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# VASCULAR DISEASES

# Mesenteric Ischemia

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**Keywords:** Disease; Blood vessels; Emergency; Bowel infarction.

## Abstract

Mesenteric ischemia is a disease of blood vessels supplying intestine. It can be classified into two types as either acute or chronic. The acute variety is life threatening emergency due to acute reduction in blood flow to intestine and may result ultimately into bowel infarction. It is mainly due to arterial embolism, arterial thrombosis, Non-Occlusive Mesenteric Ischemia (NOMI) & mesenteric venous thrombosis. One should have a high index of suspicion and diagnose it early so that fast intervention can be planned for restoring bowel blood flow before fatal infraction and gangrene occurs. Chronic mesenteric ischemia is usually due to stenosis or occlusion of proximal arterial blood supply of intestine. It is usually due to atherosclerosis. The classical symptoms are intestinal angina, defined as postprandial recurrent abdominal pain that resolves in 1 to 2 hours, associated with weight loss & aversion to food. One has to correlate clinical history, physical examination & laboratory data. Radiological imaging plays a significant role in diagnosis of acute or chronic mesenteric ischemia. If recognised early with pertinent imaging findings patients can be offered various treatment options to prevent serious and possible fatal complications that may occur due to this disease.

## Introduction

This disease entity is classified as either acute or chronic. Acute Mesenteric Ischemia (AMI) is a life threatening condition in which a sudden reduction in splanchnic blood flow jeopardizes bowel viability and may ultimately leads to bowel necrosis. Normally short episodic reduction in mesenteric blood flow is well tolerated as 25% of capillaries may be open with some reserve against changes in blood flow, but if the reduction is prolonged and there is discrepancy in demand and supply ischemia ensues [1]. Persistent ischemia may finally cause tissue damage from reperfusion injury which results in increased microvasular permeability [2].

A very high index of suspicion, early diagnosis & rapid intervention are necessary so that normal intestinal blood flow is restored before fatal bowel infarction occurs.

AMI may be due to [3,4]:

Arterial embolism
Arterial thrombosis
Non-occlusive mesenteric ischemia (NOMI)
Mesenteric venous thrombosis
Dissection
Trauma
Retroperitoneal fibrosis
Fibromuscular Dysplasia ( FMD)
Vasculitis

Chronic Mesenteric Ischemia (CMI) occurs when there is stenotic or occlusive disease involving proximal (usually ostial) part of arterial supply to bowel, usually as a result of atherosclerosis. Autopsy studies have shown a 6-10 % incidence of > 50 % stenosis in one or more mesenteric arteries, yet the incidence of symptomatic CMI is quite less [5,6].

Because of rich and redundant arterial supply of the bowel and the propensity to develop collaterals, CMI typically occurs when there is blockage of at least two of three major mesenteric blood vessels; the celiac axis, Superior Mesenteric Artery (SMA), and /or Inferior Mesenteric Artery (IMA) (**Figure 1**).

Intestinal angina is the classic presentation, defined as recurrent post-prandial abdominal pain that subsides in 1 to 2 hours, associated with weight loss and aversion to food [3]. Because intestinal angina is rare, diagnosis may be delayed and patients may be symptomatic for months or years prior to seeking medical attention.

Contrast enhanced computed tomographic and CT Angiography (CTA) is typically the initial modality of choice for diagnosing mesenteric ischemia.

CT angiography may demonstrate the exact vascular anatomy and it may also show stenosis and occlusions as well as any arterial collaterals (**Figure 2,3**). A non-contrast CT examination maybe useful for defining vascular calcification and

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should always be included. CT may also demonstrate non-vascular causes of abdominal pain such as inflammatory disease or neoplasm. Furthermore there maybe findings that will help differentiate between an acute and chronic process; bowel wall thickening, pneumatosis and porto- mesenteric gas indicate an acute process and are absent in CMI.

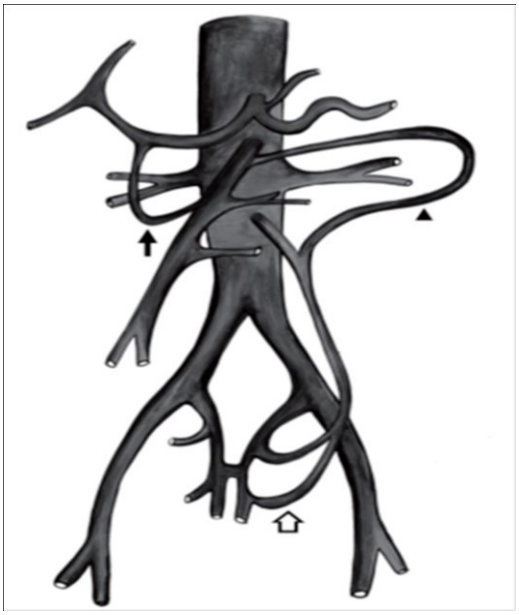

**Figure 1:** Diagram of the main collateral vessels in patients with occlusion of the celiac artery or Superior Mesenteric Artery (SMA). The pancreaticoduodenal arteries (arrow) connect the celiac artery and proximal SMA. The marginal artery of drummond and the Paracolic arcade (arrowhead) run between the SMA and the inferior Mesenteric artery (IMA). When the IMA is also occluded, the systemic Vessels (mainly the internal iliac artery) can feed the IMA (reverse flow) and the other vessels via previously described anastomoses (open arrow).

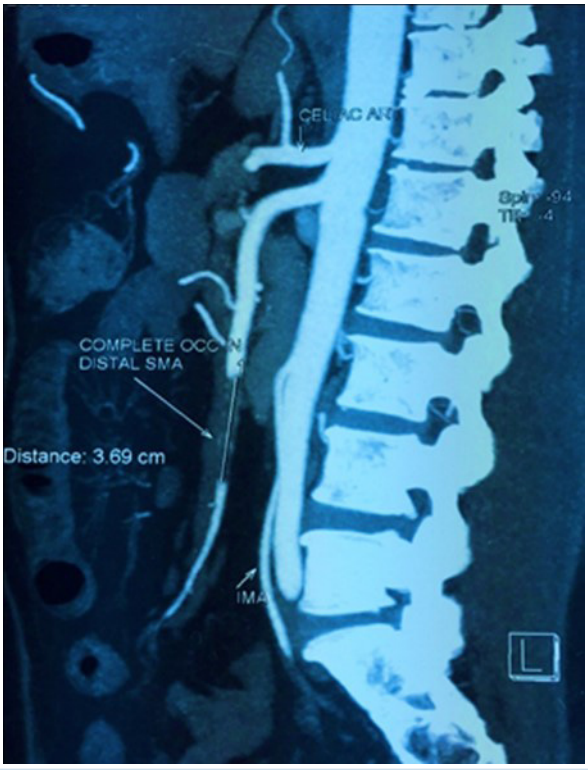


**Figure 2:** CT angiography showing embolic occlusion of Superior Mesenteric Artery (SMA).

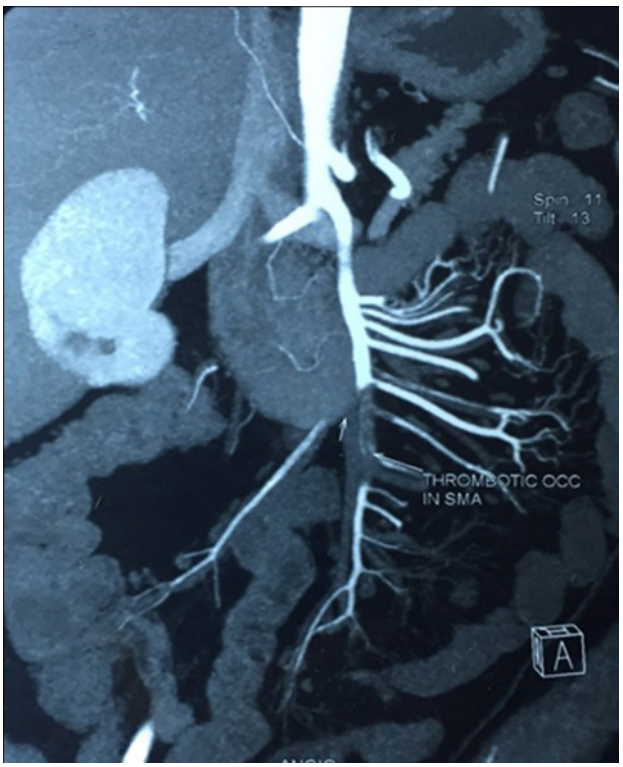


**Figure 3:** CT angio showing clot in superior mesenteric artery.

The accepted diagnostic gold standard for diagnosing either acute or chronic mesenteric ischemia is angiography (invasive). If the history suggests embolic or thrombotic occlusion with an acute presentation, the best management is prompt surgery.

As this is the best chance to save threatened, but uninfarcted bowel. Patients with a more delayed presentation may be considered for angiography, to be performed in emergency in a case of suspected AMI if there are no peritoneal signs. Angiography in such cases should be rapidly available as a delay in diagnosis could prove fatal.

Elective angiography is usually appropriate for suspected CMI; in addition to confirming diagnosis, catheter based interventions can be planned in either acute or chronic mesenteric ischemia.

For this lateral aortography and anteroposterior views along with selective angiography of celiac, SMA and IMA are typically done. Catheters used are pigtail, cobra, and hookand short and long curve sidewinder with a diameter of 4 to 5 French. One can adopt femoral or brachial route for doing angiography and further catheter based intervention.

### Acute mesenteric ischemia

There are multiple causes of AMI, but one of the most important causes is embolic arterial occlusion, implicated in up to 50% cases [7]. It typically involves SMA just distal to the middle colic artery origin (**Figure 4**).This causes vasoconstriction of the arterial branches originating beyond the embolic occlusion. The embolus is usually cardiac in origin and therefore risk factors such as myocardial infarction, arrhythmia, valvular disease and ventricular aneurysm should be considered and appropriately excluded [7].

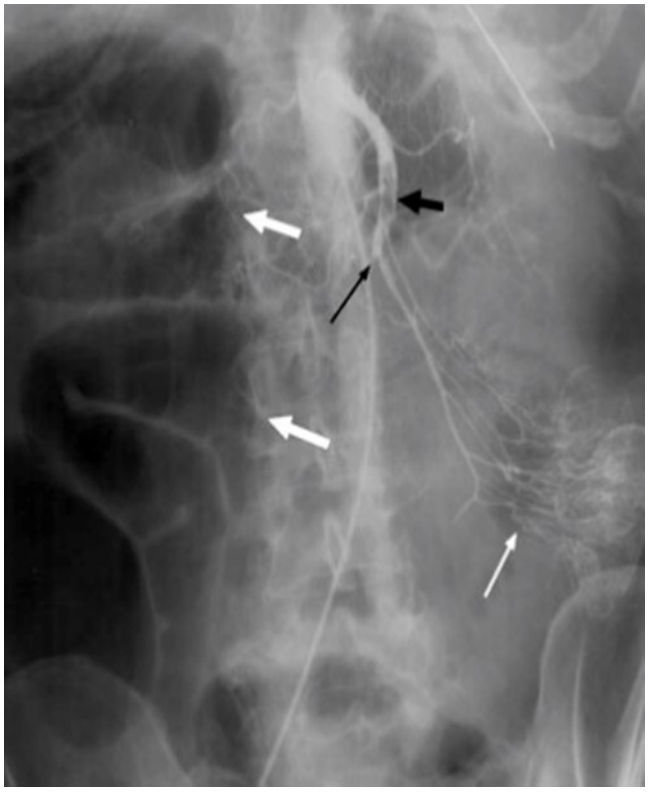

**Figure 4:** Superior mesenteric artery arteriogram shows a Large embolus that appears as a central filling defect (large Black arrow) and has occluded the distal portion of the arterial Trunk (small black arrow). A very small portion of the jejunum Remains perfused (small white arrow). Note the abnormally dilated bowel loops (large white arrows) in this patient with Acute mesenteric ischemia.

Mesenteric ischemia that results from arterial thrombosis usually occurs when a pre-existing atherosclerotic stenosis, typically at vessel origin, when reaches a critical diameter it acutely thromboses. Because the thrombotic occlusion occurs proximal to the middle colic artery, midgut arterial perfusion is completely interrupted, particularly if there are poorly formed collaterals (**Figure 5**). Thus there is usually a worse prognosis in acute ischemia due to SMA thrombosis than with embolus because emboli often lodge distally in the SMA, and allow continued partial perfusion of the proximal intestine (**Figure 6**).

Non-occlusive Mesenteric Ischemia (NOMI) results from severe and prolonged visceral arterial vasoconstriction, and may occur in setting of severe systemic illness with circulatory insufficiency and end stage shock. Thus there is no obstructive cause for the diminished perfusion, but rather there is reduced perfusion pressure. This hypotensive or hypovolemic condition may persist for hours and the intestinal mucosal permeability increases in direct proportion to the duration and severity of the vasoconstriction. This causes intestinal necrosis. This entity also occurs in response to drug intoxication e.g., cocaine, dopamine and ergot derivatives, as well as following coarctation repair. The incidence may be decreasing with the improved treatment of critically ill patients.

Intestinal ischemia that results from venous thrombosis is uncommon. One study reported that 0.38 % of laparotomies performed for “acute abdomen” revealed intestinal ischemia and only 17 % of those were from venous thrombosis [8]. Primary thrombosis have no known underlying cause but secondary thrombosis are associated with other disorders such as hypercoagulable states, trauma, abdominal surgery, neoplasm , inflammatory bowel disease, sepsis, peritonitis and pancreatitis. The process usually starts from venous arcades and extends centrally to involve the Superior Mesenteric Vein (SMV) and the portal vein, with the Inferior Mesenteric Vein (IMV) less often involved.

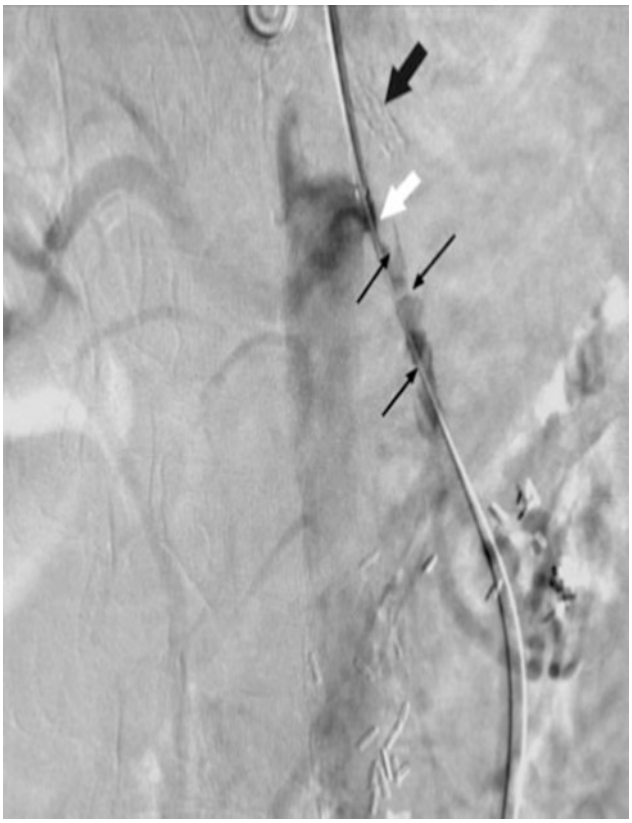

**Figure 5:** Digital Subtraction Angiography (DSA) image shows thrombosis (small black arrows) of the superior mesenteric Artery that occurred distal to a high-grade stenosis (large white arrow). There is very little perfusion distal to the Thrombosis. Note the previously placed stent in the celiac Trunk (large black arrow), which is also occluded.

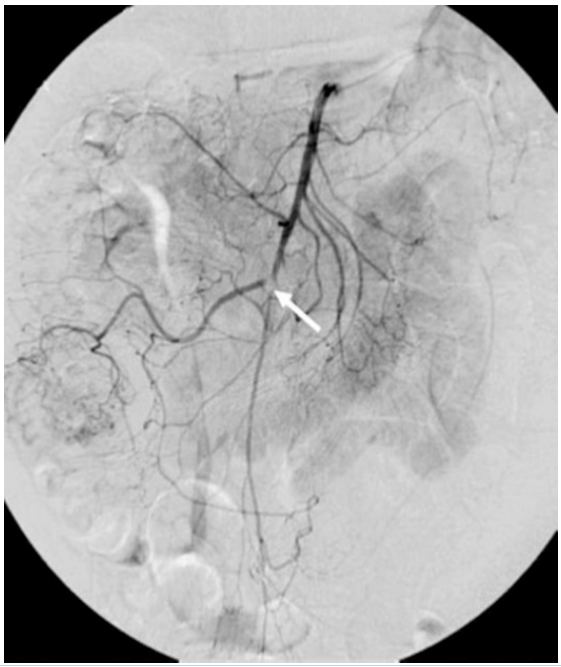

**Figure 6:** A small embolus (white arrow) has lodged distally in the superior mesenteric artery and this location allows for continued perfusion of most of the bowel in this arterial territory. This contrasts with the severe bowel ischemia that occurs with proximal embolic or thrombotic occlusion.

**Clinical features**

AMI occurs more commonly in elderly population. Those who have co morbid conditions are more prone to this disease. The onset is acute and consists of severe abdominal pain that is disproportionate to physical examination. Pain may be associated with nausea, vomiting, gastrointestinal haemorrhage, leucocytosis and acidosis (altered pH and elevated lactate levels).

**Imaging of AMI**

Plain radiographs are normal in up to 25 % cases. And when abnormal the findings are nonspecific that may be bowel dilation, pneumatosis, portal venousgas, pneumoperitoneum.

CT may show bowel dilation, wall thickening, ascites, and mesenteric oedema in early stages of AMI. Later stages may show pneumatosis, portal venousgas, and pneumoperitoneum. Other abdominal organs like kidneys may show embolism (**Figure 7**) [9].

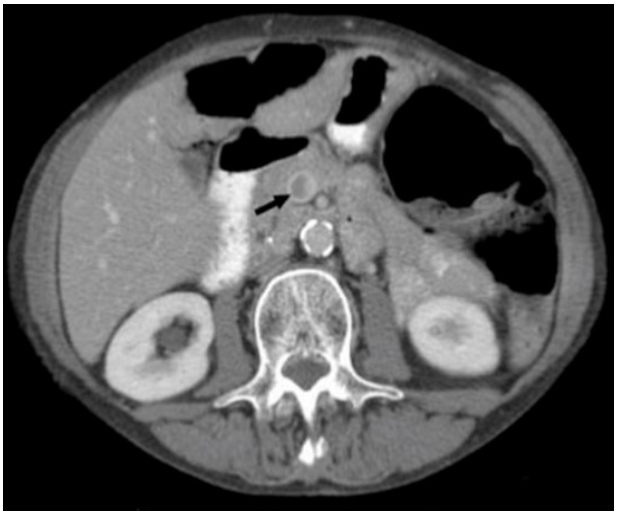

**Figure 7:** Axial image from the venous phase of a contrast enhanced computed tomography (ct) scan shows an eccentric central filling defect (black arrow) in the superior mesenteric vein representing mesenteric vein thrombosis.

CTA is more specific and will show the site of occlusion as a central filling defect in SMA or may show an abrupt point where artery is not opacified, with or without poor collaterals. While in SMA thrombosis one may see vascular calcium at origin of celiac, SMA or IMA along with well-formed collaterals. AMI from venous thrombosis may have vein showing central attenuation defects. CTA is nonspecific in NOMI although arterial calibres may be diminutive.

Gadolinium enhanced Magnetic Resonance Angiography (MRA) can also be used to define AMI.

Invasive angiography is the gold standard for detection of SMA, celiac and IMA occlusion or stenosis [10]. An acute embolus looks like a filling defect which completely or partially obstructs the artery with absent collaterals. The so called tram – track sign occurs when the embolus is partially outlined by contrast those courses between the embolus and the arterial wall (**Figure 8**). Selective mesenteric angio can be performed if the origin of vessel is patent. This is usually done by a femoral or radial access (4- 5 French). Aortography performed in anteroposterior and lateral view defines these vessels (**Figure 9**). Thereafter selective angiography may be done with cobra, hook or sidewinder catheter. The invasive and time consuming nature of angiography coupled with risk of nephrotoxicity has made CTA a better method to screen and diagnose this condition. In NOMI the angiography shows the string of sausage sign (**Figure 10**) in major arterial branches, reflux of contrast into aorta and normal venous phase. (Diffuse constriction of vessels and alternate areas of segmental constriction looks like string of sausage) [7].

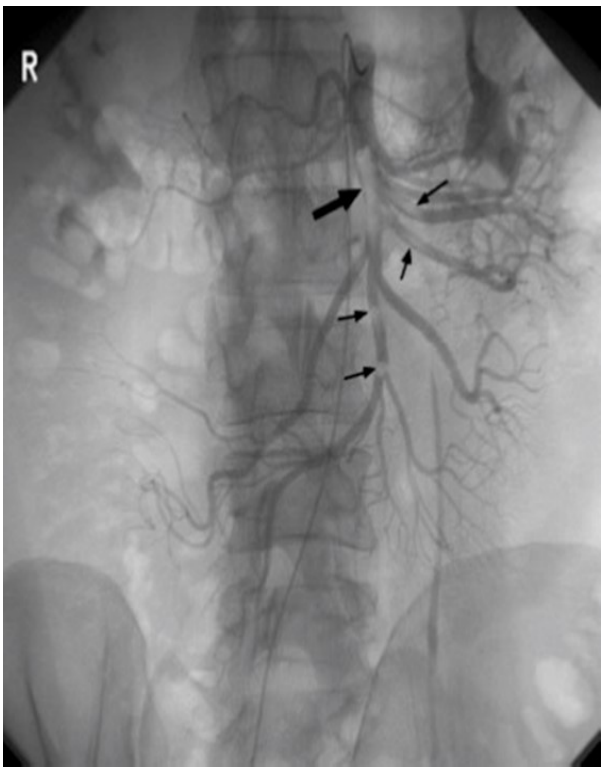


**Figure 8:** An acute embolus within the superior mesenteric artery appears as a filling defect (large black arrow) partially obstructing the artery. Smaller filling defects distally (small black arrows) represent fragmentation of the embolus. Note the so-called tram-track appearance in which the filling defects are partially outlined by contrast those courses between the emboli and the arterial wall.

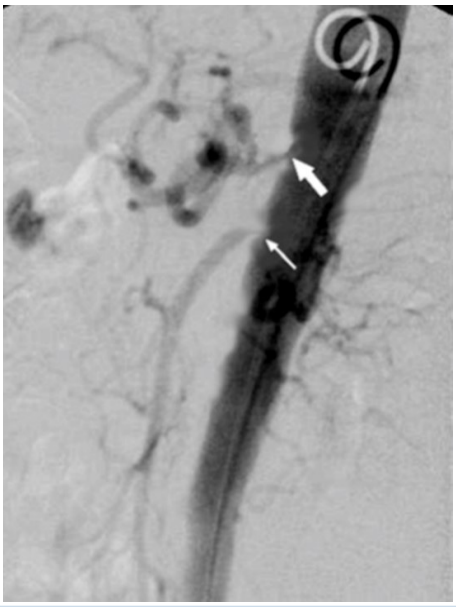

**Figure 9:** Lateral aortogram shows high-grade stenoses of the celiac (large white arrow) and superior mesenteric artery (small white arrow) origins, a typical location for an atherosclerotic etiology for chronic mesenteric ischemia. Chronic mesenteric ischemia usually occurs only when there is disease of at least two of the three major mesenteric arteries.

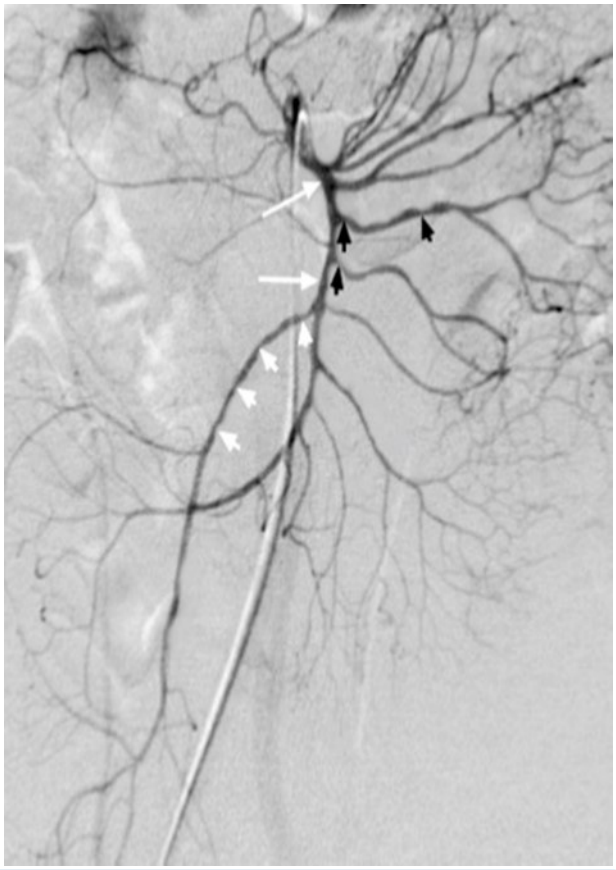

**Figure 10:** Selective superior mesenteric artery arteriogram in a patient with nonocclusive mesenteric ischemia (NOMI) secondary to vasopressor therapy shows a diminished caliber to the main arterial trunk (white arrows) and vasoconstriction of the branch vessels (black arrowheads). Alternating areas of vasoconstriction (white arrowheads) have produced a ''string of sausages'' sign.

**Treatment options**

Once the diagnosis of AMI is made, prompt treatment should be started as delay may cause fatal and irreversible bowel damage. Initial therapy should include fluid resuscitation, nasogastric tube decompression, and if possible anticoagulation and avoidance of vasoconstrictive agents [11]. Broad spectrum antibiotics should be given early. Anticoagulation is the primary therapy in mesenteric venous thrombosis, potentially with mandatory long term anticoagulation if a primary clotting disorder is discovered.

For any case of AMI, laparotomy is indicated if there are peritoneal signs. In most cases surgical embolectomy or bypass, with resection of nonviable bowel is usually necessary [12].

Catheter based therapies have been used in AMI with various degrees of success. Outcomes depend on severity of ischemia, as percutaneous therapies such as Catheter Directed Thrombolysis (CDT), or vasodilator infusion may require longer treatment times than surgery [13]. Direct intravascular vasodilator therapy should be considered in cases of NOMI; 60 mg bolus of papaverine is given selectively in SMA, followed by infusion via catheter at rates of 30 to 60 mg per hour. Recently successful treatment of NOMI was done with intravenous high dose PGE-1 infusion (0.01-0.03 microgram per kg per min) [14].

## Chronic mesenteric ischemia (CMI)

It is less common than AMI. There is failure to achieve normal postprandial hyperaemic arterial intestinal flow as a result of stenotic or occlusive disease of the mesenteric arteries. So there is an imbalance in demand and supply of oxygenated blood in gut, anabolic by-products are produced and post prandial pain analogous to angina pectoris occurs.

**Clinical aspects**

Typically history is abdominal pain after 30-45 minutes after eating, sometimes with nausea, vomiting, diarrhoea or malabsorption. With progression there is weight loss (cachexia), and fear of eating (sitophobia).Untreated cases of CMI may progress to AMI usually due to thrombosis of a high grade orificial stenosis.

**Causes are:**

Atherosclerosis
Extrinsic vascular compression ( median arcuate ligament syndrome) (**Figure 11**)
Tumour
Vasculitis
Dissection
Radiation
Fibromuscular dysplasia

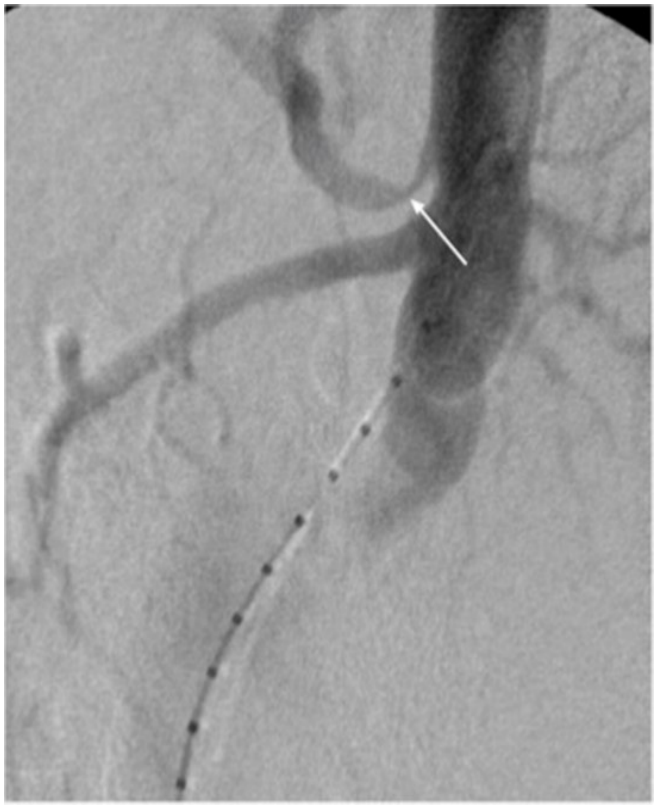

**Figure 11:** Chronic mesenteric ischemia may rarely be caused by extrinsic compression of the celiac artery by the median arcuate ligament of the diaphragm. This results in proximal arterial narrowing (white arrow) _5 to 10 mm from the origin, with a characteristic upward hooking of the celiac artery. This controversial condition may be associated with upper abdominal pain, weight loss, hyperemesis, and an epigastric bruit caused by inadequate blood flow through the celiac artery, with resultant ischemia.

**Imaging of CMI**

It can be done with initial duplex ultrasound examination which will show increased velocity (PSV) in the respective artery. Non-invasive modality such as CT Angiography (CTA) or MR angiography (MRA) very well defines stenotic or occlusive disease and presence of collateral circulation. CT may also show vascular calcification (**Figure 2,3**).

Duplex ultrasonography may show elevated fasting celiac and or SMA (PSV) peak systolic velocities > 275 cm/ second correlating to a stenosis of at least 70 %. (**Figure 12**).

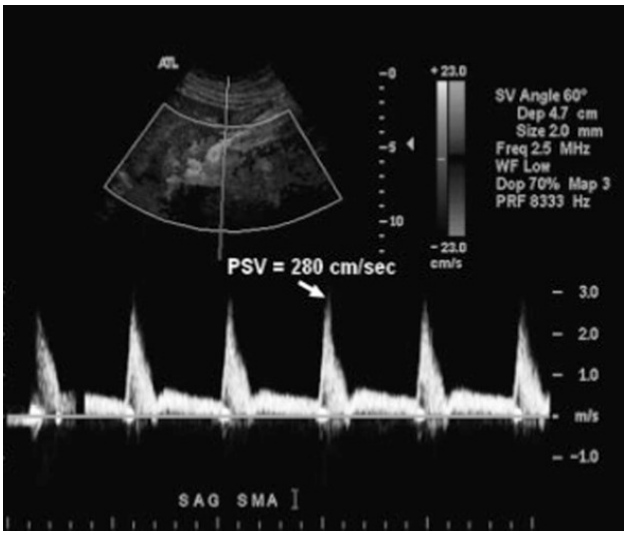


**Figure 12:** Duplex ultrasound of the superior mesenteric artery demonstrates a Peak Systolic Velocity (PSV) >275 cm/ second correlating with a stenosis of at least 70%

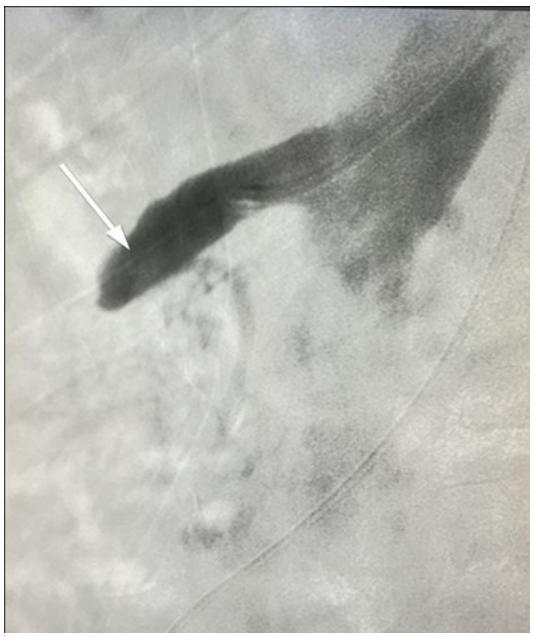

**Figure 13:** Angiography showing occluded SMA.

Angiography is often used in cases of CMI either for the confirmation of diagnosis and surgical planning or for endovascular treatment. The imaging sequence is anteroposterior (AP) and lateral aortography followed by selective mesenteric angiography.

**Treatment options**

Surgical and endovascular revascularisation are indicated both for relief of symptoms and to prevent progression to life threatening AMI. Surgical treatment options include endarterectomy, vascular bypass, and arterial reimplantation. In most cases revascularisation of only one vessel (usually SMA) is needed despite multivessel disease [15].

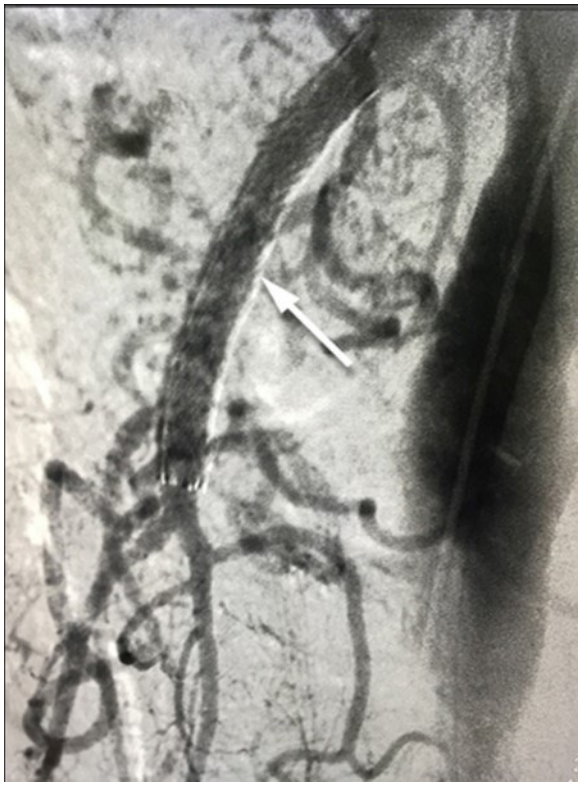

**Figure 14:** SMA after stenting ( brisk flow in branches).

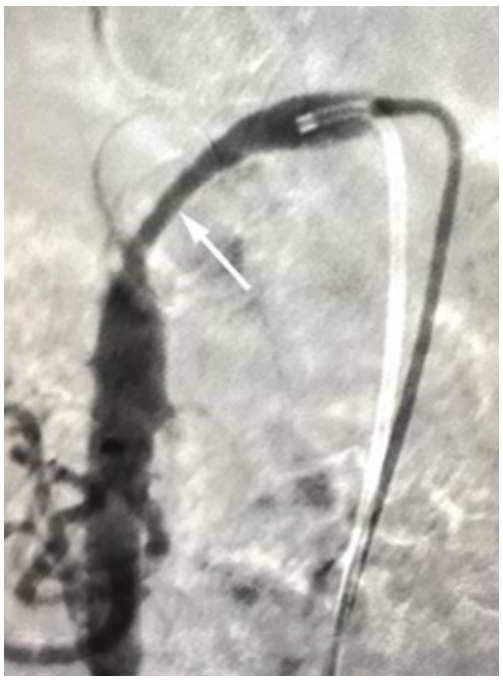

**Figure 15:** Critical SMA stenosis.

Endovascular treatment usually consists of angioplasty and stenting of stenotic of occlusive vessels with high success rates of more than 90 % [16-19]. It is done by either femoral or brachial access. A long 6 Frenchsheath is placed near the Ostia of vessel through which a small catheter and 0.014'' or 0.018'' wire is used. First predilating the lesion followed by stenting or direct stenting is usually performed. Stents used are either premounted balloon expandable or self-expanding stents (**Figure 13,14,15,16**). Recurrent stenosis occurs frequently, however requiring reinterventions. The reported cumulative primary and assisted patency rates after these treatments are lower than surgery. The long-term patency rates are (70-93%) but reported

associated morbidities of up to 29 % and mortalities of 7 %.The current recommendation is one of individualised application of surgery or endovascular treatment in accordance with individual patient's anatomic and comorbid considerations [15,16].

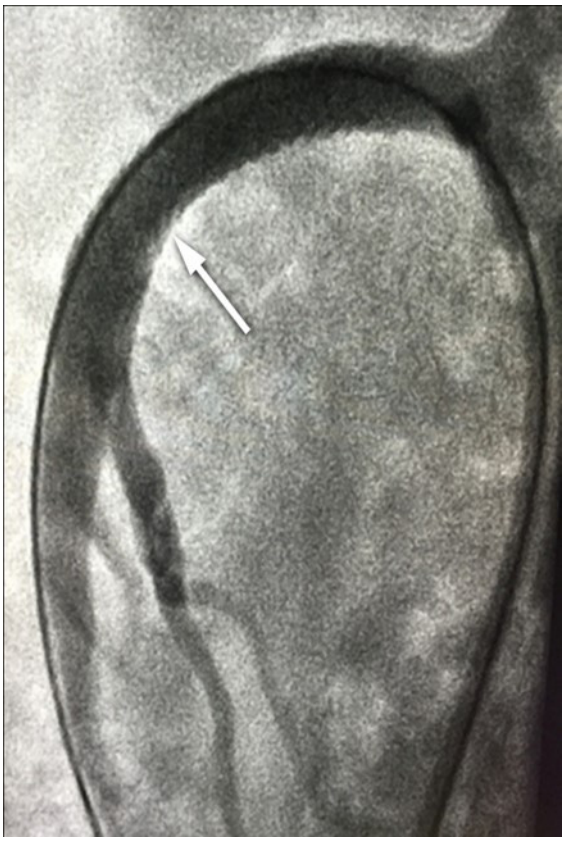

**Figure 16:** SMA after stenting.

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