American Journal of Emergency Medicine 65 (2023) 154–161

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

High risk and low prevalence diseases: Mesenteric ischemia

Kevin Molyneux, MD [a], Jennifer Beck-Esmay, MD [b], Alex Koyfman, MD [c], Brit Long, MD [d,*]

[a] Department of Emergency Medicine, Columbia University Irving Medical Center, 622 W 168th St, New York, NY 10032, USA
[b] Department of Emergency Medicine, Mount Sinai Morningside - Mount Sinai West, 1111 Amsterdam Ave, New York, NY 10025, USA
[c] Department of Emergency Medicine, UT Southwestern, Dallas, TX, USA
[d] SAUSHEC, Department of Emergency Medicine, Brooke Army Medical Center, Fort Sam Houston, TX, USA

ARTICLE INFO

Article history:
Received 24 September 2022
Received in revised form 19 December 2022
Accepted 2 January 2023

Keywords:
Mesenteric ischemia
Vascular
Gastroenterology
Gastrointestinal

ABSTRACT

Introduction: Mesenteric ischemia is a rare, frequently misdiagnosed, serious condition that carries with it a high rate of morbidity and mortality.
Objective: This review highlights the pearls and pitfalls of mesenteric ischemia, including presentation, diagnosis, and management in the emergency department (ED) based on current evidence.
Discussion: Mesenteric ischemia is an abdominal vascular emergency that includes superior mesenteric arterial embolism, arterial thrombosis, venous mesenteric ischemia, and non-occlusive mesenteric ischemia. It is associated with a variety of risk factors including older age, cardiovascular disease, hypercoagulable state, and end-stage renal disease. The presentation depends on the underlying pathophysiology. While arterial embolic disease may present with sudden, severe pain, the early stages of the disease and other forms can present with vague symptoms, including generalized abdominal pain, weight loss, vomiting, and diarrhea. Laboratory testing can suggest the disease with leukocytosis and elevated lactate, but normal values should not be used to exclude the diagnosis. The imaging modality of choice is triple phase computed tomography with non-contrast, arterial, and delayed phases. The initial ED management includes fluid resuscitation, symptomatic therapy, broad-spectrum antibiotics, and anticoagulation. Emergent consultation with a multidisciplinary team including diagnostic and interventional radiologists and cardiovascular and general surgeons is necessary for definitive treatment.
Conclusions: An understanding of mesenteric ischemia can assist emergency clinicians in diagnosing and managing this disease.

Published by Elsevier Inc.

1. Introduction

This article series addresses high risk and low prevalence diseases that are encountered in the emergency department (ED). Much of the primary literature evaluating these conditions is not emergency medicine focused. By their very nature, many of these disease states and clinical presentations have little useful evidence available to guide the emergency physician in diagnosis and management. The format of each article defines the disease or clinical presentation to be reviewed, provides an overview of the extent of what we currently know and understand, and finally discusses pearls and pitfalls using a question-and-answer format. This article will discuss mesenteric ischemia. This condition's low prevalence but high morbidity and mortality, as well as its variable atypical patient presentations and challenging diagnosis, makes it a high risk and low prevalence disease.

1.1. Definition

Mesenteric ischemia is a deadly intra-abdominal vascular condition which encompasses several different entities. The condition can be classified as arterial versus venous and as occlusive or non-occlusive [1]. Acute mesenteric ischemia comprises superior mesenteric arterial embolism, arterial thrombosis, and non-occlusive mesenteric ischemia (NOMI), while venous mesenteric ischemia is the result of mesenteric vein thrombosis [1]. An embolic cause accounts for 40–50% of cases, with arterial thrombosis accounting for approximately 25–30% of cases, non-occlusive mesenteric ischemia 20%, and mesenteric venous thrombosis 5–15% [2-4]. Rare causes include dissection, trauma, fibromuscular dysplasia, retroperitoneal fibrosis, and vasculitis [5].

1.2. Anatomy and pathophysiology

The anatomy of the intestinal system affects the underlying pathophysiology of mesenteric ischemia. The abdominal aorta branches into

* Corresponding author at: 3841 Roger Brooke Dr, Fort Sam Houston, TX 78234, USA.
E-mail addresses: Jennifer.beck-esmay@mountsinai.org (J. Beck-Esmay), Brit.long@yahoo.com (B. Long).

https://doi.org/10.1016/j.ajem.2023.01.001
0735-6757/Published by Elsevier Inc.

the celiac artery (CA), superior mesenteric artery (SMA), and inferior mesenteric artery (IMA) [6]. The CA perfuses the distal esophagus to the second part of the duodenum, the SMA the duodenum to distal transverse colon, and the IMA the transverse colon to rectum [6]. These primary vessels are interconnected with collateral networks [7]. The splanchnic and intestinal system receives 15% to 35% of cardiac output [4,6]. The mesenteric circulation has high resistance with low oxygen extraction and poor intestinal autoregulation of oxygen availability, which places it at risk for ischemia [3]. The superior mesenteric artery has a 45-degree take-off from the aorta and is affected in 85% of cases of acute mesenteric ischemia [8]. An embolism results in sudden occlusion, while thrombotic disease is associated with plaque deposition at the superior mesenteric artery origin, resulting in stenosis [9]. Plaque rupture and platelet aggregation result in sudden occlusion. Acute mesenteric thrombosis typically has a larger extent of ischemic bowel compared to acute arterial embolism, as the occlusion occurs at the origin of the SMA, whereas in embolic mesenteric ischemia the blockage is most commonly in the SMA distal to the origin of the middle colic artery [4,10]. Thus, thrombosis can lead to a more extensive area of necrotic bowel when not rapidly diagnosed and treated. Collateral circulation typically develops where there is >70% stenosis of the mesenteric arteries [11]. Non-occlusive ischemia occurs in the setting of low flow states including sepsis, hypovolemia/dehydration, cardiogenic shock, and other conditions [12]. Mesenteric venous thrombosis is due to reduced venous outflow and visceral edema. It is associated with thrombophilia; trauma; or inflammatory conditions such as diverticulitis, pancreatitis, and biliary system inflammation [3,13]. No matter the specific etiology, once acute occlusion occurs, the initial response is vasodilation within the mesenteric circulation, but vasoconstriction occurs with prolonged ischemia [14]. Ischemia first affects the mucosa, as it is furthest from the vascular supply, followed by the muscularis and serosal layers [15]. This damages the integrity of the mucosal and submucosal layers, allowing translocation of bacteria. Systemic inflammatory pathways are activated which further worsen vasospasm and contribute to ischemia [14,16]. As ischemia worsens, necrosis may occur with extensive injury to the intestinal system, followed by death [17].

1.3. Epidemiology

The prevalence of acute mesenteric ischemia approximates 0.1%, and the incidence ranges between 5.3 and 8.4 cases per 100,000 per year, with risk increasing with advancing age [18]. Approximately two-thirds of cases are due to thromboembolic occlusive mesenteric ischemia, one-sixth due to NOMI, and one-sixth due to mesenteric venous thrombosis [18]. Mesenteric ischemia was found to have a greater incidence than ruptured abdominal aortic aneurysm in a longitudinal population-based Swedish study [19]. The majority of patients develop the disease in their late 60s to 70s, with a median age of 67 years [20]. Females account for over 70% of overall cases of mesenteric ischemia, but the vast majority of these are chronic. Acute mesenteric ischemia appears to affect males and females equally [3,21,22]. Risks for developing mesenteric ischemia relate to the underlying pathophysiology. The major risk factors include older age, coronary or peripheral arterial disease, hypercoagulable state (i.e., cancer, liver disease, deep venous thrombosis, recent surgery or hospitalization, thrombophilia, etc.), and end stage renal disease requiring hemodialysis [3]. Chronic mesenteric ischemia is more common in patients older than 60 years and is three times more common in women [23,24]. Prior to the availability of current therapies, mortality rates for mesenteric ischemia were well above 50%, but this has decreased in more recent studies [25]. For all causes of acute mesenteric ischemia, studies conducted prior to 2000 indicate a mortality rate of 68.7%, while studies after 2000 indicate a mortality rate of 55.0% [26]. Patients who undergo operative therapy within 24 h of diagnosis have a mortality rate of 10.6%, but for those who undergo intervention after 24 h, mortality is over 72% [25]. In those over the age of 84 years, 7% of patients survive at 30 days, while in those <71 years, over 81% survive at 30 days [25]. Occlusive arterial acute mesenteric ischemia carries a short-term mortality rate of 51.8%, compared to 58.4% for NOMI and 24.6% for mesenteric venous thrombosis [26].

Due to the four different causes, patients can present in a variety of ways, which may lead to misdiagnosis. A large population-based Swedish study indicated that intestinal ischemia was only suspected in 33% of autopsy-confirmed cases of acute thrombo-embolic occlusion of the superior mesenteric artery [27]. In a separate study, acute mesenteric ischemia was clinically suspected in only 22% of patients who died of the disease [1]. Unfortunately, if misdiagnosis occurs, mortality drastically increases [28]. Mesenteric venous thrombosis poses a particular challenge given its often insidious onset and symptom variability; it constitutes between 6 and 9% of total mesenteric ischemia cases, with a mortality rate approximating 25% [26,29]. Furthermore, mesenteric venous thrombosis affects a younger patient population, between 40 and 60 years old, compared to other types of mesenteric ischemia, with males slightly more commonly affected [29].

2. Discussion

2.1. Presentation

The initial presentation of acute mesenteric ischemia varies, ranging from abdominal discomfort, pain, and bloating to hemodynamic compromise and death [3,30]. The “typical” presentation is an elderly female, with multiple comorbidities and abdominal pain out of proportion to examination, though this varies based on the cause of acute mesenteric ischemia [30]. Acute disease can present with sudden, severe abdominal pain, accompanied by nausea, vomiting, and occasionally hematochezia [31]. Chronic ischemia often presents with pain after eating (typically 30–60 min), nausea or vomiting, and weight loss due to sitophobia, or food fear [3]. Patients with chronic mesenteric ischemia may have undergone an extensive, unrevealing gastrointestinal evaluation prior to their presentation to the ED [3,30,32]. Small bowel ischemia initially presents with crampy periumbilical pain without reproducible tenderness until transmural ischemia develops, eventually deteriorating into intestinal necrosis with associated peritonitis, abdominal rigidity, and sepsis [1]. Patients may present with hematochezia in later stages of ischemia [32].

Mesenteric venous thrombosis can overlap between both acute and chronic mesenteric ischemia in its presentation. Given that it is due to venous thrombosis, it can present chronically with similar symptoms as chronic mesenteric ischemia and occasionally findings of portal hypertension, subacutely with increasing pain and symptoms over days to weeks, or acutely with a similar presentation as embolic disease [33]. Approximately 75% of mesenteric venous thrombosis patients are symptomatic for >48 h before their presentation, with a mean duration ranging between 6 and 14 days [33].

2.2. ED evaluation

Considering this diagnosis is integral in the ED evaluation of the patient with acute mesenteric ischemia. Laboratory testing including complete blood cell count, electrolytes, renal and liver function, lipase, lactate, venous blood gas, and electrocardiogram (ECG) are recommended. Patients may demonstrate elevated white blood cell count, hyperphosphatemia and hyperkalemia (electrolyte abnormalities associated with bowel infarction and cellular necrosis), elevated lactate, and metabolic acidemia [4]. Patients may also demonstrate elevated creatine kinase (CK) and elevated lactate dehydrogenase (LDH) [4]. However, there is no readily available laboratory test that is both sensitive and specific for acute mesenteric ischemia [4]. Imaging is recommended, but plain radiographs are not sensitive or specific for diagnosis [4]. Computed tomography angiography (CTA) with intravenous (IV)

Table 1
Antibiotic regimens for acute mesenteric ischemia [9,42-46].

Antibiotic Regimens
Ceftriaxone, cefepime, ceftazidime, ciprofloxacin, OR levofloxacin PLUS metronidazole –OR– Ertapenem, imipenem-cilastatin, meropenem, OR piperacillin-tazobactam
Vancomycin can be added if patient has MRSA risk factors

contrast should be obtained when the diagnosis is suspected (Figures 1 and 2) [4]. CTA improves detection of acute mesenteric ischemia and is associated with reduced mortality [34]. CTA should be performed as soon as possible for any patient with suspicion for acute mesenteric ischemia, even in the setting of renal failure, as the consequences of delayed or missed diagnosis are far more detrimental to the kidneys than contrast [9]. Duplex ultrasound has been evaluated and may detect stenosis of the celiac and superior mesenteric artery origin, but there are limitations associated with this test including operator dependency and anatomical difficulties [35-39].

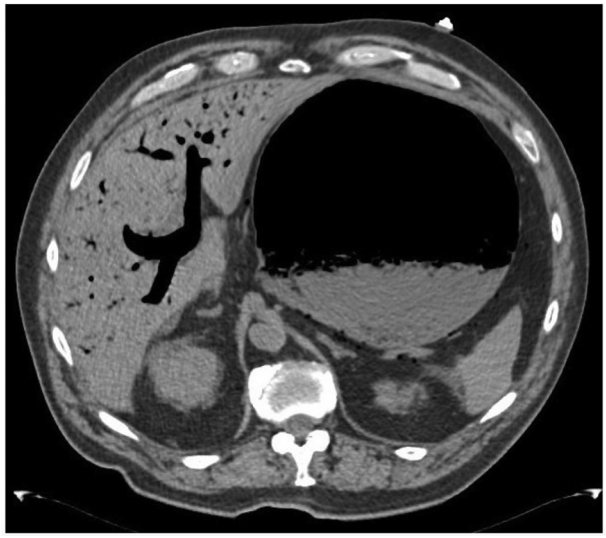

Fig. 2. CT demonstrating portal and mesenteric venous gas due to bowel ischemia [99].

2.3. ED management

The primary goals of ED management include resuscitation, symptomatic therapy, anticoagulation, administration of broad-spectrum antibiotics, and specialist consultation for definitive treatment. Aggressive fluid resuscitation is recommended due to hemodynamic compromise, volume loss, and third spacing, which may require over 10 L of IV fluid within the first 24 h of management [4]. However, volume overload should be avoided [9]. Vasopressors should also be avoided if possible and used as a last resort for patients with refractory hypotension, as they can induce intestinal vasospasm [4,9]. Symptomatic therapy including antiemetics and analgesics should be provided. Anticoagulation with unfractionated heparin should be initiated as soon as possible in patients with acute mesenteric ischemia to reduce further clot formation and propagation, but this should not delay invasive revascularization if the patient is a candidate [3,11,32]. Patients with mesenteric venous thrombosis are typically treated with anticoagulation alone for recanalization of the thrombosed vasculature and often do not require further invasive procedures [33].

Broad-spectrum antibiotics are recommended in the ED, as the loss of mucosal integrity leads to bacterial translocation out of the intestines, and antibiotic administration is associated with decreased mortality [40-42]. Given the rarity of the disease, there is a dearth of high-quality data regarding antibiotic choice for acute mesenteric ischemia specifically, and antibiotic choices are extrapolated from other intra-abdominal infectious and ischemic processes. Antibiotic choice includes most commonly metronidazole and a third-generation cephalosporin, but any broad-spectrum antibiotic regimen covering common gut bacteria is efficacious (Table 1) [32,42-46]. (See Figs. 1 and 2.)

The definitive treatment focuses on restoration of intestinal blood flow as soon as possible [47]. Early surgical and interventional radiology consultation, even before CT acquisition, is recommended if the disease is likely based on the initial clinical assessment [47]. Early surgical consultation is associated with decreased mortality, including in patients managed non-operatively, while delays in surgical consultation >24 h after disease onset and delays in operation >6 h after surgical consultation are associated with increased mortality [47]. Management of acute mesenteric ischemia depends on the underlying etiology, including arterial embolectomy, arterial bypass or stenting, arterial or venous thrombolysis, and vasodilator infusion [2]. However, patients who

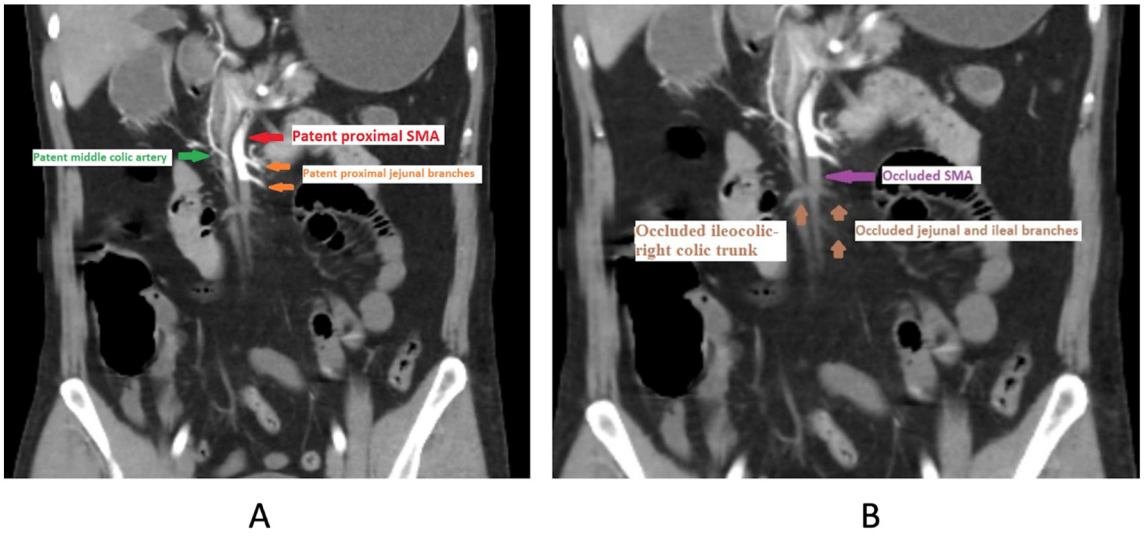

Fig. 1. CTA demonstrating occlusion of the SMA, diagnostic of acute mesenteric ischemia [98].

display evidence of peritonitis require emergent operative intervention [3,48]. Between 35 and 79% can experience severe complications including sepsis, multiorgan dysfunction and failure, acute respiratory distress syndrome, disseminated intravascular coagulation, and short gut syndrome, and thus patients should be admitted to an intensive care setting [49,50].

3. Pearls and pitfalls

3.1. What are risk factors for mesenteric ischemia that emergency clinicians should consider?

There are a variety of risk factors associated with acute mesenteric ischemia. Consideration of these risk factors in the initial clinical assessment is key in diagnosis. Table 2 lists these risk factors [3,32,51-53]. Acute embolic occlusion is the most common type of acute mesenteric ischemia. This form is commonly associated with atrial fibrillation, with concomitant emboli to other arteries. Among patients with acute mesenteric ischemia, 18% have a prior history of atrial fibrillation [52]. However, atrial fibrillation accounts for 50% of cases of embolic occlusion, with an increased incidence in patients not anticoagulated [32]. Furthermore, approximately one-third of patients with embolic acute mesenteric ischemia have a history of prior embolic event [53]. Acute mesenteric artery thrombosis accounts for 25–30% of presentations and is almost always associated with severe atherosclerotic disease; 50–70% of patients have had prior symptoms consistent with chronic mesenteric ischemia [4]. Risk factors for mesenteric venous thrombosis include prior thrombosis and any hypercoagulable state, while risk factors for nonocclusive mesenteric ischemia include any shock or hypovolemic state [3,52-56].

3.2. How do the different types of mesenteric ischemia affect presentation?

The presentation depends on the underlying pathophysiology and type of acute mesenteric ischemia. The classic "pain out of proportion" presentation may be absent in 20–25% of cases and occurs in the early phase, before transmural bowel necrosis has occurred [3,11]. Sudden onset pain is more common with acute arterial embolism due to the sudden obstruction of blood flow [3,11]. Reproducible tenderness to palpation is typically not seen until there is transmural bowel involvement causing local peritoneal irritation [3]. However, literature suggests up to 20% of patients have little to no pain initially, and 30% may have symptoms such as diarrhea and vomiting [9,45]. Thus, mesenteric ischemia should not be ruled out because the patient has symptoms such as vomiting and diarrhea. Bloody stools occur late and are found in 16% of patients [32].

Mesenteric thrombotic occlusive disease has a more varied presentation, ranging from vague abdominal pain and vomiting to acute onset severe abdominal pain; there will frequently be a prodrome of food aversion, nausea and vomiting, weight loss, and pain indicative of chronic mesenteric ischemia in these patients [11]. Weight loss may occur in up to 80% of patients who develop arterial thrombosis and is typically the result of postprandial pain leading to food fear and smaller meals [44]. Sudden pain may occur with acute occlusion [3,11].

Acute mesenteric ischemia may be heralded by antecedent symptoms consistent with chronic mesenteric ischemia. In patients with chronic mesenteric ischemia, there may be a history of postprandial symptoms (since blood flow to the intestine increases from 20% when fasting to 35% after eating), but most develop collateral circulation which prevents ischemic symptoms [57].

Patients with NOMI are typically critically ill and may have another concomitant condition causing end-organ hypoperfusion (i.e., cardiogenic shock, sepsis, severe dehydration/volume loss, etc.). These patients can present with hypotension, tachycardia, severe abdominal pain, vomiting, diarrhea, and altered mental status [3].

Table 2
Risk factors associated with AMI [32,52,54-56].

- Atherosclerosis
- Cardiovascular disease (atrial fibrillation/flutter, systolic heart failure, myocardial infarction, valvular disease)
- End stage renal disease on hemodialysis
- Endovascular aortic repair
- Hypercoagulable state (prior venous thromboembolism, chronic inflammatory disease, cancer, chronic liver disease, oral contraceptives, pregnancy, etc.)
- Older age
- Peripheral arterial disease
- Recent hospitalization/surgery
- Severe volume loss (diarrhea, sepsis, diuretic use)
- Shock or critical illness
- Tobacco use

3.3. What are deadly diseases that may present as a gastroenteritis mimic?

A wide range of conditions can present with similar symptoms [58]. Acute mesenteric ischemia is commonly misdiagnosed as gastroenteritis due to emptying of the ischemic bowel, leading to vomiting and diarrhea [44]. Acute severe abdominal pain is more likely to be associated with emergent pathology, including ruptured AAA, acute mesenteric ischemia, or perforated viscus, while insidious pain is more typical of developing inflammatory or infectious diseases like cholecystitis, appendicitis, or bowel obstruction [59]. The emergency clinician must utilize history and examination to guide further testing in the evaluation for these conditions. Table 3 lists several dangerous mimics.

3.4. How can laboratory markers assist in the ED, and what mistakes can occur with laboratory assessment?

There is no sensitive and specific laboratory test for acute mesenteric ischemia, and laboratory assessment should not be used to definitively rule in or rule out the disease. Leukocytosis is present in approximately 80–90% of patients [50]. Patients may demonstrate elevated CK and LDH, as well as metabolic acidemia [10]. D-dimer has a sensitivity of 96% and specificity of 44%, with a negative likelihood ratio of 0.12, while lactate is 86% sensitive and 44% specific [20,85]. Lactate does appear to have some utility in identifying patients who will likely have a poor outcome, as elevated lactate on admission and at 24 h after admission are both associated with increased mortality [86]. An elevated lactate >2 mmol/L is associated with irreversible intestinal ischemia [9]. However, lactate may not be elevated early in the course of the disease [20,85]. Urine intestinal fatty acid-binding protein was shown to be 92% sensitive and 80% specific for acute mesenteric ischemia in a small study, but this is not available in most EDs and has not been widely studied in acute mesenteric ischemia [87,88].

3.5. What imaging should be utilized for diagnosis?

For patients with high likelihood of acute mesenteric ischemia based on history and examination who display evidence of peritonitis or perforation, emergent consultation prior to imaging is recommended, as these patients require operative intervention, and early laparoscopy is associated with improved mortality [3,47,89,90]. Triphasic multidetector computed tomography (MDCT) (i.e., non-contrast and arterial and venous phases with parenteral contrast) should be obtained to evaluate for embolus or thrombosis in the mesenteric arteries and veins, and literature suggests a sensitivity over 94% and specificity over 96% [9,20,91-93]. Unenhanced CT can identify vascular calcification, intravascular clotting, and intramural hemorrhage, while contrast-enhanced CT can identify vascular occlusions, abnormal enhancement of the bowel wall, and the presence of embolism or infarction of other organs [94]. The venous phase is important for diagnosis

Table 3
Mimics of mesenteric ischemia [60-84].

Condition	Consideration
Appendicitis	• Presents with fever, migratory abdominal pain (from periumbilical to right lower quadrant), nausea/vomiting, anorexia.
Bowel perforation	• Associated with inflammatory diseases (such as Crohn's disease or ulcerative colitis), malignancy, immunocompromised, or obstruction. • Severe abdominal pain, nausea, vomiting.
Ischemic colitis	• Most commonly seen in females over age 65 with IBS or COPD, or in young patients with hypercoagulable state. • Abdominal pain, bloody stools/diarrhea.
Clostridioides difficile colitis	• Associated with recent antibiotic use, persistent diarrhea, and health care settings (such as nursing homes). • Diffuse diarrhea; may have abdominal pain.
Abdominal aortic aneurysm	• Often asymptomatic until rupture, which has a high mortality rate. • Classic presentation is severe abdominal or flank pain, hypotension, and a pulsatile abdominal mass.
Volvulus	• Vague, intermittent symptoms until an obstruction or intestinal necrosis occurs, which has a similar presentation to AMI.
Bowel obstruction	• Associated with adhesions, hernias, and malignancy. • Classically presents with diffuse abdominal pain, distention with vomiting, and decreased bowel movement, though nearly half of elderly patients do not have vomiting or constipation.
Diverticulitis	• Patients commonly present with left lower quadrant pain; they may have fever. • Left lower quadrant pain, lack of vomiting, and CRP ≥ 50 mg/L in the ED is highly specific for diverticulitis, but lacks sensitivity
Biliary colic/cholecystitis	• Biliary colic typically consists of intermittent right upper quadrant pain worse with meals, without associated fever or leukocytosis. • When this pain is associated with fever, nausea, and vomiting, cholecystitis should be considered.
Acute coronary syndrome (ACS)	• Atypical presentations of ACS can consist of syncope/presyncope, nausea and vomiting, or painless presentation, all of which increase the mortality due to underdiagnosis.
Pelvic inflammatory disease and tubo-ovarian abscess	• Lower abdominal and cervical pain, with vaginal discharge, dysuria, fever, and vomiting.
Neutropenic enterocolitis	• Most commonly occurs in hematologic malignancies, chemotherapy patients, and immunosuppressed patients. • Patients are often toxic-appearing, with neutropenia, abdominal pain, and fever.
Inflammatory bowel disease (Crohn's and Ulcerative Colitis)	• Two medians of age of onset: between 20 and 30 years old and then a smaller peak around 50 years of age. • Symptoms are variable and include abdominal pain, diarrhea, nausea, and vomiting, occasionally with fever and chills.
Liver abscess	• More common in elderly patients with cirrhosis or diabetes. • Abdominal pain, fever, hypotension, and malaise are typical.
Ruptured ectopic pregnancy	• Most commonly occurs in reproductive aged women with vaginal bleeding and/or abdominal/pelvic pain.
Diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar syndrome (HHS)	• 40–75% of patients have nausea, vomiting, and abdominal pain, often accompanied by polydipsia, polyuria, and weight loss. • DKA is diagnosed by acidosis, elevated glucose levels, and ketosis. • HHS can be associated with encephalopathy when the sodium is >160 mmol/L or serum osmolality is >320 mmol/-kg.
Pancreatitis	• Presents with sharp epigastric pain, nausea, vomiting. • Associated with biliary strictures, gallstones, alcohol use, hypertriglyceridemia.
Ovarian torsion	• Affects all ages, but reproductive age women is the most common population affected. • Commonly presents with lower abdominal pain associated with nausea and vomiting.
Testicular torsion	• Testicular torsion primarily affects those <18 years • Typical presentation is acute, severe testicular and abdominal pain with associated nausea and vomiting, scrotal swelling, and a high riding testicle.

of mesenteric venous thrombosis, with 90% sensitivity for triphasic CT imaging. A thrombus in the vein can be seen as a focal translucency [33]. Additionally, CTA can establish alternative diagnoses. One study found CTA was 87% sensitive and 97% specific for diagnosing another condition, most commonly small-bowel obstruction, infectious colitis, pneumonia, cholecystitis, and diverticulitis [95].

Expected findings on CTA or three-phase CT (non-contrast, angiography, delayed phase) in acute mesenteric ischemia include vascular filling defects, focal mural enhancement, bowel wall thickening, fat stranding, pneumatosis intestinalis, portal and/or peritoneal free air, and ascites [96,97]. Bowel wall thickening is not present in all causes of acute mesenteric ischemia, and the wall can be thinned if there is complete arterial occlusion given the lack of any arterial flow [94]. A halo or target appearance of the bowel wall is associated with mesenteric ischemia, indicating hyperemia and hyperperfusion, seen in acute ischemia that has undergone reperfusion or in NOMI [94]. On imaging, an acute occlusion without evidence of thickening and bowel wall enhancement indicates an earlier disease process, and therefore better prognosis, than a CTA demonstrating portal venous gas, pneumoperitoneum, and loop dilation, which are signs of a more advanced disease process and herald higher mortality [11].

NOMI should be suspected if the distribution of bowel ischemia is discontinuous and there is no occlusion visible, though no imaging modality has 100% sensitivity for detecting NOMI [2,3,61]. The most commonly affected areas in NOMI are the watershed areas at the splenic flexure and rectosigmoid junction [31].

Abdominal plain radiographs may demonstrate pneumatosis, obstruction, free air, and portal venous gas in 40% of cases, but radiographs are completely normal in 25% of overall cases and should not be used to exclude the diagnosis [4,96]. Doppler ultrasound can demonstrate proximal mesenteric vessel thrombosis with high sensitivity and specificity (85–90%), though it may be limited by bowel gas, obesity, vascular calcifications, and the pain associated with pressure applied to the abdomen. It is also operator dependent and is not recommended in place of CTA [35-39,100]. Magnetic resonance angiography may be used in patients who are unable to receive CT imaging, as it is not associated with radiation and does not require contrast. However, CTA remains the preferred modality in the acute care setting, as it is more readily available, faster, and better at detecting distal arterial stenosis and NOMI than contrast-enhanced MRA [93,101]. Catheter angiography was previously considered to be the gold standard for diagnosis but is currently used for patients in whom CTA is inconclusive. It remains a potential therapeutic intervention [3]. Finally, laparoscopy is the final means of diagnosis if other imaging modalities are inconclusive or unavailable and can also be used to assess prognosis [90].

3.6. What are the key components of ED management, and which specialists should be involved?

The ED management of acute mesenteric ischemia can be divided into stable, non-peritonitic patients, and those presenting in shock and/or with peritonitis. Definitive therapy focuses on restoring intestinal blood flow, while resecting any necrotic bowel [2]. All patients require emergent resuscitation, pain control, broad-spectrum antibiotics, anticoagulation, and specialist consultation. Where possible, a multidisciplinary team of radiology, interventional radiology, cardiovascular surgery, and general surgery specialists should be engaged to determine the best procedure for each individual patient [102].

Patients may require large amounts of fluid resuscitation—up to 100 mL/kg initially and 10–20 L in the first 24 h—due to marked volume loss and third spacing which can, in turn, worsen intestinal ischemia [4,45]. However, volume overload should be avoided. All patients should be kept NPO, as oral intake can worsen intestinal ischemia by increasing the intestinal blood demand. Unless otherwise contraindicated, unfractionated heparin should be initiated in patients with acute mesenteric ischemia [3,9,89].

For any patient with acute mesenteric ischemia who presents with evidence of peritonitis, emergent laparotomy is recommended [57]. Overall, the most common causes of death within 90 days from presentation are multiorgan failure and sepsis [32].

Patients who do not demonstrate evidence of peritonitis and are otherwise hemodynamically stable may undergo other therapeutic options. Revascularization including surgical embolectomy and bypass are possible therapies; however, there is increasing use of endovascular therapies with the interventional radiology specialist [2,103-105]. Endovascular therapy is the preferred approach in patients who do not demonstrate evidence of peritonitis, as it is less invasive and decreases rates of procedural complications [2,103-105].

Patients with mesenteric venous thrombosis should initially be managed with anticoagulation including unfractionated heparin, but if they continue to deteriorate, endovascular therapies should be considered [2]. For NOMI, the primary management is treating the underlying medical condition, providing hemodynamic support, and discontinuing medications contributing to the low flow state if possible [11]. Vasodilator agents such as prostaglandin E1 and papaverine may be used for those with NOMI, which can reduce mortality from 70% to <55% [2,4].

Table 4 lists pearls of mesenteric ischemia for the emergency clinician.

Table 4
Mesenteric ischemia pearls.

- There are four types of mesenteric ischemia: mesenteric arterial embolism, arterial thrombosis, nonocclusive, and venous thrombosis.
- The underlying pathophysiology affects the presentation. Acute disease can present with sudden, severe abdominal pain, accompanied by nausea, vomiting, and diarrhea, while chronic ischemia often presents with pain after eating and weight loss due to food fear. Hematochezia may occur in later stages.
- Pain out of proportion may not always be present, and the emergency clinician must consider this diagnosis, especially in older patients with risk factors.
- Risk factors include older age, cardiac disease, atherosclerosis, hypercoagulable state, endovascular aortic repair, renal disease requiring dialysis, and conditions associated with significant volume loss or shock state.
- There is no readily available laboratory test that is both sensitive and specific for mesenteric ischemia. Lactate elevation typically occurs once there is irreversible bowel necrosis but cannot be used to exclude the diagnosis. WBC and D-dimer may be elevated.
- Triphasic MDCTA is the most reliable imaging modality for AMI.
- CTA findings include vascular filling defects, focal mural enhancement, bowel wall thickening, fat stranding, intra-abdominal air, and ascites.
- Early surgical consultation is associated with reduced morbidity and mortality.
- Initial medical management should focus on fluid resuscitation and hemodynamic support, pain control, broad-spectrum antibiotics, and anticoagulation unless there is a contraindication.
- Definitive therapy focuses on restoring intestinal blood flow, while resecting any necrotic bowel. Patients with evidence of peritonitis require emergent operative intervention.
- Therapies for arterial mesenteric ischemia include laparotomy with surgical embolectomy and bypass as well as endovascular therapies. Anticoagulation is recommended for those with venous thrombosis, while hemodynamic support and treatment of the underlying cause are recommended in those with the nonocclusive form.

4. Conclusion

Acute mesenteric ischemia is a rare disease associated with high morbidity and mortality. It is a time-sensitive diagnosis, as rapid deterioration can ensue if there is a delay to identification and treatment. There are several types of acute mesenteric ischemia, including mesenteric arterial embolism, arterial thrombosis, nonocclusive, and venous thrombosis. The presentation varies based on the underlying pathophysiology, and the classic presentation of sudden onset abdominal pain out of proportion to examination is not always present. Other types of the disease may present more insidiously with generalized abdominal pain, vomiting, and diarrhea. Consideration of risk factors is integral to diagnosis, including older age, cardiovascular disease, hypercoagulable states, end stage renal disease, and low flow state. Laboratory analysis cannot be used to exclude the diagnosis. Triple phase CT with non-contrast, arterial, and delayed phases is the imaging modality of choice for acute mesenteric ischemia and additional intra-abdominal pathology. The initial medical management includes fluid resuscitation, symptomatic therapy, broad-spectrum antibiotics, and anticoagulation if no contraindications exist. Emergent consultation with a multidisciplinary team including diagnostic and interventional radiologists and cardiovascular and general surgeons is imperative in obtaining definitive care. Ultimately, even in the best of circumstances, acute mesenteric ischemia can be a devastating disease, but with a high index of suspicion, rapid identification, medical stabilization, and rapid access to definitive care, patient outcomes can be optimized.

CRediT authorship contribution statement

Kevin Molyneux: Writing – review & editing, Writing – original draft, Visualization, Validation, Conceptualization. **Jennifer Beck-Esmay:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision. **Alex Koyfman:** Writing – review & editing, Visualization, Validation, Supervision. **Brit Long:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Conceptualization.

Declaration of Competing Interest

None.

Acknowledgements

KM, JBE, BL, and AK conceived the idea for this manuscript and contributed substantially to the writing and editing of the review. This manuscript did not utilize any grants, and it has not been presented in abstract form. This clinical review has not been published, it is not under consideration for publication elsewhere, its publication is approved by all authors and tacitly or explicitly by the responsible authorities where the work was carried out, and that, if accepted, it will not be published elsewhere in the same form, in English or in any other language, including electronically without the written consent of the copyright-holder. This review does not reflect the views or opinions of the U.S. government, Department of Defense, U.S. Army, U.S. Air Force, or SAUSHEC EM Residency Program.

References

[1] Carver TW, Vora RS, Taneja A. Mesenteric Ischemia. Crit Care Clin. 2016;32(2):155–71.

[2] Hawthorn BR, Ratnam LA. Acute mesenteric ischaemia: imaging and intervention. Clin Radiol. 2020;75(5):398.e19–28.
[3] Clair DG, Beach JM. Mesenteric Ischemia. N Engl J Med. 2016;374(10):959–68.
[4] Oldenburg WA, Lau LL, Rodenberg TJ, et al. Acute Mesenteric Ischemia. Arch Intern Med. 2004;164(10):1054.
[5] Kim AY, Ha HK. Evaluation of suspected mesenteric ischemia. Radiol Clin. 2003.;41 (2) 327e42.
[6] Rosenblum JD, Boyle CM, Schwartz LB. The mesenteric circulation. Anatomy and physiology. Surg Clin North Am. 1997;77(2):289–306.
[7] van Petersen AS, Kolkman JJ, Meerwaldt R, et al. Mesenteric stenosis, collaterals, and compensatory blood flow. J Vasc Surg. 2014;60(1):111–9.
[8] Luther B, Mamopoulos A, Lehmann C, et al. The ongoing challenge of acute mesenteric ischemia. Visc Med. 2018;34(3):217–23.
[9] Bala M, Kashuk J, Moore EE, et al. Acute mesenteric ischemia: guidelines of the world Society of Emergency Surgery. World J Emerg Surg. 2017;12(1):38.
[10] Bradbury AW, Brittenden J, McBride K, et al. Mesenteric ischaemia: a multidisciplinary approach. Br J Surg. 2005;82(11):1446–59.
[11] Gnanapandithan K, Feuerstadt P. Review article: mesenteric ischemia. Curr Gastroenterol Rep. 2020;22(4):17.
[12] Bourcier S, Klug J, Nguyen LS. Non-occlusive mesenteric ischemia: diagnostic challenges and perspectives in the era of artificial intelligence. World J Gastroenterol. 2021;27(26):4088–103.
[13] Kumar S, Sarr MG, Kamath PS. Mesenteric venous thrombosis. N Engl J Med. 2001; 345:1683–8.
[14] Boley SJ, Brandt LJ, Sammartano RJ. History of mesenteric ischemia: the evolution of a diagnosis and management. Surg Clin North Am. 1997;77:275–88.
[15] Martinez JP, Graham AC. Abdominal and gastrointestinal emergencies. An issue of emergency medicine clinics of North America. Elsevier - Health Science; 2016. p. 328.
[16] Boley SJ, Sprayregan S, Siegelman SS, Veith FJ. Initial results from an aggressive roentgenological and surgical approach to acute mesenteric ischemia. Surgery. 1977;82:848–55.
[17] Corcos O, Nuzzo A. Gastro-intestinal vascular emergencies. Best Pract Res Clin Gastroenterol. 2013;27:709–25.
[18] Kärkkäinen JM, Acosta S. Acute mesenteric ischemia (part I) - incidence, etiologies, and how to improve early diagnosis. Best Pract Res Clin Gastroenterol. 2017;31(1): 15–25.
[19] Acosta S. Epidemiology of mesenteric vascular disease: clinical implications. Semin Vasc Surg. 2010;23(1):4–8.
[20] Cudnik MT, Darbha S, Jones J, et al. The diagnosis of acute mesenteric ischemia: a systematic review and Meta-analysis. Acad Emerg Med. 2013;20(11):1087–100.
[21] White CJ. Chronic mesenteric ischemia: diagnosis and management. Prog Cardiovasc Dis. 2011;54:36–40.
[22] Schoots IG, Koffeman GI, Legemate DA, et al. Systematic review of survival after acute mesenteric ischaemia according to disease aetiology. Br J Surg. 2004;91: 17–27.
[23] Kärkkäinen JM, Lehtimäki TT, Manninen H, et al. Acute mesenteric ischemia is a more common cause than expected of acute abdomen in the elderly. J Gastrointest Surg. 2015;19(8):1407–14.
[24] Hohenwalter E. Chronic mesenteric ischemia: diagnosis and treatment. Semin Interv Radiol. 2009;26(04):345–51.
[25] Wadman M, Syk I, Elmståhl S. Survival after operations for ischaemic bowel disease. Eur J Surg. 2000;166(11):872–7.
[26] Tamme K, Reintam Blaser A, Laisaar K, et al. Incidence and outcomes of acute mesenteric ischaemia: a systematic review and meta-analysis. BMJ Open. 2022;12: e062846.
[27] Acosta S, Ögren M, Sternby N-H, et al. Incidence of acute Thrombo-embolic occlusion of the superior mesenteric artery—a population-based study. Eur J Vasc Endovasc Surg. 2004;27(2):145–50.
[28] van den Hijkant TC, Aerts BAC, Teijink JA, et al. Challenges in diagnosing mesenteric ischemia. World J Gastroenterol. 2013;19(9):1338–41.
[29] Blumberg SN, Maldonado TS. Mesenteric venous thrombosis. J Vasc Surg. 2016;4 (4):501–7.
[30] Acosta S, Block T, Björnsson S, et al. Diagnostic pitfalls at admission in patients with acute superior mesenteric artery occlusion. J Emerg Med. 2012;42(6):635–41.
[31] Snow DC, Colbenson K. Avoiding misdiagnosis of abdominal vascular catastrophes. Emerg Med Clin North Am. 2021;39(4):769–80.
[32] Park WM, Gloviczki P, Cherry KJ, et al. Contemporary management of acute mesenteric ischemia: factors associated with survival. J Vasc Surg. 2002;35(3):445–52.
[33] Singal AK, Kamath PS, Tefferi A. Mesenteric venous thrombosis. Mayo Clin Proc. 2013;88(3):285–94.
[34] Wadman M, Block T, Ekberg O, et al. Impact of MDCT with intravenous contrast on the survival in patients with acute superior mesenteric artery occlusion. Emerg Radiol. 2010;17(3):171–8.
[35] Moneta GL. Screening for mesenteric vascular insufficiency and follow-up of mesenteric artery bypass procedures. Semin Vasc Surg. 2001;14:186–92.
[36] Moneta GL, Yeager RA, Dalman R, et al. Duplex ultrasound criteria for diagnosis of splanchnic artery stenosis or occlusion. J Vasc Surg. 1991;14:511–8.
[37] Bowersox JC, Zwolak RM, Walsh DB, et al. Duplex ultrasonography in the diagnosis of celiac and mesenteric artery occlusive disease. J Vasc Surg. 1991;14:780–6.
[38] Zwolak RM. Can duplex ultrasound replace arteriography in screening for mesenteric ischemia? Semin Vasc Surg. 1999;12:252–60.
[39] AbuRahma AF, Stone PA, Srivastava M, et al. Mesenteric/celiac duplex ultrasound interpretation criteria revisited. J Vasc Surg. 2012;55(2):428–436.e6.
[40] Kozuch PL, Brandt LJ. Review article: diagnosis and management of mesenteric ischaemia with an emphasis on pharmacotherapy. Aliment Pharmacol Ther. 2005;21 (3):201–15.
[41] de Smet AM, Kluytmans JA, Cooper BS, et al. Decontamination of the digestive tract and oropharynx in ICU patients. N Engl J Med. 2009;360:20–31.
[42] Silvestri L, van Saene HKF, Zandstra DF, et al. Impact of selective decontamination of the digestive tract on multiple organ dysfunction syndrome: systematic review of randomized controlled trials. Crit Care Med. 2010;38(5):1370–6.
[43] Wong PF, Gilliam AD, Kumar S, Shenfine J, O'Dair GN, Leaper DJ. Antibiotic regimens for secondary peritonitis of gastrointestinal origin in adults. Cochrane Database Syst Rev. 2005 Apr 18;(2):CD004539. https://doi.org/10.1002/14651858.CD004539.pub2.
[44] Singh M, Long B, Koyfman A. Mesenteric Ischemia. Emerg Med Clin North Am. 2017;35(4):879–88.
[45] Wyers MC. Acute mesenteric ischemia: diagnostic approach and surgical treatment. Semin Vasc Surg. 2010;23(1):9–20.
[46] Solomkin JS, Mazuski JE, Bradley JS. Diagnosis and Management of Complicated Intra-abdominal Infection in adults and children: guidelines by the surgical infection society and the infectious disease Society of America. Clin Infect Dis. 2010; 50:133–64.
[47] Eltarawy IG, Etman YM, Zenati M, et al. Acute mesenteric ischemia: the importance of early surgical consultation. Am Surg. 2009;75(3):212–9.
[48] Acosta S, Bjorck M. Modern treatment of acute mesenteric ischaemia. Br J Surg. 2014 Jan;101(1):e100e8.
[49] Dahlke MH, Asshoff L, Popp FC, et al. Mesenteric ischemia–outcome after surgical therapy in 83 patients. Dig Surg. 2008;25:213–9.
[50] Kougias P, Lau D, El Sayed HF, et al. Determinants of mortality and treatment outcome following surgical interventions for acute mesenteric ischemia. J Vasc Surg. 2007;46:467–74.
[51] Patel A, Kaleya RN, Sammartano RJ. Pathophysiology of mesenteric ischemia. Surg Clin North Am. 1992;72(1):31–41.
[52] Zettervall SL, Lo RC, Soden PA, et al. Trends in treatment and mortality for mesenteric ischemia in the United States from 2000 to 2012. Ann Vasc Surg. 2017;42: 111–9.
[53] Martinez JP, Hogan GJ. Mesenteric Ischemia. Emerg Med Clin North Am. 2004;22 (4):909–28.
[54] Reintam Blaser A, Acosta S, Arabi YM. A clinical approach to acute mesenteric ischemia. Curr Opin Crit Care. 2021;27(2):183–92.
[55] Acosta S, Ögren M, Sternby N-H, et al. Fatal colonic ischaemia: a population-based study. Scand J Gastroenterol. 2006;41(11):1312–9.
[56] Acosta S, Ogren M, Sternby N-H, et al. Fatal nonocclusive mesenteric ischaemia: population-based incidence and risk factors. J Intern Med. 2006;259(3):305–13.
[57] Chang JB, Stein TA. Mesenteric ischemia: acute and chronic. Ann Vasc Surg. 2003; 17(3):323–8.
[58] Blutinger E., Mamtani M., Koyfman A., et al. Gastroenteritis Mimics: What should the emergency physician consider? Available at http://www.emdocs.net/gastroenteritis-mimics-emergency-physician-consider/. Published August 21, 2017. Accessed August 22, 2022.
[59] Natesan S, Lee J, Volkamer H, et al. Evidence-based medicine approach to abdominal pain. Emerg Med Clin N Am. 2016;34(2):165–90.
[60] Shogilev D, Duus N, Odom S, et al. Diagnosing appendicitis: evidence-based review of the diagnostic approach in 2014. West J Emerg Med. 2014;15(7):859–71.
[61] Pisano M, Zorcolo L, Merli C, et al. 2017 WSES guidelines on colon and rectal cancer emergencies: obstruction and perforation. World J Emerg Surg. 2018;13(1):36.
[62] Theodoropoulou A, Koutroubakis IE. Ischemic colitis: clinical practice in diagnosis and treatment. World J Gastroenterol. 2008;14(48):7302.
[63] Czepiel J, Dróżdż M, Pituch H, et al. Clostridium difficile infection: review. Eur J Clin Microbiol Infect Dis. 2019;38(7):1211–21.
[64] Sakalihasan N, Limet R, Defawe O. Abdominal aortic aneurysm. Lancet. 2005;365 (9470):1577–89.
[65] Rubano E, Mehta N, Caputo W, et al. Systematic review: emergency department bedside ultrasonography for diagnosing suspected abdominal aortic aneurysm. Acad Emerg Med. 2013;20(2):128–38.
[66] Bauman ZM, Evans CH. Volvulus. Surg Clin North Am. 2018;98(5):973–93.
[67] Spangler R, van Pham T, Khoujah D, et al. Abdominal emergencies in the geriatric patient. Int J Emerg Med. 2014;7(1):43.
[68] Rami Reddy SR, Cappell MS. A systematic review of the clinical presentation, diagnosis, and treatment of small bowel obstruction. Curr Gastroenterol Rep. 2017;19 (6):28.
[69] Laméris W, van Randen A, van Gulik TM, et al. A clinical decision rule to establish the diagnosis of acute diverticulitis at the emergency department. Dis Colon Rectum. 2010;53(6):896–904.
[70] Gallaher JR, Charles A. Acute cholecystitis. JAMA. 2022;327(10):965.
[71] Brieger D, Eagle KA, Goodman SG, et al. Acute coronary syndromes without chest pain, an underdiagnosed and undertreated high-risk group. Chest. 2004;126(2): 461–9.
[72] Lareau SM, Beigi RH. Pelvic inflammatory disease and Tubo-ovarian abscess. Infect Dis Clin North Am. 2008;22(4):693–708.
[73] Xia R, Zhang X. Neutropenic enterocolitis: a clinico-pathological review. World J Gastrointest Pathophysiol. 2019;10(3):36–41.
[74] Feuerstein JD, Cheifetz AS. Crohn disease: epidemiology, diagnosis, and management. Mayo Clin Proc. 2017;92(7):1088–103.
[75] Ordás I, Eckmann L, Talamini M, et al. Ulcerative colitis. Lancet. 2012;380(9853): 1606–19.
[76] Roediger R, Lisker-Melman M. Pyogenic and amebic infections of the liver. Gastroenterol Clin North Am. 2020;49(2):361–77.

[77] Marion LL, Meeks GR. Ectopic pregnancy. Clin Obstet Gynecol. 2012;55(2):376–86.
[78] Umpierrez G, Korytkowski M. Diabetic emergencies — ketoacidosis, hyperglycaemic hyperosmolar state and hypoglycaemia. Nat Rev Endocrinol. 2016;12(4):222–32.
[79] Bollen TL. Acute pancreatitis: international classification and nomenclature. Clin Radiol. 2016;71(2):121–33.
[80] Sasaki KJ, Miller CE. Adnexal torsion: review of the literature. J Minim Invasive Gynecol. 2014;21(2):196–202.
[81] Laher A, Ragavan S, Mehta P, et al. Testicular torsion in the emergency room: a review of detection and management strategies. Open Access Emerg Med. 2020;12:237–46.
[82] Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic Cancer. Gastroenterology. 2013;144(6):1252–61.
[83] Banerjee A. Atypical manifestations of ruptured abdominal aortic aneurysms. Postgrad Med J. 1993;69(807):6–11.
[84] Comparato G, Pilotto A, Franzè A, et al. Diverticular disease in the elderly. Dig Dis. 2007;25(2):151–9.
[85] Cohn B. Does this patient have acute mesenteric ischemia? Ann Emerg Med. 2014;64(5):533–4.
[86] Caluwaerts M, Castanares-Zapatero D, Laterre P-F, et al. Prognostic factors of acute mesenteric ischemia in ICU patients. BMC Gastroenterol. 2019;19(1):80.
[87] Salim SY, Young PY, Churchill TA, et al. Urine intestinal fatty acid-binding protein predicts acute mesenteric ischemia in patients. J Surg Res. 2017;209:258–65.
[88] Acosta S, Nilsson T. Current status on plasma biomarkers for acute mesenteric ischemia. J Thromb Thrombolysis. 2012;33(4):355–61.
[89] Sise MJ. Acute mesenteric ischemia. Surg Clin North Am. 2014;94(1):165–81.
[90] Gonenc M, Dural CA, Kocatas A, et al. The impact of early diagnostic laparoscopy on the prognosis of patients with suspected acute mesenteric ischemia. Eur J Trauma Emerg Surg. 2013;39(2):185–9.
[91] Gopee-Ramanan P, Patlas MN, Pindiprolu B, et al. Utility of biphasic multi-detector computed tomography in suspected acute mesenteric ischemia in the emergency department. Emerg Radiol. 2019;26(5):523–9.
[92] Menke J. Diagnostic accuracy of multidetector CT in acute mesenteric ischemia: systematic review and Meta-analysis. Radiology. 2010;256(1):93–101.
[93] Oliva IB, Davarpanah AH, Rybicki FJ, et al. ACR appropriateness criteria® imaging of mesenteric ischemia. Abdom Imaging. 2013;38(4):714–9.
[94] Furukawa A, Kanasaki S, Kono N, et al. CT diagnosis of acute mesenteric ischemia from various causes. AJR Am J Roentgenol. 2009;192(2):408–16.
[95] Henes FO, Pickhardt PJ, Herzyk A, et al. CT angiography in the setting of suspected acute mesenteric ischemia: prevalence of ischemic and alternative diagnoses. Abdom Radiol. 2017;42(4):1152–61.
[96] McCarthy E, Little M, Briggs J, et al. Radiology and mesenteric ischaemia. Clin Radiol. 2015;70(7):698–705.
[97] Sugi MD, Menias CO, Lubner MG, et al. CT findings of acute small-bowel entities. Radiographics. 2018;38(5):1352–69.
[98] Arterial occlusive mesenteric ischemia. Radiopaedia.org. Obtained from. https://radiopaedia.org/cases/arterial-occlusive-mesenteric-ischaemia?lang=us; 2020. Case courtesy of Dr Mohamed Saber, Radiopaedia.org, rID: 84641.
[99] Portal and mesenteric venous gas. Radiopaedia.org. Obtained from. https://radiopaedia.org/cases/portal-and-mesenteric-venous-gas?lang=us; 2008. Case courtesy of Assoc Prof Frank Gaillard, Radiopaedia.org, rID: 2654.
[100] Ginsburg M, Obara P, Lambert DL, et al. ACR appropriateness criteria® imaging of mesenteric ischemia. J Am Coll Radiol. 2018;15(11):S332–40.
[101] Hagspiel KD, Flors L, Hanley M, et al. Computed tomography angiography and magnetic resonance angiography imaging of the mesenteric vasculature. Tech Vasc Interv Radiol. 2015;18(1):2–13.
[102] Salsano G, Salsano A, Sportelli E, et al. What is the best revascularization strategy for acute occlusive arterial mesenteric ischemia: systematic review and Meta-analysis. Cardiovasc Intervent Radiol. 2018;41:27–36.
[103] Acosta S, Sonesson B, Resch T. Endovascular therapeutic approaches for acute superior mesenteric artery occlusion. Cardiovasc Intervent Radiol. 2009.;32(5) 896e905.
[104] Falkensammer J, Oldenburg WA. Surgical and medical management of mesenteric ischemia. Curr Treat Options Cardiovasc Med. 2006.;8(2) 137e43.
[105] Arthurs ZM, Titus J, Bannazadeh M, et al. A comparison of endovascular revascularization with traditional therapy for the treatment of acute mesenteric ischemia. J Vasc Surg. 2011.;53(3) 695e8.