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MASSIVE HEMORRHAGE FROM SARCOID ULCERS IN THE STOMACH

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ALTHOUGH sarcoidosis is a systemic granulomatous disease capable of involving any organ or tissue, it has been seldom known to cause clinically significant gastrointestinal disease.

Involvement of the stomach by sarcoid granulomas was first described by Schaumann in 1936.1 It was considered rare,2,3 however, until 1958 when Palmer, by means of suction biopsies, found epithelioid-cell tubercles in the gastric mucosa of six out of 60 cases of asymptomatic generalized sarcoidosis.4 All cases had negative upper gastrointestinal x-ray studies and gastroscopic examinations.

Four cases have been described of pyloric stenosis in sarcoidosis, typically presenting the radiographic picture of linitis plastica, which have been due to granulomatous infiltrations in the distal stomach.5,6,7,8 In two other reported cases of sarcoidosis, atypical gastric ulcerations were found to overlie submucosal granulomas.8,9 Six other cases of pyloric stenosis due to granulomatous gastritis of unknown etiology, presumably representing sarcoidosis, have been described.9,10,11,12,13,14 One of these cases also had mucosal ulcerations despite a histamine-fast achlorhydria.10 In three other cases of “isolated granulomatous gastritis”, presumably representing sarcoidosis, hemorrhage was found to originate in stomachs infiltrated with epithelioid cell tubercles.12,14,15 Bleeding occurred in ulcers, erosions, or friable areas of granulomatous gastric mucosa. Granulomas were found in lymph nodes adjacent to the stomach in most cases, but demonstration of more widespread involvement was not made.

The following case of sarcoid gastric ulcer is recorded as an example of a rare cause of massive, upper gastrointestinal hemorrhage.

REPORT OF A CASE

A 55-year-old Negro porter was admitted to the Henry Ford Hospital in July, 1963, because of hematemesis. During the previous year he had suffered epigastric discomfort before meals, always relieved by food or Alka-Seltzer. He had vomited occasionally, but he had never bled. He claimed a good appetite, but he had lost about 10 pounds. He denied any previous serious illnesses except for one episode of “pneumonia” 19 years before which required several weeks of hospital care. There was a chronic morning cough productive of small amounts of phlegm. The patient denied dyspnea on exertion, allergies, or the use of medications other than Alka-Seltzer.

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Physical examination on admission revealed a thin, muscular Negro in no acute distress. Vital signs were normal. There were persistent rhonchi in both lungs. Occasional premature beats were noted. Only in the inguinal areas were palpated a few large, movable, lymph nodes. Aspiration of the stomach at first yielded brown fluid; later, frank blood was aspirated.

An emergency upper gastrointestinal x-ray examination performed without palpation was hampered by retained material in the stomach; however, several spot films showed a persistent collection of barium in the body of the stomach with decreased peristalsis in that area (Figure 1). The lower esophagus and duodenal cap appeared normal. A diverticulum was seen in the third portion of the duodenum. Emergency conditions precluded an adequate gastric analysis.

![Figure 1](image1.png)

Films representing emergency radiographic examination of the upper gastrointestinal tract showing an irregular fleck of barium in the midportion of the stomach (indicated by arrows in spot film at right).

The hematocrit was 29 per cent. In the next 36 hours, the patient continued to bleed slowly and, in spite of 1500 ml. of whole blood, the hematocrit fell to 26 per cent and early signs of shock appeared. Laparotomy was performed by Dr. Joseph Ponka. A small area of thickening was found in the serosal surface of the posterior aspect of the lesser gastric curvature at the incisura. The mucosa in this area showed several shallow ulcers with serpiginous borders (Figure 2). The largest of these measured 1.5 cm. There was considerable blood in the lumen of the stomach, the remainder of which, together with the duodenum, appeared normal. A partial gastrectomy with gastroduodenostomy was performed, and the patient made an uneventful recovery.

Microscopically there was a sparse inflammatory infiltrate about the ulcers containing an unusual number of eosinophils; numerous non-caseous granulomas with multinucleated giant cells surrounded by epithelioid cells were present in the submucosa and the upper portion of the muscular layer. Isolated granulomas were seen at a distance from the ulcers underlying normal mucosa. One omental lymph node was examined and found to contain a large number of noncaseous granulomas. No organisms were identified with acid-fast and fungus stains. A needle biopsy of the liver showed many noncaseous granulomas (Figure 3). All of the granulomas seen in the stomach, lymph nodes, and liver were morphologically compatible with those of Boeck's sarcoid.

A chest x-ray showed scattered pulmonary scars and increased volume of the right lung, but mediastinal nodes were not visibly enlarged. The white blood cell count, differential count, and urinalysis were normal. The serum calcium was 9.2 mg., the serum phosphorus was 2.8 mg., and the serum alkaline phosphatase was 1.8 Bodansky units. The total serum protein was 7.5 grams/100 ml.; the gamma globulins comprised 2.25 grams.
In the resected stomach (a), two shallow ulcers were demonstrated. Granulomas typical of Boeck's sarcoid were found beneath and adjacent to the areas of gastric ulceration (b, H and E, 90X).
Whorls of granulomas filled a perigastric lymph node (a, H and E, 90X). Multiple granulomas were abundant in a subsequent needle biopsy of the liver (b, H and E, 325X).
A bromsulfalein test yielded no retention at 45 minutes. The VDRL test was nonreactive. Skin tests with tuberculin, histoplasmin, coccidioidin, and blastomycin were negative. A skin test with mumps antigen was positive.

Biopsy of the skin six weeks after the intradermal injection of Kveim antigen showed no typical granulomas. A postgastrectomy gastric analysis yielded a low titer of free HCl; therefore, free HCl in higher concentration must have been present before operation.

Although the surgical intervention was immediately life-saving, the case was soon thereafter sadly concluded. Two months following operation, when the patient appeared to have fully recovered, he was re-admitted of acute fulminant hepatitis presumably transmitted by a virus contained in the blood with which he had been previously transfused. Despite diligently applied supportive treatment, the patient died within a week of his second admission. Permission for necropsy was denied.

**DISCUSSION**

The etiology of the disease or group of diseases known as sarcoidosis is unknown. Indeed, the morbid condition may represent a host tissue response to a variety of noxious agents rather than a disease entity in itself.

A number of affections of the stomach are characterized by chronic granulomatous inflammation. These include tuberculosis, leprosy, syphilis, regional enteritis, histoplasmosis, coccidioidomycosis, brucellosis, cat-scratch disease, eosinophilic granuloma, berylliosis, silicosis, and other foreign-body granulomas involving minerals and lipids. Granulomas have been described in lymph nodes draining areas containing malignant tumors and can be easily produced in experimental animals during the course of hypersensitivity reactions to specific antigens.

The differential diagnosis of gastric sarcoidosis is aided by the Nickerson-Kveim skin test using a reliable antigen and biopsy after six weeks, and by needle biopsy of the liver. The skin test and liver biopsy are positive in about two-thirds of cases of sarcoidosis. The gross and microscopic anatomy of an isolated granulomatous gastritis may not permit definitive diagnosis. The presence of specific microorganisms can be detected by special stains. Proliferative endarteritis suggests syphilis, and caseation points to tuberculosis. Necrosis rarely may be seen in sarcoidosis; when present, it is fibrinoid and not caseous. Polarized-light microscopy may disclose birefringent foreign bodies as causative agents. The distribution of the epithelioid cell tubercles, nonglandular inclusion bodies and tissue eosinophilia are nonspecific.

In sarcoidosis a granulomatous gastritis can exist with no gastric symptoms or with only nonspecific digestive complaints. These cases often demonstrate no abnormality on gastrointestinal films or gastroscopy.

Extensive gastric infiltration by granulomas and subsequent fibrosis can radiographically resemble antral carcinoma or linitis plastica. The symptoms may be those of pyloric stenosis. The wall of the stomach is thickened and rubbery rather than hard. The mucosa may be cobblestoned and the normal rugal pattern obliterated. Granulomas may be found in every layer of the gastric wall and may extend into the duodenum. A histamine-fast achlorhydria is almost always present in such cases.
Finally, a granulomatous gastric mucosa may become eroded and ulcerated, leading occasionally to gastric hemorrhage. Localized ulcerations in such cases permit a life-saving surgical remedy. It is recognized, of course, that granulomatous ulcerations may recur in the gastric remnant.

In the diagnosis and management of patients with sarcoidosis and gastrointestinal manifestations, two other points are worth noting. Hypercalcemia may be associated with epigastric pain and vomiting, and correction of an elevated serum calcium appears to have led to the relief of otherwise intractable symptoms. Gastrointestinal hemorrhage from esophageal varices has been ascribed to extensive hepatic sarcoidosis with secondary fibrosis and portal hypertension.

**SUMMARY**

A case of sarcoidosis with massive hemorrhage from atypical gastric ulcerations is reported. Epithelioid-cell granulomas were found in the stomach, gastric lymph nodes, and liver. The differential diagnosis of the granulomatous gastritides is discussed.

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SARCOID ULCERS


