Fatal haematemesis due to benign retrosternal goitre

D.R. Parker and A.H. El-Shabouiry

Department of Medicine, Prince Charles Hospital, Merthyr Tydfil, Mid Glamorgan, UK

Summary: The development of a goitre in the retrosternal space may result in many different symptoms due to local compression. We describe a case in which such a goitre resulted in full-thickness ulceration of the oesophagus, which presented as a fatal haematemesis. We believe that such a complication has not been previously reported.

Introduction

The development of a goitre may result in symptoms of local compression and this is particularly true in cases with retrosternal extension.1 We report a patient with a benign retrosternal goitre which caused erosion and ulceration of the oesophageal wall resulting in fatal haematemesis. To the best of our knowledge such a complication has not been reported previously.

Case report

The patient, a 74 year old female, initially presented 14 months prior to her fatal haematemesis with a 3 month history of general malaise, exertional and paroxysmal nocturnal dyspnoea and ankle oedema. She had had two partial thyroidectomies, in 1930 and 1940, and had sustained a left recurrent laryngeal nerve palsy. Clinical examination revealed thyroidec- tomy scars, a thyroid nodule (1 cm in diameter) in the left lobe, atrial fibrillation with a ventricular rate of 120 beats per minute and bilateral basal crackles.

Initial investigations showed normal serum urea, electrolytes and glucose and a normal full blood count. The ESR was 28 mm in the first hour. A postero-anterior chest radiograph revealed bilateral patchy lower lobe consolidation and a large mass occupying the superior mediastinum extending from the right hilum to the base of the neck (Figure 1).

A lateral view showed anterior tracheal deviation. Superior mediastinal tomography showed a large soft tissue mass with nodular calcification extending to both sides of the midline and confirmed the anterior tracheal shift. Thyroid function and serum calcitonin were normal but thyroid autoantibodies were strongly positive. A technetium thyroid scan and iodine-123 isotope scan showed patchy uptake in the neck presumably as a result of postoperative changes but no thyroid tissue was identified retrosternally on either of these scans. However, a technetium polyphosphate isotope bone scan did reveal increased uptake in the region of the mass. The thyroid nodule was biopsied via needle aspiration and histology revealed benign nodular colloid tissue with degenerative change and calcification. She was discharged home.

Correspondence and present address: D.R. Parker, B.Sc., M.R.C.P., University Department of Medicine, Bristol Royal Infirmary, BS2 8HW, UK.

Accepted: 18 February 1992

Figure 1 Postero-anterior chest radiograph showing superior mediastinal mass and tracheal deviation to right.
feeling well after treatment with digoxin, diuretics and antibiotics.

Fourteen months later she was readmitted with signs of cardiac failure. Soon after her arrival, she had a large haematemesis and was resuscitated successfully. Endoscopy revealed a large, fresh, pulsatile clot occluding most of the oesophageal lumen at 30 cm from the mouth. A provisional diagnosis of an aortic aneurysm or mediastinal neoplasm eroding the oesophagus was made but she suffered a second haematemesis shortly afterwards from which she could not be resuscitated. Post-mortem examination showed an oval nodular encapsulated mass 13 cm by 7 cm in diameter in the left upper mediastinum. This had eroded the oesophageal wall resulting in a 3 cm diameter ulcer. A 3 cm ulcer was also found on the medial surface of the upper lobe of the right lung. The mass was macroscopically benign thyroid tissue with focal calcification and this was confirmed on histological examination.

Discussion

Retrosternal goitre is known to cause several complications due to pressure on the structures in the narrow diameter of the thoracic inlet. These include tracheal compression, with or without stridor, Horner’s syndrome, thoracic duct obstruction, transient cerebral ischaemia due to a ‘steal’ syndrome and oesophageal compression.1–5 Oesophageal varices have been described as a complication of retrosternal goitre6 and in that case were thought to have developed because of obstruction of the superior vena cava andazygos vein as a result of fibrosis following previous goitre surgery. Blood thus flowed ‘downhill’ from the upper extremities, head, neck and thorax via mediastinal collaterals into oesophageal varices and then into the portal vein through the liver and inferior vena cava to the heart.

Such ‘downhill’ varices may bleed7 and thus should be considered in any condition causing superior vena cava obstruction, such as bronchogenic carcinoma, mediastinal fibrosis or retrosternal goitre.6 In our case, however, there was no evidence of varices at endoscopy or at post-mortem examination. A benign retrosternal goitre eroded the oesophagus causing exsanguination and we have been unable to trace a similar report in the literature. The patient had had two thyroidecotomies in the past and it is likely that postoperative adhesions and fibrosis formed a barrier in the base of the neck, so that the recurrent goitre developed in the superior mediastinum,8 producing the observed ulcers and unusual anterior tracheal displacement. In our patient, technetium and iodine-123 scanning of the thyroid gave little, if any, information regarding the retrosternal goitre but the technetium bone scan showed an uptake in the region of the mass, and this has been reported previously in a goitre with calcific degeneration.9 It is recognized that the uptake of isotope by a retrosternal goitre can be low and the diagnosis may have to be made by chest radiograph8 and confirmed by needle biopsy.

References