Anaesthetic management of intestinal obstruction

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Key points:

Intestinal obstruction is a common condition with multiple causes

Serious complications include perforation, ischaemia and sepsis

Resuscitation and pre-operative optimisation are likely to improve outcome

Remember hidden losses when considering fluid resuscitation

Obstruction of the small or large bowel accounts for 20–30% of acute adult surgical referrals. It affects all ages and is associated with significant morbidity and mortality, especially at the extremes of age. Despite improvement in diagnostic tools and availability of emergency 24 h surgical care, the management of intestinal obstruction is still a challenge. The clinical course can be protracted or it can be associated with a life-threatening complication that requires urgent treatment.

Aetiology

The spectrum of conditions leading to obstruction is enormous (Table 1). The commonest cause (45–60%) is abdominal adhesions, the small bowel being affected in > 90% of cases. There are two main types of intestinal obstruction; dynamic (peristalsis working against a mechanical obstruction) and adynamic (no peristalsis, *e.g.* paralytic ileus, or non-propulsive activity, *e.g.* pseudo-obstruction).

Table 1 Classification and differential diagnosis of intestinal obstruction

Paralytic ileus
Concentric narrowing
Crohn's disease (benign or malignant stricture)
Neoplasm (lymphoma, adenocarcinoma, other)
Diverticulitis
Resolved ischaemic enteritis
Kinking of a loop
Postoperative adhesions
Incarcerated hernia
Twisting of a loop
Caecal volvulus
Sigmoid volvulus
Midgut volvulus
Intussusception
Spontaneous
Secondary to polyp or mass
Foreign body
Ingested
Gallstone
Pseudo-obstruction

Mechanism and classification

The mechanism of obstruction is as important as the aetiology, since this influences the likelihood of bowel compromise. Four general mechanisms are recognised: (i) volvulus with torsion; (ii) incarceration in a confined space (vascular compromise and infarction likely); (iii) intrinsic and extrinsic obstruction of the lumen; and (iv) intussusception. The obstruction can be located in the high small bowel, low small bowel and large bowel – each of these presenting with different signs. Furthermore, it can be complete or incomplete and acute (several hours) or chronic (weeks).

The obstruction can be classified also by its effect on the bowel: (i) simple – lumen obstructed but the mesenteric blood flow intact; (ii) strangulated – compromised blood flow; (iii) closed loop – bowel is looped over itself and obstructed at both ends, accelerating the onset of symptoms; or (iv) pseudo-obstruction – no true mechanical obstruction exists.

Pathophysiology

The sequence of events is virtually identical regardless of the segment affected, cause or time-course (Fig. 1). In the early stages, bowel below the obstruction exhibits normal peristalsis and absorption until empty, when it contracts and becomes immobile. In an initial reaction, the bowel above the obstruction increases its blood supply and peristaltic activity in order to overcome the blockage. If the obstruction is not relieved, the bowel begins to dilate causing a reduction in the strength of peristaltic contractions, eventually becoming flaccid. This is initially protective because it prevents vascular damage.

The distension proximal to an obstruction is produced by gas (nitrogen 90% and hydrogen

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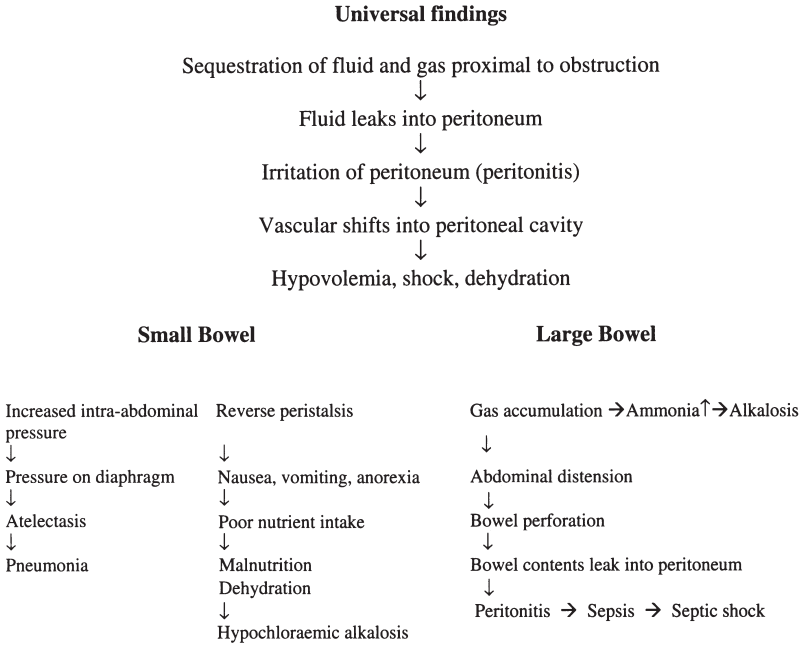

Fig. 1 Pathophysiology of intestinal obstruction.

sulphide) caused by bacterial overgrowth and fluid (digestive juices). The bowel wall becomes oedematous and, eventually, fluids leak out into the peritoneum causing peritoneal irritation, contamination and signs of peritonitis. As fluid and electrolytes are sequestered in the lumen and its absorption impeded, hypovolaemia, electrolyte imbalance, dehydration and shock occurs. The bowel continues to distend as fluid and gas continue accumulating, the intramural vessels become stretched and the blood supply compromised leading to ischaemia and necrosis. Eventually perforation occurs.

In small bowel obstruction, increased intra-abdominal pressure, if not relieved by vomiting, will lead to diaphragmatic impingement, limitation of chest wall excursion, basal atelectasis and pneumonia. Nausea, emesis and anorexia are common. Biochemical investigations reflect sodium and water loss. Chloride loss, by creating an alkalosis, exacerbates potassium losses. If the obstruction is in the high small bowel, biochemical abnormalities will mimic those of gastric outlet obstruction. If prolonged, this develops into hypovolaemic, hypokalaemic, hypochloraemic, metabolic alkalosis.

If the obstruction occurs lower in the GI tract, metabolic acidosis develops because intestinal juice contains more bicarbonate than chloride. As the bowel becomes inflamed, there is an increasing loss of protein. Starvation adds to protein losses and the consequent hypo-albuminaemia exacerbates abnormal fluid shifts.

In large bowel obstruction, accumulation of methane may lead to hyper-ammonaemia and alkalosis. However, intestinal wall ischaemia and bacterial translocation will lead to peritonitis, sepsis and septic shock which are associated with metabolic acidosis and protein loss.

Clinical features

The clinical features vary enormously depending on the site and duration of obstruction, presence of complications and medical co-morbidity of the patient. However, there are 4 cardinal features – pain, vomiting, distension and constipation.

In small bowel obstruction, signs and symptoms are evident soon after the blockade of intestinal transit starts with colicky pain localised in the epigastrium or upper quadrant in a crescendo-decrescendo fashion occurring a few minutes after eating. Hyperactive bowel sounds and distension may be detected in upper quadrants with silent lower quadrants. In a 24 h period, up to 8 l of gastrointestinal secretions can accumulate. Nausea and vomiting are early symptoms and bilious vomiting occurs if the obstruction is distal to the second part of the duodenum. Vomiting of faeculent material is related to enteric bacterial overgrowth. Constipation is classical but watery diarrhoea may occur.

In large bowel obstruction, the clinical course is more silent in the initial stages. It is heralded by abdominal discomfort and absolute constipation. The pain tends to be intermittent

and felt in the lower quadrants and not associated with eating. Localised tenderness in colonic obstruction may be a sign of impending perforation. Closed loop obstruction is often associated with right iliac fossa pain and is a surgical emergency.

Fluids and electrolyte imbalance occurs with vitamin K deficiency which can impair coagulation. Depending on the time of obstruction, the rectum will be empty and only scant watery or thin ribbon-like stools present. Intestinal obstruction is one of the causes of intra-abdominal hypertension and, if untreated, could develop into the compartment syndrome.

The commonest cause of small bowel obstruction is adhesions secondary to previous surgery. The large bowel is more commonly obstructed by malignancies and volvulus. Worrying signs are localised tenderness, generalised peritonitis, hypovolaemia, pyrexia and tachycardia.

Diagnosis

History, examination and investigations will confirm the diagnosis. A leukocytosis may help differentiate simple obstruction from strangulated and perforated bowel. Leukocyte counts of >25,000 dl^{-1} suggest mesenteric occlusion or perforation.

As well as clinical examination, plasma electrolytes, urea, creatinine, amylase and osmolality help in the assessment of dehydration or likelihood of complications. The relative deficiency of individual electrolytes can also suggest the site of obstruction (*i.e.* hypokalaemia, hypomagnesaemia, hypovolaemia or hypophosphataemia are more likely in large bowel obstruction).

Plain abdominal radiographs will confirm the diagnosis in 60% of cases. Large bowel obstruction often requires a water-soluble contrast enema to reliably distinguish between mechanical and pseudo-obstruction. Plain chest radiographs may reveal subdiaphragmatic air indicating perforation.

The main differential diagnoses are mesenteric ischaemia, acute pancreatitis and paralytic ileus. However, other causes of abdominal pain, distension and diminished bowel activity (*e.g.* myocardial infarction, pneumonia, acute sickle cell disease, ketoacidosis or drug effects) should be considered.

Anaesthetic management

After a full history, examination and analysis of the pre-operative investigations, the resuscitation needs of the patient can be assessed. Discussions with the surgical team will revolve around the urgency for surgery, need for pre-operative optimisation, pain relief and the availability of postoperative high dependency beds if deemed necessary. All cases will need some degree of fluid resuscitation to restore the circulating volume and adequate urine output. Most cases secondary to adhesions do not require surgery. About 90% will resolve spontaneously with intravenous fluids and nasogastric suction to decompress the gut.

The majority of cases of intestinal obstruction requiring surgery are categorised as 'urgent' (*i.e.* surgery needs to be performed within 24 h) allowing the procedure to be performed during routine operating hours with ready availability of all grades of anaesthetic and surgical staff. This is a NCEPOD recommendation.

Pre-operative

A full anaesthesia-related history is essential, including drugs and recent compliance. Missed medication can effect perioperative morbidity. Examination of the patient should concentrate on estimation of hydration status and other systems, as indicated by the history. Assessment guides pre-operative investigations, resuscitation, pre-optimisation and necessity for invasive monitoring.

The clinical presentation of intestinal obstruction may vary enormously, *e.g.* from mild dehydration in a well nourished patient, to a shocked, septic patient with severe abdominal pain and reduced conscious level. Fluid losses from vomiting and nasogastric aspiration can be measured but third space and intraluminal losses, especially from the large bowel, can make estimation of the true fluid deficit difficult. Concomitant hypo-albuminaemia can exacerbate loss of fluid from the vascular space. Monitoring of fluid replacement is aided by clinical assessment, hourly urine output and biochemical markers. Central venous pressure measurement is the most common invasive monitoring technique.

The goals of pre-operative fluid management are to restore vascular and interstitial volumes and to correct electrolyte and acid-base imbalances. This will normalise systemic vascular resistance and optimise oxygen delivery. The initial choice of fluid is usually crystalloid and the balanced salt solutions counteract the losses in abdominal obstruction. Colloids are more appropriate when resuscitation of the vascular space is the primary aim in the severely hypovolaemic and hypotensive patient.

Patients with intestinal obstruction may be at high risk and benefit (in terms of hospital stay, morbidity and mortality) from pre-operative optimisation after initial resuscitation on the ward. Pre-operative optimisation with inotropes and fluids

in a critical care area for only 4 h has been shown to reduce mortality (17% to 3%), length of hospital stay and postoperative complication rates in elective high-risk patients. Increased oxygen delivery and improvement in tissue perfusion may limit reperfusion injury and reduce the release of inflammatory mediators. However, these measures may require HDU admission and invasive monitoring. Although pre-optimisation has an increasing evidence-base in elective high-risk surgery, data with respect to emergency surgery are awaited. The role of invasive monitoring in the high-risk patient is well recognised and highlighted in the NCEPOD reports. Despite many years of use, the efficacy of pulmonary artery catheters is only now about to be investigated in a multicentre, randomised, controlled trial.

Intra-operative

After adequate preparation and monitoring, anaesthesia is induced using a rapid sequence induction technique with cricoid pressure. An nasogastric tube will help decompress the stomach and reduce the risk of aspiration. Cricoid pressure should be applied by appropriately trained staff. Choice of agents for induction and maintenance of anaesthesia is guided by the condition of the patient. There is a debate over the use of nitrous oxide (disruption of anastomoses secondary to increasing intraluminal pressure) and reversal with neostigmine has been implicated in anastomoses morbidity. However, there are insufficient data to allow firm conclusions and many anaesthetists use both agents.

The need for fluids and blood product replacement is dictated by the course of the operation but the use of warm fluids and measures to minimise heat loss (*e.g.* heated under- and over-blankets) are paramount. Intra-operative monitoring reflects the minimal standards set by the Association of Anaesthetists' guidelines with additional invasive monitoring as required. The oesophageal Doppler is a relatively non-invasive monitor which can be useful in assessing cardiovascular status.

The pre-operative establishment of epidural analgesia aids peri-operative pain control. A recent meta-analysis has demonstrated the benefits of epidurals and, theoretically, improved splanchnic flow may aid recovery. Evidence indicates that epidural analgesia significantly reduces the incidence of pulmonary morbidity. Opioid/local anaesthetic combinations are more effective and safer than either agents used alone.

Postoperative

The required level of postoperative care will be influenced by the patient's general condition. NCEPOD recommendations strongly suggest that an HDU is the most appropriate place, especially for elderly patients, after emergency surgery. Regardless of where postoperative care takes place, important problems to address are:

- Cardiovascular and respiratory monitoring and stabilization.
- Fluid and electrolyte balance.
- Care of the wound and antibiotic prophylaxis when indicated.
- Commencement of enteral nutrition as soon as feasible.
- Thromboembolism prophylaxis.
- Adequate pain relief to facilitate physiotherapy and prevent atelectasis and consolidation.

Epidural analgesia is regarded as the gold standard, but opioid PCA is often used especially if factors render epidural analgesia unsafe. The multimodal approach to pain control has its advantages, but care should be taken when using NSAIDs in patients who are dehydrated, salt depleted and elderly. The acute pain team can optimise safe pain relief in these patients.

Key references

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See multiple choice questions 88–90.