Chronic intestinal ischemia producing abdominal angina may be accompanied by minor small bowel abnormalities demonstrable radiographically (1, 2) and histopathologically by changes in the intestinal mucosa (2). Although erosive gastritis has been documented in association with atheromatous embolization, to our knowledge, this is the first description of such a picture occurring secondary to chronic ischemia alone. It occurred in a patient with abdominal angina and severe occlusive disease of all three splanchnic arteries.

CASE REPORT

A 69-year-old white female was admitted to the Medical Center Hospital of Vermont for evaluation of unstable angina pectoris. In addition to chest pain, during the 5 months before admission, she had also suffered epigastric and left upper quadrant pain starting about 5-10 min after meals and lasting 1-2 hr. Occasionally the pain was associated with vomiting but rarely with blood-streaking. Approximately 4-6 hr after eating, she also often had 2-4 explosive, watery stools.

The relationship between pain and meals was so consistent that during the two weeks before admission, she avoided all solid foods and drank small amounts of liquid only. Since the onset of her gastrointestinal symptoms, she had lost 20 pounds.

Medications included oral iron, nitrates, and propranolol. No aspirin, alcohol, or other gastric irritants had been taken during the few weeks prior to admission.

She was normotensive (160/80). The carotid upstroke was reduced, and there were bruits over both carotid and femoral arteries. Foot pulses were weak but present. A grade III/V holosystolic murmur was heard best at the apex of the heart. The remainder of the cardiovascular examination was normal. No abnormality was detected on abdominal examination. The stool was hemo occult positive. The hematocrit was 34%, and the erythrocyte sedimentation rate 63 mm/hr. The serum iron and total iron binding capacity were 27 μg/dl and 367 μg/dl, respectively. The serum amylase was normal at 58 units/dl. Extensive calcification of the abdominal aorta, iliac, and splenic arteries was seen on plain abdominal x-ray.

While her angina pectoris was being stabilized, investigation of her abdominal complaints included an air-contrast barium enema and sigmoidoscopy, which were normal and an upper gastrointestinal series and small-bowel follow-through which showed slight dilatation of a segment of small intestine and widened mucosal folds.

Initially, she was treated with antacids and cimetidine, but increase in her symptoms finally led to her refusal to take in anything by mouth. An esophagogastroduodenoscopy was performed after premedication with meperidine, promethazine, and diazepam. Atrophic appearing gastric rugae with irregular areas of raised mucosa of less than 5-mm elevation, multiple superficial ulcerations of 3-15 mm diameter through the stomach and duodenum, and an absence of motility were noted. The endoscopic appearances resembled those of gastric lymphoma (3), although, histologically, multiple biopsies of the stomach and the duodenum showed edema, acute inflammatory changes, and small focal deposits of bilirubin in the submucosa and lamina propria (Figure 1).

Further vascular evaluation was considered, but three days after endoscopy, the patient developed cold, blue, pulseless legs and died within a few hours. At post-mortem examination, there was marked calcified atheromatous degeneration of the abdominal aorta extending distally from above the origin of the celiac axis (Figure 2). Below the celiac axis, the lumen of the aorta was completely occluded by premortem thrombus and atheroma. Both celiac and superior mesenteric arteries were completely occluded for approximately 2 cm distal to the ostia. The inferior mesenteric artery was occluded at its aortic origin, but patent distally. The stomach was smooth with only occasional rugae present as was apparent endoscopically, with small mucosal ulcers and areas of hemorrhage (Figure 3). There was extensive ischemic discoloration of the small in-
Fig 1. Premortem duodenal biopsy showing edema of the lamina propria and infiltration with polymorphonuclear leukocytes (H&E, × 400).

testine. No other underlying pathology was found either in the gastrointestinal tract or lymphatic system. Furthermore, extensive histological examination of the stomach did not reveal any cholesterol clefts as are observed with atheromatous embolization. The only gastric changes were those of edema, inflammation, and premortem thrombus with early organization.

DISCUSSION

The fear of eating, weight loss, and diarrhea in this patient are typical symptoms of chronic intestinal ischemia, a diagnosis supported by the autopsy findings of severe aortic atheroma with occlusive disease of all three major vessels supplying the gut. The endoscopic appearance of the gastric and duodenal mucosa observed in this case, however, has not, to our knowledge, previously been described as a result of chronic ischemia.

Multiple superficial erosions of the gastric mucosa may result from atheromatous embolization (4, 5) and in that situation, infarction and hemorrhagic gastritis may occur (5–7). Embolization has also been reported in association with chronic nonspecific abdominal complaints (5), with asymptomatic blood loss (4), and "classical" gastric (8) and duodenal (9) ulcers. Endoscopic biopsies of ulcerative lesions due to embolization usually only show nonspecific inflammation, although bone-marrow biopsy may reveal the typical cholesterol clefts (10). Absence of cholesterol clefts from stomach, intestine, and bone marrow in this patient makes embolization unlikely.

Other causes of erosive gastritis include ingestion of aspirin, alcohol, and other gastric irritants, none of which had been taken during the few weeks prior to this patient's admission. Infiltrative disorders such as lymphoma, amyloid, or granulomatous processes may produce similar endoscopic appearances but were ruled out by the subsequent autopsy findings.

Multiple superficial ulcers and villous atrophy have been described in the small intestine in patients who have had abdominal angina as a result of atheromatous occlusive disease (2). Thus, it is not surprising that similar changes of chronic injury may be seen in the stomach and duodenum in a patient who had severe occlusive disease of at least the celiac and superior mesenteric vessels. We could find no reports in the literature of multiple gastric ulcerations or gastritis secondary to splanchnic vessel atherosclerosis, although an association between single peptic ulcers and atheromatous disease has been suggested (11, 12). Clearly, the superior mesenteric artery territory is more at risk since