

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 arterial, central or peripheral. The superior mesenteric vessels are the visceral vessels most likely to be affected by embolisation or thrombosis. Occlusion at the origin of the SMA is almost invariably the result of thrombosis, whereas emboli tend to lodge at the origin of the middle colic artery. Inferior mesenteric 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 infarction, vegetations on mitral and aortic valves associated with endocarditis or an atheromatous plaque from an aortic aneurysm. Primary thrombosis is associated with atherosclerosis and vasculitides, including conditions such as thromboangitis obliterans and polyarteritis nodosa. Primary thrombosis of the superior mesenteric veins may occur in association with factor V Leiden disorder, portal hypertension, portal pyaemia, sickle cell disease and in women 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 complicate 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 mesenteric ischaemia is sudden onset of severe abdominal pain in a

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. Abdominal tenderness may be mild initially, with rigidity being a late feature. Shock, with features of both hypovolaemia and sepsis, rapidly ensues. Investigation will usually reveal a profound

neutrophil leukocytosis, 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 anticoagulation 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 postoperative anticoagulation. However, the condition is usually diagnosed late in the disease process and the mortality rate is extremely high. In the young, all affected 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 sufficient intestinal digestive and absorptive function to lead relatively normal lives. In selected cases, consideration may be given to small bowel transplantation (see Chapter 91).

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