MEDICAL MEMORANDA

Pyloric Stenosis Presenting with Halitosis

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The cardinal features of pyloric stenosis are vomiting and abdominal pain, though both may be absent even in the presence of severe stenosis (Palmer, 1963). Nevertheless, gastric stasis is always present and may result in bacterial overgrowth with fermentation of retained food. This aspect of the syndrome has attracted scant attention, though two workers have noted that vomiting may be accompanied by foul-smelling eructations (Hawkins, 1963; Avery Jones *et al.*, 1968). A bad taste in the mouth has also been mentioned as a late symptom (Haubrich, 1963). We describe two patients in whom the characteristic symptoms of pyloric stenosis were absent and who presented with halitosis and malodorous flatulence. Both had an underlying gastric carcinoma.

Case Reports

Case 1.—This patient, a woman aged 47, had longstanding Crohn's disease with diffuse jejunoileal involvement. She was also introspective, with a neurotic dependence on vitamin preparations. She presented at the Queen Elizabeth Hospital, Birmingham, with a six-month history of flatulence accompanied by epigastric rumblings and complained that her "breath smelt like rotten eggs." Her diarrhoea had increased but her weight remained steady. She had vomited only six times in the previous six months. Examination showed a malnourished woman with epigastric distension, a gastric succussion splash, and increased bowel sounds. No other abnormality was detected. The haemoglobin was 13.9 g/100 ml and the E.S.R. (Westergen) 45 mm in the first hour (previously the E.S.R. had been 18 mm in the first hour). Urea, electrolytes, proteins, calcium, phosphorus, and the chest x-ray picture were normal. A short course of tetracycline instantly relieved her halitosis, which then slowly returned over the next two months. During that time a barium-meal examination showed pyloric stenosis with no apparent cause. Laparotomy (Professor G. Slaney) showed a small adenocarcinoma of the antrum 4-5 cm from the pylorus which was not apparently obstructing the outlet. The carcinoma extended through the mucosa to the serosal surface. Local lymph nodes were not affected. There was no evidence of Crohn's disease of the stomach but inactive Crohn's disease of the jejunum and ileum was noted. Partial gastrectomy was performed. The postoperative course was uneventful.

Case 2.—A man aged 67 who had previously been well presented at the Worcester Royal Infirmary with an 18-month history of

halitosis and objectionable flatulence, "as if the wind had come from the other end." Other symptoms included anorexia, epigastric swelling, nocturnal rumblings, and diarrhoea. He did not complain of vomiting at any stage but had lost 2 stone (12.7 kg). He was a fit-looking man and apart from epigastric distension the abdomen appeared to be normal. Haemoglobin was 14.4 g/100 ml, E.S.R. (Westergen) 3 mm in the first hour, and urea, electrolytes, and chest x-ray appearances were normal. Barium-meal examination showed pyloric stenosis. Gastroscopy confirmed the presence of an enormous foul-smelling gastric residue with a narrow pylorus and deformed nodular folds which, when biopsied, proved to be adenocarcinoma of the stomach. Laparotomy showed that the tumour had spread to local lymph nodes. Partial gastrectomy with removal of the nodes was performed (Mr. C. W. O. Windsor). The postoperative course was uneventful.

Comment

Classically pyloric stenosis presents with a previous history of duodenal ulceration followed by the gradual onset of vomiting. The vomit characteristically consists of large quantities of undigested food. The presence of food remnant in the vomit several days after being eaten is typical. If ulcer pain has been present it often changes to a dull ache with a feeling of epigastric fullness and rumbling as the disease progresses. Weight loss and anorexia are fairly constant features. In many cases there are no physical signs but visible peristalsis, abdominal distension, and a gastric splash are pathognomonic.

Halitosis unaccompanied by vomiting has not previously been described. In case 1 the dramatic response to tetracycline suggests that the halitosis was related to fermentation of the gastric contents by bacterial overgrowth. Furthermore, both patients had diarrhoea instead of the more usual constipation and the motions returned to normal on antibiotics in case 1. Diarrhoea occurs in 6.5% of patients with pyloric stenosis (Kreel and Ellis, 1965) and is also a feature of the blind loop syndrome (Dyer and Hawkins, 1972). Bacteria may induce diarrhoea through several mechanisms but the precise pathogenesis in pyloric stenosis has yet to be settled.

It is often difficult to find a reason for halitosis. Some patients have minor local causes in the oropharynx and others are merely neurotic, as indeed was our first patient. Nevertheless, we suggest that the symptom may occasionally herald serious disease.

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