Updated in Etiology, Risk factors, diagnosis and Management of Intestinal Obstruction

Abstract:

Bowel obstruction is an significant cause of morbidity and mortality. Bowel obstruction etiology is based on a mechanical intrinsic luminal obstruction or extrinsic compression. Laboratory appraisal of patients with suspected obstruction must include a complete blood count and metabolic panel. Hypokalemic, hypochloremic metabolic alkalosis may be noted in patients with plain emesis. Elevated blood urea nitrogen levels are consistent with dryness, and hemoglobin and hematocrit levels may be increased. The white blood cell count may be raised. Acute intestinal obstruction occurs when there is an interruption in the forward flow of intestinal contents. This interruption can happen at any point along the length of the gastrointestinal tract. Management of intestinal obstruction is focused at correcting physiologic derangements began by the obstruction, bowel rest, and removing the source of obstruction

Keywords: Bowel obstructions, Adhesive obstruction, Non adhesive obstruction, Virgin abdomen, Intestinal obstruction

Introduction

Over the last 100 years, the anatomical location of Bowel Obstruction (BO) has endured unchanged; however, the aetiological factors in small and large BO have altered significantly. With advance of time more and more elderly patients are donating with (1). But still, BO continues to be one of the most common surgical emergencies (2) encountered in overall surgery units and it continues to be a major cause of morbidity and financial expenditure (3).Peritoneal adhesions and hernia were the most common causes of BO and contributing 42.3% (4). All patients of BO are potential candidates for major abdominal surgery with long term morbidity and possible mortality. Henceforth, the decision of surgery and its timing is vital. Numerous factors are considered for taking the decision on operative or non-operative management. The factors considered are age of the patients, period of obstruction,

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volume of nasogastric aspirate, findings on the radiological imaging, earlier abdominal surgeries and malignancy.

A bowel obstruction can whichever be a mechanical or functional obstruction of the small or large intestines. The obstruction occurs when the lumen of the bowel becomes both partially or completely blocked. Obstruction frequently causes abdominal pain, nausea, vomiting, constipation-to-obstipation, and distention. This, in chance, prevents the normal movement of digested products. Small bowel obstructions (SBOs) are more common than large bowel obstructions (LBOs) and are the most frequent suggestion for surgery on the small intestines. Bowel obstructions are classified as a partial, complete, or closed loop. A closed-loop obstruction denotes to a type of obstruction in the small or large bowel in which there is complete obstruction distally and proximally in the given segment of the intestine.(5,6,7)

SBO incidence is about 350,000/annum in the USA. Etiologies include adhesions (65%), hernias (10%), neoplasms (5%), Crohn's disease (5%), and other (15%). Bowel dilatation happens proximal to obstruction primarily from swallowed air and secondarily from intraluminal fluid accretion. Dilatation increases mural tension, decreases mucosal perfusion, origins bacterial proliferation, and decreases mural tensile strength that increases bowel puncture risks. Classical clinical tetrad is abdominal pain, nausea and emesis, abdominal distention, and constipation-to-obstipation. Physical examination may reveal restlessness, acute illness, and signs of dehydration and sepsis, with tachycardia, pyrexia, dry mucous membranes, hypotension/orthostasis, abdominal distention, and hypoactive bowel sounds. Severe direct tenderness, involuntary guarding, abdominal rigidity, and rebound tenderness suggest advanced SBO, as do marked leukocytosis, neutrophilia, bandemia, and lactic acidosis. (8)

Intestinal obstruction accounts for approximately 15 percent of all emergency section visits for acute abdominal pain(9). Complications of intestinal obstruction include bowel ischemia and puncture. Morbidity and mortality associated with intestinal obstruction have declined since the arrival of more sophisticated diagnostic tests, but the disorder remains a challenging surgical diagnosis. Physicians who are treating patients with intestinal obstruction must weigh the risks of surgery with the penalties of inappropriate conservative management. A suggested method to the patient with suspected small bowel obstruction

Bowel obstruction is an important source of morbidity and mortality accounting for nearly 30,000 deaths and more than \$3 billion per year in direct medical costs; it is answerable for

approximately 15% of hospital charges for acute abdominal pain in the USA and ~ 20% of cases needing acute surgical care (10,11). Bowel obstruction etiology is grounded on a mechanical intrinsic luminal obstruction or extrinsic compression. Adynamic ileus and colonic pseudo-obstruction are produced by a lack of enteric propulsion (12). Colonic pseudo-obstruction and an adynamic ileus can be caused by drugs, trauma, postoperative period, metabolic disturbance, and other diverse basis (12,13).

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In 90% of cases, small bowel obstruction is produced by adhesions, hernias, and neoplasms (14). Adhesive small bowel obstruction represents 55–75% of small bowel obstruction cases (15). while hernias and small bowel tumors interpretation for the remainder (11). Large bowel obstruction is provoked by cancer in about 60% of cases (16); volvulus and diverticular illness are responsible of other 30% (10). Other various causes (carcinomatosis, endometriosis, inflammatory bowel disease stenosis, etc.) account for the residual 10–15% of bowel obstructions. This review emphases on the management of bowel obstruction excluding duodenal mechanical obstruction to be better included in gastric outlet obstruction unit (17).

Etiology

There are numerous potential etiologies of small and large bowel obstructions that are classified as either extrinsic, intrinsic, or intraluminal. The most mutual cause of SBOs in industrialized nations is from extrinsic sources, with post-surgical bonds being the most common. Significant adhesions can cause kinking of the bowel foremost to obstruction. It is estimated that at least two-thirds of patients with preceding abdominal surgery have adhesions. Other common extrinsic sources include cancer, which causes density of the small bowel leading to obstruction. Less common but still prevalent extrinsic reasons are inguinal and umbilical hernias. Untreated or symptomatic hernias may eventually become kinked as the small bowel protrudes through the defect in the abdominal wall and develops entrapped in the hernia sack. Hernias that are not identified or are not reducible may development to obstruction of the bowel and are considered a surgical emergency with the strangulated or imprisoned bowel becoming ischemic over time. Other reasons of SBO include intrinsic disease, which can create an insidious onset of bowel wall thickening. The bowel wall gradually becomes

compromised, forming a stricture. Crohn disease is the most mutual cause of benign stricture seen in the adult population. (18,19)

Intraluminal causes for SBOs are less mutual. This procedure occurs when there is an ingested foreign body that causes impaction inside the lumen of the bowel or navigates to the ileocecal valve and is unable to pass, forming a barricade to the large intestine. However, it is noted that most foreign bodies that pass through the pyloric sphincter will be able to permit through the rest of the gastrointestinal tract. LBOs are less mutual and compromise only 10% to 15% of all intestinal obstructions. The most mutual reason of all LBOs is adenocarcinoma, shadowed by diverticulitis and volvulus. Colonic obstruction is most usually seen in the sigmoid colon

Epidemiology

Small and large bowel obstructions are alike in incidence in both males and females. The overriding issue affecting incidence and distribution depends on patient risk factors, including but not limited to: previous abdominal surgery, colon or metastatic cancer, chronic intestinal inflammatory disease, present abdominal wall and/or an inguinal hernia, earlier irradiation, and foreign body ingestion. (20.21)

Pathophysiology

The normal physiology of the small intestine consists of the absorption of food and the absorption of nutrients. The large bowel continues to assistance in digestion and is responsible for vitamin synthesis, water absorption, and bilirubin failure. Any obstructive mechanism will hinder these physiologic components. Obstruction causes enlargement of the bowel proximal to the changeover point and collapses distally. A result of partial or complete obstruction of digested products during obstruction is emesis. Recurrent emesis can lead to fluid deficits and electrolyte abnormalities. As the disorder is left untreated and worsens, a bowel wall edema forms, and third-spacing initiates.(22)

The fundamental concerns about intestinal obstruction are its result on whole body fluid/electrolyte balances and the mechanical effect that increased heaviness has on intestinal perfusion. Proximal to the point of obstruction, the intestinal tract widens as it fills with

intestinal secretions and swallowed air.(23) Failure of intestinal fillings to pass through the intestinal tract leads to a cessation of flatus and bowel schedules. Intestinal obstruction can be broadly distinguished into small bowel and large bowel obstruction. Fluid loss from emesis, bowel edema, and loss of absorptive capacity indications to dehydration. Emesis leads to loss of gastric potassium, hydrogen, and chloride ions, and significant dehydration inspires renal proximal tubule reabsorption of bicarbonate and loss of chloride, preserving the metabolic alkalosis(24). In adding to derangements in fluid and electrolyte balance, intestinal stasis leads to overgrowth of intestinal flora, which may lead to the expansion of feculent emesis. Additionally, overgrowth of intestinal flora in the small bowel indications to bacterial translocation across the bowel wall(25).

Causes and Risk Factors

The most mutual reasons of intestinal obstruction include adhesions, neoplasms, and herniation. Adhesions resulting from prior abdominal surgery are the main cause of small bowel obstruction, secretarial for approximately 60 percent of cases(26). Lower abdominal surgeries, including appendectomies, colorectal surgery, gynecologic measures, and hernia maintenances, confer a greater risk of adhesive small bowel obstruction. Less mutual causes of obstruction comprise intestinal intussusception, volvulus, intra-abdominal abscesses, gallstones, and foreign bodies.

History and Physical Examination

Patients should be requested about their history of abdominal neoplasia, hernia or hernia repair, and inflammatory bowel disease, because these conditions rise the risk of obstruction. The trademarks of intestinal obstruction include colicky abdominal pain, nausea and vomiting, abdominal distension, and a termination of flatus and bowel movements. It is important to differentiate between true mechanical obstruction and other reasons of these symptoms. Distal obstructions allow for a superior intestinal reservoir, with pain and distension more marked than emesis, whereas patients with proximal obstructions may have slight abdominal distension but marked emesis. The attendance of hypotension and tachycardia is an

sign of severe dehydration. Abdominal palpation may reveal a enlarged, tympanitic abdomen; however, this finding may not be current in patients with early or proximal obstruction. Auscultation in patients with early obstruction reveals high-pitched bowel sounds, while those with late obstruction may present with slight bowel sounds as the intestinal tract becomes hypotonic(27).

Suspected bowel obstruction necessitates the practitioner to obtain a detailed medical history inquiring about significant risk issues related to bowel obstruction. Small and large bowel obstruction have many overlapping symptoms. Though, quality, timing, and presentation differ. Commonly in SBO, abdominal pain is described as sporadic and colicky but improves with vomiting, while the pain associated with LBO is incessant. The vomiting in SBO tends to be more frequent, in larger volumes, and bilious, which is in dissimilarity to vomiting during an LBO, which typically presents as recurrent and feculent when present. Tenderness to palpation is present in both circumstances, but with SBO, it is more focal, and with LBO, it is more diffuse. Additionally, distention is marked in LBO with obstipation more usually present. It is important to note that in certain circumstances, an LBO will mimic an SBO if the ileocecal valve is incompetent. A useless ileocecal valve can allow for the insufflation of air from the large bowel into the small bowel manufacturing symptoms of an SBO(22)

Differential Diagnosis

- Abdominal hernias
- Abdominal pain in elderly people
- Appendicitis
- · Chronic megacolon
- Colonic polyps
- Diverticulitis
- Diverticulitis empiric therapy

- · Pseudomembranous colitis surgery
- Small bowel obstruction
- Toxic megacolon(22)

Diagnostic Evaluation

Abdominal plain X-ray: Abdominal basic X-ray is the first level radiologic study. In small bowel obstruction, plain abdominal radiographic results are diagnostic in 50-60%, inconclusive in 20-30%, and misleading in 10-20% of patients (28,29). In one study after radiography, the sensitivity of bowel obstruction was meaningfully higher than after clinical assessment only: 74% versus 57%, respectively (P < 0.01). But, the positive predictive value did not differ significantly between clinical valuation only and with plain radiographs (30). In a review of 140 cases of suspected large bowel obstruction, the abdominal X-ray had 84% sympathy and 72% specificity (31)

Water-soluble contrast administration X-ray: A water-soluble contrast enema has 96% sensitivity and 98% specificity in diagnosing great bowel obstruction (31) but cannot distinguish different great bowel obstruction causes. A small bowel follow-through with water-soluble contrast is widely used in patients for paste small bowel obstruction non-operative management. Numerous systematic reviews and meta-analyses have established the utility of water-soluble difference agents in the diagnostic work-up of adhesive small bowel obstruction (32–33]. If the dissimilarity has not reached the colon on an abdominal X-ray 24 h after administration, then this is highly revealing of non-operative management failure (34). Numerous studies have shown that the use of water-soluble contrast agents precisely predicts the need for surgery with an vigorous therapeutic role (32,35,36,37). The administration of water-soluble contrast agents in adhesive small bowel obstruction is harmless in terms of morbidity and mortality, but adverse properties due to their use have been reported. Potential

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life-threatening complications are aspiration pneumonia and pulmonary edema. To evade these complications, the contrast medium should be managed when the stomach has been sufficiently decompressed through a nasogastric tube. Another potential adverse effect is that water-soluble contrast agents, because of higher osmolarity, may further desiccate a patient with small bowel obstruction, instable fluids into bowel lumen; in some children and elderly adults, the loss of plasma fluid may be adequate to cause a shock-like state (38).

Ultrasound: Small bowel obstruction can be identified with ultrasound if there are > 2.5-cm dilated loops of the bowel that are proximal to distorted loops of bowel and if there is decreased or absent peristalsis activity (39). Using ultrasound for small bowel obstruction diagnosis has 90% sensitivity and 96% specificity (40). Picturing of large bowel obstruction with ultrasound is as good as computed tomography. Computed tomography is obviously larger to ultrasound in terms of the etiologic definition for together small bowel obstruction and large bowel obstruction (41, 42). Ultrasound achieves healthier than planar abdominal X-ray in large bowel obstruction (43).

Computed tomography scan: The diagnostic correctness of computed tomography with intravenous contrast is superior to that of conservative abdominal radiography and ultrasound. In addition to its higher sensitivity and specificity, an significant benefit of computed tomography is its aptitude to deliver information about the underlying cause of obstruction or to offer information about an alternative diagnosis if no signs of bowel obstruction are current. Computed tomography leads to more accurate management and support in preoperative planning (44). Positive oral contrast physical is not needed in the diagnosis of small bowel obstruction with computed tomography because the intraluminal fluid and gas already current within the obstructed bowel are excellent dissimilarity agents. If positive oral contrast material has been given in patients with small bowel obstruction, then a late abdominal radiograph during non-operative management can measure if the contrast material has advanced to the colon. When doubts about the large bowel obstruction diagnosis persevere, a water-soluble rectal contrast agent can be managed to better envisage obstruction

Magnetic resonance imaging: To minimalize the burden of ionizing radiation in children and pregnant women, magnetic resonance imaging is a valid alternative inspection to computed tomography scan for bowel obstruction (45): potential study demonstrated a compassion of 95% and a specificity of 100% (46).

Colonoscopy: The role of colonoscopy is imperfect to the diagnosis of large bowel obstruction. The goal is to exclude other reasons for obstruction. Biopsy should be performed in cases of suspected malignancy when spare surgery has not been designated or endoscopic stent location can be expected (47).

Diagnostic Testing and Imaging (LABORATORY TESTS)

Laboratory evaluation of patients with suspected obstruction should comprise a complete blood count and metabolic panel. Hypokalemic, hypochloremic metabolic alkalosis may be noted in patients with plain emesis. Elevated blood urea nitrogen levels are reliable with dehydration, and hemoglobin and hematocrit levels may be increased. The white blood cell count may be raised if intestinal bacteria translocate into the bloodstream, causing the systemic inflammatory response condition or sepsis. The progress of metabolic acidosis, especially in a patient with an increasing serum lactate level, may sign bowel ischemia (27).

Treatment

Initial management should permanently comprise an assessment of the patient's airway, breathing, and circulation. If resuscitation is required, it should be achieved with isotonic saline and electrolyte replacement. A Foley catheter should be introduced to monitor the patient's urine production if the patient is unstable or septic. Nasogastric tube addition will allow for bowel decompression to dismiss distention proximal to the obstruction. Nasogastric tube insertion will also aid regulate emesis, allow for accurate assessment of intake and output, and lower the danger of aspiration(22).

Management of intestinal obstruction is directed at modifying physiologic derangements caused by the obstruction, bowel rest, and removing the source of obstruction.

The former is addressed by intravenous fluid revival with isotonic fluid. The use of a bladder catheter to closely display urine output is the lowest condition for gauging the capability of resuscitation; other invasive trials, such as arterial canalization or central venous pressure monitoring, can be used as the clinical situation licenses. Antibiotics are used to treat intestinal overgrowth of bacteria and translocation across the bowel wall(48). The attendance of fever and leukocytosis must quick inclusion of antibiotics in the initial treatment routine. Antibiotics should have coverage against gram-negative organisms and anaerobes, and the choice of a exact agent should be determined by local susceptibility and convenience. Aggressive replacement of electrolytes is recommended after satisfactory renal function is confirmed.

Therapy Conservative (non-surgical) therapy

Conservative treatment is the cornerstone of nonoperative organization in all patients with adhesive small bowel obstruction except there are signs of intestinal ischemia/perforation. Sign for the ideal duration of non-operative is lacking, but most authors consider a 72-h cutoff safe and appropriate (49). The mainstay of non-operative administration is nil per os and decompression with naso-gastric suction or long intestinal tube. Here has been some discussion in the literature about the use in paste small bowel obstruction of long intestinal tubes: long trilumen naso-intestinal tubes are more active than naso-gastric tubes, but they require endoscopic insertion (50). Water-soluble contrast management is a valid and safe treatment that relates with a significant reduction in the requirement for surgery in patients with adhesive small bowel obstruction with also a important reduction in the time to determination and length of stay. The administration of water-soluble difference is a safe treatment with no significant changes in complications or mortality [51,52].

Surgery

Prosthetic repair is the management of choice for most abdominal wall complicated hernias (inguinal, femoral, incisional, umbilical, epigastric, parastomal, spigelian, etc.). In circumstance of perforation/bowel resection with polluted surgical fields, suture repair is preferred due to the danger of mesh infection. Diagnostic laparoscopy may be a useful tool to assess bowel viability after reduction of complicated hernias (53). Reparation of complicated

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hernia can be performed with a laparoscopic method when no bowel resection anastomosis is needed, which normally requires a mini-open method (small laparotomy) (53). Internal hernias are cured with prompt reduction, suture repair, and bowel resection anastomosis in case of intestinal necrosis.

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