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*Case Report*

# Ischemic Bowel Disease Due to Superior Mesenteric Artery Occlusion: A Case Report

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**Abstract:** Acute mesenteric infarction is a rare but emergency disease with a high mortality rate. The rapidly restoration of intestinal blood flow is the early goal of vascular intervention. However, the unspecific presentation may confuse physicians and delay a timely diagnosis. The rate of intestinal failure in survivors is still high. Here, we present the case of an 85-year-old male presenting with acute onset progressive periumbilical cramping pain with elevated D-dimer. The abdominal computed tomography (CT) revealed severe acute superior mesenteric artery occlusion. The surgical report showed a massively ischemic small intestine that was about 250 cm with 200 mL bloody ascites. We highlight that early diagnosis and timely intervention are important for improving outcomes.

**Keywords:** acute mesenteric infarction; atrial fibrillation; superior mesenteric artery; embolectomy

## 1. Introduction

Acute mesenteric infarction is a rare but emergency disease with a high mortality rate. High rates of intestinal failure were reported in a large group of survivors [1]. The complications, such as sepsis and multiple organ failure, occurred with a high incidence in delayed diagnosis patients. A multidisciplinary and multimodal management approach were proposed to increase the survival of acute mesenteric infarction patients. Vascular intervention is the early goal to restore the intestinal blood flow. However, the unspecific presentation may confuse physicians and delay timely diagnosis. The delayed or missed diagnosis may cause bowl necrosis, the progression of sepsis, and multiple organ failure. The diagnostic tool is important to shorten the time to diagnosis. The computed tomography (CT) and ultrasound reported in previous studies were effective for diagnosis. In addition, the risk of emboli also plays an important role for physicians to keep this diagnosis in mind. Here, we present the case of a 85-year-old male presenting with acute onset progressive periumbilical cramping pain with elevated D-dimer. The abdominal CT revealed severe acute superior mesenteric artery occlusion. The surgical report showed a massively ischemic small intestine that was about 250 cm with 200 mL bloody ascites. This paper discusses the clinical and image features of acute superior mesenteric artery occlusion and highlights the importance of early diagnosis and timely intervention for improving outcomes.

## 2. Case Presentation Section

An 85-year-old male presented with acute onset progressive periumbilical cramping pain for four hours. There was no history of trauma. His past medical history was hypertension and atrial

fibrillation. The pain localized in the periumbilical area without a shift to the back or right lower abdomen. The pain was noted after the ingestion of food and accompanied with cold sweating, nausea, and vomiting. It could not be resolved by taking a painkiller, such as Diclofenac or Tramadol. He denied any fever, chills, cough, chest pain, and diarrhea. His vital signs revealed a temperature of 34.8 °C, a respiratory rate of 23 breaths/min, a heart rate of 59 beats/min, and a blood pressure of 223/98 mmHg. On physical examination, hypoactive bowel sound was noted, accompanied by whole abdominal tenderness, especially in the periumbilical area. Muscle guarding was not significantly noted. The Murphy sign was no significant tenderness, as well as at McBurney's point. Bilateral knocking pain was negative. In the extremities, no significant peripheral arterial occlusive sign was noted. The laboratory evaluation was listed in Table 1.

**Table 1.** The laboratory evaluation in the patient.

| Variables | Patient | Normal Range |
|---|---|---|
| White cell count | 10030 /μL | 3500–11000 /μL |
| Band form neutrophils | 0.0% | |
| Segment form neutrophils | 47.7% | |
| Lymphocytes | 39.3% | |
| Eosinophils | 8.2% | |
| Monocytes | 4.0% | |
| Hemoglobin | 15.8 g/dL | 12–16 g/dL |
| Platelet counts | 184000 /uL | 150000–400000 /uL |
| Blood urine nitrogen (BUN) | 26 mg/dL | 7–18 mg/dL |
| Creatinine | 2.0 mg/dL | 0.55–1.02 mg/dL |
| Sodium | 142 mmole/L | 136–145 mmole/L |
| Potassium | 3.2 mmole/L | 3.5–5.1 mmole/L |
| Glucose | 143 mg/dL | 70–100 mg/dL |
| Alanine aminotransferase | 23 U/L | 14–59 U/L |
| Lipase | 345 IU/L | 73–393 IU/L |
| Total bilirubin | 0.73 mg/dL | 0.0–1.0 mg/dL |
| Troponin I | <0.01 ug/L | <0.01 ug/L |
| FDP-D-dimer | 3548.18 ng/mL | <500 ng/mL |
| C-reactive protein | 0.24 mg/dL | 0–0.33 mg/dL |

The plain film showed no significant pneumonia patch and free air (Figure 1A). The calcification of the aorta was noted with atherosclerosis. The kidney, ureter, and bladder (KUB) study revealed a gallbladder stone and no perihepatic free air (Figure 1B). According to the above clinical symptoms and image, hollow organ perforation was initially suspected.

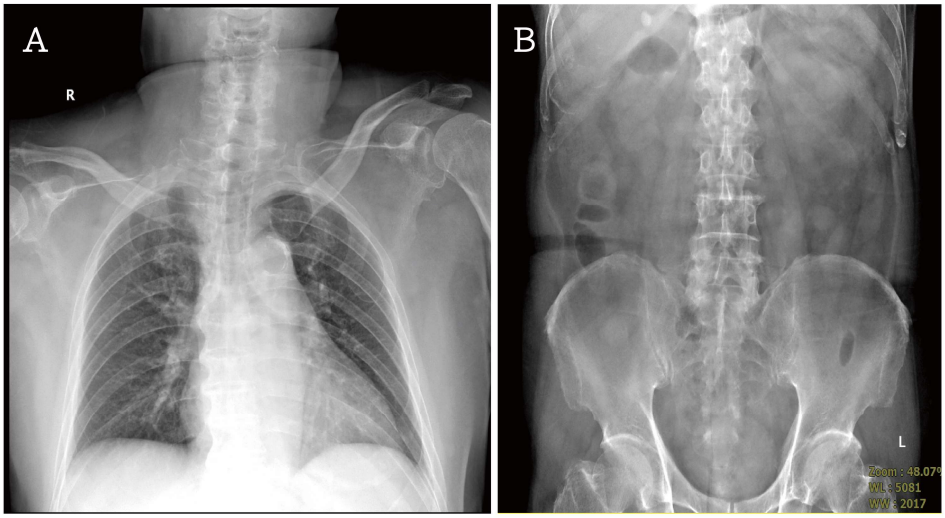


**Figure 1.** (**A**) There was no significant pneumonia patch and free air. (**B**) A gallbladder stone and no perihepatic free air were found.

A non-contrast enhanced abdominal CT was performed due to poor renal function and revealed no extra-luminal free air in the abdomen, but mild increased air in the non-distended loops of the small bowel. A 0.5 × 0.5 $cm^2$ gallbladder stone with mild local fat stranding was noted (Figure 2A,B). Cholecystitis was suspected and broad-spectrum antibiotics were administrated for the prevention of sepsis.

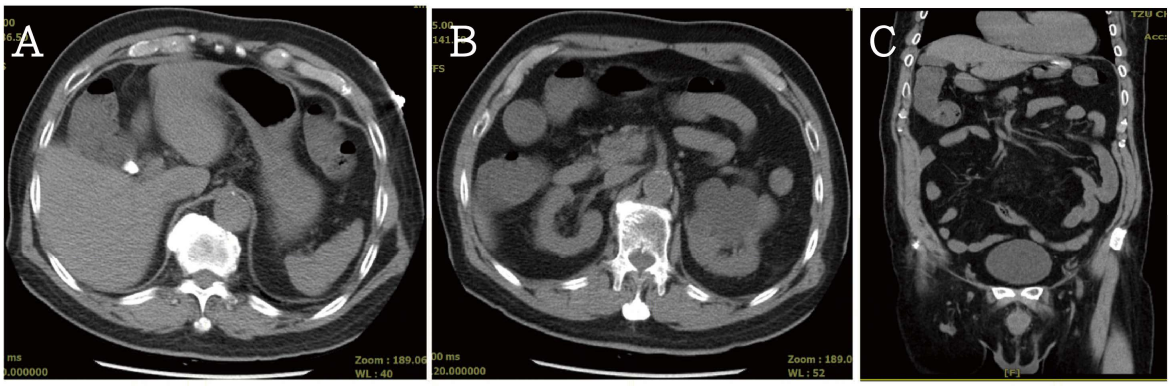


**Figure 2.** (**A**) A 0.5 × 0.5 $cm^2$ gallbladder stone with mild local fat stranding was noted in the non-contrast enhanced CT scan. (**B**,**C**) There was no extra-luminal free air in abdomen.

However, the abdominal pain was progressive and shifted to right flank pain. General muscle guarding was noted and accompanied with hypoactive bowel sound. Acute cholecystitis with perforation and impending peritonitis was suspected. An emergency laparoscopic cholecystectomy was carried out and revealed ischemia, a light purple color, venous thrombosis, and a lack of motion of whole small intestine. There was no abdominal ascites. The gallbladder was distended without local inflammation (Figure 3).

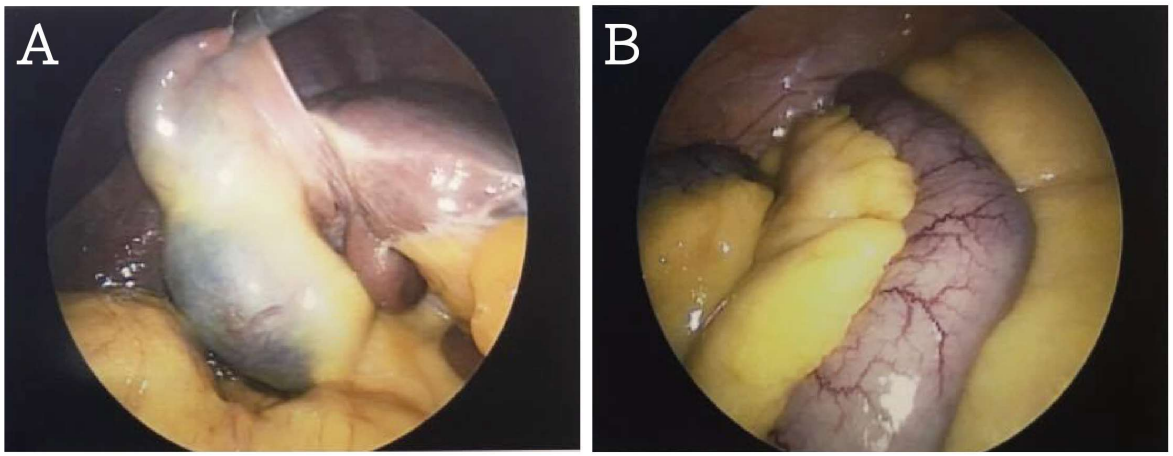


**Figure 3.** (**A**,**B**) Ischemia and a light purple and motionless small intestine was noted.

Broad-spectrum antibiotics were administrated to control sepsis. An emergency embolectomy was carried out, but the abdominal pain persisted and progressed. A urokinase pump was used, but a progression of abdominal pain was noted, accompanied with hypotension and sepsis. The physical examination revealed the progression of the whole abdominal tenderness with muscle guarding. The follow-up contrast enhanced abdominal CT showed acute superior mesenteric artery occlusion and post-operative pneumoperitoneum (Figure 4). The secondary exploratory laparatomy revealed bowel distention with 200 mL bloody ascites. The massively ischemic small intestine was about 250 cm and the relatively healthy small intestine was about 10 cm of terminal ileum and 80 cm of proximal

jejunum. The patient's family refused massive small bowel resection and requested extubation and palliative treatment. Sepsis progression was noted with hypotension. Cardiac arrest was found with pulseless electrical activity (PEA), finally. Oral informed consent was obtained from the patient.

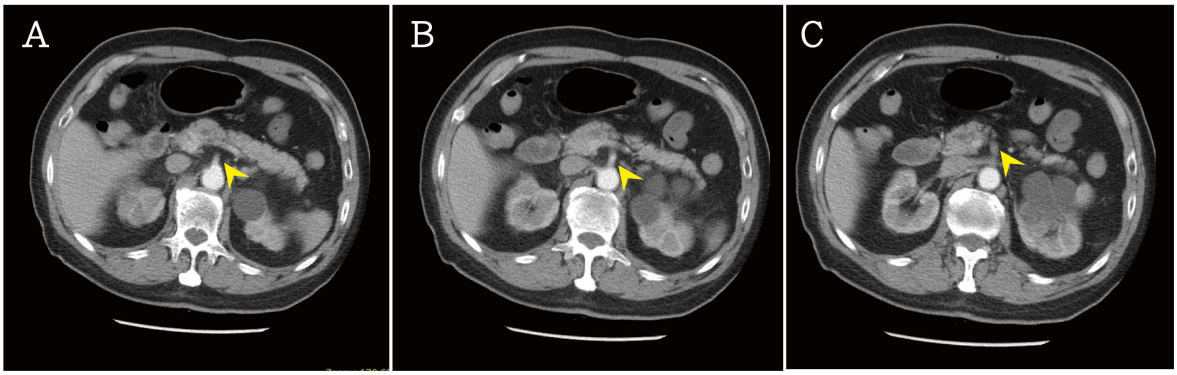


**Figure 4.** (**A**–**C**) Severe acute superior mesenteric artery occlusion was noted (arrow head).

## 3. Discussion

Acute mesenteric infarction is a rare but life-threatening disease, resulting from the impaired blood flow of mesenteric vessels, leading to bowel ischemia and gangrene formation. Acute mesenteric infarction can be divided into acute mesenteric arterial embolism and thrombosis. According to the study by Acosta et al. [2,3], embolic occlusion accounts for about 57.3% and thrombosis for about 41.3% of acute mesenteric infarction. The location of thrombotic occlusion was usually at a more proximal site [2]. Risk factors of embolic occlusion were reported, such as cardiac emboli from atrial fibrillation, sepsis, ruptured atheromatous plaque or iatrogenic injury. Atherosclerotic vascular disease, aortic aneurysm or dissection may be involved. The ileum was the most common intestinal infarction site, accounting for 96%, followed by the jejunum (76%) and colon (61%). The presentation of early clinical symptoms and signs, such as nausea, vomiting, abdominal pain and images, are nonspecific. In laboratory tests, the D-dimer is reported as a non-invasive method for acute mesenteric infarction [3]. In 2009, a prospective, non-interventional study showed the median D-dimer level in acute mesenteric ischemia was 6240 ng fibrinogen equivalent units (FEU)/mL, with a range from 960 to 5348 ng FEU/mL. In 2017, Da-Li Sun et al. reported that the combined sensitivity was 94%, the specificity was 50%, the positive likelihood ratio was 1.9, the negative likelihood ratio was 0.12, the diagnostic odds ratio was 16 and the area under the curve (AUC) was 0.81 for a total of 1300 patients studied [4]. In our case, the elevated D-dimer of up to 3548.18 ng/mL may have been a hint for the physicians to attempt to identify the cause, such as pulmonary embolism, aorta dissection, and acute mesenteric infarction.

A mortality rate of up to 67% to 80% has been reported for acute intestinal ischemia [5]. Early and aggressive therapeutic intervention can significantly reduce mortality and prevent the development of peritonitis and septic shock [6]. However, the unspecific presentation may confuse physicians and delay timely diagnosis. The elevated D-dimer in abdominal patients may be induced by acute mesenteric ischemia, acute abdominal aortic dissection, pancreatitis and acute cholecystitis. In atypical abdominal pain, the D-dimer may provide a hint of acute intestinal ischemia. We presented the case of a 85-year-old male presenting with acute onset progressive periumbilical cramping pain with elevated D-dimer. We highlighted image and laboratory studies in patients with a high risk of acute cute mesenteric infarction, especially those presenting atypical symptoms. Early diagnosis and timely intervention may improve clinical outcomes.

**Author Contributions:** M.-Y.W. and L.-C.L. wrote the paper and contributed to the organization of the figure; Y.-C.C., B.-Y.N. and Y.-L.C. provided conceptual input; G.-T.Y. and Y.-T.H. proofread and organized the manuscript. All authors reviewed the final version of the manuscript.

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

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