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Review Article

Mesenteric Ischemia

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Abstract

Mesenteric ischemia refers to ischemia of small intestine, whereas colonic ischemia refers to ischemia of large intestine. Bowel ischemia can be acute or chronic, depending upon how quickly the occlusion of vessels of intestine occurs. In acute ischemia, there is sudden occlusion of a blood vessel supplying the intestine. It is mostly embolic or thrombotic in nature and requires immediate surgery. In contrast, chronic mesenteric ischemia occurs when there is slow narrowing of blood vessels supplying the intestine, giving sufficient time for collateral circulation to develop, preventing intestinal necrosis.

Key Words: COVID-19; fiscal policy; economic growth; financial adviser, personal finance

Mesenteric ischemia

Mesenteric ischemia refers to ischemia of small intestine, whereas colonic ischemia refers to ischemia of large intestine. Bowel ischemia can be acute or chronic, depending upon how quickly the occlusion of vessels of intestine occurs. In acute ischemia, there is sudden occlusion of a blood vessel supplying the intestine. It is mostly embolic or thrombotic in nature and requires immediate surgery. In contrast, chronic mesenteric ischemia occurs when there is slow narrowing of blood vessels supplying the intestine, giving sufficient time for collateral circulation to develop, preventing intestinal necrosis.

Mesenteric blood supply:

There are 3 vessels which supply the intestine:

1. Coeliac artery
2. Superior mesenteric artery
3. Inferior mesenteric artery

There is rich collateral circulation between these vessels **(Figure 2)** so that chronic stenosis of one vessel is tolerated well. Ischemia occurs when at least two vessels are showing critical stenosis.

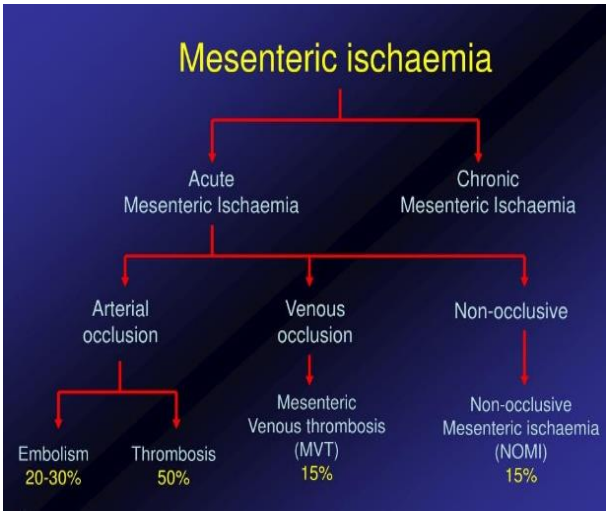

Figure 1: Chart showing the etiology of mesenteric ischemia

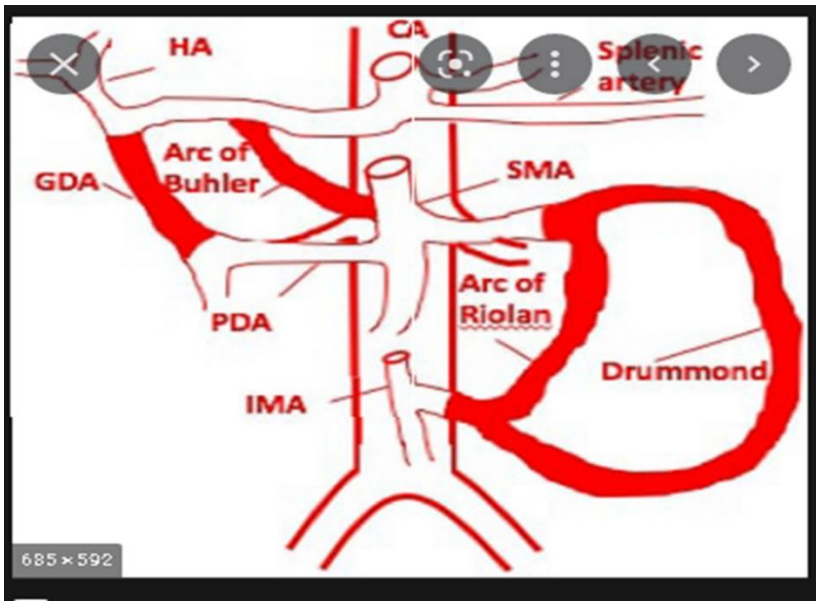

Figure 2: Collateral circulation between the three mesenteric vessels

ChronicMesenteric ischemia:

Chronic mesenteric ischemia (CMI) mostly caused by atherosclerotic narrowing of the two or more vessels supplying the gut[1,2]

Causes of CMI include:

- Atherosclerosis involving the proximal portions of the celiac, superior mesenteric, or inferior mesenteric artery.
- Dissection
- Vasculitis, especially Takayasu disease
- Fibromuscular dysplasia
- Radiation
- Cocaine abuse

One rare cause is celiac artery compression syndrome, also known as median arcuate ligament syndrome, in which intestinal ischemia is caused by compression of celiac trunk by median arcuate ligament[3].

CMI is a rare diagnosis. Moawad and Gewertz could find only 330 cases in search of 20 years literature[1]. Because many cases remain undiagnosed, the true prevalence may not really be low.

Since most of the cases are due to atherosclerosis, risk factors include diabetes, smoking, hypertension, older age, dyslipidemia and coronary artery disease.

Symptoms of CMI

Classical triad of symptoms are 1. Post prandial abdominal pain 2. Fear to eat due to anticipation of severe pain(Sitophobia) 3. Weight loss

Abdominal pain typical occurs within 10-60 minutes of food intake and may be so severe that patient fears to eat. Other symptoms include nausea, vomiting and diarrhoea.

Most of the times, diagnosis is delayed, as the disease is not suspected. Patient might have undergone ultrasound, upper and lower gastro intestinal (GI) endoscopy and many times CT coronary angio or conventional coronary angiography with possibility of post myocardial infarction (MI) angina.

Work up:

- History: High index of suspicion should be kept
- Imaging:

Duplex Ultrasound : Fasting duplex criteria for mesenteric stenosis(>70%)[4]

SMA: Peak systolic velocity of 275 cm/s or greater

Celiac artery : Peak systolic velocity of 200 cm/s or greater CMI.

CT angiography: Has sensitivity of 96% and specificity of 94% for detecting CMI[5]. It is mainly important to detect vascular disease in celiac trunk and SMA[6]. Schaefer et al found it to be the best modality in comparison to MR angiography and duplex ultrasound[7].

Magnetic Resonance Imaging/MRA: Advantage - ability to image without radiation. It has been found to accurately imagine mesenteric vessels[8,9].

Disadvantage: potential inability to accurately evaluate the IMA.

It is not considered initial investigation of choice in emergency settings[10,11].

Catheter angiography: Gold standard for diagnosing mesenteric vascular disease. Angiography can be done to confirm the diagnosis before surgery or endovascular therapy is planned.

Management: Once symptoms of mesenteric ischemia are there, revascularization is required. Open surgical repair was standard care of treatment in CMI, but at present initial approach is endovascular repair**(Figures 3-5)** in around 80% of patients[12]. It is minimally invasive, has high initial success rate and has few complications[13]. However, plain balloon angioplasty has lower success rate and high rate of restenosis, stent is almost always implanted[13,14]. Restenosis can still occur in around 40% patients and 20% may require repeat intervention[15,16]. Also, the results of angioplasty may depend upon the vessel revascularized. In a study, primary patency was better in SMA group than in coeliac artery group17.

Open surgical repair has significant post operative complications and small increase in mortality at 30 days, compared to angioplasty. However it has better long term results and low risk of recurrence at 3 years[18].

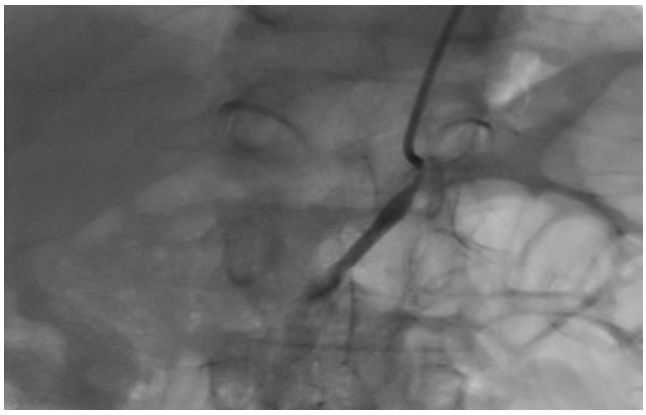

Figure 3: Tight stenosis of SMA, in a patient of CMI, missed for 8 years, in many hospitals. Upper and lower GI endoscopy, ultrasound of abdomen, coronary angiography were normal. Weight loss of 14 Kg in last two months.

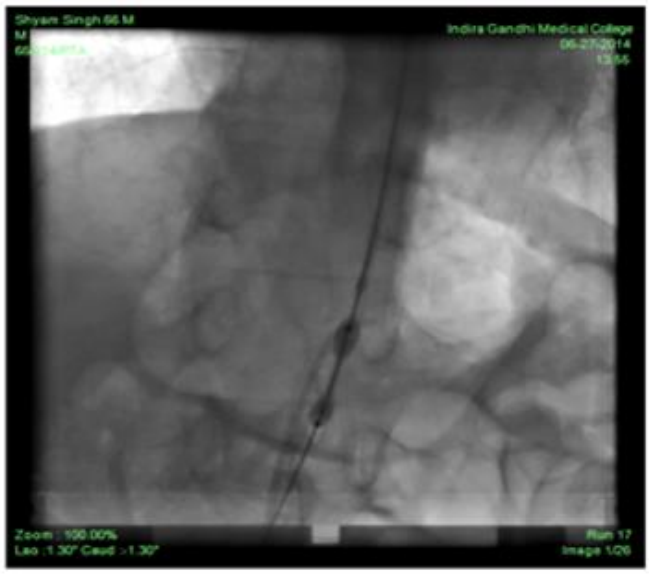

Figure 4: Implantation of stent in same patient of CMI.

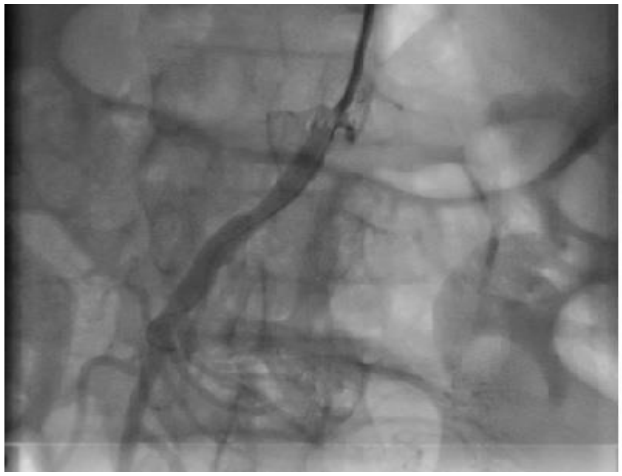

Figure 5: Wide open SMA after stenting. All symptoms disappeared. Weight gain of 4 kg in two weeks.

Acute mesenteric ischemia (AMI): It can be due to acute arterial occlusion (AAO) or mesenteric venous occlusion (MVO) or non occlusive mesenteric ischemia (NOMI). Acute mesenteric arterial ischemia (AMAI) is an surgical emergency. It is caused by embolic occlusion in 40-50% cases, thrombotic occlusion of diseased vessel in 20-35% cases and dissection of artery in around 5% cases[19]. Arterial embolism causes sudden severe pain in abdomen with associated nausea and vomiting. Thrombotic occlusion causes pain, which is more severe after meals. There may be fever, abdominal distension and local tenderness, once necrosis of bowel loops occur. The patient may present

with classical pain, out of proportion to examination, and epigastric bruit may be heard[20]. Embolic occlusion should be suspected in a patient with acute onset severe abdominal pain, who has atrial fibrillation or recent myocardial infarction. Echocardiography should be done to see for cardiac source of embolism. Fortunately, overall incidence is low, accounting for .09-.2% of patients admitted to the emergency department[21,22]. It is uncommon cause of acute abdomen but difficult to diagnose. CTA has a sensitivity of 71-96% and a specificity of 92-94% for AMI.

Management: heparin should be started in patients with AMI. Because of high risk of infection, antibiotics should be started. Oral intake is avoided as it can increase ischemia[23]. Thrombolytic therapy may be rewarding, if given within 8 hours of symptom onset, if signs of bowel necrosis and peritonitis are not there [24]. Revascularization is the cornerstone of treatment of AMI, otherwise the mortality is very high. In one study of 104 patients, mortality within 30 days was 64% n patients who were not revascularized, compared to 42% in vascularized patients[25]. Outcome in AMI is determined by intestinal viability. Non viable intestine may result in multi organ failure and ultimately may be fatal. So after initial stabilization laprotomy is performed, intestinal viability is assessed, non viable intestine is resected and revascularization is performed by means of embolectomy or arterial grafts. Nowadays endovascular procedures may be an alternative to surgery **(figure 6-9).** Though there are no head to head studies comparing surgery and endovascular approach in AMI, some studies show less need for surgery, less bowel necrosis and less mortality with endovascular approach[26]. However, open surgery helps in assessing the viability of the intestine and so taking appropriate decision, especially when endovascular approach is not available[27]. Patients with resection of large segment of small bowl can suffer from small bowel syndrome and intestinal failure, which is associated with poor quality of life[2]

Figure 6: Abdominal aortogram in a patient of AMI. SMA shows total cut off just after origin. Coeliac atery shows 95% stenosis at origun.

Figure 7: Stenting of SMA in same patient of AMI.

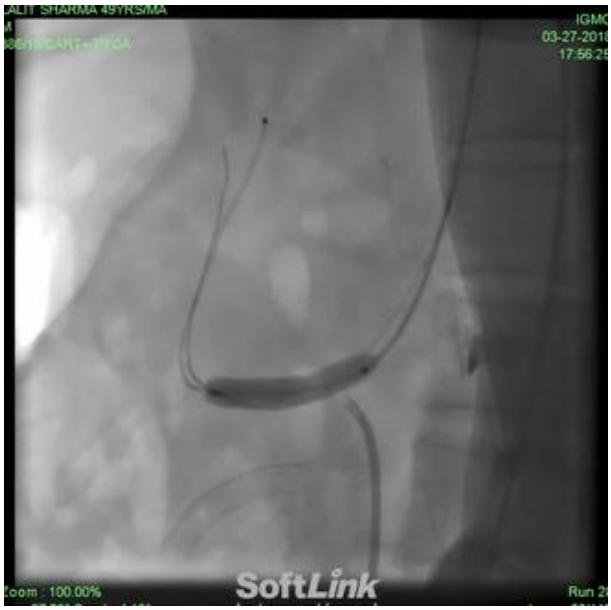

Figure 8: Stenting of celiac artery in the same patient of AMI.

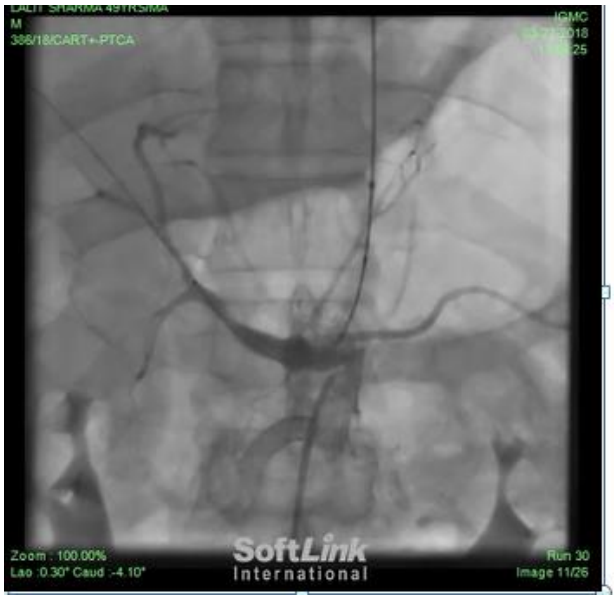

Figure 9: Good result in both celiac artery and SMA after stenting. Patent had developed necrosis of 6 feet small intestine, for which surgical resection and temporary iliostomy was required after the stenting.

Non occlusive mesenteric ischemia: It is caused by severe reduction in mesenteric perfusion due to hypovolemia (due to fluid or blood loss), heart failure and septic shock. There is secondary vasospasm. Pian in NOMI patients is more diffuse and episodic, with poor cardiac functions. Some authors have suggested hemodialysis is a risk factor for NOMI[29,30]. It can also occur in patients with septic shock, being treated with high dose vaso- active drugs. NOMI accounts for around 25% cases of AMI[31]. Treatment of NOMI is treatment of primary cause. Patients who develop intestinal necrosis will require laprotomy irrespective of cause.

Mesenteric vein thrombosis: It is due to hypercoagulable state. In primary MVT, there is no identifiable cause for coagulation. MVT can occur after ligation of splenic vein, portal vein or superior mesenteric vein after surgery for penetrating abdominal injury. It can also be secondary to pancreatitis, sickle cell disease or malignancy. In MVT, there is efflux of fluid into the bowel wall and lumen resulting in hypovolema and hemoconcentration. There is decreasd outflow of blood due to venous thrombosis, which impede the arterial blood flow and so ischemia of intestine. MVT affects younger population. Duration of symptoms may be longer than typical cases of AMI. CT and MRA are diagnostic. Treatment is mostly anticoagulation. Systemic thrombolysis is rarely indicated.30 day mortality is 13-15% with anticoagulation and 25% without. Surgery may be required for intestinal necrosis. Early use of heparin improves survival[32].

Conclusion

Mesenteric ischemia van be chronic or acute. CMI is difficult to diagnose as patient has varied presentation. High index of suspicion is must. Patient with post prandial abdominal pain with all routine investigations inconclusive, and significant weight loss should be investgated for CMI. AMI is an emergency, and could be due to acute mesenteric artery

occlusion, NOMI or due to MVT. Here also high risk of suspicion is required. In suspected cases, early CT angio or MRA should be done. Delay in diagnosis is important cause of mortality.

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