Mesenteric Ischaemia and Ischaemic Colitis

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16.1 Mesenteric Ischaemia

Acute mesenteric ischaemia is a catastrophic abdominal emergency. The critical feature is a sudden interruption to mesenteric blood flow which often leads to bowel infarction and death. Most mesenteric ischaemia is acute but there can be chronic presentations.

Mesenteric vascular compromise can be arterial (embolic, thrombotic), venous (thrombotic), or non-occlusive (vascular narrowing).

Embolism is the commonest cause of mesenteric ischaemia. The majority of emboli arise from the left atrium in patients with atrial fibrillation. The superior mesenteric artery is most commonly affected.

Thrombosis has a worse prognosis and usually occurs in the presence of atherosclerosis. There is often a history of fear of eating, intestinal angina, and weight loss.

Non-occlusive disease results from hypoperfusion including congestive cardiac failure and shock. Surgical patients may have intense vasoconstriction due to treatment of shock with vasoconstrictors. Arterial vasospasm may persist even after correction of the underlying event. Overall prognosis is poor.

Acute mesenteric ischaemia due to venous thrombosis most commonly affects the superior mesenteric vein. It is very uncommon. Causative factors are hypercoagulable states (e.g., Factor V Leiden mutation and Protein C, S, antithrombin III deficiency) or acquired hypercoagulable states (e.g., malignancy, portal hypertension, oral contraceptives, intra-abdominal sepsis, and post abdominal surgery).

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F. Piscioneri et al. (eds.), *Emergency Surgery for Low Resource Regions*,
Hot Topics in Acute Care Surgery and Trauma,
https://doi.org/10.1007/978-3-030-68099-2_16

16.1.1 Presentation

Severe abdominal pain that is out of proportion with the clinical signs is present in over 95% patients. Other early symptoms include nausea, vomiting, and diarrhoea. Late symptoms are indicative of progression of the ischaemia and include bloody diarrhoea, abdominal distension, and eventually signs of peritonitis (fever, tachycardia, and shock).

16.1.2 Pathophysiology

The ischaemic insult leads to mucosal barrier disruption which has two main effects.

Bacteria, toxins, and vasoactive compounds are released and lead to systemic inflammatory response syndrome (SIRS) which can lead to multi organ dysfunction syndrome (MODS) and eventually death.

Large amounts of protein-rich fluid is released into the gut leading to hypovolaemia which if untreated leads to shock.

If there is complete vessel obstruction with no collateral flow, then absolute ischaemia will occur. There is then a rapid progression with mucosal sloughing in the first 3 h. At this stage, the damage is still reversible. Within 6 h there is transmural necrosis, gangrene, perforation, and peritonitis.

16.1.3 Investigations

Blood findings are non-specific and include haemoconcentration, leucocytosis, metabolic acidosis, and lactic acidosis.

Plain abdominal x-rays may show dilated bowel loops, 'thumb-printing' sign indicating bowel wall oedema and thickening, gas in the bowel wall (pneumatosis intestinalis), and gas in the portal vein.

CT angiogram is the main imaging modality. It can demonstrate occlusions and narrowing in the mesenteric arteries, bowel wall oedema, gas in the bowel wall and in the portal vessels, and pneumo-peritoneum.

16.1.4 Management

Look for history and examination consistent with ischaemic bowel. Pain will be out of proportion with the clinical signs. Look for risk factors.

If peritoneal signs are present, it is likely that there is a breach of the intestinal wall and the patient should have an immediate laparotomy.

If there are no peritoneal signs, the patient proceeds along a diagnostic pathway. Once acute mesenteric ischaemia has been established the following treatment should be commenced;

- oxygen therapy
- fluid resuscitation
- nasogastric tube to decompress the stomach and bowel rest
- broad-spectrum antibiotics
- heparin 5000U IV followed by a therapeutic infusion
- invasive monitoring in HDU setting for the more severely ill patients
- treat arrhythmia/heart failure

16.1.5 Surgical Exploration

Approach is via a midline laparotomy. Necrotic bowel should be excised. Only consider re-anastomoses if there is good blood supply to the bowel ends and the patient is not requiring circulatory support with vasopressors or inotropes. If there is any doubt about the security of a primary anastomosis the bowel ends should be closed and a second look laparotomy performed the next day to assess the viability and to perform a bowel anastomosis. During this time the patient's physiology is able to be restored in the intensive care unit. If there is extensive bowel necrosis with little or no residual healthy bowel the situation becomes unsalvageable and is best to close the patient and proceed to palliation.

If there is no obvious infarction but there are signs of ischaemia then there are two options. If vascular expertise is available angiography and clot retrieval should be performed. If this service is not available then the patient should be closed, continued with heparin infusion, and closely monitored. Further deterioration warrants a second look laparotomy.

Intravenous papaverine should be considered for 24 h in those patients who have had a revascularisation procedure as arterial spasm can continue after revascularisation has been successful.

If a second look laparotomy is mandatory, such as in the case of having to perform a bowel anastomosis, the abdomen can be left open with a suitable covering such as a vacuum dressing. There is no place for routine open abdomen. Close observation and selective second look laparotomy is the preferred course of action. This should be performed within 24–48 h.

16.1.6 Non-Occlusive Mesenteric Ischaemia

Treatment is non-operative unless peritoneal signs develop. In such situations, the patient requires a laparotomy and surgical management of the findings.

Non-operative management consists of;

- correction of underlying disorder (e.g., treatment of shock)
- elimination of vasopressors
- optimising fluid status and cardiac output
- arterial catheter infusion of vasodilator (e.g., papaverine)

16.2 Mesenteric Venous Thrombosis

Treatment is mainly non-operative with heparin infusion. A search should be made for a hypercoagulable state and this should be managed long-term. Any development of peritoneal signs should be treated with laparotomy and bowel resection as required.

16.3 Low Resource Regions

Mesenteric ischaemia can be managed in limited-resource regions. The diagnosis can be made clinically and the patient managed either operatively or non-operatively according to the abdominal signs.

Mortality, even in the best of centres, approaches 80%.

16.4 Ischaemic Colitis

Ischaemic colitis is defined as a sudden and often temporary reduction in colonic blood flow that is insufficient to meet the metabolic needs of parts of the colon.

The colon derives its blood supply from the superior mesenteric and inferior mesenteric arteries. The Marginal Artery provides collateral circulation in the mesentery between the two arterial systems. There are watershed areas of potential diminished blood flow at the splenic flexure and the recto-sigmoid junction. The blood supply to the colon is less than that of the rest of the gastrointestinal tract and thus is more vulnerable during periods of hypotension.

It affects males and females equally, is more common in the left colon, and in the over 60 age group. It has similar risk factors as small bowel ischaemia but in addition can be affected by bowel obstruction and colonic infections.

Hypoxia leads to tissue necrosis with an inside to out progression. Transmural infarction can occur in 8–12 h.

16.4.1 Presentation

The presentation is in three phases.

The initial hyperactive phase is with severe abdominal pain and usually blood stained loose stools. This is then followed by paralytic phase characterised by the pain diminishing but becoming more continuous and generalised, the abdomen distending, and bowel sounds becoming absent.

In 10–20% of patients, there is progression to a shock phase which features a very large fluid, protein, and electrolyte transfer through damaged gangrenous mucosa, metabolic acidosis, and shock. These patients require urgent surgical intervention to manage the necrotic bowel and associated peritonitis.

16.4.2 Diagnosis

History and examination are usually suggestive of the diagnosis. In the early stages, pain is out of keeping with the physical signs but with progression patient develops features of peritonism.

Laboratory investigations include;

- white cell count above 20,000
- elevated lactate, LDH, and ALP
- metabolic acidosis
- stool culture to rule out an infective cause

16.4.3 Imaging

Plain abdominal x-ray may show dilatation of a segment of the colon in the early stages. This later progresses to loss of haustrations and pneumatosis of the colonic wall.

CT scanning is a valuable investigation tool. Things look for include;

- disruption of the mesenteric vessels indicative of thromboembolism
- irregular narrowing of the bowel lumen with associated bowel wall oedema
- pneumatosis in both the bowel wall and the portal venous system
- dilatation of the bowel proximal to the ischaemic segment

Colonoscopy can be used if the diagnosis is uncertain. It should only be used in the early stages and can show mucosal ischaemia or necrosis. It should not be used once the patient develops signs of bowel atonia as the risk of perforation of potentially necrotic bowel wall is extremely high.

Barium enema is less diagnostic than CT scanning. Features include thickened bowel wall, segmental luminal narrowing, with stricture formation in the latter stages of the disease.

16.4.4 Surgical Management

There is a role for laparoscopy in the diagnostic workup. It is useful for ruling out gut ischaemia where other investigations have been inconclusive. The other role for laparoscopy is to determine the extent of established ischaemia in order to evaluate whether the patient is a suitable candidate for laparotomy and bowel resection, or whether the disease is so extensive that the best course of action is palliation thus avoiding an unnecessary laparotomy.

About 20% of patients will require surgical intervention whilst the remainder can be managed non-operatively with bowel rest until there is return of intestinal function and diminution of pain.

Acute surgical intervention is indicated for patients with peritoneal signs and those with persistent fevers and sepsis. All affected bowel should be resected and the mucosa of the resected margins inspected for viability. Questionable ischaemia should also be resected unless there is an extensive disease where a second look procedure can be done at 24–48 h to recheck viability of the colon. Primary anastomosis is usually avoided as it results in a high risk of anastomotic leakage in the acute setting. The usual procedure being an end colostomy with the distal segment either closed off or formed into a mucous fistula. If there is an extensive disease of the colon then total colectomy and terminal ileostomy are performed.

At the second look procedure anastomosis can be considered if there is clinically viable tissue and the patient is not requiring inotropic or vasopressor support.

In those patients that require surgical intervention, the mortality rate approaches 50–75%.

There is a small role for elective surgery in patients who have developed strictures or in those where bleeding and diarrhoea persists.

16.4.5 Low Resource Regions

Patients with ischaemic colitis can be well-managed in low resource regions. The diagnosis can usually be made clinically and most cases can be treated non-surgically. In moribund patients, a mini laparotomy can be performed to inspect the bowel to help determine the extent of disease and necrosis. This can often be done with simple local anaesthetic and sedation.