Case reports

Thyrotoxic heart disease presenting as unilateral pulmonary consolidation

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Thyrotoxic cardiomyopathy tends to occur in the elderly with mild but longstanding thyrotoxicosis. Although many patients are in atrial fibrillation, the classical features of Graves' disease are often absent. Fatigue and dyspnoea on exertion are common, but symptoms and signs of overt heart failure are unusual. We report a case of total unilateral pulmonary consolidation secondary to thyrotoxic heart failure.

Case report
A 66-year-old woman was admitted with anasarca. She described progressive leg and abdominal swelling for four months, but denied any orthopnoea or paroxysmal nocturnal dyspnoea. Her exercise tolerance, however, was reduced and she had been aware of intermittent, fast, regular palpitations for two months. She denied chest pain and there was no history of hypertension. Two years prior to admission she developed hyperglycaemic non-ketotic coma, but made a full recovery and her diabetes mellitus was well controlled on glibenclamide 2.5 mg daily.

On examination she appeared anxious. There was pitting oedema to the umbilicus with ascites. The heart rate was 100/min in sinus rhythm. She was apyrexial and there was no cough or sputum production. The blood pressure was 130/80 mmHg and the jugular venous pressure elevated by 6 cm. The apex was displaced to the 6th intercostal space 2 cm lateral to the midclavicular line and a third heart sound was audible. There was reduced movement of the right hemithorax, with dullness to percussion at the right base and bronchial breathing over the whole right side of the chest. The left hemithorax was entirely normal. Apart from ascites, the abdominal examination was unremarkable. There were no thyroid eye signs, proximal myopathy or pretibial myxoedema.

The full blood count and biochemical profile were normal apart from a raised alkaline phosphatase at 394 iu/l (normal range 86–280). Chest radiography on admission revealed cardiomegaly, consolidation of the whole right lung with air bronchograms and a possible small right pleural effusion but there was no abnormality on the left (Figure 1A). Ultrasound imaging of the chest confirmed the intra-alveolar consolidation of the entire right lung and identified a small, right-sided pleural effusion, but attempted aspiration was unsuccessful. The ECG was normal, but echocardiography showed an enlarged and poorly functioning left ventricle. Her free thyroxine index was 204 (normal range 70–125) and the free triiodothyronine index was 5.2 (normal range 1.7–2.7) with a reduced triiodothyronine uptake of 86 (normal range 92–117). There was a high titre of thyroid cytoplasm antibodies.

Figure 1. Posteroanterior chest radiographs, before treatment showing complete opacification of the right hemithorax (A), and when euthyroid (B).
She was treated initially with diuretics and carbimazole, and subsequently radioactive iodine. The jugular venous pressure fell, the oedema regressed and her chest signs resolved over a fortnight. As she became euthyroid, all her cardiorespiratory symptoms disappeared and she required no further diuretic treatment. Follow-up echocardiography and chest radiography were normal (Figure 1b).

Discussion
Hyperthyroidism can undoubtedly cause decompensation in pre-existing heart disease. Furthermore, pure thyrotoxic heart failure can occur in isolation and is characterized by a reversible haemodynamic deterioration. Although the presence of extensive, yet entirely occult, coronary artery disease cannot be excluded in this case, the predominant and probably sole cause of our patient's heart failure was hyperthyroidism.

Asymmetrical pulmonary shadowing is a well recognized feature of heart failure, but unilateral alveolar oedema is rare. Like pleural effusion secondary to cardiac failure, unilateral pulmonary oedema is nearly always on the right. It is often transient and may be related to dependency. However, it seems unlikely that consolidation of the severity and chronicity seen in the present case could be explained by the patient lying predominantly on her right side. Fraser and Paré have suggested that left-sided cardiac enlargement might physically impede blood flow in the left pulmonary artery, thereby reducing capillary volume. Asymmetrical pulmonary hypertension certainly accounts for the 'inappropriate' distribution of oedema in patients with unilateral thromboembolic disease, emphysema and pulmonary artery anomalies. Unequal capillary blood flow also explains the unilateral chronic pulmonary oedema seen occasionally following the surgical creation of left-to-right shunts.

Thyrotoxicosis is well known as a cause of atrial fibrillation, but this case emphasizes the importance of considering hyperthyroidism as a cause of heart failure despite sinus rhythm and being aware of the reversibility of thyrotoxic cardiomyopathy. In addition, it illustrates the marked asymmetry of chest radiographic changes occasionally seen in pulmonary oedema.

References

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Sterebral perforation of the colon following postoperative analgesia

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Sterebral ulceration and associated perforation of the large bowel results from trauma caused by impacted inspissated faecal material. It is a remarkably unusual condition, but should be suspected in any patient who presents with signs of a perforated viscus and is faecally impacted. Most reported cases involve elderly patients, often bedridden and suffering from chronic constipation. This report concerns a young man with a previously normal bowel habit who developed constipation associated with postoperative analgesia.

Case report
A 47-year-old man presented as an emergency with a six-hour history of severe abdominal pain. His bowels had not been open for 72 hours. Lower abdominal pain developed suddenly while he was attempting to defaecate and rapidly became severe and generalized. Three weeks previously he had had a single-vessel coronary artery bypass graft for ischaemic heart disease and had made an initially uneventful recovery. However, he had been taking regular, thrice daily doses of dihydrocodeine 60 mg, mainly for discomfort in his median sternotomy wound. His past gastro-intestinal history included intermittent upper abdominal discomfort for many years, which was relieved by cimetidine, but barium studies had revealed no abnormality.

On examination he looked ill, was in pain, shocked, hypotensive with a tachycardia, but was apyrexial. He had a distended, rigid, tender, silent abdomen. Rectal examination was markedly tender and revealed a rectum filled with hard faeces.

Investigations performed included full blood count, electrolytes and amylase, all of which were normal. There were no acute ischaemic changes on the electrocardiogram. An erect abdominal X-ray showed free gas under both hemidiaphragms and considerable faecal loading throughout the colon. He was resuscitated with intravenous fluids, analgesia, steroids (2 g methylprednisolone) and antibiotics (1 g ceftaxime and 500 mg metronidazole). At laparotomy he was found to have widespread faecal peritonitis arising from a ragged perforation at the rectosigmoid junction, through which faecal scybala were emerging. There was gross faecal loading of the colon, but no sign of any colonic pathology at the site.

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