Ménétrier's disease and trichobezoar of stomach—an unusual association

A. K. CHAKRABARTI
M.D.

K. JOSHI
M.D.

T. D. SINGH
M.D.

A. K. MALIK
M.D.

Departments of Pathology and Gastroenterology, Postgraduate Institute of Medical Education and Research, Chandigarh-160012, India

Summary

Hypertrophic gastropathy (Ménétrier's disease) is a clinical and pathological curiosity. It is often seen in association with a variety of other disease processes. The authors here present a previously undescribed association of Ménétrier's disease with trichobezoar in the stomach and small intestine.

KEY WORDS: Ménétrier's disease, trichobezoar, stomach.

Introduction

In 1888, Ménétrier described a group of patients with enlarged gastric rugae due to mucosal hypertrophy which involved part or whole of the stomach giving an appearance of cerebral convolutions. The enlarged rugal folds are not attributable to lymphoma, infiltrative carcinoma or non-neoplastic diseases such as syphilis, tuberculosis and sarcoidosis. It is this condition which has been termed Ménétrier's disease or hypertrophic gastropathy (Scharschmidt, 1977). The cause of the disease is unknown. It is rarely familial and has no regular association with other diseases, although an association with caustic ingestion and bile reflux has been described in some cases (Catanzaro, Weeks and Kafka, 1962). A combination of trichobezoar and Ménétrier's disease has so far not been described. The significance of this association, though not understood, is briefly discussed.

Case report

A pregnant 24-year-old female was admitted in November 1980 with a history of mild colicky pain in the upper abdomen, vomiting and a gradually increasing mass in the upper abdomen for 5 months. Pain was relieved by vomiting.

Examination revealed a dehydrated and moderately anaemic patient, with an irregular mobile, non-

pulsatile mass of the shape of the stomach palpable in the upper abdomen and umbilical region. Another mobile mass of the same consistency was felt in the right iliac fossa. Due to her poor general condition surgery could not be undertaken. Abdominal aspiration revealed foul-smelling fluid. She died of septicemia 7 days after admission.

Autopsy findings

There was a 'J-shaped' mass in the stomach (Fig. 1) composed of coiled black hair measuring 25 × 10 cm and weighing 1900 g. The body of the stomach close to the greater curvature showed multiple prominent rugal folds with well-circumscribed, circular nodules 2 cm in diameter (Fig. 2).

Histologically, the nodular lesions showed elongated tortuous gastric pits with superficial gastritis, hyperplasia of mucosal cells, and cystically superficial gastritis, hyperplasia of mucosal cells, and cystically dilated glands with mucin in the lumen which penetrated into the submucosa. The muscularis mucosae separated the cystically dilated glands. There was moderate lymphomononuclear cell infiltration in the lamina propria. On the basis of these findings Ménétrier's disease was diagnosed.

Another separate trichobezoar (5 × 2.5 cm) was found 5 cm proximal to ileo-caecal valve (Fig. 3). There was irregular ulceration in the ileal mucosa at the same location with a small perforation. The latter had resulted in serositis and purulent ascites.

Discussion

The aetiopathogenesis of Ménétrier's disease, even after almost a century of its first description, remains uncertain. Various causative agents such as physical irritants, dietary factors, allergic reactions, bacteria and their toxins, neurogenic and congenital factors

FIG. 1. J-shaped trichobezoar in the stomach (25 × 10 × 10 cm) and a smaller one in the small intestine (5 × 2.5 cm).

have been considered in the pathogenesis of the disease, but none proven (Fieber, 1955). Association of the disease with Zollinger-Ellison syndrome and multiple endocrine adenomata has been described, where the change in the gastric mucosa is attributed to stimulation from a gastrin secreting tumour or other neurogenic or hormonal influences (Kenney, Dockerty and Waugh, 1954). Although the detailed pathogenesis is unknown, there appears to be a common factor of some source of intense stimulation to the growth of the gastric mucosa. By detailed

histological analysis, Butz (1960) postulated that the histological changes develop from disruption and loss of continuity of the muscularis mucosae combined with hyperplasia of epithelial cells. The pits undergo metaplasia to mucous secreting cells, become cystically dilated and herniate through the muscularis mucosae.

Malignant transformation as well as spontaneous remission of Ménétriér's disease has been described (Chisud, Hersh and Colcher, 1964; Schindler, 1965; Lesser, Falchuk and Singer, 1975).

Trichobezoar causing the complications of nutritional oedema, gastric perforation and acute intestinal obstruction are well documented (Osmond and Price, 1951; Wine, 1957). Intestinal trichobezoar causing obstruction is well known but perforation is rare (Malhotra and Manchanda, 1957). Interestingly, our patient had the symptoms of gastric outlet obstruction complicated by perforation of the small intestine and peritonitis.

Gastric trichobezoar has also been described in association with multiple gastric polyps and malignant change in one of the polyps (Charache, Polayes and Behr, 1957). However, a combination of trichobezoar and Ménétriér's disease has so far not been described.

Although association between the two conditions in our case is perhaps coincidental, we suggest that Ménétriér's-like change in the gastric mucosa can possibly occur due to a number of stimuli including the physical and chemical irritant actions of a trichobezoar.

Acknowledgment

Authors are thankful to Dr Basil C. Morson, St Mark's Hospital, London for his opinion on histopathology of the gastric lesions.

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(Accepted 14 December 1982)