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ANATOMY OF THE SMALL INTESTINE

ANATOMY OF THE SMALL INTESTINE

Although the duodenum is anatomically indistinguishable from the rest of the small intestine, in surgical terms it may be regarded as a distinct structure and is discussed in Chapter 67 The small intestine lies between the duodenojejunal (DJ) flexure and the ileocaecal valve (Figure 74.1). It is di ffi cult to establish the length of the small intestine. It varies widely between subjects (it is said to be longer in men) and estimates gathered at surgery , at postmortem and during radiological investigations have been noted to vary widely , even in the same individual. Most studies however describe a range between 300 and 850 /uni00A0 cm. The proximal 40% of the small intestine is referred to as the jejunum and the remainder is the ileum. There is no clear demarcation between jejunum and ileum, as the character of the small intestine changes gradually from proximal to distal. The jejunum tends to hav e a wider diameter and a thicker wall, with more prominent mucosal folds (valvulae conniventes), while the ileum has a thicker, more fatty mesentery with more complex arterial arcades. The ileum also contains larger aggre gates of lymph nodes (Peyer’s patches), which can occasionally become the lead points in childhood intussusception. The small intestine has a rich blood supply , derived from the superior mesenteric artery (SMA), while venous drainage is via the portal venous system. The superior mesenteric vein joins the splenic vein to form the portal vein, w hich drains into the liver, carrying absorbed nutrients from the bowel for pro cessing. The lymphatic drainage of the small intestine follows the arterial supply . The small intestine has a rich autonomic innervation aris ing from the splanchnic nerves, which contribute a dense net Valvulae conniventes describes a fold of mucous membrane that passes across two-thirds of the bowel circumference. Johann Conrad Peyer , 1653–1712, Professor of Logic, Rhetoric and Medicine, Scha ff hausen, Switzerland, described the lymph follicles in the intestine in 1677. work of sympathetic fibr es around the SMA and its branches. Referred pain from the small intestine is usually felt in the peri - umbilical region (T10). The blood and nerve supplies to the small intestine run in its mesentery , which is attached to the posterior abdominal wall and runs obliquely downwar ds to the . right between the DJ flexure to the left of the second lumbar vertebra and the right sacroiliac joint (Summary box 74.1 ; see Chapter 65). Summary box 74.1 Important features of small bowel anatomy /uni25CF /uni25CF /uni25CF

The importance of non-surgical management of small • The principles of small intestinal surgery • That complex intestinal problems are best managed by a • Comprises jejunum and ileum Has valvulae conniventes Blood supply from SMA

Bacterial overgrowth

Bacterial overgrowth

The small intestine can become colonised with bacteria normally confined to the colon if there is stasis resulting in delayed bacterial clearance (blind loop syndrome; 74.9). Similar complications may result from chronic small bowel obstruction, jejunal diverticulosis and ileocolic fistu lation. Overgrowth in the upper small intestine results in fat malabsorption due to the deconjugation of bile salts, while vitamin B12 deficiency results from overgrowth more distally . Ther e is usually relatively little e ff ect on carbohydrate or protein metabolism. If steatorrhoea occurs, other serious malabsorption features may follow , including glossitis, osteo malacia, paraesthesia and peripheral neuropathy . Eugen (Jeno) Alexander Pólya , 1876–1944, surgeon, St Stephen’s Hospital, Budapest, Hungary . Improvement normally follows after intermittent therapy with oral antibiotics; metronidazole, ciprofloxacin, tetracycline and rifaximin are commonly used. Definitive treatment is sur - gical if the anatomical abnormality can be cor rected, but this is not always possible. -

(f) (b) self-emptying: no de /f_i ciency occurs; (c) long afferent loop stasis (f) ‘stenosis–anastomosis loop’ syndrome.

Benign

Benign

The majority of small bowel neoplasms are benign, comprising adenomas, lipomas, haemangiomas and neurogenic tumours. They are frequently asymptomatic and identified incidentally , James Israel , 1848–1926, first found sulphur granules in pus from a discharging sinus in a man’s neck. Later, he became a famous Berlin urologist. John Law Augustine Peutz , 1886–1968, Chief Specialist for Internal Medicine, St. John’s Hospital, The Hague, The Netherlands. Harold Joseph Jeghers , 1904–1990, Professor of Internal Medicine, The New Jersey College of Medicine and Dentistry , Jersey City , NJ, USA.). - but can present with intussusception, small bowel obstruction and bleeding that may cause anaemia or may even be overt. Where these lesions do cause anaemia, the cause can be di ffi cult to diagnose, as CT or small bowel contrast studies do not show them easily . Capsule endoscopy or small bowel endoscopy have been used successfully where the facilities exist. Symptomatic lesions can be treated by small bowel resection. Peutz–Jeghers syndrome This is an autosomal dominant condition characterised by melanosis of the mouth and lips, with multiple hamartoma - tous (benign tumour-like malformations resulting from faulty development in an organ) polyps in the small bowel and colon (Figure 74.3). Melanin spots can also occur on the digits and perianal skin. Mutation of the STK11 gene on chromosome 19 has been found in a proportion of patients. Long-term follow-up of the original family described by Peutz has shown reduced survival as a consequence of complications of bowel obstruction and the development of a range of cancers. Regular colonic surveillance should be performed and female patients should attend breast and cervical screening. Despite the increased risk of malignancy in general, malignant change in the polyps themselves is uncommon. Resection may be indi - cated, however, for heavy and persistent or recurrent bleeding or intussusception. Polyps may be removed by enterotomy or, at laparotomy , snared via a colonoscope introduced via an enterotomy . Heavily involved segments of small intestine may occasionally be resected.

Figure 74.3 Melanin spots on the lips of a patient with Peutz–Jeghers syndrome (courtesy of Major PCM Manta, Indian Medical Service).

Small bowel malignancy is rare, classically presents late and is most often diagnosed after surgery for small bowel obstruction. Four types will be considered, which account for over 99% of small bowel malignancies: adenocarcinoma, neuroendocrine tumours (NETs), lymphomas and gastrointestinal stromal tumours (GISTs). Adenocarcinoma Small bowel adenocarcinoma is more often found in the jeju num than in the ileum and, although the aetiology is unknown, it is more common in patients with CD, coeliac disease, familial adenomatous polyposis, hereditary non-polyposis colon cancer and Peutz–Jeghers syndrome. The tumours present with anaemia, overt g astrointestinal bleeding, intussusception or obstruction. Prognosis is poor, particularly in patients with CD, in whom these tumours often present late because the symptoms are commonly mistaken for those of CD and treated conservatively . When diagnosed, surgical treatment is a resec tion of 5 /uni00A0 cm of non-involved bowel either side of the lesion and the a ff ected mesentery (Figure

74.4). A right hemicolectomy is likely to be required for tumours of the distal ileum. Neuroendocrine tumours NETs (previously known as carcinoid tumours) occur through out the gastrointestinal tract, most commonly in the appendix, ileum and rectum in decreasing order of frequency . They arise from Kulchitsky cells at the base of intestinal crypts (of Lieberkuhn). The primary is usually small, although significant lymph node metastases can occur. In up to one-thir d of cases of small bowel NETs, the tumours are multiple. They may produce dense fibrosis in the surrounding tissues, resulting in distortion and scarring of the bowel and associated mesentery , giving them a characteristic radiological appearance. NETs can produce a number of vasoactive peptides, most commonly 5-hydroxytryptamine (serotonin), but also histamine, prosta glandins and kallikrein. When they metastasise to the liver, the ‘carcinoid syndrome’ can become evident because the vasoactive substances escape the filtering actions of the liver. The clinical syndrome itself consists of reddish-blue cyanosis, flushing attacks, diar rhoea, borborygmi, asthmatic attacks and, eventually , pulmonary and tricuspid stenosis (Summary box 74.3). Classically , the flushing attacks are induced by alcohol. Surgical resection is usually su ffi cient for patients with pri mary disease, but the incidence of recurrence is significant. Summary box 74.3 Carcinoid syndrome /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Nikolai Kulchitsky , 1856–1925, Professor of Histology , Kharkov , Ukraine, who left Russia after the Revolution of 1917 and later worked at University College, London, UK. He described these cells in 1897. Johann Nathaniel Lieberkühn , 1711–1756, physician and anatomist, Berlin, Germany , described these glands in 1745. Thomas Hodgkin , 1798–1866, lecturer in morbid anatomy and curator of the museum, Guy’s Hospital, London, UK, described lymphadenoma in 1832. Denis Parsons Burkitt , 1911–1993, Irish-born surgeon who worked in Kampala, Uganda. - The extent of disease can be assessed preoperatively using oct - reotide scanning (somatostatin receptor scintigraphy), which - may detect otherwise clinically unapparent primary and sec - ondary tumours. Plasma markers of tumour bulk, such as chromogranin A concentrations, may be useful mar kers of disease recurrence, as well as of prognostic value. tic resection can be carried out in patients with meta - Hepa static disease. The treatment has been transformed by the use - of octreotide (a somatostatin analogue), which reduces both flushing and diarrhoea, and octreotide cover is usually used in tients with a carcinoid syndrome who have surgery to prevent pa a carcinoid crisis resulting from liberation of vasoactive sub - stances following handling of the tumour. NETs generally grow more slowly than most metastatic malignancies and patients may live with metastatic disease for many years. The tumour is not usually sensitive to chemo- or radiotherapy (see Chapter 69). Lymphoma Small bowel lymphoma may be primary or, more commonly , - secondary to systemic lymphoma. The incidence of small bowel lymphoma is increased in patients with CD and immunodeficiency syndromes. The classification of lymphoma is beyond the scope of this chapter but a number of points are worth noting briefly . It is rare for Hodgkin’s lymphoma to a ff ect the small bowel and most Western-type lymphomas are non-Hodgkin’s B-cell lymphomas. They usually present with anaemia, bleeding, perforation, anorexia and weight loss. - Small bowel T-cell lymphoma can develop in patients with coeliac disease. It usually presents with worsening of local the patient’s diarrhoea, pyrexia of unknown origin and obstructive symptoms. Mediterranean lymphoma is found mostly in North Africa and the Middle East and is often wide - spread at diagnosis. Burkitt’s lymphoma can aggressively a ff ect the ileocaecal region, particularly in children. The mainstay of treatment for these conditions is chemotherapy; however, sur - equired for obstruction, perforation or bleeding. gery may be r

Diarrhoea Palpitations Bronchospasm Tricuspid regurgitation Facial/upper chest /f_l ushing Figure 74.4 Small bowel adenocarcinoma.

GISTs are mesenchymal tumours and the distinction between benign and malignant types is di ffi cult even on histological examination. Increased size and high levels of c-kit (C D 1 1 7) staining are associated with malignant potential. GISTs are found most commonly in the stomach but can be found in other parts of the gut. They occur most commonly in the 50- to 70-year age group. Although the cause is unknown, patients with neurofibromatosis have an increased risk of developing these types of tumour. Patients may be asymptomatic and the tumour may present as an incidental mass on a CT scan. Symptoms include lethargy , pain, nausea, haematemesis or melaena. Surgery is the most e ff ective way of treating GISTs as the tumour is radioresistant and is not sensitive to conventional chemotherapy. Imatinib is a tyrosine kinase inhibitor that has been shown to be e ff ective in advanced cases and may also have a role in adjuvant treatment. It may be used preoperatively to reduce tumour size; however, the involuting tumour may perfo rate, precipitating a surgical crisis (see Chapters 11 and /uni00A0 67

CONDITIONS CAUSING MALABSORPTION Coeliac disease

CONDITIONS CAUSING MALABSORPTION Coeliac disease

Coeliac disease is the most common cause of malabsorption in the UK with a reported prevalence of 1:1800, although this may be an underestimate. It is characterised by a hyper- trophic small bowel mucosa with atrophic villi and deep crypts. Loss of surface area and brush border enzymes results in malabsorption. Coeliac disease is caused by an abnormal immune response to gluten, a cereal protein, although the exact mechanism remains unclear. There is a genetic component, as the disease is more common in first-degree relatives and has an associa - hildren, coeliac disease presents with tion with HLA-B8. In c steatorrhoea and growth retardation. In adults, it may result in diarrhoea and weight loss but many patients simply present tients develop a charac - with iron deficiency anaemia. Some pa teristic skin rash (dermatitis herpetiformis). The diagnosis is usually made after an endoscopic duo - denal biopsy allows pathological examination of the mucosa. A /uni00A0 blood test for immunoglobulin A anti-tissue transglutaminase (IgA tTGA) is relatively sensitive and specific for diagnosing - coeliac disease, making it the preferred test for detection. Mea surement of IgA antiendomysial antibodies (anti-EMA) should be used as a confirmatory test. A duodenal biopsy is usually - indicated to confirm the diagnosis. The biopsy usually shows flattening of the mucosa, marked inflammatory changes and characteristic findings of intraepithelial lymphocytes. All tests should be performed while the patient is on a gluten- containing diet as false-negative tests may occur if on a gluten- free diet. Patients with coeliac disease may develop an acute inflam matory condition of the small intestine (ulcerative jejunoileitis) and have an increased risk of small bowel lymphoma and ade nocarcinoma. T he main treatment for coeliac disease is the withdrawal of gluten from the diet by avoiding wheat, rye and barley . Sur gery does not usually play a role in the management of disease and is primarily reserved for resection of malignancy .

(d) (e) Figure 74.9 Common types of blind loop: (a) self- /f_i lling: de /f_i ciency occurs; in Pólya gastrectomy; (d) jejunal diverticula; (e) intestinal stricture causing stasis;

CONNECTIVE TISSUE DISORDERS Intestinal diverticula

CONNECTIVE TISSUE DISORDERS Intestinal diverticula

Diverticula (hollow outpouchings) are a common structural abnormality that can occur from the oesophagus to the recto sigmoid junction. Small bowel diverticula may be congenital or acquired. In congenital diverticula all three coats of the bowel are present in the wall of the diverticulum (e.g. Meckel’s diverticulum). Acquired diverticula These often develop in the jejunum and arise from the mesen teric side of the bowel as a result of mucosal herniation at the point of entry of the blood vessels, where there is a potential defect in the muscularis layer. Jejunal diverticula can vary in siz and are frequently multiple. They are commonly asymptomatic and present as an incidental finding at surgery or on radio logical imaging. However, they can result in malabsorption, due to bacterial overgrowth, or present as an acute abdominal emergency if they become inflamed or perforate. Bleeding from a jejunal diverticulum is a rare complication (compar with sigmoid diverticular disease). Elective resection of an a ff ected small bowel segment causing malabsorption can be e ff ective, provided there is only a limited amount of jejunum involved. If perforated jejunal diverticulitis is found at emer gency laparotomy , a small bowel resection should be performed and a decision made between primary anastomosis and stoma formation. This will depend on the degree of contamination, physiological stability and local resources for managing a patient with a high-output jejunostomy . Complications resulting from extensive jejunal diverticulo sis can be extremely di ffi cult to treat. In severe cases, much of the proximal small intestine may be involved, e ff ectively pre cluding resection. Prolonged antibiotic therapy for bacterial overgr owth may be preferable, and antibiotics (metronidazole, Johann Friedrich Meckel (the younger), 1781–1833, Professor of Anatomy and Surgery , Halle, Germany , described the diverticulum in 1809. -). ciprofloxacin, rifaximin) may be rotated in an attempt to avoid antibiotic resistance. Limited resection, leaving remaining seg - ments of a ff ected jejunum, may be feasible, but may also fail - to deal adequately with bacterial overgrowth, recurrent attacks of inflammation or bleeding. Meckel’s diverticulum A Meckel’s diverticulum is a persistent remnant of the vitello - intestinal duct and is present in about 2% of the population. It is found on the antimesenteric side of the ileum approximately - 60 /uni00A0 cm from the ileocaecal valve and is classically 5 /uni00A0 cm long (2% prevalence; 2 feet [60 /uni00A0 cm] from ileocaecal valve; 2 inches [5 /uni00A0 cm] long). A Meckel’s diverticulum is a congenital diverticulum e (Figure 74.5). It contains all three coats of the bowel wall and has its own blood supply . It may be vulnerable to obstruction - and inflammation in the same way as the appendix; indeed, when a normal appendix is found at surgery for suspected appendicitis, a Meckel’s diverticulum should be looked for by examining the small bowel,

particularly if free fluid or pus ed is found (see Chapter 76). In approximately 20% of cases, the mucosa of a Meckel’s diverticulum contains heterotopic epithelium of gastric, colonic or pancreatic type. The presence of heterotopic mucosa may predispose to the development of - complications (Summary box 74.4). The vast majority of Meckel’s diverticula are asymptomatic and a Meckel’s diverticulum is notoriously di ffi cult to visualise erticulum may however with contrast radiology . Meckel’s div present clinically in the following ways. - Haemorrhage . If gastric mucosa is present, peptic ulcer - ation can occur and present as painless dark rectal bleeding or - melaena. If the stomach, duodenum and colon are excluded as a source of bleeding by endoscopy , radioisotope scanning with te a Meckel’s diverticulum. technetium-99m may demonstra

Figure 74.5 Meckel’s diverticulum.

appendicitis, although if perforation occurs the presentation may resemble a perforated duodenal ulcer. Intussusception . A Meckel’s diverticulum can be the lead point for ileoileal or ileocolic intussusception. Chronic ulceration . Pain is felt around the umbilicus, as the site of the diverticulum is midgut in origin. Intestinal obstruction . A band between the apex of the diverticulum and the umbilicus (also part of the vitello intestinal duct) may cause obstruction directly , or by predis posing to the development of a volvulus around it. Perforation . (Figure 74.6). When found in the course of abdominal surgery , a Meckel’s diverticulum can safely be left alone, provided it has a wide mouth and is not thickened. When there is doubt, it can be resected. The finding of a Meckel’s diverticulum in an inguinal or femoral hernia has been described as ‘Littre’s hernia’. Summary box 74.4 Features of Meckel’s diverticulum /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Meckel’s diverticulectomy A broad-based Meckel’s diverticulum should not be amputated at its base and invaginated (as for an appendix), as there is the risk of stricture and of leaving heterotopic epithelium behind. It is safer simply to excise the diverticulum, either by resecting the diverticulum and suturing the defect at its base or by performing a limited small bowel resection with anastomosis. This can also be achieved with a linear stapler–cutter. If the base of the diverticulum is indurated, it is on balance safer to perform a limited small bowel resection of the entire involved segment, followed by an anastomosis.

Remnant of vitellointestinal duct Occurs in 2% of patients, 5 /uni00A0 cm (2 inches) long, 60 /uni00A0 cm (2 feet) from the ileocaecal valve, 20% heterotopic epithelium Should be looked for when a normal appendix is found at surgery for suspected appendicitis If a Meckel’s diverticulum is found incidentally at surgery, it can be left provided it has a wide mouth and is not thickened Can be a source of gastrointestinal bleeding if it contains ectopic gastric mucosa

Chronic small intestinal ischaemia

Chronic small intestinal ischaemia

Chronic small intestinal ischaemia almost invariably results from atherosclerosis and a ff ects the proximal superior mesen teric and coeliac vessels. Patients classically present with symptoms of severe central abdominal pain that comes on within 30–60 minutes of eating (mesenteric angina). Weight loss and diarrhoea due to malabsorption may also occur. The condition may be di ffi cult to diagnose and is often overlooked. The presence of significant vascular disease on CT is common in elderly patients and in those with severe vascular disease and should not necessarily be assumed to indicate that abdominal symptoms are attributable to chronic ischaemia. Treatment is usually by selective visceral angiography , with stenting/angio plasty and, where this is not possible, bypass surgery . Smoking cessation is imperative and patients are usually anticoagulated.

Complications of stomas

Complications of stomas

Stoma complications are underestimated and common (Summary box 74.6). On occasion, these complications require surgical revision. Sometimes, this can be achieved with an incision immediately around the stoma, but on occasion reopening the abdomen and freeing up the stoma may be necessary . Repair of parastomal hernias is particularly tech nically challenging and the recurrence rate is high. Simple suture of the parastomal hernia is associated with an almost 100% risk of recurrence and transfer to the opposite side of the abdomen, or insertion of a piece of prosthetic material within the abdominal wall around the stoma may be necessary (see Chapter 64). Summary box 74.6 Stoma complications /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF -

Figure 74.8 Spouted ileostomy in the right iliac fossa. Skin irritation Stenosis Prolapse Parastomal hernia Retraction Bleeding Ischaemia Fistulation

ENTEROCUTANEOUS FISTULA

ENTEROCUTANEOUS FISTULA

- An abnormal connection between the small intestine and the skin can occur as a result of CD, radiotherapy or abdominal trauma, but the condition most commonly follows a surgical - complication – either a leak from an anastomosis or an inadver - coeliac tent enterotomy . At least 50% of small bowel enterocutaneous fistulae develop after surgery in which no small bowel has been resected as a result of injury to the intestine during division of adhesions. The frequency of this complica tion has been shown to increase with the number of previous laparotomies. Management of patients with an enterocutaneous fistula can be very challenging, especially when the fistula output Figure is high (defined as >500 /uni00A0 mL of e ffl uent/day). The majority of low-output fistulae can be expected to heal spontaneously , - provided there is no distal obstruction or disease at the fistula site. Reasons for failure of spontaneous healing also include epithelial continuity between the gut and the skin and an associated complex abscess. The management of fistulae is based on well-established principles (‘SNAP’; see Summary box 74.7). An early return - to theatre to try to treat the problem definitively (i.e. by ished patient is doomed to failure. Summary box 74.7 Principles of management of enterocutaneous fistulae (SNAP) /uni25CF /uni25CF /uni25CF /uni25CF Infected collections are best identified at CT (Figure 74.10 and can be drained percutaneously . Skin protection is import ant as small bowel e ffl uent is caustic. Nutritional support must include fluid and electrolytes, which can be lost in high quan tities from a proximal fistula, as w ell as carbohydrates, protein, fat and vitamins. Judgements have to be made between enteral and parenteral feeding: enteral feeding has advantages, but if the fistula is proximal or high output total parenteral nutrition will be requir ed. Defining fistula anatomy is best done after careful discussion with the radiologist; a sequence of contrast studies (follow-through, fistulogram and enema) may well be required to define bowel length and plan a surgical strategy . Surgery can be extremely technically demanding and an anastomosis should be avoided in the presence of continuing intra-abdominal sepsis or when the patient is hypoalbuminae mic (<32 /uni00A0 g/dL).

S, elimination of Sepsis and skin protection N, Nutrition – a period of parenteral nutrition may well be required A, Anatomical assessment P , de /f_i nitive Planned surgery

End-ileostomy

End-ileostomy

An end-ileostomy is formed after a colectomy without anas - tomosis, when it may later be reversed, or after panproctocol - ectomy , when it is permanent. The ileum is normally brought through the rectus abdominis muscle. Careful attention should be paid to the terminal ileal mesentery to ensure tha t it is not too bulky . The use of a spout was originally described by Brooke; this should project some 2–4 /uni00A0 cm from the skin surface (Figure 74.7). A disposable appliance is placed over the ileos - - tomy so that it is a snug fit at skin level. There may be an ‘ileostomy flux’ while the ileum adapts to the loss of the colon. While ileostomy output can amount to 4–5 litres per day , losses of 1–2 litres are more common. A con - sistent ileostomy output in excess of 1.5 litres is usually asso - ciated with dehy dration and sodium depletion in the absence of intravenous therapy . Up to 20% of patients may require readmission for the trea tment of dehydration after creation of an ileostomy but the stools thicken in a few weeks and are usu ally semisolid in a few months. The help, skill and advice of the stoma care nurse specialist are essential. Modern appliances are unusual have transformed stoma care and skin problems (Figure 74.8). Complications of an ileostomy include prolapse, retraction, stenosis, bleeding, fistula and parastomal hernia.

Figure 74.7 Construction of an end-ileostomy. The diagram is orientated such that the upper aspect of the stoma is to the right, thus when the sutures are tied the everted stoma is angled slightly inferiorly. (Reproduced with permission from O’Connell PR, Madoff RD, Solomon MJ (eds). Operative surgery of the colon rectum and anus , 6th edn. Boca Raton, FL: CRC Press, 2015.)

Human immunodeficiency virus

Human immunodeficiency virus

Human immunodeficiency virus (HIV) infection is associated with a number of proctological problems (see Chapter 80 Intestinal complications are common after the development of acquired immunodeficiency syndrome (AIDS), when opportunistic organisms can cause gastroenteritis (Summary box 74.2). HIV may also cause a specific enteropathy . Treat ment is directed towards the relevant organism and surgery should be avoided if possible. Summary box 74.2 Opportunistic intestinal infections in patients with AIDS /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Bacterial Viral Salmonella Cytomegalovirus Shigella Protozoal Yersinia Cryptosporidium Campylobacter Giardia Mycobacterium avium– Fungal intracellulare (MAI) Candida albicans

I N F E C T I V E E N T E R I T I S Campylobacter

I N F E C T I V E E N T E R I T I S Campylobacter

Infection with Campylobacter jejuni (a Gram-negative rod with a distinctive spiral shape) is the most common form of bacte - rial gastroenteritis in the UK, typically acquired from eating infected poultry . It causes diarrhoea and abdominal pain and may mimic an acute abdomen. Severe cases may resemble ulcerative colitis, with rectal bleeding and colorectal ulcer - ation, causing diagnostic di ffi culty . The organism is fastidious in culture and may take several days to isolate in the laboratory . - Toxic dilatation and ev en disintegrative colitis have rarely been reported to occur. Treatment is generally supportive as the condition usually resolves without antibiotics. It is a notifiable disease.

INFLAMMATORY BOWEL DISEASE

INFLAMMATORY BOWEL DISEASE

The term ‘inflammatory bowel disease’ is reserved for conditions characterised by the presence of idiopathic intes tinal inflammation; conditions such as infective or ischaemic enteritis are, by definition, excluded. Crohn’s disease (CD) is the only known ‘inflammatory bowel disease’ that a ff ects the small intestine and is covered in detail in Chapter 75 . Burrill Bernard Crohn , 1884–1983, gastroenterologist, Mount Sinai Hospital, New Y ork, NY , USA, along with Leon Ginzburg and Gordon Oppenheimer, described regional ileitis in 1932.

Jejunum Ileum (proximal (distal 40%) 60%) Terminal ileum Figure 74.1 Portions of the small bowel and their relationship to the colon.

Introduction

Introduction

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Learning objectives

Learning objectives

To appreciate: The basic anatomy and physiology of the small intestine intestinal problems • The range of conditions that may affect the small intestine • To understand: The aetiology and pathology of common small intestinal multidisciplinary team • conditions The principles of investigation of small intestinal symptoms •

Loop ileostomy

Loop ileostomy

A loop ileostomy is often used for defunctioning a low rectal anastomosis or an ileal pouch. A knuckle of ileum is exterior - ised through a skin trephine in the right iliac fossa. An incision is made in the distal part of the knuckle, and this is then pulled over the top of the more proximal part to create a spout on the proximal side of the loop with a flush distal side still in continuity . This allows near perfect defunctioning , but also the possibility of restoration of continuity , by taking down the - spout and reanastomosing the partially divided ileum. The advantage of a loop ileostomy over a loop colostomy is the ease with which the bowel can be brought to the surface. Care is needed when the ileostomy is closed such that suture line obstruction does not occur. Closure of a loop ileostomy can be a technically challenging pr ocedure, particularly if there are dense adhesions resulting from previous surgery . -

PHYSIOLOGY OF THE SMALL INTESTINE

PHYSIOLOGY OF THE SMALL INTESTINE

- The principal function of the small intestine is the digestion of food and absorption of nutrients, water and electrolytes. Carbohydrates and proteins are broken down in the intestinal lumen by pancreatic enzymes, but the final hydrolysis takes place at the brush border of the jejunum, after which these nutrients are absorbed. Fats are digested chiefly by the actions of pancreatic lipase and bile salts. The products of fat diges - - tion, fatty acids and monoglycerides, separate from bile salts in the jejunum and are absorbed for further processing. The jejunum is the principal site for digestion and absorption of - fluid, electrolytes , iron, folate, fat, protein and carbohydrate, - but the absorption of bile salts and vitamin B12 only occurs in the terminal ileum, where there are specific transporters. If the jejunum is resected, the ileum can assume all the required absorptive functions, but resection of the terminal ileum will result in a diminished bile salt pool, vitamin B12 deficiency and may lead to deficiency of the fat-soluble vitamins A, D, E and K. The ileum also plays an important role in water absorption, possibly because the tightness of the intercellular junctions supports a concentration gradient across its lumen. Significant ileal resection therefore commonly results in very troublesome diarrhoea. The small intestine plays an important role in the metab olism of plasma lipoproteins, as it is the main site of synthesis of high-density , low-density and very low-density lipoproteins (HDL, LDL, VLDL). These particles transport most of the absorbed dietary fat to the systemic circulation via the lymph. The small bowel also synthesises intestinal hormones such as glucagon-like peptides GLP-1 and 2, peptide YY and motilin, which interact with the enteric nervous system to modulate intestinal function, growth and di ff erentiation.

SHORT BOWEL SYNDROME INTESTINAL FAILURE

SHORT BOWEL SYNDROME/ INTESTINAL FAILURE

Intractable diarrhoea with impaired absorption of nutrients following resection or bypass of the small intestine, ultimately leading to progressive malnutrition, is referred to as short bowel syndrome. When a patient is unable to maintain satisfactory fluid, electrolyte or nutritional homeostasis without intra venous administration of fluid, electrolytes or nutrients they are said to have intestinal failure. The mainstay of treatment for intestinal failure is parenteral nutrition. The most common causes of short bowel syndr ome are resection resulting from the management of CD and its com plications (which accounts for almost half of cases), mesenteric vascular thr ombosis, radiation enteritis and tumours. Although features of short bowel syndrome usually appear w hen there is less than 200 /uni00A0 cm of small bowel, the length and nature of the remaining intestine are also important. In general, diseases that result in short bowel syndrome tend to preferentially a ff ect the distal small intestine, and there is some evidence that the ileum, with its tighter intercellular junctions and consequently better fluid absorptive capacity , can assume the functions of a missing jejunum, but not vice versa. While the ileocaecal valve used to be considered important with regard to preservation of absorptive function, it is more likely that this is a reflection of the associated preservation of the distal ileum and right colon than the valve itself.) - - Patients with an intact colon are relatively protected from the e ff ects of massive small bowel resection because of the abil - ity of the colon to absorb not only fluid and electrolytes but also a modest amount of nutrient energy . Patients with as little as 100–200 /uni00A0 cm of jejunum anastomosed to an intact colon may therefore be able to maintain satisfactory macron utrient, fluid and electrolyte status, although they will, of course, be at risk of fat-soluble and B12 vitamin deficiencies and will also generally need oral nutritional supplements of trace elements, vitamins and minerals. Some (but not all) patients with 50–100 /uni00A0 cm of - small intestine and an intact colon will have intestinal failure, as will almost all patients with 50 /uni00A0 cm or less of jejunum anas - tomosed to an intact colon. In contrast, most patients with less than 200 /uni00A0 cm and virtually all with less than 100 /uni00A0 cm of small intestine ending in a stoma will have intestinal failure and will require long-term par enteral nutrition. Medical management of patients with short bowel syn - drome relies on the use of antidiarrhoeal agents (loperamide and codeine phosphate), drugs to reduce diarrhoea related to bile-salt malabsorption (cholestyramine), drugs to reduce the - increased gastric acid secretion resulting from the loss of the small bowel ‘brake’ on gastric acid production (pr oton pump inhibitors) and enteral and parenteral vitamin and trace ele - ment supplements. Although there has also been interest in the use of drugs to promote intestinal adaptation, such as growth - hormone, glutamine and, most recently , glucagon-like peptide 2 agonists, the mainstay of trea tment for short bowel syndrome remains home parenteral nutrition (HPN). The development of this treatment in the late 1960s enabled the majority of patients with short bowel syndrome to enjoy a reasonably good quality of life, with long-term survival related

principally to the underlying disease. HPN is, however, expensive and demand - ing and patients are at risk from catheter-related complications (notably catheter-related sepsis and occlusion), as well as meta - bolic complications (fibrotic liver disease, gallstones, metabolic bone disease and kidney stones). Surgical procedur es designed to improve the surface area or reduce the speed of transit of the r emaining small intestine (and thus improve absorptive capacity) have shown some promise in children, but their place in managing adults with

Figure 74.10 Computed tomography (CT) scan in a patient with a complex enterocutaneous /f_i stula and an intra-abdominal abscess being drained with a CT-guided catheter.

In some patients, the loss of venous access resulting from the complications of long-term intravenous feeding or the development of progressive liver dysfunction may represent indications for small bowel transplantation. The results of small bowel transplantation have progressively improved and 5-year patient survival now exceeds 80% in some centres (see Chapter 91). Bland KI, Sarr MG, Büchler MW et al. (eds.) Surgery of the small bowel. Handbooks in General Surgery . London: Springer-V erlag, 2011. Keighley MRB, Williams NS. Keighley & Williams’ surgery of the anus rectum and colon, 4th edn. Boca Raton, FL: CRC Press, 2018. Slade DAJ, Carlson GL. Takedown of enterocutaneous fistula and complex abdominal wall reconstruction. Surg Clin North Am 2013; 93 : 1163–83. Soop M, Carlson GL. Intestinal failure: In: Herold A, Lehur P-A, Matzel KE, O’Connell PR (eds). European manual of medicine: coloproctology , 2nd edn. Berlin: Springer, 2017.

STOMAS

STOMAS

A stoma is an artificial opening made in the bowel to divert faeces and flatus outside the abdomen, where they can be collected in an external appliance. Depending on the purpose Bryan Nicholas Brooke , 1915–1998, Professor of Surgery , St George’s Hospital, London, UK. temporary or permanent (Summary box 74.5). - Summary box 74.5 Stomas /uni25CF /uni25CF /uni25CF - /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF - /uni25CF

May be colostomy or ileostomy May be temporary or permanent Temporary or defunctioning stomas are usually fashioned as loop stomas An ileostomy is spouted; a colostomy is /f_l ush Ileostomy ef /f_l uent is usually liquid, whereas colostomy ef /f_l uent is usually solid Ileostomy patients are more likely to develop /f_l uid and electrolyte problems An ileostomy is usually sited in the right iliac fossa End-colostomy is usually sited in the left iliac fossa Whenever possible, patients should be counselled and sited by a stoma care nurse before operation

Stoma bags and appliances

Stoma bags and appliances

Stoma output is collected in a disposable adhesive bag. Ileostomy appliances tend to be drainable bags that are left in place for 48 hours, while colostomy appliances are simply changed two or three times each day . A wide range of such bags is currently available. Many now incorporate an adhesive backing, which can be left in place for several days. In most hospitals, a stoma care service is available to o ff er advice to patients, to acquaint them with the latest appliances and to provide the appropriate psychological and practical help.

TUMOURS OF THE SMALL INTESTINE

TUMOURS OF THE SMALL INTESTINE

Small bowel tumours are rare and in total account for less than 10% of gastrointestinal neoplasia.

Tuberculosis of the intestine

Tuberculosis of the intestine

Tuberculosis, like CD, can a ff ect any part of the gastrointestinal tract. The sites a ff ected most often are the ileum, proximal colon and peritoneum. There are two principal disease presentations. Ulcerative tuberculosis Ulcerative tuberculosis develops secondary to pulmonary tuberculosis and arises as a result of swallowing tubercle bacilli. Multiple ulcers, lying transversely , develop in the terminal ileum and the overlying serosa is thickened, reddened and covered in tubercles. Patients typically present with diarrhoea and weight loss, although subacute obstruc tion and even local perforation and fistula formation can occur. A barium follow-through or computed tomography (CT) examination fails to show filling of the distal ileum, caecum and the ascending colon as a result of narrowing of the ulcerated segment (Figur e 74.2). A course of antituberculous chemotherapy usually leads to cure, provided the pulmonary tuberculosis is adequately treated. Surgery is usually undertaken only in the rare event of a perforation or complete intestinal obstruction. Esche - - - - - Hyperplastic tuberculosis This is caused by the ingestion of Mycobacterium tuberculosis by patients with a high resistance to the organism. The infection usually occurs in the ileocaecal region, although solitary and multiple lesions in the distal ileum are also sometimes seen. The infection establishes itself in lymphoid follicles, and the resulting chronic inflammation causes thickening of the intestinal wall and narrowing of the lumen. There is early involvement of the regional lymph nodes, which may caseate. Unlike in CD, abscess and fistula formation are uncommon. Patients usually present with attacks of abdominal pain and intermittent diarrhoea. There is incomplete ileal obstruction, leading to stasis and bacterial overgrowth. This in turn causes steatorrhoea, anaemia and loss of weight. P atients may present with a mass in the right iliac fossa and vague ill health. The di ff erential diagnosis is that of an appendix mass, lymphoma, carcinoma of the caecum, CD, tuberculosis or actinomycosis. A barium follow-through or small bowel enema will show a - long narrow filling defect in the terminal ileum (which may result in a di ff erential diagnosis of CD). CT will also demon - strate the narrowed segment with proximal distension and the associated lymphadenopathy . When the diagnosis is clear and the patient has not yet developed obstructive symptoms, treatment with antituberculous medication is advised and may be curative . Where obstruction is present, or the possibility of CD or lymphoma requires clarification, ileocaecal resection is often required (see Chapters 6 and 65).

Figure 74.2 Ileocaecal tuberculosis showing dilatation of the distal ileum and stricturing of the terminal ileum and caecum (courtesy of Dr. VK Kapoor, Delhi, India).

Abdominal actinomycosis is rare. It is caused by infection with Actinomyces israelii and usually develops several weeks after an apparently straightforward perforated appendicitis. An abscess develops and spreads to the retroperitoneal tissues and the adjacent abdominal wall, eventually becoming the seat of multiple indurated discharging sinuses. At first, the discharge from the sinuses is thin, watery and ino ff ensive, but it may later become thicker and malodorous. Secondary fistulation may occur and the tissues may become extensively indurated and woody . In

contrast to tuberculosis, however, mesenteric lymph nodes are not involved and the lumen of the intestine is not narrowed. Haematogenous spread via the portal vein may lead to multiple liver abscesses. Pus should be sent for bacteriological examination, which will reveal the characteristic sulphur granules. Penicillin or co-trimoxazole treatment is required and should be prolonged and in high dosage.

VASCULAR ANOMALIES OF THE INTESTINE Mesenteric isc

VASCULAR ANOMALIES OF THE INTESTINE Mesenteric ischaemia

Mesenteric vascular disease may be classified as acute intestinal ischaemia – with or without occlusion – venous, chronic arte rial, central or peripheral. The superior mesenteric vessels are the visceral vessels most likely to be a ff ected by embolisation or thrombosis. Occlusion at the origin of the SMA is almost invariably the result of thrombosis, whereas emboli tend Alexis Littre , 1658–1746, surgeon and Lecturer in Anatomy , Paris, France, described Meckel's diverticulum in a hernial sac in 1700, 81 years before Meckel was born. - - lodge at the origin of the middle colic artery . Inferior mesen - teric artery involvement is usually clinically silent because of a rich collateral circulation. SMA emboli may be carried from the left atrium in atrial fibrillation, the left ventricle after mural myocardial infarc - tion, vegeta tions on mitral and aortic valves associated with endocarditis or an atheromatous plaque from an aortic aneu - rysm. Primary thrombosis is associated with atherosclerosis and vasculitides, including conditions such as thromboangitis obliterans and poly arteritis nodosa. Primary thrombosis of the superior mesenteric veins may occur in association with fac - tor V Leiden disorder, portal hypertension, portal pyaemia, sickle cell disease and in w omen taking the oral contraceptive pill. A specific form of 'non-occlusive mesenteric ischaemia' (in which the vessels are normal but flow is critically reduced) may complica te critical illness, possibly because of alterations in splanchnic blood flow . Irrespective of whether the occlusion is arterial or venous, haemorrhagic infarction occurs. The mucosa is especially sensitive to ischaemic injury because of its high metabolic activity . The intestine and its mesentery become swollen and oedematous, especially with venous occlusion. Bloodstained fluid exudes into the peritoneal cavity and bowel lumen. The changes develop rapidly and irreversible injury , ranging in severity from mucosal necrosis and sloughing to full-thickness infarction, usually occurs within 6 hours at most. If the main trunk of the SMA is involved, the infarction usually covers an area from just distal to the DJ flexure to the splenic flexure. Usually , a branch of the main trunk is implicated and the area of infarction is smaller. - Clinical features The most important clue to an early diagnosis of acute mesen - teric ischaemia is sudden onset of severe abdominal pain in a to

Figure 74.6 Gangrenous Meckel's diverticulitis.

typically in the central abdomen and is out of all proportion to the physical findings. Persistent vomiting and defecation occur early , with the subsequent passage of altered blood. Abdomi nal tenderness may be mild initially , with rigidity being a late feature. Shock, with features of both hypovolaemia and sepsis, rapidly ensues . Investigation Investigation will usually reveal a profound

neutrophil leuko cytosis, a severe metabolic acidosis and raised blood lactate. A contrast-enhanced CT scan will show bowel wall enhancement absent or reduced and there may be free fluid in the abdomen. Gas may be present within the intestinal wall and occasionally in the mesenteric and portal vein, a late and ominous sign. Treatment Mesenteric venous thrombosis may be treated by anticoagu lation with close monitoring. An immediate laparotomy with embolectomy or revascularisation of the SMA by vascular bypass may be considered in early cases of arterial ischaemia, followed by postopera tive anticoagulation. However, the condition is usually diagnosed late in the disease process and the mortality rate is extremely high. In the young, all a ff ected bowel should be resected, whereas in the elderly or infirm the situation may be deemed incurable. Where the demarcation between viable and non-viable bowel is uncertain a planned relook laparotomy may be useful. After extensive enterectomy , it is usual for patients to require intravenous nutrition. The young, however, may sometimes develop su ffi cient intestinal digestive and absorp tive function to lead relatively normal lives. In selected cases, consideration may be given to small bowel transplantation (see Chapter 91).

Ye r s i n i a

Ye r s i n i a

Yersinia enterocolitica is a Gram-negative rod that can infect the terminal ileum, appendix, ascending colon and mesenteric lymph nodes, and can cause a granulomatous inflammatory process that may mimic CD. Yersinia typically causes a fever and gastroenteritis, but may persist and cause a terminal ileitis, which, on occasion, may perforate. The diagnosis - may be made on stool culture, but is more often confirmed serologically . If discovered at laparotomy , the terminal ileum and mesenteric nodes will look thickened and inflamed and a lymph node biopsy can be taken for diagnostic purposes. The disease is normally self-limiting, but responds to treatment with co-trimoxazole or chloramphenicol antibiotics. Salmonella are a family of Gram-negative rods that can cause a range of enteric infections. Salmonella gastroenteritis is typically caused by Salmonella enteritidis from poultry and is most often a self-limiting illness comprising headache, fever and watery diarrhoea. When severe, antibiotics and hospitalisation and intravenous fluids may be needed. The diagnosis is based on stool culture. Shigella and enteropathogenic strains of richia coli may cause similar diarrhoeal illnesses. Typhoid fever is caused by Salmonella enterica and presents with fever and abdominal pain after an incubation period of 10–20 days. Over the next week, the patient can develop distension, diarrhoea, splenomegaly and characteristic ‘rose spots’ on the abdomen caused by a vasculitis. Typhoid is a systemic infection and diagnosis of typhoid is confirmed by culture of blood or stool. Treatment is by antibiotics, usually chloramphenicol. A number of surgical complications can result, including paralytic ileus, intestinal haemorrhage, free ileal perforation and cholecystitis. Invasion of the systemic circulation, which is a character istic feature of salmonellosis, may cause severe Gram-negative sepsis, resulting in septic shock. Some patients develop meta static sepsis, including septic arthritis and osteomyelitis, menin gitis, ence phalitis, disseminated intravascular coagulation and pancreatitis. Perforation of a typhoid ulcer characteristically occurs during the third week of the illness, although it is sometimes the fir st clinical sign of the disease. The ulcer is parallel to the long axis of the gut and is usually situated in the distal ileum. Perforation r equires surgery to wash out and close the ulcer and intestinal resection is usually avoided. In unstable patients, notably with evidence of septic shock, the bowel should be exte riorised and the perforation closed after recovery . Paratyphoid infection (with Salmonella Paratyphi A) resembles typhoid fever and is treated in a similar manner (see Chapter 6).