OPEN ACCESS TEXTBOOK OF GENERAL SURGERY

MESENTERIC ISCHAEMIA

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INTRODUCTION

Mesenteric ischaemia results from hypoperfusion of the gut, most commonly due to occlusion, thrombosis or vasospasm. The clinical sequelae are dependent upon the number of vessels affected, the adequacy of collateral circulation and the duration of the insult. Early diagnosis and treatment is imperative, otherwise outcomes can be catastrophic. Mesenteric ischaemia can either be acute or chronic.

ACUTE MESENTERIC ISCHAEMIA

EPIDEMIOLOGY

Acute mesenteric ischaemia (AMI) is relatively uncommon, but probably remains under diagnosed.
Incidence of AMI: ~ 12.9/100,000 person years
Median age at presentation is 70 years, with an exponential increase in incidence of AMI with age. There is no significant gender difference.

AETIOLOGY

There are four principal causes of acute mesenteric ischaemia:

- Arterial embolism (usually cardiac, due to atrial fibrillation (AF) or mural thrombus following myocardial infarction)
- Arterial thrombosis (on pre-existing atherosclerosis of the coeliac axis or more commonly SMA)
- Venous thrombosis
- Non-occlusive mesenteric ischaemia (NOMI).
- Other rare causes include aortic dissection, aneurysmal disease and vasculitides.

The commonest cause of AMI is embolic occlusion of the SMA (50% of all cases); 20% are due to thrombosis and 15% are due to NOMI.

ACUTE MESENTERIC ARTERY OCCLUSION

PATHOPHYSIOLOGY

Three main arteries supply the bowel:

- Coeliac axis - to the foregut (distal oesophagus to the second part of duodenum)
- Superior mesenteric artery - to the midgut (third part of duodenum to mid-transverse colon)
- Inferior mesenteric artery - to the hindgut (mid-transvers colon to rectum).

The intestine is able to compensate for a 75% acute reduction of blood flow for up to 12 hours without substantial injury but at pressures less than 40mmHg the gut becomes ischaemic. After 15 minutes of absolute ischaemia, changes in villi can be seen; after 3 hours the mucosa sloughs off and after 6 hours, trans mural necrosis occurs, resulting in perforation, sepsis and death. The effects of arterial occlusion however may be mitigated by the presence of collaterals, more common in thrombosis than emboli, since they have time to develop as a result of the underlying stenotic disease. Thrombotic occlusion tends to be more proximal than embolic occlusions, and the extent of bowel infarction more severe.

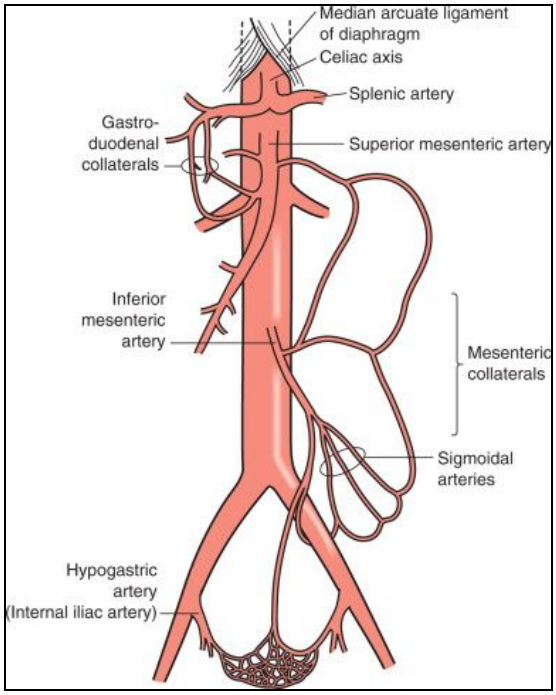

Fig. 1 Mesenteric artery anatomy and collateral

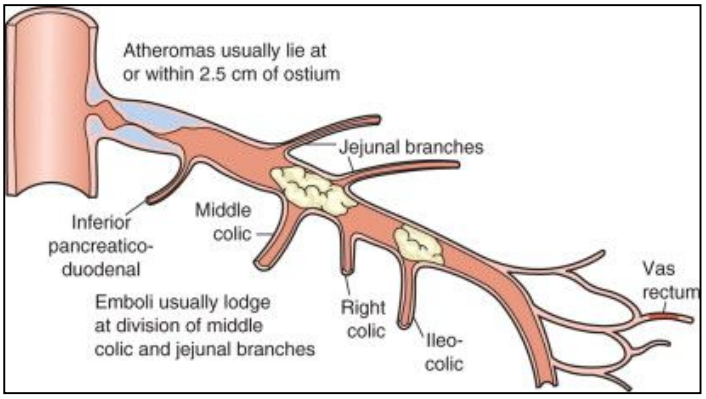

Fig. 2 Typical location of SMA obstruction in patients with embolic and thrombotic occlusion

CLINICAL PRESENTATION

Severe abdominal pain

- Pain is disproportionate to clinical abdominal findings.
- Sudden onset pain.
- Located centrally or in the epigastrium.
- Pain may be colicky initially and then becomes constant in nature.

Associated nausea and vomiting and diarrhea, which may contain blood (late sign)

A history of recent cardiac events such as arrhythmias (i.e. atrial fibrillation) or recent myocardial infarction may be present.
Patients may typically have other manifestations of diffuse atherosclerotic occlusive disease or a history of chronic mesenteric ischaemia.

On examination

- Acutely ill patient with hypotension and tachycardia.
- Abdominal distention may be present.
- Bowel sounds may be normal or absent.
- Peritonitis is a late sign suggesting bowel infarction or perforation.

INVESTIGATIONS AND DIAGNOSIS

Laboratory investigations:

- WCC, CRP and serum amylase may be raised but are nonspecific
- Metabolic acidosis with high serum lactate

Imaging investigation:

- Plan abdominal X-ray may show dilated small bowel loop, bowel wall oedema and gas in the bowel wall and portal venous system. Free air may be seen if perforation has occurred.
- CT Angiogram (93% sensitivity and 96% specificity)
    - Filling defect in coeliac axis or SMA
    - Pneumatosis intestinalis
    - Portal vein gas
    - Bowel wall thickening
    - Solid organ infarction

  CTA can also assess vascular anatomy and other pathology.

MANAGEMENT

General management

- Aggressive fluid resuscitation, correction of electrolyte imbalance, urinary catheter, broad-spectrum antibiotics.
- Heparin IVI, bolus 5000U followed by continues infusion to maintain an aPTT 2 x normal (heparin prevents thrombus propagation)

Surgical management

- Exploratory laparotomy
    - Is there ischaemic bowel (or another diagnosis)?
    - How much bowel is salvageable (is this compatible with life)?
    - Is revascularization an option?
- All ischaemic bowel needs to be resected. A minimum of 50-70cm small bowel is required to maintain life. If this is not possible a decision to close the abdomen and palliate the patient is appropriate. If bowel viability is questionable a relook laparotomy can be done 24-48hr later.
- If bowel appears salvageable consider revascularization.
    - SMA embolectomy if embolic cause.
    - SMA bypass if thrombotic cause.
- Endovascular management options are available but require careful patient selection.
    - Aspiration thrombectomy.
    - Thrombolysis.
    - Angioplasty and stenting.

PROGNOSIS

Thirty-day mortality ranges from 32 to 81% with a mean of 68%
Factor associated with poor outcome

- Age
- Extensive bowel necrosis
- Peritonitis and bowel perforation
- Co-morbidities

AMI is associated with significant morbidity and mortality and the only way to achieve meaningful improvements in outcome is to rapidly diagnose and treat the underlying aetiology.

MESENTERIC VEIN THROMBOSIS (MVT)

- MVT accounts for 5 – 10% of AMI.
- Common underlying causes associated with a risk for MVT
    - Thrombophilia (e.g. factor V Leiden, antithrombin III deficiency, anticardiolipin antibodies)
    - Portal hypertension
    - Intra-abdominal malignancies
    - Pancreatitis
    - Pregnancy
- Symptoms are similar to AMI but with a more insidious onset presenting over weeks or months with vague abdominal pain.
- CT is the mainstay of diagnosis with 90% accuracy.
- Treatment is systemic anticoagulation.
- Patient should be closely monitored for signs of worsening bowel ischaemia, perforation and systemic inflammation.
- Patient with MVT should undergo hypercoaguable workup to identify any underlying aetiology.

NON-OCCLUSIVE MESENTERIC ISCHAEMIA (NOMI)

- NOMI develops in patients with severe systemic illness associated with shock and

multi-organ failure.

- Typically in patients in ICU with cardiac dysfunction on inotrope support causing severe intestinal vasospasm.
- Management is usually non-operative, and treatment of the underlying pathophysiological process.
    - Optimize patient haemodynamics.
    - Elimination of inotrope therapy.
    - Correction of systemic factors contributing to shock.
    - Intra-mesenteric infusion of papaverine may be beneficial to relief the vasospasm.
- Mortality in patients with NOMI is high at 70 – 80%.

CHRONIC MESENTERIC ISCHAEMIA (CMI)

Epidemiology

There are no population-based studies to indicate the true incidence of CMI, but symptomatic disease is quite rare. At least two of the visceral arteries need to be affected to cause symptoms due to the good collateral supply. CMI is commoner in women than men and most patients present between 50 and 70 years of age.

Aetiology and Pathology

About 95% of CMI is due to atherosclerosis and usually affects the origins of the mesenteric vessels. Risk factors of atherosclerosis also apply to these patients.

Clinical Presentation

'Mesenteric angina', a stereotypical pattern of post-prandial, pain peri-umbilical, usually occurs ~ 30 min after a meal. It generally resolves thereafter, only to recur with subsequent meals.

- Weight loss due to fear of food
- Nausea and vomiting

On examination the patient is thin and abdominal bruit may be heard, together with other stigmata of generalized arterial disease.

Diagnosis and Investigations

Diagnosis requires a careful history and exclusion of other illnesses such as intra-abdominal malignancy, chronic pancreatitis, and gastric ulcer.

- Laboratory investigation are usually non-specific
- Imaging investigations include
    - Duplex ultrasound
    - CTA or MRA
    - Digital subtraction angiogram

The advantage of a CT scan is that it may demonstrate other pathology to account for the patient's symptoms.

Management

Management can be either an endovascular or open technique.

Endovascular

- Balloon angioplasty and stenting.
- A strategy attractive in these patients with poor nutrition and multiple co-morbidities.

Open surgery

- Re-implantation technique
- Endarterectomy
- Bypass from the infrarenal or suprarenal aorta or iliac artery.
- Long term graft patency is excellent and exceed 90%

Most vascular surgeons would offer an endovascular first approach where applicable. Patient with asymptomatic disease should be managed with best medical therapy.

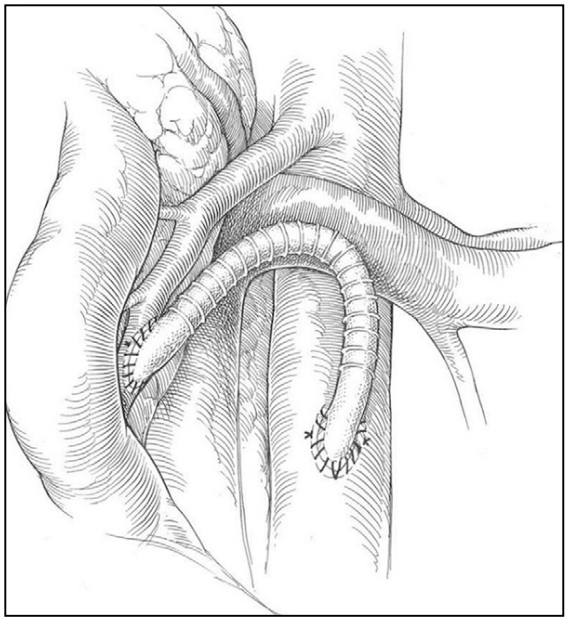

Fig. 3: SMA bypass with prosthetic graft