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ACUTE MESENTERIC ISCHEMIA – A CLINICAL REVIEW

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Abstract

AMI *(acute mesenteric ischemia) is basically a serious vascular emergency which requires urgent analysis and intervention to sufficiently reinstate mesenteric blood flow and to cure bowel necrosis and death of the patient.*

The primary cause is diverse, and the prognosis depends upon the precise pathological findings. Besides the procedure in understanding the AMI (acute mesenteric ischemia) pathogenesis and the modern treatment modalities development, AMI remains a specific and diagnostic challenge for medical professionals and clinicians, and the diagnosis delay contributes to the persistence high rate of mortality. Initial analysis and prompt operative treatment both are essential to enhance the clinical outcomes.

In a broader view, AMI (acute mesenteric ischemia) may be categorized further as venous or arterial. As an arterial disease, it sub-divided into NOMI (non-occlusive mesenteric ischemia) and OMAI (occlusive mesenteric arterial ischemia; where OMAI further divided in AMAE (Acute Mesenteric Arterial Embolism) and AMAT (Acute Mesenteric Arterial Thrombosis). On the contrary, acute mesenteric ischemia as the venous disease can be MVT (mesenteric venous thrombosis).

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1.0. INTRODUCTION:

AMI (acute mesenteric ischemia) is probably a fatal and severe vascular emergency with complete 60% to 80% rate of mortality. Acute mesenteric ischemia includes a pathophysiologic processes group which has a general endpoint – bowel necrosis. The patients' survival rate has not enhanced considerably in the last seventy years and its main factor is the constant implication in analysis and recognition of the patient's condition before bowel infarction happens. Clinical performance is non-specific generally and may be featured by an early inconsistency between minimal clinical findings and severe abdominal pain (Acosta, 2014).

The physical analysis does not dependably distinguish between infarcted and ischemic bowel. Issues such as peritonitis, ileus, gastrointestinal bleeding and pancreatitis can also mask the early symptoms and signs of acute mesenteric ischemia. AMI risk factors and also its clinical course vary as per the experience of the pathologic condition. As there is a rapid progress of bowel ischemia to irrevocable metabolic derangements ensue, severe bowel necrosis heading to a chain of events which terminate in death or several organ dysfunctions. The suitable use of therapeutic methods with a proper diagnosis for the swift restoration of blood flow is the main point to declining the high rate of mortality associated with acute mesenteric ischemia (Bala et al., 2017).

2.0 Pathophysiologic Processes

The splanchnic circulation obtains 25% (approximately) of the resting with 35% of postprandial cardiac results. Including 70% of the flow of mesenteric blood also directed to submucosal and mucosal layers of the bowel, including remnants supplying the serosal and muscularis layers. The physiologic features of splanchnic flow of blood are complicated and insufficiently understood. A different main factor interacts to deliver the intestinal tract including an applicable blood supply share, comprising the (intrinsic) metabolic and myogenic and (extrinsic) humoral and neural regulatory system (Bala et al., 2017).

The reactive hyperemia, pressure flow autoregulation, and hypoxic vasodilation are contemplated intrinsic regulators and have instantaneous fluctuations responsibility in splanchnic blood flow. In the theory of metabolism, delivery of oxygen rather than blood flow triggers an adaptive alteration in the splanchnic flow. An inequality between the demand and supply of oxygen tissues will raise local metabolism concentration; such as potassium, hydrogen, carbon dioxide and adenosine, ensuing in hyperemia and vasodilation (FINK, CHAUDHURI and DAVIS, 2015).

On the contrary, the myogenic study proposes the act of arteriolar tension receptors to manage vascular resistance in relation to transmural pressure. A severe decline in perfusion pressure is rewarded for by a decline in arteriolar tension wall, thus handling the splanchnic flow of blood. The extrinsic neural factor of splanchnic regulation of circulation includes the initiated vasoconstrictor fibers. Extreme vasoconstrictor fibers activation by adrenergic motivation results in small vessels vasoconstriction and a decline in the mesenteric flow of blood. After extended periods of adrenergic vasoconstriction, there is an increase in blood flow, probably by the adrenergic stimulus, which performs as a protective response (Bala et al., 2017).

After adrenergic stimulus cessation, comprehensive hyperemia generates the triphasic response. Though multiple kinds of neural stimulus (such as; histaminergic, vagal, cholinergic and sympathetic) may further affect the gut, adrenergic limb regarding autonomic nervous system is the main and likely the sole neural impact on splanchnic circulation. Norepinephrine and elevated degrees of epinephrine generate severe vasoconstrictions by the stimulus of adrenergic receptors. Some other pharmacologic complexes which delink splanchnic flow of blood comprise vasopressin, digoxin, and phenylephrine. Low dose dopamine produces splanchnic vasodilation, however, higher dose further lead to stimulating vasoconstriction adrenergic receptors. Papaverine, dobutamine, adenosine, sodium nitroprusside and fenoldopam mesylate are exogenous agents which may elevate mesenteric blood flow. Additionally, different naturally happening agents may assist as splanchnic vasodilators, comprising acetylcholine, nitric oxide, histamine, leukotrienes, glucagon, thromboxane analogs and a range of gastrointestinal hormones. Accordingly, the impacts of prostaglandins are different and the splanchnic circulation is managed by a complicated array of pharmacologic and physiologic features, which described through below-mentioned Table 1.

Table 1. Physiologic and Pharmacologic Factors Regulating Mesenteric Blood Flow (Extrinsic Regulatory System)

Decrease Blood Flow	Increase Blood Flow
Humoral (Endogenous and Exogenous)	
Epinephrine (high dose)	Epinephrine (low dose)
Norepinephrine (moderate to high dose)	Norepinephrine (low dose)
Dopamine (high dose)	Dopamine (low dose)
Phenylephrine	Dobutamine
Vasopressin	Sodium nitroprusside
Angiotensin II	Papaverine
Digoxin	Nitric oxide
Neural	
α-Adrenergic receptors	β-Adrenergic receptors
Dopaminergic receptors	

(Source: Bala et al., 2017)

2.1 Reperfusion Injury

Tissue hurt due to change in mesenteric blood flow is frequently the output of cellular injury linked with reperfusion. Comprehensive gaps of mesenteric ischemia indicate to an elevation in microvascular potentially, therefore extended ischemia further leads to the commotion of intestinal mucosal hindrances, basically through the functions of imprudent "oxygen metabolites and polymorphonuclear neutrophils". The blood flow reduction level that the bowel may abide without triggering this reperfusion remarkable mechanism (Ranjan, 2018).

Only 1/5 of the mesenteric capillaries are exposed at any given period of time and consumption of normal oxygen may be managed with only 20% of the maximal flow of blood. When the splanchnic flow of blood is reinstated, delivering relatively consistent oxygen consumption, oxygen extraction increases over a larger level of flow of blood rates. Therefore, when the flow of blood decrease according to the threshold level, consumption of oxygen declined and oxygen debt succeeds (Kim, Shim and Lee, 2014).

3.0 Causes

Acute mesenteric ischemia may be recognized into further four particular kinds based on some causes, as mentioned in Table 2.

Table 2. Clinical Features of Acute Mesenteric Ischemia

Cause	Incidence, %	Presentation	Risk Factors	Treatment
Arterial embolism	40-50	Acute catastrophe	Arrhythmia, myocardial infarction, rheumatic valve disease, endocarditis, cardiomyopathies, ventricular aneurysms, history of embolic events, recent angiography	Embolectomy, papaverine, excise infarction
Arterial thrombosis	25	Insidious onset with progression to constant pain	Atherosclerosis, prolonged hypotension, estrogen, hypercoagulability	Papaverine, thrombectomy, excise infarction, revascularization
Nonocclusive	20	Acute or subacute	Hypovolemia, hypotension, low cardiac output status, α-adrenergic agonists, digoxin, β-receptor blocking agents	Treat cause first, papaverine, excise dead bowel
Venous thrombosis	10	Subacute	Right-sided heart failure, previous deep vein thrombosis, hepatosplenomegaly, primary clotting disorder, malignancy, hepatitis, pancreatitis, recent abdominal surgery or infection, estrogen, polycythemia, sickle cell disease	Thrombectomy, excise dead bowel, heparinize, long-term complication

(Source: Bala et al., 2017)

3.1 Arterial Embolism

First, the most frequent cause is "Arterial Emboli" of AMI and is liable for 40% to 50% same cases; it mostly originated from a cardiac resource. Infarction or myocardial ischemia, endocarditis, atrial tachyarrhythmias, ventricular aneurysms, cardiomyopathies, and valvular ailments are major risk factors for the growth of the mural thrombus, which further embolize the mesenteric arteries. A mesenteric artery embolus, rarely, can happen in the time period or after angiography of the cerebral or coronary circulation. Highly visceral arterial emboli differently stay in the SMA (superior mesenteric artery) due to the aorta at a slanting angle. Therefore, 15% of arterial emboli happen at the SMA origin, 50% lodge distally to the base of the central colic artery, the main branch of SMA (superior mesenteric artery). Closely, 1/3 of the patients with SMA (superior mesenteric artery) embolus have specifically a history of an antecedent embolic event (Ranjan, 2018).

3.2 Arterial Thrombosis

Acute mesenteric thrombosis is liable for 25%, almost, of all ischemic happenings. About all the mesenteric ischemia caused by arterial thrombosis happens in the severe atherosclerotic disease setting, as per general site near the SMA origin. Normally, this condition patients may tolerate the visceral artery obstruction ad the slow developing atherosclerosis nature permits the growth of significant indemnities. Infarction or bowel ischemia follows when the end remaining visceral artery or significant collateral artery blocks. The amount of infarction or bowel ischemia is generally higher than that with embolism, ranging from duodenum to the transverse colon. The mortality ranges of perioperative about 70% to 100%, it happens due to the delay in the analysis, the massive nature of the bowel ischemia or infarction, and the requirement of highly complicated surgical revascularization (Seeger, 2017).

3.3 Non-occlusive Mesenteric Ischemia

Almost 20% of the patients specifically with mesenteric ischemia also have the non-occlusive disease. The pathogenesis of NOMI (non-occlusive mesenteric ischemia) is feebly implied but frequently contains a lesser cardiac result which linked with diffuse mesenteric vasoconstriction. Splanchnic vasoconstriction in accordance with hypovolemia reduced hypotension; the cardiac result and vasopressor accurately describe the variance between the other forms and entity of AMI(Kim, Shim and Lee, 2014).

3.4 Mesenteric Venous Thrombosis

MVT (mesenteric venous thrombosis) is the least general reason for mesenteric ischemia, and it representing up to only 10% of overall patients suffered from mesenteric ischemia and 18% from those having AMI. Previously, in many cases, there were thoughts from secondary to other intra-abdominal pathologic situations such as intra-abdominal sepsis, malignancy, and pancreatitis and were categorized as idiopathic (Ranjan, 2018).

4.0 Diagnosis

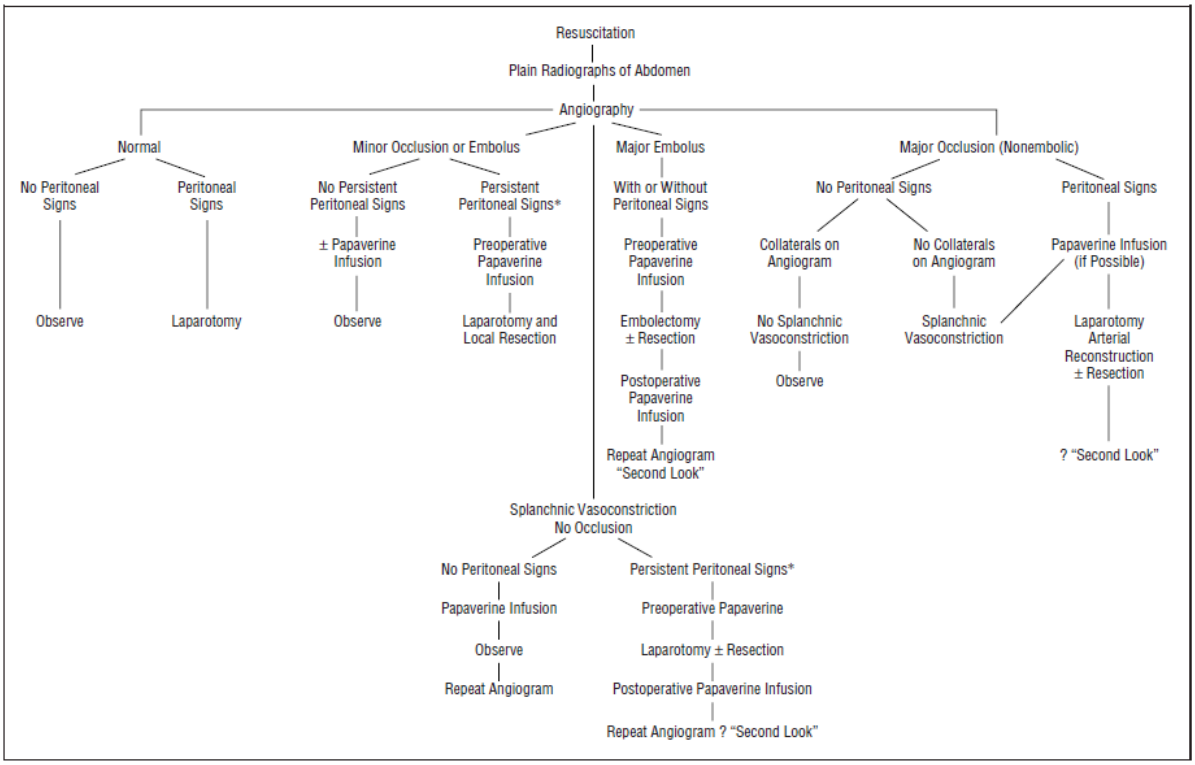
Due to AMI may ensue to serious intestinal infarction swiftly; prompt analysis and treatment are paramount. An excessive index of suspicion in the compatible history setting and physical examination attends as the foundation to initial mesenteric ischemia diagnosis. Once it is supposed, the medical professionals should act swiftly to assure the diagnosis and introduce the proper treatment. AMI (acute mesenteric ischemia) must specifically be considered in the disparity diagnosis when a patient age is older than sixty years and also has a history of atrial fibrillation, congestive heart failure, recent myocardial infarction, arterial emboli, abdominal pain or postprandial and weight loss; it is primarily seen for a specific abdominal pain and need to a physical examination. The survival rate is approximately 50% when analysis happens within twenty-four hours after the recognition of symptom but further dropped swiftly to 30% or less if the time of diagnosis is delayed (Kim, Shim and Lee, 2014).

The common abnormalities in the laboratory are hemoconcentration, metabolic acidosis, and leukocytosis, specifically with lactate concentrations and intense anion gap. High-grade serum amylase, lactate dehydrogenase, aspartate aminotransferase, and creatin phosphokinase are often analyzed at the demonstration but there is no sufficient sensitivity or particular to be analyzed. Hyperkalemia and hyperphosphatemia are basically associated with bowel infarction and considered late signs. In AMI, the diagnosis on a plain abdominal radiograph is nonspecific and in the initial stage of this disease, almost 25% of patients showed normal analysis of abdominal radiography. There are some abnormalities in radiographic also such as thickening or thumb-printing of bowel loops, but it happens in lesser than 40% of patients. Peritoneoscopy is also vital for investigating AMI according to venous thrombosis. Serosanguineous fluid, specifically in older patients' abdominal cavity with pain, hemoconcentration and leukocytosis also expressively advised for MVT (Seeger, 2017).

5.0 Diagnosis and Treatment

Preoperative mortality in patients experiencing revascularization specifically for AMI levels from 44% to 90%. Long-term outputs, as published data, showed the results after positive revascularization are very limited and generally diagnosis is not advantageous for patients of chronic mesenteric ischemia. It is also observed that recurrence is usually not unfamiliar and it transfers a poor diagnosis. The limited proportion of patients endures massive bowel resection and grows the syndrome of short-gut; it is also observed that it require long-term to small-bowel transplantation or complete parenteral alimentation.

When the analysis happened there must be a focus on treatment and it should be originated without any delay. Treatment should comprise the active resuscitation and underlying condition's treatment, with support directed at declining the associated vasospasm and curing propagation of the plotting process intravascular and diminishing the reperfusion injury. There is an algorithm of treatment summarizing the procedure in below-mentioned Figure:



(Source: Kim, Shim and Lee, 2014)

IV fluid resuscitation with blood products and crystalloids must be initiated swiftly to amend the deficit level and metabolic derangement. Ideally, fluid resuscitation must start before crystalloids and angiography and (maybe) managed through the amount as high as 100 mL/kg. Accordingly, broad-spectrum antibiotics must be provided as soon as possible. Specifically for severe mesenteric embolism, the embolectomy standard through a transverse arteriotomy in the proximal SMA must be executed. The arteriotomy is locked, after embolectomy, with interrupted non-absorbable polypropylene sutures. If the reason of severe mesenteric embolism is in qualm or if SMA thrombosis is supposed, then there must give some preference to the longitudinal arteriotomy (Seeger, 2017).

According to this approach, if a medical supervisor feels that a bypass is necessary, the longitudinal arteriotomy may be utilized as the distal bypass site graft anastomosis. Accordingly, if the flow is sufficiently restored due to bypass the longitudinal arteriotomy may be blocked by angioplasty patch to confirm that the luminal diameter is not conceded. Specifically, for AMI from arterial thrombosis which is by the cause of atherosclerotic disease, the vascular bypass graft mostly recommended by medical professionals. The graft may instigate from the supraceliac or infrarenal aorta. To evade the probable

intra-abdominal infectivity from perforated and infarcted bowel there must be revascularization need to perform while utilizing an autologous vein (Yudin and Veselyy, 2016).

6.0 CONCLUSION:

Acute mesenteric ischemia is basically a stimulating clinical issue with varied reasons, which frequently results in deferred analysis and treatment. A very strong clinical distrust, specifically considered as an aggressive method should be assumed while dealing with this situation due to the result critically based on quick analysis and treatment. With an optimized understanding of the AMI pathogenesis and the accessibility of the level of diagnostic and interventional methods and adjuvant pharmacotherapies, there must be some improved results may achieve.

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