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Tracheal compression relieved by cardiomyotomy

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Achalasia of the oesophagus is a rare cause of acute thoracic inlet compression.[1] We describe a case of previously undiagnosed achalasia presenting in a young man with symptoms consistent with tracheal compression of insidious onset, in whom the maximum flow-volume loop showed a pattern compatible with severe variable intrathoracic airway obstruction that improved after cardiomyotomy.

Case report

A 19-year-old man presented to the outpatient department with a two-year history of gradually increasing shortness of breath on exertion, noisy breathing, recurrent coughing, particularly at night, and a "crackly" retrosternal sensation on inspiration. Only on direct questioning did he admit to dysphagia, which had been present for about three years and which was associated with nocturnal regurgitation of food.

Physical examination showed a well-nourished man who was not breathless at rest. During tidal inspiration and expiration a soft low-pitched wheeze was audible, which became stridorous during deep breathing. The posteroanterior chest radiograph (fig 1) showed a convex right paramediastinal shadow extending from the sternoclavicular joint to the level of the eighth rib posteriorly. On the lateral view the tracheal air column was bowed anteriorly. Barium swallow examination showed that this displacement was a result of gross distension of the oesophagus from the cardia to the pharyngo-oesophageal junction, the appearances being consistent with achalasia.

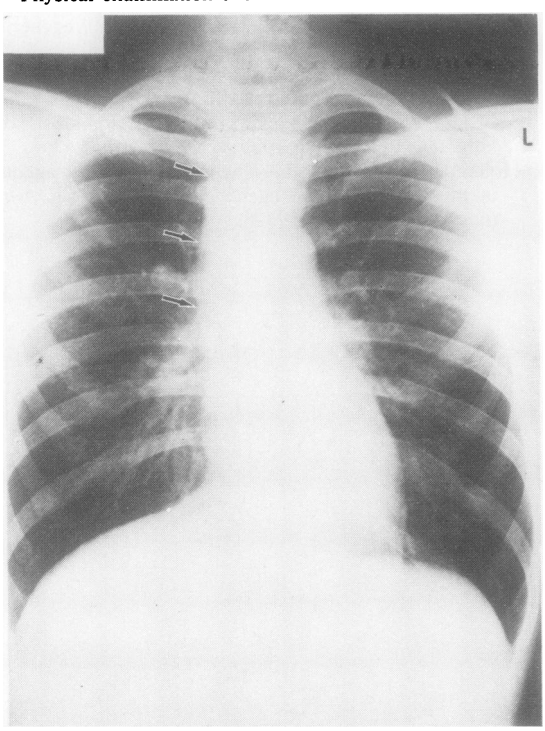

Fig 1 *Chest radiograph showing features suggestive of achalasia: right paramediastinal shadow (arrowed) and absent gas shadow in the fundus of the stomach.*

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Lung function testing carried out preoperatively on different days showed a severe obstructive impairment of ventilatory capacity, which did not alter appreciably after salbutamol inhalation: FEV_1 1·62 litres (41% of predicted value[2]), forced vital capacity 4·70 l (103%), FEV_1/FVC 34% (40%). Residual volume as assessed by helium dilution and single-breath transfer factor for carbon monoxide were both normal: 0·93 l (87%) and 34 ml/min/mm Hg (108%). The shape of the maximum expiratory-inspiratory flow-volume curve (fig 2) was consistent with severe variable intrathoracic airway obstruction.[3]

At endoscopy the oesophagus was grossly dilated and contained a large amount of turbid material. Bronchoscopy showed the lumen of the trachea to be ovoid in cross-section with considerable narrowing anteroposteriorly. After recovery from thoracotomy and cardiomyotomy (performed by Mr BA Ross) the patient gradually became less breathless and experienced less difficulty with swallowing. One year after operation he achieved the following normal spirometric values: FEV_1 4·14 l

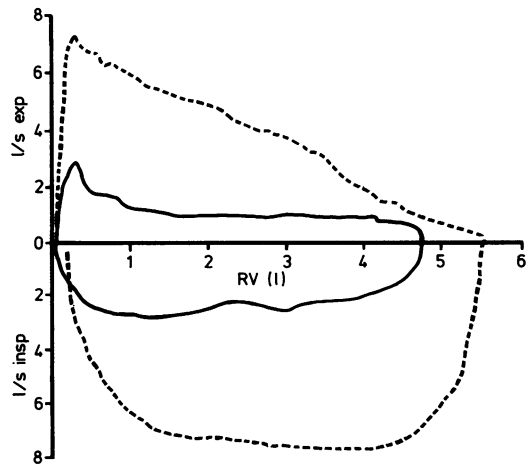

Fig 2 *Maximum expiratory-inspiratory flow volume loops before _____ and one year after . . . . . cardiomyotomy.*

(105% of predicted normal value[2]), forced vital capacity 5.45 l (119%), FEV_1/FVC 76% (89%). The appearance of the maximum flow-volume loop at this time was also considerably improved (fig 2).

Discussion

The association of achalasia with respiratory symptoms resulting from aspiration of the oesophageal contents has been known for many years.[4] Achalasia may also—though rarely—cause a syndrome of acute thoracic inlet compression, variously characterised by dyspnoea and stridor of sudden onset, swelling of the neck, and considerable dilatation of the oesophagus as shown on plain chest radiographs.[1 5 6] The notable features of our case were that the onset of dyspnoea and stridor was insidious and that although there were only slight changes on the plain chest radiograph tracheal compression was severe, as indicated by the degree of impairment of ventilatory capacity at presentation. In previous reports of acute stridor in achalasia[1 6] upper oesophageal distension by trapped air has been clearly instrumental in causing tracheal compression and it has been postulated that an incompetent cricopharyngeal muscle permits air to enter the oesophagus on inspiration but traps it in expiration by shutting off in the manner of a pinch-cock valve.[7] There was no clear radiographic evidence of oesophageal air trapping in our patient and we suggest that prolonged distension by food and fluid contents alone may have been sufficient to narrow the trachea by causing its forward displacement against the manubrium sterni. Improvement in maximum expiratory and inspiratory flow rate occurred gradually during the 12 months after cardiomyotomy. This may have been the result of slow restoration of normal tracheal geometry after the relief of oesophageal pressure. Repeated bronchoscopy to confirm this was not considered justifiable; but if the cartilaginous rings had been compressed for some years they are likely to have taken months to return to their normal shape.

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