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Case report

Irradiation-induced motor disorder of the oesophagus

J A C THORPE, C OAKLAND, I P ADAMS, and H R MATTHEWS*

From East Birmingham Hospital and the General Hospital, Birmingham

SUMMARY This case report describes the late development of an achalasia-like disturbance of oesophageal motility following irradiation to the neck for a pharyngeal lymphosarcoma. The radiological, manometric and endoscopic findings are recorded as well as laboratory investigations showing evidence of complete vagal denervation.

Irradiation-induced oesophageal dysfunction is uncommon. The following case report demonstrates an achalasia-like disturbance of oesophageal motility caused by irradiation to the neck with resulting vagal denervation.

Case report

A 64 year old male presented in February 1981 with a two year history of intermittent dysphagia for both liquids and solids, of increasing severity. Attempts at swallowing were invariably accompanied by choking attacks and aspiration into the bronchial tree. Although his appetite was normal he had lost 12 kg (2 st) in weight.

In 1941, after a history of sore throat, nasal blockage, and high dysphagia, he was referred to an ENT consultant. A hard tumour was found growing from the posterior pharyngeal wall and biopsies were taken. Histology revealed a typical nasopharyngeal lymphosarcoma. He underwent a course of irradiation with a tumour dose of 3680 Röntgens, which was given in five treatments over 14 days. This was applied bilaterally from the mastoid process to the clavicle as well as in the anteroposterior plane. He was followed up for 20 years by the Christie Hospital and Holt Radium Institute and had no recurrences throughout this period.

Subsequently he developed progressive bilateral weakness in the muscles of his neck, pharynx, tongue, and in the muscles of his right hand. In 1976, electromyographic studies of his neck muscles confirmed bilateral damage to cervical nerve roots C3 and C4. This was considered to be due to radiation neuropathy or myelopathy and not to any progressive neurological disorder.

On admission, there was a severe cervical scoliosis with bilateral wasting of anterior and posterior neck muscles together with gross wasting of trapezius and to a lesser extent supraspinati. The right half of his palate, tongue, and right vocal cord were paralysed with impairment of speech. A liquid barium swallow showed a dilated oesophagus throughout its entire length (Figure). Barium swallow with a solid bolus demonstrated difficulties in bolus formation and initiation of swallowing. Transit of the bolus was markedly delayed in the upper two-thirds of the oesophagus with absence of peristaltic activity. There was no delay at the oesophagogastric junction. Oesophageal manometry was performed using the low compliance Arndorfer capillary-infusion technique. This demonstrated a hypotensive upper oesophageal sphincter (15 mm/Hg, normal range 70–140 mm/Hg) and a normotensive non-relaxing lower oesophageal sphincter (22 mm/Hg, normal range 15–30 mm/Hg). Peristalsis in the body of the oesophagus was absent. A Hollander test was performed. Insulin was administered at 0·2 units/kg, and an adequate hypoglycaemic response obtained (glucose basal 5 mmol/l, minimum 1·4 mmol/l). There was no increase in volume or acid concentration of stomach aspirates. Basal acid concentration 116 mmol/l, maximum acid concentration 107 mmol/l. These results indicate a complete vagotomy. Oesophagoscopy showed a dilated organ and a smooth narrowing at the oesophagogastric junction. A large

* Address for correspondence: H R Matthews, Department of Thoracic Surgery, East Birmingham Hospital, Bordesley Green East, Birmingham, B9 5ST.

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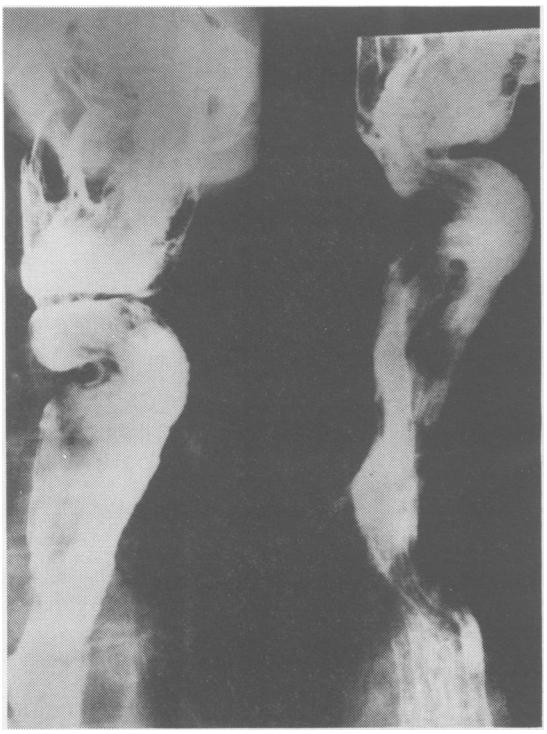

Figure *Barium swallow showing posteroanterior lateral projections of upper oesophagus demonstrating a diffusely dilated and atonic oesophagus.*

mercury bougie (20 mm) passed into the stomach without difficulty. There was no evidence of oesophagitis, stricture, or carcinoma.

These investigations indicated that the main functional problem was in the mouth and upper oesophagus and was not amenable to any form of surgical treatment. As the upper oesophagus was dilated and the upper oesophageal sphincter pressure low, it was felt that cricopharyngeal myotomy would confer no benefit.

Discussion

The term 'achalasia' originated from the Greek 'failure of relaxation' and was first coined in 1913 by Sir Cooper Perry.[1] Achalasia is a condition of unknown aetiology characterised manometrically by failure of relaxation of the lower oesophageal sphincter with synchronous or reduced motor activity in the body of the oesophagus. It is regarded as a disease of neuromuscular origin and histological studies demonstrate a reduction or absence of ganglion cells in Auerbach's plexus. Histological changes are also seen in the extraoesophageal vagus nerves which resemble Wallerian degeneration. A reduction of cell bodies has been identified in the dorsal motor nucleus of the vagus at necropsy.[2] Aperistalsis in the oesophagus after high vagal section was first produced in rabbits in 1839 by Reid *et al*, and confirmed by other workers in different animals.[1] It also occurs in Chagas's disease, which is caused by an infestation by *Trypanasomiasis crusi*. In the chronic form, megaoesophagus is one of the pathological features and histology shows loss of ganglion cells in Auerbach's plexus with some preganglionic changes in the vagus nerve and central nervous system.[3] Gastric secretory tests have demonstrated decrease of gastric secretion.[4] This may not necessarily be due to truncal vagal denervation alone, as there can be a decrease in acid output when the ganglion cells of the stomach are reduced. This is known to occur in Chagas's disease and in very severe achalasia.

In this patient radiation to the neck appears to have led to complete vagal denervation in addition to damage to the upper cervical spinal roots and other cranial nerves – for example, hypoglossal nerve. Other forms of damage to the vagus nerve can also produce achalasia-like syndromes – for example, malignant extension from carcinoma of the stomach, involvement of the vagus by enlarged inflammatory lymph nodes, and various neurological syndromes resulting from penetrating injuries to cranial nerves which were described during the first and second world wars – for example, Vernet's, Villaret's, and Sicard's syndromes. These cause similar clinical features to those seen in this case. There have been few studies describing the effect of radiation on the oesophagus, although failure of lower oesophageal sphincter relaxation has been noted clinically in patients with radiation oesophagitis.[5] In this case the area of irradiation was remote from the oesophagus and could not have been caused by direct damage to the oesophageal wall.

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