

FEVER, FLOOD WATER, AND GASTRITIS

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Case Report

A 76-year-old woman was admitted with a 1 week history of diffuse, intermittent abdominal pain and nonbilious emesis after her home had been destroyed by a flood. She reported watery diarrhea coupled with occasional bouts of melena, as well as subjective fever on the day of admission. Past medical history was notable for diabetes mellitus, hypertension, and Paget's disease. Medications included aspirin and omeprazole.

Upon arrival in the emergency room she was febrile to 39°C, but other vital signs were normal. The remainder of her physical examination was notable only for epigastric tenderness and heme-positive stool. Nasogastric lavage was negative for gross blood, white blood cell count was 24,300 cells/cu mm and hematocrit was 32.8%. She was hypokalemic with a potassium level of 2.1 meq/dL. An abdominal computed tomography (CT) revealed pneumatosis and thickening of the gastric wall at the greater curvature (Figure 8-1). Her blood cultures grew *Clostridium butyricum* within 24 hours and stool studies several days later grew *Aeromonas caviae*.

The patient was treated conservatively with broad-spectrum antibiotics for *Clostridium*-induced emphysematous gastritis in the setting of a recent *Aeromonas*-induced gastroenteritis (likely from flood water). She had a rapid clinical improvement, and a repeat CT scan 5 days later showed a normal gastric wall (Figure 8-2). An upper endoscopy 1 week after admission demonstrated gastritis with scattered areas of punctate hemorrhage at which time she was completely asymptomatic. Biopsies of the gastric mucosa were completely unremarkable.

Discussion

The patient had emphysematous gastritis, which is an uncommon infection of the gastric mucosa by gas-producing organisms (eg, *Clostridium*) with only 42 reported cases in the literature. This disease entity was first described clinically by Fraenkel in 1889 and as a radiological diagnosis by Weens in 1946.¹ This gastric mucosa and acidity usually provide efficient barriers against microorganisms; however, infection can occur following mucosal penetration or by hematogenous spread. Factors that may predispose one to emphysematous gastritis include corrosive ingestion, alcohol abuse, recent gastroenteritis, recent gastroduodenal surgery, immunosuppression, and dia-