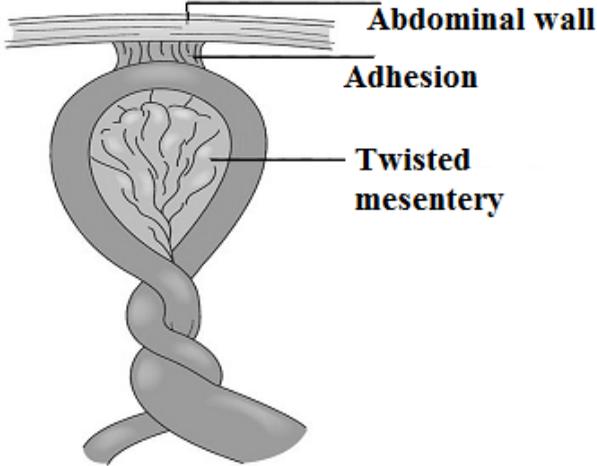


Figure 15. Schematic illustration of a closed-loop obstruction.

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 because of the twisting of the mesentery

The obstruction is due to adhesions (92 %), phlegmon or abscess, intussusception (2.5–4 %), or internal hernia. The treatment is conservative in the absence of bowel ischemia or mechanical obstruction. Nasogastric decompression, intravenous fluid therapy, and even parenteral nutrition for up to 10–14 days is indicated if the patient is stable and exhibiting clinical and radiologic improvement continues. After this time further improvement is unlikely and operation should be performed.

5. LARGE BOWEL OBSTRUCTION (LBO)

Obstruction – mechanical interruption of the flow of intestinal contents (adults):

1. Neoplasms (60 % of cases).

Cancer of the colon and rectum is predominantly (95 %) adenocarcinoma it may start as a benign polyp but may become malignant, invade and destroy normal tissues, and extend into surrounding structures (*Fig. 16*). Cancer cells may break away from the primary tumor and spread to other parts of the body (most often to the liver).

2. Volvulus: the sigmoid; the cecal; others are rare.
3. Diverticulitis with stricture: the sigmoid; the cecal; others are rare.
4. Intussusception.

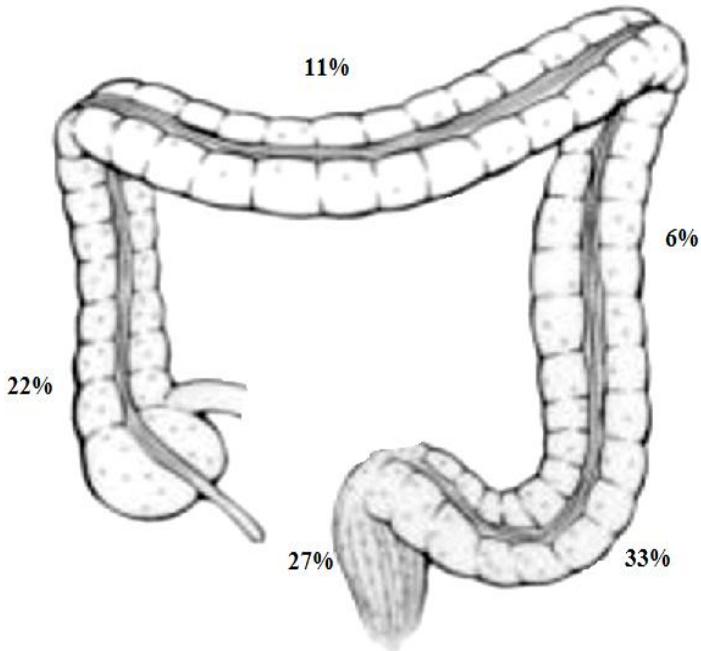


Figure 16. Cases of neoplasms of the colon and rectum

5.1. PATHOPHYSIOLOGY

As in small bowel obstruction, large bowel obstruction (LBO) results in an accumulation of intestinal contents, fluid, and gas proximal to the obstruction. Obstruction in the large bowel can lead to severe distention and perforation unless some gas and fluid can flow back through the ileal valve. Large bowel obstruction, even if complete, may be undramatic if the blood supply to the colon is not disturbed. If the blood supply is cut off, however, intestinal strangulation and necrosis (ie, tissue death) occur; this condition is life threatening. Dehydration occurs more slowly than in the small intestine because the colon can absorb its fluid contents and can distend to a size considerably beyond its normal full capacity.

5.2. CLINICAL MANIFESTATIONS

Large bowel obstruction differs clinically from small bowel obstruction in that the symptoms develop and progress relatively slowly. In patients with obstruction in the sigmoid colon or the rectum, constipation may be the only symptom for days. Loops of large bowel become visibly outlined through the abdominal wall, and the patient has crampy lower abdominal pain. Finally, fecal vomiting develops. Symptoms of shock may occur.

5.3. ASSESSMENT AND DIAGNOSTIC FINDINGS

Diagnosis is based on symptoms and on x-ray studies. Abdominal x-ray studies (flat and upright) show a distended colon. Barium studies are contraindicated (*Fig. 17, 18*).

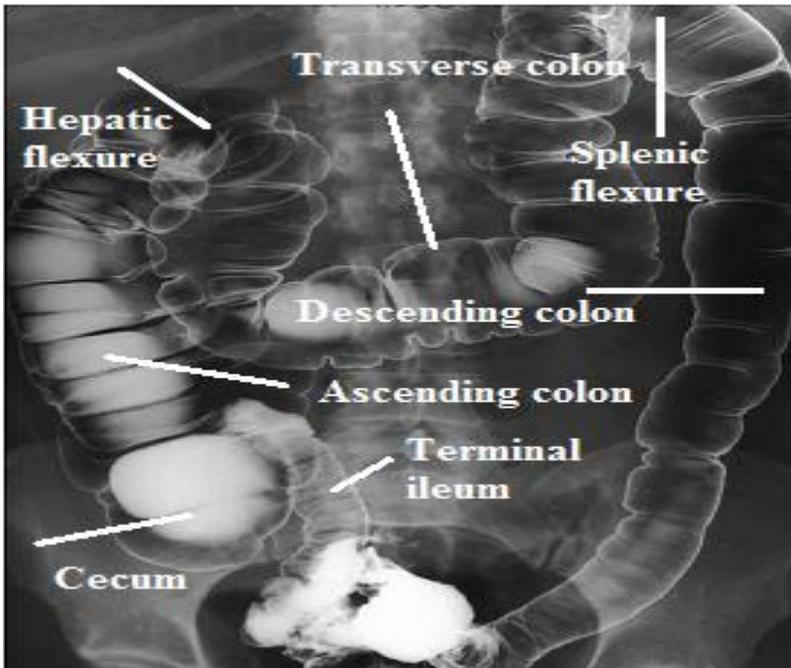


Figure 17. Abdominal X-ray. Small bowel obstruction

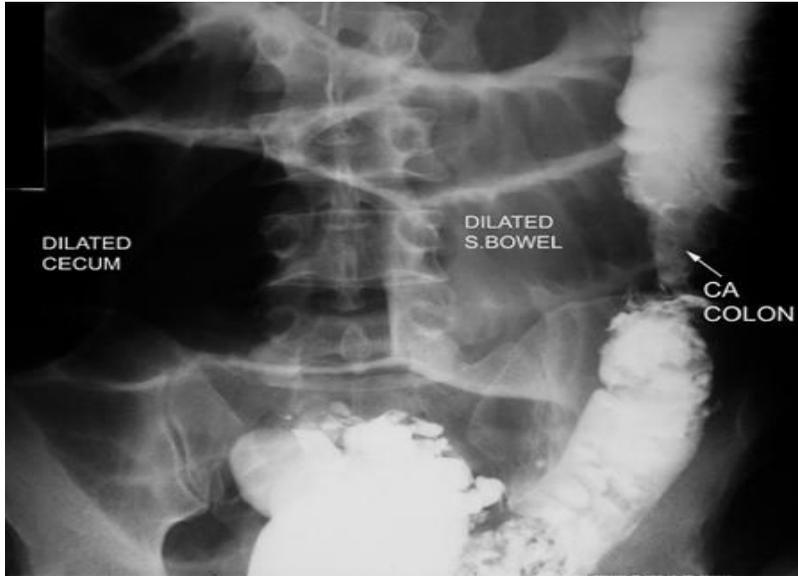


Figure 18. Large bowel obstruction – cancer of the descending colon (arrow)

5.4. MEDICAL MANAGEMENT OF THE NEOPLASMS OF THE COLON

- A colonoscopy may be performed to untwist and decompress the bowel.
- Neoplasms of the right colon: a cecostomy: in which a surgical opening is made into the cecum, may be performed for patients who are poor surgical risks and urgently need relief from the obstruction (*Fig. 19*).

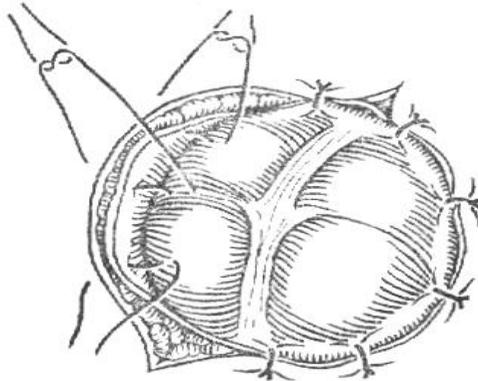


Figure 19. Cecostomy

➤ Surgical resection of the right colon – right hemicolectomy (*Fig. 20*). Colic resection is easy to perform because of the mobility of the right colon.

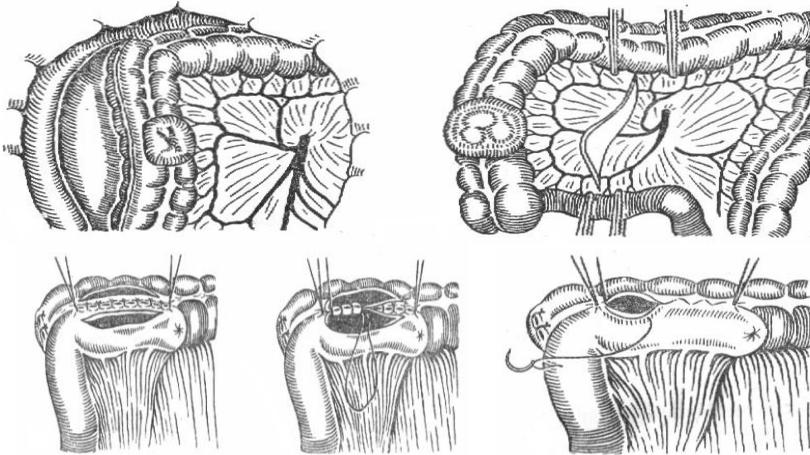


Figure 20. Right hemicolectomy

➤ Neoplasms of the transverse colon: surgical resection of the colon and a cecostomy.

➤ Surgical resection to remove the obstructing lesion. A temporary or permanent colostomy may be necessary. An ileoanal anastomosis may be performed if it is necessary to remove the entire large colon. When gangrene is associated with peritonitis a **Hartmann's procedure** may be performed (*Fig. 21*).

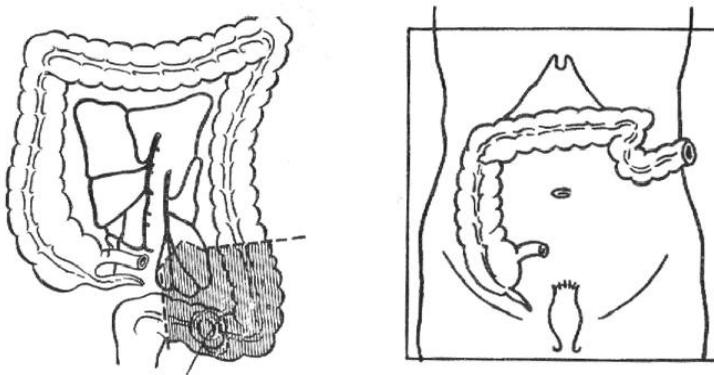


Figure 21. Hartmann's procedure

5.5. VOLVULUS OF THE COLON

Obstruction caused by twisting of the intestines more than 180 degrees about the axis of the mesentery (*Fig. 22*).

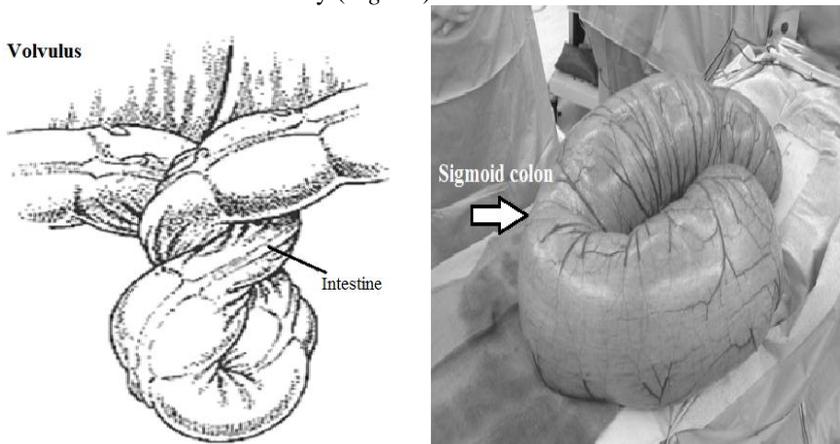


Figure 22. Volvulus of the colon

Pathophysiology of obstruction:

- With mechanical obstruction, air and fluid accumulate in the bowel lumen. Results in increase of intestinal intraluminal pressure.
- This further inhibits absorption and stimulates influx of water and electrolytes into lumen.
- Initially, there is increase in peristaltic activity. But as process progresses, coordinated peristaltic activity diminishes along with contractile function.
- Gives rise to dilated and atonic bowel proximal to point of obstruction.
- With progression, patient may actually appear to improve clinically with less
 - Frequent and crampy pain.
 - Effect of mechanical obstruction causes an initial increase in blood flow.
 - With unrelieved obstruction, blood flow diminishes leading to breakdown of mucosal barriers and increased susceptibility to bacterial invasion and ischemia.

Volvulus of the right colon. Acute volvulus of the right colon and terminal ileum (cecal volvulus) accounts for 1–10 % of all intestinal obstructions and 18–44 % (~25 %) of all cases of colonic volvulus. Autopsies have shown that in 11–22 % of the population the right colon is sufficiently mobile to allow the development of volvulus. Adhesions from previous surgery, congenital band, pregnancy, malrotation and obstructing lesions of the left colon have been discussed as triggers (*Fig. 23*).

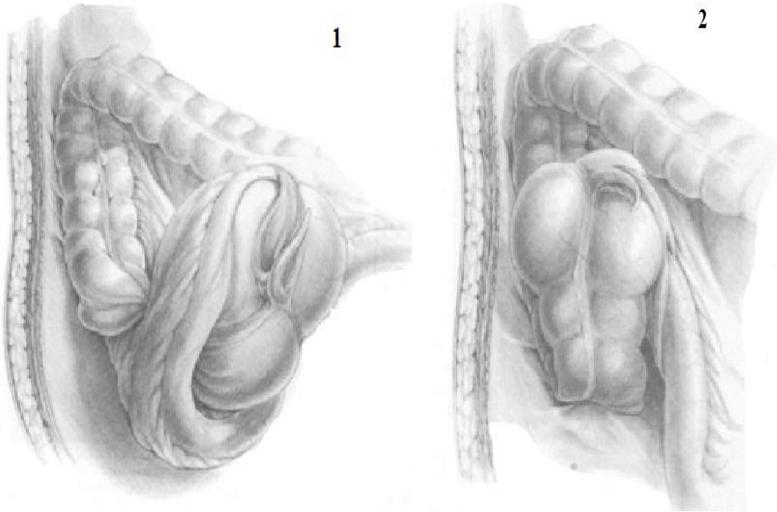


Figure 23. Types of volvulus of the right colon:

- 1 – axial torsion type (twist 180–360 degrees on longitudinal axis of ascending colon (distal ileum and ascending colon); associated with bowel compromise, ischemia, and perforation);
- 2 – cecal bascule (cecum folds anteriorly on ascending colon; may result in intermittent obstructive symptoms)

Female predominance has been frequently reported; many authors observed a significantly higher frequency in elderly women.

Clinical features. The diagnosis is not easy; the clinical picture is that of bowel obstruction with a tympanic mass extending from the right lower to the right upper quadrant. Radiological examination of the abdomen remains the key to diagnosis: typically, the caecal shadow is absent from the lower quadrant, the caecal air fluid level may be seen in the left upper quadrant mimicking the stomach shadow (*Fig. 24*).

Cecum is greatly dilated, distended small bowel loops are often present, and the terminal ileum may be filled with air and visualized in an abnormal position to the right of the distended cecum. A single, long air-fluid level is usually noted in the distended cecum, there may be a relative absence of gas in the transverse and left colon.

Many authors advocate a preoperative water-soluble contrast enema examination to confirm the diagnosis and to exclude a concomitant obstructing lesion of the left colon, but this examination is potentially dangerous.

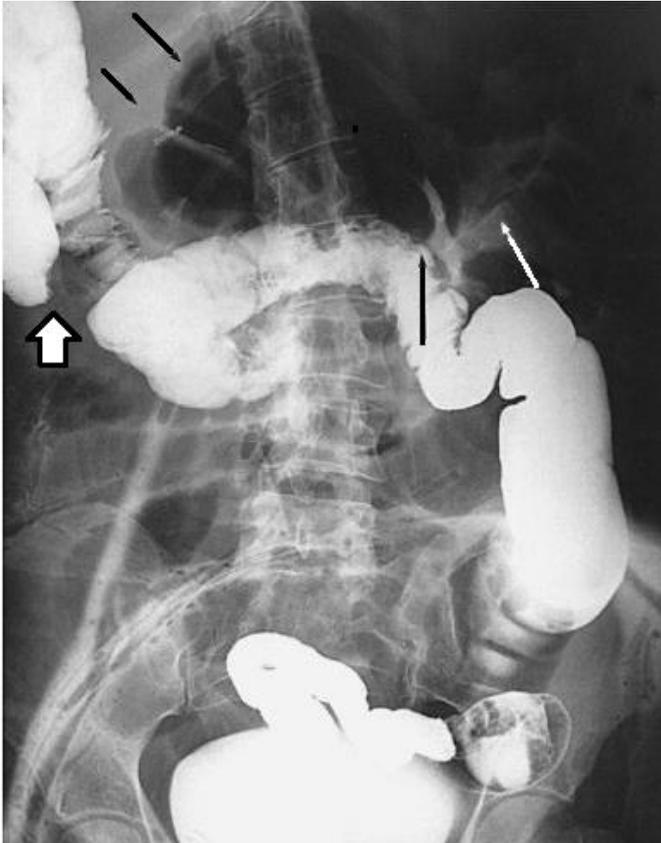


Figure 24. Barium enema of cecal volvulus: contrast stops at hepatic flexure (arrow head) and air filled cecum crosses midline of abdomen in LUQ

Treatment. The treatment of choice for volvulus of the right colon without gangrene is discussed controversially. Cecostomy and cecopexy (*Fig. 25*) have been recommended but postoperative mortality and recurrence rates were high. Total resection of the right colon eliminates the risk of recurrence. Colic resection is easy to realise because of the mobility of the right colon. Postoperative mortality after resection is about 8 % without gangrene and 26 % with gangrene.

Many authors advocate right hemicolectomy as the method of choice for the treatment of volvulus of the right colon, even in the absence of gangrene.

Volvulus of the transverse colon and the splenic flexure. Volvulus of the transverse colon and the splenic flexure is uncommon. Less common area for volvulus (~4%). Transverse colon volvulus associated with mobile right colon, distal obstruction, chronic constipation, congenital malrotation of the midgut.

Diagnosis is generally made at laparotomy. The principles of treatment – resection of transverse colon with colostomy.

Volvulus of the sigmoid. The sigmoid colon is the most common site for colonic volvulus occurring often in patients over 60 years with a history of chronic constipation. Other contributing factors may include neurologic or psychiatric disorders (neuropsychotropic drugs alter the bowel motility), adhesions

from previous surgery, and pregnancy. Acute volvulus sigmoid colon accounts for 1–10% of all intestinal obstructions and 60% of all cases of colonic volvulus. Rotation of a segment of bowel around its mesenteric axis that is sufficient to cause a complete or partial obstruction of the lumen and a variable degree of impairment to its vascular supply. Can only occur if the two ends of the segment that are twisted are in close approximation.

Clinical features. The symptoms of volvulus of the sigmoid are colicky abdominal pain, complete constipation, and gross, usually asymmetric distention of the abdomen. Physical examination reveals marked abdominal tenderness frequently with a palpable tympanitic mass. Rectal examination commonly shows absence of feces. If gangrenous changes have occurred, tachycardia, toxicity and peritonitis may be present.

Diagnosis. A plain X-ray examination of the abdomen reveals dilated loops of large bowel forming the “omega loop” sign, with the convexity of the loop lying away from the site of obstruction. Pointing towards the obstruction is the “bird's beak”. Volvulus can be confirmed in 80% by a plain-film of the abdomen (*Fig. 26*) and barium enema (“bird's beak” or ace of spades – pathognomonic of volvulus) (*Fig. 27*).

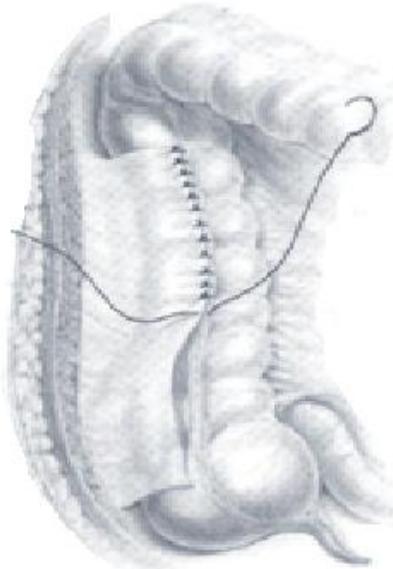


Figure 25. Cecopexy

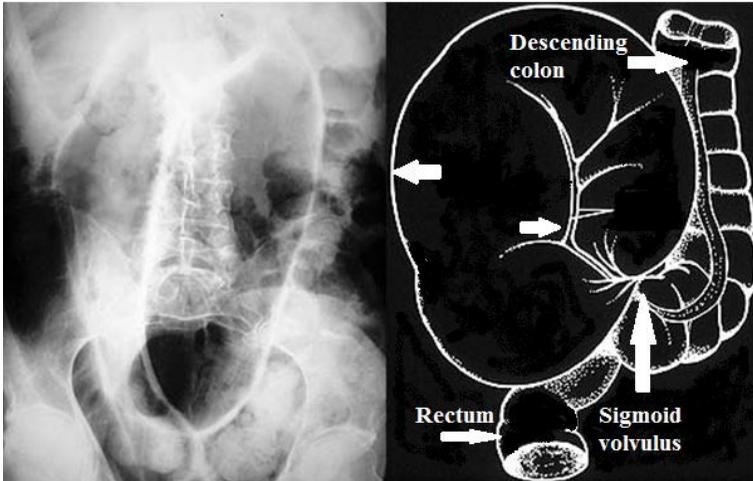


Figure 26. Plain-film radiograph of abdomen: shows a very dilated single loop of colon in the left abdomen with both ends toward the pelvis and the center superiorly positioned – looking like a “bent innertube”

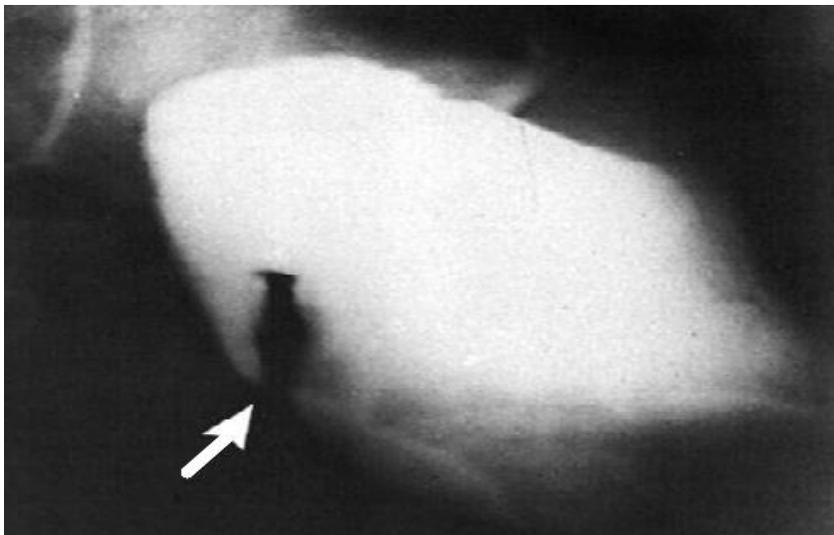


Figure 27. Radiograph of abdomen: “bird’s beak” on barium enema (arrow)

Mesocolon “whirl” sign around the superior mesenteric artery is pathognomonic of volvulus on CT scan (*Fig. 28*).

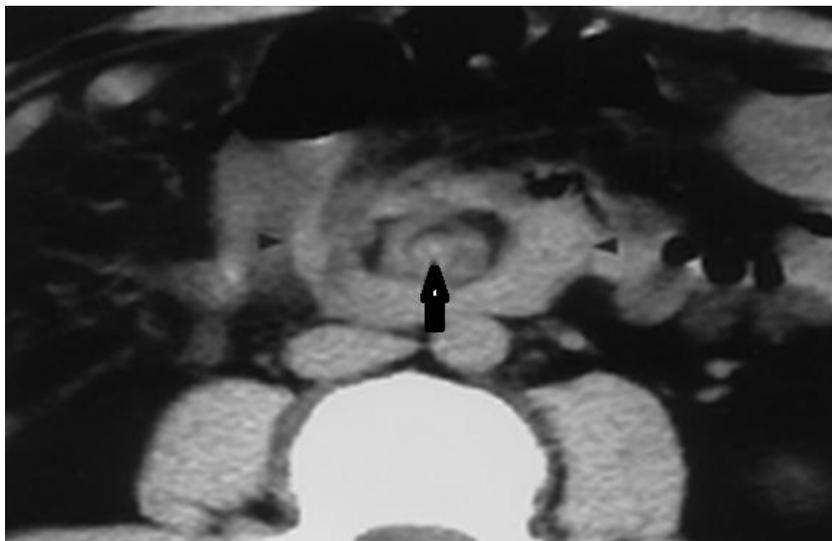


Figure 28. CT scan: mesocolon whirl (arrow)

Treatment. The initial treatment is an attempt at deflation and untwisting of the sigmoid loop by the passage of a well lubricated rubber rectal tube through a sigmoidoscope with the patient in the lateral knee-chest position. After decompression, the involved segment usually undergoes spontaneous detorsion, the immediate escape of flatus and liquid faeces through the sigmoidoscope or catheter indicates that the obstruction has been removed. The tube is left in place for 48 hours. This treatment can be expected to be successful in 80 % of patients. If the segment is viable at exploration simple detorsion should be performed, further treatment should be considered because of the high recurrence rate (35–60 %). Elective resection of the sigmoid loop following adequate preparation with primary anastomosis should be performed through laparotomy or laparoscopic approach.

If decompression is unsuccessful or if mucosa of doubtful viability is seen, immediate laparotomy is mandatory. The resection may be accompanied by primary anastomosis with peroperative colonic lavage in most of the cases. When gangrene is associated with peritonitis a Hartmann's procedure may be performed, eventually accompanied by Mikulicz's drainage. Colostomy alone is contraindicated because it will not prevent recurrent volvulus.

Differential diagnosis:

- Colon cancer.
- Diverticular disease.
- Extrinsic compression from metastatic carcinoma.
- Hernia

- Intussusception .
- Fecal impaction.
- Paralytic ileus.
- Toxic megacolon.

5.6. OTHERS

Endometriosis. Endometriosis may affect rectum and sigmoid of fertile female patients. The intestine is involved in 12–37 % of cases. Intestinal endometriosis is usually asymptomatic and complete obstruction of the bowel lumen occurs in less than 1 % of cases. Endometriosis usually does not produce complete obstruction but endometriosis may extend circumferentially round the bowel producing a stenosis. Symptoms related to the menstrual cycle and the length of the history may suggest the diagnosis of endometriosis. At laparotomy endometriosis may mimic a constricting colon carcinoma. Establishing an accurate pre- and peroperative diagnosis is very difficult.

Fecal impaction. Fecal impaction is a common cause of obstruction in elderly, chronically ill, bed-ridden patients and can be detected by rectal examination. When the colon has been emptied of fecal material by enemas repeated over a period of several days, colonoscopy or barium contrast enema may be necessary to exclude other causes of obstruction.

6. MESENTERIC ISCHEMIA

Mesenteric ischemia is a condition characterized by high mortality. It does not occur very frequently but when it does, the diagnosis is often made too late. Several new approaches have been suggested but mortality still remains in the same high range for the last decades.

6.1. DEFINITION, PATHOGENESIS AND EPIDEMIOLOGY

Mesenteric ischemia is defined as a condition in which the supply of oxygen is too small to satisfy the needs of the intestines.

Ischemia can affect the small intestine, the colon or both. It can be acute and chronic.

Most often mesenteric ischemia is classified as occlusive or non-occlusive.

Probably the most common cause of acute occlusive mesenteric ischemia is strangulation. This variant will, however, not be dealt with in this chapter. Other common causes of occlusive intestinal ischemia are arterial emboli, arterial thrombosis, complications of aortoiliac surgery and venous thrombosis. Unusual causes of occlusive mesenteric ischemia include trauma and small vessel disease. Acute non-occlusive mesenteric ischemia occurs as a consequence of other critical diseases such as shock and heart failure. Non-occlusive intestinal ischemia can also be induced pharmacologically by e.g. digitalis and vasoactive drugs. Chronic ischemia can be caused by atherosclerosis, fibromuscular

dysplasia, inflammatory disease (Takayasu), and be a consequence of radiation injury. It can also be congenital as a part of aortic coarctation.

The pathogenesis in occlusive ischemia is most often an abrupt occlusion of a major vessel resulting in a significant reduction in intestinal blood flow. It is, however, not unusual that one finds two of the three supplying major arteries to be occluded without signs of mesenteric ischemia. That probably requires that the process has taken some time to allow collateral blood flow to develop and that the remaining vessels are healthy. In the elderly patient with atherosclerotic vessels, which is a common situation in the western world, sudden obstruction of blood flow by an embolus in the superior mesenteric artery is likely to impair blood flow enough to cause bowel infarction. If ischemia is total or near total it takes 8–16 hours to develop transmural infarction. This is, thus, the time frame during which the diagnosis has to be made and appropriate actions started in order to allow measures to prevent bowel infarction. After this period removal of dead bowel is all that can be achieved surgically and the prognosis is then dependent on the extent of bowel necrosis.

In the non-occlusive forms time course and extent of bowel injury is less easy to predict. Small intestinal blood flow can be reduced to about half of normal and the bowel can compensate for this reduction in oxygen delivery by increased oxygen extraction. (As a consequence the liver might become quite hypoxic). If blood flow is about 5 % of normal or less then ischemia is total or near total. In between, e.g., reduction down to 10–40 % of the normal level, ischemia causes mucosal injury but not transmural infarction even if it becomes quite prolonged. This mucosal injury is considered important in the sense that it further exaggerates the underlying shock situation by release of various toxic factors from the intestine including bacteria and endotoxin – a process often referred to as translocation. Impairment of the intestinal immune function and the associated liver ischemia may also contribute to the aggravation of the underlying disease. The mucosal injury caused by this degree of intestinal ischemia, is likely to be exacerbated at reperfusion by increased generation of oxygen free radicals.

The colon seems to be affected less often by ischemia than the small intestine. One exception is focal non-occlusive colonic ischemia affecting the splenic flexure. The ischemic episode often remains undiscovered and these patients are later found to have a stricture at barium enema. This stricture sometimes can be mistaken for a neoplastic one. Colon is also affected in ischemia caused by surgery for abdominal aortic aneurysm.

The frequency of acute mesenteric ischemia is low. It has been reported that less than 1 % of all acute laparotomies are performed because of acute mesenteric ischemia. Arterial thrombosis and embolus make up the two most common occlusive causes of acute mesenteric ischemia (approximately 50 and

33 %, respectively). Most often the patient with a mesenteric embolus has an atrial fibrillation as the source of the embolus. Venous mesenteric thrombosis is the cause of acute mesenteric ischemia in about 10–15 % of all cases. The onset of ischemia may be significantly prolonged in some patients with venous thrombosis.

6.2. CLINICAL STAGES

Irrespective of etiology four clinical stages are usually recognized. As mentioned above in arterial embolism the onset of symptoms is often very quick and the progression of symptoms rapid while the process can take several days following venous thrombosis.

The first stage is *the hyperactive stage*. This is characterized by the intermittent severe pain which starts immediately after occlusion of the vessel. Frequently there is passage of loose stools, sometimes with blood, and vomiting. Usually there is a discrepancy between the often very severe pain and the few findings on physical abdominal examination. Ischemia causes hyperperistalsis reflected in hyperactive bowel sounds on auscultation.

The paralytic stage. The pain is usually diminishing but becomes more continuous and diffuse. During this stage the size of the abdomen increases and it becomes generally tenderer and there are no bowel sounds at auscultation.

The stage of disarranged fluid balance. Fluids containing proteins and electrolytes start to leak through the mucosal as well as the serosal side of the gut. When the bowel becomes necrotic peritonitis develops. The fluid loss is usually massive. In this stage the patient does not differ much from other patients suffering from peritonitis of other causes.

The Shock stage. In this stage patients are rapidly deteriorating with severe alterations in the fluid balance and the situation soon goes over into irreversible shock.

6.3. DIAGNOSIS

Rapid diagnosis is essential in order to improve the high rate of detrimental outcome in acute mesenteric ischemia. A high degree of suspicion is the single most important factor in order to achieve diagnosis while treatment still could be corrective.

When the patient is in the hyperactive symptomatic stage, at laparotomy the gut could still be saved if an embolectomy, thrombectomy or a reconstruction could be performed.

Plain X-ray is unspecific until very late when gas can be seen in the bowel wall and in the mesenteric veins.

Duplex ultrasonography can be diagnostic in the hyperactive stage but in the later stages there is usually too much gas to allow reliable readings.

Angiography can be diagnostic and has been advocated strongly by some. Others have argued angiography may cause unacceptable delays in the

handling of these patients. If angiography is performed and if there is a non-occlusive disease the catheter can be used for local infusion of vasodilating drugs as papaverin as advocated by *Boley* and co-workers.

Ischemia following aortoiliac reconstructive surgery constitutes a special situation. Clinical colonic ischemia occurs in 2.3 % of all cases increasing to 7.3 % after surgery for ruptured aortic aneurysm with the patient in shock. If the patients are routinely followed up by endoscopy mucosal ischemia is seen in about 10% of the cases.

Early passage of stools postoperatively, especially if bloody, is an important warning sign. Other signs that might indicate ischemia include failure to improve as expected, increasing creatinine concentrations in serum and extensive thrombocytopenia. The vast majority of the ischemic lesions following surgery for aortic aneurysm are within reach by the sigmoidoscope.

6.4. TREATMENT

If the diagnosis is made before bowel gangrene has developed (or if there is only a minor localized gangrenous area which could be resected although surrounded with ischemic but not yet gangrenous intestine) and if there is a localized obstruction an embolectomy or thrombectomy is the best treatment, although not supported by RCTs (*Fig. 29*).

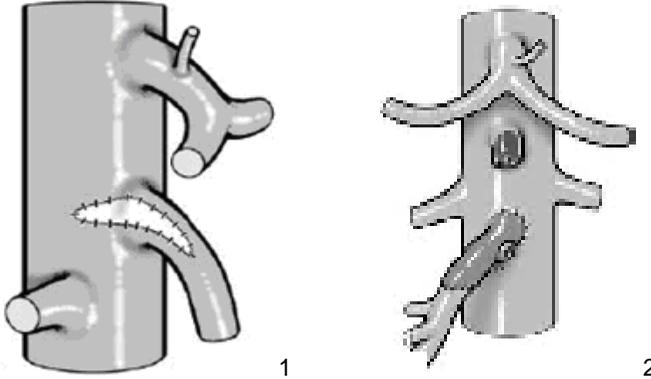


Figure 29. Open endarterectomy and reconstruction by piece of autologous vein (1). Cutting and reimplantation with reconstruction by autologous vein (2) by M. Betzler, 1998

If there is a central stenosis in the superior mesenteric artery with low flow after thrombectomy, this is best treated by implanting the infrapancreatic part of the artery end to side in the infrarenal aorta or by a short bypass. Reconstruction of the superior mesenteric artery close to the aorta is warned against because of the difficult anatomic position (*Fig. 30*).

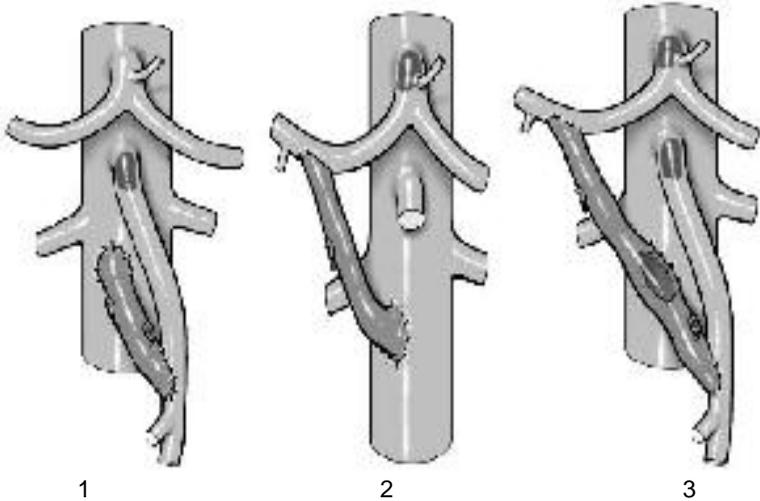


Figure 30. Variants of autologous vein shunts by M. Betzler, 1998:
 1 – between aorta and superior mesenteric artery;
 2 – between aorta and common hepatic artery;
 3 – aorta-hepatico-mesenteric shunts

Following this type of surgery, with or without simultaneous bowel resection, the rule should be to perform a second look 12–24 – hours later. This decision should be made at the time of the first operation and should basically not be discussed thereafter.

Mortality following second look surgery is generally 65–85 %. It is, however, 100% if nothing is done.

In transmural colonic ischemia following aortic aneurysm surgical resection of the affected bowel segment should be performed. Closing the distal end of the colon blindly and an end colostomy (the Hartmann procedure) is then a safe operation. If the bowel segment is removed before signs of peritonitis are visible the prognosis is excellent.

If there is significant bowel infarction at the time of surgery all that could be done is removal of the dead gut.

In case that the remaining part of the gut would allow oral nutrition and a normal life, resection should be performed. It is often advised to perform a “second look” 24 hours later and this decision should be made at the time of primary surgery, as stated above. If, however, the entire small intestine is gangrenous and the patient has peritonitis it is most often too late to save the life of the patient and often nothing is done at laparotomy in such cases.

7. PSEUDOObSTRUCTION (OGILVIE'S SYNDROME)

Ogilvie's syndrome – dilation of the bowel in the absence of a causative anatomic lesion. Distention of colon with signs and symptoms of colonic obstruction without a mechanical cause for the obstruction. May be acute or chronic: *acute*: usually involves only colon, and more commonly affects patients with chronic renal, respiratory, cerebral or cardiovascular disease; *chronic*: can affect other parts of the GI tract and tends to recur.

Pseudoobstruction may be primary or secondary: *primary* pseudo-obstruction – a motility disorder (familial visceral myopathy; diffuse disorder involving autonomic innervation of intestinal wall); *secondary* associated with neuroleptics, opiates, metabolic illness, myxedema, uremia, lupus, scleroderma, Parkinson's, traumatic retroperitoneal hematomas.

7.1. DIAGNOSIS

- Water soluble contrast enema (can differentiate between mechanical and pseudoobstruction).
- Colonoscopy (can also be used for treatment).

7.2. INITIAL TREATMENT

- Resuscitation.
- Parasympathomimetic.
- Rectal tube/enemas /exams (work in most).
- Colonoscopic decompression (80–90 % effective).
- Surgery (cecostomy vs. resection) – cecum > 12 cm or peritoneal signs.

Навчальне видання

**Абдомінальна хірургія
Змістовний модуль 1**

ГОСТРА КИШКОВА НЕПРОХІДНІСТЬ

Методичні вказівки для студентів та інтернів

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