Achalasia-like disturbance of oesophageal motility following truncal vagotomy and antrectomy

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Summary

This report describes a patient who developed radiological and manometric evidence of an achalasia-like disturbance of oesophageal motility following trans-abdominal truncal vagotomy and antrectomy. The patient presented with severe dysphagia which spontaneously resolved after 8 weeks. Subsequent barium swallow and oesophageal manometry demonstrated a return to normal function.

KEY WORDS: achalasia, oesophagus, vagotomy, antrectomy dysphagia.

Introduction

Transient dysphagia has been described following truncal and selective vagotomy for peptic ulceration. There are considered to be 3 main aetiologies, namely haematoma, oesophagitis and lower oesophageal sphincter spasm. In the latter instance barium swallow may show the changes of achalasia, but previous reports have described either normal oesophageal manometry, or lower oesophageal sphincter spasm only. The oesophageal manometry in this patient was unique in its apparent resemblance to true achalasia.

Case report

A 41-year-old man presented with an 8-year history of recurrent epigastric pain and haematemesis. In 1975 he uneventfully recovered after a proximal gastric vagotomy for duodenal ulceration. He remained asymptomatic for 3 years, but his symptoms then recurred and were incompletely controlled by antacids and cimetidine. A barium meal showed a normal oesophagus and stomach and a deformed duodenal cap. Fibreoptic endoscopy confirmed a normal oesophagus and revealed a small

lesser curve gastric ulcer and a recurrent duodenal ulcer. Multiple biopsies from the gastric ulcer revealed chronic inflammatory changes only. Gastric studies demonstrated a basal acid output of 10 mmol/hr and, with insulin-induced hypoglycaemia, a maximum acid output of 31.8 mmol/hr. A gastrin assay was normal (less than 5 pmol/litre). In view of his history, severity of symptoms and high acid output, truncal vagotomy and antrectomy were performed. The patient made an uneventful recovery and was discharged home.

He was re-admitted 2 weeks later, complaining of total painless dysphagia of 48 hr duration. A barium swallow showed changes typical of achalasia, with a dilated lower oesophagus above a beaked, narrowed oesophago-gastric junction and unco-ordinated synchronous contractions of the lower two-thirds of the oesophagus (Fig. 1a). Oesophageal manometry was performed using the Andorfer hydraulic capillary infusion system and demonstrated a lower oesophageal sphincter pressure of 50 mmHg (normal range 15-25 mmHg) with incomplete relaxation on swallowing, and synchronous non-peristaltic activity in the body of the oesophagus (Fig. 2). Oesophagoscopy revealed a smooth stricture at the gastro-oesophageal junction with normal mucosa. This was readily dilated to a 24 French gum elastic bougie. After oesophageal dilatation he was able to swallow a soft diet and was discharged home. His dysphagia resolved after 8 weeks and he rapidly regained weight. Subsequent barium swallow (Fig. 1b) and oesophageal manometry (Fig. 3) demonstrated a return to normal function. He remains asymptomatic and well one year postoperatively.

Discussion

Post-vagotomy dysphagia was first described by Moses (1947). The incidence of dysphagia after vagotomy has since been variously reported as 1% to

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FIG. 1. Barium swallows. (a) Achalasia-like appearance, (b) return to normal.

FIG. 2. Oesophageal manometry tracing—achalasia-like changes. Note high sphincter pressure and incomplete relaxation on swallowing, in association with non-peristaltic contractions. LOS = lower oesophageal sphincter, WS = wet swallow, DS = dry swallow.

FIG. 3. Oesophageal manometry tracing—return to normal. Note normal sphincter pressure, reflex relaxation with swallows, and peristaltic contractions. LOS = lower oesophageal sphincter, WS = wet swallow, DS = dry swallow.

33%, the largest series indicating an incidence of 1% (Guillory and Claggett, 1967). If pre-existing oesophageal problems are excluded, the causes of post-vagotomy dysphagia may be classified as: (1) Perioesophageal oedema or haematoma with rare progression to a constricting fibrous ring requiring surgical intervention (Bruce and Small, 1959; Postlethwaite, Kim and Dillon, 1969; Spencer, 1975; Suleiman, Maglad and Hobsley, 1979); (2) Postoperative reflux oesophagitis, which is more frequently seen if concomitant gastrectomy necessitates use of a naso-gastric tube (Beal, 1948; Clarke, Penre and Ward, 1965; Johnson, 1965) and (3) Spasticity of the lower oesophageal sphincter due to vagal denervation, which radiologically mimics achalasia (Moses 1947; Harris and Miller, 1960). Typically, this last group presents with painless dysphagia commencing 7 to 14 days postoperatively (Alexander-Williams and Woodward, 1967). Vagal denervation may explain the higher incidence of dysphagia after trans-thoracic vagotomy (Wirthlin and Malt, 1972). Carveth, Schlegel and Code (1962) demonstrated that denervation of the lower oesophagus in dogs is only achieved by section of the vagi at the level of the lung hila. It is unknown if this occurs in man. The analogy with achalasia has been challenged by Silber (1969), who demonstrated lower oesophageal spasm, but normal peristalsis in the oesophageal body, in 15 patients. Edwards (1970) was also unable to demonstrate any neurogenic abnormality in 8 patients with dysphagia following truncal vagotomy. The mechanism of this transient complication of vagotomy is unexplained, but may be due to partial vagal denervation of the lower oesophagus. The expected immediate effect of denervation may not present for some days, because the patient is deprived of a normal diet in the postoperative period. In contrast to classical achalasia, where there is diminution or

absence of ganglion cells in Auerbach's myenteric plexus, the myenteric plexus is likely to remain intact. Little is known about the effect of gastrin, or its withdrawal, on the denervated or partially denervated smooth muscle of the lower oesophagus, and this may play a role when antrectomy accompanies vagotomy.

Interpretation of manometry in such cases requires careful analysis as the lower oesophageal sphincter region is oedematous and may obstruct the flow of fluids, not only from the oesophagus into the stomach, but also from the manometry tube, giving an impression of a raised lower oesophageal sphincter pressure. If the lower oesophagus is full of fluid the manometry tube orifices will lie in a common cavity and apparent synchronous non-peristaltic activity will be recorded. The barium swallow and oesophageal manometric tracing obtained in this patient both showed characteristics typical of achalasia. However, the manometry tracing suggested some peristalsis in the upper oesophagus, with a resting pressure less than intragastric pressure, and this mitigated against a diagnosis of achalasia. Spontaneous resolution of both symptoms and abnormal investigative findings excluded a true achalasia.

Conclusions

This paper describes a patient in whom both manometric and radiological data suggested an achalasia-like disturbance of oesophageal motility which appeared after a trans-abdominal truncal vagotomy and antrectomy. It is important to distinguish this condition from other oesophageal disorders as it is invariably self-limiting. The value of oesophageal manometry in demonstrating the precise nature of the abnormal postoperative oesophageal motility is stressed.

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