

INTRODUCTION

The small intestine is a specialized absorptive hollow conduit between the pylorus of the stomach and the caecum of the large intestine. It is divided into 3 sections: duodenum, jejunum and ileum based on anatomical and physiological considerations. This chapter aims to cover the basic anatomy and physiology as well as cover in greater depth the relevant surgical pathologies of the small intestine.

ANATOMY

Embryology

In order to understand the anatomy it is relevant to mention the basic embryology. The small intestine (other than the first part of the duodenum) develops as part of the embryological midgut structures. The midgut develops around the superior mesenteric artery which supplies the small intestine. The small intestine spends part of foetal life herniated outside the abdomen, rotates around the axis of the artery and returns to the abdominal cavity. During this time it is attached to the yolk sac by the yolk stalk. Some surgical pathology, relevant to the small intestine, (e.g. umbilical hernia, intestinal malrotation, Meckel's diverticulum) has its origins in abnormalities of this early developmental process.

Macroscopic anatomy

The small intestine begins with the duodenal C-loop as it encircles the head of the pancreas. The duodenum is the shortest part of the small intestine (25cm) and is divided into four sections: (see fig 1: illustration of duodenal C-loop configuration) superior(1), descending(2), horizontal(3) and ascending(4). It is

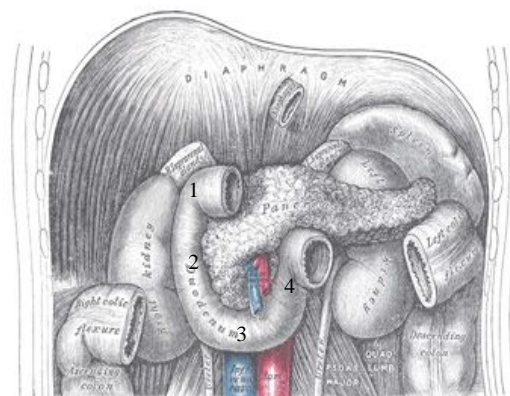


Figure 1: Illustration of duodenal C-loop configuration

the widest and only portion of the small intestine that is partially retroperitoneal. It does not have a mesentery but is fixed to the posterior abdominal wall and demarcated at its end by a peritoneal fold - the ligament of Treitz. The bile and pancreatic ducts enter and discharge their contents into the descending part of the duodenum via the ampulla of Vater on the posteromedial wall.

In adult patients, the jejunum and ileum are around 5 metres long and make up the remainder of the small intestine by a 2/5th (jejunum) to 3/5th (ileum) ratio. They are suspended from the posterior abdominal wall by a mesentery and are not as clearly demarcated as the duodenum. There is a more gradual transition from the jejunum's larger thicker walled circumference to the ileum's smaller circumference and thinner wall. There is also a gradual change in the mesenteric vascular arcade configuration.

The arterial blood supply to the small intestine (except the first part of the duodenum) is via the superior mesenteric artery. Venous drainage is via the superior mesenteric vein which joins the splenic vein to form the portal vein. The nerve supply is both sympathetic from splanchnic nerves and parasympathetic via the vagus. It

is insensitive to most painful stimuli but sensitive to distension. Lymphatic drainage as a general rule follows the arteries.

Microscopic anatomy

In order to allow effective absorption and digestion, the wall of the small intestine is made up of 4 layers: mucosa, submucosa, muscularis and serosa. The mucosa has an incredibly large absorptive surface area multiplied by circular mucosal folds termed plicae circulares (the valvulae conniventes seen on abdominal x-ray). These folds are more numerous in the jejunum. The surface area is further increased by multiple villi and microvilli that cover the plicae circulares surface.

PHYSIOLOGY

The small intestine's primary function is digestion and absorption of carbohydrates, fats, protein, vitamins, water and ions. The duodenum accepts the high acid contents from the stomach as well as exocrine secretions from the liver and pancreas. This intestinal chyme within the lumen is propelled forward by peristaltic waves of contractions from the duodenum into the jejunum which has a primarily absorptive role. The intestinal contents continue into the ileum which has absorptive function as well as being concerned with the enterohepatic circulation of bile salts and absorption of the vitamin B12-intrinsic factor complex. The small intestine plays a significant endocrine role through the secretion of multiple hormones (eg. gastrin, cholecystokinin, secretin, motilin, vasoactive intestinal polypeptide) and has a part in immune function through the ileal lymphocyte aggregates known as Peyer's patches.

PATHOLOGY

Small bowel obstruction

Small bowel obstruction (SBO) is the inability of intestinal contents to progress along its normal intestinal course. Mechanical obstruction (of particular interest to the surgeon) of the small intestine needs to be differentiated from non-mechanical/paralytic ileus which is generally treated medically. Bowel obstruction may be **partial**, with the ability to pass gas and/or some liquid, or **total** with complete obstipation. Correctly identifying the patient with mechanical small bowel obstruction and in particular the patients that will benefit from surgery is crucial to avoiding delays that may significantly increase patient morbidity and mortality.

Aetiology

SBO, in contrast to large bowel obstruction, is more often caused by benign rather than malignant lesions. The aetiology of SBO is varied and more easily recalled if categorized as lesions occurring extrinsic to the bowel wall, in the bowel wall and intraluminal. (see table 1: causes of small bowel obstruction) By far the majority of cases however are caused by **adhesions** and **hernias**. It is likely that as the rate of elective hernia repair and general surgery increases in developed countries, the more often adhesions rather than irreducible hernias emerge as the top cause. Neoplastic disease as a cause of obstruction is likely to be metastatic peritoneal disease rather than a small bowel primary tumour. Primary neoplasms occurring in the small bowel wall are rare but may be a lead point in intussusception. Intussusception (see fig. 2: intussusception) is telescoping of one part of the intestine into itself. It is more commonly seen in children presenting with the classical colicky abdominal pain, a palpable abdominal mass and bleeding per rectum. When found in adults there is usually a pathological lead point i.e. tumour but in children usually no obvious lead

Causes of small bowel obstruction		
Extrinsic	Adhesions	
	Hernia <ul style="list-style-type: none"> • External eg. inguinal, umbilical • Internal eg. diaphragmatic 	
	Neoplastic	
	Intra-abdominal sepsis/abscess <ul style="list-style-type: none"> • Ruptured appendix/diverticulum 	
Wall	Congenital <ul style="list-style-type: none"> • Malrotation • Cystic fibrosis • Meckel's diverticulum 	
	Inflammatory <ul style="list-style-type: none"> • Crohn's disease 	
	Infectious <ul style="list-style-type: none"> • Tuberculosis • Actinomycosis 	
	Traumatic <ul style="list-style-type: none"> • Haematoma • Ischaemic stricture 	
	Neoplastic <ul style="list-style-type: none"> • Primary • Metastatic 	
	Other <ul style="list-style-type: none"> • Intussusception • Endometriosis • Radiation stricture 	
	Lumen	Gallstone
		Bezoar
		Foreign body
		Enterolith

Table 1: Causes of small bowel obstruction



Figure 2: Intussusception

point is found. Paediatric patients may also develop obstruction due to volvulus of the small intestine caused by congenital malrotation whereby failure of fixation of the intestine to the posterior abdominal wall occurs. The midgut segment (see embryology) is prone to volvulus around the narrow pedicle containing the superior mesenteric artery.

Strictures of the bowel wall may be caused by inflammatory conditions such as Crohn's disease, radiation injury and infectious causes (eg tuberculosis). Matted groups of lymph nodes may also cause obstruction in patients with abdominal tuberculosis. Crohn's disease is treated medically but can require surgery for complications (e.g. bowel obstruction, fistulae).

Haematomas may occur in the bowel wall in trauma patients or may occur spontaneously in patients taking anticoagulants.

Gallstones may cause obstruction by entering the small intestine through a cholecystenteric fistula and usually lodge in the terminal ileum, unable to pass the ileocaecal valve.

Pathophysiology

Once an obstruction occurs in the small bowel, peristalsis increases in an attempt to push intestinal contents past the obstruction. As this fails, peristaltic waves eventually decrease and the small intestine expands. It distends with gas and fluid proximal to the obstruction. The gas accumulates due to increased bacterial proliferation and swallowed air (nitrogen is poorly absorbed). There is increasing oedema of the bowel wall and transport of fluid and electrolytes is impaired. Dehydration and hypovolaemia occur due to extensive fluid sequestration. Vomiting also accentuates the fluid deficit. Intraluminal pressure rises and venous return in the bowel wall is impaired. Eventually there is decreased arterial blood flow and gangrenous perforation

may occur. Other organs may become compromised as the patient enters septic shock. Abdominal distension elevates and splints the diaphragm and impairs respiration.

Diagnosis

The diagnosis of mechanical SBO is based on an appropriate and comprehensive evaluation of the history and examination of the patient with SBO. It is confirmed by plain abdominal x-ray. The clinical presentation varies according to the level and duration of the obstruction.

History

The patient's history is an important part of diagnosing SBO and helps to convey clues to the underlying cause of the obstruction. The following are all relevant to the diagnosis:

- previous surgery
- hernias that are now irreducible
- malignancy or severe loss of weight
- inflammatory bowel disease
- previous irradiation
- prior episodes of SBO

Symptoms of SBO may vary according to the site of the obstruction and include the following:

- abdominal discomfort/colicky pain
- abdominal distension
- nausea and bile stained vomiting
- obstipation (may have initial diarrhoea)

Nausea and vomiting are more likely in patients with more proximal obstruction. Patients presenting with distal SBO and presenting later in the course of obstruction may have faeculent vomiting.

Clinical

Vital signs may initially be normal then deteriorate with tachycardia, hypotension and pyrexia. Localised tenderness and pyrexia are indicative

of perforation. During abdominal examination look for the following:

- gaseous distension (may be absent in higher obstruction)
- previous surgical scars (adhesions)
- early vigorous peristalsis (later silent)
- masses (peritoneal metastases, tuberculous mass)
- signs indicating possible malignancy: periumbilical mass (Sister Mary Joseph nodule), right supraclavicular (Virchow) node, hepatosplenomegaly
- localised rebound, tenderness and guarding suggest perforation
- examine hernia sites for irreducibility
- blood on rectal examination (intussusception/infarction) or masses

Investigations

Plain abdominal x-rays showing dilated loops of small bowel (>3cm in diameter) and the absence of dilated large bowel usually clinch the diagnosis of SBO but do not necessarily identify the cause. An erect abdominal x-ray will reveal multiple air-fluid levels in a step-like pattern. (The presence of up to two air-fluid levels is considered normal.) Small bowel is generally identifiable occupying the central portion of the abdominal x-ray with the distinctive valvulae conniventes traversing the entire diameter of the bowel. The large bowel is peripheral with haustral markings that extend only partially across the bowel. The x-ray may also detect foreign bodies, a gallstone in the small intestine or air in biliary tree (suggesting cholecystenteric fistula). Any free air on the abdominal x-ray indicates perforation and necessitates urgent surgery. Oral contrast media can confirm the presence of total obstruction.

Complex small bowel obstruction (multiple recurrent obstructions, severe underlying medical problems, radiation enteritis, partial small bowel obstruction) may require additional studies to pinpoint the diagnosis. Computerised tomography may be helpful in diagnosing carcinoma, other extrinsic lesions (e.g. intraabdominal abscess) and more accurately determining the level of obstruction. It will also detect irreversible necrotic bowel.

Measuring the serum electrolytes does not help diagnose bowel obstruction but will help identify the extent of dehydration. The white cell count may or may not be elevated and haemoconcentration may be present. Acidosis and a raised lactate are not conclusive but are indicators of bowel necrosis.

There are however, no investigations that conclusively exclude presence of ischaemic bowel and the decision to operate relies on a thorough assessment of the patient and their evolving condition.

Treatment

Treatment of the patient with suspected SBO must begin immediately on presentation. Aggressive early fluid resuscitation with appropriate replacement of electrolytes is vital before any definitive surgery can be contemplated. Immediate intravenous access is necessary for both fluid replacement and antibiotics if perforation is suspected. Patients must be kept nil per mouth with a nasogastric tube and urinary catheter placed. The nasogastric tube drains the stomach contents of both liquid and gas and helps decrease further vomiting and bowel distension. Non-mechanical bowel obstruction must be excluded as a differential diagnosis. Unless recurrent adhesive obstruction, post operative obstruction, Crohn's disease or abdominal carcinomatosis

is present it is likely that urgent surgery will be required after aggressive resuscitation.

Adhesive bowel obstruction is managed conservatively by 'drip and suck' (intravenous line and nasogastric tube) as long as there is no suspected perforation. If a patient shows no signs of improvement or deteriorates then surgery for adhesiolysis may be required. This is usually done via open laparotomy but in select cases may be done laparoscopically. All patients requiring surgery for bowel obstruction must be adequately consented and must be informed of the possibilities of an alternative pathology (eg carcinoma) and the possibility of a stoma. Ideally all patients undergoing laparotomy for bowel obstruction should be seen and counselled by a trained stoma therapist and the appropriate stoma sites marked before surgery. At surgery adhesions are released, any non-viable bowel is resected and anastomoses or stomas made as required.

Hernias are usually reduced by the standard hernia incision depending on the site (i.e. inguinal, umbilical). The bowel must be adequately assessed for viability before being reduced.

Foreign bodies require a laparotomy and an enterotomy (incision through the bowel wall) to remove.

Small bowel perforation

Small bowel perforation may occur secondarily to bowel obstruction but may also occur spontaneously in other conditions. (see table 2: causes of spontaneous small bowel perforation) Immunocompromised patients may present a particularly challenging diagnostic problem. Patients with HIV may present with perforation but few clinical signs other than general malaise. Their immune system may be too compromised to mount an appropriate response. They are high risk surgical candidates but can do well with urgent surgery. Consider

particularly typhoid, tuberculosis and cytomegalovirus in these patients.

Causes of spontaneous small intestinal perforation
Tuberculosis
Typhoid
Cytomegalovirus
Malignancy
Crohn's disease
Steroids
Radiotherapy

Table 2 Causes of spontaneous small intestinal perforation

Short bowel syndrome

Patients with short bowel syndrome present after extensive small bowel resection with incapacitating diarrhoea, steatorrhoea and malnutrition. The clinical expression is variable depending on the extent and site of resection. It is inevitable in patients with 100cm or less of small intestine remaining as there is not enough absorptive area left to maintain a healthy nutritional status. Most of these patients will require lifelong total parenteral nutrition with small bowel transplant being the only other alternative for a select few in highly specialized units in the developed world.

Small intestinal haemorrhage

Other than duodenal ulcers, bleeding from the small intestine is fairly uncommon. Patients presenting with blood or malaena per rectum are appropriately investigated for peptic ulcer disease and colorectal pathology before small intestinal causes are considered.

Small intestinal haemorrhage may be due to vascular abnormalities of the mucosal wall, small bowel Crohn's disease or an ileal diverticulum of Meckel. Meckel's diverticulum presents more commonly in children. It

occurs in 1-2% of the general population and is more often found to be an incidental finding at surgery than the cause for concern. It is a congenital remnant of the proximal part of the embryonic yolk stalk and consists of a pouch projecting from the antimesenteric border of the small intestine (see fig. 3: Ileal diverticulum of Meckel). It is usually located around 40cm from the ileocaecal junction. It may be free or less commonly attached by a cord to the umbilicus. It more commonly presents with bleeding but may clinically mimic appendicitis or present with obstruction.

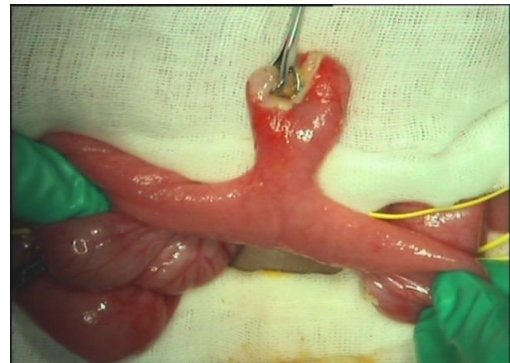


Figure 3: Ileal diverticulum of Meckel

Small bowel neoplasms

Benign (gastrointestinal stromal tumours (GIST's), adenomas, lipomas) and malignant (adenocarcinomas, carcinoids, lymphoma) are particularly rare. Metastatic neoplasms (e.g. melanoma, renal, breast) affecting the small intestine are more common than primary tumours. Malignant primary neoplasms are more likely to present with symptoms of SBO, bleeding or perforation than benign lesions.



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