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Isolated superior mesenteric artery dissection: An updated review of the literature

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ABSTRACT

Isolated superior mesenteric artery dissection (ISMAD) is a rare but potentially life-threatening cause of acute abdominal pain. Owing to the availability of computed tomography angiography, more cases have been detected during screening for acute abdomen in recent years. With increasing knowledge of ISMAD, a better management strategy is being developed. To enhance our understanding and improve treatment outcomes of ISMAD, a systematic literature review was conducted with a focus on diagnosis and management strategies based on existing evidence.

1. Introduction

Isolated superior mesenteric artery dissection (ISMAD) refers to dissection occurring solely in the superior mesenteric artery (SMA), which typically occurs spontaneously and does not involve the aorta. Initially considered rare, ISMAD now appears to be on the rise, particularly among the Chinese population, owing to the widespread availability of computed tomography angiography (CTA).[1-4] Symptoms of ISMAD can vary; however, in most cases, patients present with moderate to severe acute abdominal symptoms. Diagnosis relies on modern imaging techniques. Early recognition and appropriate management are critical because ISMAD can be catastrophic. Although our understanding of its etiologies and pathologies remains limited, significant improvements in its diagnosis and treatment have occurred over the past decade.[5,6] This study aimed to systematically review the current diagnosis and management strategies for ISMAD based on the existing literature.

2. Epidemiology

ISMAD was first reported by Bauersfeld in 1947.[7] While it was once deemed rare,[8] recent advances in imaging technology have led to an increase in ISMAD diagnoses over the past decade[1,9] with the majority of cases reported in East Asia.[1,9,10] Recent reviews show that approximately 3000 cases of ISMAD have been reported worldwide, with a mean patient age of 55.1 years (range 49–68 years) and a male predominance (89%).[11,12]

3. Etiology

Although the causes and underlying etiology of ISMAD have not yet been fully characterized, it appears to be multifactorial, consisting of systemic diseases and anatomical and genetic components.

3.1. Systemic disease

There is a strong association between ISMAD and systemic diseases such as atherosclerosis, medial degeneration, and fibromuscular dysplasia.[13,14] Coincident celiac and renal arterial dissections and aneurysms have also been reported,[15,16] providing further evidence of a systemic component of the disease. Moreover, hypertension and a history of smoking are common among ISMAD patients.[17,18] These findings suggest that ISMAD has a strong systemic component.

3.2. Anatomic characteristics

The anatomy of the SMA is a reported risk factor for ISMAD. The mean aortomesenteric angle ($59.7 \pm 21.4°$ vs $48.2 \pm 16.8°$; $p < .001$) and SMA maximum curvature (0.084 ± 0.078 mm-1 vs 0.032 ± 0.023 mm-1; $p < .001$) were higher in ISMAD patients than controls.[19] The convexity of SMA after bending causes shear stress against the anterior wall, the most common site of ISMAD.[1]

3.3. Genetic component

In a single case report, ISMAD was hypothesized to be linked to the chromosomal locus 5q13–14.22. However, no genetic studies to date have confirmed this hypothesis.[20]

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4. Pathophysiology

In most cases of ISMAD, the dissection is localized in the curved portion (1–3 cm away from the root) of the SMA.[1,21,22] Hemodynamic studies demonstrated that the distribution of wall shear stress in the SMA can cause degenerative changes in the anterior wall of the SMA, including thinning of the media, a reduced smooth muscle cell count and elastin level, translamellar mucoid extracellular matrix accumulation, elastic fiber fragmentation, and smooth muscle cell nuclei loss or disorganization.[18,19,23]

5. Pathology

Pathological examinations were conducted of only five patients with ISMAD; among them, only one showed signs of fibromuscular dysplasia, while the other four were idiopathic.[14,24] In addition, medial degeneration was found in patients with ISMAD.[13]

6. Clinical manifestations

The clinical manifestations of ISMAD are nonspecific.[1] Patients may be asymptomatic if only a small dissection without vascular supply is compromised. However, among established diagnosed patients, the majority experienced moderate to severe abdominal pain (91%).[1,10] Other symptoms include nausea, vomiting, diarrhea, and bloody stools.[1,4] However, no clinical symptoms or physical examination findings are pathognomonic for ISMAD.[25] Signs of peritoneal irritation may occur, indicating the presence of pan-peritonitis or intestinal necrosis, but this is a very delayed complication of ISMAD. In rare cases, careful auscultation may reveal a vascular murmur in the peri-navel region caused by turbulent blood flow in the false lumen. This finding was reported in 17.2% (5/29) of patients with ISMAD.[26]

7. Imaging studies

Contrast-enhanced computed tomography (CT) is typically the first-line imaging modality for patients with acute abdominal pain. However, in cases of highly suspected ISMAD or equivascular initial CT scan findings, CTA may be preferred. For patients with severe contrast allergies and no time for premedical treatment, color Doppler flow imaging (CDFI) can serve as an alternative diagnostic tool.[1,27] Although magnetic resonance angiography (MRA) is an option for ISMAD diagnosis, it is not commonly used because of its time-consuming nature. Digital subtraction angiography (DSA) is reserved for patients requiring intervention.[1,27]

7.1. CT angiography

CT with intravenous contrast is the most commonly used screening tool for acute abdominal pain. CTA is used to further characterize the nature of the dissection and follow patients. CTA can be used to assess ISMAD and assess the abdominal organs, which can be helpful when formulating a differential diagnosis. This feature has made CTA the most commonly used modality for screening patients with clinically suspected ISMAD.[1,2]

Although ISMAD is diagnosed in approximately 95% of patients during the initial work-up for an acute abdomen with a CT scan using intravenous contrast,[1] CTA is the preferred imaging modality for the further characterization of the nature and extent of the dissection, complications of ISMAD, visceral organ status, and follow-up.

The direct signs of ISMAD on CTA are a double cavity of the SMA, a true lumen that is compressed by the false lumen, the presence of an intimal flap, and the presence of entry and re-entry points.[2,28] The indirect signs of ISMAD on CTA are SMA thrombosis, SMA stenosis or obstruction, the formation of collateral circulation, thickening or thinning of the intestinal wall, unenhanced or poorly enhanced intestinal wall due to ischemia, intestinal gas accumulation, and intestinal dilatation. Although these indirect signs are of little significance in diagnosing ISMAD, they indicate that intestinal ischemia or necrosis has occurred and provide guidance for follow-up treatment.[3,27]

7.2. Color Doppler flow imaging

CDFI examinations are indicated for the follow-up of patients with ISMAD.[29,30] CDFI, including color doppler ultrasound (CDU) and contrast-enhanced ultrasound (CEUS), can show the true and false lumen, entry and re-entry sites, dissection location, intimal flap location, and hemodynamic changes.[3,31] CEUS is reportedly more effective than CDU at detecting the false lumen and entry and re-entry sites; thus, CEUS is commonly used in combination with CDU.[29] CDFI examinations can be affected by many factors such as operator experience and skill, patient obesity, and large amount of gas in the bowel. Despite being noninvasive, radiation-free, and simple to perform,[29] CDFI is less effective at demonstrating dissections located in the distal main trunk of the SMA and cannot be used to assess the degree of intestinal ischemia.

7.3. Magnetic resonance angiography

MRA can be used as an alternative diagnostic tool in patients with severe contrast allergies (unsuitable for CTA), obesity, or large amounts of gas in the bowel (unsuitable for CDFI).

7.4. Digital subtraction angiography

Although DSA is an excellent tool for diagnosing ISMAD and can demonstrate the degree of stenosis of the true lumen, diameter of the false lumen, and entry and re-entry sites of the false lumen, it can also show the collateral circulation. However, DSA plays a minimal role in establishing a diagnosis. DSA is usually performed for endovascular intervention and assists in treatment decision-making.[28] The disadvantages of DSA are its inability to determine a thrombosed false lumen and visceral organ status.[28]

When performing DSA, posteroanterior and lateral angiography should be performed with additional oblique or three-dimensional angiography, if necessary, to fully delineate the ISMAD. Angiography of the common hepatic and inferior mesenteric arteries is also required to show the collateral circulation.

8. Classification

ISMAD is a pleiomorphic disease for which a systematic classification is required. In recent years, many classification systems for ISMAD have been proposed, including classifications by Sakamoto,[32] Yun,[25] Zerbib,[33] Li,[34] Luan,[27] Xiong,[35] and Yoo.[36] However, consensus is lacking regarding which system should be used.[40]

The original Sakamoto classification is based on the imaging appearance of the false lumen and categorizes ISMAD into four types.[32] However, this system does not consider the condition of the true lumen, such as in cases of total thrombotic occlusion of the SMA. Yun's classification is based on angiographic findings and divides ISMAD into four types depending on the presence of false luminal flow and true lumen patency in the dissected segment. Subsequent classifications have been built on these previous systems, but are used less frequently because they are more complicated.

Therefore, a simple classification based on the symptoms, signs, and imaging findings of ISMAD is required; however, such a classification is lacking.

9. Treatment

The treatment plan is tailored to the patient's clinical signs and symptoms as well as imaging findings that provide valuable insights into the condition.[37,38,41] Depending on the specifics of the case, treatment

options may include conservative measures, such as medication and physical therapy, or more invasive interventions, such as endovascular or open surgery.[42]

9.1. Conservative therapy

Conservative therapy is indicated for patients with ISMAD type I, type IIa with a false lumen diameter ≤2 cm,[43,44] type IIb, or type III, according to the Chinese expert consensus on ISMAD management.[42]

Conservative therapy consists of observation alone or fasting, gastrointestinal decompression, pain control, strict blood pressure control (≤120/80 mmHg), and lactulose syrup treatment.[39,40] Fasting should be used in patients with moderate to severe abdominal pain[45] with a gradual switch to liquid, semiliquid, and normal diets once it has subsided. Patients with abdominal discomfort or pain after meals should be advised to eat multiple small meals throughout the day consisting of easily digestible food. Gastrointestinal decompression can be performed in patients with abdominal distension. For patients with moderate to severe abdominal pain, analgesics should be administered; however, strong analgesics should be avoided, as they can mask the patient's condition. Blood pressure should be controlled to ≤120/80 mmHg to prevent rupture of the false lumen. Finally, prophylactic antibiotic therapy should be considered in patients with severe abdominal pain and increased white blood cell and neutrophil counts.

Anticoagulation or antiplatelet therapy prevents secondary thrombosis of the true lumen in patients with ISMAD. However, a meta-analysis of 35 articles and 727 patients demonstrated that anticoagulation or antiplatelet therapy did not improve clinical outcomes in patients with ISMAD.[46] These therapies are not conducive to thrombosis in the false lumen and interfere with SMA remodeling.[40] Therefore, conventional anticoagulation or antiplatelet therapy is not recommended for patients with ISMAD unless evidence is noted of thrombosis in the false lumen or severe stenosis in the true lumen.

Although most cases of ISMAD can be managed successfully with conservative therapy, this treatment strategy fails in approximately 20% of cases.[45] The failure of conservative therapy is defined as the persistence or aggravation of symptoms or signs, progression of ISMAD on images, or occurrence of intestinal necrosis or mesenteric artery rupture after conservative treatment.[40] Patients for whom conservative therapy fails will require active intervention.[9]

9.2. Endovascular therapy

Endovascular therapy is indicated for patients with type IIa ISMAD with a false lumen diameter >2 cm,[43,44] patients without intestinal necrosis for whom conservative therapy has failed,[47] and patients with SMA rupture, especially those with limited SMA rupture or life-threatening disease.[48]

Endovascular therapy can consist of stent placement, stent-assisted coiling, or coiling alone.[49,50] Stent placement is most commonly used in patients with ISMAD.[1] Stent-assisted coiling can be used for patients in whom stent placement has failed and blood flow persists in the false lumen after the procedure.[40] Coiling alone is sometimes used to embolize the false lumen.[50]

To date, no stents have been developed specifically for treating ISMAD.[40] Bare stents alone can be used in most patients; these stents can open the stenosed true lumen and promote false lumen thrombosis.[39] Covered stents can be used in cases of SMA rupture or dissection in the SMA ostium. Distal edge stenosis may occur after patients are treated with stents.[51] Thus, antiplatelet therapy should be administered to patients undergoing stent placement.[52]

9.3. Open surgical therapy

Open surgical therapy is indicated for patients with intestinal necrosis, those with SMA rupture, those who are not candidates for endovascular therapy, or those in whom endovascular therapy has failed.[53]

Open surgical therapy may consist of bypass surgery, vascular repair, endarterectomy plus patch angioplasty, fenestration, or other techniques.[43,53] Regardless of the technique used, open surgery is becoming an uncommon treatment option; most reported cases of open surgical treatment for ISMAD occurred in the 1990s.[54,55] In China, only 3.2% of patients with ISMAD underwent open surgical therapy in recent years.[1] With the continuing development of endovascular technology, open surgery will become even more uncommon in the future.

10. Clinical outcomes

Most patients with ISMAD have good clinical outcomes, according to the literature, regardless of the presence of symptoms.[4,45,56] A meta-analysis of 51 articles and 721 patients reported an ISMAD-related mortality rate of just 0.69% during a mean of 26.5 months of follow-up.[10]

10.1. Short-to medium-term outcomes

Most patients with ISMAD present with abdominal pain that usually resolves within 2 weeks after treatment.[4,40] Chronic abdominal discomfort or pain occurs in some patients; these symptoms will be resolved with the formation of collateral circulation among the SMA, celiac artery, and inferior mesenteric artery.[57] Clinicians should monitor patients for the occurrence of intestinal necrosis and SMA rupture despite their rarity in patients with ISMAD.[58]

10.2. Long-term outcomes

In patients treated with conservative therapy, complete SMA remodeling is common, even in patients with severe stenosis of the true lumen.[45,56] For those who undergo stent placement, the long-term stent patency is 90% (9/10), with no symptoms occurring, even in the case of occlusion.[59] For patients who undergo open surgical therapy, the long-term outcomes are good in patients without short bowel syndrome.[57]

11. Clinical follow-up

Patients with ISMAD require regular follow-up, as this is a dynamic disease process.[3] Clinical follow-up should include assessments of symptoms, signs, and images to evaluate ISMAD status. The follow-up strategy should be based on the patient's status and treatment method. Imaging should be performed whenever a symptom or sign relapse is observed.

For patients who undergo conservative therapy, clinical follow-up should be scheduled for the first month after diagnosis and annually thereafter until complete mesenteric artery remodeling has occurred or for a maximum of 2 years if no changes in ISMAD are noted.[3,40] For patients who undergo endovascular therapy, clinical follow-up should be scheduled for 3 and 6 months after the procedure. Because of the risk of stent restenosis and occlusion, patients who undergo stent placement should be scheduled for follow-up every 3–5 years to assess stent patency.[49,60] For patients who undergo open surgery, clinical follow-up should be scheduled for 1 and 6 months after the procedure, with further follow-up visits scheduled based on the results of the previous visit.

CTA and CDFI can be performed during follow-up.[28,29] CTA is recommended for patients with complex or unstable ISMAD, whereas the combination of CDU and CEUS is recommended for patients with simple and stable disease, those who underwent stent placement and/or coiling (as the metal material could cause artifacts on CTA, complicating the assessment of stenosis), those with an allergy to iodinated contrast agents, and those with renal insufficiency. The use of CDFI instead of CTA for long-term ISMAD surveillance should be considered, especially

considering the decreased radiation, contrast, and costs associated with ultrasound imaging.[3,29]

12. Conclusions

ISMAD is being increasingly detected in recent years; however, its etiology remains unclear. The diagnosis of ISMAD is primarily based on CTA findings. Treatment decisions should be based on the patient's symptoms and signs as well as the morphological characteristics of ISMAD on imaging. Conservative therapy is suitable in most ISMAD cases. The clinical outcomes of ISMAD are good in most patients regardless of symptoms. Nevertheless, patients with ISMAD require regular follow-up until complete SMA remodeling has occurred or for a maximum of 2 years if no changes in ISMAD are noted. Both CTA and CDFI are recommended for assessing ISMAD status.

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Declaration of competing interest

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