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### In Brief



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Many clinicians will have encountered patients with large bowel obstruction (LBO) on a ward or in the operating room and will appreciate the challenges posed by such presentations. Establishing the cause of an obstruction is vital given the high associated morbidity and mortality.

Although less common than small bowel obstruction, LBO (25% of all intestinal obstructions) poses more immediate risks, (eg, perforation and subsequent peritonitis). Common causes of LBO have been extensively documented within the published literature.

Here, we aim to describe less frequently encountered and understood etiologies that should be considered when more common pathologies have been excluded. We aim to assist clinicians tasked with investigating and managing their patients. We will consider a range of aspects relating to LBO before focusing on rare aetiologies.

Accounts of bowel obstruction go back several millennia: One of the first cases was described 3400 years before the Common Era (BCE) in the Ebers Papyrus from ancient Egypt. Accounts from the 16th and 17th centuries document the use of a stoma, and by the late 19th century exteriorisation of the bowel via ileostomy, cecostomy, or colostomy was often reported.

Surgery as we know it today owes much to the work of surgeons at the turn of the 20th century. Technological advancement throughout the 20th century has extended the knowledge base and the investigations, equipment, and management strategies available. Clinical diagnostics are more sensitive and surgery is safer and less invasive, resulting in the reduction in mortality in patients with LBO.

The large bowel is involved in approximately 25% of cases of acute bowel obstruction. Although LBO occurs less frequently than small bowel obstruction, it accounts for approximately 2% to 4% of emergency surgical admissions. Colorectal cancer is now the most common cause of LBO in developed countries.

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

The course of the large bowel extends from the terminal ileum to the anus. The large bowel derived from midgut structures is supplied by branches of the superior mesenteric artery. Branches of the inferior mesenteric artery supply the distal one-third of the transverse colon and descending colon. Drainage is primarily by the portal vein, which is formed by the confluence of the splenic vein and superior and inferior mesenteric veins and innervated by sympathetic nerves which follow the course of the superior and inferior mesenteric arteries.

LBO can occur anywhere along the course of the large bowel but is most common within the descending colon. 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 extra-luminal. In complicated obstruction the circulation to the bowel is compromised, resulting in ischaemia, necrosis, and perforation.

Absolute constipation (90%), abdominal pain (74%), and distension (65%) are the most frequently occurring signs and symptoms of LBO. The prognosis depends largely upon the cause of the obstruction. Morbidity rates across all causes of small bowel obstruction and LBO vary widely, from 6% to 47%, with mortality rates of 2% to 19%.

When LBO is suspected, laboratory tests followed by radiological imaging augment the clinical history and examination. Erect chest radiograph and plain abdominal films assess for dilatation of the large bowel and pneumoperitoneum. Contrast enema is indicated in certain cases only. CT of the abdomen is more sensitive.

Acute LBO is a challenging surgical emergency. Management centres on fluid resuscitation, insertion of a nasogastric tube, and prompt identification of the underlying cause. Conservative, endoscopic, and surgical management strategies all have a role to play. Management depends on many factors including the patient, nature, and severity of presentation, and the centre's expertise.

Several rarer causes of LBO have been described. Infective causes of LBO are infrequently encountered, but typically relate to granulomatous disease. Causes include *Actinomyces*, *Mycobacterium tuberculosis*, and *Borrelia burgdorferi*. Helminths are encountered widely globally, with the gastrointestinal system most commonly affected. Obstruction may occur directly as a result of mechanical obstruction by a mass of worms or secondary to a lead point triggering volvulus or intussusception, or as a result of intraluminal granulomatous inflammation causing stenosis or occlusion. Schistosomiasis, *Ascaris lumbricoides*, *Strongyloides stercoralis*, *Trichuris trichiura*, *Taenia saginata*, *Echinococcosis*, and *Anisakiasis* have been implicated in cases of LBO.

Fungal infections of the bowel are well-documented and tend to occur in the immunocompromised patient. 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* have been reported.

Luminal obstruction of the large bowel secondary to a foreign body has been well documented. The nature of such objects is varied, ranging from corporeal matter, including hair and gallstones, to organic material and synthetic articles. Intraluminal blockage can occur via ingestion or insertion of an object per rectum, or from extrinsic compression. Various artefacts have been reported, including fruit and/or vegetables, sex toys, light bulbs, and bottles. If conservative methods fail, endoscopy, transanal extraction, and surgery have roles in the management of these patients.

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 and small intestine. Various types of bezoar exist; phyto, lacto, pharmaco, tricho, and food boluses. Phytobezoars are the most common, caused by the accumulation of undigested plant and fibrous material. Obstructing large bowel seed bezoars due to wild banana, jaboticaba, poppy, pumpkin, watermelon, sunflower, and pomegranate have been described.

Drug or body packing is a widely reported cause of LBO, first described by Deitel and Syed in 1973. The practice relates to the internal concealment of illicit drugs, most commonly cocaine or heroin. Ischemia, perforation, and drug toxicity (body packer syndrome) make this a precarious condition.

LBO secondary to a gallstone has been reported, with nearly 40 “gallstone sigmoid ileus” or “gallstone colesus” cases since 2000. Two mechanisms for gallstone sigmoid ileus exist: cholecystocolonic fistula and, less commonly, a stone traversing the ileocaecal junction via a cholecystoenteric fistula. 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.

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%. Injuries to the transverse and sigmoid colon are more common given their anterior placement. LBO may occur as a result of different mechanisms: compression between abdominal wall and vertebrae and/or intra-abdominal organs; deceleration injury; laceration; or devascularisation to bowel or mesentery.

Diseases capable of forming adhesions or scarring internally are theoretically able to obstruct the large bowel. Inflammatory bowel disease is an obvious potential etiology. Other documented inflammatory causes include endometriosis, Churg–Strauss syndrome, Behcet’s disease, sarcoidosis, distal intestinal obstructive syndrome secondary to cystic fibrosis, pancreatitis, appendicitis, encapsulating peritoneal sclerosis, and giant cell phlebitis. Management options vary from conservative to endoscopic to surgical.

Adenocarcinoma of the colon accounts for 50% to 60% of LBO cases. Other malignancies and benign pathologies make up a small percentage. Malignant bowel obstruction is estimated to occur in 10% to 28% of colorectal cancers and 5.5% to 42% of ovarian, stomach (6% to 19%), pancreas (6% to 13%), bladder (3% to 10%), and uterine (3% to 11%) malignancies, with breast cancer and melanoma also described. They may cause intraluminal obstruction, intramural obstruction, or extrinsic compression. Cases of lymphoma, leiomyosarcoma, and Kaposi’s sarcoma have also been described.

Giant inflammatory polyps are a recognised association of inflammatory bowel disease. Some mucosal polyps are capable of occluding the bowel lumen. Colonic lipomas, neurofibromatosis, lymphangiomas, and haemangiomas have all been reported.

Various causes of extrinsic compression to the large bowel, including foreign bodies, localized or disseminated malignancy, or benign lesions (eg, uterine fibroids) have been documented. Bladder calculi, urinary retention, ovarian cystadenofibroma and teratomas, aneurysms, leiomyoma and pregnancy have also been described.

Both investigations and surgery may potentially cause LBO. Although adhesions are an important and relatively common cause of small bowel obstruction, they are a rare cause of LBO. Barium impaction, radiotherapy, percutaneous endoscopic gastrostomy, and colonoscopy represent other iatrogenic causes.

A number of congenital conditions may manifest with LBO. Meconium ileus is an early clinical manifestation of cystic fibrosis which leads to LBO in the newborn. Small left colon syndrome, also known as meconium plug syndrome, can give rise to obstruction due to inability of the colon to propel meconium through the intestinal tract. Colonic atresia may cause congenital LBO, most commonly due to a vascular insult on the mesentery. Malrotation, Hirschsprung’s disease, hernia, and Ladd’s bands are other reported causes.

Approximately 1% to 5% of all cases of adult intestinal obstruction are caused by intussusception, mostly within the small bowel (50% to 80%). It commonly occurs in areas acting as junctions between freely moving and fixed segments of the gastrointestinal tract. The etiology of intussusception can be classified as benign, malignant, or idiopathic; 66% of large bowel intussusception cases are caused by a malignant lead point, with colorectal adenocarcinoma the most common cause.

Reported cases of hernia as a cause of LBO are relatively rare. Inguinal, diaphragmatic, lumbar, internal, and ventral hernias are all implicated in colonic obstruction.

This monograph both informs and educates readers with regard to the history, pathophysiology, breadth of LBO presentations, and 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 here is not to give an exhaustive list of every individual case ever published, but to highlight the extent and diversity with which LBO may manifest. Management strategies of these rare presentations 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.