15.10.7 Effects of massive bowel resection 2911

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15.10.7 Effects of massive bowel resection 2911 T. whipplei DNA is detectable in various body fluids and tissues. Sampling should be performed depending on the clinical manifest ations: small-bowel biopsies for digestive symptoms, synovial fluid and/or biopsy for articular involvement, blood and/or cardiac valve for endocarditis, cerebrospinal fluid for neurological manifestations, and aqueous humour for uveitis. When Whipple's disease is suspected, screening may also be per formed on the basis of the combined analyses of saliva and faeces using specific quantitative real-time PCR. If both were positive, the suspicion of Whipple's disease is high, whereas if they are both nega tive, classic Whipple's disease can be excluded. The diagnosis of classic Whipple's disease is based on positive periodic acid–Schiff (PAS) staining of duodenal biopsies (Fig. 15.10.6.1), but PAS staining can be positive in other circumstances, such as mycobacterium infection. Immunohistochemical analysis using specific antibodies allows the direct visualization of bacteria in samples and has sensitivity and specificity superior to those of PAS staining. 18F-fluorodeoxyglucose positron emission tomography images demonstrating small-bowel hypermetabolism seem to be associated with classic Whipple's disease. Such images in an investigation of un explained weight loss, diarrhoea, or fever of unknown origin might suggest the need to consider and investigate for Whipple's disease. Western blot serology can help differentiation of PCR-positive asymptomatic carriers, who have a strong immune response, and patients with classic Whipple's disease, who lack or have a low im mune response, but this technique is complex and can be performed only in the chapter authors' laboratory. Culture of this fastidious bacterium from human specimens is currently also only performed in the authors' laboratory. Management and prognosis Whipple's disease is a lifelong disease. Failures, relapses, and rein fections caused by different T. whipplei strains have been observed in patients who have apparently been cured. Relapses have been also reported as late as 20 years after the initial diagnosis, and these may occur in organs other than those previously involved. Until recently, treatment was empirical, but the recent culture of T. whipplei has allowed antibiotic susceptibility testing to guide treatment protocols. Trimethoprim–sulfamethoxazole, which for a long time was regarded as the mainstay of treatment, must be avoided due to its poor efficiency and its association with failures and relapses. Currently, for classic Whipple's disease, on the basis of

antibiotic susceptibilities and the follow-up of patients, doxy cycline (200 mg/day) and hydroxychloroquine (200 mg three times/day) for 12 months is the best treatment. This should be fol lowed by long-term (possibly lifelong) treatment with doxycycline because potentially fatal relapses can occur, as well as reinfections with new T. whipplei strains. For localized chronic infections, a combination of doxycycline and hydroxychloroquine for a duration of 12 to 18 months, followed by an extended follow-up, has been proposed, but the recent obser vation of evolution of localized T. whipplei endocarditis into classic Whipple's disease should also lead to consideration of longer treat ment for localized chronic infections. No specific treatments or recommendations are available for acute infections and chronic carriage. FURTHER READING Elchert JA, Mansoor E, Abou-Saleh M, Cooper GS (2019). Epidemiology of Whipple's disease in the USA between 2012 and 2017: a population-based national study. Dig Dis Sci, 64, 1305–11. Fenollar F, Lagier JC, Raoult D (2014). Tropheryma whipplei and Whipple's disease. J Infect, 69, 103–12. Keita AK, et al. (2015). High prevalence of Tropheryma whipplei in Lao kindergarten children. PLoS Negl Trop Dis, 9, e0003538. Lagier JC, Cammilleri S, Raoult D (2016). Classic Whipple's disease diagnosed by 18F-fluorodeoxyglucose PET. Lancet Infect Dis, 16, 130. Lagier JC, et al. (2014). Treatment of classic Whipple's disease: from in vitro results to clinical outcome. J Antimicrob Chemother, 69, 219–27. Lagier JC, Raoult D (2018). Whipple's disease and Tropheryma whipplei infections: when to suspect them and how to diagnose and treat them. Curr Opin Infect Dis, 31, 463–70. Ramharter M, et al. (2014). Prevalence and risk factor assessment of Tropheryma whipplei in a rural community in Gabon: a community- based cross-sectional study. Clin Microbiol Infect, 20, 1189–94. Vinnemeier CD, et al. (2016). Tropheryma whipplei in children with diarrhoea in rural Ghana. Clin Microbiol Infect, 22, 65.e1–65.e3. 15.10.7 Effects of massive bowel resection Stephen J. Middleton, Simon M. Gabe,
and Raymond J. Playford ESSENTIALS Major vascular events involving the superior mesenteric artery and small-bowel volvulus are the commonest reasons for adults to re- quire massive intestinal resection. The ability of the residual bowel to adapt after resection varies greatly between patients, but common postoperative problems include sepsis, diarrhoea (or high-output stoma losses), fluid and electrolyte imbalance, malnourishment (protein–energy malnutrition, mineral and vitamin deficiencies), gall- stones, renal stones, and psychological illness. Where appropriate, oral nutrition, initially consisting of low-volume polymeric feeds administered by nasogastric or enteral tube, should be started within the first few days of surgery. Small-volume, frequent, solid or semisolid meals with low long-chain triglycerides and (when colon is in continuity) oxalate content should be introduced subsequently, and isotonic electrolyte solutions given as required. Oral multivitamin and mineral supplements are usually needed, and vitamin B12 injections may be required. There should be regular long-term monitoring of fat- soluble vitamins (A and D), vitamin B12, folate, magnesium, zinc, and bone status. Long-term intravenous nutrition is sometimes needed. Growth factor administration, especially glucagon-like peptide-2 analogues, may stimulate bowel adaptation. Small-bowel surgery can sometimes offer a modest increase in length for patients with dilated bowel. Those who are dependent on peripheral nutrition and develop complications such as loss of venous access or liver disease should be considered for intestinal transplantation.

section 15 Gastroenterological disorders 2912 Introduction Massive loss of the intestine occurs as a result of surgical resection leaving a residual length that is insufficient to support all aspects of nutrition adequately. These patients initially enter an acute phase with sudden loss of enteral function in association with a variety of other comorbidities either resulting from or being the cause

of their intestinal catastrophe. If they survive this period they will enter a chronic phase of intestinal failure when their health and well-being will depend upon careful nutritional support, which often includes intravenous feeding, as well as physical, psychological, and intestinal rehabilitation. During this phase, a plan for long-term management is made, the aims of which will depend particularly upon the re habilitation potential of the remaining intestinal tract. The skills and facilities for effective management of the acute phase need to be widely available as patients usually present as emergencies to their nearest hospital. The chronic phase is often better managed in specialized centres. The management and prog nosis of patients is greatly influenced by the presence of the colon in continuity with the remaining small intestine. This chapter is pre dominantly concerned with adult patients, although much of it will also apply to paediatric patients. Aetiology The conditions that most frequently lead to sudden massive intes tinal surgical resection resulting in a short bowel in adults are mes enteric ischaemia and small-bowel volvulus. Crohn's disease is a common cause of short bowel, but this usually results from multiple segmental resections which lead incrementally to short-bowel syn drome due to a combination of reduced length and reduced func tion within the remaining segments arising from factors such as mucosal fibrosis. Other causes include surgery to resect desmoid tumours and trauma. Lifestyle changes and medication to reduce ar terial disease, prudent limitation of surgical intervention in Crohn's disease, and careful surgical technique to avoid subsequent volvulus after colectomy or bariatric surgery may help reduce the incidence of these events. In children, the main causes include necrotizing en terocolitis, gastroschisis, and intestinal volvulus. Pathophysiology Water and electrolyte depletion The consequences of intestinal resection are influenced by the re gion (or regions) of the gut that has been lost. Patients with short- bowel syndrome commonly fall into one of three groups based on their residual gastrointestinal anatomy (Fig. 15.10.7.1). The stomach may secrete 1 to 2 litres of acidic fluid each day, which is normally reabsorbed in the small bowel and colon. The proximal 100 cm of jejunum is net secretory, such that the water volume excreted via a stoma in this segment will be greater than that in an ingested meal. The sodium concentration of most meals varies between 10 and 40 mmol/litre, and due to the contribution of gastro intestinal secretions this gradually increases to around 90 mmol/ litre at the duodenojejunal flexure. On reaching the terminal ileum, the concentration has risen to about 140 mmol/litre, largely due to water absorption in the more distal intestine. Compared to the ileum, the jejunum is less able to absorb water and sodium against a concentration gradient due to rapid back-diffusion into the lumen to create an iso-osmolar luminal fluid mixture. In contrast, the ileum is far less permeable and can mount a considerable concentration gradient across the mucosal surface. Furthermore, jejunal sodium absorption is coupled to glucose and amino acids, in contrast to the ileum which is free from these constraints and can also increase ab sorption in response to aldosterone. Taken in combination, these regional differences in intestinal function result in an adverse effect following loss of the distal small intestine that is greater than losing a similar amount of prox imal intestine. The presence of colon in continuity can compen sate to some degree for water and electrolyte losses and may also encourage adaptation (which includes increased villus height and crypt depth) of the remaining intestine to enhance absorption via mediators such as glucagon-like peptide (GLP)-2, which is released by colonic enterochromaffin L cells in response to malabsorbed nutrients. Changes in gut hormones, motility, and secretion Other potentially important pathophysiological changes include hypergastrinaemia. This is unlikely to cause gastric hypersecretion for more than a few weeks, although many patients benefit from treatment with a proton pump inhibitor in the longer term. Gastric emptying is often rapid in patients who have lost the colon and ileum, probably due to depletion of peptide tyrosine-tyrosine (PYY)- releasing

enterochromaffin cells. Pancreaticobiliary secretion is not greatly affected, but loss of more than 100 cm of terminal ileum leads to malabsorption of secondary bile acids. These act as secretagogues in the colon, increasing mucosal secretion, and fat malabsorption results from depletion of the bile acid pool. Patients with a jejunocolonic anastomosis frequently have rapid small bowel transit due to both reduced intestinal length and loss of the ileum, which has inherently slower transit. It is thought that the early arrival of liquid nutrients in the colon triggers a ‘colonic brake’, mediated by PYY, that reduces the transit rate of the solid meal components more proximally. Loss of the colon is associated (a) (b) (c) Fig. 15.10.7.1 The three types of patient with short bowel syndrome. (a) Jejunocolonic—small intestine and proximal colon have been resected and the small bowel anastomosed to the colon with loss of the ileocaecal valve and some of the proximal colon. (b) Jejunostomy—the small bowel has been shortened by resection(s) and the shortened small bowel ends in a jejunostomy. (c) Ileocolonic—the small bowel has been shortened by resection(s) and the shortened small bowel is anastomosed to residual terminal ileum, leading to the large bowel through the ileocaecal valve in the normal way.

15.10.7 Effects of massive bowel resection 2913 with reduced intestinal adaptation due to lower levels of GLP-2 and faster transit, probably caused by lower levels of PYY. Vitamins and micronutrients Loss of more than 50 cm of terminal ileum can result in vitamin B12 malabsorption and deficiency. Other important nutrients that are particularly prone to deficiency after small-bowel resection include magnesium, zinc, iron, biotin, and selenium. Fat malabsorption is common and can lead to deficiency of essential fatty acids and the fat-soluble vitamins (A, D, E, and K). Management Massive intestinal resection initially results in a high-output state as gut motility returns in the early days after surgery. This acute phase predominantly requires urgent attention to water and electro lyte balance, control of sepsis, and establishment of safe nutrition. Subsequently the more insidious issue of progressive undernutrition, weight loss, and nutrient deficiencies may develop. Finally, with cor rect management, the major nutritional deficiencies are corrected and minor adjustments are needed to optimize long-term outcome and symptoms control. The management can usefully be divided into acute and chronic phases. Acute phase Sepsis Following massive intestinal resection, patients are often critically ill and have uncontrolled sepsis. Adequate nutritional support will be impaired by the presence of sepsis, the source of which needs to be identified and resolved as a priority. Further surgical intervention should be avoided if possible until sepsis is controlled. Radiological drainage of infected fluid collections and appropriate antimicrobial treatment should be the mainstay of treatment. Infection of intra venous feeding lines is a common complication unless scrupulous care is maintained at all times. Specialist microbiological advice is advisable to select the best antibacterial and antifungal agents. Nutritional support During the first few weeks after massive intestinal resection, management of fluid and electrolyte balance is challenging (Table 15.10.7.1). Oral nutrition Oral or enteral nutrition should ideally be started within the first few days after surgery. Patients will usually have a nasogastric or other enteral feeding tube in place. A low-volume polymeric feed is pref erable, which can be stopped when the patient is able to take oral nutrition. Subsequently, small-volume, frequent, solid or semisolid meals should be introduced. The introduction of luminal nutrition tends to exacerbate diar rhoea or increase stomal output. Patients may enter a high-output state and lose quantities in the region of 10 litres per day. Adequate fluid replacement depends upon accurate estimation of losses based on knowledge of those expected according to the remaining intes tine, and measured losses of water and electrolytes from the stoma, urine, and other sites such as abdominal drains and nasogastric tubes. Patients are often critically ill at this stage and abnormal renal losses may need to be

identified and accounted for. Particular attention should be given to abnormal acid–base balance, which is often insidious, as are magnesium and zinc defi ciencies. Early signs of hypomagnesaemia include cramps and par aesthesiae. This can be accentuated by proton pump inhibitors as these medications decrease magnesium absorption. Daily body weights are useful but may not be available in crit ically ill patients. Analysis of urine for sodium concentration and osmolality can assist in the estimation of sodium and water balance, but may be unreliable in the acutely ill patient due to acute kidney injury, and the therapeutic response to finding sodium depletion is inherently reactive rather than preventative. Clinical assessment is often made difficult as peripheral oedema may result from the acute illness rather than indicate intravascular volume overload. Thirst can be a useful guide, as can central venous and arterial blood pres sures. In practice, close observation and use of all these elements is usually adequate to provide enough information to institute an ef fective programme of water and electrolyte replacement. Oral hypotonic or hypertonic fluids should be restricted to 1 litre of hypotonic fluids and 1 litre of an electrolyte solution initially, increasing the restriction further if bowel/stoma output remains high despite appropriate medical management. This is often difficult to achieve as patients commonly have an insatiable thirst, but con sumption of hypotonic fluids will lead to a net loss of water and so dium from the short bowel. For patients receiving enteral tube feeds, the sodium content can be increased by adding sodium chloride to achieve a sodium concentration of 90 to 100 mmol/litre. Feeds with a high osmolality should also be avoided (e.g. elemental feeds). Stomal losses can be ameliorated with the use of proton pump inhibitors, H2 antagonists, loperamide, and codeine phosphate, limiting fluid intake, and drinking an electrolyte solution; taking food and drink separately can also help. Octreotide or its analogues can be used (but may increase the risk of gallstones and liver dys function) in patients with a particularly high output. Patients with an ileal remnant can occasionally benefit from fludrocortisone. In very high-output states (>8 litres/day), it is often necessary, in the initial 1 to 2 weeks, to restrict oral intake to sips of isotonic fluid (i.e. <200 ml per day) and administer nutritional support parenterally. Intravenous nutrition Following massive resection of the small bowel, the function of re maining intestine will be impaired due to the postoperative state, comorbidity such as sepsis, and it will not have had adequate time to start the process of adaptation. For these reasons, many patients will initially be rendered almost entirely dependent on parenteral nutritional support. Patients who are most likely to require paren teral support at this stage include those who are malnourished with little reserve, patients with a prolonged ileus, patients with a stomal Table 15.10.7.1 Management of a high-output state Fluid intake Restrict hypo-osmolar fluid Administer oral glucose-saline solution Antimotility agents Loperamide Codeine phosphate Antisecretory agents Proton pump inhibitors Octreotide

 output of more than 1.5 litres/day, and patients with a short bowel (<100 cm to a jejunostomy or <50 cm to the colon). This can be started on the second or third postoperative day. Anatomy Having secured adequate nutritional support and sepsis control, attention can be refocused to establish the amount and nature of remaining intestine. This usually requires cross-sectional imaging, contrast studies, and (sometimes) endoscopic examination to deter mine gut viability. Surgical procedures During the acute phase, only the most urgent surgery should be undertaken. Examples include the removal of dead gut or necrotic infected tissue that cannot be radiologically drained. Early recon structive surgery to bring the colon back into continuity with a je junal remnant should only be considered if conditions are ideal, which is rarely the case in the scenario of massive resection. Chronic phase Feeding and adaptation As the patient’s condition improves and becomes more stable, efforts should be made

to minimize parenteral nutrition and maximize enteral and oral feeding to encourage intestinal adapta tion. Specific mixtures of electrolyte, sugar, and water which pro mote absorption can often be taken as drinks or given enterally (Table 15.10.7.2). Enteral tube feeding into stomach, jejunum, or via a mucus fistula into a portion of defunctioned distal intestine can be helpful from both the provision of nutrients and also to en courage intestinal adaptation. Hyperadaptation can be induced by the administration of ex ogenous growth factors such as GLP-2 analogues. These were ini tially administered to patients who had already achieved an adapted steady state, but they are now being considered for use earlier in the adaptation process (Fig. 15.10.7.2). Nutritional requirements may reduce with time, due to adaptation, and overfeeding may occur if parenteral nutrition is not adjusted. Socialization The sudden loss of a large portion of the intestine is a cata strophic event with far reaching effects on patients' social life and psychological well-being. Psychological support is essential, and ef forts should be made to facilitate social contact. Education in self- directed care and patient engagement in clinical decision-making are beneficial to long-term outcome. Long-term treatment plan After a period long enough to allow the maximum restoration of en teral nutrition, an assessment of the patient's prognosis can be made. In some cases it may be immediately obvious that the intestinal remnant is too short to provide any prospect of independence from parenteral nutrition, but for others, increases in absorption due to adaptation can continue for about a year (Fig. 15.10.7.2). Bowel length measurements are taken from the duodenojejunal flexure. Patients may manage without parenteral nutrition if the jejunal remnant is longer than 50 cm with colon in continuity or greater than 100 cm to a jejunostomy. Isotonic electrolyte mixes (with a sodium concentration of about 100 mmol/litre, approxi mating to the concentration in jejunostomy fluid) will be required according to the length and function of the remaining jejunum (Table 15.10.7.3). Patient involvement and education is vital to maximize the possibility of weaning from parenteral nutrition, and as with any chronic condition, assessment for psychiatric symp toms such as depression may be important. Gradual undernutrition (protein–energy malnutrition) may occur during this period and can be insidious. Hypomagnesaemia Table 15.10.7.2 Electrolyte mixes commonly used in the United Kingdom Electrolyte solution Amount Measure Comments Concentration (mmol/L) St Mark's electrolyte solution Sodium bicarbonate 2.5 g 1 heaped 2.5-ml spoonful Make up to 1 litre with water Flavourings are best added when it is being prepared Make up a fresh solution every day Keep chilled Na: 90 K: 0 Cl: 60 HCO3: 30 Glucose: 60 Glucose powder 20 g 6 level 5-ml spoonfuls Sodium chloride 3.5 g 1 level 5-ml spoonful Dioralyte solution (double usual strength) Sodium chloride 470 mg Amounts present in one sachet of Dioralyte Reconstitute 2 sachets with 200 ml of water (double usual strength) Make up a fresh solution every day Keep chilled Na: 120 K: 40 Cl: 120 Citrate: 20 Glucose: 180 Potassium chloride 300 mg Disodium hydrogen citrate 530 mg Glucose 3.56 g Early treatment Late treatment Hyperadaptation Accelerated adaptation Spontaneous adaptation Resection Bowel function 1 year 2 years 3 years Fig. 15.10.7.2 Intestinal adaptation following massive intestinal resection and the effects of early and late treatment with GLP-2.

15.10.7 Effects of massive bowel resection 2915 is common and is treated with oral or intravenous magnesium supplements (intravenous: magnesium sulphate; oral: magnesium glycerophosphate or aspartate). Sodium depletion may require attention. Oral 1-α-hydroxycholecalciferol may be required for vitamin D deficiency. Bone health should be monitored and steps taken to prevent osteoporosis. Loss of the ileocaecal valve above residual colon and factors leading to small-bowel stasis may result in small intestinal bacterial overgrowth. This may further impair nutrient absorption in general and is particularly associated with hypomagnesaemia and vitamin B12 deficiency. When long-term parenteral nutrition is required, efforts should be made to provide this

through a specialist centre. A dedicated single- lumen tunnelled intravenous feeding catheter should be placed with the tip situated at an appropriate high-flow site (low superior vena cava or superior vena cava/right atrial junction) to reduce the in cidence of line-related venous thrombosis. Patients need rigorous training in line care if complications are to be avoided. Surgery Optimization of intestinal function Surgical restoration of intestinal continuity should be reconsidered when the patient is stable. This can enhance intestinal absorption and also encourage adaptation. Inclusion of the colon and/or a seg ment of small intestine into the active digestive tract may greatly en hance fluid and electrolyte balance, which is often the most difficult element of intestinal function to restore. A few patients may be suitable for bowel lengthening proced ures: those with dilated intestinal remnants can be considered for procedures such as the STEP (serial transverse enteroplasty) and LILT-Bianchi (longitudinal tailoring and lengthening) procedures, which should be performed only in highly specialized centres and after careful case selection. GLP-2 analogues may be of use for those requiring minimal par enteral nutrition or only fluid and electrolytes, and can result in a significant reduction or removal of the need for intravenous sup port. Their current use is limited, but they have potential for more widespread application as clinical experience with them increases and costs fall. Treatment of small-bowel bacterial overgrowth, which may com plicate short bowel, often leads to improved intestinal function. Transplantation Patients who have irreversible intestinal failure and complications of parenteral nutrition such as loss of venous access or intestinal failure-associated liver disease may be candidates for intestinal trans plantation. Patient survival after isolated intestinal transplantation has greatly improved over the last 10 years and in well-performing centres is now greater than 60% and in some centres greater than 85% at 5 years. The survival rate of patients undergoing multivisceral transplantation is lower, as in addition to the intestine, other organs such as the liver, pancreas, duodenum, and (often) stomach and kidney are transplanted in a cluster graft. Patients commonly have complex preoperative comorbidities and the operative procedure is much more complicated. The postoperative management of patients is particularly challenging and requires good cooperation between numerous medical and surgical specialists. Survival on parenteral nutrition remains better than after trans plantation in most centres, and is the best option for most patients. However, if the recent improvements in survival figures are sus tained at 10 years, transplantation will become a reasonable alter native to parenteral nutrition as the primary treatment for many patients during the next decade. Long-term complications Colonic oxalate absorption is often increased due to malabsorbed fatty acids binding luminal calcium which would otherwise form insoluble calcium oxalate. Additionally, bile salt malabsorption in creases mucosal permeability to oxalate and bacterial degradation of oxalate is reduced. This may result in renal oxalate stones, which occur in about 25% of patients with jejunocolonic anastomosis. Strategies to avoid this include a low-oxalate diet with restricted long-chain triglycerides, and oral calcium supplements. Pigment gallstones are common (about 45% of patients with short small Table 15.10.7.3 Estimated long-term fluid and nutritional supplements and common complications in patients with short bowel Jejunum-colon Jejunostomy Length of remaining jejunum (cm): <50 Parenteral nutrition Parenteral nutrition and IV electrolyte solution <100 Oral/enteral nutrition IV electrolyte solution ± parenteral nutrition <150 No supplement Nutritional supplement (oral/enteral) and oral sodium/glucose solutiona <200 No supplement Oral sodium/glucose solutiona Renal calculi (calcium oxalate) 25% None Biliary calculi (pigment) 45% 45% d-lactic acidosis Infrequent No Adaptation Yes No a Oral sodium/glucose solutions are approximately iso-osmolar. Note: estimates vary between individuals. Patients with jejunoileal anastomosis generally have fewer complications than jejunocolonic anastomosis. Patients with a high-output enterocutaneous fistula can be considered in the same group as those with an end jejunostomy.

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