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Unusual causes of large bowel obstruction

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Introduction

Large bowel obstruction (LBO) is defined as a surgical emergency where a mechanical interruption (either complete or partial) occludes the flow of intestinal contents.[1] Understanding the varying etiologic causes of LBO is important for clinicians and surgeons when tailoring management to each patient. Knowledge of large bowel anatomy, embryology, and pathophysiology is vital when investigating and treating LBO.

Many clinicians will have encountered patients with LBO on a ward or in the operating room and will appreciate the challenges posed by such presentations. Although less common than small bowel obstruction (25% of all intestinal obstructions[2]) LBO poses more immediate risks in the form of perforation and subsequent peritonitis. Establishing the cause of an obstruction is paramount, given the high associated morbidity and mortality,[3] in order to facilitate the guidance of treatment. Recent studies highlight high morbidity and mortality rates of 42% to 46% and 13% to 19%, respectively, following operation.[3,4]

LBO accounts for nearly 2% to 4% of all surgical admissions.[5] Colonic malignancy remains the most common cause of LBO, representing approximately 60% of cases.[3,6] Other prevalent etiologies relate to adhesions, diverticulosis, hernia, inflammatory bowel disease (IBD), and volvulus.[7]

Within the published literature, much has been documented regarding the common causes of LBO. However, it is the etiologies we see less often and understand less that form the focus of this review.

We aim to highlight the causes of LBO, which although encountered less frequently, should be considered when more common pathologies have been excluded. Utilizing the surgical sieve, we break down this large subject into more manageable segments. Multiple searches were conducted on each subtopic using different search engines that included PubMed, Google Scholar,

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Medline, and Embase. The subsequent cases were reviewed; and those both relevant and pertinent to the monograph were included.

This review aims to help clinicians tasked with investigating and managing such patients. We will consider a range of aspects relating to LBO, including the historical context, epidemiology, anatomy, clinical presentation, investigations, and management before focusing on rare etiologies.

History of LBO

LBO Before the Common Era (BCE)

Accounts of bowel obstruction go back several millennia. The Ebers Papyrus is thought to have described 1 of the first ever cases 3400 years before the common era (BCE) in ancient Egypt.[8] Hippocrates (born 460 BCE) felt that bowel obstruction fell within the domain of the physician, and this practice was accepted up until the 18th and 19th centuries. He recognized conditions such as sigmoid volvulus and intussusception, managing the disorders with air insufflation.[9,10] However, it was another Greek named Praxagoras who is credited with performing the first operation for small bowel obstruction by creating an enterocutaneous fistula in 350 BCE.[11]

LBO in the postclassical era

Surgery has been performed for centuries, with the first successful suturing of the bowel reported by Saliceto in the 13th century.[12] Accounts from the 16th and 17th centuries document the use of a stoma, and by the late 19th century, exteriorization of the bowel via ileostomy, cecostomy, or colostomy was more frequently reported.[13] Pillore of Rouen in 1776 carried out the first elective colostomy for rectal cancer.[14] Reybard successfully undertook a sigmoid resection with immediate anastomosis in 1823 without anesthetic. By 1899, Billroth had performed a colonic resection and the formation of a colostomy.[15]

LBO in the early modern era

Modern day surgery owes much to the work of the surgeons at the turn of the 20th century. At this time Frank Thomas Paul and Johannes von Mikulicz-Radecki popularized the exteriorization of bowel for tumor resection of colonic left-sided tumors (Paul-Mikulicz operation).[15] Mikulicz reported a 43% reduction in mortality rate with primary anastomosis to 12.5% by using exteriorization.[15] Sir William Arbuthnot Lane pioneered the safe practice of colectomy in patients with colonic inertia.[16]

Stomas represent a valuable adjunct in the surgeon's arsenal when managing patients with LBO in the emergency setting. Current indications for stomas include lavage, exteriorization, defunctioning, and diversion. Current practice dictates that a patient is defunctioned if there is concern relating to the anastomosis or there is evidence of perforation. Defunctioning diverts intestinal flow thereby preventing potential leakage of intestinal contents through an anastomosis or a diseased segment of bowel.[17,18]

In 1900, Gibson reported that 35% of intestinal obstructions were due to strangulated hernias (47% mortality rate), while 19% were due to adhesive bands.[19] We now know that the incidence and prevalence of LBO differ markedly.

LBO in the modern era

Technological advancement throughout the 20th century has seen many changes to both the knowledge base surrounding LBO as well as the investigations, equipment, and management strategies available.

An important differential of LBO remains Ogilvie syndrome or pseudo-obstruction caused by disruption to the innervation of the colon.[20] Ogilvie observed 2 patients with retroperitoneal tumors invading the splanchnic plexus and concluded that such infiltration resulted in unopposed parasympathetic innervation of the distal colon.[20] This differs from true mechanical obstruction in that motility disturbance results in stasis of intestinal contents as opposed to luminal compression or occlusion.

Although first described in 1948 by Ogilvie, the etiology remains unclear, with more than 50 potential causes. Intestinal pseudo-obstruction is linked to a variety of conditions and most commonly occurs in the elderly. Pelvic/gynecologic surgery, electrolyte imbalances, and infection represent some of the etiologic causes.[21] Nonoperative management involves fluid resuscitation, nasogastric tube insertion, and correction of any electrolyte imbalance. Pharmacologic management revolves around intravenous neostigmine administration. This acts as a reversible acetylcholinesterase inhibitor, thereby increasing acetylcholine and promoting colonic motor activity.[22] Insertion of a flatus tube and surgery remain options in the event that a patient does not respond to other less-invasive management strategies.

The first single contrast agent colonic examination was reported by Schule in 1904 using bismuth subnitrate.[23] Over the following decades, barium contrast studies were refined to become a staple investigation of patients with colonic pathology. Double contrast barium enemas (using air and contrast) became ever popular by the 1960s and 1970s, helping clinicians diagnose colonic neoplasms and polyps with greater accuracy than ever before. Nevertheless, toward the end of the century, its role in modern diagnostic radiology began to diminish owing to the advent, development, and refinement of colonoscopy, magnetic resonance imaging (MRI), and computed tomography (CT) imaging.

In 1986, Dudley and Paterson-Brown reported that pseudo-obstruction and mechanical LBO cannot be distinguished based on radiographs alone.[24] This evidence resulted in changes to the management algorithm of LBO with the introduction of contrast enemas prior to laparotomy, helping to reduce unnecessary surgery and associated morbidity.

Röntgen discovered X-rays in 1895 by and they have played a vital role in clinical diagnostics since. Abdominal radiographs have been a mainstay of radiological investigations for LBO. However, in recent years there has been a switch to CT as the more sensitive diagnostic tool.[6] CT dates back to 1971, when Godfrey Hounsfield pioneered the technology at the Atkinson Morley Hospital, London, UK. Rapid advances in the field of CT have continued through the 1970s, 1980s, and into the 21st century.[25]

Endoscopy in the form of colonoscopy or flexible sigmoidoscopy plays a valuable diagnostic and therapeutic role in patients with LBO. Although Bozzini first attempted endoscopy in 1806, it was Drs Niwa and Yamagata (Tokyo University) in the 1960s who developed colonoscopy, as we know it today. Subsequently, Dr William Wolff and Dr Hiromi Shinya further pioneered the device's advancement after 1968.[26] Dohmoto and colleagues first used colonic stents in 1990 as a palliative adjunct to manage patients with colonic malignancy.[27] Colonoscopy has the potential to be both diagnostic and therapeutic in the setting of LBO. This technique can help distinguish true obstruction from pseudo-obstruction. Biopsies, lithotripsy, polyp removal, air insufflation, and the insertion of stents can all be undertaken without submitting a patient to the risks of general anesthesia or surgery.

Laparoscopic and minimally invasive surgery is an evolving field which clinicians are utilizing ever more when managing patients with LBO. Benefits include shorter hospitals stays, less (postoperative) pain, and decreased morbidity.[28] It was not until 1987 that the first laparoscopic cholecystectomy was performed by Mouret.[29] Following this, Jacobs and colleagues carried out a laparoscopic right hemicolectomy in 1990.[30] Subsequent technological innovations have seen such practice become widely accepted and incorporated into the general and emergency surgeon's practice.[31]

Over the past 150 years, significant advances have been made across all areas of medicine and surgery. Surgery is now safer and less invasive, while clinical diagnostics are more sensitive. Such developments have been fast paced and have resulted in a reduction in mortality for patients with LBO. Nonetheless, clinicians are still practicing reactive medicine rather than

preventative. Just as the surgeons in the 19th century encountered patients with LBO so do the surgeons of today. The future is unknown, however with history as an aid, there are likely to be more advances as technology continues to drive innovation and creativity through the 21st century.

Epidemiology of LBO

Distribution of LBO

The large bowel is involved in approximately 25% of cases of acute bowel obstruction.[32] Although LBO occurs less frequently than small bowel obstruction, it accounts for approximately 2% to 4% of emergency surgical admissions.[5,33] LBO is associated with a higher morbidity and mortality, often due to delayed diagnosis or treatment.[7]

Due to the changing caliber of the large bowel and fecal contents within it, the majority of cases of LBO are due to obstruction on the left side of the colon,[7] which reflects the pathophysiology of the most common causes of LBO. More than 75% of colorectal cancers causing obstruction are distal to the splenic flexure.

Determinants of LBO

There has been a significant change in the etiology of LBO. Colorectal cancer is now the most common cause of LBO in developed countries.[34] According to the National Institute for Clinical Excellence, up to 30% of cases of colorectal cancer present in the emergency setting, with approximately 15% of patients presenting with LBO.[35] Volvulus is the second most common cause, accounting for approximately 10% of cases. This contrasts with some developing countries, where volvulus is the most common cause,[36] accounting for approximately 85% of the LBO workload.[37] Indeed, a century ago, volvulus was the most common cause of LBO globally,[32] but increased longevity in developed countries has resulted in a higher incidence of cancer overall, and therefore higher rates of associated complications including LBO.

The incidence of LBO in men and women is similar, although this varies by cause. In 1 study that looked at LBO secondary to colorectal carcinoma, the sex distribution showed a male predominance, with men accounting for approximately 57% of patients.[38] This preponderance for men is to be anticipated. Data published from the office for national statistics reported that, of the 34,025 cases of colorectal cancer in the UK registered in 2014, more than 55% were men.[39] In the study by Suan, the mean age of patients with LBO was approximately 62 years.[38] Again, this also reflects increasing longevity.

Factors that increase the risk of LBO are mainly attributable to the cause. For example, LBO is more common in elderly patients, mainly due to the higher incidence of malignancy, diverticulitis, and other causative pathology in this age group. In patients with colorectal cancer, increasing time from diagnosis is associated with an increased risk of LBO.[40] Poor mobility, mental illness, and living in an institutional environment are also considered risk factors for LBO, and are particularly associated with the development of volvulus.[37] Individuals with predisposing conditions such as IBD, or those that have had radiation for previous cancer are also at higher risk of LBO,[41] as are those who have undergone previous abdominal surgery.[42]

Embryology

The embryologic gastrointestinal tract is divided into the pharyngeal gut, foregut, midgut, and hindgut. These subdivisions originate from the endodermal yolk sac following craniocaudal and lateral folding, to give rise to the primitive gut.[43] The large intestine is formed from these primitive midgut and hindgut structures.

The midgut starts at the entrance of the common bile duct into the duodenum and ends at the caudal one-third of the transverse colon. At 5 weeks, the vitelline duct connects the midgut

to the yolk sac.[43] The midgut subsequently elongates to form the primary intestinal loop that continues to have a connection with the yolk sac through the vitelline duct. Later failure of the vitelline duct to obliterate gives rise to a Meckel's diverticulum.

Over the following weeks, the primary intestinal loop rotates 270° anticlockwise around the axis of the superior mesenteric artery. Due to rapid growth, the loop temporarily herniates into the umbilical cord. If the loop does not return into the abdomen, this is known as an omphalocele.

The caudal section of the midgut eventually gives rise to the cecum, appendix, ascending colon, and proximal transverse colon. By week 6, the cecal bud has moved from the right upper quadrant to the right iliac fossa, allowing the ascending colon to assume its final position on the right side of the abdomen.

During rotation of the midgut, the hindgut is carried with it to adopt a final position on the left. It commences at the distal one-third of the transverse colon and extends to the ectodermal part of the anal canal.[44] At the termination of the hindgut, an endodermal pouch known as the cloaca is shared with the developing urogenital tract. If the membrane around the cloaca does not break down, this results in an imperforate anus.

The hindgut can also be rendered aganglionic if neural crest cells fail to migrate to their final position. Because the bowel that arises from this aganglionic segment is deficient in both submucosal and myenteric plexuses, obstruction ensues due to unopposed tonic contraction. It manifests in the newborn with a grossly distended abdomen and failure to pass meconium, a condition known as Hirschprung's disease.[43]

Anatomy

The course of the large bowel extends from the terminal ileum to the anus.[45] At 1.5m long, the large intestine consists of the cecum, ascending colon, transverse colon, descending colon, and sigmoid colon. It has epiploic appendices on its surface, which consist of fat enveloped in peritoneum.[46] The walls of the large bowel contain 3 bands of longitudinal muscle known as the teniae coli that begin at the base of the appendix and terminate at the rectosigmoid junction. As the teniae coli are approximately a foot shorter than the length of the bowel itself, they contract it into sacculations, known as haustra. The ascending and descending colon are retroperitoneal, whereas the transverse and sigmoid colon are intraperitoneal.[46]

The parts of the large bowel derived from midgut structures are supplied by branches of the superior mesenteric artery, which divide into the ileocolic, right colic, and middle colic vessels. The latter one-third of the transverse colon and descending colon are supplied by branches of the inferior mesenteric artery, the left colic, and sigmoid arteries. The marginal artery of Drummond forms an anastomotic network of arteries that passes along the mesenteric border of the colon.

The large bowel is drained primarily by the portal vein, which is formed by the confluence of the splenic vein and superior and inferior mesenteric veins.[46] The portal vein returns venous blood to the inferior vena cava, via the liver.

The large bowel is innervated by sympathetics, which follow the course of the superior and inferior mesenteric arteries.[45]

Cecum

At 7.5cm long and wide, the cecum lies in the right iliac fossa. The ileocecal valve closes when the cecum distends, due to constriction of the frenula of the ileal orifice.[47] This prevents reflux of cecal contents into the ileum. The ileocolic artery, derived from the superior mesenteric artery, supplies the cecum. Venous drainage of the cecum is via the ileocolic vein, which drains into the superior mesenteric vein and, later, the portal vein. The lymphatics of the cecum follow the arterial supply and drainage is along the ileocolic nodes to the superior mesenteric lymph nodes.[47]

Ascending colon

The ascending colon passes superiorly toward the liver and curves at the hepatic flexure to become the transverse colon. The lumen of the ascending colon is narrower than at the cecum.[47]

The ascending colon is supplied by the ileocolic and right colic arteries, which are derived from the superior mesenteric artery, and is drained by the ileocolic and right colic veins, which pass into the superior mesenteric vein.[47]

Transverse colon

Not only is the transverse colon the longest segment of the large intestine, it is also the most mobile.[47] It runs from the hepatic to the splenic flexure, where it is attached to the diaphragm by the phrenicocolic ligament.

This segment is supplied primarily by the middle colic artery but receives a supply from the marginal artery of Drummond.[47] The superior mesenteric vein drains the transverse colon and lymphatics are via the middle colic nodes. Just proximally to the splenic flexure, the arterial supply is poor. This is because this area of the transverse colon lies at the transition point of the superior to inferior mesenteric vessels, and marks the boundary between midgut and hindgut structures.[47]

Descending and sigmoid colon

The descending colon continues into the S-shaped sigmoid colon. In contrast to the descending colon's short mesentery, the sigmoid colon has a long mesentery that offers it greater mobility.[47] This renders the sigmoid colon more prone to volvulus. Length of mesosigmoid and the width of its base are strongly associated with increased risk of sigmoid volvulus. Other risk factors include diabetes, neuropsychiatric diagnoses, and immobility.

The left colic and sigmoid arteries supply the descending and sigmoid colon and are derived from the inferior mesenteric arteries. Venous drainage occurs through the inferior mesenteric vein to the portal vein. Following the arterial supply, lymphatic drainage is via the intermediate colic lymph nodes to the inferior mesenteric nodes.[47]

Pathophysiology of LBO

Physiology of the large bowel

The large bowel primarily functions to store and concentrate fecal matter prior to defecation. The ileocecal sphincter regulates entry of chyme into the cecum. It is closed in the resting position but opens upon contraction of the terminal ileum to allow chyme to enter the cecum.[48] Any distension of the colon results in a reflex contraction of the ileocecal sphincter to prevent retrograde propulsion of fecal matter back into the small intestine.[48]

Approximately 1500 mL of chyme enters the colon every day. Sodium is actively transported into the blood with subsequent osmotic absorption of water.[49] The longer the fecal matter remains in the large intestine, the more water is absorbed.[50] The colon is also involved in potassium secretion and the excretion/absorption of bicarbonate and chloride, respectively.[49]

A complex bacterial ecosystem (with a population of approximately 10^{13} colony-forming units)[51] is present in the colon that metabolizes undigested polysaccharides and produces small quantities of essential vitamins.[52] Flatus is a by-product of this fermentation process.[50]

Propulsion and eventual expulsion of fecal matter through the large intestine requires an effectively functioning muscular apparatus. This consists predominantly of smooth muscle and

Table 1
Differential diagnosis of large bowel obstruction.[54]

Intraluminal	In the wall	Extraluminal
Intussusception	Congenital • Stenosis • Atresia • Imperforate anus	Mass compression • Abscess • Pregnancy • Neoplasia • Cysts • Urinary retention • Vascular malformation
Impacted matter • Feces • Foreign bodies • Medications • Bezoars • Meconium • Gallstones • Worms • Enteroliths	Inflammatory • Diverticulitis • Ulcerative colitis • Crohn's disease Neoplasia • Benign • Malignant Trauma/Iatrogenic • Hematoma • Stricture • Radiation	Adhesions Hernias Volvulus Pancreatitis Endometriosis

interstitial cells of Cajal directly controlled by the enteric nervous system but with inputs from both the sympathetic and parasympathetic nervous systems.[53] The discrete circular and longitudinal layers of smooth muscle coordinate to produce rhythmic and synchronized peristalsis of luminal contents.[53]

Pathophysiology

LBO is the occlusion of the lumen of the colon with subsequent dilation of the proximal bowel from swallowed air and chyme.[54] It can occur anywhere along the course of the large bowel but is most commonly within the left colon.[54] LBO can be functional (pseudo-obstruction) or mechanical, which can broadly be classified by the location of the obstruction in relation to the bowel wall: intraluminal, mural, or extraluminal (Table 1). LBO can also be classified according to whether partial or complete and simple or complicated. Complete obstruction blocks passage of all contents, whereas partial allows the passing of some liquid and/or gas through the obstructing point. In complicated obstruction, the circulation to the bowel is compromised resulting in ischemia (loss of blood supply), necrosis (tissue death), and perforation (loss of bowel wall continuity).

Colorectal carcinoma is the most common cause of LBO, accounting for 55%.[54] This is followed by volvulus (11%) and diverticulitis (9%).[54] Reports have suggested the reverse in developing countries, where volvulus has been reported as the most common cause, followed by colorectal carcinoma.[55]

The response of the colon to an obstruction depends upon the competency of the ileocecal valve. The large bowel is able to decompress into the small bowel in the presence of an incompetent valve. Approximately 75% of patients have a competent ileocecal valve that prevents colonic decompression, resulting in a closed loop obstruction.[6] In these circumstances, chyme can enter but not exit the colon, resulting in dilatation and an increase in wall tension progressing to ischemia, necrosis, and perforation without intervention.[6] Once ischemia starts, it takes just 3-4 hours before necrosis of the mucosal villi and 6 hours until transmural infarction takes place, shortly progressing to perforation.[56] Laplace's law states that the intraluminal pressure required to stretch the wall of a hollow tube is inversely proportional to its radius. Because the

cecum has the largest diameter of the large bowel, it therefore demands the least amount of pressure to cause distension and subsequent perforation.[57]

Clinical presentation of LBO

The clinical presentation of LBO includes a wide spectrum of signs and symptoms. Absolute constipation, abdominal pain, vomiting, nausea, and bloating are all recognized presenting complaints in LBO.[2] The exact nature of the acute presentation is dependent on the underlying cause; absolute constipation (90%), abdominal pain (74%), and distension (65%) tend to be the most frequently occurring signs and symptoms documented in the literature.[2] Vomiting tends to occur much later in the clinical course of LBO when compared with small bowel obstruction.[58] Strangulation or progression to bowel ischemia, necrosis, or perforation might be indicated by continuous abdominal pain or point tenderness, fever, tachycardia, and signs of peritonitis on physical examination.[2] The presentation of bowel ischemia can be relatively vague but must be considered if there is evidence of sepsis, an elevated serum lactate level, or a dilated cecum.[58]

Morbidity and mortality of LBO

The prognosis of patients with LBO depends largely upon the cause of the obstruction. Morbidity rates across all causes of both small bowel obstruction and LBO vary widely from 6% to 47%, with mortality rates varying from 2% to 19%.[2] Factors affecting these complication rates include age greater than 75 years, male sex, comorbidities, nonviable strangulation, previous operations, and malignancy.[3,59,60] Malignant bowel obstruction (MBO; obstruction caused by intra-abdominal malignancy, most commonly colorectal or ovarian) carries a very poor prognosis with a life expectancy of only 1-9 months.[61,62] Many in this group of patients suffer prolonged nausea, vomiting, and abdominal pain, and more than one half of those discharged will be readmitted to the hospital.[61]

Prompt management of patients with LBO can help pre-empt developing life-threatening complications with subsequent increased risk associated with drastic surgical intervention and risk of mortality. Rates are reported to vary from 7% to 42% with strangulation, 20% with ischemia, 8% with necrosis, and 2%[2] with perforation.

Much of the morbidity associated with LBO is related to the cause and subsequent management. Many patients will require colostomy formation, particularly in those with hemodynamic instability, perforation, or a poor premorbid state.[58] Colostomy formation is often associated with significant complications, specifically parastomal hernias, stenosis, retraction, prolapse, a reduction in quality of life, and low rates of reversal.[63] A proportion of patients are deemed suitable for segmental colectomy. These patients are at risk of anastomotic leak, wound infection, and stoma formation.[58] Finally, a select number of patients may undergo subtotal colectomy but are likely to suffer (ongoing) problems with frequent bowel movements, dehydration, and dietary restrictions.[58]

Investigating LBO

Laboratory

Once a thorough history and physical examination have been undertaken, laboratory tests are useful adjuncts to investigate the clinical state of the patient.[1] A full blood count, urea, electrolytes, coagulation studies, and C-reactive protein should be performed.

Full blood count allows the clinician to assess for raised white blood cells, which can suggest an inflammatory or infectiouis etiology, in addition to perforation.[34] In cases where malignancy is the cause, a microcytic anemia may also be present.

Due to third space losses, overwhelming sepsis, or dehydration, electrolyte imbalances may be present, the extent of which are dependent upon the duration of obstruction. Particular attention should be paid toward potassium, which is often reduced. In addition, serum urea and creatinine may be elevated as a result of dehydration.

C-reactive protein is likely to be raised in many causes of intestinal obstruction due to the ensuing inflammatory response. Coagulation studies may also be deranged in patients who are septic from perforation.

An arterial blood gas can also yield helpful information when evaluating these patients. Not only does this provide rapid analysis of electrolytes, but also generates a value for lactate and acid-base status that is vital to assess for ischemia and shock.

Radiological

Imaging poses a particular challenge in evaluating patients with suspected LBO. One in 3 patients in whom LBO is deemed a clinical probability based on history, physical examination, and radiographs, transpire to have no obstruction. In contrast, 20% of patients who are thought to have pseudo-obstruction (Ogilvie's syndrome) actually have mechanical LBO.[64] These statistics reflect the complex difficulties in discriminating between true obstruction and pseudo-obstruction radiologically.

First line radiological tests should include an erect chest radiograph and abdominal plain film. The presence of pneumoperitoneum (air under the diaphragm) is suggestive of visceral perforation, and therefore increases the likelihood that urgent intervention is required.

On an abdominal film, LBO is demonstrated by dilatation of the colon. The typical appearance seen in LBO is distension of the colon up to the level of the obstruction, with distal collapse. Commonly used diameters suggestive of pathology include 10- to 12-cm cecum, 8-cm ascending colon, and 6.5-cm rectosigmoid. In closed loop obstruction with a competent ileocecal valve, distension beyond 10 cm puts the patient at significant risk of perforation.

Radiographs may also provide evidence of pneumotosis intestinalis (intramural gas), a key stigma of ischemic colon. Despite this, pneumotosis is not pathognomonic of transmural infarction, and therefore should be interpreted merely as high risk for potential necrosis.[65] Abdominal radiographs can also help in assessing the chronicity of the condition. The presence of an air-fluid level is indicative of a more acute process, given that the fluid has had insufficient time to be absorbed.[6]

Plain radiographs have a sensitivity of 75% in identifying volvulus, where a coffee-bean appearance of the section of affected colon is seen. Cecal vs sigmoid volvulus can be differentiated by the rotation of right-sided colon to the left in cecal volvulus, whereas dilated colon points toward the right in sigmoid volvulus. When sigmoid volvulus is strongly indicated, flexible or rigid endoscopy can be both diagnostic and therapeutic, however it is commonly troublesome to cross the level of obstruction with the endoscope.[66]

Conversely to proximal obstruction, where distension terminates at the transition point and the distal colon is free of gas, it can be difficult to detect this change in more distal obstruction. In order to make the distinction between low obstruction and ileus, it is helpful to position the patient in the lateral decubitus position, to allow gas to enter the sigmoid and rectum.[67] Following this maneuver, consequent rectal collapse is suggestive of mechanical obstruction rather than ileus but further imaging is required if this distinction cannot be made.

Contrast enema can be used to further investigate the type of obstruction.[68] When the diagnosis is unclear on plain abdominal radiographs, contrast can be used to demonstrate a "bird's beak" appearance in cases of volvulus, including more unusual sites of volvulus such as the transverse colon.[6] Contrast enema improves the sensitivity and specificity compared to plain radiographs alone from 84% and 72%, respectively, to 96% and 98%.[68] The Association of Coloproctology of Great Britain and Ireland (ACPGBI) states that it is particularly useful in excluding Ogilvie's syndrome, which is difficult to exclude on plain radiographs alone, and in confirming the precise site of obstruction.[66] If the precise site of obstruction can be

demonstrated, this obviates the need for cross-sectional imaging. If perforation or peritonitis is likely, however, contrast enema should be avoided. In addition, failure to retain the enema is not uncommon[69] and as water-soluble contrasts are hyperosmolar, they can lead to dehydration, which can potentiate electrolyte and fluid disturbances.

CT is able to elicit the diagnosis of LBO in more than 90% of patients.[66] LBO appears as dilatation proximal to the level of obstruction at the so-called transition point, before distal collapse becomes apparent.[66] Colonic dilatation is evident on CT, using the same pathologic parameters as plain radiographs. CT has the ability to provide greater diagnostic information than contrast enema, particularly as it can identify the disease leading to extrinsic compression. Megibow found CT to be more sensitive and have a better negative predictive value when compared to contrast enema.[70]

In patients with LBO due to volvulus, the CT "whirl sign" can assist in determining the location of the volvulus. This is due to rotation of the bowel around its mesentery at the site of obstruction, with the mesenteric vessels creating the whirl appearance.[71] Macari and colleagues found a highly significant association between location of the whirl sign and location of the volvulus ($P < 0.0001$).[72] In patients with a right twist on CT, this was indicative of a cecal volvulus in 93.3%, whereas a left twist was indicative of sigmoid volvulus in 100%.

Jaffe and Thompson have reported on CT findings in colonic intussusception leading to LBO.[6] These include distended colon with transmural thickening, intraluminal telescoping, and a curvilinear appearance of fat due to invagination. Commonly, the bowel appearance in intussusception is analogous to a target in cross-section or a sausage longitudinally.

Furthermore, CT can provide a whole array of information where there is diagnostic uncertainty as to the etiology of the obstruction. For some of the rarer causes of LBO, CT is able to show evidence of infectious sequelae, bezoars, and extrinsic compression from atypical sources. Consequently, when the etiology is unclear, CT can provide evidence of the cause and assist in planning intervention.

There are a number of important investigations to carry out when LBO is a differential diagnosis. Laboratory tests, followed by radiological imaging, augment the clinical history and physical examination. Erect chest radiograph and plain abdominal radiographs help assess for dilatation of the large bowel and pneumoperitoneum. Contrast enema is indicated in certain cases only; however, CT of the abdomen is more sensitive and helps plan preoperatively.

Management

Acute LBO is a challenging surgical emergency. The patient is likely to be dehydrated; the bowel is unprepared, edematous, and may have a compromised blood supply. These factors increase the risk of postoperative complications, including anastomotic leaks[73] or stoma complications.[74]

LBO requires early identification to allow timely intervention in order to reduce the incidence of postoperative morbidity and mortality. In this section, we review the initial management of LBO, followed by cause-specific management. Since there are many causes of LBO, we will deal with the 3 most common causes: colorectal cancer, diverticular strictures, and volvulus.[75]

Initial management

Regardless of cause, the principles of initial management of LBO are the same and often referred to as "drip and suck." Fluid resuscitation with correction of electrolyte abnormalities ("drip") is required to prevent hypovolemic shock from edema and fluid sequestration. This can be guided by carefully monitoring the urine output via a catheter and urometer bag, clinical assessment of fluid status, and blood test results. Patients are kept nil-by-mouth and a nasogastric tube can be inserted to decompress the bowel[58] ("suck") in an open loop system. Decompression

provides symptomatic relief by reducing the buildup of gas and bowel secretions, and reduces the risk of aspiration.[75] Empirical treatment with broad-spectrum antibiotics is important, especially if ischemia is suspected, which should cover gram-negative aerobes and gram-negative anaerobes. Referring to local hospital antibiotic policies for specific antibiotic choice is advised. After adequate resuscitation, the definitive treatment of LBO thereafter depends upon both the underlying pathology and the patient's clinical status.[75]

Malignant LBO

Nonsurgical management

Many patients with malignant LBO may not be fit for surgery, and in the emergency setting, surgical intervention is associated with a mortality rate of up to 10%. As such, there are nonsurgical options that should be considered.[75] These may be appropriate for patients with malignant LBO without peritonitis, or for patients with advanced disease, or for those who would be medically unfit for surgical excision.[66] However, patients who are acutely unwell, septic, or peritonitic will likely require emergency surgery.[58]

Alternatives to surgery include tumor debulking, endoscopic colonic decompression with decompression tubes, or self-expanding metallic stents (SEMS). Endoscopy forms a major role in the nonsurgical management of patients with LBO.

Endoscopic laser therapy has been shown to be effective in debulking colorectal cancers causing LBO, and is most suitable for nonsurgical candidates.[76] A study by Brunetaud and colleagues reviewed data from 272 patients treated at a French center for rectosigmoid cancer with endoscopic laser ablation. They reported a success rate of 85% for palliation of symptoms, and a complication rate of 2%, with functional improvement continuing for an average of 10 months postprocedure.[77] Alternative methods for endoscopic tumor ablation include argon plasma coagulation and snare polypectomy. However, their reported success is limited as documented by the Standards of Practice Committee of the American Society of Gastrointestinal Endoscopy (2010).[76]

Colonic decompression can be achieved with the temporary placement of a decompression tube or a SEMS, which can be used for both symptom control in the palliative setting or as a "bridge to surgery."[78] Colonic stenting has increasingly become a more attractive alternative to emergency laparotomy in patients with left-sided malignant LBO, as it has the ability to convert an emergency situation into a safer elective operation.[66] In 1 retrospective study that compared the outcomes of stent insertion used as a bridge to surgery with emergency laparotomy, it was found that SEMS reduced the rates of stoma creation and postoperative morbidity without compromising the prognosis of the interval operation.[79] One systematic review of the literature showed high levels of both technical success (92%) and clinical success (88%) of stent insertion; palliation was successfully achieved in 90% of the 336 cases analyzed, and 85% of the 262 insertions used as a bridge to surgery were successful.[78] The risks associated with stent insertion include perforation, stent migration, and repeat obstruction. These occurred at a rate of 4%, 10%, and 10%, respectively, with repeat obstruction occurring most commonly in the palliative group. The UK-based ACPGBI recommends SEMS as the most appropriate palliative treatment for patients with uncomplicated left-sided obstruction, and it provides a cost-effective alternative to resection or colostomy formation for patients with colorectal cancer.[66] Recent evidence from a multicenter randomized controlled trial found no difference in oncologic outcome for patients undergoing colonic stenting as a bridge to surgery compared to emergency surgery, and with lower rates of stoma formation associated with colonic stent use, this affirms the role of stenting as a bridge to surgery.[80]

Defunctioning colostomy or ileostomy in the context of LBO remains an important option for surgeons. When faced with a hostile abdomen, exteriorization of the bowel proximal to the obstruction provides a safe option to allow resumption of intestinal flow. Such surgery may prove definitive in some cases; in others, this may enable a patient to be stabilized and all for further investigations and diagnosis to take place.

Surgical management

Patients who are acutely unwell, septic, or peritonitic will likely require emergency surgery.[58] There is significant debate about the optimum surgical management of MBO. As such, the ACPGBI and Royal College of Surgeons have published position statements and guidelines recommending different surgical options for patients who would benefit from surgery.

Resection and primary anastomosis has long been considered the surgical treatment of choice for right-sided tumors because it offers the patient a definitive procedure with no need for stoma formation or further surgery.[63] For left-sided tumors, a staged procedure consisting of defunctioning colostomy followed by resection with anastomosis and stoma closure has typically been employed.[66] However, this view has been challenged over the last 30 years. Several studies have compared outcomes for left-sided vs right-sided obstruction, all managed with resection and primary anastomosis. Results demonstrate that resection with primary anastomosis is as safe for left-sided obstruction as it is for right-sided obstruction, in the absence of contamination,[66,81,82] and in 1 study, the mortality rate following resection and primary anastomosis was lower in left-sided obstruction than right.[82] Indeed, the ACPGBI now recommends resection with primary anastomosis for uncomplicated left-sided bowel obstruction.[66] However, it is a technically demanding procedure, with the risk of anastomotic leak,[83] which can lead to significant postoperative morbidity with longer hospital stay. Therefore, in patients who are hemodynamically unstable, or where there is gross contamination and peritonitis from perforated bowel, or bowel is nonviable with ischemia, there is still a role for primary resection with end colostomy (ie, a Hartmann's procedure). This is often considered the safest surgical procedure for high-risk patients with left-sided obstruction.[35] Since there is no risk of anastomotic leak and a lower mortality rate.[58] However, this procedure is not without its risks; morbidity from stomal complications and issues associated with quality of life are relatively common.[58] Furthermore, approximately one half of patients will be unable to have their colostomy reversed.[63]

The use of laparoscopy in the management of acute SBO is well documented. However, its use in the management of acute LBO is lacking. One study by Gash and colleagues looked at the outcome of laparoscopic surgery in acute LBO and found it to be safe with a low complication rate, reducing the length of hospital stay.[84] However, due to the lack of high quality evidence, its use is not currently widely recommended.

Diverticular strictures

Diverticular strictures causing LBO often necessitate surgical intervention.[75] Options for intervention are similar to those for MBO, with the choice of SEMS followed by an elective procedure, with or without defunctioning ileostomy or resection with primary anastomosis.[85] Weighard highlights currents trends in the surgical management of diverticulitis as: "maintaining intestinal continuity, using a laparoscopic approach, and controlling infection acutely to bridge patients to later one-stage procedures."[86]

The principles of surgical intervention are resection of the whole sigmoid colon and anastomosis with viable descending colon and the upper rectum. There is an increased risk of recurrence if the sigmoid colon is incompletely resected.[87] The importance of directly visualizing the large bowel lumen even in the presence of CT confirmed diverticular disease is demonstrated by Elmi and colleagues.[88] Of the 402 patients who underwent both CT and colonoscopy, 2.2% had a colonic cancer in the presence of diverticulosis.

Volvulus

Volvulus most commonly affects the sigmoid colon. After adequate resuscitation, optimum initial management of uncomplicated sigmoid volvulus is with endoscopic decompression.[89] The 2 most common methods for decompression include flexible sigmoidoscopy and colonoscopy. If endoscopic decompression is successful in detorting the bowel, and there are no signs of ischemia, it is possible to insert a rectal tube to prevent recurrence of the volvulus, and plan

for elective surgery thereafter. If endoscopic decompression fails, the visualized bowel looks ischemic, or the patient is septic, an emergency laparotomy is indicated, commonly leading to a Hartmann's procedure.[90]

Unusual causes of LBO

Having now discussed LBO as a whole, including the historical context, epidemiology, anatomy, clinical presentation, investigations, and management, we now move on to focus on the rarer causes documented in the literature.

Infection

Infection within the large bowel is not uncommon, however disease significant enough to cause obstruction is. This segment addresses bacterial, fungal, and helminthic induced LBO. Given the rarity of such causes, few reports exist let alone reviews or meta-analyses, thereby hindering significant epidemiologic analysis of each factor.

Bacteria

Infective causes of LBO are infrequently encountered, but those that are documented tend to relate to disease associated with granulomas. Actinomycosis is an unusual cause of LBO, and is as a result of granulomatous, slow growing, filamentous, gram-positive bacteria.[91] Before the 1970s, most cases resulted either spontaneously or from the surgical opening of the bowel (eg, perforated appendicitis/appendicectomy). After the 1970s gynecologic sources (eg, intrauterine contraceptive devices) are the more common cause.[92] Infection is typified by a progressive inflammatory response which is contiguous and insidious with multiple connecting abscesses and an aggressive desmoplastic process.[93] Reports relate to the associated use of intrauterine contraceptive devices with resultant infection often mimicking malignancy.[94-96] In these cases, extrinsic compression from the actinomyces infection resulted in LBO. Antonelli describes a diffuse infiltrative process involving the adnexa, uterus, and sigmoid colon being identified on laparotomy. Segmental resection with stapled anastomosis and a defunctioning transverse loop colostomy were performed. Metronidazole and ampicillin were commenced postoperatively.[94] The other cases also required significant surgery in view of raised inflammatory markers. Other cases relating to Actinomycosis and large bowel exist, with recurrent themes relating to the presence of intrauterine devices, extrinsic bowel compression (particularly at the sigmoid colon), and the requirement for significant surgery.[97-99]

Mycobacterium tuberculosis (TB) is often regarded as one of medicine's great imitators,[100] and has been reported as an underlying cause in cases of LBO. Abdominal TB can be divided into 4 types: tubercular lymphadenopathy, peritoneal TB, gastrointestinal TB, and visceral TB.[101] Although the ileocecal region and peritoneum are the most common areas for abdominal TB, the pathogen can affect any part of the gastrointestinal tract.[102] Segmental colonic TB is seen in 9.2% of all abdominal TB, while multifocal disease occurs in one-third of patients with abdominal TB.[102,103] The incidence is increased in immunocompromised and acquired immunodeficiency syndrome (AIDS) patients.[101] The mainstay of treatment relates to antibiotic therapy, and even patients with intestinal strictures respond well to this therapy.[104] Consequently, obstruction is a rare presentation with pain and bleeding as the more common symptoms. Demetriou and colleagues report a 46-year-old Somalian woman with systemic symptoms and vomiting. CT highlighted a 10-cm obstructing annular mass in the transverse colon with a concurrent mass in the cecum. An extended right hemicolectomy was performed, with histology demonstrating caseating, transmural granulomatous inflammation consistent with tuberculosis.[105] There are a number of other reports, and recurring themes relate to the utilization of endoscopy in attempting

to directly visualize/pass the site of obstruction, swift commencement of antituberculous medications, and surgery when endoscopic measures fail to alleviate symptoms.[106,107]

One report suggests *Borrelia burgdorferi* (Lyme disease) resulted in a prolonged episode of pseudo-obstruction, as a result of reversible autonomic neuropathy.[108] Conservative management with antibiotics resolved the symptoms. Similar Lyme disease cases relating to pseudo-obstruction secondary to autonomic dysfunction are published.[109,110] A case of Whipple's disease caused by the gram-positive bacterium, *Tropheryma whippelii* has been documented. This resulted in low intestinal obstruction and hemorrhage due to mesenteric retraction, attributable to enlarged intra-abdominal lymph nodes.[111]

Other granulomatous diseases such as *bartonella, cryptococcus,* leprosy, and rheumatic fever have had no reported cases of LBO. Cases of pseudomembranous colitis with associated colonic obstruction secondary to *Clostridium difficile* and *Staphylococcus aureus* have been reported.[112,113] In such cases, mucosal inflammation obstructs the lumen, however good response to antibiotic therapy is reported and limits the role of surgery. As evidenced, bacteria are capable of producing LBO. Both extrinsic and luminal pathophysiology is documented. Clinical diagnosis and management requires the utilization of endoscopy, microbiology, pathology, and both medical and surgical teams working together in these challenging cases.

Helminth

Helminths are encountered widely around the world, with the gastrointestinal system most commonly affected.[114] Obstruction may occur directly as a result of mechanical obstruction from a mass of worms, secondary to a lead point triggering volvulus or intussusception, or as a result of intraluminal granulomatous inflammation causing stenosis or occlusion.

The management will depend on the severity of obstruction, response to conservative measures, and antihelmintic medication. Uysal highlights a variety of helminths implicated in gastrointestinal disease requiring either endoscopic or surgical intervention (majority do not relate to LBO). These include *Ascaris lumbricoides*, *Anisakiasis*, *Strongyloides stercoralis*, *Taenia saginata, Fasciola hepatica, Enterobius vermicularis, Necator americanus, Ancylostoma duodenale*, *Trichuris trichiura*, and *Wuchereria bancrofti*.[114]

Schistosomiasis is an acute on chronic parasitic disease caused by the trematode worm and is prevalent in tropical and subtropical countries. There are 5 main *Schistosoma* species that infect the intestinal and urogenital tracts, *Schistosoma*: *mansoni, japonicum, mekongi*, *guineensis*, and *haematobium*.[115] A number of cases are reported within the literature relating to *Schistosoma* induced LBO. Atik and colleagues report a case of perforation secondary to obstruction that required a left hemicolectomy.[116] Many cases of obstruction mimic colon cancer.[117] Bessa and colleagues highlight a cohort of 40 patients that required surgical intervention after medical management had failed, 3 of which had LBO.[118]

A. lumbricoides is 1 of the commonest colonizing worms found within the human alimentary tract and also 1 of the largest. Although reported more frequently as a cause of small bowel obstruction in childhood[119] particularly in underdeveloped countries, 1 case of adult LBO has been documented. Filling defects within the transverse colon on barium enema helped confirm the diagnosis. A course of mebendazole successfully treated the 73-year-old patient without the need for surgery.[120]

S. stercoralis, another tropical nematode commonly known as threadworm, has numerous documented cases of small bowel obstruction occurring as a result of infection[121,122] and particularly within children.[123] However, there are no reported incidences of LBO.

T. trichiura, also known as hook worm, is capable of causing LBO. With more than a quarter of the world's population thought to harbor the parasitic worm, this helminth is encountered particularly in tropical parts of Asia, Africa, and South America.[124] Bahon and colleagues report the case of an 84-year-old woman who developed a pseudotumor with proliferative surrounding inflammatory tissue secondary to heavy trichuris infection in France.[125] The resultant LBO required laparotomy and an ileocolectomy. Another case documents a large tangled mass

of worms that had caused obstruction and perforation to the ascending colon in a 25-year-old Puerto Rican man.[126] Similar findings have been recounted in a 33-year-old Japanese woman who also required an ileocecal resection and end-to-end anastomosis, after a 5-cm tumor in the proximal ascending colon was removed.[127] Histologic examination confirmed the presence of *trichuris* infection. Two cases of *trichuris* induced ileocolonic and colocolonic intussusception have also been reported.[128,129]

Karanakis and colleagues highlighted the bovine tapeworm *T. saginata* in a case of LBO secondary to volvulus from impacted worms.[130] A Hartmann's procedure was performed; however, on the second day postoperatively the patient died from respiratory complications.

Echinococcosis is caused by the tapeworms *Echinococcus granulosus* and *Echinococcus multilocularis,* which are harbored in the intestines of carnivores such as dogs and foxes. Where cystic echinococcosis is endemic, incidence rates in humans can exceed 50 per 100,000 persons; the prevalence levels are as high as 5%-10% in parts of Argentina, central Asia, China, East Africa, and Peru.[131] Restivo and colleagues document LBO secondary to the migration of a calcified 6-cm hydatid cyst (echinococcosis) via a hepatocolonic fistula in a similar mechanism to gallstone sigmoid ileus.[132] Endoscopic fragmentation proved successful in alleviating the obstruction. Another report relates to a 17-year-old man who attended with 5 days of abdominal pain. Ultrasound revealed dilated large bowel loops and an elongated, linear, hypoechoic tubular structure with well-defined echogenic walls in the transverse colon, which was actively moving. Treatment with albendazole resulted in resolution of his symptoms and a large dead tapeworm was passed 5 days later.[133]

Anisakiasis relates to the infestation of the marine nematode *Anisakis*. The parasite, although widely found, is most commonly diagnosed in Japan and the Netherlands.[134] This is somewhat unsurprising given that it is found in raw and smoked fish, both of which are popular in these countries. A 25-year-old woman in California presented with abdominal pain, vomiting, and abdominal distension having eaten ceviche 2 days earlier and sashimi 3 weeks prior to that. CT revealed a heterogeneous mass in the mesenteric side of the ascending colon and dilated small bowel loops. Exploratory laparotomy revealed a 3-cm mass and confirmed obstruction; a subsequent ileocolectomy was performed. Serology for immunoglobulin E specific to anisakiasis was highly positive, confirming the diagnosis.[135]

Helminth induced LBO is seldom seen. However, where endemic, the differential of LBO secondary to worm pathology should be considered. The management appears tailored to each center's expertise. As evidenced by the cases highlighted, not every patient can be managed conservatively. Endoscopy and surgery have important roles both diagnostically and therapeutically.

Fungus

Fungal infections of the bowel are well documented and tend to occur in the immunocompromised patient.[136] The most common fungi responsible for gastrointestinal infection are *Candida* and *Aspergillus*, although neither has been documented to cause LBO. However, cases of basidiobolomycosis, histoplasmosis, and cryptococcosis are reported (Table 2).

Nemenqani and colleagues present a 3-patient case series from Saudi Arabia relating to basidiobolomycosis, caused by the fungus *Basidiobolus ranarum*.[137] The authors describe that all patients presented with colonic masses initially felt to be malignancy. Biopsies demonstrated inflammatory tissue and granulomas in association with fungal hyphae. Two patients were managed medically; however, 1 required a right hemicolectomy due to obstruction with impending perforation. A similar case of basidiobolomycosis masquerading as colon cancer was successfully treated with oral itraconazole in Arizona.[138] Additional adult cases include a 24-year-old woman whose LBO resolved spontaneously with voriconazole and prednisolone[139] and another 24-year-old woman who was treated with itraconozole, similarly not requiring surgery.[140]

Histoplasmosis masquerading as a colonic tumor with resulting LBO has been reported in an immunodeficient patient.[141] A similar case in a non immunocompromised 52-year-old woman is reported from Mexico in an area endemic for histoplasmosis.[142] A fungating near obstructing

Table 2
Cases of helminth-induced large bowel obstruction.

Age (years)	Sex (M/F)	Location	Pathogen	Immuno compromised (Y or N)	Site of mass	Management
27	F	South Korea	Cryptococcosis	Y	Ascending colon	Amphotericin B No surgery
27	M	Thailand	Histoplasmosis	Y	Colon (exact site not stated)	Amphotericin B Surgery (not stated)
52	F	Mexico	Histoplasmosis	N	Transverse colon	Subtotal colectomy with ileosigmoid anastomosis. Amphotericin B then oral itraconazole.
77	M	Saudi Arabia	Basidiobolomycosis	N	Cecum/ ascending colon	Extended right hemicolectomy Oral itraconazole
19	F	Saudi Arabia	Basidiobolomycosis	N	Ileocecal junction to hepatic flexure	Laparotomy—not amenable to resection, biopsied omentum, and mass. Amphotericin B then oral itraconazole
20	M	Saudi Arabia	Basidiobolomycosis	N	Hepatic flexure	Voriconazole
56	M	Arizona, USA	Basidiobolomycosis	N	Recto sigmoid and cecum	Oral itraconazole
24	F	Saudi Arabia	Basidiobolomycosis	Y	Descending and sigmoid colon	Voriconazole and prednisolone
24	F	India	Basidiobolomycosis	N	Rectum	Oral itraconazole

F, female; M, male.

mass in the transverse colon was identified on colonoscopy. Owing to concerns of malignancy and the obstructing nature of the mass, surgical resection was undertaken and subsequent systemic antifungal therapy commenced. Cases of ulceration within the large bowel have been reported,[143,144] while other cases of gastrointestinal histoplasmosis induced obstruction relate to the small bowel.

A 27-year-old immunocompetent Korean woman presented with intermittent melena. CT and subsequent colonoscopy confirmed an ulcerating fungating mass in the ascending colon. Biopsies confirmed colonic cryptococcosis. Systemic intravenous amphotericin B for 4 weeks and a further 6 weeks orally negated the need for surgery.[145]

Mycosis induced LBO is extremely rare. When obstruction is encountered, particularly in the immunocompromised patient this cause should be considered. As with other presentations of LBO, appropriate evaluation and exclusion of more common causes is required. Commencement of systemic antifungals appears a mainstay of treatment but does not replace the necessity of surgery in certain cases.

Foreign body

Luminal obstruction of the large bowel secondary to a foreign body has been well documented. The nature of such objects is varied and can range from corporeal matter including hair and gallstones, to organic material and synthetic man-made articles.

Occlusion of the lumen can occur as a result of intraluminal blockage, from ingesting or inserting an object per rectum, or from extrinsic compression. Khanna and Khanna describe LBO secondary to an intravaginal foreign body compressing the rectum, later found to be a brick measuring 15 × 12 × 12 cm.[146]

The majority of foreign bodies ingested or inserted do not occlude the lumen of the large bowel. This is due to the wide diameter and the fact that objects will have already traversed the stomach, small bowel, and ileocecal junction if ingested. Therefore, for obstruction to occur, bowel luminal narrowing, foreign body stasis, and associated inflammation are all causative factors.

A variety of objects have been reported in the literature including fruit/vegetables,[147] sex toys,[148] light bulbs,[149] and bottles.[150] Where conservative methods fail, endoscopic, transanal extraction, and surgery all have roles in the management of these patients.[151]

Bezoar

Bezoars are solid masses of indigestible material within the gastrointestinal system. They account for between 0.4% and 4% of all gastrointestinal obstructions, with the commonest sites being the stomach or small intestine.[152] Various types of bezoar exist: phyto, lacto, pharmaco, tricho, and food boluses. Conservative approaches such as the use of enemas and manual disimpaction is considered in uncomplicated cases, while endoscopy and surgery are indicated when these measures fail or in the emergency setting.[153]

Phytobezoars are the commonest type of bezoar and relate to the accumulation of undigested plant and fibrous material. Cases causing LBO are well documented, with treatment modalities varying according to the clinical scenario. One reported case required a laparotomy for distal sigmoid obstruction.[153] Yoon and colleagues highlight successful endoscopic resolution for similar obstructing phytobezoars.[154,155]

Seed-based bezoars have also been reported. A sesame seed bezoar in a 79-year-old man, with a pre-existing benign stricture, required laparotomy and bowel resection.[156] Similarly, an underlying stricture required dilatation in a case of LBO secondary to Box Mrytle seed bezoar in India.[157] Other documented obstructing large bowel seed bezoars relate to wild banana,[158] jaboticaba,[159] poppy,[160] pumpkin,[161] watermelon (child),[162] sunflower (child),[163] and pomegranate.[164] Single seed or “stone” obstructions of the large bowel have been reported. A peach stone impacted within a malignant adenocarcinoma stenosis of the sigmoid is reported by de Matos Filho.[165] Exploratory laparotomy was required, with a significant portion of the large bowel from cecum to sigmoid excised after it was found to be distended and necrotic as a result of the malignant bezoar obstruction.

Other large bowel phytobezoars causing obstruction relate to popcorn kernels,[166] gummy bears (child),[167] and cloth fibers.[168]

Trichobezoars relate to hair occluding a lumen and have only been documented to cause LBO in 1 case.[169] The majority of trichobezoar presentations relate to the stomach and small bowel as a result of trichophagia. Rapunzel syndrome is a recognized condition that describes a gastric trichobezoar with a tail extending into the small bowel that can cause obstruction.[170] Lactobezoar or “milk curd obstruction” has seldom been seen, with the majority relating to gastric obstruction in premature infants.[171] However, Frietas does highlight a case of lower intestinal obstruction in a child secondary to lactobezoar at the level of the transverse colon.[172] Conservative measures such as lavage and enteral feeding appear to be valuable adjuncts when managing such a patient.

Pharmacobezoars occur when capsules, pills, or husks aggregate within the lumen. Yeliseeti and colleagues (2017) report an 81-year-old man with multiple co-morbidities who was found to be obstructing at the rectosigmoid junction on CT. Endoscopic resolution proved successful in removing the clump of pills.[173] Another case relates to psyllium seed husks used as a bulk forming laxative by a 23-year-old woman obstructing the right colon.[174] Magnesium oxide pills causing rectal obstruction in a 75-year-old woman necessitated a Hartmann's procedure before subsequent enterotomy.[175] Cholestyramine has been implicated in a case of mid transverse colon obstruction.[176] Other medications are reported in causing bezoars, but without luminal obstruction.[177]

Authors report foreign bodies inserted per rectum as a cause of LBO. If conservative measures are unsuccessful then surgical intervention is required.[178] Surinder and Sharma highlight 2 cases where laparotomy was required after a plastic bottle and a broom handle had become impacted within the sigmoid colon, respectively.[179]

Acute malignant obstruction is reported secondary to an impacted chicken bone, which was successfully treated with endoscopic removal.[180] A similar case related to beef bones.[181] Another report highlighted a malignant stricture in the left colon that was complicated by the swallowing of dentures resulting in LBO.[182] Similarly the ingestion of a dental crown proved significant to cause obstruction at the splenic flexure in another patient.[183] A separate case from Turkey relates to the ingestion of dentures without malignant stricture that resulted in perforation of the sigmoid colon.[184]

Drug packing

Drug or body packing is a widely reported cause of LBO, first described by Deitel and Syed.[185] This practice relates to the internal concealment of illicit drugs, most commonly cocaine or heroin. Although a finding more frequently relating to the stomach and small bowel, large bowel packing can carry significant morbidity.[186] Ischemia, perforation, and drug toxicity (body packer syndrome) make this a precarious condition.[187]

Gallstone

Gallstone ileus is a well-documented condition relating to a cholecystoenteric fistula enabling a gallstone to obstruct the lumen of the small bowel. LBO secondary to a gallstone has been reported, with nearly 40 reported "gallstone sigmoid ileus" or "gallstone coleus" cases since 2000.[188] Two mechanisms for gallstone sigmoid ileus are possible: cholecystocolonic fistula and, less commonly, a stone traversing the ileocecal junction via a cholecystoenteric fistula. The condition appears to have preponderance toward women and in patients older than 60 years of age.[188] The size of stones impacting varies (2.3-7 cm in maximum diameter) and it is postulated that 3 factors are required for gallstone sigmoid ileus to occur: cholelithiasis causing a cholecystoenteric fistula; a gallstone large enough to obstruct the bowel lumen; and narrowing of the bowel.[189] Differing management modalities are available including conservative, endoscopic, lithotripsy, and surgery. In a review conducted by Farkas and colleagues 74% of patients ultimately required some form of surgical intervention.[188] The study concluded that endoscopy and lithotripsy offer practical first line strategies, although surgical intervention should not be delayed if nonoperative measures fail or in an emergency.

After ingestion of foreign objects, bezoars, and gallstones, the majority will traverse the gastrointestinal tract without issue. Those that obstruct require management tailored to the patient and center. Conservative and endoscopic measures are used as first line strategies, where possible. However, in the emergency setting or when other modalities have failed, surgery remains a valuable option.

Trauma

Trauma to abdominal viscera rarely causes LBO. Hematoma, perforation, and peritonitis represent the common sequelae of penetrating injuries. For LBO to arise, stenosis or occlusion of the lumen is required. Colonic injury following blunt abdominal trauma is unusual, with an incidence of 0.3%.[190] Injuries to the transverse and sigmoid colon are more common given their anterior placement.[191] LBO may occur as a result of different mechanisms: compression between the abdominal wall and vertebrae/intra-abdominal organs; deceleration injury; laceration; or devascularization to the bowel or mesentery.

A delayed presentation of transverse colon stenosis causing LBO has been reported secondary to blunt abdominal trauma. Adem and colleagues reported the case of a 20-year-old man who suffered a work related accident in Turkey.[192] A heavy weight fell on the patient's abdomen 20 days prior to presenting with colicky abdominal pain and constipation. Stenosis within the distal transverse colon was confirmed on CT and colonoscopy. Endoscopic balloon dilatation acted as a temporary measure with a laparoscopic transverse colectomy and anastomosis being performed 3 months later. Other cases of post traumatic colonic strictures are documented secondary to seatbelt injury.[193] In this instance, the driver attended the emergency department with abdominal pain and vomiting 1 day after an accident while driving an excavator. Colonoscopy confirmed a tight stricture in the transverse colon. Although biopsies did not confirm neoplasia, this diagnosis could not be excluded. Consequently, a laparoscopic-assisted transverse colectomy was performed.

Obstruction secondary to intramural hematoma is well documented within the literature, with 2 mechanisms suggested. One describes chronic stenosis as a result of scarring from hemorrhage and blood supply interruption.[194,195] The other describes acute obstruction secondary to the hematoma itself.[196-198] Balaguera and colleagues highlight that 70% of the 40 or so reported cases of acute colonic intestinal hematoma are secondary to blunt trauma.[199] Other causes include anticoagulation, bleeding disorders, and endometriosis.

Traumatic injury to the diaphragm predisposes to herniation. Recorded cases relate to large bowel herniation causing obstruction following such injury.[200,201] In the case series recorded by Cruz, 4 adults had surgical confirmed defects in the left hemidiaphragm causing LBO following stab injuries. Bhatti and colleagues report the case of a 28-year-old man who, 9 months after a stab wound to the left chest, required laparotomy for LBO. He was found to have perforations in the cecum and transverse colon with a nonviable loop of transverse colon stuck in a 4×4 cm left diaphragmatic hernia. Closure of the hernia with a nonabsorbable suture, resection of the necrotic bowel, sutured repair of the perforations, and a loop ileostomy were performed. The majority of bowel containing diaphragmatic defects relate to the left side.[202]

Other trauma-induced cases relate to abdominal wall hernia.[203,204] Belgers and colleagues highlight 2 cases relating to delayed presentations. In 1 case, a 44-year-old man presented to the emergency department with swelling in the left flank, which on CT revealed a 5×9 cm herniation of the colon over the iliac crest. Five years before he had fallen from a height of 3 m and was buried under a collapsed wall. A mesh repair of the defect was performed and the patient made a full recovery. The second concerned a 64-year-old man who had suffered a 15-cm laceration and penetrating wound from a bull's horn 4 months prior. Local exploration and mesh repair resolved his symptoms.[203] Mahajna documents a 55-year-old man involved in a road traffic accident. CT confirmed a distal aortic tear and abdominal wall rupture in the right upper quadrant. Thoracotomy was initially performed successfully, however the patient subsequently developed LBO a day later. A right hemicolectomy was required after the hepatic flexure was found strangulated in a 7-cm abdominal wall defect.[204]

Kumar conducted a literature review pertaining to more than 50 cases of traumatic abdominal hernia. Seventy-two percent of patients presented immediately, with 35% of patients suffering handlebar injuries and 25% with crush injuries. The majority of these patients undergo operation, with 66% straightaway and 24% after a period of conservative management. The majority require laparotomy with either primary suture or mesh repair of the defects, however there are

cases of laparoscopic repair.[205] Immediate or delayed presentation with symptoms indicative of LBO following traumatic abdominal injuries should raise the possibility of this diagnosis. Surgery in the form of laparotomy when compared to the laparoscopic approach appears to be the favored management of clinicians.

Pancreatitis is a recognized cause of LBO. A patient with trauma-induced pancreatitis following a motorcycle accident resulting in LBO has been reported by Bolam.[206] Temporizing loop colostomy was performed prior to subsequent resolution of bowel continuity 2 months later.

Obstruction in burn patients is extremely rare. Cirrode highlights 3 cases, with total burn surface areas ranging from 20% to 40% that suffered sigmoid perforations requiring surgery.[207] Cecal perforation is also documented within the literature.[208] It is suggested that burn patients are prone to develop severe intestinal complications as a result of decreased splanchnic circulation.[207,209]

Inflammatory/autoimmune LBO

Diseases capable of forming adhesions or scarring internally are theoretically able to obstruct the large bowel. Management options vary from conservative to endoscopic to surgical. From an autoimmune viewpoint, IBD represents an obvious potential etiologic cause, however other inflammatory causes do exist.

Endometriosis is a common documented source of nonmalignant intra-abdominal adhesions. An estimated 8%–15% of women have the condition, but this accounts for less than 1% of all cases of bowel obstruction.[210] In a couple of large studies of patients with endometriosis, obstruction occurred in between 0.1% and 0.7% of cases.[211,212] The condition relates to functioning endometrial tissue found outside of the uterus. Intestinal endometriosis often manifests as a submucosal tumor or luminal stenosis, because it mainly involves the muscularis propria and subserosa or mesentery.[210] We have highlighted some cases of LBO secondary to endometriosis and their subsequent management. A 35-year-old woman with grade 4 endometriosis was found obstructed at the rectosigmoid junction. Endoscopic stenting proved successful in decompressing the patient. Four months later an elective recto-sigmoid resection and bilateral salpingoophorectomy and hysterectomy were successfully performed.[213] Another case from the United States highlighted the utilization of a stapled closure of a perforated cecum and diverting transverse loop colostomy in a case of endometriosis induced sigmoid colon obstruction.[214] Endometriosis induced LBO and perforation complicated by ventriculoperitoneal shunt was reported where the patient developed meningitis due to intra-abdominal fecal contamination.[215] Other cases relating to LBO secondary to endometriosis highlight neoplasm as the main differential.[216] This is not unexpected given that the rectosigmoid is the most common site of intestinal endometriosis. This explains why presentation is often with bleeding per rectum, diarrhea or constipation, colonic mass and/or bowel obstruction resembling a colorectal neoplasm.[217]

Distal intestinal obstructive syndrome (DIOS) relates to partial or complete intestinal blockage and occurs in up to 15.9% of cystic fibrosis (CF) patients.[218] DIOS is characterized by abnormally viscid mucofeculent material in the terminal ileum and ascending colon. The majority of such cases relate to the small bowel. However, Nassenstein and colleagues highlight a LBO case of DIOS in a 34-year-old man with end stage CF who required laparotomy.[219] Following lung transplantation the patient developed an acute abdomen. Contrast CT demonstrated massive dilatation of the small bowel and proximal colon. A homogenous mass in the proximal ascending colon with no evidence of extrinsic compression was noted. At laparotomy, the diagnosis of DIOS was confirmed.

We have touched upon trauma-induced LBO secondary to pancreatitis previously. Colonic complications of pancreatitis are well documented which include, adhesions, fistula, and hemorrhage, although obstruction is infrequent. LBO secondary to pancreatitis most commonly relates to the transverse colon and splenic flexure where a severely inflamed pancreatic body and tail can cause subsequent pressure necrosis. Another pathophysiological hypothesis put forward by Hunt and colleagues suggests that mesenteric ischemia associated with a severe attack of pan-

creatitis may cause fibrosis and stricture formation.[220] Yoo and colleagues highlight the case of a 43-year-old man who was initially successfully managed conservatively, however subsequently required a left hemicolectomy due to psuedocysts in the tail of the pancreas causing concomitant wall thickening and narrowing of the proximal descending colon.[221] Pascual and colleagues highlight the difficulty in recognizing LBO as many mimic malignancy,[222] and associated morbidity is evidenced by Chung and colleagues.[223]

Encapsulating peritoneal sclerosis is a serious complication of continuous ambulatory peritoneal dialysis. The condition relates to the fibrosis and thickening of the peritoneal membrane that forms an encapsulating cocoon around the abdominal contents.[224] One of the most serious complications relates to bowel obstruction. The majority of cases described relate to small bowel obstruction, however Choi and colleagues highlight a case of LBO.[225] Reported surgical options for LBO caused by encapsulating peritoneal sclerosis include adhesiolysis (with membrane excision), resection, and anastomosis.[226]

Gastrointestinal manifestations of systemic autoimmune disease are recognized in the literature and vary widely. Infarction, ulceration, perforation, and hemorrhage are well documented,[227] however bowel obstruction and, in particular, colonic obstruction are rare. Vaglio reports an atypical case of Churg-Strauss syndrome with associated LBO. A stenosing “apple core lesion” in the ascending colon was revealed by barium enema in a 60-year-old woman who presented with low-grade pyrexia and abdominal pain. Elevated serum IgE and positive antinuclear antibodies were noted. A laparotomy was undertaken with resection of the terminal ileum, cecum, and ascending colon. Histology demonstrated an eosinophil rich inflammatory infiltrate with necrotizing small vessel vasculitis and eosinophilic granulomas, all characteristic of Churg-Strauss syndrome.[228]

Behcet’s disease is a rare condition in the western hemisphere affecting less than 1 per 100,000 in the UK, but is more common in the Middle East, with a prevalence of 20-420 per 100,000.[229] This chronic multisystem vasculitic disease is relapsing and remitting in nature and classically associated with orogenitial ulcers, cutaneous lesions, and ocular and articular involvement.[230] Although gastrointestinal features are encountered, obstruction is extremely rare. A case from Malaysia highlights bowel obstruction in the context of Behcet’s disease.[231] A 35-year-old woman with the systemic features of Behcet’s disease presented with abdominal pain and distension. An abdominal radiograph demonstrated multiple dilated loops of small bowel and an emergency laparotomy and right hemicolectomy with primary stapled anastomosis is undertaken. Intraoperatively, a constricting stricture was noted at 10 cm from the ileocecal junction as well as multiple deep serosal punctum extending into mucosal ulcers along the length of the ascending colon. Another case required a subtotal colectomy for multiple stenotic lesions that were histopathologically confirmed as Behcet’s disease.[232]

A case of giant cell phlebitis caused an ischemic stricture of the large intestine in a 16-year-old girl resulting in LBO.[233] It is supposed that such vasculitic conditions cut off the blood supply to a segment of bowel resulting in ischemia and subsequent stricturing.

Crohn’s disease and ulcerative colitis (UC) make up the 2 primary conditions that clinicians refer to as IBD. UC affects only the colonic mucosa, whereas Crohn’s disease has full thickness involvement of the bowel wall. This factor means that deep ulceration, fistula formation, and stricturing are all potential sequelae of Crohn’s disease, while UC has a greater risk of malignant transformation.

Cases of LBO are reported in the literature relating to UC. Four patients from a study of 644 UC patients in Boston between 1969 and 1979 had LBO as a result of strictures or pseudopolyps.[234] Zeki and colleagues highlight a 19-year-old woman who developed subacute LBO secondary to a giant inflammatory polyp at the splenic flexure.[235] An extended right hemicolectomy with primary anastomosis proved successful in definitively managing the patient. Similar cases relating to large pseudopolyps and UC causing LBO have been reported.[236-238] Gumaste highlights a 5% risk (70 of 1156 patients) of UC patients developing strictures, of which 53 were benign.[239] Right-sided strictures appear more likely to be malignant. Obstructive symptoms and stricture formation after 20 years of known UC also appear factorial in malignant disease.

Pathogenesis of stricture formation in UC patients is uncertain, however fibrosis formation, contraction, and hypertrophy of the muscularis mucosa are both suggested mechanisms.[240] Ignjatovic highlights varying management options for UC strictures that include endoscopic balloon dilatation and surgery. No medical management is currently available.[241]

The prevalence of Crohn's related large bowel stenosis ranges from 7% to 15%, and from 20% to 40% in patients with small bowel disease.[242] Similarly to UC, the pathophysiology of intestinal strictures is unclear, but it is theorized that strictures develop from prolonged inflammation and fibrosis leading to luminal narrowing.[243] Given that the incidence of Crohn's disease ranges from 3.1 to 14.6 per 100,000[244] and that up to 90% of patients require surgical intervention during their lifetime,[245] it is unsurprising that LBO is a well-documented complication.[41]

Unlike tuberculosis, sarcoidosis causes noncaseating granulomas. A case of 2 synchronous obstructing lesions at the rectum and splenic flexure secondary to sarcoid was treated conservatively with prednisolone.[246] A separate report highlighted the need for laparotomy and right hemicolectomy to treat an ascending colon lesion pathologically shown to be sarcoid.[247]

Appendicitis and associated mucocele formation may present as LBO. Opreanu states that dense adhesions from the inflamed appendix tip containing the mucocele caused stricturing to the ascending colon.[248] Bowel was resected proximal to the adhesive band to the ileum and a standard anastomosis was carried out.

Neoplastic, intraluminal, extrinsic compression LBO

Adenocarcinoma of the colon accounts for 50%-60% of LBO cases, however other malignancies and benign pathologies make up a small number of cases. MBO is estimated to occur in 10%-28% of colorectal cancers and 5.5%-42% of ovarian malignancies.[249] Stomach (LBO in 6%-19%), pancreas (LBO in 6%-13%), bladder (LBO in 3%-10%), and endometrium (LBO in 3%-11%) have all been described, with breast cancer and melanoma reported less frequently.[250] Much like other conditions, the same pathophysiology applies to this subgroup, whether causing intraluminal obstruction, intramural obstruction, or extrinsic compression.

Carcinoma of the pancreas is reported as a cause of MBO. A 68-year-old man with weight loss, alternating constipation, and obstructive jaundice was felt initially to have an obstructive colon cancer. The patient's cecum perforated 5 days after admission and he subsequently died. Postmortem examination demonstrated a large carcinoma of the head of the pancreas that had completely invaded the transverse colon.[251] Other cases relating to carcinoma in the pancreatic tail[252,253] and metastatic pancreatic cancer have been reported.[254]

Yu and colleagues highlight a case of gastric adenocarcinoma in a 59-year-old woman with the first presentation being LBO. The patient had not opened her bowels for 15 days and reported some mild right epigastric pain. CT revealed a dilated, fluid filled colon with a thickened wall and adjacent fat infiltration with a tumor infiltrating the transverse colon. Upper gastrointestinal endoscopy highlighted a raised and thickened gastric mucosa in the antrum; biopsies confirmed poorly differentiated adenocarcinoma. A radical distal gastrectomy and right colectomy was successfully carried out.[255] A similar case of directly invading gastric cancer that responded to paclitaxel is documented by Fukuda and colleagues.[256]

Transitional cell carcinoma (TCC) of the bladder is a known cause of LBO. Rohloff and colleagues describe the case of a 49-year-old man who had undergone a radical cystoprostatectomy (for a T2, grade III bladder TCC) 1 year prior to presenting with LBO.[257] Having presented with abdominal distension and weight loss, a sigmoidoscopy was aborted, as the scope was unable to pass. Barium enema revealed an apple core stenosing lesion in the descending and sigmoid colon, which necessitated left hemicolectomy and end colostomy. Histology demonstrated TCC within the tumor. One other case relating to isolated bladder cancer metastasis causing intussusception within the transverse colon with subsequent LBO is recorded.[258] Following colonic resection and primary anastomosis in this case, histology highlighted a pedunculated sarcomatoid bladder carcinoma originating from the colonic mucosa.

Anorectal malignant melanoma accounts for 1% of all anal malignancies.[259] Obstructing tumors of the rectum are rare.[260]

Lymphoma accounts for less than 0.5% of colorectal malignancies.[261] Wong highlights male sex and increasing age as relevant factors. Hodgkin's B-cell lymphoma appears to be the most common histopathologic cause. Obstruction is reported with annular stricturing necessitating surgical excision.[262] Although the ileocecal region is the most common site for lymphoma of the colon, other positions such as splenic flexure and cecum are highlighted within the literature causing LBO.[263] Combined modality treatment is advocated by the Milan Cancer Institute, with systemic chemotherapy and surgery suggested as valuable adjuncts in gastrointestinal non-Hodgkin's lymphoma patients.[264] However, it should be noted that in this study no patients were acutely obstructed. In contrast, in the cases of intussusception at the ileocecal junction caused by mucosa associated lymphoid tissue (MALT)[265] and B-Cell non-Hodgkin's lymphoma,[266] emergency surgery (both right hemicolectomies) was indicated.

Kaposi sarcoma (KS) is a rare cancer that primarily affects elderly men of Eastern European and Mediterranean origin. There is also an association with immunosuppressed and transplant patients. Herpes simplex 8 virus plays a causative role in the disease of such patients. Commonly multiple purple/blue firm plaques develop on the hands and feet, which can subsequently spread to involve viscera or mucosa in 10% of patients.[267] A case of KS of the rectum in a 28-year-old African-American man with AIDS causing obstruction is reported. The patient presented with rectal pain, bleeding, and abdominal distension. A low CD4 count of 79/μL was attributed to poor compliance with highly active antiretroviral therapy. Colonoscopy could not proceed due to a large friable mass 2-cm proximal to the anal verge. Biopsies confirmed the diagnosis of KS. The patient was treated with radiotherapy.[268] No other cases of KS obstructing the large bowel have been reported.

Leiomyosarcomas are infrequently encountered and represent 0.1% of all colorectal malignancy.[269] These aggressive tumors arise from the smooth muscle cells within the intestinal wall and are more often seen in men. A case relating to the ascending colon of an 89-year-old woman is reported from Spain in which a 4.5-cm subocclusive tumor was unable to be managed conservatively necessitating an emergency right hemicolectomy.[270] Rao and colleagues highlight some of the few other cases of leiomyosarcoma induced LBO.[271]

Malignancies other than colorectal cancers represent an important differential when faced with LBO. As evidenced, the management varies somewhat depending on cause. However, histopathologic diagnosis is not always achievable in the acute setting when treating LBO. Consequently, surgery is readily employed to treat such patients. In those patients with subacute or partial obstruction, evaluation with radiological and endoscopic investigations is paramount since some malignancies may respond to chemotherapy, radiotherapy, or endoscopic management. Thus, surgery may not be needed.

Benign intraluminal causes

Giant inflammatory polyps are a recognized association of IBD. Some mucosal polyps are capable of occluding the bowel lumen and causing subsequent LBO. Marques and colleagues highlight such an instance in the case of a 23-year-old man with a background of UC.[272]

Other benign conditions such as colonic lipomas are reported as causative agents in LBO. The incidence of colonic lipomas ranges from 0.2% to 4.4% and may mimic other more sinister diseases when presenting.[273] Lipomas may cause obstruction themselves[274] or act as a lead point for colonic intussusception.[275] Approximately 90% of lipomas are found in the submucosa and are prevalent in up to 4.4% of the population.[273] Giant lipomas, classified as larger than 4 cm, are a relatively common cause of intussusception. Sarker highlights a 10-cm lipoma within the descending colon as a lead point for intussusception.[276] Agarwal and colleagues describe a left-sided mass with associated symptoms of obstruction which prompted surgery, and histopathologic examination subsequently confirmed it as a lipoma.[277]

Neurofibromatosis has a well-documented gastrointestinal component, with up to 25% of patients with type 1 disease developing gastrointestinal manifestations. A review of gastrointestinal obstruction in von Recklinghausen's disease highlights 25 patients between 1972 and 2013 of which 7 related to the large bowel.[278] "Tail gut cysts" or cystic hamartoma is reported as causing LBO in 2 cases.[279] Both cysts were in the pelvis, causing obstruction as a result of extrinsic compression. Such cysts are generally benign but rarely can show malignant transformation. Surgical intervention was required in both patients. A further case of a single hamartoma measuring 9.5-cm causing colonic obstruction within the transverse colon is described by Park and colleagues.[280]

Lymphangioma is an uncommon, benign bowel pathology pertaining to malformations of the lymphatic system and characterized by thin-walled cysts. The majority are found in children (90%) on the head or neck.[281] A case of intussusception secondary to lymphangioma is described[282] and Lepre and colleagues highlight the need for emergency surgery in a 73-year-old man due to anemia.[283] An ascending colon mass was noted on CT scan; however, due to hemodynamic instability an urgent laparotomy and right hemicolectomy was performed.

Hemangiomas have similarly been described to masquerade as malignancy causing mechanical bowel obstruction. Bowel hemangiomas are most commonly found in the small intestine, rectum, and sigmoid. Hemangiomas typically mainfest with rectal bleeding, however obstruction is reported in up to 20% of patients.[284] A case relating to LBO within the transverse colon requiring colectomy with end-to-end anastomosis in a 62-year-old man has also been reported.[285]

Extrinsic compression

A variety of conditions are capable of causing extrinsic compression to the large bowel, including foreign bodies, localized or disseminated malignancy, or benign conditions such as uterine fibroids. Many of these cases have been highlighted in other sections of this paper. Nevertheless, the diversity of causes highlights a range of interesting differentials for the clinician faced with an atypical case.

Symptomatic bladder calculi usually present with obstructed urinary flow. Nonetheless, LBO attributed to a large bladder calculus measuring 14 × 12.5 cm is reported in Malaysia. The stone was found to be compressing the rectum and an emergency vesicolithotomy was performed through a midline incision.[286] Equally unusual is the case of a stag-horn calculus silently perforating adjacent to large bowel, resulting in obstruction. This presentation related to a 63-year-old woman with a short history of abdominal distension, pain, and absolute constipation. CT revealed a 1.5 × 1.0 × 2.5 cm perforated extrarenal fragment of calculus positioned adjacent to the proximal descending colon. Diffuse inflammation resulted in eventual obstruction requiring a left hemicolectomy and left nephrectomy.[287]

LBO secondary to urinary retention of the bladder is recognized.[288-290] Mac Giobuin and colleagues highlight benign prostatic hypertrophy as another etiologic factor causing urinary retention and subsequent compression of the sigmoid against the sacrum.[291] Lissidinni reports a large primitive cystic structure originating from the seminal vesicles in an elderly man. At laparotomy this was found to compress both bladder and rectum and only diagnosed on subsequent histopathology.[292] A large, single, 27-cm right renal cyst proved capable of compressing the large bowel, which was successfully managed with ultrasound-guided drainage.[293] Polycystic kidneys are similarly reported to cause LBO, with 1 case necessitating nephrectomy due to cyst infection, hemorrhage, and luminal compression.[294] Another caused strangulation and necrosis after the mesentery had become overly stretched, resulting in occlusion of the mesenteric veins and necessitating a jejuno-transverse colostomy.[295]

Cases of LBO secondary to ovarian cystadenofibromas[296] and teratomas[297] are documented. Both cases failed to settle with conservative measures and required surgical intervention with resection of the respective masses causing compression.

Leiomyomas are benign smooth muscle tumors that are capable of causing MBO. Leiomyomas or fibroids are often encountered within the uterus. A large (11 × 9 × 7.5 cm) uterine fibroid

was found at laparotomy to be extrinsically compressing the large bowel at the rectosigmoid junction in a 44-year-old patient.[298] A case of sigmoid volvulus is described in a young woman who was found to have pedunculated uterine fibroids intraoperatively.[299]

Aneurysms may cause bowel obstruction by a variety of mechanisms. Ruptured aortic aneurysms with large retroperitoneal hematomas can extrinsically compress the bowel.[300,301] An unusual case of a ruptured left internal iliac artery aneurysm resulted in rectosigmoid compression and LBO. Decompression of the aneurysm and a femoral-femoral bypass were required to revasculavize the lower limb.[302]

Following operative intervention distal bowel ischemia and stricturing are recognized complications,[303] however cases of acute LBO are exceedingly rare. Lane and Bentley highlight 2 cases, 1 of which responded to dilatation, while the other required fashioning of an end colostomy.[304] Gunasekera and colleagues highlight a case of complete obstruction due to ischemia of the rectum following abdominal aortic surgery.[305]

Intestinal pseudo-obstruction in pregnancy is a recognized cause of LBO,[306] particularly in the third trimester. However, other cases are encountered: band adhesion from a previous appendicectomy complicated 1 pregnancy,[307] while both sigmoid[308] and cecal volvulus[309] are documented in pregnant women. A case of a 27-year-old woman with a double uterus who had given birth via Cesarean Section developed LBO secondary to the sigmoid colon becoming incarcerated between the 2 uterine bodies.[310] At laparotomy, adhesiolysis of the incarcerated sigmoid colon and amputation of the smaller uterus took place.

Iatrogenic causes of LBO

Although we have touched on cases previously in other sections (eg, following aortic aneurysm surgery), iatrogenic induced LBO should be considered, particularly in patients with significant surgical histories. Both surgery and investigations have the potential to cause LBO.

Postoperative adhesions

Although adhesions (fibrous bands between 2 or more anatomical structures) are an important and relatively common cause of small bowel obstruction, they are a rare cause of LBO, with few cases reported in the literature.[42,307,311-317] Adhesions can either be iatrogenic or congenital, with the formation of postoperative adhesions the most commonly encountered complication of abdominal and pelvic surgery.[318] Only a minority of the reported LBO cases caused by adhesions can be considered iatrogenic, with the remainder as a result of an anatomical remnant or other abnormality.[312] This can include an epiploic appendage becoming inflamed, adherent to the abdominal wall and subsequently constricting to the large bowel.[42] Other examples include remnants such as a vitelloumbilical cord or mesourachus.[42]

Iatrogenic adhesional LBO has only been reported following nephrectomy, renal transplant, appendicectomy, laparoscopic converted to open inguinal hernia, and laparoscopic cholecystectomy.[42,307,311,312] Sites of obstruction were in the sigmoid, mid-transverse colon, hepatic flexure, and cecum, respectively.[42,307,311,312]

Barium impaction

Inspissated barium following use in contrast gastrointestinal imaging is a rare but notable potential cause of LBO.[319] Kurer and colleagues performed a systematic review, which identified 32 reported cases from 1950 to 2006 of LBO secondary to impacted barium (barolith).[320] Impaction is a relatively late complication and can occur following any gastrointestinal barium investigation, although the greatest risk follows a barium meal due to the amount of barium used.[320]

Contributory factors include low residue diet, dehydration, poor colonic motility, electrolyte disturbance, and Ogilvie's syndrome.[320] Since 2006 there have only been 5 further cases of LBO in adults precipitated by a barolith described.[321-325] The interval from barium investigation to presentation with LBO varied from 2 days to 24 months, with 50% presenting at over 5 weeks from the initial investigation.[320] The left colon (16 cases) was the most common site of barolith impaction across all the cases documented in the literature.[320-323] Treatment varied across all the reported cases and included manual evacuation, enema, gastrograffin, lactulose, endoscopy, and laparotomy, with either colostomy, colectomy, or colostomy.[320-325]

Radiotherapy

Radiation induced injury to the gut occurs in approximately 5% of irradiated patients with intra-abdominal or pelvic malignancy.[326] In these cases, radiation colitis commonly results 6 months to 5 years postradiotherapy treatment and is most commonly located in the rectosigmoid region.[326-328] Diffuse fibrosis in the affected bowel results from radiation induced vascular damage with subsequent persistent local ischemia.[327] This causes diffuse fibrosis in the lamina propria and submucosa, and forms the basis for the complications associated with chronic radiation colitis: proctitis, hemorrhage, fistula, stricturing, obstruction, perforation, and cancer.[327] Chronic radiation colitis, apart from being a precancerous lesion, can lead to (typically partial) LBO that can often be managed conservatively with nasogastric suction and parenteral support.[327] Intestinal stricture formation is a commonly recognized complication of radiation therapy, and can exacerbate anastomotic stricturing following resection surgery. Surgical management of LBO following radiotherapy can be particularly challenging due to diffuse fibrosis and alterations in the micro and macroscopic structure of the intestine.[327] There is a significant increase in the risk of anastomotic leak if the tissues involved have been previously irradiated.[327]

Percutaneous endoscopic gastrostomy

LBO has been reported following insertion of percutaneous endoscopic gastrostomy (PEG) tubes.[329-331] One case highlights LBO that occurred following the tube being cut flush with the skin. The remaining inner part of the tube remained in the stomach, with the intention to be passed through the GIT without complication. Two weeks after insertion the patient required a laparotomy. Intraoperatively, an inflammatory tumor was identified around the remaining PEG material causing obstruction in the sigmoid colon.[329] Buluş and colleagues reported a case in which the PEG tube was pulling the anterior wall of the stomach and transverse colon against the abdominal wall resulting in subsequent colonic compression.[330]

Colonoscopy

A case of post colonoscopy LBO is reported. Diverticulosis and multiple sessile polyps in the sigmoid colon were highlighted and biopsied. Immediately after the endoscopic investigation, the 56-year-old man complained of colicky abdominal pain. Radiographs confirmed dilated loops of large bowel. Immediate repeat colonoscopy demonstrated an edematous sigmoid at the site of the previous biopsies, which the scope could barely pass through due to spasm and edema. Following the repeat colonoscopy, the patient's symptoms resolved.[332]

Congenital causes of LBO

There are a number of congenital conditions that can manifest with LBO. Meconium ileus is an early clinical manifestation of CF, which leads to LBO in the newborn.[333] Stagnant meconium

obstructs the ileum, leading to congestion, and has a soap bubble appearance on plain radiograph. Although the primary site of disease occurs in the ileum, colonic obstruction can ensue due to the development of a microcolon—bowel with a narrow lumen but of normal length.[334] This occurs as meconium does not reach the colon and therefore the growth stimulus is removed. Through the use of a diagnostic and therapeutic contrast enema, the meconium ileus is usually alleviated, and with this, the microcolon often returns to a normal caliber.

Small left colon syndrome, also known as meconium plug syndrome, can give rise to obstruction due to the inability of the colon to propel meconium through the intestinal tract.[335] The syndrome is associated with maternal diabetes and magnesium tocolysis and can be clinically difficult to distinguish from meconium ileus (small left colon syndrome has no association with CF). As with meconium ileus, it commonly resolves following administration of a contrast enema for diagnostic purposes.

Colonic atresia can also account for congenital LBO, most commonly due to a vascular insult on the mesentery.[336] Neonates with colonic atresia tend to present with bilious vomiting, abdominal distension, and failure to pass meconium. These patients have microcolon evident distal to the level of obstruction due to lack of stimulation and require emergency surgical intervention.

Colonic atresia is known to be associated with Hirschprung's disease. Hirschprung's disease occurs due to failure of development of the Meissner's and Auerbach's plexuses in the wall of the colon, leading to proximal obstruction. This gives rise to an aganglionic segment of bowel, with dilatation of proximal bowel loops. Diagnosis is achieved via a rectal biopsy. Management requires segmental resection of the affected bowel.

Malrotation, or failure of the midgut to rotate 270° around the superior mesenteric artery in utero, can lead to intestinal obstruction in the pediatric population (and more rarely in adults). An unusual cause of malrotation leading to midgut volvulus is reported in a 40-year-old woman who had 100 cm of small bowel, cecum, and ascending colon resected due to ischemia.[337] Such patients tend to present with subacute obstructive symptoms with multiple hospital attendances until a critical event occurs leading to irreversible ischemia of the affected bowel segment.

There have been cases reported where Ladd's bands have not only led to small bowel obstruction but also obstruction of the colon. Small bowel obstruction arising from malrotation is commonly associated with Ladd's bands—peritoneal bands, which in healthy individuals are attached to the cecum in the right lower quadrant. However, in malrotation, they appear along with the cecum in the right upper quadrant, traversing the duodenum and leading to small bowel obstruction. Raphaeli and colleagues describe colonic obstruction at the hepatic flexure in a previously healthy teenager secondary to a Ladd's band.[338] The patient was treated in the same manner as in small bowel obstruction, with division of the Ladd's band and a Ladd's procedure.

A limited number of cases have been described that attribute obstruction to congenital anomalous bands.[42] Lin and colleagues report the case of a 6 month old child with no history of previous surgery that had extraluminal obstruction secondary to a congenital mesocolic band compressing the proximal sigmoid.[317] Lysis of the band was achieved through laparotomy and the patient recovered well without further complication. A further case of a congenital band leading to obstruction was described in an 8-year-old child with trisomy 13 and intestinal malrotation, who had a segment of mesourachus bound by adhesions to the mesentery.[339] A mesourachus occurs when the vestigial urachus connects to the anterior abdominal wall by mesentery, a rare occurrence. In this case, the combination of mesourachus and intestinal malrotation led to severe obstruction of the descending colon.

Another rare cause is congenital solitary intestinal fibromatosis. Fibromatoses are of mesenchymal origin and appear histologically as spindle cell tumors that rarely affect the gastrointestinal tract. Numanoglu and colleagues describe a 6 day old neonate who presented with vomiting and abdominal distension.[340] At laparotomy, the stenosing intestinal fibromatosis had led to obstruction of the proximal transverse colon, which was treated successfully by segmental resection.

Obstruction due to stenosis has also been reported secondary to a Meckel's diverticulum.[341] A barium enema in a 17-year-old boy with no history of abdominal surgery showed extrinsic compression of the ascending colon. Laparoscopy was performed, demonstrating a raised and compressed region of colon due to a Meckel's diverticulum with a fibrous band attaching to the umbilicus.

Where congenital defects are concerned, bowel may herniate through 1 of 2 foramens in the diaphragm: the more anteriorly situated right-sided foramen of Morgagni and the posteriorly situated left-sided foramen of Bochdalek. Herniation of abdominal viscera through these congenital defects into the thorax may cause mild respiratory or gastrointestinal symptoms and is often discovered incidentally on chest radiograph. Presentation with bowel obstruction is relatively rare but well documented in the literature.[342]

Bochdalek hernias are rarely detected in the adult population. Ekanayake and colleagues report on a 28-year-old man who presented with bowel obstruction that was later attributed to an incarcerated hernia of Bochdalek containing ischemic transverse colon and splenic flexure.[343] The male patient underwent repair of the hernia, plus an extended right hemicolectomy with ileocolic anastomosis. Kugai and colleagues report a case of an elderly woman presenting with features of intermittent LBO secondary to a Bochdalek diaphragmatic hernia. The chest radiograph showed an elevated left hemidiaphragm and CT revealed large bowel loops in the left hemithorax. The herniated bowel was reduced into the abdominal cavity with excision of the hernia sac, requiring both transthoracic and transabdominal approaches.[344]

Two cases of LBO secondary to Bochdalek hernias have been reported in pregnancy. In 1 case, the female patient presented at 23 weeks gestation and underwent resection of the gangrenous portion of large bowel with primary anastomosis.[345]

Hernia of Morgagni represents 2%-3% of congenital diaphragmatic hernias—a defect in the anterior of the diaphragm due to incomplete fusion of the pleuroperitoneal membrane, which can lead to herniation of abdominal viscera into the thorax.[346] Hernias of Morgagni commonly manifest with vague epigastric fullness and pain, but rarely can lead to complete intestinal obstruction. McBride and colleagues describe LBO detected on CT secondary to an undiagnosed congenital diaphragmatic hernia of Morgagni.[342] The patient had a closed loop obstruction due to a competent ileocecal valve and required reduction of the hernia and an extended right hemicolectomy.

Overall, congenital causes of LBO are an infrequent finding, however they should certainly be considered in the symptomatic neonatal and pediatric population. In neonates, important differentials include meconium ileus, meconium plug syndrome, colonic atresia, Hirschprung's disease, and malrotation. Other less common causes reported in the literature in both the pediatric and adult patients include congenital anomalous bands, Ladd's bands, solitary intestinal fibromatosis, stenosis secondary to Meckel's diverticulum, and congenital diaphragmatic hernia.

Intussusception

Approximately 1%-5% of all cases of adult intestinal obstruction are caused by intussusception, although most occur within the small bowel (50%-80%).[347,348] It commonly occurs in areas acting as junctions between freely moving and fixed segments of the gastrointestinal tract. They can be classified accordingly: enteroenteric (small bowel only), colocolic (large bowel only), ileocolic (prolapse of terminal ileum into the ascending colon), or ileocecal (the ileocecal valve acts as the lead point).[347] Approximately 90% of cases of adult intussusception are secondary to an underlying pathologic lesion serving as a lead point in the bowel wall.[347] The etiology of intussusception can therefore be classified as benign, malignant, or idiopathic (Table 3).

Adults presenting with LBO secondary to intussusception tend to present with relatively nonspecific symptoms, differing from the classic pediatric presentation (triad of abdominal pain, hematochezia, and a palpable tender mass), including nausea, vomiting, bleeding per rectum, a change in bowel habit, constipation, and abdominal distension.[347] Abdominal CT is currently considered the radiologic investigation of choice to identify intussusception, with characteristic

Table 3
Benign and malignant causes of adult intussusception resulting in large bowel obstruction.

Benign	Malignant
Congenital • Bands[349] • Persistent ascending and descending mesocolons[350] • Colon duplication[351]	Primary tumor • Colorectal adenocarcinoma[352] • Carcinoid tumor[353] • Mixed adenoneuroendocrine carcinoma (MANEC)[354] • Ovarian fibrosarcoma[355] • Mucinous adenocarcinoma[356] • Primary colonic melanoma[357] • Leiomyosarcoma[358] • Fibrous histiocytoma[359] • Angiosarcoma[360] • Liposarcoma[361] • Diffuse large B cell lymphoma[362] • Burkitt's lymphoma[363] • Non-Hodgkin's lymphoma[364]
Benign tumor • Adenoma[365,366] • Peutz-Jeghers syndrome[367] • Lipoma[368,348] • Neurofibroma[369] • Lymphangioma[370] • Hemangioma[371] • Pseudopolyp[372] • Hamartoma[373,374]	
Iatrogenic • Colonoscopy[375]	Secondary tumor • Pleomorphic carcinoma[376] • Sarcomatoid bladder carcinoma[258] • Melanoma[377] • Esophageal adenocarcinoma[378]
Other • Scleroderma[379] • Intestinal endometriosis[380] • Pneumatosis intestinalis[381] • Eosinophilic colitis[382] • Arteriovenous malformation[383] • Hypertrophic Peyer's patches[384] • Lymphoid hyperplasia[385]	

features including a "sausage shaped mass" and mesenteric vessels within the bowel lumen.[347] Approximately 66% of large bowel intussusception cases are caused by a malignant lead point, with colorectal adenocarcinoma the most common cause.[386]

Management of intussusception causing LBO is broadly considered to warrant prompt laparotomy.[366] This is in contrast to the management of pediatric patients, where attempts at hydrostatic reduction would be considered first line, due to the risk of an underlying pathology in adults. There has been debate in the literature concerning whether to reduce an intussusception prior to resection, as it has been postulated that the reduction may result in dissemination of malignant cells, although there is no clear evidence.[366]

Hernia

Hernia is a common cause of small bowel obstruction[2] and the most common cause of strangulation in patients presenting with small bowel obstruction.[387] However, cases reporting hernia as a cause of LBO are relatively rare. Here, we review cases of LBO secondary to inguinal, diaphragmatic, lumbar, internal, and ventral hernias.

Inguinal hernia

Inguinal hernias are the most common type of hernia,[388] accounting for approximately 75% of hernias, with a lifetime risk of 27% in men, and 3% in women.[388] The contents most commonly found in inguinal hernias include the small bowel and omentum. Although inguinal hernias are a relatively common cause of small bowel obstruction, with the most serious complication being strangulation, it is a rare cause of LBO, with right-sided hernias in particular rarely containing large bowel.[389] There are several cases of LBO secondary to inguinal hernia documented in the literature, however a small proportion of these were complicated by colonic malignancy as the

more likely cause of the obstruction.[390] Thus careful interpretation of the radiographic evidence is imperative in patients presenting with inguinal hernia, to determine the cause of obstruction.

Bali and colleagues report a case of an elderly man presenting with LBO secondary to an irreducible right inguinal hernia. The abdominal radiograph showed dilated loops of large bowel and a CT revealed sigmoid colon in the hernia sac. At laparotomy, the sigmoid colon was found to be ischemic and was resected with formation of an end colostomy.[389]

Diaphragmatic hernia

As documented above, LBO can occur secondary to diaphragmatic hernia. Both congenital defects in the diaphragm and traumatic injury can result in LBO.

Lumbar hernia

Lumbar hernia is an uncommon type of abdominal wall hernia, and a rare cause of LBO, making it a challenging presentation to sort out. Lumbar hernia may protrude through 1 of 2 different defects in the posterior abdominal wall: the superior lumbar triangle of Grynfeltt-Lesshaft[391,392] or the inferior lumbar triangle of Petit,[393] which occurs less frequently than superior hernias.

There are 4 case reports documenting LBO secondary to lumbar hernias. Hide and colleagues report a case of an elderly woman presenting with LBO secondary to a lumbar hernia causing incarceration of the descending colon. She presented with absolute constipation, vomiting, and mild abdominal pain but nothing was found on palpation. An abdominal radiograph showed features in keeping with LBO, but it was a barium enema that showed herniation of the descending colon through a narrow orifice. Only at laparotomy was a hernia through the left inferior lumbar triangle found, which contained incarcerated bowel. The surgeons were able to reduce the bowel, which was found to be viable, and the hernial orifice was repaired.[394]

Internal hernia

The most common type of internal hernia is a paraduodenal hernia, although this rarely causes bowel obstruction, accounting for less than 5% of all cases.[395] The majority of case reports documenting bowel obstruction secondary to paraduodenal hernia contain small bowel,[396] and they are most commonly left-sided,[395] where bowel protrudes through the foramen of Landzert located posterior to the fourth part of the duodenum.[395]

Bhatti and colleagues describe a case of a young woman presenting with LBO secondary to a left paraduodenal hernia. The patient presented with features in keeping with LBO, but due to deterioration she underwent an emergency laparotomy before CT imaging was done. The cecum and ascending colon were found to be dilated and gangrenous in the hernial sac, necessitating resection with formation of a stoma.[396]

Ventral hernia

There are several cases in the literature reporting LBO secondary to Spigelian hernia. Spigelian hernias account for 1%-2% of all hernias and are a rare type of abdominal wall hernia. Approximately 20% of cases report incarceration, however this is most commonly pertaining to small bowel. Salemis and colleagues report a case of an obese young man presenting with LBO secondary to an incarcerated Spigelian hernia containing ascending colon. A high level of suspicion is required in such cases as more than one-third of patients may not have a palpable mass. Since the defect is often small, there is a high risk of strangulation, and surgical repair is imperative.[397]

Conclusion

This review both informs and educates readers with regard to the history, pathophysiology, and breadth of LBO presentations as well as subsequent management. Although many of the presentations highlighted are one of a kind, it is not unfathomable to think that one might encounter an unusual case of LBO in one's own career. Although the vast majority of obstructions relate to commonly seen conditions, it is important to consider the differential in each patient.

The purpose of this paper was not to give an exhaustive list of every individual case ever published, but to highlight the extent and diversity with which LBO may manifest. We have demonstrated the differing etiology behind LBO, however it is important to remember that the pathophysiology of this condition remains the same. Obstruction occurs via extrinsic compression, mural stricturing, or intraluminal occlusion and these mechanisms remain, regardless of cause.

Obstruction to the small bowel is encountered more commonly both clinically and in the published literature. When conducting the literature searches it is apparent that many of the presentations documented within our paper also affect the small bowel. This is to be expected given the relative mobility and smaller luminal diameter. However, this only succeeds in illustrating the rarity of LBO outside of those conditions frequently encountered.

Management strategies for these rare presentations of LBO vary but appear tailored to each individual case. Conservative, endoscopic, and surgical interventions all have important roles when managing patients with LBO. Although technological progress and improved patient safety have advanced patient care significantly over the past 100 years, LBO continues to have a high associated morbidity and mortality rate.

This review provides an up-to date insight into the infrequently seen presentations of this common condition. To our knowledge, no other in-depth review on this topic has been published. Although many factors have been touched on, new etiologies will likely present in the future and provide clinicians with fresh challenges when managing the patient with an unusual LBO. We hope that this review will serve as an aid-memoir to clinicians when faced with diagnostic uncertainty in such patients.

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