Journal of Medical and Health Studies
ISSN: 2710-1452
DOI: 10.32996/jmhs
Journal Homepage: www.al-kindipublisher.com/index.php/jmhs

JMHS
AL-KINDI CENTER FOR RESEARCH AND DEVELOPMENT

| RESEARCH ARTICLE

Gut Warning: Acute Mesenteric Ischemia as The First Sign of Hidden Atrial Fibrillation

Bayan S. Mohamed Alalawi[1], Fatema Mohamed Shehab[2], Zakeya A. Salman[2], Sara Ebrahim Alabood[3], Fatema Mohamed Almadhoob[3], Yusuf Ahmed Almalki[4], Fatema Mohamed Alam[5], Safa Husain Eksail[6], Hawra Ali Salman[6], Zahra Ali Salman[6], Fatima Adel Abdulla[2], and Marwa Mohamed Ali[3]

[1]*Bahrain Defense Force Hospital, Riffa, Bahrain*
[2]*Eastern Health Cluster, Dammam, Saudi Arabia*
[3]*Salmaniya Medical Complex, Manama, Bahrain*
[4]*Dream Reem Medical Center, Qalali\Askar, Bahrain*
[5]*Alkawthar Medical Center, Budiya, Bahrain*
[6]*Mansoura University, Faculty of Medicine, Mansoura, Egypt*
Corresponding Author: Bayan S. Mohamed Alalawi, **Email:** bayanmohd6@gmail.com

| ABSTRACT

Some surgical abdomens may present with severe pain that appears disproportionate to physical examination findings, a red flag that clinicians must not overlook, as early mucosal ischemia can progress to transmural necrosis and eventual signs of peritonitis. Atrial fibrillation may remain clinically silent until it manifests through a first embolic event, as illustrated in this case. This report highlights the life-saving value of maintaining a low threshold for abdominal CT angiography in patients with atrial fibrillation. We present a 64-year-old male who arrived with acute, severe abdominal pain of sudden onset, persisting for approximately five hours prior to hospital admission. Laboratory findings of leukocytosis and lactic acidosis indicated acute intestinal injury and early tissue hypoxia, prompting a high index of suspicion for acute mesenteric ischemia which was later confirmed by CT angiography of the abdomen. Initial management included intravenous fluids, broad-spectrum antibiotics, rate control, analgesia, and gastrointestinal decompression, followed by definitive treatment via endovascular embolectomy. The patient demonstrated an excellent recovery, with postoperative angiography confirming successful reperfusion, and was discharged after an uncomplicated ICU course on long-term anticoagulation therapy.

| KEYWORDS

Atrial Fibrillation, Thrombosis, Embolism, Thrombo-embolic Event, Acute Mesenteric Ischemic, Acute Abdomen

| ARTICLE INFORMATION

ACCEPTED: 12 October 2025 **PUBLISHED:** 27 October 2025 **DOI:** 10.32996/jmhs.2025.6.6.1

Introduction

Acute mesenteric ischemia is a rare yet catastrophic surgical emergency stemming from sudden cessation of intestinal blood supply, which if not urgently reversed progresses to ischemia, gangrene and often fatal outcomes [1]. It comprises approximately 0.09 to 0.2 percent of emergent surgical admissions but carries a mortality rate exceeding fifty percent [1]. This entity may manifest as either an occlusive form or a non-occlusive variant. The occlusive type commonly arises from an arterial embolus accounting for roughly half of cases or from in-situ arterial thrombosis contributing to fifteen to twenty-five percent of cases, typically involving the superior mesenteric artery; mesenteric venous thrombosis represents a less frequent cause, seen in five to fifteen percent [1]. The non-occlusive form emerges when profound systemic hypoperfusion or intense vasoconstriction — as occurs in shock states, advanced heart failure or during administration of vasopressors — critically diminishes splanchnic

perfusion [2]. In typical embolic cases the occlusion lodges three to ten centimetres distal to the origin of the superior mesenteric artery, thereby often sparing the proximal jejunum and segments of colon [1]. By contrast, thrombosis tends to occur at the arterial origin in patients with longstanding atherosclerotic disease and may present against a background of weight loss or postprandial abdominal pain reflecting chronic mesenteric ischemia [1]. Venous thrombosis is relatively uncommon and is frequently precipitated by hypercoagulable states, intra-abdominal inflammation or prior surgery [3]. Non-occlusive mesenteric ischemia accounts for about twenty to twenty-five percent of cases and typically occurs in patients who are critically ill, such as those with cardiac or septic shock [2]. Patients afflicted with acute mesenteric ischemia classically complain of sudden, severe abdominal pain that appears out of proportion to the findings on physical examination [4]. The pain is generally diffuse, constant and intense, often accompanied by nausea, vomiting or diarrhoea [4]. Early in its course the abdominal wall may remain soft despite severe pain, a paradox that confounds diagnosis. As ischemia advances to infarction peritoneal signs emerge, rectal bleeding may ensue and systemic toxic features develop including tachycardia, hypotension and leukocytosis [4]. Laboratory workup is often nonspecific; serum lactate and D-dimer levels may rise yet lack specificity or sensitivity [5]. Arterial blood gas analysis frequently reveals metabolic acidosis with elevated lactate when infarction is advanced [5]. Definitive diagnosis hinges on a high clinical index of suspicion and prompt cross-sectional imaging: contrast-enhanced CT angiography is the investigation of choice [6]. CT angiography enables rapid detection of arterial occlusions or venous thrombosis, bowel wall hypoenhancement, pneumatosis intestinalis, portal venous gas or other signs of bowel infarction [6]. Delays in diagnosis exact a grave cost; each six-hour delay in CT angiography roughly doubles mortality [6]. Conventional plain radiographs and biomarkers contribute little diagnostic value, and clinicians should expedite CT angiography in any patient with suspected disease [6]. Atrial fibrillation constitutes the most common sustained cardiac arrhythmia in adults worldwide and is marked by disorganized atrial electrical activity and loss of coordinated atrial contraction [7]. In atrial fibrillation the atria quiver ineffectually and fail to propel blood into the ventricles effectively, leading to stasis, especially in the left atrial appendage, a locus prone to thrombosis [7]. Chronic atrial fibrillation engenders progressive atrial structural and functional remodeling, promoting endothelial dysfunction and hypercoagulability that render the left atrial appendage the origin of most thrombi [8]. Approximately ninety percent of cardioembolic strokes in atrial fibrillation trace back to left atrial appendage thrombi [8]. Clinically, atrial fibrillation manifests as an irregularly irregular pulse, and patients may experience palpitations, dyspnoea, fatigue or chest discomfort; yet up to one-third of episodes remain silent or only recognized after complications arise [7]. The arrhythmia confers a four- to five-fold increase in stroke risk, and about fifteen to twenty percent of all ischemic strokes are attributed to atrial fibrillation [8]. While the central nervous system is the most frequent target of thromboembolism, representing ninety to ninety-two percent of events, approximately eight to ten percent involve embolization to peripheral or visceral arteries [9]. When extracerebral emboli occur they typically involve the limbs or kidneys, but mesenteric and splenic infarctions are well documented [9]. A systematic review estimated the annual incidence of atrial fibrillation-related acute mesenteric ischemia at approximately 0.14 percent with lethality nearing seventy percent, while limb ischemia occurred at about 0.4 percent [9]. Hence, although less frequent than cerebral infarction, embolic mesenteric ischemia is a recognized and serious complication of atrial fibrillation. Management of acute mesenteric ischemia must be emergent and tailored to the underlying etiology. The initial steps include aggressive intravenous fluid resuscitation, correction of acid-base and electrolyte derangements, and commencement of broad-spectrum antibiotics to maintain perfusion and counter bacterial translocation [10]. Full-dose anticoagulation, typically in the form of intravenous heparin, is indicated in all suspected cases — particularly when embolism or venous thrombosis is suspected — unless contraindications exist [10]. In the presence of peritoneal signs or imaging evidence of infarcted bowel, urgent laparotomy is mandatory to resect nonviable intestine and attempt restoration of mesenteric flow [10]. Surgical or endovascular embolectomy may re-establish perfusion in cases of superior mesenteric artery embolus, and thrombosis may require bypass grafting in selected patients [10]. Endovascular therapies, including catheter-directed thrombolysis or stenting, are increasingly utilized particularly in embolic occlusions and have in some series yielded lower rates of bowel loss and mortality compared with open surgery [10]. After revascularization, a planned second-look laparotomy at 24 to 48 hours frequently allows identification of further evolving necrosis. In non-occlusive mesenteric ischemia the primary goal is correction of the precipitating systemic insult, restoration of cardiac output, withdrawal of vasopressors and institution of anticoagulation [11]. Mesenteric venous thrombosis is managed primarily with anticoagulation unless infarction mandates surgical intervention [11]. In every scenario prompt recognition and timely restoration of intestinal perfusion significantly reduce mortality [10]. Therapeutic strategies for atrial fibrillation focus on symptom control and prevention of thromboembolism. Rate control via beta-blockers, calcium channel blockers or digoxin is often adequate in older or minimally symptomatic individuals, whereas rhythm control using antiarrhythmic agents or electrical cardioversion is pursued in younger or more symptomatic patients [12]. In cases of refractory or recurrent atrial fibrillation, catheter ablation has proven to be an efficacious means to restore sinus rhythm [12]. Anticoagulation decisions are guided by CHA_2DS_2-VASc scoring, and current practice warrants long-term oral anticoagulants for most patients with an annual stroke risk of two percent or higher [13]. Direct oral anticoagulants are preferred over warfarin in nonvalvular atrial fibrillation for their improved safety profile and ease of use [13]. For patients at high bleeding risk nonpharmacologic interventions — such as left atrial appendage occlusion — may be considered [14]. In real-world settings patients presenting with embolic phenomena often turn out to harbor occult left atrial thrombi on echocardiography, necessitating a collaborative multidisciplinary approach involving cardiology and vascular surgery. The distinctiveness of this

case lies in the fact that atrial fibrillation manifested initially as acute mesenteric ischemia rather than being known beforehand. While embolic mesenteric infarction is typically seen in patients with established arrhythmia, this scenario emphasizes the capacity of atrial fibrillation to remain clinically silent until it presents catastrophically via visceral embolism. The phenomenon of silent or "lone" atrial fibrillation is well documented and this instance underscores the importance of considering occult cardiac embolic sources when mesenteric ischemia arises without clear cause. To the best of our knowledge acute mesenteric ischemia as the inaugural presentation of previously undiagnosed atrial fibrillation is seldom reported. This report enriches the literature by showcasing a dramatic gastrointestinal herald of subclinical atrial fibrillation and reinforcing the crucial value of early diagnosis and a multidisciplinary therapeutic approach in improving outcomes in mesenteric ischemia [1][6].

Case Presentation

Patient's history and Physical Examination

This case describes a 64-year-old male who presented with a chief complaint of acute, severe abdominal pain of sudden onset, persisting for approximately five hours prior to hospital admission. The pain began abruptly while the patient was at rest in his residence and was characterized as constant, cramp-like, and severe, rated 9 out of 10 in intensity. The discomfort was unrelieved by over-the-counter analgesics and bore no apparent relation to food intake or body posture. Associated symptoms included nausea without episodes of vomiting, as well as mild, non-exertional shortness of breath. He denied any concurrent chest pain, palpitations, syncope, changes in bowel habits, melena, hematochezia, or urinary complaints such as dysuria. His past medical history was significant for poorly controlled hypertension over the past 12 years, type 2 diabetes mellitus for which he was taking metformin 1000 mg twice daily, and dyslipidemia managed with atorvastatin 20 mg daily. He had no prior surgical history, did not smoke, and reported no alcohol consumption. There was no notable family history of cardiovascular, gastrointestinal, or thromboembolic disorders. On initial examination, the patient appeared acutely unwell, visibly distressed, anxious, and diaphoretic. He was afebrile, with a heart rate of 132 beats per minute, hypotensive with a blood pressure of 96 over 62 mmHg, respiratory rate of 22 breaths per minute, and oxygen saturation of 97% on ambient air. Abdominal examination revealed a soft but diffusely tender abdomen with minimal guarding and rigidity, findings that appeared disproportionately mild in comparison to the patient's marked clinical distress. Cardiovascular, respiratory, neurological, and extremity examinations were within normal limits, with the exception of an irregularly irregular peripheral pulse, raising suspicion for an underlying arrhythmia.

Investigations

A bedside 12-lead electrocardiogram demonstrated an irregularly irregular rhythm, absence of discernible P waves, and a variable R–R interval, findings consistent with newly identified atrial fibrillation with a rapid ventricular response, recorded at a rate of 135 beats per minute. Relevant laboratory investigations are summarized in Table 1. A contrast-enhanced computed tomography angiography (CTA) of the abdomen was subsequently performed, revealing a well-defined intraluminal filling defect within the superior mesenteric artery, located just distal to its origin. This was accompanied by reduced perfusion of the small intestine, mild segmental bowel wall thickening, and the presence of pneumatosis intestinalis, indicative of ischemic insult. A transthoracic echocardiogram was also obtained, showing evidence of left atrial enlargement with preserved left ventricular ejection fraction and no visualization of intracardiac thrombi or valvular vegetations. Based on the clinical presentation and radiographic findings, a definitive diagnosis of acute mesenteric ischemia was established, attributed to arterial embolism likely secondary to the newly recognized atrial fibrillation, despite a negative transthoracic echocardiographic assessment for a visible intracardiac thromboembolic source.

Test	Result	Normal Range
Hemoglobin	14.2	12-16 g\dL
WBC	$14.2x10^9$	$4.0\text{-}11x10^9$\L
Platelets	$290x10^9$	$150\text{-}450x10^9$\L
Sodium	135	135-145 mmol\L
Potassium	4.5	3.5-5.0 mmol\L
Creatinine	1.8	0.6-1.2 mg\dL
ALT	58	<40 U\L
AST	65	<40 U\L
Troponins	0.08	<0.04 ng\mL
CRP	52	<5 mg\L
Serum Lipase	48	0-160 U\L
Serum Amylase	35	23-85 U\L
Serum Lactate	6.1	0.5-2.2 mmol\L

VBG	Mild metabolic acidosis with respiratory compensation	-

Table 1: results of relevant laboratory investigations.

Management course

The patient was initiated on supplemental oxygen at a flow rate of 4 liters per minute via nasal cannula. Simultaneously, intravenous resuscitation was commenced with 1000 milliliters of normal saline administered over the course of one hour. Empiric broad-spectrum antimicrobial coverage was provided with intravenous piperacillin-tazobactam, and analgesia was achieved through titrated doses of intravenous fentanyl. A nasogastric tube was inserted for gastrointestinal decompression. For acute rate control of atrial fibrillation, two slow intravenous boluses of metoprolol, each 5 mg, were administered. Anticoagulation was promptly initiated with an intravenous bolus of unfractionated heparin at 80 units per kilogram, followed by a continuous infusion at 18 units per kilogram per hour, targeting an activated partial thromboplastin time (aPTT) between 60 and 80 seconds. The patient was placed under continuous cardiac telemetry and urinary output monitoring. As definitive management, an emergent endovascular thromboembolectomy of the superior mesenteric artery was successfully performed within four hours of hospital presentation, with post-procedural angiography confirming restoration of mesenteric perfusion. The postoperative course was uneventful and included intensive care unit observation. The patient was transitioned to oral anticoagulation with apixaban 5 mg twice daily and continued rate control with oral metoprolol 50 mg twice daily. He remained nil per os for 24 hours, followed by cautious reintroduction of oral fluids and gradual progression to solid food. Spontaneous reversion to sinus rhythm was noted 48 hours post-intervention. Clinical improvement was marked by normalization of serum lactate levels within 36 hours. The patient was discharged home in stable condition on hospital day eight, with arrangements for cardiology follow-up and long-term anticoagulation, given a CHA_2DS_2-VASc score of 4.

Discussion

Severe abdominal pain that appears grossly disproportionate to the physical examination must always raise suspicion for a life-threatening intra-abdominal event, particularly acute mesenteric ischemia (AMI), where this discordance reflects the early phase of mucosal ischemia before transmural involvement or peritonitis sets in [1,4]. This clinical mismatch remains one of the most diagnostically valuable clues in the early stages of ischemic bowel disease, where prompt recognition can significantly alter patient outcomes [2]. As ischemia progresses rapidly, relying solely on physical findings may delay intervention and worsen prognosis. Therefore, the presence of significant distress or intractable abdominal pain—especially when coupled with systemic signs like leukocytosis or metabolic acidosis—should substantially lower the threshold for cross-sectional imaging [5]. Elevated lactate levels, though nonspecific, often serve as a surrogate marker of tissue hypoperfusion and correlate with the severity of ischemia, particularly when interpreted in conjunction with other laboratory and radiological findings [5]. Early imaging is indispensable, and contrast-enhanced computed tomography angiography (CTA) has become the diagnostic modality of choice, capable of identifying mesenteric arterial or venous occlusion, diminished bowel wall enhancement, pneumatosis intestinalis, mesenteric stranding, and portal venous gas with both high sensitivity and specificity exceeding 90% [6]. Importantly, studies show that each six-hour delay in obtaining CTA significantly increases mortality, reinforcing the urgency of early imaging in high-risk patients [6]. Epidemiologically, the etiologic spectrum of AMI includes non-occlusive causes related to low-flow states (approximately 20%), arterial thrombosis associated with atherosclerotic disease (20–30%), and embolic events, which account for 40–50% of all cases [1,2]. Among embolic causes, atrial fibrillation is the most common underlying rhythm disturbance, with thrombus formation typically occurring in the left atrial appendage due to stasis during fibrillatory activity [7–9]. The superior mesenteric artery (SMA), owing to its large caliber and nearly straight trajectory from the abdominal aorta, is particularly vulnerable to embolic insult, second only to cerebral vasculature in embolic stroke events [1,9]. While transthoracic echocardiography (TTE) is commonly employed to evaluate for cardiac embolic sources, its sensitivity in detecting thrombi or vegetations—particularly within the left atrial appendage—is limited. The absence of identifiable thrombus on TTE does not exclude a cardioembolic origin, especially in patients with newly diagnosed atrial fibrillation [8]. In such scenarios, transesophageal echocardiography (TEE) should be strongly considered, as it provides enhanced visualization of atrial structures and can identify thrombi missed by TTE [8,9]. In terms of management, immediate anticoagulation with intravenous unfractionated heparin is considered standard in suspected or confirmed AMI cases, as it helps to stabilize the embolus, prevent propagation, and reduce the risk of recurrent embolization [10]. Where feasible, endovascular techniques such as catheter-directed thromboembolectomy or localized thrombolysis have gained increasing favor due to their minimally invasive nature and favorable outcomes, particularly in the absence of intestinal necrosis or peritoneal signs [10]. Surgical exploration remains indicated in cases of bowel infarction, perforation, or generalized peritonitis, where delayed resection can be fatal [1]. Mortality in untreated embolic SMA occlusion may reach 60–80%, but early identification and reperfusion interventions can reduce this risk to below 30% [1,10]. Post-revascularization care includes fluid resuscitation, nutritional support, and close hemodynamic monitoring, with a multidisciplinary approach involving vascular surgery, intensive care, and cardiology. Long-term secondary prevention hinges on effective anticoagulation. In this case, the use of a direct oral anticoagulant (DOAC) such as apixaban was

appropriate, as DOACs have demonstrated superior safety and at least equivalent efficacy compared to warfarin in patients with non-valvular atrial fibrillation [13]. The ARISTOTLE trial, among others, has shown a 21% relative risk reduction in stroke and systemic embolism, along with reduced major bleeding risk, in patients treated with apixaban versus warfarin [13]. However, in patients with valvular atrial fibrillation—particularly those with mechanical heart valves or rheumatic mitral stenosis—warfarin remains the gold standard due to its established efficacy and extensive historical use [13,14]. The failure to perform TEE in this case highlights a common clinical limitation, as excluding a cardiac embolic source without comprehensive imaging may overlook residual thrombi or predisposing cardiac pathology. Ultimately, this case underscores the imperative to maintain a high index of suspicion for embolic causes in any patient presenting with acute abdominal symptoms and concurrent or newly detected atrial fibrillation. Abdominal pain, when sudden and severe, may not signify a primary gastrointestinal disorder, but rather an extra-abdominal vascular catastrophe. Clinicians should therefore expand their diagnostic lens to consider cardioembolic etiologies, recognizing that atrial fibrillation may first manifest through ischemic events beyond the brain. In such cases, early diagnosis, timely imaging, prompt anticoagulation, and appropriate reperfusion therapy are critical to improving survival and reducing long-term morbidity [1,4,6,10].

Conclusion

Never disregard pain that is out of proportion to physical examination findings, as it may indicate early mucosal ischemia preceding transmural necrosis. Although lactic acidosis is nonspecific, it can support the diagnosis of tissue hypoxia when interpreted within the broader clinical context. Atrial fibrillation may remain clinically silent until its first embolic event; therefore, pulse palpation is essential in patients presenting with abdominal pain. In the setting of atrial fibrillation, clinicians should maintain a low threshold for imaging and consider acute mesenteric ischemia early in the differential diagnosis, as prompt abdominal CT angiography can be life-saving.

Funding: This research received no external funding.

Conflicts of Interest: The authors declare no conflict of interest.

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References:

1. 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*. 2022;17(1):54.
2. Clair DG, Beach JM. Mesenteric Ischemia. *N Engl J Med*. 2016;374(10):959–968.
3. Acosta S. Mesenteric venous thrombosis with transmural bowel infarction: a population-based study. *J Vasc Surg*. 2010;51(1):59–63.
4. Oldenburg WA, Lau LL, Rodenberg TJ, Edmonds HJ, Burger CD. Acute mesenteric ischemia: a clinical review. *Arch Intern Med*. 2004;164(10):1054–1062.
5. van den Heijkant TC, Aerts BA, Teijink JA, Buurman WA, Luyer MD. Challenges in diagnosing mesenteric ischemia. *World J Gastroenterol*. 2013;19(9):1338–1341.
6. Menke J. Diagnostic accuracy of multidetector CT in acute mesenteric ischemia: systematic review and meta-analysis. *Radiology*. 2010;256(1):93–101.
7. Hindricks G, Potpara T, Dagres N, et al. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation. *Eur Heart J*. 2021;42(5):373–498.
8. January CT, Wann LS, Calkins H, et al. 2023 ACC/AHA/HRS Guideline for the Management of Patients With Atrial Fibrillation. *J Am Coll Cardiol*. 2023;81(13):1376–1482.
9. Balsam P, Tymińska A, Ozierański K, Grabowski M, Opolski G. Systemic embolism in atrial fibrillation: risk factors and prevention. *Kardiol Pol*. 2016;74(10):979–987.
10. Acosta S, Björck M. Modern treatment of acute mesenteric ischaemia. *Br J Surg*. 2014;101(1):e100–e108.
11. Kumar S, Sarr MG, Kamath PS. Mesenteric venous thrombosis. *N Engl J Med*. 2001;345(23):1683–1688.
12. Kirchhof P, Camm AJ, Goette A, et al. Early rhythm-control therapy in patients with atrial fibrillation. *N Engl J Med*. 2020;383(14):1305–1316.
13. Lip GYH, Banerjee A, Boriani G, et al. Antithrombotic therapy for atrial fibrillation: CHEST guideline and expert panel report. *Chest*. 2022;162(5):1120–1203.
14. Reddy VY, Doshi SK, Kar S, et al. 5-Year Outcomes After Left Atrial Appendage Closure. *J Am Coll Cardiol*. 2017;70(24):2964–2975.