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Mesenteric Ischemia: Diagnosis and Management

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Abstract

Mesenteric ischemia is a rare etiology of abdominal pain caused by impaired blood flow to the mesenteric circulation. This condition has acute and chronic variations and is associated with high mortality and morbidity. Early diagnosis is needed to determine management and prevention efforts towards a worse prognosis. The main principles of management of mesenteric ischemia are the restoration of bowel perfusion and resection of the necrotic bowel. This review will discuss the basic materials, diagnosis, and patient management strategies based on the conditions and causes of mesenteric ischemia.

Keywords: mesenteric ischemia, necrosis, thrombosis, embolism

INTRODUCTION

Intestinal ischemia occurs due to insufficient blood flow in the mesenteric circulation. This condition is considered a vascular emergency if it occurs in the acute phase, which is associated with high morbidity and mortality [(1)]. Although rare, this condition is reported to have a surgical emergency rate of 0.09% - 0.2% and a mortality rate ranging from 30% - 90% [(2)]. Patients with mesenteric ischemia usually present with gastrointestinal symptoms, especially abdominal pain. However, these symptoms are non-specific and diagnostic tests may be inconclusive. This makes it difficult to establish the diagnosis of mesenteric ischemia. Early diagnosis is necessary to prevent a worse prognosis so that prompt and appropriate management can also be carried out [(1,3)]. This review will discuss mesenteric ischemia, how to diagnose it, and the management based on the conditions and causes of ischemia extracted from the existing literature.

METHOD

In-depth electronic searches were performed with Google Scholar. Furthermore, extensive research was carried out on relevant journals and references. The search only included publications in English and Indonesian. The search included reviews and original articles. Articles written in languages other than English and Indonesian and articles with limited access were not eligible for consideration. The following keywords were used: mesenteric ischemia, necrosis, thrombosis, and embolism. Mendeley software was used to compile the articles obtained. Mendeley software was also used to remove some of the duplicate citations found during searches in several databases. The full text of each article was read and summarized after the search results were sorted by title and abstract.

RESULT

Twenty-three studies were included in this brief review. The author read the full text of the selected publication. A secondary search of cited citations was also carried out to ensure that all relevant papers were included. Extraction of data from each study was carried out using Microsoft Word, and data extraction was checked and confirmed by the authors before being summarized and compiled into a brief review.

DISCUSSION

Anatomy and physiology

There are three main arterial branches that supply the blood vessels of the colon and small intestine, namely the celiac trunk, superior mesenteric artery (SMA) and inferior mesenteric artery (IMA) (Figure 1). The celiac trunk carries blood to the distal esophagus and descending duodenum. SMA supplies blood to the transverse and ascending duodenum, jejunum, ileum, and colon at the level of the splenic flexure. The IMA supplies the distal colon to the superior part of the rectum [1,4,5].

In addition to the main arteries, there are collateral pathways that provide blood supply to the intestine, such as the gastroduodenal artery, the marginal artery of Drummond, and the arcade of Riolan (intestinal arterial arcade) [1,4].

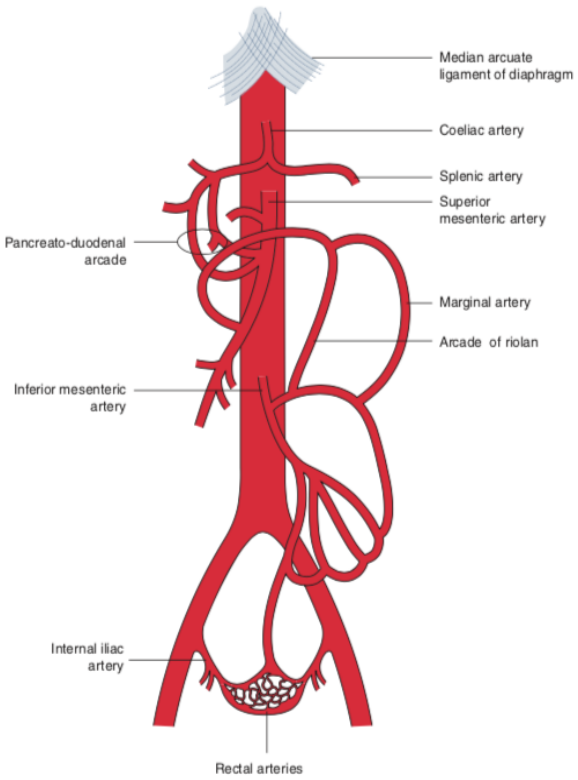

Figure 1. Mesenteric artery anatomy [4]

Venous return flows parallel to the arteries. The inferior mesenteric vein joins the splenic vein, which then joins the superior mesenteric vein to form the main portal vein [1].

Pathophysiology

Under normal circumstances, the human intestine receives 20% of the cardiac output. Autoregulation of intestinal perfusion maintains tissue viability in the presence of a systemic blood pressure of 70 mm Hg. If blood pressure is too low, local myogenic autoregulation is overtaken by systemic autoregulation and local protective mechanisms fail. This results in the occurrence of ischemia in the intestinal wall. Ischemic lesions can trigger the process of releasing inflammatory mediators such as cytokines, platelet-activating factors, and tumor necrosis factor [1,6,7].

In the early stages of ischemia, clinically, there will be complaints of sudden spasmodic abdominal pain. After 3-6 hours, there is a painless interval phase due to intramural pain receptor damage caused by prolonged hypoperfusion. Acute circulatory failure causes irreversible mucosal ischemia with leukocyte infiltration. Damage to the mucosal barrier can cause bacterial invasion of the intestine, resulting in bacteremia and sepsis [4,5].

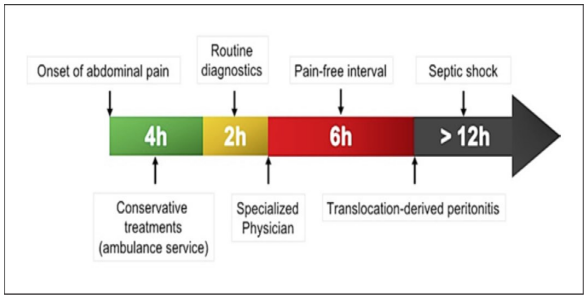

Figure 2. Clinical manifestations of mesenteric ischemia based on the time course of the disease [3]

Ischemic damage to the intestinal wall is divided into several stages, namely:

- Stage I : limited to the mucosa
- Stage II : extends into the submucosa and layers of the muscularis propria
- Stage III : entire lining of the intestine (transmural infarction)

Mesenteric blood flow increases 30-150% after a meal because of the increased demand for oxygen during digestion. Complaints occur after eating in patients with early stages of chronic mesenteric ische-

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mia. In the advanced stage, patients usually complain of permanent abdominal symptoms exacerbated by eating. This occurs due to insufficient blood flow during the pre-prandial period [4].

Acute mesenteric ischemia

As many as 90% of cases of ischemia are acute. This condition is an emergency and can cause irreversible hypoperfusion within hours [1]. Acute mesenteric ischemia has a mortality rate ranging from 50-70%. Heart failure, atrial fibrillation, coronary heart disease, arterial hypertension and peripheral vascular disease have been reported to be risk factors for acute mesenteric ischemia. Acute ischemia due to arterial embolism or thrombosis, venous occlusion, or nonocclusion [1,3,8].

Embolic and thrombotic acute mesenteric ischaemia (EAMI/TAMI)

Arterial occlusion is most often due to thromboembolism. Embolism usually originates from the left atrium as a result of atrial fibrillation. Other causes of heart disease include myocardial infarction, mitral stenosis, arrhythmias, ventricular aneurysms, and valvular endocarditis [5]. Embolism usually occurs in SMA because it has a wider diameter and smaller angle than other arteries. EMI is characterized by sudden abdominal pain in patients >70 years of age and a history of atrial fibrillation. The location of abdominal pain can vary. Other symptoms can include nausea and vomiting. Peritonitis may result from infection progressing to transmural infarction. Symptoms that appear can include fever, bloody diarrhea, and shock [1,5].

Occlusion in TAMI is gradual. Symptoms that appear are called mesenteric angina, namely postprandial abdominal pain and nausea, causing patients not to want to eat, feel full easily, and lose weight. The main risk factors for TAMI are atherosclerosis and dyslipidemia [1,8].

Venous acute mesenteric ischaemia (VAMI)

VAMI is more common in younger patients with milder symptoms. Fifty per cent of patients have risk factors such as a history of deep vein thrombosis or pulmonary embolism. Distal venous thrombosis is more at risk for hemorrhagic infarction compared to proximal veins. This is due to collateral connections between the mesenteric and systemic veins in the proximal part [1].

VAMI is not associated with postprandial symptoms. This type of ischemia is usually characterized by subacute abdominal pain lasting up to 2 weeks. Other symptoms may include flatulence, abdominal distension, fever, and the presence of blood on stool examination [1].

Non-occlusive mesenteric ischaemia (NOMI)

Intestinal ischemia can occur due to decreased intestinal perfusion caused by shock states such as sepsis, hemorrhagic, or cardiogenic. The shock causes a drop in systemic blood pressure, which results in reflex vasoconstriction of the mesenteric arteries to divert blood supply to the brain and heart [1,9].

Some patients with mesenteric ischemia caused by NOMI have risk factors, including age > 50 years, history of myocardial infarction, congestive heart failure, aortic insufficiency, cardiopulmonary bypass, kidney or liver disease, or major abdominal surgery [1,9]. Incidence increases in patients on chronic hemodialysis with subsequent hypovolemia and intestinal vasospasm in patients after cardiac surgery [1,10]. The diagnosis of NOMI can be suspected in patients with mesenteric hypoperfusion due to shock and vasoconstrictor drugs if there is an unexpected deterioration in the clinical condition. Symptoms may include acute abdominal pain, bloating, abdominal distension, and blood on stool examination. NOMI can also be a silent disease in critically ill patients. Findings on CT may mimic other intestinal diseases such as enteritis and colitis [1,6,9].

Table 1. Causes of mesenteric infarction by etiology [1]

Arterial Occlusion	Venous Occlusion	Nonocclusion
Arrhythmia	Infiltrative	Hypovolemia
myocardial infarction	Neoplasm	Hypotension
Valve disease	Inflammation (acute pancreatitis, appendicitis, diverticulitis, peritonitis)	Low cardiac output
Atherosclerosis	Hypercoagulable disorder	Chemotherapy
	Right heart failure	Immunosuppression
Prolonged hypotension	Infiltrative	Drugs that cause vasoconstriction (digitalis, vasopressin, adrenaline, noradrenaline) Medications that cause hypotension (diuretics, antihypertensives, antidepressants)

Diagnosis

Physical examination and laboratory results are not sensitive and specific enough to diagnose mesenteric ischemia. So the clinician must have a broad differential diagnosis in patients with complaints of abdominal pain [1,6]. Examination of serum lactate can describe the presence of anaerobic metabolism in ischemic tissue. However, this examination is non-specific [11]. Investigation in the form of multidetector computed tomography (MDCT) is accurate and non-invasive and has high specificity and sensitivity. This examination is a first-line imaging method for diagnosing mesenteric ischemia [1,2,9].

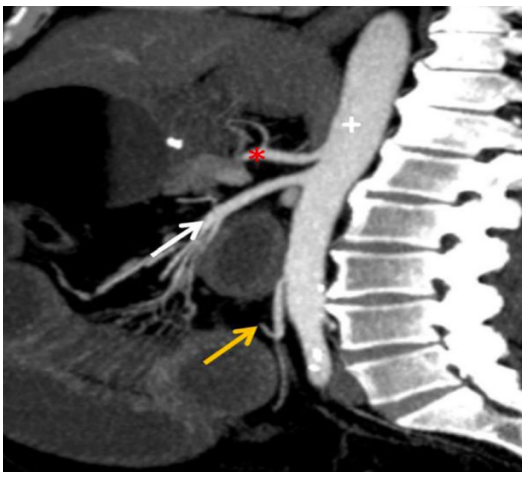

Figure 3. Main mesenteric artery anatomy on sagittal CT scan: celiac trunk (*), SMA (white arrow), IMA (orange arrow), abdominal aortic branch (+)[1]

The MDCT image is taken from the hepatic dome to the perineum so the entire intestine can be seen. Triphasic CT performed was precontrast scanning, arterial and venous phase. A non-contrast CT scan may be performed before a contrast CT scan to detect vascular calcifications, intravascular thrombi, and intramural hemorrhage. Contrast CT can identify the presence of mesenteric vessel thrombus, abnormal intestinal wall enhancement, and embolism or infarction in other organs. CT angiography can accurately depict mesenteric vascular anatomy and can differentiate occlusive and non-occlusive mesenteric ischemia [1,8,12].

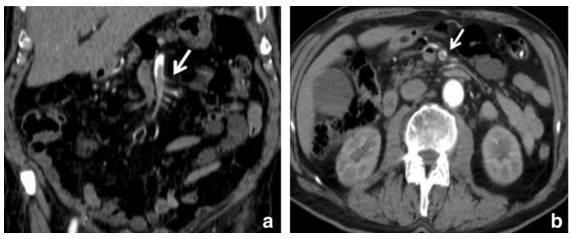

Figure 4. Abdominal contrast-enhanced CT in a 54-year-old female patient with SMA thrombosis (white arrows) (a) coronal section, (b) axial section [1]

The CT appearance of mesenteric ischemia varies depending on the pathogenesis of bowel ischemia, the location and extent of ischemia, the state of the collateral circulation, the degree of acuteness, duration and presence of superimposed bowel wall infection, or the presence of

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bowel perforation. If bleeding or edema occurs, the bowel wall may thicken up to 15 mm. In contrast to cases of arterial occlusion or transmural infarction, intramural nerves and intestinal musculature are damaged, resulting in a "paper thin wall" appearance, namely dilation and loops filled with gas or liquid with extreme thinning of the intestinal wall. Dilatation occurs due to intestinal peristaltic disturbances due to ischemic damage to the intestinal wall. Intestinal ischemia may manifest as hypoattenuation or hyperattenuating [1,2,9].

On non-contrast CT, hypoattenuation indicates bowel wall edema, whereas hyperattenuation indicates intramural hemorrhage and infarction. On CT with contrast, "pale ischaemia" or loss of bowel enhancement may be found, whereas in the postperfusion period, bowel hyperenhancement or "shock bowel" may be found [1,5].

Treatments

Treatment is given based on the patient's clinical status and radiological findings. Emergency surgery was performed on a patient with an unstable condition and signs of peritonitis. If there are no signs of peritonitis or intestinal gangrene, pharmacotherapeutic intervention with local fibrinolysis or embolectomy can be performed in cases of peripheral embolism [3,12].

Other treatments, such as administration of fluids, systemic anticoagulants, correction of electrolytes and blood gases, and antibiotics, can be given. Anticoagulants in the form of heparin 5000 units were started intraoperatively and continued with a controlled dose. Broad-spectrum antibiotics should be given early to prevent the progression of infection that can lead to sepsis. Close monitoring is also necessary to monitor for possible secondary organ failure [3,5,6].

Surgery

Surgery is performed when there are signs of peritonitis, central SMA occlusion, or failure of endovascular action. The primary goal of surgery is to obtain arterial reperfusion prior to bowel resection. Resection is performed on parts of the intestine where irreversible ischemia has occurred [3,6,8,13].

Endovascular

The endovascular technique is performed in hemodynamically stable patients without signs of peritonitis. This action aims to reopen the main branches of the artery so that the part that is still occluded can be perfused and produce good collateral growth. This treatment consists of catheter aspiration embolectomy angiography and catheter lysis with recombinant tissue plasminogen activator, urokinase, or pharmacotherapy with prostaglandin E1. In cases of thrombosis, recanalization is followed by stent placement [2,14–16].

NOMI management

In hemodynamically stable patients, catheter angiography is recommended as therapy. Vasodilators are injected so that they can restore vascular spasms. Immediate surgery is performed on patients with suspected gangrene [8–10].

Differential diagnosis

Ischemia colitis

Ischemia colitis and mesenteric have the same etiological factors. However, the symptoms that appear are different. In colitis, symptoms often appear within a few hours, abdominal pain is moderate, and accompanied by sanguineous diarrhea. The therapy given is conservative [1].

Thrombosis Mesenteric Vein

Thrombosis can occur in stagnant blood flow, hypercoagulability, and vascular disorders. Venous thrombosis can also cause irreversible bowel wall damage if it is centrally located and affects the periphery. There is 3 pathogenesis that causes intestinal venous thrombosis, namely direct injury caused by acute pancreatitis, inflammatory bowel disease, or surgical trauma; local venous congestion due to portal hypertension or congestive heart disease; and thrombophilia. Symptoms are non-specific and depend on the extent of the thrombosis. CT with biphasic contrast can be performed to support the diagnosis. Surgery is per-

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formed on patients with signs of peritonitis. Thrombolysis with an antegrade transmesenteric catheter can be performed intraoperatively to assist local thrombus lysis. In stable patients without signs of peritonitis, recanalization was performed via transjugular transhepatic access with or without a transjugular intrahepatic portosystemic shunt (TIPS). Anticoagulation with intravenous unfractionated heparin can be given to patients with an initial diagnosis without signs of peritonitis [3,6,17,18].

Chronic mesenteric ischemia

Chronic mesenteric ischemia occurs in as many as 10%. This type can cause complaints of postprandial abdominal pain, fear of eating, and weight loss. Chronic ischemia can also develop into acute again. The increasing incidence of cardiovascular disease in old age, obesity, and diabetes mellitus contributes to the increased incidence of chronic mesenteric ischemia [6,19].

Etiology

Causes of chronic mesenteric ischemia are divided into occlusive and nonocclusive types (NOMI). Occlusive chronic ischemia is caused by atherosclerosis, median arcuate ligament syndrome (MALS), vasculitis, or mesenteric venous thrombosis (MVT). Atherosclerosis is the most common occlusive cause. Associated risk factors are smoking, hypertension, diabetes, hypercholesterolemia, and a family history of cardiovascular disease [4,20].

MALS, formerly called Dunbar syndrome, is a symptomatic eccentric compression of the celiac trunk by the median arcuate ligament (MAL). Fibrous arches of the ligaments unite with the diaphragmatic crura. Compressions become more painful during maximal expiration [21]. Vasculitis is a rare cause of chronic ischemia. This condition often occurs in patients with polyarteritis nodosa, immunoglobulin (Ig) A vasculitis, and Takayasu arteritis. MVT can cause an acute condition due to obstruction. This cause is rare because collateral veins usually form more quickly [4,20].

Chronic NOMI occurs in patients with typical symptoms in the absence of ischemic mucosal stenosis or occlusion. This type is characterized by low-grade ischemia associated with heart failure, pulmonary hypertension, chronic obstructive pulmonary disease, mesenteric arterial vasospasm, low blood flow conditions (eg, patients with chronic renal failure on hemodialysis) and chronic anemia [4,20].

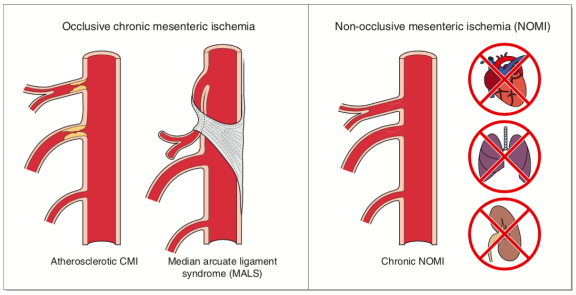

Figure 5. Etiology of Chronic Mesenteric Ischemia [4].

Diagnosis

Symptoms of chronic mesenteric ischemia include postprandial abdominal pain, starting from 10-30 minutes after eating and lasting up to 1-2 hours. Patients usually adapt their diet to prevent postprandial pain by eating smaller portions. This can also lead to fear of eating and weight loss at a later stage. Other complaints can be diarrhea and nausea. On physical examination, an abdominal bruit may be found [4,8,19,20,22].

On laboratory examination, there are no specific biomarkers indicating the occurrence of chronic mesenteric ischemia. Computed tomography angiography (CTA) and contrast-enhanced magnetic resonance angiography (CE-MRA) are the gold standards for examining patients with suspected chronic mesenteric ischemia. CTA can detect the presence of atherosclerotic plaques and calcifications. This test has a sensitivity of 100% and a specificity of 95-100% [4,19,20,23].

Functional tests by visible light spectroscopy (VLS) are performed during gastrointestinal endoscopy. Measurement

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of oxygen saturation in the upper gastrointestinal mucosa was performed with an endoscope connected to a VLS oximeter. In diagnosing chronic ischemia, VLS has a sensitivity of 90% and a specificity of 60% [4,20].

Management

Revascularization is indicated in patients with chronic mesenteric ischemia to relieve symptoms, improve quality of life, and increase life expectancy to prevent bowel infarction. Endovascular revascularization is the treatment of choice in chronic ischemia. Stent placement is also performed with vascular stenosis. Revascularization surgery is performed when endovascular techniques fail, are not performed because of extensive occlusion and calcification, or contraindications to radiation or contrast media [8]. Treatment for MALS consists of surgical removal of the MAL, fused diaphragmatic sections, and celiac plexus excision. If stenosis of the celiac artery persists afterwards, bypass surgery or endovascular therapy may be performed [4,20,21].

Differential diagnosis

Chronic mesenteric ischemia has a similar differential diagnosis of abdominal pain such as chronic pancreatitis, celiac disease, duodenal ulcer, abdominal malignancy, and irritable bowel syndrome (IBS) [19].

CONCLUSION

Mesenteric ischemia occurs due to a lack of blood flow in the mesenteric circulation, which causes the metabolic needs of the intestine to be unfulfilled. Ischemia can be either acute or chronic. In the acute phase, it is an emergency and can be life-threatening. The clinical presentation is non-specific, making it difficult to diagnose directly, and other modalities are needed for a proper diagnosis. Selection of fast and appropriate management is also needed based on the cause of ischemia, the patient's clinical condition, and the availability of health facilities. This is important to improve the quality of life and prevent patient mortality.

REFERENCES

1. Florim S, Almeida A, Rocha D, Portugal P. Acute mesenteric ischaemia: a pictorial review. Insights Imaging. 2018;9(5):673–82.
2. Olson MC, Fletcher JG, Nagpal P, Froemming AT, Khandelwal A. Mesenteric ischemia: what the radiologist needs to know. Cardiovasc Diagn Ther. 2019;9(4):S346–59.
3. Kühn F, Schiergens TS, Klar E. Acute Mesenteric Ischemia. Visc Med. 2020;36(4):256–63.
4. Terlouw LG, Moelker A, Abrahamsen J, Acosta S, Bakker OJ, Baumgartner I, et al. European guidelines on chronic mesenteric ischaemia – joint United European Gastroenterology, European Association for Gastroenterology, Endoscopy and Nutrition, European Society of Gastrointestinal and Abdominal Radiology, Netherlands Association of Hepa. United Eur Gastroenterol J. 2020;8 (4):371–95.
5. Amini A, Nagalli S. Bowel Ischemia. In Treasure Island (FL); 2022.
6. Bala M, Kashuk J, Moore EE, Kluger Y, Biffl W, Gomes CA, et al. Acute mesenteric ischemia: Guidelines of the World Society of Emergency Surgery. World J Emerg Surg. 2017;12(1):1–11.
7. Zafar M, Farooq M, Abousamra A, Marshall A, Whitehead M. Mesenteric ischaemia following posterior myocardial infarction. Clin Med J R Coll Physicians London. 2021;21 (4):E423–5.
8. Kärkkäinen JM. Acute Mesenteric Ischemia: A Challenge for the Acute Care Surgeon. Scand J Surg. 2021;110(2):150–8.
9. Pérez-García C, De Miguel Campos E, Gonzalo AF, Malfaz C, Pinacho JJM, Álvarez CF, et al. Non-occlusive mesenteric ischaemia: CT findings, clinical outcomes and assessment of the diameter of the supe-

rior mesenteric artery. Br J Radiol. 2018;91(1081).
10. Sakamoto T, Lefor AK, Kubota T. Non-occlusive mesenteric ischaemia associated with anorexia nervosa. BMJ Case Rep. 2019;12(5):10–2.
11. Isfordink CJ, Dekker D, Monkelbaan JF. Clinical value of serum lactate measurement in diagnosing acute mesenteric ischaemia. Neth J Med. 2018;76(2):60–4.
12. Rehman ZU, Khan R. Acute Mesenteric Ischaemia: Case report, technical considerations and innovative approaches. J Pak Med Assoc. 2019;69(10):1557–8.
13. Janež J, Klein J. Multidisciplinary diagnostic and therapeutic approach to acute mesenteric ischaemia: A case report with literature review. SAGE Open Med Case Reports. 2021;9.
14. Berard X, Brizzi V. Acute Mesenteric Ischaemia: The Importance of Knowing When, Where, and What To Do. Eur J Vasc Endovasc Surg [Internet]. 2019;57(6):850. Available from: https://doi.org/10.1016/j.ejvs.2019.02.006
15. Sakamoto T, Kubota T, Funakoshi H, Lefor AK. Multidisciplinary management of acute mesenteric ischemia: Surgery and endovascular intervention. Vol. 13, World Journal of Gastrointestinal Surgery. 2021. p. 806–13.
16. Altintas Ü, Lawaetz M, de la Motte L, Riazi H, Lönn L, Lindh M, et al. Endovascular Treatment of Chronic and Acute on Chronic Mesenteric Ischaemia: Results From a National Cohort of 245 Cases. Eur J Vasc Endovasc Surg Off J Eur Soc Vasc Surg. 2021 Apr;61(4):603–11.
17. Acosta S, Salim S. Management of Acute Mesenteric Venous Thrombosis: A Systematic Review of Contemporary Studies. Scand J Surg. 2021;110(2):123–9.
18. Sulger E, Dhaliwal HS, Goyal A, Gonzalez L. Mesenteric Venous Thrombosis. In Treasure Island (FL); 2022.
19. Patel R, Waheed A, Costanza M. Chronic Mesenteric Ischemia. In Treasure Island (FL); 2022.
20. van Dijk LJD, Noord D van, de Vries AC, Kolkman JJ, Geelkerken RH, Verhagen HJM, et al. Clinical management of chronic mesenteric ischemia. United Eur Gastroenterol J. 2019;7(2):179–88.
21. Sultan SA, Acharya Y, Mustafa M, Hynes N. Two Decades of Experience With Chronic Mesenteric Ischaemia and Median Arcuate Ligament Syndrome in a Tertiary Referral Centre: A Parallel Longitudinal Comparative Study. Cureus. 2021;13 (December 2002).
22. Terlouw LG, van Noord D, van Walsum T, van Dijk LJD, Moelker A, Bruno MJ. Early risk stratification of patients with suspected chronic mesenteric ischaemia using a symptom and mesenteric artery calcium score based score chart. United Eur Gastroenterol J. 2021;9(5):626–34.
23. Miklosh B. Diagnostic and therapeutic approaches in chronic mesenteric ischemia have improved. United Eur Gastroenterol J. 2020;8(4):369–70.