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CASE REPORT

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Acute mesenteric ischemia secondary to complete AV block and acute systolic heart failure: a case report

Jian Cui[1*]

Abstract

Background Acute mesenteric ischemia (AMI) usually presents with abrupt and severe abdominal pain associated with nausea and vomiting. This case is notable due to the occurrence of AMI secondary to acute systolic heart failure caused by new onset complete heart block.

Case Presentation A 65-year-old male presented with sudden onset epigastric pain. ECG showed complete AV block and acute ischemia, and a subsequent CTA revealed complete occlusion of the mid superior mesenteric artery. His emergent left heart catheterization showed non-occlusive coronary artery disease. The patient underwent emergent laparotomy and SMA thrombectomy. Postoperative complications included worsening congestive heart failure and persistent bradycardia, requiring a permanent pacemaker. The patient was discharged to a skilled nursing facility in stable condition.

Conclusions This case highlights the diagnostic challenges of AMI in the setting of acute heart failure and new onset AV block mimicking acute cardiac events and emphasizes the importance of a multidisciplinary approach in managing such complex cases.

Keywords Acute mesenteric ischemia, Complete AV block, Acute systolic heart failure, Case report

Introduction

Acute mesenteric ischemia (AMI) usually presents with abrupt and severe abdominal pain associated with nausea and vomiting. It may be caused by an arterial embolus (40–50% of cases), an arterial thrombus (20–35%), or a venous thrombus (5–15%) [1]. In acute embolic mesenteric ischemia, the emboli typically originate from a cardiac source and frequently occur in patients with atrial fibrillation or following myocardial infarction [2]. Acute thrombosis occurs in patients with underlying mesenteric atherosclerosis, which typically involves the origin of the mesenteric arteries while sparing the collateral branches [3]. The authors describe an interesting case of AMI believed to be secondary to acute systolic heart failure caused by new onset complete heart block. The authors also discuss the difficulties in the diagnosis and management.

Case presentation

A 65-year-old male Caucasian presented to our facility with sudden onset epigastric pain in the last 4 h. Pain severity was 10/10, non-relenting, associated with nausea, vomiting and diarrhea. The patient was sweating

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profusely. He reported no chest pain, back pain or shortness of breath.

He denied previous cardiac/pulmonary medical conditions. He was not taking any medications. He had been a heavy smoker with 2 pack/day for the last 30 years and he has no history of recent alcohol use. He denies a significant family history.

His physical exam: Vital signs: T was 97.4 °F (36.3 °C); BP was 178/74, PR was 45/min, RR was 16/min and oxygen saturation was 97% room air.

He was sick-looking and sweating, in moderate acute distress. Lungs: clear to auscultation bilaterally. Heart: Bradycardic, S1 and S2 noticeable, no gallop. Abdomen: soft, minimal epigastric tenderness, no guarding or rebound pain, hyperactive bowel sounds.

Investigation and treatment

His ECG showed complete AV block with VR 48/min, LVH, and significant T wave abnormalities from inferior and lateral leads. (Fig. 1).

Point-of-care Echo showed LVEF at 25%, moderate to severe global hypokinesis, anterior wall akinesis, no left ventricular thrombosis, no right heart strain, no pericardial effusion, or B-lines. Surprisingly he did not present any symptoms or signs of acute heart failure such as shortness of breath, lung crackles or wheezing, or lower extremity edema.

Pertinent labs: Troponin 0.143 ng/ml (normal < 0.017), BNP 12,539 pg/ml (normal < 900) and lactic acid 2.1 mmol/L.

Heparin drip was started and cardiology was consulted. Decision was to take him to emergent left heart catheterization (LHC). The ED provider also considered the possibility of acute mesenteric ischemia, though patient denied history of atrial fibrillation and previous chronic abdominal pain. He was taken to CTA chest, abdomen and pelvis first before taken to cath lab.

His CTA Images were reviewed by ED physician and they clearly showed complete occlusion of mid superior mesenteric artery (Fig. 2a and d). Subsequently, a diagnosis of AMI complicated by acute congestive heart failure was made. His complete AV block may be preexisting, related to his underlying cardiomyopathy or acute due to increased vagal tone secondary to the AMI. Positive CT finding was discussed with the cardiologist, general and vascular surgeons and final decision was laparotomy and SMA thrombectomy immediately after left heart catheterization. His left heart catheterization showed non-occlusive moderate coronary artery disease and subsequent aortogram further confirmed the occluded mid superior mesenteric artery (Fig. 3). Transvenous pacer was inserted in cath lab due to his worsening bradycardia.

His CTA of abdomen and pelvis official reading revealed no abdominal aortic aneurysm dissection or occlusion, occlusion of the mid SMA, occlusion of the IMA at its origin, high-grade short segment stenoses at the left common iliac artery, proximally and distally, high-grade short segment stenosis at the left renal artery, small bowel does not demonstrate bowel wall thickening

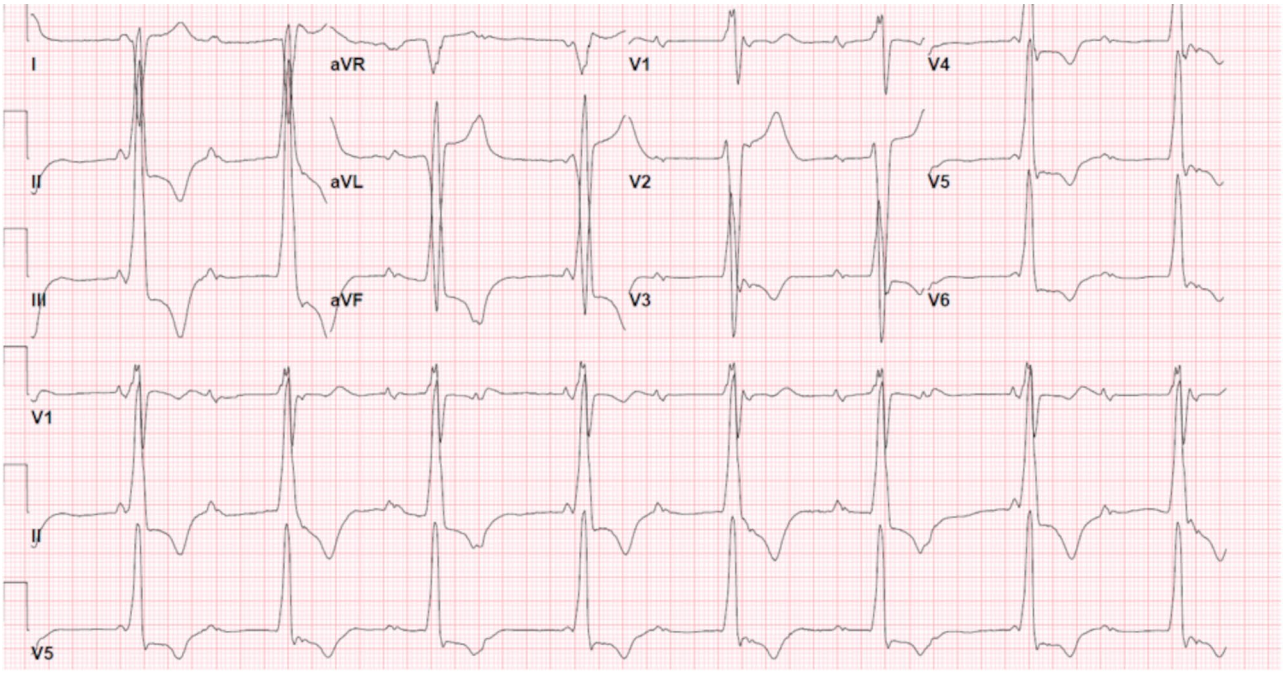
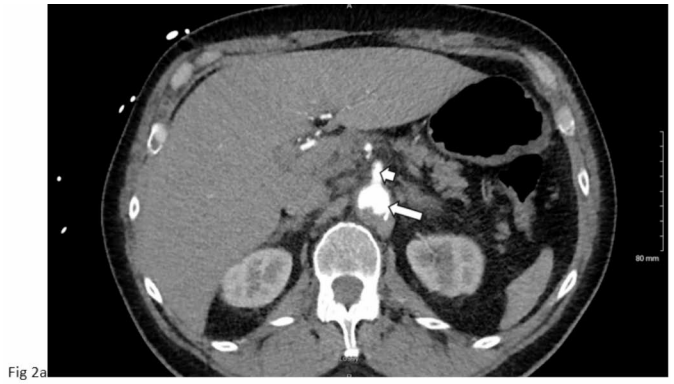
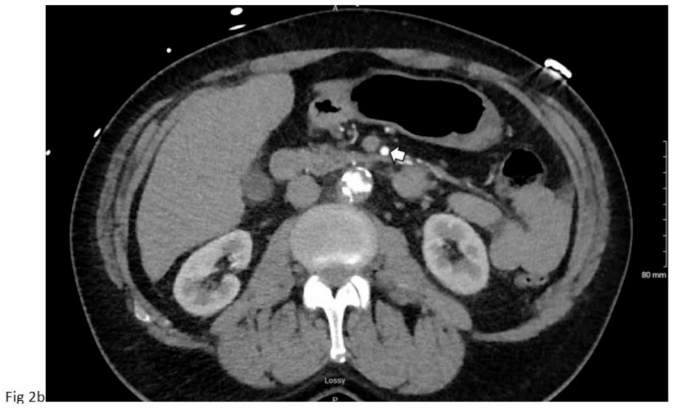
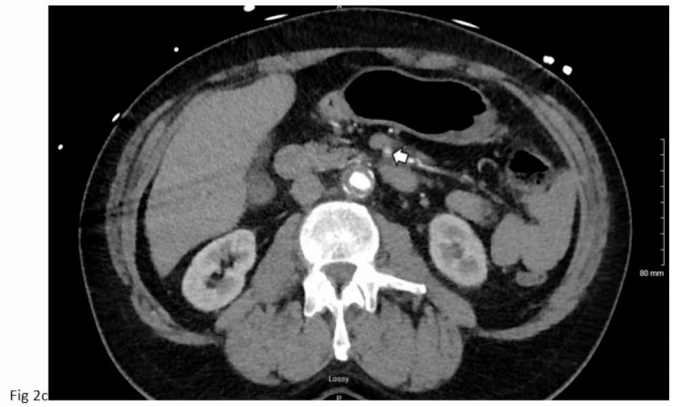


Fig. 1 EKG of complete AV block







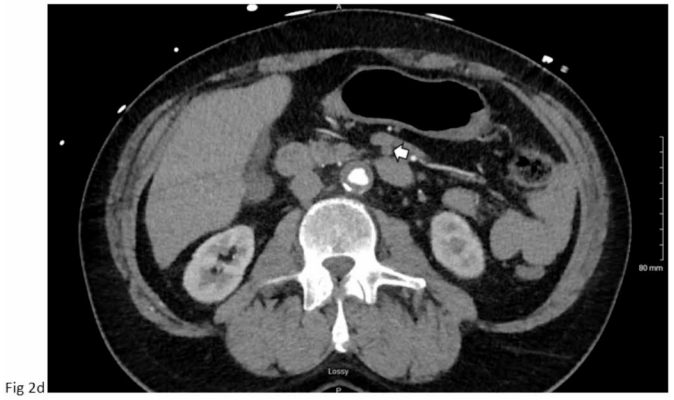


Fig. 2a Patent SMA (small arrow) arising from the abdominal aorta (large arrow)
b: Patent proximal SMA (small arrow)
c: Decreased contrast filling in mid SMA (small arrow) indicating occlusion
d: Complete absence of contrast suggesting complete occlusion of SMA (small arrow)

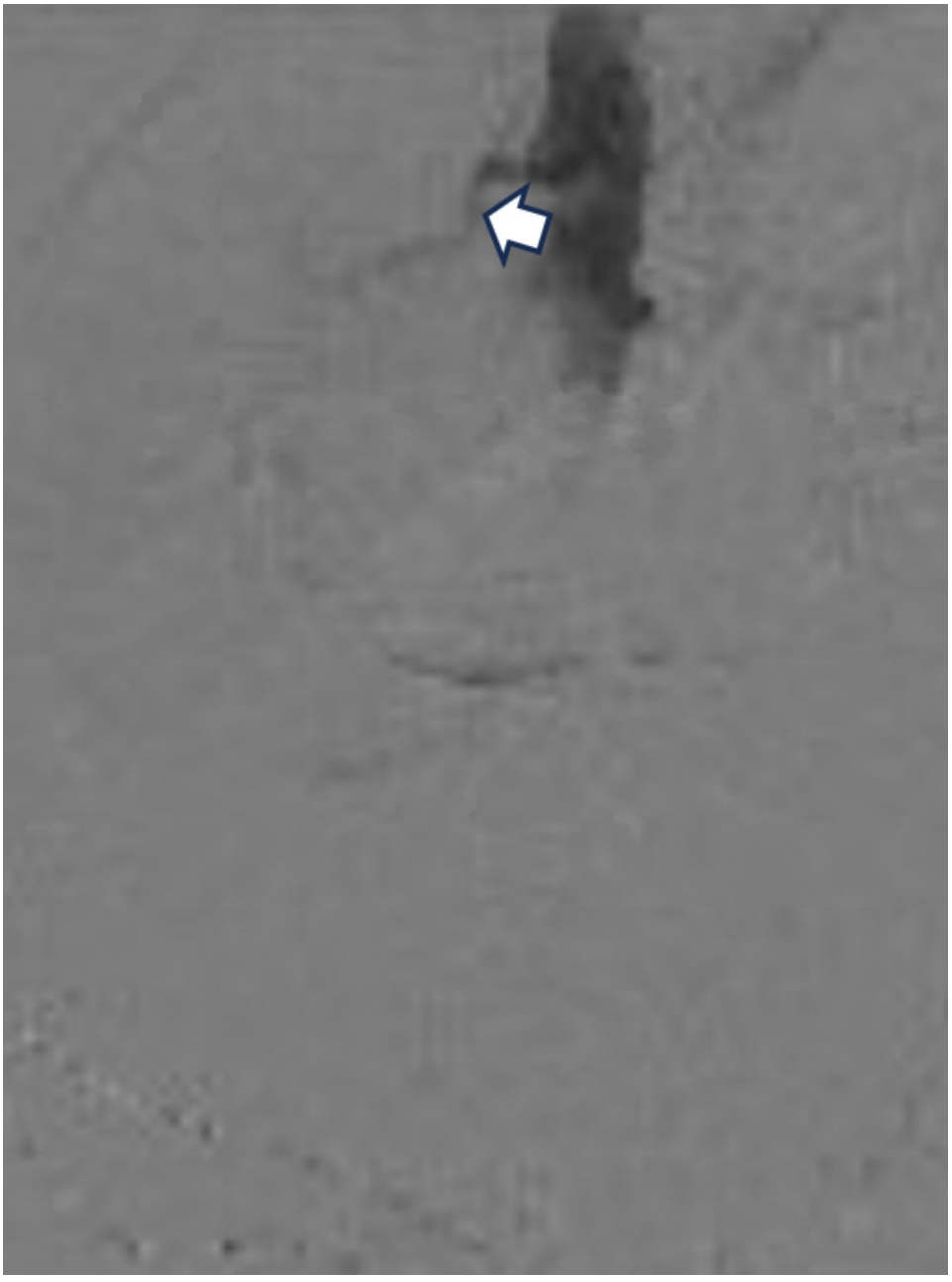


Fig. 3 Aortogram with SMA (arrow) occlusion

or pneumatosis to suggest ischemia. No mesenteric edema. No free air or free fluid.

His troponin and lactic acid levels improved slightly to 0.035 ng/mL and 1.6 mmol/L, respectively, over 2 h. HbA1c was 6.0, and his lipid panel was as follows: total cholesterol of 140 mg/dL, LDL of 97 mg/dL, HDL of 28 mg/dL, and triglycerides of 77 mg/dL.

The patient underwent exploratory laparotomy and SMA thrombectomy with multiple large blood clots retrieved. Although CTA showed significant atherosclerotic disease of aorta, no significant SMA atherosclerosis was seen during surgery. His postoperative formal Echo revealed: Left ventricle chamber size is mildly enlarged. Global hypokinesis. Wall motion abnormality: Akinesis anteriorly. Akinesis of the septum. The ejection fraction is 35% (+/- 5%) by visual estimate. Ejection fraction is moderately reduced (35–40%). Diastolic function is abnormal. The findings are consistent with ischemic cardiomyopathy. A moderate size, spherical, layered and mobile thrombus is present at the apex. It measures 1.9 cm. Mild spontaneous contrast. No mass is present. His postoperative course was complicated by worsening

congestive heart failure, left ventricular thrombus and persistent significant bradycardia requiring a permanent pacemaker. He was eventually discharged in stable conditions to a skilled nursing facility.

Discussion and conclusions

Acute mesenteric ischemia (AMI) is a relatively uncommon but potentially devastating condition that generally presents in patients over 60 years of age, having a male predominance (3:1). Acute mesenteric ischemia may be occlusive (most commonly) or nonocclusive (NOMI) [4].

Roughly, 50% of all cases of AMI are due to acute mesenteric embolism [1, 5]. Mesenteric emboli can originate from the left atrium, associated with cardiac dysrhythmias such as atrial fibrillation, the left ventricle with global myocardial dysfunction associated with poor ejection fraction, or cardiac valves due to endocarditis. Occasionally, emboli are generated from an atherosclerotic aorta. Emboli typically lodge at points of normal anatomic narrowing, and the SMA is particularly vulnerable because of its relatively large diameter and low takeoff angle from the aorta. The majority of emboli lodge 3 to 10 cm distal to the origin of the SMA, thus classically sparing the proximal jejunum and colon [6, 7].

We did not have a suspicion of AMI at first as he had no history of atrial fibrillation or coronary artery disease, which made the acute mesenteric embolism less likely. Although a heavy smoker, he reported no symptoms of chronic mesenteric ischemia such as postprandial pain, weight loss, or "food fear," suggesting acute on chronic mesenteric arterial thrombosis was also less likely. When his ECG was done, we even suspected that he may have acute pancreatitis as ischemic ECG findings have been well known to present in pancreatitis [8] given the absence of chest pain.

His abnormal ECG, however, led to an ED point-of-care Echo, which showed global dyskinesia with LVEF of 25% with regional wall motion abnormalities (RWMAs) which was confirmed by a subsequent formal echo study. His cardiomyopathy was thought to be ischemic, though his LHC showed non-occlusive moderate coronary artery disease. His complete AV block seemed to be pre-existing instead of AMI-related enhanced vagal tone and the patient eventually received a permanent pacemaker before discharge. Infiltrative cardiomyopathies, such as sarcoidosis [9] and amyloidosis [10], are also recognized causes due to their propensity to involve the conduction system, leading to AV block. Additionally, Lyme disease, a known infectious cause of myocarditis [11], can result in both cardiomyopathy and AV block, especially in endemic areas. Left ventricular non-compaction (LVNC), a rare congenital condition characterized by a spongy myocardial texture, can also present with cardiomyopathy and conduction abnormalities, including AV block [12]. These differential diagnoses should be considered in the evaluation of such patients presenting with both cardiomyopathy and AV block.

Although his ED point-of-care echo did not show left ventricle thrombosis, during SMA thrombectomy, multiple large clots were retrieved and the vascular surgeon believed the patient's AMI was caused by a thromboembolic event. Postoperative echo showed he had mural thrombosis and persistent left ventricle global dyskinesia with RWMAs, which further supported the above hypothesis. It is expected that complete atrioventricular (AV) block and cardiomyopathy can contribute to acute superior mesenteric ischemia, but the most common mechanism of such ischemia is through NOMI, acute AMI such as in this case is rare.

In such a patient with suspected AMI, significant EKG changes, systolic heart failure, and complete AV block, the clinical decision-making involves risks of both action and inaction as well as priorities of actions. Traditionally, obtaining the diagnosis of AMI set in motion the process of emergent laparotomy, arterial thrombectomy, and possible bowel resection [13], especially for patients with overt peritonitis [14]. While exploratory laparotomy and arterial thrombectomy are often the definitive treatments, particularly in cases with bowel necrosis or uncertain diagnosis, percutaneous mechanical thrombectomy has emerged as a minimally invasive option [14, 15]. This technique can be particularly beneficial in patients with high surgical risks or when rapid revascularization is necessary to restore mesenteric blood flow. However, its use is typically limited to early presentations where there is minimal bowel ischemia and no signs of perforation or peritonitis. In this patient, given the severity of ischemia and the need for definitive diagnosis and management, exploratory laparotomy remained the most appropriate approach, though percutaneous options may be considered in selected cases depending on the clinical context.

Abbreviations

AMI	Acute Mesenteric Ischemia
AV	Atrioventricular
CTA	Computed Tomography Angiography
ECG	Electrocardiogram
ED	Emergency Department
LHC	left heart catheterization
LVEF	Left Ventricular Ejection Fraction
NOMI	Nonocclusive Mesenteric Ischemia
RWMAs	Regional wall motion abnormalities
SMA	Superior Mesenteric Artery

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Author contributions

Jian Cui (JC) analyzed and interpreted the patient data regarding the acute mesenteric ischemia and heart failure. JC performed the literature review and was a major contributor in writing the manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

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Not applicable.

Consent for publication
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Authors' information
Not applicable.

Competing interests
The authors declare no competing interests.

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