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Case Report

A 57-year-old woman with a stroke and left-sided pleural effusion

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ABSTRACT

Severe hypothyroidism is associated with a wide spectrum of pulmonary and cardiovascular disorders. However, it rarely presents with pleural effusion. A 57-year-old woman presented with a stroke and left-sided pleural effusion was suspected of having hypertensive cardiomyopathy. During the treatment with diuretics, her renal function deteriorated and she developed a complete respiratory insufficiency, which led us to the definitive diagnosis. Therapy with thyroid hormone resulted in significant clinical improvement and the pleural effusion disappeared. Myxedema is easily missed when myxedema and pitting edema are simultaneously present in one subject. Severe hypothyroidism should be considered in the differential diagnosis of unexplained pleural effusion.

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1. Introduction

Severe hypothyroidism is associated with a wide spectrum of pulmonary and cardiovascular disorders. However, it rarely presents with pleural effusion. We will now describe a case in which severe hypothyroidism presented with a stroke, left-sided pleural effusion and a combination of pitting edema and myxedema which responded well to thyroid hormone substitution.

2. Case report

A 57-year-old woman came into the Emergency Department with depressed consciousness and left-sided hemiplegia. She had a short history (a few months) of peripheral edema of the lower extremities and fatigue. Her medical history included untreated hypertension. She was a non-smoker and took no medication. On hospital admission, the patient's heart rate was 75 beats/min and regular, BP was 210/120 mmHg, and the transcutaneous oxygen saturation was 92% on 5 L of oxygen via nasal cannula. Her face was swollen and pale coloured. There was distension of the jugular veins, abdominal shifting and dullness consistent with ascites and pitting edema of the lower extremities. Auscultation of the lungs revealed an absence of breathing sound on the left side. Normal heart sounds were observed. Her neurological examination showed a Glasgow Coma Scale of E2M6V2, with left-sided hemiplegia and

left-sided Babinsky reflex. A chest radiograph showed massive pleural effusion on the left side, with a shift of the heart and mediastinum to the right side (Fig. 1). Her significant initial laboratory values are displayed in Table 1. Thoracocentesis showed pale yellow-coloured fluid, chemistry showed transudative characteristics, culture was negative and no malignant cells were observed. Computed tomography (CT) scan of the brain demonstrated an intracerebral haemorrhage in the right hemisphere (Fig. 2). Echocardiography revealed severe concentric left ventricle hypertrophy (wall thickness >20 mm), most likely due to severe and long-standing untreated hypertension, 2 cm of pericardial effusion without signs of tamponade or elevated right filling pressures.

A presumptive diagnosis of hypertensive cardiomyopathy with left and right-sided decompensatio cordis was made and she was started on intravenous diuretics. The pleural effusion was treated with an indwelling chest drain. The cerebral haemorrhage was supposed to be secondary to her severe hypertension. After a few days of treatment with diuretics, her kidney function deteriorated, but she continued to suffer from dyspnoe. Her arterial CO₂ pressure increased and her pitting edema subsided, but did not resolved completely and changed into non-pitting edema. These symptoms favoured a diagnosis of hypothyroidism with secondary hypoventilation and myxedema. Thyroxin (T₄) level appeared to be 0.0231 ng/dL and thyroid stimulation hormone (TSH) >100 mIU/L.

After a gradual correction of the hypothyroidism, the patient showed significant clinical improvement with resolving pleural effusion, the renal function improved and the alveolar hypoventilation disappeared. Her ascites and edema of the lower extremities gradually dispersed. We therefore concluded that the

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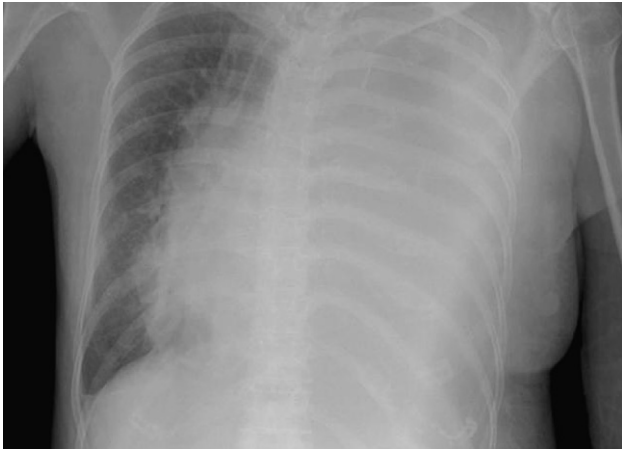


Fig. 1. Posterior anterior chest X-ray obtained on the day of admission showing massive pleural effusion at the left side, with a shift of the heart and mediastinum to the right side.

peripheral edema had been caused by a combination of myxedema and pitting edema due to right-side decompensatio cordis. In our clinical judgement, the pleural effusion was not only due to the hypertrophic cardiopathy, but mainly due to hypothyroidism.

After a few weeks, she was discharged from hospital and transferred to a rehabilitation centre. A few months later, she was able to walk using a stick.

3. Discussion

This case report describes an unusual presentation of massive pulmonary edema caused by severe hypothyroidism. Severe hypothyroidism is characterized by a wide spectrum of pulmonary and cardiovascular disorders; we will review the foremost items.

3.1. Pulmonary effects of hypothyroidism

Hypothyroidism can lead to pleural effusion, alveolar hypoventilation, respiratory muscle weakness and sleep apnea.

Pleural effusion as an initial clinical presentation of hypothyroidism is very unusual. The definitive diagnosis is often delayed^{1–4} and should be considered in the differential diagnosis of unexplained pleural effusion. The first case of a patient with pleural effusion associated with hypothyroidism was reported by Scheier-son and Katz in 1958. They described a 73-year-old man with 'myxedema' and right pleural effusion that resolved following thyroid hormone replacement.⁵

Pleural effusion can be an isolated finding in hypothyroidism or it can be associated with ascites, pericardial fluid and heart failure. Pleural effusions can be unilateral as well as bilateral,⁵ transudative or exudative.⁴ The literature contains different accounts of the amount of pleural effusion, which varies from a small, asymptomatic amount⁵ to massive pleural effusion,^{1–4} such as described in our patient.

Severe hypothyroidism is associated with a depression of both the hypoxic and the hypercapnic ventilatory drive, which may

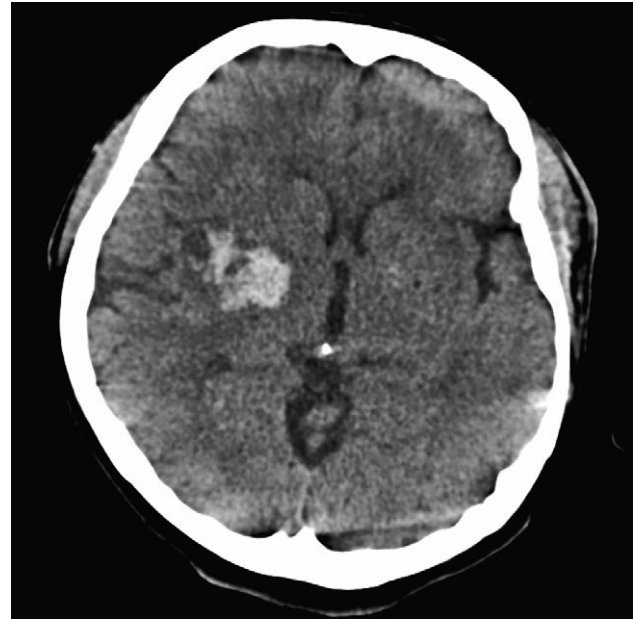


Fig. 2. CT scan of the brain showing an intracerebral haemorrhage in the right hemisphere.

cause alveolar hypoventilation. This can contribute to the development of a myxedema coma.⁶

Patients with hypothyroidism may develop diaphragmatic muscle weakness that can range from a mild impairment to severe dysfunction, with marked resting dyspnea and chronic hypercapnia.⁶

Hypothyroidism can predispose patients to sleep apnea, possibly as a result of narrowing of the upper airways by macroglossia secondary to deposition of mucopolysaccharides.⁶

3.2. Cardiovascular effects of hypothyroidism

The cardiovascular effects of hypothyroidism include haemodynamic changes, a decrease in cardiac contractility, an increase in peripheral vascular resistance, pericardial effusion, and alterations in lipid metabolism.

The loss of the inotropic and chronotropic effects of thyroid hormones leads to a reduction in both stroke volume and heart rate resulting in a decrease in cardiac output at rest. Echocardiographic studies have revealed left ventricular diastolic dysfunction, consistent with dilated cardiomyopathy, which can lead to advanced heart failure.⁷

Of particular interest is the fact that when myxedema and pitting edema simultaneously present in one subject, one of these phenomena is easily missed, as was the case with our patient.

Hypothyroidism causes a decrease in the levels of endothelial nitric oxide synthetase. This leads to an increase in the peripheral vascular resistance and results in diastolic hypertension.⁸ In normotensive patients, blood pressure increases are minor. The blood pressure of patients with hypertension, however, may increase significantly.⁶

Pericardial effusion can occur, causing enlargement of the cardiac silhouette on chest X-rays. Pericardial effusions do not compromise the cardiac output; cardiac tamponade is extremely rare. Treatment with thyroid hormone corrects the haemodynamic changes and restores the heart to a normal size.

Hypothyroidism is also associated with elevations of total and low-density lipoprotein (LDL) cholesterol resulting in an increased

Table 1
Initial laboratory values.

Haemoglobin	10.6 g/dL
Creatinine	1.11 mg/dL
pH (arterial)	7.29
PaO ₂ (obtained on 5 L oxygen)	63 mmHg
PaCO ₂	60 mmHg

risk for atherosclerosis, cardiovascular disease and an apparent increase in risk of stroke.⁸

In conclusion, we report a subject with severe hypothyroidism for whom it was difficult to make a correct diagnosis on account of the atypical presentation with massive pleural effusion and the combination of pitting edema and myxedema. Severe hypothyroidism should be considered in the differential diagnosis of unexplained pleural effusion.

Conflict of interest

None.

Funding

None.

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