Haematemesis in Ménétrie’s disease

R. J. DICKINSON  
B.Sc., M.R.C.P.  
A. T. R. AXON  
M.D., M.R.C.P.

Gastroenterology Unit, The General Infirmary, Leeds LS1

Summary

Three patients with Ménétrie’s disease presented with massive haematemesis. One patient died. Ménétrie’s disease may be associated with gastrointestinal haemorrhage and although it is rare it should be borne in mind as a cause of upper gastrointestinal bleeding. In one patient an elevated serum gastrin was found and the possible significance of this is briefly discussed.

Introduction

Ménétrie’s disease, a recognized cause of protein-losing gastroenteropathy, is rarely considered as a cause of haematemesis. Three patients with Ménétrie’s disease seen at the General Infirmary at Leeds in whom massive haematemesis was the presenting complaint are described.

Case reports

No. 1

A 53-year-old woman was admitted with haematemesis. There was a past history of haematemesis 3 years previously. Investigations showed: haemoglobin 63 g/dl; serum albumin 39.4 g/l; serum calcium 2.20 mmol/l; gastroscopy revealed hypertrophic greater curve rugae with adherent blood clot but no evidence of ulceration; gastric function tests revealed a basal acid output of 0 mEq/hr and peak acid output 1632 mEq/hr (total); the gastric juice was notably mucus rich; serum fasting gastrin was 135 pg/l (upper value of 95% confidence limits 70 pg); 3 further haematemeses followed before a Billroth I gastrectomy was performed.

No. 2

A 53-year-old man was admitted with a massive haematemesis and melaena. Investigations showed Hb 8.3 g/dl. Laparotomy and Billroth I gastrectomy were performed soon after admission for continuing haemorrhage. Postoperatively he developed renal failure and died.

No. 3

A 48-year-old man was admitted to another hospital with massive haematemesis necessitating transfusion of 12 pints of blood. The patient gave a past history of recurrent abdominal pain and dyspepsia. No cause was found for the gastrointestinal haemorrhage. Investigations at the General Infirmary at Leeds included double contrast barium meal which, in spite of poor coating due to an excess of mucus, showed characteristic greater curve changes of Ménétrie’s disease. At endoscopy a diffuse superficial erosive gastritis was seen in association with hypertrophy of the rugae of the greater curve.

Examination of the excised stomachs of patients 1 and 2 confirmed the presence of giant rugae of the body of the stomach with sparing of the antrum. Histology of the operative specimens of patients 1 and 2 and superficial endoscopic biopsies from patient 3 showed the characteristic microscopic appearances of Ménétrie’s disease with hyperplasia of the mucosa and elongation and cystic dilatation of the glands. All specimens also showed superficial erosive gastritis.

Discussion

A confident diagnosis of Ménétrie’s disease can be made for patients 1 and 2. The characteristic mucosal histology (Jones et al., 1972) was present in full-thickness sections of the operative specimens and in addition patient 1 was shown to have the hypochlorhydric mucus-rich gastric juice which is typical of the disease (Scott et al., 1975). In patient 3 only superficial biopsies were obtained which may be insufficient for a firm diagnosis (Jones et al., 1972) but the clinical and investigational findings make this diagnosis likely.

Ménétrie’s disease is rarely cited as a cause of haematemesis, although this complication is recognized (Palumbo, Rugtiv and Cross, 1951) and should be considered in any case of haematemesis in which rugal hypertrophy is found at endoscopy or barium meal. Bleeding probably occurs as a result of superficial gastritis (Jones et al., 1972) and although it may cease spontaneously, emergency surgery may be necessary in the face of continued life-threatening haemorrhage. That one of these patients died underlines the potential severity of the complication.
The cause of the disease is unknown. A lack of the trophic effect of gastrin has been suggested (Scott et al., 1975), but serum gastrin in patient 1 was high and was within the range that might be expected for patients with gastritis with antral sparing, which may occur in Ménétrier's disease (Jones et al., 1972) and hypochlorhydria (Korman, Strickland and Hansky, 1971). The authors are not aware of other reports on gastrin levels in Ménétrier's disease and consider that this finding merits further study.

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References


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R. J. Dickinson and A. T. Axon

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