Acute thoracic inlet obstruction in achalasia of the oesophagus

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McLean, R. D. W., Stewart, C. J., and Whyte, D. G. C. (1976). *Thorax*, 31, 456–459.
Acute thoracic inlet obstruction in achalasia of the oesophagus. A case of acute thoracic inlet obstruction presenting as a rare complication of achalasia is described. The probable mechanism, diagnosis, and management are discussed.

Patients with achalasia tolerate considerable distension of the oesophagus remarkably well. Respiratory symptoms are usually due to pulmonary aspiration rather than to a space-occupying mechanism. Stridor due to tracheal compression is very rare. Bello *et al.* (1950) first described such a case, and Guistra, Killoraw, and Wasgatt (1976) described two cases. We now report a further patient with acute tracheal compression and thoracic inlet obstruction.

CASE REPORT

A 70-year-old woman was admitted to the Gynaecological Unit in February 1974 for repair of a procidentia. Achalasia of the oesophagus had been diagnosed radiologically 10 years previously when she had complained of a choking sensation after meals. Over the succeeding years her symptoms had been mild and intermittent except on two occasions when she had experienced severe respiratory distress with cyanosis; on each occasion her symptoms had resolved spontaneously. She had never admitted to any significant dysphagia and surgical treatment had been declined.

The chest radiograph before repair of the procidentia demonstrated a fluid level within a dilated oesophagus (Fig. 1).

In preparation for the operation a naso-oesophageal tube was passed, the patient having been pre-oxygenated, while still awake, and breathing spontaneously. Pancuronium bromide (Pavulon), 5 mg, and 1% methohexitone sodium, 60 mg, were given intravenously. Intubation was performed using an 8.0 mm tracheal tube. Anaesthesia was maintained using N₂O/O₂ (50%/50%) and inter-

mittent halothane (range 0.5% to 1%). Neuromuscular block was reversed with 2.5 mg neostigmine and 1.20 mg atropine. Extubation was uneventful, there being no postoperative stridor. The patient removed the oesophageal tube on arrival in the recovery ward.

Half an hour later she developed rapidly increasing respiratory distress with inspiratory and expiratory stridor. A tense bilateral swelling of the neck appeared, involving both anterior and posterior triangles. On each side it extended from the horizontal ramus of the mandible to the clavicle, and laterally from the trachea to the scalenus anterior. The neck veins were engorged with deep cyanosis of the face. The swelling was resonant on percussion; otherwise it resembled a very large goitre.

Apart from tracheal stridor the lung fields were clear on auscultation. The pulse became rapid and thready, the blood pressure falling to 50 mmHg systolic. An endotracheal tube was passed with difficulty. High inflation pressure was required to achieve adequate ventilation. Attempts to pass a suction catheter down the endotracheal tube failed, the tube appearing to be kinked about mid-tracheal level.

An erect chest radiograph (Fig. 2) showed that the oesophagus was grossly distended, this air-containing viscus being defined over the right hemithorax. There was a fluid level and a mottled opacity at the level of the carina. Above this the distended oesophagus passed in a sigmoid shape from the apex of the right hemithorax to the left side of the neck, also above and to the left at the level of cricopharyngeus. The lung fields were otherwise clear.

FIG. 1. *Radiograph of chest before operation.*

FIG. 2. *Radiograph of chest during acute symptoms.*

An emergency oesophagoscopy was carried out. The cricopharyngeal sphincter presented as a tight transverse and slightly crescentic slit, being displaced anteriorly by a tense swelling. Considerable pressure was required to pass a stiff catheter into the oesophagus. This produced a dramatic relief of symptoms, with rapid resolution of the neck swelling and the associated thoracic inlet obstruction. A barium swallow later confirmed a grade IV achalasia (Fig. 3). A Heller's cardiomyotomy was

FIG. 3. Barium swallow showing grade IV achalasia of the oesophagus (oblique projection).

carried out in May 1974. This was performed by the abdominal route. Anaesthesia was uneventful. The oesophageal hiatus was repaired by posterior suturing of the crus. The phrenico-oesophageal ligament was repaired, maintaining 5 cm of intra-abdominal oesophagus. Three oesophagogastric sutures, to accentuate the gastro-oesophageal angle, were added as an antireflux mechanism.

COMMENT

In the more severe grades of achalasia the oesophagus lengthens, often assuming a sigmoid

shape, as well as enlarging circumferentially. d'Abreu, Collis, and Clarke (1971) have drawn attention to incompetence and paralysis of the cricopharyngeal muscle in this condition. This allows the negative intrathoracic pressure to draw air into the oesophagus during inspiration. In this case there was gross dilatation and elongation of the oesophagus, extending behind the trachea and above and to the left of the cricopharyngeal sphincter. It would appear that the respiratory effort following anaesthesia allowed excessive air to be drawn into the oesophagus, causing distension, the cricopharyngeal sphincter acting as a flutter valve, thus allowing air entry during the more powerful inspiratory effort, and preventing its escape during expiration. Progressive distension of the oesophagus caused forward displacement, angulation, and compression of the trachea with obstruction, this being facilitated by the soft membranous posterior portion of the trachea.

The space-occupying effect on the neck veins produced the clinical picture of thoracic inlet obstruction and contributed to the low-output cardiac state.

The radiographic appearances in this case are characteristic, particularly the air-fluid level above a mottled opacity due to food and secretions. Radiological investigation plays an important part not only in confirmation of the state, but also in alerting the clinician to the diagnosis. On the chest radiograph a right-sided convex shadow may be seen extending from the mediastinum usually at mid-chest level. The margin may be curvilinear or scalloped. A fluid level may be noted in the erect radiograph. The stomach gas bubble is usually absent. In the presence of an obstructed, dilated, and tortuous oesophagus, air may be defined at the cervicothoracic level; this has a characteristic curvaceous pattern. The lateral radiograph may indicate compression and forward displacement of the larynx and trachea.

A useful sign, when present (stressed by Moser and McCuiston (1957)), is the presence of a crescentic air shadow high in the thorax. This is due to air which is trapped between the wall of the oesophagus and food and secretions within the oesophagus. Direct demonstration of the lesion entails the ingestion of opaque medium. The tortuous, dilated oesophagus and the beak-like smoothly tapered distal end of the viscus showing a slow intermittent milking of the medium into the stomach are diagnostic. Where there is danger of inhalation, dionosil or other absorbable medium should be used instead of barium. In cases of acute

tracheal compression, relief of symptoms is dramatic after decompression of the oesophagus. 'Blind' passage of a naso-oesophageal tube is likely to prove unsuccessful. In view of the tightness and displacement of the upper oesophageal sphincter, immediate oesophagoscopy with the passage of a catheter under direct vision is a safer and more certain method. Postoperative retention of a naso-oesophageal tube for 24 hours is advisable in patients with achalasia to prevent a complication such as occurred in this case. In view of the possibility of this complication, it is advisable to deal with the achalasia before contemplating surgery for non-urgent conditions.

We are indebted to Mr. S. W. Liggett, FRCS, FRCOG, under whose care this patient was originally admitted, for permission to publish this case.

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