Adenocarcinoma of the Stomach in Association with Menetrier's Disease

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Abstract. Menetrier's disease is an uncommon lesion which may have malignant potential. This report documents a case of gastric malignancy arising in a patient with long-standing Menetrier's disease and emphasizes that close follow-up of patients with this condition is necessary because of the possible development of gastric malignancy.

Key words: Stomach, rugal hypertrophy — Stomach, adenocarcinoma — Stomach, Menetrier's disease.

Giant hypertrophy of the gastric mucosa was originally described as a distinct pathologic entity by Menetrier in 1888 [1, 2]. The original monograph included one case of "sheet-like polyadenomas" with malignant transformation. A number of other cases of coexistent Menetrier's disease and adenocarcinoma of the stomach have been reported, suggesting the possibility of malignant potential in this condition. We recently had the opportunity to examine a patient with Menetrier's disease who subsequently developed gastric adenocarcinoma.

Case Report

A 51 year old white man first came to his physician with abdominal distress in 1965. Barium meal examination at the time showed enlargement of rugae involving the gastric fundus and body (Fig. 1A). He was treated conservatively and remained relatively symptom-free for 12 years. Roentgen studies in 1976 showed no change in the appearance of the enlarged gastric rugae (Fig. 1B). During the winter and spring of 1977, the patient noted intermittent epigastric pain and a 6 to 7 lb weight loss. He subsequently experienced one episode of hematemesis followed by 3 days of melena and was then referred to our institution for evaluation.

Physical examination disclosed mild tenderness in the mid-epigastrium but no palpable masses. Laboratory evaluation included a hemoglobin level of 10.7 gm/100 ml, reticulocyte count of 3.6%, and total serum protein of 5.5 gm/100 ml with an albumin level of 3.10 g/100 ml. Urinalysis revealed only a trace of protein. Carcinoembryonic antigen was elevated at 6.7 ng/ml. Gastric analysis disclosed absence of hydrogen ion in the fasting basal state, although acid secretion was present following histamine stimulation. Liver function studies, sulfer-colloid liver-spleen scan, and chest radiograph were normal. A barium meal study showed large rugae involving the gastric fundus and body. In addition, a 6-cm mass involved the greater curvature (Fig. 2A). Ulceration was not evident. Endoscopy confirmed the presence of large gastric rugae, but because of distortion from the enlarged mucosal folds a mass was not appreciated. A superficial biopsy revealed nonspecific chronic inflammation and was inadequate for definitive diagnosis.

At operation the mass was palpable along the greater curvature and a metastatic nodule was noted on the liver surface. Pathologic examination of frozen sections from the mass indicated adenocarcinoma, and a segmental gastric resection was performed.

Pathologic examination of the resected stomach showed a 6-cm, fungating, ulcerated mass along the greater curvature. Surrounding the mass were enlarged gastric rugae with multiple polypoid-like structures along the mucosal surface (Fig. 2B). Microscopic sections of the mass showed disorganized glandular epithelium with mitotic figures and pleomorphic nuclei deeply invading the underlying muscularis and approaching the serosal surface. Sections of gastric folds demonstrated thickened gastric mucosa, increased length and tortuosity of gastric glands, and dilatation of the crypts in some areas. A diffuse infiltrate of chronic inflammatory cells was present within the lamina propria and adjacent mucosa. These features were consistent with the entity Menetrier originally described as "sheet-like polyadenomas" with associated adenocarcinoma.

Discussion

The question of malignant potential in Menetrier's disease has been debated in the literature since Menetrier described the first case.

Menetrier observed that most cases of this entity evolve to a certain degree, become stationary, and persist as a benign lesion. He felt, however, that the
hypertrophic lesions were susceptible to further evolution and malignant transformation. Bartlett and Adams [3] in 1950 reported a case of Menetrier’s disease and commented that the large hyperplastic polyps should be regarded as potentially precancerous on the basis of metaplastic changes observed.

In 1954 Palumbo et al. [4] reviewed 4 cases of giant hypertrophic gastritis showing the progressive changes of inflammation, hyperplasia, anaplasia and mucosal infiltration. Their fourth case demonstrated histologic findings consistent with Menetrier’s disease and two areas of ulceration. The epithelium bordering these ulcers showed tumor-like proliferation with glandular invasion of submucosa and muscularis. They hypothesized that these multiple areas of carcinoma illustrated the malignant potential of Menetrier’s disease. Chusid et al. [5] subsequently reviewed 12 cases of carcinoma of the stomach associated with various forms of hypertrophic gastritis. They added a case of giant rugal folds progressing to multiple polyposis and carcinoma. Similar cases have been described [6–10]. Two additional case reports described localized Menetrier’s disease with concurrent adenocarcinoma arising at a separate focus in the stomach [11, 12]. These authors felt that the association was coincidental. Menetrier’s disease has also been reported in association with carcinoma of the pancreas [13], multiple endocrine adenomas [14], and hepatocellular carcinoma [15].

Although the exact occurrence of Menetrier’s disease in the general population is difficult to determine, on the basis of pathologically verified reported cases it appears to be an uncommon condition [16]. At least 20 cases of associated carcinoma have been