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Challenges in Management of Mesenteric Ischemia

Review

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Abstract – Mesenteric ischemia is a complex disease characterized by insufficient blood supply to the mesentery and, therefore, the intestinal tissue, which continues to confuse doctors. Despite its low frequency, consistent clinical manifestations of pathology and radiological findings that are difficult to establish, can delay life-saving conditions in acute cases and make the quality of life of patients less stable. worse when it goes undiagnosed. This review is to summarize recent research updates and emerging intervention strategies for acute and chronic mesenteric ischemia. Acute and chronic mesenteric ischemia pose diagnostic and therapeutic challenges for practitioners. Computed tomography angiography remains the diagnostic method of choice in both cases. Open surgical procedures are the gold standard in acute mesenteric ischemia, while endovascular procedures are preferred in cases of chronic mesenteric ischemia.

Key Words – Mesenteric Ischemia; Revascularization.

I. Introduction

Mesenteric ischemia is a serious condition of insufficient blood flow to the mesentery and intestines that continues to confuse doctors. It is classified as "acute" or "chronic" depending on the duration of the hypoperfusion. Acute mesenteric ischemia includes three different types of insults that cause a decrease in blood flow in the mesenteric artery and ultimately lead to hypoperfusion of the intestine [1,2,3,4]. These lesions may include arterial emboli, often resulting from dislodged thrombus from the left atrium, left ventricle, cardiac valves, or proximal aorta [2,3,4]. In addition, they may arise from acute thrombosis in patients with preexisting chronic mesenteric ischemia due to atherosclerotic disease triggered by abdominal trauma, infection, thrombosed mesenteric aneurysms, and aortic or mesenteric dissections [2,3,4]. Mesenteric artery occlusion accounts for approximately 67-95% of acute mesenteric ischemia [3,4]. Other causes of acute mesenteric ischemia include mesenteric venous stasis, which can lead to venous thrombus formation [3,4]. This event can be idiopathic, seen in a hypercoagulable state, or secondary to a malignant disease or surgical procedure years ago [3,4]. Increased resistance to blood flow can lead to edema of the intestinal wall, which can promote ischemic events [4]. Occult mesenteric ischemia is considered as the result of hypoperfusion of splanchnic vessels and vasoconstriction [3,4]. A recent study found that the majority of cases of acute ischemia are usually complications in the superior mesenteric artery, which is followed by non-occlusive mesenteric ischemia[5]. Despite the low rate of acute mesenteric ischemia, accounting for only 0.09-0.2% of all hospital admissions in the United States [1], the clinical evolution of the disease and the radiological diagnosis are difficult to initiate and may be delayed to save lives in acute settings and destroying the quality of life of those who suffer from the disease when it is undiagnosed .

Chronic mesenteric ischemia differs from acute mesenteric ischemia, usually resulting from peripheral vascular disease where the metabolic needs of the intestine exceed the supply [6]. In the fasting state, approximately 20% of cardiac output is directed to the

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Vol. 47 No. 1 October 2024, pp. 42-56

mesenteric artery [6]. However, postprandial blood pressure increases by 100-150% for 3-6 hours due to vasodilation of mesenteric vessels that begins 3-5 minutes after ingestion [6]. Obstructed or stenotic vessels and chronic mesenteric ischemia reduce this postprandial hyperemic response, resulting in a mismatch between oxygen supply and demand, nutrient availability, and inadequate ingestion [6]. This may lead to symptoms such as pain, malabsorption, and delayed bowel emptying [6]. Although mesenteric artery stenosis is common (affecting up to 10% of the population over the age of 65), chronic mesenteric disease is rare, representing less than 1 in 1000 people. It affects patients aged 5^{th} to 7^{th} decade and has a high female/male ratio [7]. Patients may present with concomitant manifestations of atherosclerotic disease, like limb ischemia, cerebrovascular disease, and cardiovascular disease, among others. Risk factors that may predispose mesenteric vessels to atherosclerosis include diabetes, hypertension, smoking, and dyslipidemia [6,7,8].

There are other rare etiologies of chronic mesenteric ischemia, including fibromuscular dysplasia, medial arcuate ligament syndrome, vasculitis, connective tissue disease. The clinical relationship between known diseases and the presentation of abdominal symptoms is important to establish a diagnosis [9]. A recent study showed that patients with chronic mesenteric ischemia may also suffer from intestinal dysbiosis, which may resolve postoperatively [10]. This study highlights the importance of a comprehensive immune system for intestinal homeostasis and microbiome modulation [10]. The method of diagnosis and management of acute and chronic mesenteric ischemia is different depending on the chronic disease of the disease, always researching and repeating the traditional method. This review will review the most common clinical manifestations of chronic and acute mesenteric ischemia and review recent studies regarding their diagnostic criteria and preferred medical and surgical treatment. In addition, it provides a unique and comprehensive algorithm to assist in the early diagnosis and intervention of chronic and acute mesenteric ischemia. The main purpose of this review is to complement the recent studies that challenge the traditional diagnosis and intervention to help in the rapid diagnosis and effective treatment of mesenteric ischemia in the hope of reducing the number of high morbidity and mortality associated with this disease.

1. RISK FACTORS FOR NOMI

Currently, it is believed that high risk factors for NOMI include incomplete visceral trauma caused by cardiogenic shock, septic shock and dehydration, activation of the sympathetic response in conditions of low perfusion, increased heart rate and mesenteric arterial vasoconstriction. Other common risk factors include myocardial infarction, benign arrhythmias, valvular heart disease, major surgery, major trauma, and end-stage renal disease requiring long-term hemodialysis.[11-13] Regardless of the etiology of the shock, those who exhibit splanchnic dilatation by marked vasoconstriction and systemic vascular resistance will increase by more than 40% [14]. Studies have shown that if the cardiac output is reduced by 56%, the lower mesenteric blood flow will be reduced by 83% due to a significant increase in vascular resistance [15]. In the case of persistent hypotension, the autoregulatory system does not maintain adequate blood flow to the intestines. Also, the use of vasopressins such as norepinephrine and epinephrine can cause perfusion damage to the intestinal mucosa, leading to NOMI, [16-18] so ICU patients with unexplained abdominal pain or abdominal distension should be suspected NOMI. The role of enteral nutrition in the development of intestinal ischemia in critically ill patients is still controversial. In a recent post-NUTRIREA-2 study of 2410 patients with acute respiratory distress syndrome and shock,[19] all patients were not assigned to receive nutritional or parenteral nutrition. The results showed that nutritional factors, use of dobutamine, Simplified Acute Physiology Score II (SAPS II) ≥ 62, and hemoglobin ≤ 109 g/L were independently associated with AMI, suggesting that in the case of multiorgan failure and small heart output and / or height. SAPS II score, entral feeding should be delayed or used with caution in patients with airway obstruction who require a vasoconstrictor.

2. NOMI CLINICAL PRESENTATION

AMI does not have well-defined clinical manifestations, the first patients may have severe abdominal pain, nausea and vomiting, and symptoms of gastrointestinal bleeding may occur when intestinal necrosis occurs at a high level. Therefore, it is easy to confuse it with strangulated intestinal obstruction, abdominal hernia, digestive tract perforation and other diseases, causing missed wrong diagnosis. NOMI has an insidious onset and can present clinically with abdominal pain, abdominal distension, gastric residual volume >500 ml, intra-abdominal pressure >15 mmHg, loss of bowel, and even peritonitis and shock in severe cases. [20,21]. In

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2022, the World Society of Surgery (WSES) guidelines for AMI highlighted that NOMI, as one of the subtypes of AMI, often presents with pain and paroxysmal pain. Unexplained abdominal pains or gastrointestinal bleeding may be the only sign of acute intestinal ischemia in patients with NOMI, but about 25% of patients admitted to the ICU may not experience it. Patients with diarrhea and bacteremia after cardiopulmonary resuscitation should be suspected of NOMI, regardless of abdominal pain. Right abdominal pain is accompanied by brown or shiny stool, which is suggested to be NOMI.

Table 1 Clinical, laboratory and imaging markers of NOMI

Items	Symptoms and signs
Clinical	Abdominal bloating/Ileus
	Abdominal pain and tenderness
	Severe abdominal pain
	Gastric residue volume > 500 mL
	Intrabdominal pressure > 15 mmHg
	Absent bowel sounds
	Peritonism (focal/diffuse)
	Oliguria
	Tachycardia
	Hypotension/shock
Laboratory	Leukocytosis
	Hyperlactacidemia
	Metabolic acidosis
Radiological	Mucosal thumb printing
	Dilated bowel
	Thickened bowel wall
	Mucosal hypoperfusion on CTA
	Intestinal pneumatosis
	Portal venous gas
	Pneumoperitnoeum

CTA, computed tomography angiography; NOMI, non-occlusive mesenteric ischemia.

Adapted from Al-Diery *et al.*[21]

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Vol. 47 No. 1 October 2024, pp. 42-56

3. NAME OF BIOMARKERS

Currently, there are no biomarkers that can accurately predict or diagnose AMI. In recent years, some studies have shown that the use of factors such as intestinal fatty acid binding protein (I-FABP), serum α-glutathione S-transferase (α-GST) , and ischemia-modified albumin can be used to diagnose AMI.

3.1 I-FABP

The fatty acid binding agent is a lipid chaperone protein system, which is unique to the intestine. When the integrity of intestinal epithelial cells is destroyed, I-FABP is released into the blood and the level of I-FABP is related to the degree of injury.[14]. One study evaluated the value of I-FABP alone and in combination with traditional biomarkers in the evaluation of NOMI.[22] The results showed that the sensitivity of the NOMI leukocyte test was 40% and the specificity was 85.9%. The sensitivity of NOMI diagnosis by D-Dimer is 52% and the specificity is 87.3%. The sensitivity of NOMI detection by lactic acid is 60% and the specificity is 88.7%.Sensitivity, specificity and area under the curve (AUC) of NOMI detection by I-FABP are 76%, 80.3% and 0.805, respectively. Another multicentre research study to determine the value of I-FABP and citrulline in the diagnosis of NOMI showed that in the case of NOMI complicated by intestinal necrosis, the blood level of I-FABP increased significantly (AUC = 0.83).[23] The sensitivity, specificity, negative predictive value and positive predictive value for the diagnosis of intestinal necrosis were 70%, 85%, 58% and 90%, respectively, while the difference in plasma citrulline content was not significant. statistics. However, the use of I-FABP in diagnosing ischemic bowel disease is still limited. I-FABP levels are increased in other types of intestinal diseases (such as acute enterocolitis, Crohn's disease, and simple intestinal obstruction) and kidney failure. Some NOMI patients have low I-FABP levels due to intestinal cell depletion, which directly affects the diagnosis of NOMI.[24-25]

3.2 α-GST

GST is a type of catalytic enzyme found in the intestinal mucosa, which has a short half-life and is involved in oxidative stress. When intestinal mucosal cells were damaged by ischemia and hypoxia, α-GST expression in serum increased. α-GST is considered as a biomarker for mesenteric ischemia, and measuring the level of α-GST can provide useful diagnostic information for mesenteric ischemia, therefore, it may have important clinical applications in diagnosis. and monitoring of ischemia of the small intestine.[26]. Treskes et al. found that α-GST increased in patients with AMI, with a sensitivity of 67.8% and a specificity of 84.2%.[27] There is evidence that α-GST plays an important role in the auxiliary diagnosis of AMI, and the occurrence of AMI can be completely excluded if the concentration of α-GST is less than 4 μg/L. [28,29] However, since it is not only in the intestine but also in the renal and hepatic epithelial cells, it is not enough to evaluate the damage of the intestinal mucosa by α-level alone.[30] So far, there are few studies about α-GST in the context of NOMI alone, and the clinical significance of α-GST in these patients needs to be further clarified in the future.

3.3 Ischemia-modified albumin

Ischemia-modified albumin is one of the potential biomarkers for the diagnosis of AMI.[31] In the physiological state, there is a binding site at the N-terminus of human serum albumin, which can bind to transition metal ions (e.g. cobalt). Under conditions of hypoxia and ischemia, the structure of the N-terminal of the protein will change. This may be due to its exposure to reactive oxygen species, which reduces its ability to bind to iron ions. A clinical study showed that albumin levels altered by ischemia were significantly higher in patients with AMI than in controls [32]. Polk et al. showed that 12 patients with AMI had higher ischemia-modified albumin levels than patients with non-mesenteric ischemia among 26 included subjects.[33]. A meta-analysis (including a case-control study of 7 cases of thromboembolic occlusion of the superior mesenteric artery) showed that the sensitivity and specificity of albumin ischemia changed in the diagnosis of AMI was 94.7% (95%) interval [CI], 74.0%- 99.9%) and 86.4% (95% CI, 65.1%-97.1%).[27] Since NOMI is only one type of AMI, the clinical value of ischemia-modified albumin in patients with isolated NOMI remains to be further evaluated in patients, and the study population focused on all types of IAM, The clinical value of ischemia-modified albumin in patients with isolated NOMI remains to be further investigated in patients whose study is focused on all etiologies.

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4. NOMI IMAGEING EXAMINATION

4.1 Computed tomography angiography

Clinical practice has confirmed that computed tomography angiography (CTA) is currently the best method for diagnosing AMI. As soon as patients suspect AMI, they should undergo a CTA test immediately. The presentation of the CTA examination is closely related to the pathophysiological changes.[34-36] Computed tomography angiography shows edema and thickening of the intestinal wall, as well as an abnormal increase in the mucosal layer due to increased vascular permeability. In addition, signs of intestinal wall ischemia can appear due to the poor distribution of arterial blood, which is used to reduce the intestinal wall. Ascites and mesenteric thickening are also angiographic signs of AMI during computed tomography (CT). The pathophysiological changes in the central part of AMI are those that are localized in the intestinal submucosa and tissue layer, such as intestinal wall edema, ulceration, hemorrhage, and necrosis. Computed tomography angiography shows paralysis of the intestine, and the gray area on the wall of the intestine is dilated and thick, which is reduced by ischemia. At this time, even if the blood flow of the intestine is restored, there is still a risk of intestinal fibrous stenosis and frequent bowel obstruction. The main pathological changes in the final stage of AMI are increased capillary permeability, intestinal wall necrosis, and bacterial translocation. On the other hand, CTA will see dilated and thin bowel wall, sometimes showing air bubbles between the bowel wall, mesentery and portal vein, or even a free abdominal gas sign. According to Yu et al., arterial vasoconstriction is seen in CTA images of patients with NOMI, such as stenosis of the superior mesenteric artery branch and vasospasm leading to a "beaded" or "sausage" appearance.[37] Compared with the previous computed tomography (CT) or healthy controls, the diameter of the superior mesenteric artery in patients with NOMI is very small, and the diameter < 4 mm is helpful for diagnosis. Because NOMI usually occurs in low perfusion conditions, the portal and systemic venous systems may be reduced or collapsed, but pre-CT revascularization may prevent cases. It should be noted that if CT angiography does not confirm mesenteric ischemic disease and doctors strongly suspect AMI, mesenteric arteriography should be performed to confirm or refute the diagnosis as soon as possible.[38] . In patients with renal insufficiency, if AMI is suspected, CT angiography should also be performed, because the consequences of late diagnosis will be worse.[39]

4.2 View more images

An abdominal radiograph is often the first imaging test in patients with acute abdominal pain, but the radiographic diagnosis of AMI is not specific, especially in the early diagnosis of AMI. Only when intestinal infarction occurs and the intestine shows free gas in the abdominal cavity can an X-ray film be taken with good results. Negative x-ray film cannot rule out AMI, so an abdominal x-ray film has little meaning.[40] Diagnosis of B-mode ultrasound has high requirements for the operator, and it is easily affected by the accumulation of intestinal gas and edema. Although various CT scans can detect indirect signs such as the intestinal wall of the intestine, intestinal obstruction, content accumulation, and mesenteric vascular accumulation, it is useful for the early diagnosis of AMI, experienced operators are often required to operate and explain it. research findings.[41] Arterial angiography used to be considered the best method for diagnosing AMI, but it is expensive, complicated and invasive, so it is rarely used to diagnose AMI, but it is often used in early treatment time.

5. ACUTE MESENTRIC ISCHEMIA

5.1. Clinical presentation

The clinical presentation of acute mesenteric ischemia manifests as pain that does not correspond to physical examination findings [42]. As the disease progresses, the intestinal mucosa may extend to the intestinal wall, corresponding to the onset of necrosis [42]. The duration and characteristics of the pain are related to the underlying pathophysiology, and thromboembolic causes often cause sudden onset or gradual onset over a few days, while mesenteric ischemia is tending to continue to move slowly, it varies in strength and stability [42]. The clinical examination can be different in the case of non-occlusive mesenteric ischemia, and unexplained abdominal cramps or gastrointestinal bleeding are sometimes the presenting symptoms, especially in sedated patients in the intensive care unit, in about 25% of cases [43].

Patients with severe mesenteric ischemia often have other symptoms of severe disease, including septic shock, cardiac problems, and respiratory distress, which may require the use of vasopressor drugs [43]. A recent study has established a link between pain or

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Vol. 47 No. 1 October 2024, pp. 42-56

pain requiring morphine with acute mesenteric ischemia [44]. Obviously, cases of acute mesenteric ischemia have also been recorded in patients with COVID-19, which is attributed to high thromboembolic events and small vessel thrombosis associated with hypercoagulability and retention and showing higher hospitalization rates than patients without COVID-19 [45,46]. Identifying risk factors for acute mesenteric ischemia can improve the health status and facilitate early diagnosis. A recent study has highlighted several demographic characteristics and factors associated with this pathology. It is common in elderly patients, especially those with many systemic diseases, including heart disease, endocrine and metabolic diseases, kidney disease, digestive disease, respiratory disease, cerebrovascular disease, vascular disease and cancer [47]. Furthermore, critically ill patients with non-obstructive mesenteric ischemia who are prescribed vasoconstrictor agents may increase vasoconstriction of the superior mesenteric artery, potentially contributing to organ infarction [48]. Interestingly, the role of atherosclerosis as a risk factor for non-occlusive mesenteric ischemia has been debated in the literature [49].

5.1.2. Diagnosis

The early and correct diagnosis of acute mesenteric ischemia is important to obtain the best results, since the delay in diagnosis is the main cause of the mortality rate of the disease between 30% and 70% . Intestinal ischemia should be considered for any critically ill patient with unexplained deterioration [43,50].Although blood tests can confirm the clinical diagnosis of acute mesenteric ischemia, abnormal or often abnormal findings in normal laboratory values should not prevent rapid imaging in cases of high clinical suspicion. Studies have examined various biomarkers and blood tests and their correlation with the diagnosis, severity, and prognosis of acute mesenteric ischemia patients. For example, the CRP/albumin ratio has been identified as a strong predictor of in-hospital mortality, surpassing white blood cell count, neutrophil/lymphocyte ratio, and lactate levels [51]. However, other studies have shown that markers such as L-lactate, D-dimer, leukocytosis and neutrophil/lymphocyte ratio may not have strong predictive value [52]. Elevated lactate levels should increase the clinical suspicion of mesenteric ischemia, but may also be nonspecific. High lactate levels and tolerance are associated with high mortality rates in acute mesenteric ischemia [53]. In addition, the diagnostic value of D-dimer has been questioned, and studies suggest that they can serve as exclusion tests but lack specificity [54]. There has been an increase in plasma I-FABP and intestinal necrosis in patients with non-invasive mesenteric ischemia [55].

The presence of peritoneal signs on physical examination is an indication for abdominal/emergency surgical examination to evaluate for intestinal perforation, necrosis, and other complications [43]. Severe suspicion of acute mesenteric ischemia without peritoneal signs requires advanced imaging to warrant further investigation [43,56]. A plain radiograph of the abdomen can exclude the presence of intra-abdominal free air indicating bowel perforation, but is considered non-specific for intestinal ischemia [56]. Computed tomography (CT) angiography is the considered initial imaging in patients with high suspicion of acute mesenteric ischemia [57]. The World Association of Emergency Surgery and the American College of Radiology strongly recommend the rapid use of CT angiography, as studies have shown that every 6 hours of delay increases the rate of death [21,26]. Oral contrast should not be used because it may obscure the mesenteric vessels and enhance the intestinal wall [56]. The standard CT method for large mesenteric ischemia usually includes a biphasic method, the first of which includes the angiographic segment that is important from the whole stomach using a high number of different injections [58].

Early diagnostic tests can detect vascular calcification, hyperattenuating intravascular thrombus, and intramural hemorrhage [43]. The subsequent arterial phase with contrast can reveal the defect filling the vein or the area of infarction in the tissue corresponding to the vein disease, planning the surgery [58]. Venous systems can reveal hyper attenuation in the venous system regarding thrombi [43,58]. Multiple reconstructions can help determine the origin of the mesenteric artery [43]. This biphasic method showed high sensitivity (96-100%) and specificity (94-100%) in the diagnosis of acute mesenteric ischemia [58]. Although some studies have evaluated the value of the triphasic CT method in addition to the non-contrast method, no significant benefit has been found over the traditional biphasic test [58]. Dual-energy CT is an emerging tool that improves intraluminal contrast, which can be particularly useful in situations where time or contrast is a problem [57,58]. In rare cases of mesenteric ischemia, computed tomography angiography can reveal intestinal ischemia and free fluid in the presence of patent mesenteric vessels [143]. Mesenteric venous thrombosis can be manifested as signs of intestinal wall, pneumatosis, splenomegaly, and ascites, and portal and mesenteric venous gas indicates intestinal obstruction [52]. Several CT radiological parameters for acute mesenteric ischemia have been identified by recent meta-analyses [59,60]. These include narrowing of the intestinal wall, reduction or lack of improvement of the intestinal

wall, intestinal dilatation, pneumatosis intestinalis, portal mesenteric venous gas and acute arterial occlusive mesenteric ischemia . Intestinal wall, portovenous gas, and mesenteric venous gas are specific for the diagnosis of transmural intestinal necrosis [59,60].

Studies are expecting the involvement of the colon in about 28% of the total final mesenteric Ischemia. Wall thickening is the most common CT sign in such cases. Occlusion of the inferior mesenteric artery is a major risk factor for colonic damage. Colon involvement on CT has been associated with increased morbidity and mortality in cases of acute mesenteric ischemia (AMI) [61]. Although magnetic resonance imaging (MRI) can be considered in the diagnosis of acute mesenteric ischemia and can provide high sensitivity, CT is preferred due to its availability, speed and low cost [56]. If the diagnosis is still uncertain despite CT or MR angiography, catheter-based arteriography is recommended [43]. Doppler ultrasound is very useful in detecting large mesenteric ischemia due to gas in the bowel lumen and can clearly see the mesenteric vessels in obese patients [57].

5.1.3. Medical Management

Medical management of acute mesenteric ischemia involves a comprehensive approach that includes several procedures. Rapid rehabilitation includes bowel decompression, volume resuscitation, hemodynamic monitoring and support, correction of electrolyte disturbances, symptom management, prophylaxis, and anti-inflammatory drugs [56]. These procedures should be considered as temporary measures in anticipation of surgery. Any time the surgical intervention can be fatal. The inflammatory response following the dissection of the intestinal wall can lead to significant capillary permeability. Therefore, adequate crystalloid rehydration is important to improve bowel function while awaiting surgery. The loss of the mucosal barrier puts patients at risk of bacterial translocation, requiring early initiation of various antibiotic treatments [62].

Medical management of non-occlusive mesenteric ischemia depends on the underlying cause of intestinal hypoperfusion [43,63]. In such cases, rehydration remains essential to improve intestinal perfusion, maintain and improve cardiac output, and avoid vasoconstrictor agents [43]. In cases where the etiology is unclear, systemic antibiotics and unfractionated heparin may be considered [43]. Other treatment methods can also be used such as catheter placement of vasodilatory antispasmodic agents such as papaverine hydrochloride [63,64]. Continuous intravenous infusion of prostaglandin E1 has shown promise to improve mortality rates in patients with early signs of non-occlusive mesenteric ischemia [43,65]. A recent study shows that continuous prostaglandin infusion can reduce lactate accumulation and improve survival in non-occlusive mesenteric ischemia [66]. A recent study showed that direct parenteral anticoagulant therapy improves the prognosis in patients with intestinal obstruction [67]. A study involving animal models suggested that single-dose pretreatment with albendazole might decrease the inflammatory response and increase the ischemia threshold after the induction of a mesenteric reperfusion injury. The study reported a decrease in pro-inflammatory cytokines in the treated group [68]. More studies are needed to confirm the effects of albendazole in humans before its implementation in clinical practice.

5.2 Surgery versus endovascular revascularization

A recent national study in Germany has defined a therapeutic approach for acute mesenteric ischemia. In most surgical patients, visceral surgery is preferred over endovascular intervention [69]. However, visceral surgery is preferred, especially in patients with a high risk of complications, such as intestinal ischemia, perforation or peritonitis, so it is associated with a high 7 Comparative studies between open and endovascular procedures have shown similar results in patients with indications for laparotomy [70,71]. Logically, the open method has made it possible to extend the response time [70]. Although some small studies report that it will improve the endovascular intervention [70,72,77,74]. lower rates of gastric rupture, reduced need for parenteral nutrition, shorter hospital stay, and lower cost compared to open surgery [70,74,75,76].

6. CHRONIC MESENTRIC ISCHEMIA

6.1. Clinical presentation

Chronic mesenteric ischemia, commonly referred to as "intestinal angina," is characterized by recurrent and relapsing pain due to small bowel hypoperfusion in patients with multivessel mesenteric artery stenosis. or occlusion [77]. Other less well-defined symptoms include weight loss, food aversions, nausea, vomiting, early satiety, and irritability such as diarrhea or constipation [77].

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A history of other symptoms related to atherosclerotic disease spread, such as angina, stroke/transient ischemic disease, or lower extremity, may be present [77]. Physical examination can be vague and sensitive, with a mild tenderness that is not specific or safe, but it is usually normal for most patients [77]. Abdominal murmurs can be diffuse in approximately 50% of patients, but should not be considered as the only diagnostic feature [77].

6.2. Diagnosis

The chronically poor discovery is not weak because of other difficulties to adopt her disease to an earner ship of his illness [77]. These conditions include peptic ulcers, cholecystitis, malignancy, and gastroparesis [77]. Arterial imaging is important to diagnose chronic mesenteric ischemia and to detect stenosis or occlusion of the great mesenteric vessels [77]. CT angiography is the first non-invasive method preferred, because it can detect stenotic or closed vessels and at the same time exclude other pathologies [77]. Its sensitivity is high (100%) and its specificity (95%) [78]. Common CT findings in chronic mesenteric ischemia are stenosis of the mesenteric vessels, thickening of the bowel wall, the presence of free peritoneal fluid and large vascular lesions [78]. Duplex ultrasound is another reasonable method for diagnosis [77]. A peak systolic velocity ≥ 275 cm/s for the superior mesenteric artery and ≥ 200 cm/s for the celiac artery indicates a stenosis greater than 70% and is important for diagnosis [77]. The sensitivity of duplex ultrasound in chronic mesenteric ischemia is 72 to 100% and the specificity is 77 to 90% [78]. Endoscopic duplex ultrasound has been shown to have a higher sensitivity than transabdominal duplex ultrasound, but it is rarely used in the clinic and is not considered standard of care [79].

Digital subtracted arterial angiography can be used to confirm the diagnosis if it is unclear and a treatment plan is established [77]. This allows selective catheterization and pressure relief in stenotic vessels, helping to define the important hemodynamic quality of these lesions [80]. Magnetic resonance angiography (MRI) is also considered as an alternative to computerized tomography (CT) for detecting stenotic or mesenteric vessels, but direct comparisons between these two methods are limited. However, CT angiography is generally superior to contrast-enhanced magnetic resonance angiography in coronary arteries [81]. A recent retrospective study developed and tested new markers to assess the severity of mesenteric artery disease [82]. This is a six-weighted score called the "CSI score", with "C" for the celiac artery, "S" for the superior mesenteric artery and "I" for the inferior mesenteric artery, and is based on the number of vessels related and the extent and degree of stenosis or obstruction of the visceral arteries involved [82]. The study found that elevated patients had higher rates of coronary heart disease, chronic kidney disease, and peripheral arterial disease [82]. A statistical analysis showed that most participants with high-grade symptoms underwent endovascular intervention, open surgery, or open replacement after endovascular treatment for chronic mesenteric ischemia [82]. Patients with higher scores also had higher mortality rates [82]. CSI signifies a positive ability to speak for nerves and associations and associations and needs and needs, surgery in surgery and death [82]. Scoring tools such as these, in addition to imaging findings, patient surgical candidacy, and interventionalist preferences or skills, can aid in determining optimal and individualized therapeutic intervention strategies.

6.3 Management

Interventions in patients with chronic mesenteric ischemia are aimed at reducing symptoms, improving nutritional status, and preventing intestinal infarction [78]. Lifestyle changes are important and include encouraging patients to manage risk factors such as blood sugar, smoking cessation, controlling blood pressure, improving diet and adequate exercise [78]. Conservative management usually includes antiplatelet therapy and high-dose statins, but all patients requiring surgery must undergo revascularization [78]. A meta-analysis comparing dual antiplatelet therapy with mono antiplatelet therapy after endovascular arterial revascularization found no significant benefit in restenosis or stent thrombosis, but showed an increased risk of bleeding with dual antiplatelet therapy [83]. For some patients, oral antibiotics such as rivaroxaban may be considered [84]. One study found that low-dose rivaroxaban plus aspirin reduced cardiovascular events and adverse events compared with aspirin alone without an increase in major bleeding [84]. The Society for Vascular Surgery recommends revascularization in symptomatic patients with chronic mesenteric ischemia, with the aim of reversing symptoms and improving quality of life [85]. The primary target of revascularization should be the superior mesenteric artery, with the celiac artery and inferior mesenteric artery considered secondary targets [85]. Asymptomatic patients with severe mesenteric stenosis should be discussed with the need for revascularization [85]. Endovascular revascularization is

recommended as the first treatment in patients with resectable lesions, opening up surgical revascularization for patients with unresectable lesions by endovascular intervention [83]. Patients with untreated chronic mesenteric ischemia face a 5-year mortality rate of more than 100% [77].

Surgical revascularization is preferred in the first place; However, the endovascular approach has a lower rate of intrahospital cardiac and cerebrovascular diseases, a shorter hospital stay, less intrahospital complications, and a higher cost [70,71,77]. They have become the best treatment method for lesions that can be resected by endoscopy [83]. Obliterating the aortic valve, small vessels, large calcifications, tandem lesions, and degree of stenosis may lead to endovascularization [85]. The skill and preference of the operator determine the endovascular technique chosen to restore patency in stenotic blood vessels [85]. The Journal of Vascular Surgery reports that balloon-encased expandable stents have replaced traditional balloon angioplasty due to long-term improvements [85]. After revascularization, most patients continue to have symptomatic relief for 5 to 10 years, but restenosis can occur in 20 to 40% of patients and require revascularization [78]. High-dose statins after endovascular intervention are associated with an improvement in primary patency [86]. A serial duplex ultrasound examination at 3-6 intervals is recommended to evaluate restenosis or recurrence of symptoms [78]. If the vessel is unable to undergo endovascularization, an open surgical procedure may be considered [85]. Possible approaches include antegrade bypass of the supraceliac aorta, retrograde bypass of the common iliac artery (or infrarenal aorta), aortic endarterectomy, and open retrograde mesenteric stenting [85]. Few studies have compared the methods directly, but individual studies have shown their high rate of improvement in technology and clinical practice [85]. Several studies have evaluated the results of various interventions for chronic mesenteric ischemia. Patient diseases often play an important role in long-term mortality. Factors such as myocardial infarction, malignancy, age, and chronic lung disease have been associated with different mortality rates depending on the treatment regimen [86]. However, comparative studies suggest that endovascular revascularization can have a similar effect to open surgical procedures, and show a high rate of technical failure in time and the return of symptoms and needs for regeneration [87]. Despite the high early and late success of the technology associated with open surgical procedures, they have been associated with a high rate of serious adverse events and 30-day mortality. [88]

II. CONCLUSION AND FUTURE PROSPECTIVES

Acute and chronic mesenteric ischemia continues to pose a great challenge to diagnosticians and practitioners due to the often unspecific presentation that delays diagnosis. and results in a high death rate. Early detection and revascularization are important for optimal outcomes, better survival and quality of life. A good understanding of pathophysiology, diagnostic methods and treatment options are important for health professionals to detect pathologies early and improve outcomes through timely intervention. Biphasic CT angiographic protocol is necessary for the diagnosis of both pathologies. New studies continue to refine the diagnostic criteria for both conditions. Early signs of transmural intestinal necrosis and acute mesenteric ischemia, such as those that have recently been associated with the intestinal wall, reduced or absent improvement of the intestinal wall, intestinal dilatation, pneumatosis intestinalis, and the presence of a mesenteric portal venous. gas, can help practitioners determine the best course of treatment for a patient. Another study can be done to determine whether endoscopic ultrasound is a possible diagnostic tool for chronic mesenteric ischemia, because it can be a secondary procedure if transabdominal ultrasound or CT angiography is something equal. Traditionally, blood and urine tests have played a low-suspicious role in the evaluation and diagnosis of acute mesenteric ischemia. However, recent studies are trying to find useful tests. Preoperative evaluation of lactate, creatinine, I-FAB, ischemia-modified albumin, procalcitonin, or IL-6 may help in determining the extent of disease and the need for open surgery instead of endovascularization surgery, but it more studies are needed to confirm this. information. It may be useful to combine abnormal laboratory values and imaging findings into diagnostic and diagnostic criteria, such as the CSI score for chronic mesenteric ischemia, to determine which treatment would be best in a different situation.

As revascularization procedures for acute and chronic ischemia progress and the popularity of endovascularization procedures increases, the importance of risk stratification and prognosis increases. Open surgery must be available for reference time and optimal intervention for acute mesenteric ischemia, because subsequent laparotomy for necrosectomy is often necessary with endovascularization. Hybrid interventions including standardized approaches to blocked vessels may have a role to play, but more

studies are needed to design standardized strategies. Adjunctive treatment may be indicated for acute mesenteric ischemia, because the temporary benefits of prostaglandin and infusions of albendazole are seen in small patients and animals, respectively. Other trials in human patients are needed to determine their specific contribution to whether infusions before surgery delay revascularization. In patients with chronic mesenteric ischemia, endovascular procedures are becoming the preferred method of treatment, and rightfully so. They are less invasive and have shown functional results similar to open surgery. However, more research is needed to identify patients at risk for in-stent stenosis and the need for reoperation, because their rate of failure is high. These patients may benefit from open surgery rather than an endovascular approach. Further studies are needed to compare the results of different open surgical procedures, as none have been done recently, possibly due to the popularity of the endovascular approach. The high mortality rate of acute and chronic mesenteric ischemia prompts doctors to know the best diagnostic and treatment methods at present. Early approval and intervention is essential to achieve the best results. The advent of new diagnostic techniques and revascularization for acute and chronic diseases provides many opportunities for new research or future support. As shown above, more studies have been done on acute mesenteric ischemia than chronic, making future studies focus on the latter. A larger scale of studies is needed to support the current retrospective study, given the lack of interest in it. However, it can be difficult to understand this due to the rarity of these conditions.

CONFLICT OF INTEREST

All authors declare no conflicts of interest.

AUTHOR CONTRIBUTION

Authors have equally participated and shared every item of the work.

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