A 78-year-old woman presented to the emergency department with a 12-h history of sudden-onset abdominal pain. She had vomited after the pain started, and she had also had two episodes of diarrhoea. Until this time, she had been well, although she was known to be in atrial fibrillation and took digoxin 125 mg daily.

On examination, she was distressed and obviously in pain. Baseline observations revealed a pulse of 110 bpm, irregularly irregular, blood pressure of 95/60 mm Hg, respiratory rate of 28 breaths/min, and temperature of 37.3°C. Her chest was clear, heart sounds were normal (irregular rhythm), and the jugular venous pressure was not elevated. Abdominal examination was unremarkable, with a soft abdomen and minimal tenderness despite severe pain, and normal bowel sounds.

The investigations shown in Table 25.1 were performed by the admitting surgeon.

Electrocardiogram (ECG) revealed atrial fibrillation with no other acute changes. Erect chest X-ray revealed normal lung fields and no free gas under the diaphragm. Abdominal radiography was unremarkable except for minimal small-bowel distension.

**Question 1**

Which of the following is the most unlikely diagnosis?

A. Acute ulcerative colitis.
B. Pancreatitis.
C. Mesenteric venous thrombosis.
D. Acute mesenteric ischaemia (AMI).
E. Diabetic ketoacidosis.
Table 25.1. Investigations performed by the admitting surgeon.

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urinalysis</td>
<td>No abnormality</td>
</tr>
<tr>
<td>Biochemistry</td>
<td>Na⁺ 139 mmol/l</td>
</tr>
<tr>
<td></td>
<td>K⁺ 4.6 mmol/l</td>
</tr>
<tr>
<td></td>
<td>Creatinine 112 mmol/l</td>
</tr>
<tr>
<td></td>
<td>Glucose 6.1 mmol/l</td>
</tr>
<tr>
<td></td>
<td>Amylase 2000 IU/l</td>
</tr>
<tr>
<td>Haematology</td>
<td>Haemoglobin 12.3 g/dl</td>
</tr>
<tr>
<td></td>
<td>White cell count 27,000</td>
</tr>
<tr>
<td></td>
<td>Platelets 235,000</td>
</tr>
<tr>
<td>Arterial blood gas</td>
<td>pH 7.21</td>
</tr>
<tr>
<td></td>
<td>pCO₂ 3.2 kPa</td>
</tr>
<tr>
<td></td>
<td>pO₂ 9.4 kPa</td>
</tr>
<tr>
<td></td>
<td>HCO₃⁻ 17 mmol/l</td>
</tr>
<tr>
<td></td>
<td>Base excess –8</td>
</tr>
</tbody>
</table>

**Question 2**

What are the most common causes of AMI?

A. Renal failure.
B. Atrial fibrillation.
C. Multi-organ failure.
D. Anti-phospholipid syndrome.
E. Atherosclerotic disease

**Question 3**

Which of the following tests are of use in the acute management of a patient with AMI?

A. Echocardiography.
B. Lateral-view mesenteric angiography.
C. Thyroid function tests (TFTs).
D. Non-contrast computed tomography (CT) scanning.
E. Mesenteric vessel duplex Doppler.

At this point, the patient was taken to the high-dependency unit, where the following measures were undertaken: high-flow oxygen therapy by mask (15 l/min), continuous ECG monitoring, central venous pressure (CVP) monitoring, urinary catheter inserted to monitor urinary flow hourly, and infusion of 4 litres of fluid resuscitation. Intravenous broad-spectrum antibiotics and an anticoagulant dose of...
Intravenous heparin were also given. After 2 h of resuscitation, the patient’s blood pressure was 130/85 mm Hg, pulse 100 bpm and CVP +8 cm water. She was still in a lot of pain despite 10 mg of diamorphine, and she was still tachypnoeic. Repeat blood gas and blood count investigations were as in Table 25.2.

Because the patient was persistently acidotic with an elevated white count and in severe pain, she was taken to the operating theatre for an emergency laparotomy. Almost the entire small bowel and most of the large bowel were found to be ischaemic but viable. There was a pulse in the proximal superior mesenteric artery (SMA) but nothing was palpable beyond the origin of the middle colic vessel.

**Question 4**

What operative options are available to achieve restoration of flow to the bowel?

A. Full heparinisation.
B. Catheter thrombectomy.
C. Axillofemoral bypass.
D. Mesenteric bypass with a vein graft.
E. Mesenteric bypass with prosthetic graft.

Clot was removed successfully from the SMA. However, despite the majority of the bowel receiving a good blood supply, several areas remained dusky in appearance.

**Question 5**

What features of the bowel’s appearance determine whether it is viable?

A. The presence of peristalsis.
B. Lack of foul odour from the peritoneal cavity.
C. Serosal sheen.
D. Mesenteric pulsation.
E. Active bleeding from the cut surface of the bowel at the time of resection.
**Question 6**

Having determined that an area of the bowel is non-viable, what action should you take?

A. Revascularise the bowel, then remove that which is non-viable.
B. Remove the non-viable bowel, then revascularise the remaining bowel.
C. Resect all non-viable bowel and primarily anastomose ends; then close the abdomen.
D. Close the abdomen and start the patient on an intravenous infusion of diamorphine.
E. Resect all non-viable bowel and exteriorise viable ends; plan relook laparotomy.

**Commentary**

The incidence of AMI is approximately 1/100,000 in the UK and USA [1, 2]. AMI is a life-threatening vascular emergency. It accounts for 0.1 per cent of emergency admissions [3], and based on several large series it has a mortality of between 60 and 100 per cent [3–7]. Females are affected twice as often as males, and the median age at presentation is 70 years [6].

The clinical presentation is often not as clear-cut as described in the case above. However, some if not all of the described features will be present. One must have a high index of clinical suspicion in anyone aged over 55 years who presents with abdominal pain out of proportion to the physical signs elicited on abdominal examination [8]. The diagnosis should also be considered in patients with known peripheral arterial disease and abdominal pain. The triad of abdominal pain, a cardiac source of embolus and gut emptying, as described by Klass [9], make AMI the most likely diagnosis. In addition to this triad, the finding of a marked leucocytosis, metabolic acidosis and hyperamylasaemia are also suggestive of AMI. It is also not unusual for there to have been a history of previous embolic events [8]. Pancreatitis can be difficult to differentiate from AMI, and laparotomy is indicated if suspicion of AMI is aroused. [Q1: E]

Defining the aetiology of AMI is important as the different causes have different treatments. The most common presentation, as described in our case, is of superior mesenteric embolus; this accounts for about 50 per cent of all cases [3, 5–7]. The usual source for these clots is the atria in patients in atrial fibrillation or the ventricle if the patient has recently sustained a myocardial infarction. Another potential source of emboli is atheroma from the aortic wall following radiological procedures in which catheters and guidewires have been passed up the aorta. The consequence of such an event can be catastrophic, as the mesenteric circulation has not had time to develop a collateral circulation.

The next most frequent aetiology is SMA thrombosis, which accounts for between 25 and 50 per cent of cases [3, 5–7]. This results from progression of atheromatous disease at the origin of the SMA. It is important to note that a long-standing stenosis may have caused symptoms of chronic mesenteric ischaemia in the months before its ultimate occlusion [10]. Therefore, in a patient with pre-existing symp-
toms of mesenteric ischaemia, sudden onset of abdominal pain should be regarded as AMI until proven otherwise.

Non-occlusive mesenteric ischaemia (NOMI), first described by Ende in 1958 [11], is the next most frequently encountered condition, occurring in about 20 per cent of cases [3, 5, 6]. In this situation, the patient is often critically ill from another cause and the mesenteric ischaemia is due to vasoconstriction leading to reduced flow in the splanchnic circulation. This may be due to cardiogenic shock, hypovolaemia or vasoconstricting inotropes. Even after reversal of shock, mesenteric hypoperfusion may persist for several hours [12, 13].

Mesenteric venous thrombosis (MVT) is the least common cause and accounts for about 5 per cent of AMI [14–16]. The thrombotic process is thought to start in the superior mesenteric vein and spreads to the portal vein; the inferior mesenteric vein is usually spared. The onset of symptoms is more insidious and may have a history of several days. It is caused by the same provoking factors that one finds in any thrombotic situation: sluggish flow, clotting abnormality and vessel wall damage (Virchow’s triad). It is associated most commonly with hypercoagulable states, abdominal trauma or intra-abdominal sepsis [17–20]. The diagnosis is often made at laparotomy and encompasses a spectrum of severity from segmental mesenteric venous thrombosis to the entire portal vein being thrombosed.

Diagnostic confirmation of AMI poses a dilemma. Should one delay in order to confirm a suspicion and risk converting a salvageable situation into a non-salvageable one [21]? There is little evidence on which to base sound advice. However, if the patient is cardiovascularly stable with minimal symptoms, and one has prompt access to angiography, then this provides accurate diagnosis (Fig. 25.1). Some authors recommend colour-flow duplex at the bedside during the resuscitation phase [22, 23]; this procedure is less time-consuming than angiography, but it requires considerable skill that is not always available. Furthermore, good views are often hampered by obesity and/or overlying bowel gas.

Transthoracic echocardiography is useful in identifying a cardiac source for emboli and may help in making the decision regarding postoperative anticoagulation. However, it is not as sensitive as transoesophageal echo in searching for left atrial emboli and may waste valuable time.

Contrast-enhanced CT may be of use in identifying mesenteric venous thrombosis [24]. This will not prevent laparotomy as bowel resection may well be necessary. If there is clear evidence of peritonism and a high index of suspicion for AMI, then the patient should be resuscitated rapidly and this should be followed by urgent laparotomy. [Q3: B, E]

In order to answer Question 4, one has to be confident of the aetiology of AMI. In our case, there is embolus in the SMA. The abdomen should be approached through a long midline incision, which will afford excellent exposure. Having entered the abdomen, a quick survey of the viscera and extent of ischaemia should give some information on the aetiology of the AMI (see below). At this point, the main aim of surgery is to restore flow to the ischaemic viscera if viable. In order to do this, the transverse mesocolon is elevated and the ligament of Treitz identified and the fourth part of the duodenum mobilised. The root of the mesentery is palpated to feel for the SMA pulse. If, as in our case, the AMI is due to an embolic event, then a proximal SMA pulse should be palpable and the duodenojejunal flexure and proximal few centimetres of jejunum should be viable. The emboli usually lodge at a variable site 3–8 cm
from the origin of the SMA, usually at the point where the middle colic artery arises [6, 8, 21] (Fig. 25.2a, b).

In order to expose the SMA, the inferior leaflet of the mesocolon is entered along the course of the vessel and a 5-cm section of artery is dissected out and slung with Silastic sloops. At this point, if the patient has not been heparinised previously, then they should be given an intravenous dose of 5000 units of unfractionated heparin. A longitudinal arteriotomy is made in the cleared SMA (on the left margin of the vessel, to permit easier graft placement, should a bypass be necessary) [21], and a size 3 or 4 Fogarty embolectomy catheter is passed up and down the vessel to retrieve the embolus. Once adequate backward and forward flow has been achieved, the vessel should be flushed with heparinised saline; the arteriotomy can then be closed primarily or with a patch (depending on size), using a 6/0 or 7/0 Prolene suture.

At this point, the anaesthetist should be warned that you are about to reperfuse the viscera. Metabolites that have accumulated in the ischaemic viscera will pass rapidly from the mesenteric venous circulation into the systemic circulation, which can precipitate circulatory collapse and, over several hours, result in the development of systemic inflammatory response syndrome (SIRS). There is no point in revascularising dead bowel; indeed, this is dangerous. Irrefutably dead bowel should be removed before revascularisation. It is important to note that if, on
opening the abdomen, the entire small bowel is black and irreversibly ischaemic, then the most appropriate thing to do is close the abdomen and keep the patient comfortable with morphine: death will usually follow within a matter of hours.

In the event that there is no pulse palpable at the SMA origin, then the revascularisation strategies are similar to those for chronic visceral ischaemia (see Chapter 24). In this case, attempts at thrombectomy will fail as the catheter will not cross the occlusion. The options available are retrograde bypass from the infrarenal aorta to the SMA, antegrade bypass from the supracoeliac aorta (with concomitant revascularisation of the coeliac trunk if it is occluded), aortomesenteric endarterectomy, or side-to-side anastomosis between the SMA and aorta. Prosthetic material should be avoided since transmural migration of bacteria is likely to contaminate the graft. Also, if the lower aorta and iliac systems are heavily calcified, then it may be better

Fig. 25.2. a, b Patchy mid-gut infarction due to atherosclerotic occlusion of superior mesenteric artery.
to select the supracoeliac aorta for the inflow site of the bypass. The use of a temporary shunt for immediate reperfusion while the bypass is being constructed seems sensible [21].

At this point in the procedure, the viscera need to be inspected again and any dubiously viable bowel resected. If the patient is otherwise young and fit, then it may be better to resect all the necrotic bowel, exteriorise the remaining ends, and consider long-term total parenteral nutrition or small-bowel transplantation. If there is no evidence of arterial compromise and a pulse is palpable in the SMA, then one should suspect either NOMI or MVT.

NOMI usually results in widespread patchy ischaemia. Treatment entails resection of obviously non-viable bowel, attempting to be as conservative as possible. The remaining bowel must be inspected at a second-look laparotomy 24–48 h later. During surgery, several strategies have been described to improve mesenteric flow: a combination of systemic dopamine and an opiate epidural [25], papaverine infusion (30–60 mg/h) into the SMA either via direct puncture fine bore or angiographically placed catheter [13, 26, 27]. At relook laparotomy, the extent of the ischaemia should be reassessed; treatment may need to be abandoned if the gangrene is progressing. Mortality of this condition is depressingly high at 70–80% despite the therapeutic measures outlined above [13].

The classic laparotomy findings in MVT are ascites and swollen omentum with bowel infarction. Like NOMI, MVT is managed by resection of non-viable bowel, which is most frequently sharply demarcated and found in the mid-small bowel. Again, second-look laparotomy is mandatory. Anticoagulation with heparin and then warfarin is mandatory in view of the high incidence of recurrent MVT. Investigation of any underlying prothrombotic disorder along with long-term anticoagulation improves survival [28, 29].

Thrombolysis has been used successfully in MVT in two studies where the diagnosis had been established by non-invasive means and peritoneal signs had not developed [30, 31]. Venous thrombectomy has also been reported to be successful in a handful of cases [19, 32–34]. This procedure is likely to be difficult, as the peritoneum is usually oedematous. [Q4: B, D]

The classic features of ischaemia are oedema, loss of peristalsis, loss of surface sheen, staining of the serosa, absent mesenteric pulsation, or frank gangrene with or without perforation (Fig. 25.3). The decision at surgery is, after revascularisation (providing it was appropriate or feasible), how much small bowel to resect. Once there is good flow to the viscera, then reversibly ischaemic segments should declare themselves viable and the rest will need to be resected. Other adjuncts to inspection and palpation are continuous-wave Doppler, pulse oximetry and fluorescein dye [35]. The next question is whether to exteriorise or anastomose the open ends of the bowel. The arguments against exteriorising are that many stomata may be necessary and they do not guarantee that the intervening segments may not subsequently become ischaemic; however, this is extremely safe and avoids the complication of a necrotic anastomosis. Performing primary anastomoses and leaving the abdomen open and covering it with a see-through bag (a cut-open bag of saline) [36] allows direct visualisation of the viscera at all times; second-look laparotomy can then be planned on the appearance of the gut [36]. If all looks well by 72 h and the patient’s condition is stabilising, then they can be returned to theatre for planned abdominal closure. [Q5: A, C, E], [Q6: B, E]

The short-term management of these cases is demanding on staff and time, but if best results are to be achieved, then no short cuts can be taken.
AMI is a treatable vascular emergency. It requires a high index of clinical suspicion, rapid aggressive resuscitation and diagnostic manoeuvres to determine the specific underlying cause. This will allow a prompt, directed revascularisation procedure after optimisation of cardiac performance, or correction of a hypercoagulable state. This effort is directed at maximising the amount of salvageable bowel. These strategies are the cornerstones for a successful outcome in this life-threatening vascular catastrophe.

References