Abrupt onset of abdominal pain

What was causing this patient's epigastric abdominal pain, which began several days after he'd been hospitalized for acute pericarditis?

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The authors reported no potential conflict of interest relevant to this article.

The views expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, or the US government.

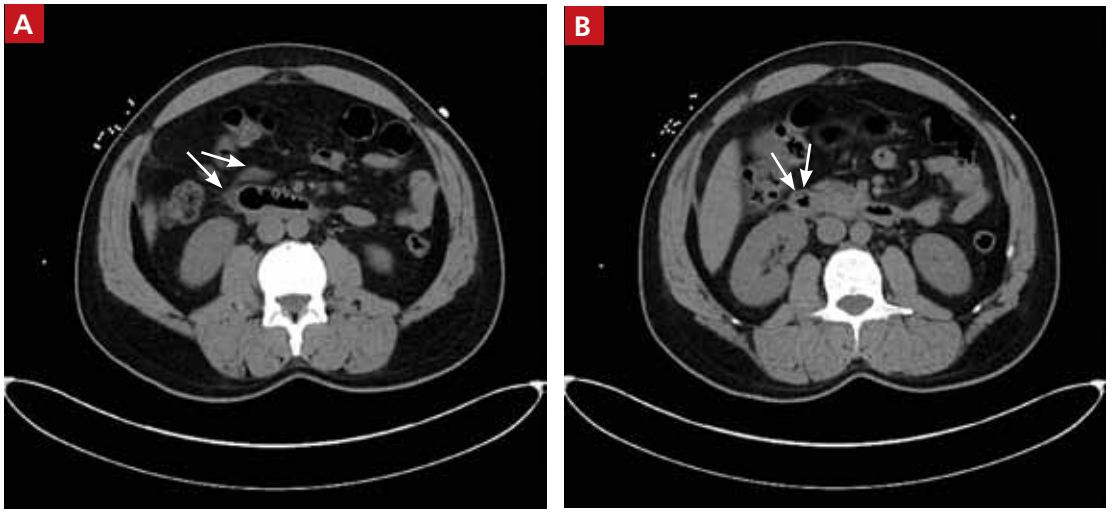
SIX DAYS AFTER BEING DISCHARGED FROM THE HOSPITAL for treatment of acute pericarditis, a 49-year-old man came to our clinic for a follow-up appointment. The patient still had midsternal chest discomfort and dyspnea. He also reported new epigastric abdominal pain, which had begun 2 days earlier. The patient's wife indicated that he'd had a low-grade fever since discharge, but the patient denied any chills, hematemesis, melanotic stools, diarrhea, or constipation.

The patient's medical history was significant for gastroesophageal reflux, hyperlipidemia, osteoarthritis, sleep apnea, and obesity. Along with newly prescribed indomethacin for the pericarditis (50 mg tid), he was also taking simvastatin. In addition, he occasionally took ibuprofen (800 mg/d) for osteoarthritis of his knee.

On physical examination the patient appeared uncomfortable. Auscultation of his lungs and heart was unremarkable. His abdomen was mildly distended, with hyporesonant bowel sounds and involuntary guarding upon palpation of the upper abdomen. The location of the pain was 2 cm below the xiphoid process. The patient was admitted to the hospital for further work-up, which included computed tomography (CT) (**FIGURE 1A AND 1B**).

- WHAT IS YOUR DIAGNOSIS?
- HOW WOULD YOU TREAT THIS PATIENT?

FIGURE 1
The patient's pain was 2 cm below the xiphoid process



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Antibiotics for a perforated GI tract should cover for gram-negative enteric bacteria and anaerobes.

Diagnosis: Duodenal perforation caused by indomethacin

The CT scans revealed inflammation (arrows, **FIGURE 1A**) and thickening of the second and third portion of the duodenum and the presence of extraluminal air at the site of the perforation (arrows, **FIGURE 1B**). There was also free fluid along the right paracolic gutter and into the pelvis. We diagnosed a small intestinal perforation in this patient, which was likely caused by a nonsteroidal anti-inflammatory drug (NSAID)-induced ulcer.

How NSAIDs affect the GI tract

NSAIDs inhibit cyclooxygenase (COX), the enzyme responsible for prostaglandin production. Specifically, the COX-1 enzyme is responsible for the production of prostaglandins in the gastrointestinal (GI) tract. Prostaglandins play an important role in protecting the GI mucosa. By inhibiting the synthesis of prostaglandins, the permeability of the GI tract is increased and the natural protective barrier of the mucosa is destroyed.[1]

Gastroduodenal damage is a well-known adverse effect of NSAIDs. Ulcers have been noted on upper endoscopy in regular NSAID users, and the risk of developing a symptomatic ulcer and complications increases with every year of regular NSAID use.[2] Ulcers in the GI tract can be complicated by perforation.[1]

Patients will complain of sudden onset abdominal pain

Important clues in the patient history for a perforated GI tract include sudden onset of severe abdominal pain that may present initially as epigastric pain and progress to generalized abdominal pain that may radiate to one or both shoulders.[3] Physical exam findings for a perforated GI tract may include abdominal tenderness and rigid abdomen; fever and tachycardia may also be present.[3] Concern for possible abdominal perforation should be evaluated with a complete blood count, basic metabolic panel, and radiographic studies.[3]

The differential diagnosis of epigastric abdominal pain includes: pancreatitis, gastritis, gastric/duodenal ulcer, and obstruction. Elevated serum lipase levels or CT findings of pancreatic inflammation assist in the diagnosis of pancreatitis. Upper endoscopy is key to identifying cases of gastritis and ulcers in the GI tract. And an abdominal radiograph that shows dilated loops of bowel will confirm suspicions of obstruction.

Stabilize the patient

Acute management of GI perforation begins with stabilizing the patient and determining the need for surgical intervention or medical management. Should a patient have a persistent air leak, surgery is the mainstay of treatment.[3] If the perforation has healed, the patient should be medically managed.

Therapy may require placing a nasogastric tube and holding any oral intake until abdominal pain and perforation resolves. Medications in the acute treatment should include proton pump inhibitors (PPIs) and antibiotics. Antibiotics should cover for gram-negative enteric bacteria and anaerobes.

How to prevent NSAID-induced injury. PPIs used with nonselective NSAIDs appear to reduce the likelihood of gastric ulceration.[4] Similarly, H_2-receptor antagonists (H_2RA) also inhibit gastric acid secretion, and high doses of them significantly reduce the incidence of gastric ulcers.[4] However, standard doses of H_2RAs have not been shown to be effective in reducing the risk of NSAID-induced gastric ulcers.[4] Misoprostol, a synthetic prostaglandin E1 analogue that inhibits gastric secretion and protects the gastric mucosa is also an option; it has been used in combination with nonselective NSAIDs to counteract the increase in GI permeability.[4]

What about using COX-2 inhibitors instead? The COX-2 inhibitors have a lower risk of gastrointestinal tract injury.[2] Gastric ulcers, GI bleeding, and complications from ulcers have been shown to be less common with COX-2 NSAIDs compared with nonselective NSAIDs.[5] A review of the literature suggests that for patients with previous GI bleeding, COX-2 inhibitors are comparable to an NSAID paired with a PPI in preventing GI bleeding; pairing a COX-2 inhibitor with a PPI, however, appears to provide the greatest defense.[2,4]

Consider preventive steps. Patients at risk for complications are likely to benefit

from the simultaneous use of prophylactic agents with NSAID therapy. Risk factors for NSAID-related GI complications include: a previous GI event; older age; simultaneous use of anticoagulants, corticosteroids, or other NSAIDs (including low-dose aspirin); high-dose NSAID therapy; and chronic debilitating disorders (especially cardiovascular disease).[6]

Our patient improved with medical management

Our patient was evaluated by general surgery during his hospital admission, but because he was stable—with a healing duodenal perforation—we opted to manage him medically. We started him on esomeprazole (40 mg bid) along with ciprofloxacin (500 mg bid) and metronidazole (500 mg tid) for the microperforation of the duodenum. The patient was also scheduled for an outpatient endoscopic evaluation. JFP

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