Acute abdomen: intestinal obstruction

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Abstract

Intestinal obstruction is a common and dangerous surgical emergency that is associated with a high mortality if managed inappropriately; results are usually good if it is diagnosed and treated early. Obstruction of the small intestine and large intestine are fundamentally different in their cause and management (although differentiation between the two merges in distal small-intestine and proximal large-intestine obstruction). In general, obstruction of the small intestine presents with colicky abdominal pain and vomiting, whereas distension and absolute constipation tend to be more common in obstruction of the large intestine. The causes of intestinal obstruction can be mechanical or paralytic in origin, and can be subdivided into small- and large-intestinal obstruction. Obstruction of the large intestine accounts for about 80% of all intestinal obstructions (the most common cause in 'developed countries' is adhesions), whereas adenocarcinoma of the colon is the most common cause of obstruction of the large intestine. A number of pathophysiological changes occur in intestinal obstruction, and these changes determine the clinical picture. Obstruction of the small intestine is characterized by dehydration and hypovolaemia due to fluid losses; obstruction of the large intestine is influenced by the competency of the ileocaecal valve. These features are diagnosed clinically and by radiography, but the decision to operate is a clinical (based on abdominal examination and clinical suspicion of potential pathology). Despite improvements in management, the mortality ranges from 10–30% (depending on type of obstruction and the presence of perforation) in the UK.

Keywords acute abdomen; decompression; emergency surgery; intestinal obstruction; intussusception; paralytic ileus; pseudo-obstruction; strangulated obstruction; volvulus

Intestinal obstruction is a common and potentially dangerous surgical emergency that is associated with high mortality if managed innappropriately. The clinical features are vomiting, constipation, abdominal distension and pain; the relative magnitude of each differs according to the cause and site of intestinal obstruction. Successful management requires early diagnosis and treatment, with meticulous balance of fluid and electrolytes and, if appropriate, timely surgical intervention.

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Aetiology

In general, the aetiology of intestinal obstruction can be divided into those causes in which vigorous peristalsis is associated with mechanical obstruction resulting from physical occlusion or distortion of the intestinal lumen, and conditions of intestinal 'paralysis' in which peristalsis is absent or disordered, without a mechanical obstruction. Each mechanism can be further divided into obstruction of the small and large bowel (Table 1 and Figure 1). Postoperative adhesions are the most common cause of intestinal obstruction in 'developed' countries and account for about 40% of all cases; inguinal hernia is the most common cause of intestinal obstruction in 'developing' countries.

Mechanical obstruction of the small intestine: about 80% of intestinal obstruction involves the small intestine. Mechanical obstruction of the small intestine can be due to a variety of causes.

Luminal – foreign bodies, faecoliths, gallstones, bezoars, parasites, polypoidal tumours.

Intrinsic – atresia, inflammatory strictures (tuberculosis, Crohn's disease), tumours.

Extrinsic – adhesions, hernias, volvulus, intussusception, bands, inflammatory or neoplastic masses.

Mechanical obstruction of the large intestine: in developed countries, colon cancer is the most common cause of mechanical obstruction of the large bowel, with a benign stricture due to colonic diverticulitis, inflammatory bowel disease, and sigmoid volvulus accounting for other presentations. Less commonly, mechanical obstruction of the large bowel may be due to colonic intussusception, radiation- and ischaemia-induced strictures and external hernias. It is exceedingly rare for adhesions to cause obstruction of the large bowel because the large intestine is mainly a retroperitoneal structure.

'Paralytic' obstruction of the large and small intestine: peristaltic failure develops as a result of disordered neuromuscular transmission within the myenteric plexuses of the intestinal wall. This leads to the development of a 'floppy' and inert intestine, with accumulation of fluid and gas, vomiting, absolute constipation and absent bowel sounds. This condition is termed 'paralytic ileus' if it affects the small intestine, whereas colonic involvement is termed 'pseudo-obstruction'.

Paralytic ileus may develop as a consequence of abdominal surgery (where it usually lasts for <72 hours), as a consequence of intra-abdominal infection, metabolic disorders (e.g. uraemia, hypokalaemia) or as a reflex response to retroperitoneal (e.g. spinal) injuries.

Acute colonic pseudo-obstruction (Ogilvie's syndrome) may be caused by the same abnormalities that lead to paralytic ileus, but may also complicate acute neurological disease (e.g. stroke, subarachnoid haemorrhage), pneumonia, and be associated with drug (e.g. tricyclic antidepressants) use. Metabolic causes (e.g. diabetic ketoacidosis, hyponatraemia) are less common.

Causes of intestinal obstruction and paralytic ileus

	Common	Less common	Rare
Mechanical	Adhesions	Sigmoid diverticular disease	Tumours of the small bowel
	External hernias	Crohn's disease	Gallstone ileus
	Colorectal carcinoma	Intussusception	
		Volvulus	
Paralytic 'ileus'	Postoperative (up to 72 hours)	Pseudo-obstruction (Ogilvie's syndrome)	Metabolic/biochemical (e.g. ketoacidosis, severe hypokalaemia)
	Pancreatitis	Drugs (opiates, anticholinergics)	
	Mesenteric infarct	Retroperitoneal haemorrhage	

Table 1

Pathophysiology

A number of significant pathophysiological changes occur in intestinal obstruction and these changes determine the clinical manifestations.

Obstruction of the small intestine

The small intestine has two functional components: a proximal part (which has a predominantly secretory role) and a distal segment (absorption role). One-fifth of the total fluid of the body is secreted and reabsorbed through the intestine daily, and interference with this process may rapidly cause fluid sequestration in the bowel, with massive depletion of fluid and electrolytes.

In complete obstruction, the bowel distal to the point of obstruction is emptied of its fluid and gas content by absorption and therefore collapses. The bowel proximal to the obstruction distends with gas and fluid, which is persistently augmented by the continuous secretion of biliary, pancreatic and gastrointestinal juices.

Relative frequency of underlying diagnosis of intestinal obstruction

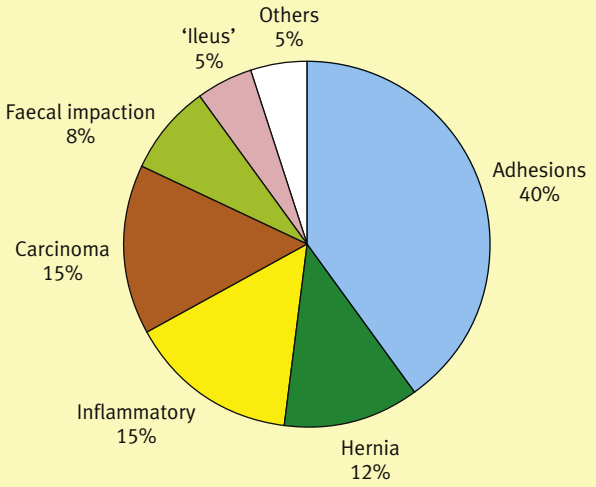

Figure 1

In the early stages, the major source for the intestinal gas is swallowed air, which is responsible for about 70% of the gas in the distended bowel. The major component of atmospheric air is nitrogen which, unlike oxygen, is poorly absorbed. With time, the amount of oxygen in the bowel steadily falls and this is associated with a concomitant rise in carbon dioxide. Gas arising from bacterial fermentation becomes increasingly important if the obstruction is not relieved. This process is responsible for the production of other gases (e.g. hydrogen sulphide, ammonia), which lower the partial pressure of nitrogen within the lumen, and so establish a gradient for the further diffusion of nitrogen from the congested vessels in the mucosa into the lumen.

Large quantities of isotonic fluid pass into the bowel lumen and, because this fluid is not reabsorbed, it is lost from the extracellular compartment. Initially, the fluid may contain recognizable food, but this is soon replaced by turbid, bile-stained fluid and gradually the colour of the intestinal juice darkens. Fermentation and digestion by proliferating micro-organisms within the obstructed intestine are responsible for the faeculent odour of bowel contents. (In health, the bacterial count of the small bowel is very low, but the stagnant, protein-rich fluid of the obstructed small intestine provides an ideal culture medium and micro-organisms proliferate rapidly).

The rate of movement of salt and water from the blood to the lumen increases, whilst the rate of movement from the lumen to the blood remains static or decreases. Thus, absorption is halted, and net secretion of fluid into the obstructed lumen occurs, resulting in further losses of fluid and electrolytes.

Excretion of urinary sodium and water is reduced (in order to maintain plasma volume), resulting in oliguria and movement of fluid from the interstitial space into the intravascular space, which helps to preserve plasma volume.

A large deficit in total-body potassium may occur but, because of shifts in potassium between the extravascular and intravascular compartments, hypokalaemia usually develops late. Vomiting accentuates deficits of fluid and electrolytes and may impair acid–base balance (see page 194). Profound hypovolaemia is an important cause of death in inadequately treated obstruction of the small bowel and may contribute to complications in patients undergoing surgery without effective resuscitation.

As the intestine becomes progressively distended with gas and fluid, intraluminal pressure rises and venous drainage from the bowel may become impaired. This causes congestion and

oedema of the mucosa, contributing to further loss of intraluminal fluid and additional loss from the serosal surface of the bowel into the peritoneal cavity. Peristalsis becomes more vigorous in an endeavour to overcome the resistance imposed by the obstruction. Eventually, the intestinal smooth muscle becomes fatigued, at which time bowel sounds cease to be heard.

Obstruction of the large intestine

The pathophysiology of obstruction of the large bowel is influenced by the competency of the ileocaecal valve. In about 20–30% of patients with obstruction of the large bowel, the ileocaecal valve becomes incompetent and colonic pressure is relieved by reflux of colonic contents into the ileum. This results in distension of the colon and the small intestine and, ultimately, faeculent vomiting.

If the ileocaecal valve remains competent, a closed loop is formed between the valve and the point of obstruction, resulting in progressive colonic distension as the ileum continues to empty gas and fluid into the obstructed segment. Intraluminal pressures may reach very high levels, impairing the circulation of the bowel wall, resulting in the rapid development of gangrene and colonic perforation; this usually occurs in the caecum because the wall of the right colon is thinner than that of the left and its calibre is greater. When the intraluminal pressure rises, distension and wall tension are maximal in the distended caecum. If the caecal diameter reaches 15 cm, the threat of ischaemic necrosis and perforation is so great that colonic decompression must be initiated immediately.

Specific types of mechanical obstruction

Strangulated obstruction of the intestine is a surgical emergency and occurs if there is impairment of the blood supply to the bowel wall. A trapped or acutely angulated segment of bowel may have its venous drainage impaired, causing capillary engorgement. This greatly increases tension within the bowel wall, which eventually compromises arterial inflow, ultimately resulting in arterial occlusion; haemorrhagic infarction of the bowel and bleeding into the lumen follows. Bacteria may invade the damaged wall and the peritoneal cavity, causing localized and eventually generalized peritonitis with frank perforation of the involved loop. Strangulated obstruction characteristically starts acutely and is painful. The presence of shock, leukocytosis or signs of peritoneal irritation should raise the suspicion of strangulation, which requires emergency surgical exploration because the effects of strangulation are rapidly fatal if untreated.

A closed-loop obstruction is a form of mechanical obstruction in which a segment of bowel is isolated by closure of both of its ends. This may occur as a result of a loop of small bowel becoming trapped in a hernia or twisting about an unyielding band, causing a volvulus. The rapidity of the onset of symptoms and signs depends of the length of the involved segment and the number of organisms in the lumen. Tension rises rapidly and devitalization and rupture swiftly follow if the intestinal contents are heavily colonized or the length of the affected loop is short.

Volvulus is an axial rotation of a segment of the intestine about its mesentery. Complete volvulus results in a closed-loop obstruction and early mesenteric vascular occlusion. Volvulus may be primary (when it occurs as a result of a congenital malrotation of the bowel) or secondary (when it occurs due to twisting of the bowel around an adhesion band or a stoma). Large-bowel volvulus most commonly affects the sigmoid colon and caecum.

Intussusception is a telescoping (or invagination) of a loop of proximal intestine (intussusceptum) inside the immediately adjacent segment (intussuscipiens). Most intussusceptions are ileocolic, and less commonly ileo-ileocolic or ileo-ileal.

Intussusception is rare, except in infants aged between three and nine months. In most cases, it is thought that an inflamed Peyer's patch acts as the 'lead point' of the intussusception, in the terminal ileum; other pathology provides the lead point (e.g. neoplasm, polyp, Meckel's diverticulum, submucosal lipoma) in older children and adults. As with volvulus, strangulation frequently occurs at an early stage.

Clinical features of intestinal obstruction

The clinical presentation of intestinal obstruction depends upon the level, site and nature of the obstruction.

Symptoms: obstruction of the small intestine usually presents acutely with colicky, periumbilical abdominal pain. Vomiting is usually an early symptom in proximal obstruction of the small bowel, but may take longer to develop with more distal obstructions. The degree of abdominal distension varies greatly, depending on the site of obstruction; proximal obstruction results in little or no abdominal distension, while distal obstruction produces more significant abdominal distension. Absolute constipation is a very late sign in obstruction of the small bowel because colonic contents are emptied normally initially. Constant, severe pain of sudden onset is an ominous symptom and indicates strangulated or infarcted bowel. In contrast to mechanical obstruction, paralytic ileus is usually painless.

Obstruction of the large bowel usually presents with absolute constipation and marked abdominal distension; nausea and vomiting are late signs. Pain is not usually a predominant symptom but, when it occurs, tends to be in the lower abdomen. Constipation becomes the predominant symptom the more distal the obstruction, and is described as 'absolute' if neither flatus nor faeces are passed.

Physical signs of intestinal obstruction are dehydration, abdominal distension and 'obstructed' hyperperistaltic bowel sounds. Examination is initially directed at establishing the general condition of the patient. At presentation, the patient may show signs of dehydration, with a dry, furred tongue, sunken dull eyes, a characteristic foetor and decreased tissue turgor—all suggest loss of extracellular fluid. In simple obstruction, peripheral circulation is maintained until a late stage, when tachycardia, hypotension and cold, clammy peripheries indicate the onset of hypovolaemic shock.

Abdominal distension tends to be mainly central in obstruction of the small bowel because the dilated loops lie one above the other in a 'ladder' pattern. Distension is mainly in the flanks or in the upper abdomen in colonic obstruction.

Abdominal tenderness is not a feature of uncomplicated intestinal obstruction and its presence should alert the clinician to the possibility of impending strangulation. Abdominal masses suggest a neoplastic or inflammatory process. Hernial orifices (especially in the groins and periumbilical region) should be carefully examined because an unsuspected strangulated hernia is an important cause of obstruction of the small bowel. Obstructive bowel sounds are invariably present at some stage, but may disappear if the obstruction remains unrelieved or if strangulation develops.

Rectal examination confirms the presence or absence of faeces. A rectal tumour may be detected and extrarectal masses within the pelvis may also be palpable.

Investigations

Investigations are required to assess the general condition of the patient, confirm the diagnosis, and establish an underlying cause.

Blood tests: the haemoglobin concentration and packed cell volume will confirm dehydration. The white cell count is usually normal or slightly elevated, except in strangulation or perforation, when it is markedly raised.

A rising concentration of urea in blood may be due to simple dehydration, but may also indicate acute renal impairment if dehydration is severe or prolonged. Serum concentrations of sodium and chloride may fall with severe gastrointestinal electrolyte losses. Whole-body depletion of potassium is common, but hypokalaemia is not, and an abrupt rise of potassium levels in serum may indicate strangulation.

Metabolic derangements tend to be more severe with obstruction of the small bowel owing to the larger volumes lost with vomiting and small-bowel secretions that are sequestered in the lumen.

Imaging

Radiographs – a plain supine radiograph of the abdomen usually shows dilated, gas-filled loops of bowel, which may allow assessment of the likely level of obstruction.

- Dilated jejunum has valvulae conniventes (Figure 2), which are seen as parallel lines spanning the entire width of the bowel lumen.
- An obstructed ileum appears cylindrical and valvulae conniventes may be seen less clearly.
- Typically, the distended colon creates a 'picture-frame' outline of the abdominal cavity. The colon has haustral markings, which do not traverse the entire diameter of the lumen. The bowel is collapsed and the rectum does not contain gas below the level of the obstruction.

Rarely, the plain radiograph shows features that can allow a specific diagnosis to be made (e.g. calcified gallstone, swallowed foreign body). An erect radiograph of the chest or a lateral decubitus radiograph of the abdomen may show free intraperitoneal gas if obstruction has been complicated by perforation.

Contrast studies can be helpful, and a water-soluble contrast meal can assist in the management of complex obstruction of the small bowel. This may be particularly important in cases of adhesive obstruction, where passage of contrast to the caecum by four hours strongly suggests that the obstruction will settle spontaneously and that conservative management of an otherwise stable patient is appropriate.

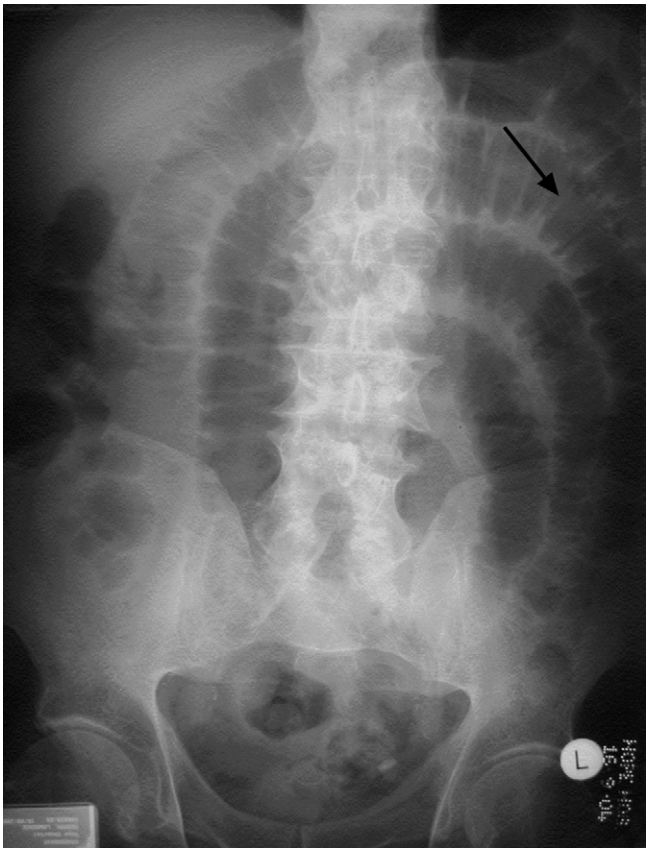

Figure 2 Plain radiograph of the abdomen showing obstruction of the small bowel, with dilated jejunum and valvulae conniventes (arrow).

Many surgeons in the UK regard a contrast enema mandatory in the management of obstruction of the large bowel. This investigation distinguishes between a mechanical cause and pseudo-obstruction and, in the former, may identify the site of obstruction and underlying pathology. Water-soluble contrast is sometimes used instead of barium, which could cause an intense peritonitis if an unexpected perforation occurred.

Management

The principles of the management of intestinal obstruction are provision of analgesia, intestinal decompression, intravenous administration of fluid and electrolytes and, if appropriate, surgery. Up to 75% of patients with adhesive obstruction of the small bowel will settle with conservative treatment, so it is attempted initially in all patients without overt signs of strangulation. Indications for surgery for adhesive obstruction of the small bowel may be facilitated by contrast radiology (see above).

However, it is unusual for obstruction of the small bowel to resolve without surgery if it has not done so within 48 hours of conservative management.

Decompression: a nasogastric tube should be inserted to decompress the proximal bowel, and to better estimate fluid and electrolyte requirements. Decompression of the stomach is essential

to reduce the substantial chance of aspiration (e.g. during anaesthesia).

Replacement of loss of gastrointestinal fluid: intravenous cannulation should be instituted immediately and fluids (i.v.) continued until daily requirements of fluid and electrolytes can be met orally. The amount of fluids and electrolytes required for satisfactory fluid and electrolyte balance may vary considerably, depending on the level and duration of the obstruction. Hartmann's solution or 0.9% saline with 20 mmol potassium/l is used to replace the loss of sodium, chloride, water, and potassium. Nasogastric losses should be replaced litre-by-litre with intravenous saline and potassium. Great care must be taken not to precipitate cardiac failure or severe imbalance of other electrolytes (notably serum magnesium) in the elderly and frail.

Urine output is an invaluable guide to hydration status, and a urinary catheter should be inserted in all patients with significant dehydration; measurement of central venous pressure may be of value in those with heart failure or those in shock (where assessment of hydration status can be difficult). Twice-daily (or even more frequent) estimations of urea and electrolytes in plasma may be needed during rehydration, and estimation of requirements may be facilitated by measurement of urinary excretion of sodium. Patients with severe dehydration or strangulation may require admission to a HDU, resuscitation with high-flow oxygen, invasive monitoring and, in the case of strangulation, antibiotic therapy (i.v.) before laparotomy.

Surgery is indicated if there:
- is a primary underlying disease process that must be treated (e.g. a hernia, obstructing carcinoma)
- is failure of adhesive obstruction to settle
- are signs of peritoneal irritation.

Patients with small intestinal obstruction and nothing to suggest adhesions (e.g. no previous abdominal or pelvic surgery) require early laparotomy. Colonic obstruction can usually be investigated with an urgent colonic contrast study in the absence of closed-loop obstruction.

Surgery should be undertaken when the patient has been resuscitated as fully as possible, but this may require careful judgement (e.g. in the presence of strangulation when it may not be possible to resuscitate the patient completely until gangrenous bowel has been resected). The incision should afford direct access to the expected site of obstruction and the standard approach is a midline laparotomy (though previous surgical incisions may influence this).

The surgical procedure will depend upon the cause of obstruction. Obstructed intestine must be handled very gently and adhesions divided carefully because postoperative morbidity and mortality are increased considerably if bowel is inadvertently injured. Ideally, obstructing tumours should be resected, but occasionally this is not possible and a bypass procedure may be required. Obstructing foreign bodies or gallstones can usually be 'milked' cephalad and removed with ease through a transverse, proximally-placed enterotomy made in healthy bowel.

The surgeon must decide if a strangulated loop of bowel is viable once the obstruction has been relieved. It may be helpful to wrap the loop of affected bowel in a warm, saline-soaked pack and leave it for several minutes. The pack is removed and the bowel inspected, and is considered viable if there is:
- sheen remaining on the serosal surface
- return of normal pink colour
- clear peristalsis
- pulsation in mesenteric vessels.

The bowel is not viable if:
- the sheen is lost
- it has become purple, dark green or black in colour
- it emits an odour
- does not transmit peristalsis
- there are thrombosed mesenteric vessels.

Under these circumstances, the bowel should be resected and an anastomosis between healthy and viable bowel ends performed or the ends exteriorized, depending upon the patient's condition and the level of bowel resected.

In colonic obstruction, the initial aim is to decompress the obstructed segment in order to prevent perforation. In mechanical obstruction, this almost always requires surgery. The obstructing lesion should be removed and continuity of the large bowel restored (if possible). For tumours or other obstructing lesions of the right colon, a one-stage right hemicolectomy with an ileotransverse anastomosis usually achieves both objectives (as long as the patient is fit to withstand the procedure, and the obstructing lesion is resectable). In the presence of perforation and gross contamination, an anastomosis is not advisable and the proximal bowel should be brought onto the surface as an

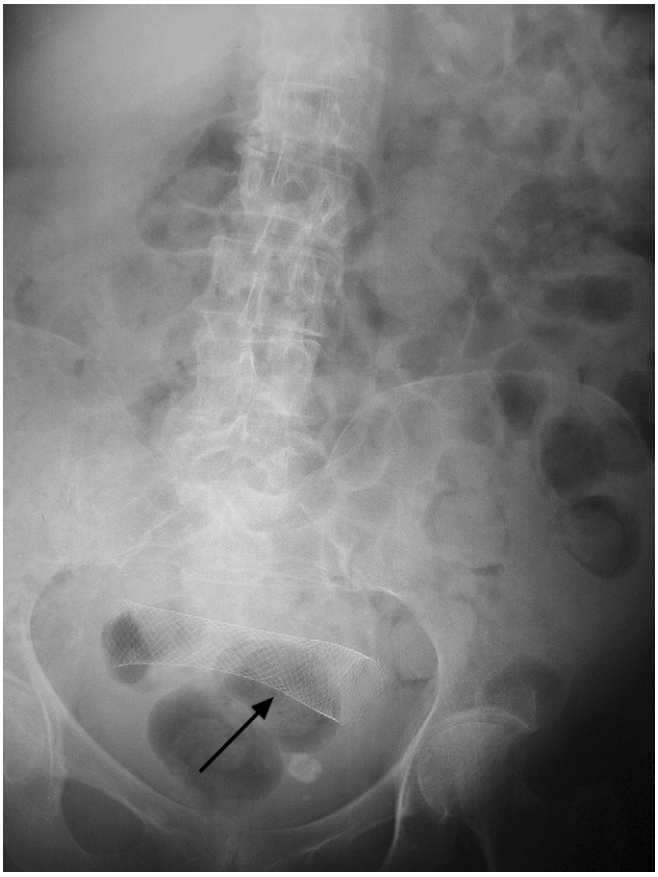

Figure 3 Plain radiograph of the abdomen showing a rectosigmoid colonic stent *in situ* (arrow).

end ileostomy. The continuity of the bowel can then be restored when the patient has fully recovered.

The management of obstructing lesions of the left colon is more controversial. There are two main approaches:

- resection of the tumour with a temporary end colostomy (a second operation is required a few months later for colorectal reanastomosis)
- resection with primary anastomosis and on-table colonic irrigation.

Proponents of a one-stage approach argue that the combined overall mortality rate of a staged approach is greater than that of a single procedure. This challenges the view that resection and primary anastomosis in the presence of an obstructed and unprepared bowel are associated with an increased risk of anastomotic dehiscence. In many cases, the anastomosis is protected by a loop ileostomy or a tube caecostomy to allow decompression in the early postoperative period.

The surgical management of obstruction of the left colon is beyond the scope of this review. However, a substantial proportion of patients never have intestinal continuity restored after creation of a 'temporary' end ileostomy and highly experienced colorectal surgeons are more likely to undertake a one-stage procedure.

Medical management: management of mechanical obstruction may be possible without surgical exploration under certain circumstances. Sigmoid volvulus can be decompressed with a colonoscope and/or flatus tube, while hydrostatic reduction of intussusception is often possible with a contrast enema. For obstructing colonic lesions where the patient is unfit for surgery or where palliation is the objective, an expandable metal stent may be placed across the obstruction radiologically or endoscopically (Figure 3).

Management of paralytic ileus and pseudo-obstruction – acute disorders of intestinal motility are usually a reflection of other, coexisting serious illnesses. In general, if a mechanical cause of intestinal obstruction has been excluded, these conditions should be treated conservatively, with replacement of fluid and electrolytes and nasogastric decompression. Attention should be focused on the underlying cause which may require treatment (e.g. diabetic ketoacidosis). Cholinergic stimulants (e.g. neostigmine) together with adrenergic blockers are rarely used to restore intestinal motility; colonic decompression with a colonoscope and/or flatus tube may be appropriate. Surgery is occasionally undertaken in colonic obstruction if caecal tenderness develops due to progressive caecal dilation; a tube caecostomy may be required until colonic motility has recovered.

Prognosis

The mortality associated with acute, uncomplicated obstruction of the small bowel may be as high as 10% in the UK; the mortality associated with strangulated obstruction is much greater. The mortality associated with colonic obstruction is about 30%, and increases significantly if the caecum has perforated. The prognosis is worse still in elderly patients, especially if they are in poor general health. ◆

