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Authors

Strait, Adrienne
Gasper, Warren
Dhaliwal, Gurpreet

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CLINICAL PRACTICE
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The Resolution of Abdominal Pain: an Ominous Sign of Mesenteric Ischemia

Adrienne Strait, BA[1], *Warren Gasper, MD*[2,3], *and Gurpreet Dhaliwal, MD*[4,5]

[1]School of Medicine, University of California San Francisco, San Francisco, CA, USA; [2]Department of Surgery, University of California San Francisco, San Francisco, CA, USA; [3]Vascular Surgery Section, San Francisco Veterans Affairs Medical Center, San Francisco, CA, USA; [4]Department of Medicine, University of California San Francisco, San Francisco, CA, USA; [5]Medical Service, San Francisco Veterans Affairs Medical Center, San Francisco, CA, USA.

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INTRODUCTION

Acute mesenteric ischemia requires rapid diagnosis and treatment to reduce its substantial morbidity and mortality. In a patient over 75 years of age, it is a more common cause of an acute abdomen than appendicitis or a ruptured abdominal aortic aneurysm.[1] Despite efforts to improve early diagnosis, the mortality of patients with acute mesenteric ischemia remains between 60 and 80%.[2,3]

CASE PRESENTATION

A 70-year-old man presented to the Emergency Department after 6 weeks of progressive post-prandial abdominal pain. Over the last 3 weeks, he had nausea, vomiting, non-bloody diarrhea, food aversion, and a 6.8-kg weight loss. Over the last week, his symptoms had progressed to the point that he was unable to eat or drink water or take medications without severe abdominal pain; in addition, the frequency of vomiting and diarrhea had increased to 4–6 episodes every day. On presentation, his abdominal pain was an 8 out of 10 in severity.

His medical history included hypertension, hyperlipidemia, myocardial infarction, coronary artery bypass grafts, and peripheral artery disease treated with right femoral thromboendarterectomy and patch angioplasty. Six weeks prior to admission, he was diagnosed with multiple myeloma for which he had been prescribed ixazomib, lenalidomide, and dexamethasone. He was also taking oxycodone and ibuprofen for his abdominal pain. He discontinued all medications (except for lenalidomide and dexamethasone) 2 weeks prior to presentation, suspecting they could be exacerbating his symptoms, but there was no improvement.

The patient was afebrile with blood pressure 112/70 mmHg, heart rate 103 beats/min, respiratory rate 16 breaths/min, and oxygen saturation 97% on room air. He was cachectic and had a soft, non-distended abdomen without tenderness or guarding. The white blood cell count (WBC) was 14,060 cells/μL with an absolute neutrophil count of 11,260 cells/μL. Other laboratory tests included normal liver biochemical tests and lipase, creatinine 0.95 mg/dL, C-reactive protein 100 mg/L (0.2–7.5), carbon dioxide 20 mmol/L (24–32), lactate 3.6 mmol/L (0.5–2.2), and troponin 0.05 (0.00–0.03). An electrocardiogram showed normal sinus rhythm with left ventricular hypertrophy. His tachycardia and lactic acidosis resolved with fluid administration, and his pain decreased after 4 mg of intravenous morphine and intravenous ondansetron. A fecal PCR assay for 22 enteric pathogens was negative.

The team ordered an abdominal contrast CT on admission, but there were logistical barriers to obtaining the study after-hours. Given the concern for chronic, but not acute, mesenteric ischemia, as well as the improvement in his lactic acidosis, tachycardia, and pain, the team planned to obtain imaging the next morning. The patient received 2 mg of intravenous morphine for his abdominal pain overnight.

The following day (hospital day 2), the patient reported resolution of his abdominal pain and had a soft, non-tender abdomen. He ate a clear liquid diet and asked to go home. His WBC had decreased to 11,780 cells/μL. A contrast CT of the abdomen and pelvis (Fig. 1) showed a distended gallbladder with mild wall enhancement and dilated loops of the small bowel in the left upper quadrant; a discussion with the radiologist revealed there were no signs of mesenteric ischemia. General surgery was consulted. Later in the evening, an additional finding of occlusive atherosclerotic disease at the origin of a combined celiac/superior mesenteric artery (SMA) trunk was noted on the CT report (Fig. 2); a non-urgent consultation with vascular surgery was planned for the next day.

Overnight, his abdominal pain recurred. On the morning of hospital day 3, he had a newly distended abdomen with diffuse tenderness and guarding. The WBC was 9,980 cells/μL and the aspartate aminotransferase was newly elevated at 79 U/L (5–35). An abdominal ultrasound showed a sludge-filled gallbladder with no stones or surrounding fluid. The vascular surgery service was consulted. That afternoon, the oxygen saturation decreased to 70%. His arterial blood gas (ABG) was 7.18/23/75/8.2 on a non-rebreather mask, and he was emergently intubated for acute hypoxic respiratory failure. A

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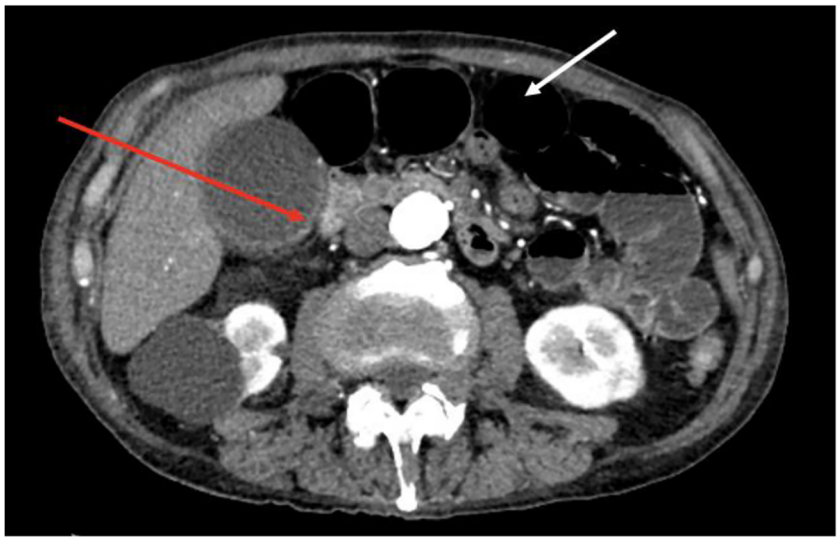

Figure 1 Contrast computed tomography of the abdomen and pelvis on hospital day 2 demonstrated a distended gallbladder with mild wall enhancement (red arrow) and dilated loops of the small bowel in the left upper quadrant (white arrow).

chest X-ray was normal. Serum lactate was 13.2 mmol/L, and creatinine had risen to 1.32 mg/dL. His blood pressure was difficult to measure accurately. The patient was transferred to the intensive care unit (ICU), where lactated Ringer's solution, bicarbonate, piperacillin/tazobactam, and norepinephrine were administered.

A repeat ABG was 7.28/23/402/10.7 on 100% FiO_2 with a serum lactate of 14.3 mmol/L. A CT of the abdomen and pelvis with contrast (Fig. 3) showed extensive hypoperfusion of the entire small bowel and ascending colon with pneumatosis, dilated small bowel, and hepatic portal venous gas. The patient was diagnosed with acute-on-chronic mesenteric ischemia. General surgery performed an exploratory laparotomy that revealed a grossly necrotic right colon from the hepatic flexure to the ileocecal valve, an ischemic ileum and distal jejunum, and a necrotic gallbladder. Right ileocolectomy and open cholecystectomy were performed. Vascular surgery performed balloon embolectomy and stenting of the combined celiac/SMA trunk. Prior to closure, the patient suffered a cardiac arrest with pulseless electrical activity (PEA). After return of spontaneous circulation and return to the ICU, he had refractory hypotension and suffered

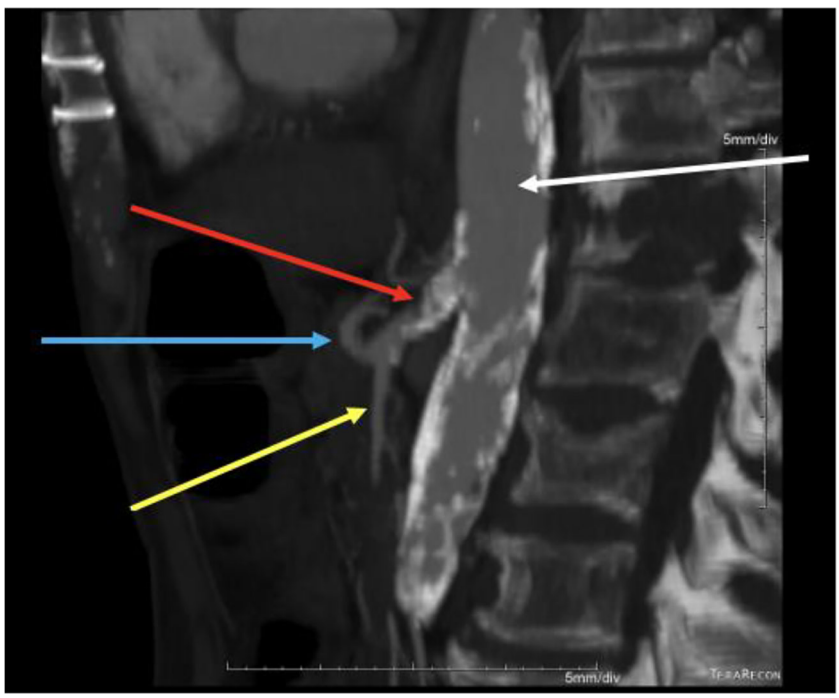

Figure 2 Contrast computed tomography of the abdomen and pelvis on hospital day 2 showed occlusive atherosclerotic disease at the origin of a combined celiac/superior mesenteric artery trunk (red arrow). The white arrow denotes the aorta, the blue arrow denotes the celiac artery, and the yellow arrow denotes the superior mesenteric artery.

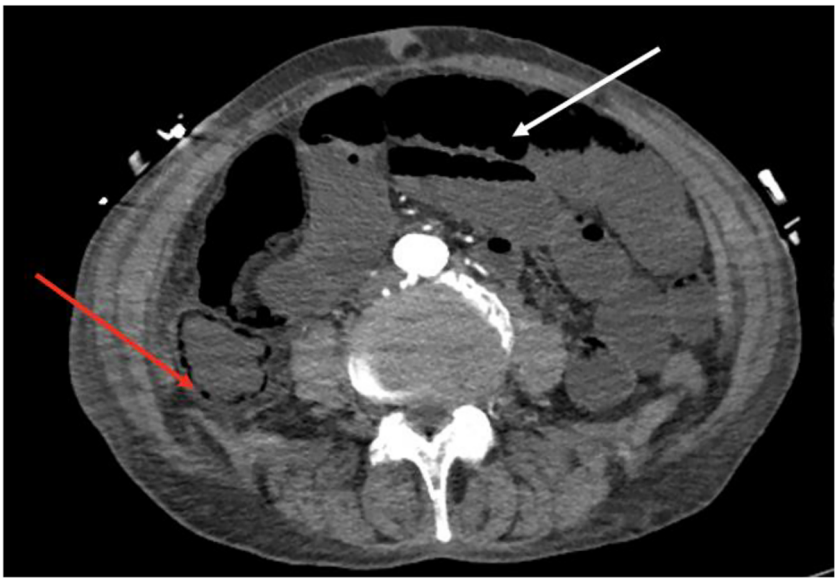

Figure 3 Contrast computed tomography of the abdomen and pelvis on hospital day 3 revealed hypoperfusion of the entire small bowel and ascending colon with pneumatosis (red arrow), dilated small bowel (white arrow), and hepatic portal venous gas (not visualized on this image).

another PEA arrest. Cardiopulmonary resuscitation was unsuccessful. Pathology revealed transmural necrosis of the ileum, colon, appendix, and gallbladder and severe mesenteric atherosclerosis. Blood cultures obtained at ICU admission grew *Escherichia coli*.

DISCUSSION

Chronic mesenteric ischemia was initially suspected based on the weeks of post-prandial pain, weight loss, and widespread atherosclerosis. The resolution of his pain overnight lowered the medical team's suspicion of acute mesenteric ischemia. An understanding of the anatomy and pathophysiology gives insight into why this change can be ominous, not reassuring.

The symptoms of early acute mesenteric ischemia vary depending on the acuity, extent of arterial obstruction, and bowel wall layer (mucosa, submucosa, muscularis, serosa) involvement.[1] Patients with acute mesenteric ischemia experience visceral pain as the intestinal layer furthest from the blood supply, the mucosa, becomes ischemic from thrombosis, embolism, dissection, or vasospasm. When the muscularis and serosa remain perfused, there is no peritoneal irritation. Ischemia of the mucosa with perfusion of the other layers leads to a soft abdomen with severe pain and mild focal tenderness but no rigidity or involuntary guarding (i.e., "pain *out of proportion* to exam"). As the ischemia progresses, the muscularis and serosa also become ischemic, which causes peritoneal irritation, leading to peritoneal signs commensurate with the abdominal pain ("pain *in proportion* to exam"). In between the early and late phases of ischemia, there may be a deceptive pain-free interval lasting 3 to 6 hours that has been attributed to ischemia of the intramural pain receptors.[4]

Our patient experienced a pain-free interval during hospital day 2 before a marked recurrence of pain on the morning of day 3. Our case emphasizes the importance of recognizing this period of quiescence as an ominous sign of progressive bowel ischemia that requires urgent surgical intervention in order to prevent the ensuing and irreversible bowel necrosis.

Computed tomographic angiography (CTA) is the imaging study of choice for diagnosis of acute mesenteric ischemia and provides information about the extent of vessel stenosis or occlusion. Other imaging findings may be present in acute mesenteric ischemia depending upon the etiology and stage of the ischemia including mural hypoenhancement, thickening or thinning of the bowel wall, mesenteric stranding, and bowel

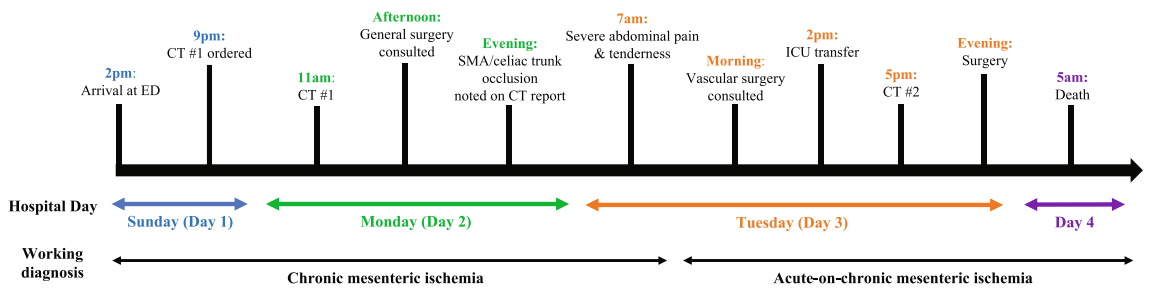

Figure 4 A timeline of the patient's hospital course demonstrating key events, diagnostic steps, and the change in the general medicine team's working diagnosis.

dilatation.[5,6] While bowel wall thickening is the most sensitive indicator of ischemia, it is the least specific as it can also result from inflammatory and infectious conditions. Pneumatosis and portal venous gas are signs of end-stage bowel necrosis.

The first CT scan in this case showed small bowel dilatation and a distended gallbladder, which shifted the team's attention away from acute mesenteric ischemia. In retrospect, both imaging findings, in the setting of a high suspicion for chronic mesenteric ischemia and the demonstration of a combined celiac/SMA trunk, should have *increased* the concern for acute ischemia. Small bowel dilatation is a non-specific imaging finding associated with acute mesenteric ischemia that is likely related to hypomotility of the ischemic segment of intestine.[5,6] The severely stenosed combined celiac/SMA trunk (an anatomic variant) explains how the distended gallbladder (which is supplied by the celiac artery) with mild wall enhancement was an ischemic gallbladder and an overlooked sign of his transition from chronic to acute mesenteric ischemia.

The standard approach to the management of acute mesenteric ischemia includes heparin, fluid resuscitation, bowel rest, and surgical revascularization. Broad-spectrum antibiotics are recommended to reduce the risk of sepsis from bacterial translocation across the injured mucosal barrier.[7] Rapid revascularization is a cornerstone of treatment and can be performed through an open (bypass grafting, embolectomy, or endarterectomy) or endovascular (stenting or thrombolysis) technique depending on the etiology of the acute mesenteric ischemia. An assessment of bowel viability is necessary regardless of revascularization technique. A systematic review of published case series demonstrated that the endovascular approach may lead to lower short-term mortality and morbidity;[8] however, there was selection bias in the included studies.

LEARNING FOR NEXT TIME

In order to learn more from the case, our team studied the course of events (Fig. 4), presented the case at internal medicine morbidity and mortality conference, and spoke with the vascular surgeons, who also attended the conference.

A key lesson from this case is that the temporary resolution of abdominal pain in a patient with suspected chronic mesenteric ischemia can be an ominous (rather than reassuring) sign that signals the transition to acute-on-chronic mesenteric ischemia. Analgesics can also account for a reduction in abdominal pain. However, it is unlikely that two doses of morphine in a patient who used oxycodone regularly would lead to a sustained pain-free interval.

We also learned that an imaging finding of nearly occlusive atherosclerotic disease of the SMA in a patient with recent abdominal pain (even if acute mesenteric ischemia was not suspected) warrants prompt evaluation by the vascular surgeons. When we (G.D.) heard about this finding from the radiologists, we should have requested urgent surgical evaluation that evening instead of the next day. The surgeons shared an analogy that we found particularly helpful: nearly occlusive, stable disease of the SMA can be thought of like a tight stenosis of the left main coronary artery. Both require close monitoring in an asymptomatic patient, but when any referable and progressive symptoms develop, there is a very low threshold to revascularize as the stenosis can quickly progress to complete occlusion and irreversible ischemia.

Corresponding Author: *Adrienne Strait, BA; School of Medicine, University of California San Francisco, San Francisco, CA, USA (e-mail: Adrienne.Strait@ucsf.edu).*

Conflict of Interest: *Dr. Dhaliwal reports receiving honoraria from ISMIE Mutual Insurance Company and GE Healthcare.*

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