

Acute Mesenteric Ischemia at a Glance

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Abstract

Acute mesenteric ischemia (AMI) is also outlined as an unforeseen interruption of the blood provide to a section of the little bowel, resulting in ischemia, cellular harm, enteral death, and eventually patient death if untreated(1). Acute mesenteric ischemia (AMI) means the unforeseen decrease of enteral intromission, which may result in obstructive or non obstructive reduction of blood vessels or blood flow. Acute mesenteric ischemia results from the reduction in enteral blood flow that regularly leads to intestine death and is related to a high mortality. Acute mesenteric ischemia could also be occlusive or non-occlusive (NOMI), further defined with the main pathological component as mesenteric venous thrombosis (5–15%), mesenteric arterial embolism (50%) or mesenteric arterial occlusion (15–25%)(2).

Mesenteric ischemia includes inflammatory injury, insufficient blood supply and consequently necrosis of the small intestinal walls. Mesenteric ischemia can be divided into chronic (CMI) and acute mesenteric ischemia (AMI), with the further division into four categories (3). Therefore, acute mesenteric ischemia (AMI) will be caused as a result of non-occlusive causes (NOMI), blood vessel occlusion, blood vessel embolism and mesenteric venous thrombosis (MVT), like hypo-perfusion due to mesenteric arterial vasoconstriction or to low rate of cardiac output(4). Due to transmural injury, reversible ischemia, perforation, and subsequent necrosis Bowel damage may vary from minimum lesions and it has direct relation to the mesenteric blood flow reduction (5). Mesenteric ischemia (MI) with elevated mortality rates i.e 24-94% and it accounts for 0.1% of all hospital admissions is an uncommon medical condition(6). In more

than 95% of cases, chronic mesenteric ischemia (CMI) is associated with generalized atherosclerosis disease, with all major mesenteric arteries presenting occlusion or stenosis. Mesenteric ischemia (MI) could be a ruinous event with mortality rates up to 67–90%(7). Interestingly, almost 20% of total diagnosed acute mesenteric ischemia cases were patients having ages lesser than forty years. The estimated incidence of acute mesenteric ischemia has been calculated to almost 4.3–10/10,000 (8). It is reported that probably 20% patients will have evidence of emboli to other vasculature systems of body, 75% of patients will have atrial fibrillation and 30% patients were have a history of embolic events (9).

The records of 128 patients diagnosed with acute mesenteric ischemia at Kemal Atatürk University, Faculty of Drugs, Department of General Surgery between Jan 2000 and July 2007 has been reviewed in terms of mortality and morbidity along with other patient factors. 26 of these cases studied were having age lesser than 40 years (10). The records consisted of clinical findings like modes of presentation, clinical features and other laboratory markers. It has been concluded that mesenteric ischemia in patients below forty years older is present but comparatively very rare. If unnoted, mesenteric ischemia proves to be fatal even in younger age groups. Therefore, throughout the study of young patients with non specific abdominal pain, it is essential to think about and keep in consideration with the presence of predisposing factors, young patients could suffer from acute mesenteric ischemia (11).

Literature shows documentation of few cases of mesenteric ischemia at younger ages with evidence of trend showing hard drug abuse. Hard drug abuse was rumored to be a number one reason behind acute mesenteric ischemia in the young population(12, 13). In a study, it has been reported that 3 cases were reportable with a number one cause. In this study, the predisposing

factors were anti-thrombin III deficiencies and fibrin, protein C and Takayasu arteritis, that cause a hypercoagulable state. Protein C deficiency is reportable to be the explanation for the cause of mesenteric venous occlusion (14, 15). Additionally, to an underlying vasculitis or hypercoagulable state, significant smoking might dispose of the young patient to mesenteric ischemia and mesenteric arterial occlusive disease(16).

With overall mortality of 60-80%, acute mesenteric ischemia (AMI) could be a probably fatal vascular emergency, and it is reported that the incidence has been rising day by day (17, 18).Of these patients who survive, 20-60% develop short gut syndrome. 35-30% were account for acute mesenteric occlusion for all ischemic events and nearly always happens within the setting of severe atherosclerosis disease (19) Around 50-75% of the cases who present in this manner have had previous symptoms which are in concordance with chronic mesenteric ischemia.

Mesenteric Arterial Anatomy

The SMA and the arterial blood vessel origins from the arteria are in shut proximity to the arterial blood vessel origins within the higher abdomen. The celiac axis lies slightly below the arterial blood vessel hiatus of the diaphragm. The proximal parts of the arterial blood vessel and SMA are every engulfed in a neurological tissue and bodily structure of liquid body substance. These vessels are comparatively skinny walled compared with extremity arteries. The major peritoneum veins and the vena portae also are skinny walled. The inferior mesenteric artery (IMA) arises from the left anterior lateral side of the mid-infrarenal arteria. The blood offer of the gut is split into four major areas. Abnormal patterns of peritoneum blood vessel anatomy occur in approximately 15% of people and, though a replaced right artery from the SMA is one in all the foremost ordinarily reportable, there are a range of anomalies which are reportable(20).

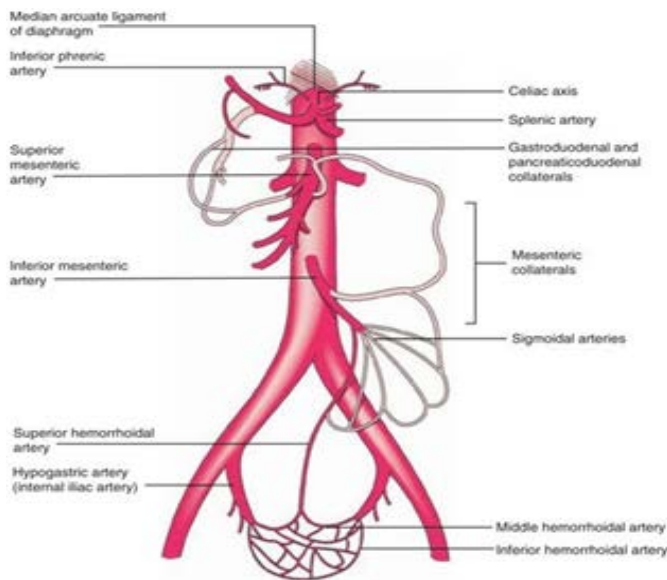


Figure 1: Mesenteric Arterial Anatomy Demonstrating Collateral Flow Between Major Branches of the Aorta

Every mesenteric vessel makes an anastomosis with adjacent sections of intestinal walls via communicating collateral vasculature. These collaterals have variable ability of blood flow and supply inadequate blood supply to adjacent areas in acute occlusion. The gastroduodenal artery which is a branch of the common hepatic artery, communicates through an anastomosis with branches of the inferior mesenteric artery. The dorsal arteries, a branch of the arterial blood vessel, communicates with the help of anastomosis among anterior and posterior pancreaticoduodenal arcades via a right crosswise branch of the dorsal arteries (Kirk's arcade). The arc of Buhler, which is a gift present in 1-4% of people, parallels the arteries proximally connecting the celiac axis to the SMA. There are also be arcs of Barkow, collateral pathway inside the peritoneum between the epiploic arteries of the SMA and splenic artery(21).

Pathophysiology of Acute Mesenteric Ischemia

The major mechanisms leading to mesenteric ischemia is bated blood supply to the tiny internal organ. The reduction in blood supply may be due to:

- Occlusion
- Systemic hypoperfusion

- Spasm within the mesentery

When blood flow through the mesentery gets interrupted, collateral channels are formed which try to compensate for the loss of blood flow. However, in the case of a prolonged decrease in blood flow, constriction of these vessels happens resulting in bated blood flow within the collaterals, that were at foremost defense against protection against ischemia(22). It is worth mentioning that mesenteric vessel constriction stays until the blood flow is renovated, which explains the utilization of dilator in the treatment of acute mesenteric ischemia (23). Ischemia associated injury to the mesentery is either caused due to reperfusion injury once the ischemia happens for a short amount of time or hypoxic injury once the ischemia happens for a longer time period (24).

Mesenteric Arterial Occlusion

Reduced blood flow through the blood vessel happens when the vessel, in the main trunk of SMA, gets blocked by an embolism that has originated from the left side of heart or from a clot that has formed over a thrombus, trauma or infection (25). Strangulation of the small bowels that compromise their blood supply momentarily and repeatedly due to small clots or emboli can lead to bowel ischemia in future.

Mesenteric Venous Occlusion

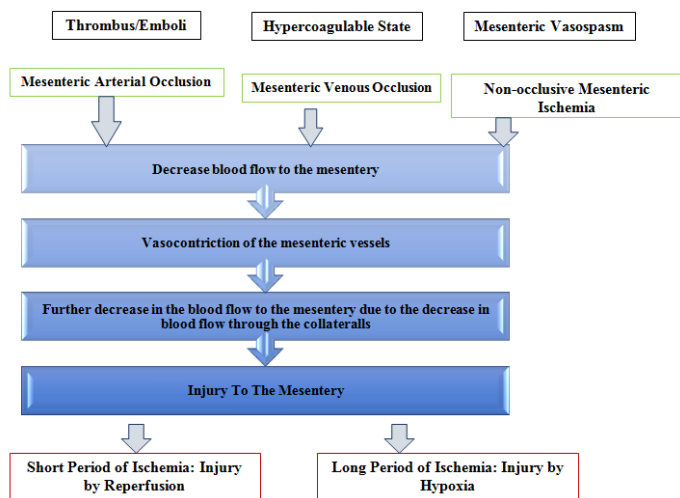
Decreased blood flow to the mesentery in venous occlusion happens with a sequence of events totally different from that of arterial occlusion. In hypercoagulable states, whether or not heritable or nonheritable, a clot of blood is created within the venous mesenteric vasculature. Once the blood clot is created, the vascular resistance to the venous blood flow of the mesentery will increase and it cause severe fluid movement across the vasculature. This results in systemic hypotension and edema that results in generalized hypo

perfusion and loss of oxygen supply to various vital organs including the intestine (26).

Non-Occlusive Mesenteric Ischemia

Non-occlusive mesenteric ischemia takes place when the mesenteric blood flow decreases secondary to generalized hypoperfusion caused by secondary peritoneum vasospasm. Non-occlusive acute mesenteric ischemia happens mainly in elderly who have multiple CVDs risks and CVD conditions in the presence of hypoperfusion secondary to some specific medications, sepsis, MI, congestive cardiac failure exacerbation, single or multi organ failure. Once this decrease in circulation takes place, the mesenteric blood flow decreases to such an extent that rest of the vasculature is unable to compensate for the loss of supply (27). One of the reasons behind non-occlusive acute mesenteric ischemia is cocaine (28).

Below is shown the flow chart depicting the complete pathophysiology of acute mesenteric ischemia.



Diagnostic Modalities

Due to the various modes of manifestation of this condition, it is usually misdiagnosed that leads to serious morbidity and high mortality. Laboratory studies add to little in making a definite diagnosis. Initially, complete cell count could also be normal however, leukocytosis or leftward shift could be seen later with progression of the

disease. The rise in enzyme amylase and lactose dehydrogenase is also present. Metabolic acidosis however, nonspecific disorder, is present as there is generalized shortage of supply and anaerobic respiration at a larger scale taking place in the body(29, 30). Additionally, PT and aPTT ought to be checked and particularly once MVT takes place, patients should be checked for protein S and C and antithrombin III deficiencies, abnormalities in lupus anticoagulant, medication, anticardiolipin protein and blood platelet aggregation (31).

Treatment of Mesenteric Ischemia

The treatment of the condition is based on the etiology and the cause so either medical or surgical. However, if viscus has completely lost its blood supply, the sole treatment is surgical resection of the necrotic segments of viscus. In non-occlusive mesenteric ischemia, wherever there's no complete blockage of the arteries providing the viscus, the treatment is medical therapy. Individuals are subjected to emergency admission to the hospital for revival with intravenous fluids resuscitation, optimization of their vessel perform and careful observance of laboratory tests. Nasogastric tube decompression and anticoagulant heparin medication medical aid may additionally be used to optimize introduction, severally and to limit stress on the viscus.

Surgical revascularization remains the treatment of selection for mesenteric ischemia associated with associate degree occlusion of the vessels supplying the bowel, however, vascular interventional radiological techniques and thrombolytic medical treatment have an evolving role(32). If the ischemia has progressed to such an extent that the affected intestinal segments appear unhealthy, a surgical procedure of these segments is performed because they can be a source of sepsis. Sometimes, clearly necrotic bowel segments are removed at the time of primary surgery, and a second-look

operation is planned to view and assess bowel segments that are borderline and can be salvaged once revascularization (32).

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