

Previously treated oesophageal achalasia re-presenting with stridor

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Abstract

Achalasia is a motility disorder of the oesophagus that typically presents with dysphagia, regurgitation and chest pain. A rare presenting symptom is stridor. A case of previously treated achalasia re-presenting with stridor is described and associated imaging presented.

Key words: Oesophageal Achalasia; Airway Obstruction; Respiratory Sounds; Trachea

Introduction

Achalasia is a motility disorder of the oesophagus characterised by impairment of relaxation of the lower oesophageal sphincter and aperistalsis of the oesophagus. Patients usually present between the ages of 30 and 60 years with chest pain, resulting from the vigorous, non-peristaltic, simultaneous contractions of the oesophageal body. Later symptomatology includes dysphagia and regurgitation of retained solids and liquids due to oesophageal dilatation. A rare symptom of achalasia is stridor,^{1–8} secondary to tracheal compression. There has been only one previously reported case of treated achalasia re-presenting at a later date with stridor.¹ We report the case of a 78-year-old man presenting with intermittent stridor three years after oesophageal dilatation for achalasia.



FIG. 1

Chest radiograph showing a grossly dilated thoracic oesophagus.

Case report

A 78-year-old man presented with a history of progressive intermittent stridor. He had been diagnosed with achalasia three years previously for which he underwent successful treatment with oesophageal dilatation. Past medical history also included conservative treatment for peptic ulcer disease.

At presentation he was dyspnoeic with marked stridor. Chest radiography (Figure 1) showed gross dilatation of the upper oesophagus. Flexible nasendoscopy revealed an inflamed glottis with gross swelling posteriorly causing compression of the larynx. Intravenous steroids were commenced and his symptoms settled rapidly over the following 24 hours. A computed tomography (CT) scan (Figure 2) showed massive oesophageal dilatation with compression of the trachea within the superior mediastinum. Four days following admission he developed acute, severe abdominal pain, and owing to the previous history of peptic ulceration and current treatment with steroids, a perforated ulcer was suspected. Laparotomy confirmed the clinical findings, and in addition the oesophagus was found to be grossly dilated. The surgical

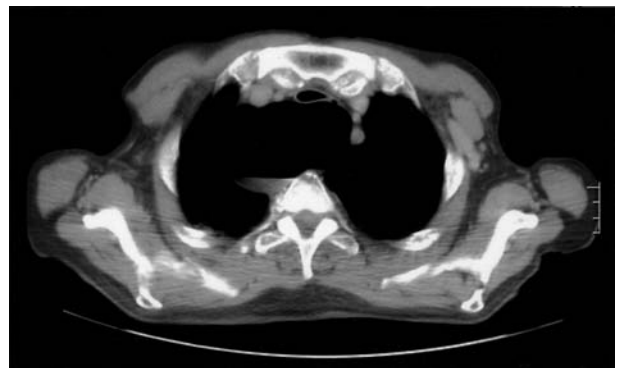


FIG. 2

CT thorax showing gross dilatation of the oesophagus causing compression of the trachea.

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intervention included patch repair of the perforation and a cardiomyotomy and antral wrap. The post-operative course was complicated by intermittent tachyarrhythmias, respiratory failure and subsequent multiple organ dysfunction syndrome (MODS). The patient died on the intensive care unit 12 days after admission.

- **This is a case report of a patient with achalasia of the oesophagus presenting with stridor**
- **The stridor in this case was secondary to tracheal compression and occurred following previously successful treatment with oesophageal dilatation**

Discussion

This case demonstrates that previously treated achalasia may re-present with serious consequences. Achalasia should be included in the differential diagnosis in all patients presenting with stridor, particularly in those with a past history of the disorder. There has been one previously reported case of achalasia presenting with stridor despite previous effective oesophageal dilatation.¹ The initial investigations in this case included a barium swallow, which showed cricopharyngeus spasm. A cricopharyngeus myotomy was performed and the patient remained asymptomatic at the time of publication.

In our patient the use of intravenous steroids caused resolution of the presenting symptoms. Previous cases in the literature have reported relief from the passage of a nasogastric tube;^{1,3-5} emergency oesophagoscopy;^{2,7-8} or from insertion of a cardiac needle into the neck swelling.⁶ On examination with flexible nasendoscopy our patient was noted to have inflammation of the glottis; the aetiology of this is unknown but chronic oesophageal reflux secondary to achalasia is a likely causative factor. As mentioned previously cricopharyngeal myotomy has been helpful for resolution of symptoms in other reported cases, but this is likely to enhance upper overspill and result in worsening of any intercurrent inflammation of the glottis. Therefore, in the presence of glottis inflammation, interventions other than cricopharyngeal myotomy should be considered.

Although the use of steroids helped in the acute management of our patient's symptoms, by relieving the inflammation of the glottis, they may also have been a contributing factor in the perforation of his duodenal ulcer, therefore we reiterate the need for caution when using steroids and advise regular re-evaluation of their continued requirement when used. The definitive treatment of achalasia should be prompt, by either oesophageal dilatation of the lower oesophageal sphincter or by surgical cardiomyotomy.

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