Megaoesophagus Presenting as Stridor

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Abstract

Acute airway obstruction is a rare presentation of megaoesophagus. Megaoesophagus is commonly caused by achalasia; however, we present a case of megaoesophagus caused by idiopathic dysmotility disorder. Decompression with a wide bore nasogastric tube avoided a scenario of difficult intubation or emergency front of neck access

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TITLE OF CASE

Megaoesophagus presenting as stridor

AUTHORS

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DESCRIPTION

A female presented to the Emergency Department with a four-day history of progressive breathlessness and intermittent coughing episodes followed by stridor. She reported no dysphagia or odynophagia. Examination revealed a marked inspiratory stridor but no respiratory compromise. Flexible nasolaryngoscopy showed no obvious supraglottic swelling; however, the larynx was rotated towards the right, which corresponded with her neck examination which revealed a soft palpable swelling along the left-hand side. Her past medical history included hypothyroidism, atrial fibrillation, and small bowel ischaemia requiring laparotomy and small bowel resection 4 years prior. A chest X-ray showed a markedly dilated oesophagus throughout its length which was shown to be chronic when compared to previous X-rays (Figures 1 & 2). Computed tomography revealed oesophageal dilatation causing mass-effect and compression upon the tracheobronchial tree and larynx. Tracheal narrowing was noted to be 3–4 mm in the upper mediastinum (Figure 3). She was treated with nebulised adrenaline and intravenous dexamethasone in the Emergency Department, which settled her stridor. She was admitted to the Otolaryngology Ward for observation overnight. Upon discharge, she underwent a barium swallow (Figure 4), demonstrating marked cricopharyngeal spasm causing significant luminal narrowing but no significant holdup of barium. The remainder of the oesophagus was chronically dilated and appeared atonic but there was prompt emptying into the stomach. The findings were atypical of achalasia, there were no obstructing lesions identified and it was felt that chronic dysmotility was most likely. She was followed up at 6 months and reported no further symptoms. A gastroenterology review suggested that the patient's presentation was due to an atonic hypo-motile oesophagus with a degree of dysmotility. The patient declined further investigations. One year following her initial presentation, she attended the Emergency Department with biphasic stridor and signs of respiratory distress. Flexible nasolaryngoscopy again identified a large swelling in the left hypopharyngeal compressing the larynx (Video 1). She was transferred to theatre for intubation +/- tracheostomy; however, during transfer, her stridor settled and compression on the larynx lessened (Video 2), allowing safe insertion of a wide-bore nasogastric tube under flexible nasolaryngoscopic visualisation via the non-compressed right pyriform fossa. Aspirating the air from the dilated oesophagus resulted in complete decompression of her larynx (Video 3) and the stridor disappeared. Following observation overnight, the nasogastric tube was removed, the patient commenced a soft diet and was discharged home. An alert has been put on her electronic patient record so that if she presents again with this problem, the team are aware of the successful management method described above which avoided the need for intubation or front of neck access. The patient has opted out of surgery to address her oesophageal condition as she is asymptomatic between episodes. Megaoesophagus is rare, most frequently presents with dysphagia and regurgitation of food and is most commonly caused by achalasia. A literature search identified only one previous case of obstructing megaoesophagus secondary to idiopathic dysmotility disorder; however, this case revealed the stridor to be secondary to an impacted food bolus, whereas our case did not demonstrate any obstructing lesions.[1-4]

LEARNING POINTS

Acute airway obstruction is a rare presentation of megaoesophagus Megaoesophagus is commonly caused by achalasia; however, we present a case of megaoesophagus caused by idiopathic dysmotility disorder Decompression with a wide bore nasogastric tube avoided a scenario of difficult intubation or emergency front of neck access

CONSENT STATEMENT

Approval from an ethics committee was not required in this case; however, verbal and written informed consent was obtained from the patient prior to submission for publication.

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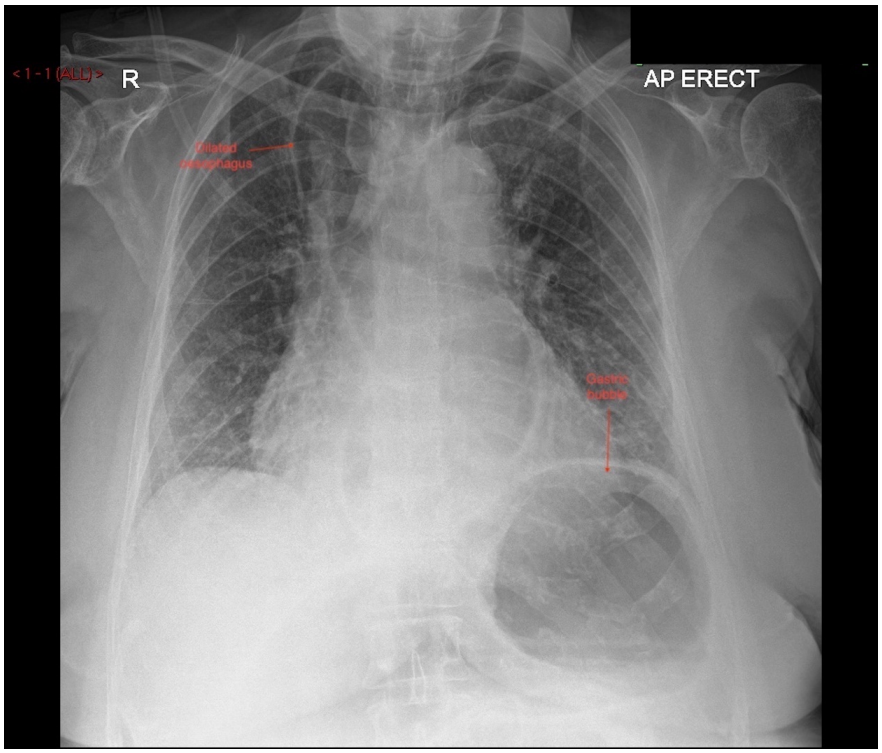
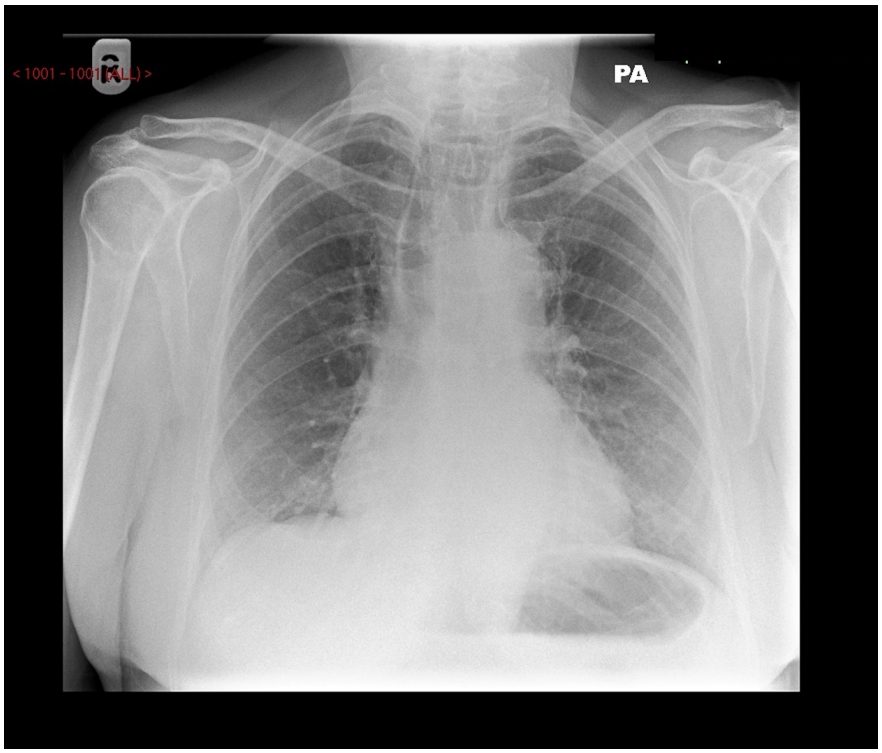
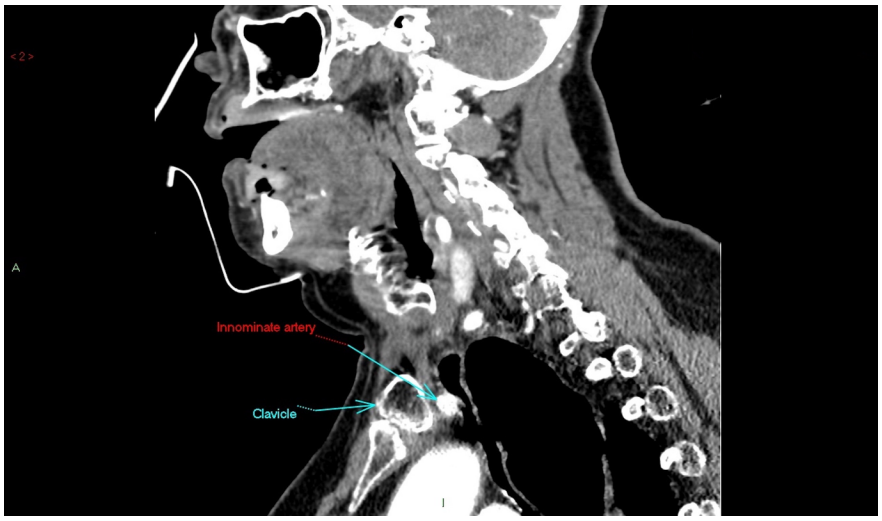
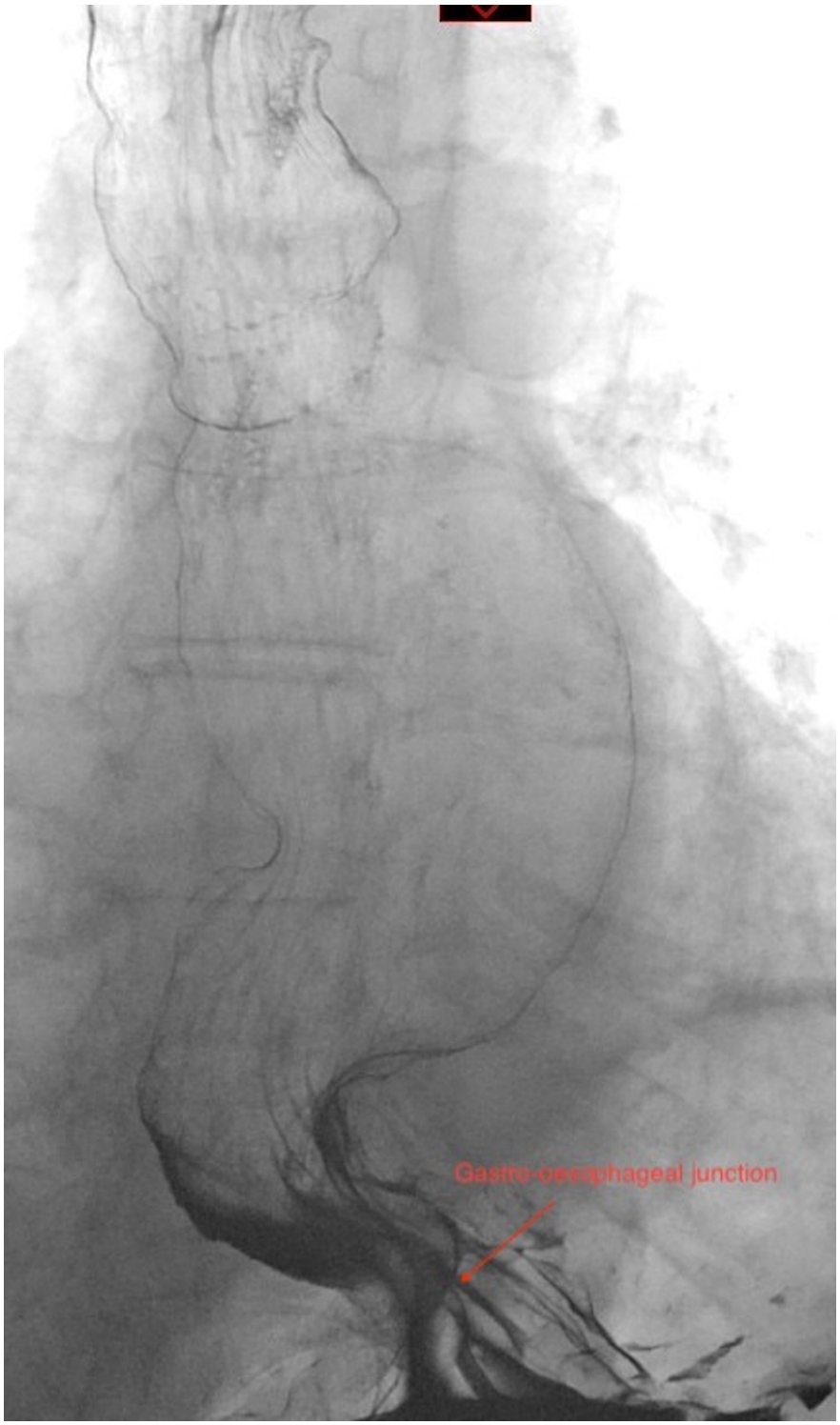
FIGURE/VIDEO CAPTIONS

Figure 1: A dilated oesophagus and the presence of a large gastric fundus air bubble were observed on chest X-ray. These findings would not be typical of achalasia where a fundal gas bubble would not be present. **Figure 2:** A previous chest X-Ray demonstrated longstanding dilatation of the oesophagus **Figure 3:** A contrast CT scan of the neck demonstrated the dilated oesophagus causing tracheal compression **Figure 4:** Barium swallow demonstrated a normal gastroesophageal junction, which excluded a diagnosis of achalasia **Video 1:** Flexible nasolaryngoscopy demonstrating a compressive swelling at the left hypopharynx. **Video 2:** Repeat flexible nasolaryngoscopy performed after transfer to theatre prior to any intervention showing slightly less compression. **Video 3:** Flexible nasolaryngoscopy showing a wide bore nasogastric tube inserted into the oesophagus via the right pyriform fossa to decompress the larynx.









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