Neoplastic autovagotomy causing gastric stasis

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Summary
Neoplastic autovagotomy causing atonic gastric stasis is extremely rare. Two cases are reported in which marked gastric stasis was complicated by a bezoar and a gastric volvulus respectively. Both cases were associated with a left hilar bronchial carcinoma, and malignant invasion of the vagus nerve may have been the underlying cause.

Introduction
Autovagotomy causing gastric stasis appears to be a rare occurrence. In diabetes mellitus, it is well recognized that autonomic dysfunction may lead to atonic gastric dilatation (Rundles, 1945). However, direct vagus nerve involvement with malignant disease is extremely rare and there have been only 2 previous reports in the literature (Kirks and Szemes, 1971; Allan, Willson and Lee, 1977).

Two cases of bronchial carcinoma are presented in which neoplastic autovagotomy is postulated as being the underlying cause of marked gastric stasis. This led to the formation of a gastric bezoar in one case and a gastric volvulus in the other.

Case 1
In February 1973, a 73-year-old retired farmer was investigated for dyspnœa. A plain chest film suggested a left hilar mass but tomography, sputum cytology and rigid bronchoscopy were normal. Examination had revealed a mitral pansystolic murmur and echocardiography confirmed mitral incompetence due to a ruptured chorda. He was commenced on digoxin and diuretics and remained well until 1976 when he presented with a 2-month history of dysphagia, postprandial fullness, vomiting stale food and weight loss.

Barium meal showed a distended atonic stomach containing a large amount of food residue including some lead shot (Fig. 1). The pylorus and duodenum appeared normal although there was some delay in gastric emptying. These appearances were thought possibly to be due to a gastric bezoar and he was admitted for further investigation. At endoscopy, there was gross gastric residue with large masses of partially digested food. At laparotomy, the stomach was grossly distended and atonic but the pylorus was entirely normal with no evidence of scarring or narrowing. The duodenum was mobilized and a pyloroplasty performed, following which there appeared to be free flow into the duodenum.

The postoperative chest film showed enlargement of the left hilar mass (Fig. 2) and oat cell carcinoma cells were found in the sputum. Postoperatively, there was evidence of inappropriate secretion of antidiuretic hormone (serum sodium 112 mmol/l; plasma osmolarity 247 mmol/l; urine osmolarity 740 mmol/l). This responded to fluid restriction. Skull X-rays were normal and hepatic and bone scans showed no evidence of metastatic disease. A course of local radiotherapy (2340 rad in 6 fractions over 2 weeks) was given. This was followed by cyclical chemotherapy consisting of CCNU* 50 mg/m², cyclophosphamide 500 mg/m² and methotrexate 40 mg/m² as part of a MRC study. This caused considerable improvement in the X-ray appearances and he has remained well at follow-up for 18 months apart from developing complete heart block which has required insertion of a permanent pacemaker.

Case 2
A 76-year-old gentleman was admitted complaining of vomiting copious amounts of ‘coffee ground’ fluid accompanied by central constant abdominal pain. For the previous 3 months, he had suffered from anorexia, weight loss, back ache and a slight cough. He had had no previous dyspepsia and his bowels had been regular and he had had no urinary symptoms. He was a long-standing heavy smoker but had had no previous medical history of note.

On examination, he was ill, cachectic, had obviously lost weight and was dehydrated but had no lymphadenopathy or finger clubbing and his chest sounded clear. However, his abdomen was markedly distended with apparent ascites and scanty bowel

* 1-(2-chloroethyl)-3-cyclohexyl-1-nitrosourea.
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sounds. He was tender in the epigastrium but with no guarding or rigidity. Rectal examination was normal. Haemoglobin was 11.8 g/dl, ESR 82 mm/hr and WBC was 9100/mm. Chest X-ray showed some shadowing at the left hilum (Fig. 3) and abdominal X-ray showed no evidence of obstruction. Peritoneal paracentesis was performed and a large amount of straw-coloured fluid drained which was repeatedly negative on cytology. However, the following day, the fluid draining from the peritoneal drain suddenly became dark green and the patient's condition deteriorated. A gastrograffin swallow showed an extremely large dilated stomach (Fig. 4)

At laparotomy, an extremely dilated stomach was found which had undergone an 180° organoaxial volvulus and led to ischaemic necrosis in the upper half of the greater curvature and fundus where the line of the volvulus lay (Fig. 5). There were 2 perforations in this area with a subphrenic collection and an acutely infarcted spleen. No obvious primary cause of the dilatation of the stomach was found. A Pólya gastrectomy and splenectomy were performed.

Postoperatively, the patient progressed slowly and the shadowing on his chest X-ray was investigated. Tomography and sputum cytology confirmed this as being a squamous cell carcinoma of the bronchus. The patient was eventually able to be discharged for convalescence but had to be readmitted almost immediately and his general condition rapidly deteriorated and he died. Permission for post-mortem was refused.

Discussion

It is now well recognized that truncal vagotomy causes not only decreased acid secretion but also delayed gastric emptying and stasis (Dragstedt and Owens 1943; Dragstedt, Camp and Fritz, 1949; Schlicke 1963; Colmer, Owen and Shields, 1973; Donovan, 1976). For this reason, truncal vagotomy is always combined with a drainage procedure – either pyloroplasty or gastroenterostomy.

Fig. 1. Barium meal of patient 1 showing dilated stomach with food residue and lead shot.
In the first case, the gastric stasis was complicated by the formation of a bezoar as were the 2 previously reported cases of autovagotomy. Gastric bezoars have been reported following vagotomy even when combined with a drainage procedure (Moseley 1967; Rippin and Orda, 1972; Mir and Mir, 1973). Their formation is thought to be due to reduced gastric mobility with delayed emptying and stasis and reduction in acid output. The stomach becomes so distended and atonic that it is incapable of expelling its contents even through an adequate stoma.

The second case was complicated by an acute organo-axial gastric volvulus with strangulation and perforation. This in itself is a rare occurrence (Tanner, 1968) and has a high mortality (Wastell and Ellis, 1971). It is suggested that long-standing gross gastric dilatation following autovagotomy had led to an acute volvulus which had precipitated the sudden deterioration in his condition. Perforation of the stomach followed strangulation due to the acute volvulus and explained the free gastric contents which appeared from the peritoneal cannula. An acute volvulus occurring in this way has not previously been reported following vagotomy, although as Dalgaard (1952) pointed out, distension of the stomach predisposes to volvulus by approximating the pylorus and the cardia, and has been observed after a large meal.

Diabetic autonomic neuropathy may cause abnormalities of sweating, sphincter and bladder function and gastrointestinal motility, in particular gastric dilatation (Rundles, 1945; Howland and Drinkard 1963).

Direct vagal involvement with malignant disease may occur quite frequently but is probably seldom
Fig. 3. Chest X-ray of case no. 2 showing left hilar mass.

Fig. 4. Gastrografin swallow of case no. 2 showing an extremely dilated stomach and cannula draining peritoneal cavity.
clinically significant. Carcinoma of the upper oesophagus involving the sensory branches of the vagus can cause pain (Hoover, 1938) and it is well recognized that involvement of the recurrent laryngeal branch of the vagus causing vocal cord paralysis may occur with bronchial carcinoma (Henderson, Bozko and van Nostrand, 1974). However, complete autovagotomy appears to be a rare occurrence and there are only 2 previous reports in the literature (Kirks and Szemes, 1971; Allan et al., 1977). One case was an oesophageal carcinoma and the other a bronchial carcinoma and both were complicated by bezoar formation and, at post-mortem, evidence of extensive perineural invasion of vagal branches by neoplastic tissue was obtained.

Below the level of the lung roots, both vagus nerves form an extensive oesophageal plexus (Last, 1973). It would appear, therefore, that for autovagotomy to occur, extensive involvement by tumour of either both trunks or the whole of this plexus would be necessary. This may explain why autovagotomy is not a common occurrence (Allan et al., 1977).

Although there is no histological proof that neoplastic autovagotomy was responsible in either of the present cases, it seems more than likely. In both cases, there was radiographic and operative evidence of gross gastric distension in the presence of a normal pylorus. The left hilar masses in each case were shown to be bronchial carcinomas which, in one case, has responded to treatment.

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References