VOMITING, DIARRHEA, AND CONSTIPATION

VOMITING

The occurrence of vomiting may be the result of both gastrointestinal (GI) and non-GI causes. Categorization of the cause of vomiting is often segregated by the presence or absence of abdominal pain. Benign causes of vomiting often do not present with abdominal pain. These benign causes of vomiting are often the results of medications (including chemotherapy), motion sickness, food poisoning, infectious gastroenteritis, hepatitis, upper GI bleeding, postoperative ileus, or acute central nervous system disease. A thorough history of present illness as well as determination of associated symptoms may very well identify the etiology for these benign causes of vomiting.

More concerning causes of vomiting are those associated with abdominal pain. These often include potentially serious conditions that may require surgical intervention, including gastrointestinal obstruction, mesenteric ischemia, pancreatitis, biliary colic, and perforated intestine causing peritonitis, as in the case of perforated appendicitis. The presence of bilious emesis is more concerning for a bowel obstructive process, or protracted ileus from complications of one of the above-noted diagnoses, and often warrants more acute evaluation and assessment.

Gastric obstruction is a common occurrence in children but a rare and concerning diagnosis in adults. Infants may develop a progressive obstruction of the gastric pylorus in a disease of hypertrophic pyloric stenosis. This process, which has some genetic and environmental contributions to its development, leads to the progressive hypertrophy of the pylorus to the point of near-complete obstruction. Presentation of these children often occurs before the third month of life with symptoms of persistent non-bilious vomiting and often severe dehydration and classic metabolic alkalosis from electrolyte loss. Surgery is indicated for a myotomy of the abnormal muscle, resolving the obstructed stomach. Gastric obstruction of the stomach in adults may be from obstruction of the pyloric channel from peptic ulcer disease and ulceration, or from the obstruction of gastric outflow from malignant tumor burden. Endoscopic assessment of the symptoms of gastric outlet obstruction with the symptoms of non-bilious emesis will diagnose the condition by endoscopic biopsy.

Obstruction of the small intestine often leads to the clinical picture of vomiting with the possibility of associated abdominal pain. The presentation of a bowel obstruction often includes the presence of abdominal discomfort and distention. Interview of the patient with these findings must include questioning with regards to the character of the vomitus. The presence of yellow or green material suggestive of bile should alert the physician to the potential of an obstructive process at a location distal to the ampulla of Vater. Additional clinical findings of more severe abdominal pain are concerning for an advanced obstructive process that may include bowel incarceration or intestinal volvulus with or without associated strangulation and ischemia of the involved intestine. The presence of localized peritoneal signs is concerning for intestinal ischemia, but the lack thereof, does not exclude the potential for early ischemia being present.

Common etiologies for small bowel obstruction include adhesive bands causing obstruction following any type of intra-abdominal surgery as well as bowel incarceration from an associated hernia, including internal, ventral, inguinal, and femoral hernias. In the patient with a history of prior abdominal surgery and the presence of a small bowel obstruction, three-fourths of these obstructive events will be related to postoperative adhesions. This type of bowel obstruction may occur early following intra-abdominal surgery in two-thirds of cases, with another one-third of cases occurring within one year of the surgical procedure. Aside from these two principle etiologies for bowel obstruction, other processes leading to bowel obstruction are rarer including obstruction caused by tumor, intussusception, Crohn's disease, bezoar, or intestinal volvulus.

Fluid and Electrolyte Abnormality

The main electrolyte abnormality associated with persistent vomiting is that of metabolic alkalosis. This electrolyte abnormality results from the net loss of gastrointestinal chloride. Vomiting from the etiology of infantile hypertrophic pyloric stenosis or gastric outlet obstruction induces the initial loss of hydrogen chloride (HCl). Normal secretion of HCl into the stomach lumen is balanced by the absorption of bicarbonate. With vomiting, the loss of hydrogen chloride then results in a net alkalosis. Mild alkalosis can be addressed by the kidneys at the glomeruli level with secretion of the bicarbonate ion accompanied by sodium and potassium. With progressive volume depletion from ongoing vomiting, loss of extracellular volume worsens leading to a reduced ability of filtration and excretion of the bicarbonate ion at the glomeruli level and thus progressive alkalosis. More protracted vomiting with associated extracellular volume loss and dehydration will stimulate the renin-angiotensin II-aldosterone feedback response. This leads to bicarbonate reabsorption, attempts at sodium reabsorption to balance the extracellular fluid loss, and results in hydrogen ion secretion, thus confounding and increasing the bicarbonate reabsorption. The resulting elevation in aldosterone levels will lead to an accompanied potassium loss which further exacerbates hydrogen ion secretion and reabsorption of bicarbonate. This state leads to a paradoxical aciduria in attempts to normalize sodium and potassium absorption at the expense of the hydrogen ion.

Evaluation

Initial evaluation of the presence of gastrointestinal obstruction, aside from history and physical examination, should include a serum electrolyte panel and blood glucose to discern any electrolyte abnormality secondary to the primary process or a hyperglycemic cause for intestinal ileus. Elevation in liver enzymes or pancreatic enzymes may help suggest a hepatobiliary or pancreatic etiology, respectively, to the vomiting process. Plain abdominal radiography of the abdomen will help discern a possible bowel obstruction process or concerns of intestinal perforation. Plain films of the abdomen should include the patient in a supine position as well as either an upright or left lateral decubitus position to evaluate for possible pneumoperitoneum. Computed tomography remains the study of choice given its sensitivity and specificity for a bowel obstruction, with its ability to detect intestinal distention as well as transition points of obstructed and non-obstructed intestine along the intestinal tract. Additionally, computed tomography may elucidate the etiology for the obstructive process resulting from either a mass effect, an inflammatory process, intraluminal masses, and/or intussusception.

Management

Initial intervention for the treatment of bowel obstruction should consist of aggressive resuscitation for the treatment of dehydration as well as correction of associated electrolyte disorders. The use of an isotonic crystalloid solution like normal saline will provide the needed chloride supplementation as well as the free water to address the extravascular fluid imbalance. Hemodynamic monitoring should be utilized until stable. Achievement of appropriate urine output will provide the best indicator of correction of the associated dehydration.

Nasogastric decompression should be utilized to decompress the stomach and the proximal intestinal tract. This provides comfort to the patient through gastric decompression and from the avoidance of additional vomiting. In addition, it provides assessment of the character of the gastric contents that may indicate complete obstruction by the presence of bilious material. Serial abdominal exams should be performed in an effort to identify any progressive signs or symptoms that would indicate intestinal ischemia and early peritonitis. For this reason, the use of narcotic medications should be avoided to minimize masking peritoneal irritation that could indicate early signs of intestinal ischemia and need for surgical intervention. The relief of obstruction related to an adhesive process in the first few months after an operation approaches 90% but is less successful thereafter. Failure to respond to non-operative management or the progression of symptoms that may indicate intestinal ischemia that would warrant operative intervention by way of abdominal exploration and correction of the obstructive process.

Some centers are using hyperosmolar solution via the NGT after a period of decompression. This is followed by another abdominal plain film to see if the contrast has progressed. If the contrast has progressed into the colon, then the patient is likely to have resolution of their symptoms in the following 24-48 hours. However, if the contrast does not make it into the colon by 24 hours, then it is unlikely the obstruction will resolve without surgery. Some surgeons use this method to more quickly decide who needs an operation and who will resolve without one.

DIARRHEA

Classification for diarrhea is often described as malabsorptive, watery, and/or inflammatory. Many diarrheal diseases may have more than one mechanism for the resulting increase in stool volume and stool weight. Most infectious causes for diarrhea often will be in the "watery" diarrheal classification for the increased chloride secretion and often inhibition of sodium absorption in the colon leading to a secretory diarrheal process. Diarrhea related to inflammation may cause the inhibition of sodium absorption within the intestine and a secretory process. Inflammatory diarrhea may also include underlying damage to the enterocyte cell membrane with resultant reduction in sugar absorption and peptide hydrolization leading to further reduction in sodium chloride absorption. Severe inflammation may progress to underlying vascular damage and ulceration, leading to an exudative loss from capillaries that will contribute to the diarrhea.

Aside from solute-related, osmotic-related, and malabsorptive etiologies for diarrhea, the causes of diarrhea with potential surgical importance occur with either extensive inflammation and/or

ulceration and vascular impact from infectious processes, e.g. Clostridium difficile infection, or the hallmark inflammatory diarrhea of the inflammatory bowel disease entities.

Pseudomembranous Colitis

The diarrheal process that results from a *Clostridium difficile* infection is a result of the impact of toxins on the colonic mucosa and the associated cellular inflammation and cell death. This process is also known as pseudomembranous colitis given the endoscopic appearance of the colon surface. Diagnosis is typically made by testing stool for *C diff* toxin and also by visualizing pseudomembranes on sigmoidoscopy. In the more advanced cases of *Clostridium difficile* colitis, early symptoms of fever, abdominal pain, and abdominal distention may progress to signs of overwhelming infection with hypotension and shock. Fulminant colitis may result in clinical sepsis from overwhelming infection that causes mucosal-barrier breakdown, bacterial translocation across the intestinal wall, and bacteremia; which may lead to patient mortality if unrecognized and untreated. Aggressive fluid resuscitation as well as antibiotic treatment with both oral vancomycin and intravenous metronidazole should be initiated as first line therapy. The treatment of fulminant colitis and vigilance for sepsis may require intensive care unit stay for appropriate patient monitoring. Close monitoring of the patient's response to treatment or lack thereof should be utilized to determine signs of failure in medical management, including worsening acidosis, renal insufficiency and other end organ failure. The decision for surgical intervention in the treatment of fulminant colitis remains complex and is based on disease symptomatology and severity, as well as, response to medical management (or lack thereof). Cases requiring surgical intervention require an abdominal colectomy with ileostomy with intent to preserve a short rectal stump at the level of the peritoneal reflection for future reconnection. Overall mortality rates for patients diagnosed with *Clostridium difficile* colitis have increased five-fold over the last 15 years, depicting more virulent strains and toxins of *Clostridium difficile* and the associated colitis.

Ischemic Colitis

Ischemic colitis (IC) is a disease process that may lead to the presence of diarrhea and has the potential for surgical intervention. This condition is distinct from acute mesenteric ischemia, where occlusion of major blood vessels to the intestine causes acute ischemia and need for immediate intervention. Ischemic colitis is generally a disease of small blood vessels with a more vague set of complaints and less definitive presentation than that of acute mesenteric ischemia. Presenting signs and symptoms often include abdominal cramping with mild to moderate lower abdominal pain that is poorly localized. It is often associated with the urge to defecate and hematochezia and/or bloody diarrhea within 24 hours of the onset of abdominal pain. The classic signs of abdominal pain, tenesmus, and hematochezia may only present in approximately one half of those patients with this condition. Risk factors would include atherosclerosis, septic shock or hypotension, transabdominal vascular surgery, and hypercoagulable states. Medications that may be associated with the onset of ischemic colitis include opioids, cocaine, and some immunomodulators. IC usually is associated with nausea, vomiting, and low-grade fever. Areas affected are usually the "watershed" locations of the colon including the ileocecal distribution, the splenic flexure, and the rectosigmoid region. The majority of cases are self-limited with appropriate supportive care.

Diagnosis relies heavily on an elevated clinical suspicion based upon the presenting signs and symptoms of the patient. Computed tomography of the abdomen is the main method for diagnosis of the colonic disease, with radiologic findings suggestive of colitis through demonstration of bowel wall thickening and adjacent soft tissue fat stranding and the presence of ascites. More severe disease may be indicated by the presence of pneumatosis, portal venous gas, and/or the presence of megacolon. With a suggestion of ischemic colitis by radiographic imaging, colonoscopy should be utilized to confirm the diagnosis through visualization of the colonic mucosa for signs of ulceration or ischemia. Biopsies should be taken of the involved areas of colitis. Endoscopy is contraindicated in cases presenting as acute peritonitis or with extensive signs of ischemia including pneumatosis, pneumoperitoneum, or portal venous gas.

Greater than three-fourths of patients with ischemic colitis respond to conservative treatment with bowel rest, fluid resuscitation, and broad-spectrum antibiotics, including aerobic and anaerobic coverage to address the risk of bacterial translocation. Fluid resuscitation should be optimized to achieve and maintain hemodynamic stability. Serial monitoring of the patient's condition, abdominal examination, and imaging is required to identify any progression of disease or complicating colonic necrosis or perforation that would indicate the need for surgical management. Surgical management of complicated ischemic colitis would focus on identification of the involved segments of intestine and resection of the frankly necrotic or perforated bowel segments and intestinal diversion by way of a colostomy or proximal ileostomy. Full extent of disease may be difficult to determine and may dictate a second-look laparotomy within 24 hours for reassessment of colon viability.

Complicated Ulcerative Colitis

Ulcerative colitis is an inflammatory bowel disorder that is defined by inflammation confined to the mucosal level of the intestine. It is a chronic disorder that involves the rectum and variable lengths of more proximal colon in a contiguous manner. It is this manner of contiguous involvement of the colon that allows for its differentiation from Crohn's disease. Presenting symptoms often include diarrhea and bloody stools. Pain is an uncommon symptom of the disease unless there is extension of the inflammation to the serosal level of the intestine. The gold standard test for its diagnosis remains endoscopy with multiple biopsies of the colon extending from the rectum to the distal terminal ileum, in order to exclude Crohn's disease. Alternative causes for diarrhea and rectal bleeding, including an infectious etiology, should be excluded before considering the diagnosis of ulcerative colitis.

The mainstay of treatment for ulcerative colitis lies in the use of immunosuppressive medications including acetylsalicylic acid, steroids, antipyrine or pyrimidine compounds, or newer immunologic therapy. The disease often becomes a chronic affliction that can lead to a medically refractory state or that of steroid dependence. The development of intractability of the disease process to medical management or complicating side effects of long-term therapy would indicate the need for surgical intervention. Severe disease leading to fulminant colitis and toxic megacolon may occur in 5-15% of patients with ulcerative colitis. Toxic colitis or megacolon refers to the signs of fever, tachycardia, and leukocytosis in a patient with ulcerative colitis concurrent with the presence of significant colonic distention greater than 8 cm in

diameter. The development of toxic colitis places the patient at risk for colonic perforation and will indicate the need for urgent surgical management. The risk of significant hemorrhage is unlikely but may also be an indication for urgent surgical intervention.

The cumulative risk of colorectal cancer in patients with ulcerative colitis, often with extensive involvement of the colon, is nearly 2% at 10 years, reaching 10% at 20 years. This risk increases to a 50-75% cumulative risk by 30 and 40 years of disease, respectively. This risk conveys the need for endoscopic surveillance on an annual basis beginning 10 years from the onset of symptoms. Endoscopic biopsy findings for high-grade dysplasia or malignancy is an additional indication for surgical intervention.

As ulcerative colitis is a process localized to the colon; performance of colectomy is essentially curative. Under urgent circumstances performance of a subtotal abdominal colectomy either open or laparoscopically will eradicate the majority of the disease. An end ileostomy would be performed in conjunction with colectomy until resolution of the acute disease in the remnant rectum. Final restorative surgery will include the anastomosis of the small intestine to the remnant of rectum leaving the patient with functional continence. The use of an ileal pouch to provide a fecal reservoir aids patient's continence of the liquid fecal material. In cases of more elective interventions related to intractable disease, a total proctocolectomy may be performed with anal anastomosis, with or without diverting ileostomy. After healing of the anastomosis, the diverting ileostomy is reversed. With the continued risk of malignancy in any remaining rectum, there remains the need for ongoing surveillance of any residual rectum.

Crohn's Disease

Crohn's disease is signified by a chronic transmural inflammatory disease of the gastrointestinal tract. It is not confined to any particular segment of the gastrointestinal tract and may affect any single or multiple portions of the gastrointestinal tract concurrently or sequentially. Given the various potential locations of disease within the gastrointestinal tract, surgical management of this disease is not curative. Thus, surgical intervention is reserved for management of the complications of the disease. The secondary goal for any surgical intervention associated with Crohn's disease is to maintain any functional segments of intestine for appropriate electrolyte and caloric absorption. Its presenting signs often include diarrhea with associated abdominal pain. Symptoms may also include anorexia and weight loss, nausea and/or vomiting, hematochezia, and potential findings from the chronic nature of the disease, including growth failure and vitamin and nutritional deficiencies.

Similar to ulcerative colitis, diagnosis is made through endoscopic evaluation of the gastrointestinal tract with biopsy to determine the presence of transmural inflammation as a hallmark of Crohn's disease. At initial presentation, up to 40% of patients will have terminal ileal disease, while 20% will have isolated colonic disease. Ten percent may initially present with perianal disease alone, which is manifest by multiple perianal fistulae and is referred to as a watering can perineum. Medical management is the mainstay of treatment for Crohn's disease with the use of anti-tumor necrosis factor-type biologic agents and steroid treatment. Newer agents that affect the immune process and its contribution to the chronic inflammatory process have demonstrated a significant impact in the treatment of Crohn's disease.

Surgical management of Crohn's disease focuses on the treatment of the complications of this disease and/or its intractability. Failed medical management of active disease, intestinal obstruction, fistula formation, intestinal perforation and/or abscess formation would lead to the acute need for surgical intervention. More chronic findings of disease intractable to medical management, growth retardation, and poor response to maximal medical therapy would also indicate the need for surgery. Surgical intervention is then dependent upon the presenting features of the disease process but often entails surgical resection of the actively involved segment of intestine, minimizing excessive resection. Strictureplasty of chronically involved, scarred segments of intestine without active disease may be offered to mitigate a stricture **(see Figure 1)** while maintaining the absorptive surface area of the involved segment of intestine. Surgical interventions may be performed on an open or minimally invasive approach depending upon the nature of the complicating process.

FIGURE 1 - Crohn's Stricture (photo provided courtesy of Dr. Jeremy Lipman)
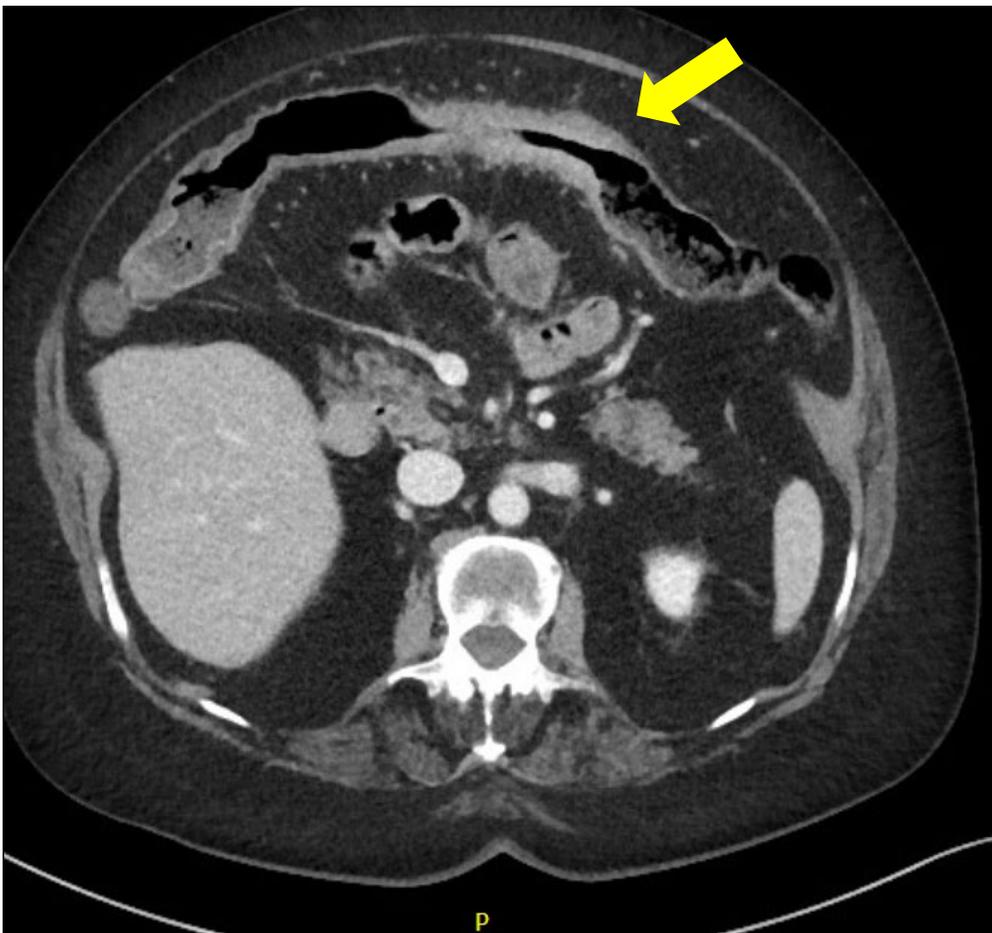
CONSTIPATION

Constipation and Large Intestine Obstruction

Constipation, or the failed or reduced ability to pass fecal material, is reported in about 20% of the population. Overall constipation may be divided into pathophysiologic categories of constipation with normal transit, slow colonic transit, or evacuation disorders. In children an additional diagnosis of the lack of intrinsic nerves to the myenteric plexus of the intestine, Hirschsprung's disease, can also be a contributor to failed passage of stool. In Hirschsprung's disease, the failure to pass stool often occurs within the first 24 hours of life, however, delayed identification of this diagnosis has been made beyond 1 year of age.

In general, treatment of slow colonic transit constipation focuses on the use of a high fiber diet to aid colonic transit. Evacuation disorders often entail dysfunction of the anal sphincter from a variety of potential etiologies, including iatrogenic injury to the sphincter from prior surgery. Anorectal anatomic abnormalities, such as a rectocele, should be addressed as first line management for these types of evacuation disorders. Laxative treatment is often reserved for the treatment of constipation with no underlying anorectal or anatomic disorder that would be improved with stimulatory function.

Large bowel obstructions have a largely different spectrum of etiologies compared to those for small bowel obstruction. Some etiologies including inflammatory bowel disease, incarcerated hernia, adhesion-related obstruction, and foreign bodies are similar between the small and large intestine obstructive processes. The causes of large bowel obstruction are often categorized as functional or mechanical obstructions. The vast majority of mechanical large bowel obstructions are related to neoplastic processes resulting in near-complete or complete obstruction from tumor burden. Unique etiologies for colonic obstruction include adenocarcinoma, diverticulitis, inflammatory bowel disease from ulcerative colitis, infection, and fecal impaction.

Large bowel obstruction manifests with a variety of symptoms, mostly related to the chronicity of the obstructive process. Early signs and symptoms will include abdominal pain, abdominal distention, and possible obstipation. Vomiting is usually a later sign of large bowel obstruction that results from the propagation of luminal distention from the level of obstruction into the small bowel. Clinically concerning symptoms of perforation including peritonitis, sepsis, and shock may indicate urgency in the needed treatment and surgical intervention.

Early management still consists of proximal decompression by way of a nasogastric tube, intravenous fluid resuscitation and correction of electrolyte imbalance, and possible utilization of empiric antibiotic coverage depending on the presumed cause of the obstructive symptoms. Early management should consist of a thorough abdominal examination to evaluate for any signs of an acute abdomen that would warrant urgent surgical intervention. Radiographic imaging of the abdomen should consist of a flat abdominal radiograph as well as an upright or cross-table lateral X-ray of the abdomen. **(see Figure 2)**.

FIGURE 2 - Relevant Diagnostic Studies

- Malignancy[1]

The multi-plane imaging of the abdomen helps to discern the potential location of the obstructive process, as well as the possible presence of intra-abdominal free air. Cross-sectional imaging by way of computed tomography additionally assists in possible delineation of the obstructive process as well as the location of the obstruction. **(See Figure 3.)** Measurement of cecal dilation is important in patient assessment, as a diameter 12 cm or greater would signify greater risk of ischemia from intraluminal pressure and the potential for perforation.

FIGURE 3 - Diverticular Stricture (photo provided courtesy of Dr. Jeremy Lipman)
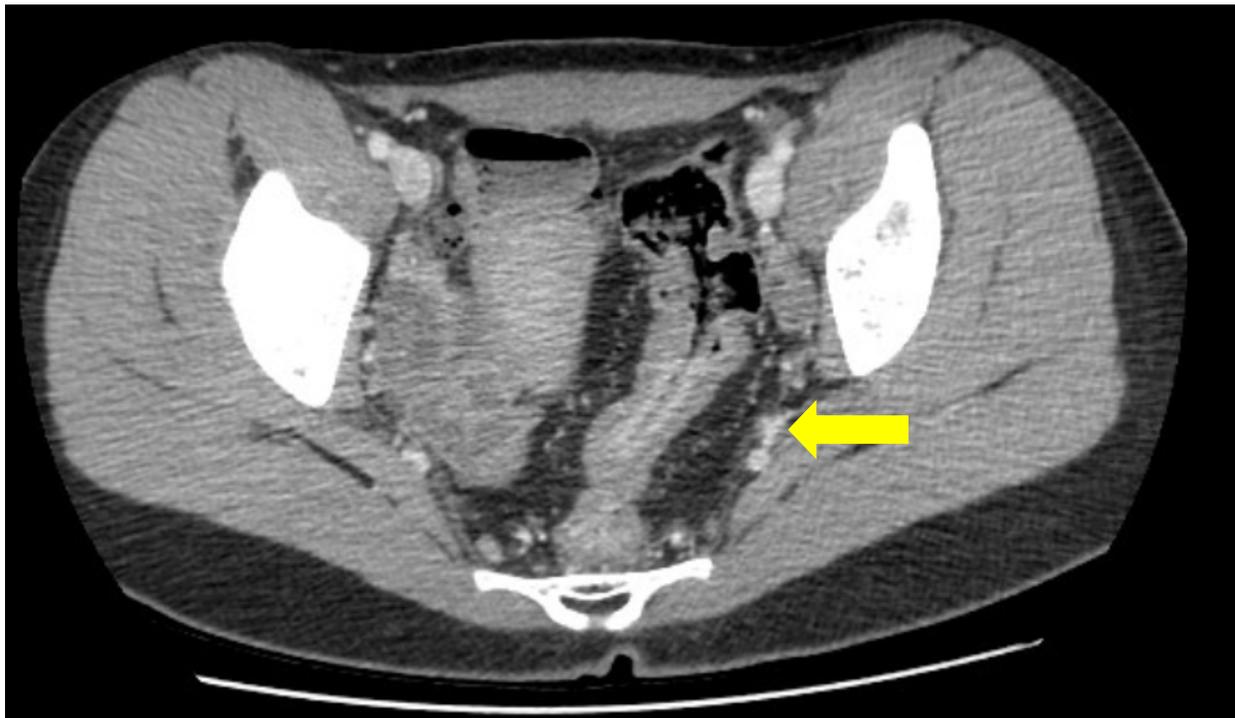
Each etiology for large bowel obstruction will dictate a specific management and possible surgical considerations in the management of that process. In general, surgical excision of the involved segment may be required. Intestinal diversion by way of a loop colostomy or ileostomy for decompression of the proximal intestine may be required if the underlying process is not amenable to direct surgical extirpation, thus allowing for adjuvant treatment of the underlying process. Conditions such as sigmoid volvulus **(see Figure 4)** may be approached by decompressive endoscopy which allows for detorsion of the colon and relief of the obstruction. Successful detorsion with endoscopy approaches 95% in patients without signs of an acute abdomen or intestinal ischemia. There is a high rate of recurrence of sigmoid volvulus and, thus, there should be consideration of a semi-elective sigmoid resection during the presenting hospital course, following successful reduction.

FIGURE 4 - Sigmoid Volvulus[1]

Treatment of a functional obstructive process such as pseudo-obstruction requires surgery only as a last resort. Correction of any electrolyte or fluid imbalance should be the primary consideration in management. Investigation for the contribution of opiates or antimotility medications as a cause of the disorder should be performed and addressed if identified. Colonic decompression may be required by way of endoscopy to relieve the associated intraluminal pressure. Intractable functional obstruction would then warrant surgical intervention by way of a subtotal colectomy and end ileostomy.

Basic Postoperative Care

Usually, after elective colonic resection, patients are encouraged to resume a diet quickly and to expedite their post-operative recovery. However, patients who had an urgent resection for a large bowel obstruction are usually managed differently. These patients typically have significantly slower return of bowel function and a higher incidence of post-operative ileus, likely related to the dilation of their bowel. Nevertheless, early enteral nutrition is associated with fewer postoperative complications. Fluid and electrolyte management is important during this time. Patients are monitored for common post-operative complications, including:

- Superficial surgical site infection
- Intra-abdominal abscess
- Venous thromboembolic events
- Atelectasis and pneumonia
- Anastomotic leak – it is important to note that even in patients who have a proximal ileostomy for fecal diversion can still have an anastomotic leak. The sequelae of a leak, however, are usually significantly less than in a patient who was not diverted due to the majority of enteric contents exiting through the ileostomy. This is the principal reason behind performing proximal fecal diversion. The timing of the leak is often post-operative day 5-7 and therefore one must not assume all is well in the first few days.

Patients with an ileostomy require special attention, particularly to their bowel function. As the ileostomy begins to function it is important to ensure they do not become hypovolemic or have electrolyte disturbances. If an ostomy produces more than around 1000 ml per day, the patient can be at risk for dehydration. Medications can be used to slow the ostomy output when needed. Patients with an ostomy will also require teaching by their healthcare team regarding its care and management.

Optional Content

Surgical intervention for colorectal carcinoma focuses on either the complete resection of the cancer or palliative intervention for unresectable lesions. The premise for resectional therapy includes resection of the malignancy along with negative margins as well as the removal of the surrounding draining lymph node basin. It is often the draining lymph node basins that dictates the extent of colonic resection that is often of a greater length of resection than would be required to achieve negative margins. The lymph node basin for such a malignancy can be determined based upon the vascular inflow and outflow for a particular lesion, along with the contributing venous drainage pattern. This then allows one to map out a planned resection for potential cure. For example, a tumor of the ascending colon would include the lymphatic basin along the entire right colic artery and vein, thus requiring a complete cecal and right colon resection to the level of the hepatic flexure. Depending on the location of sigmoid cancers, either a left hemicolectomy or a sigmoid colectomy may be performed to eliminate the affected lymph node basin. Surgical intervention may be performed with either an open or laparoscopic approach based upon surgeon training.

Surgical staging at the time of colon resection should also be performed to evaluate any evidence for metastatic disease. Areas of direct extension from the intestinal tract should be resected en bloc with the surgical specimen. Evaluation of the liver for metastatic disease should also be performed. Patients with node-positive, stage III, colon cancer should be considered for postoperative adjuvant chemotherapy. Commonly utilized chemotherapy regimens include oxaliplatin complying with fluoropyrimidine. Adjuvant radiation therapy is not indicated for colon cancer. Surveillance for colon adenocarcinoma would include follow-up clinical assessments and CEA level every 3 months for the first 2 years after treatment and then every 6 months for the next 3 years. Annual cross-sectional imaging with computed tomography is also recommended as well as annual colonoscopy.

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