

**Reversible diaphragmatic dysfunction in primary hyperparathyroidism**

Sirs,

Neuromuscular involvement is a rare complication of primary hyperparathyroidism (PHP). We report here a case of a reversible diaphragmatic dysfunction as a complication of PHP suspected from a chest CT and confirmed by ultrasonography.

A 21-year-old woman was hospitalised for dyspnea and thoracic pains that had lasted for two weeks. She had no particular medical history. She was afebrile, and became breathless following physical exercise. Her blood pressure and pulse were normal. A physical examination revealed bilateral basal thoracic condensations. Cardiac investigations were normal, and

no other abnormalities were found; there was no limb weakness, saturation was normal at rest and desaturation appeared with exertion. Antibiotic therapy had no effect on her symptoms.

Laboratory analyses revealed normal haemoglobin (12.7 g/dl), and neutrophils (4.64 G/l). Other results were as follows: C-reactive protein: 11 mg/l (n <5mg/l), serum calcium: 3.69 mmol/l (normal 2.25–2.5 mmol/l), serum phosphate: 0.40 mmoles/l (normal 0.85–1.4 mmoles/l), and serum creatinine: 60 µmoles/l. Primary hyperparathyroidism was confirmed by a high level of parathyroid hormone at 257 ng/ml (normal 15–65 ng/ml). Investigations for infections were negative, and a bronchial fibroscopy produced normal findings.

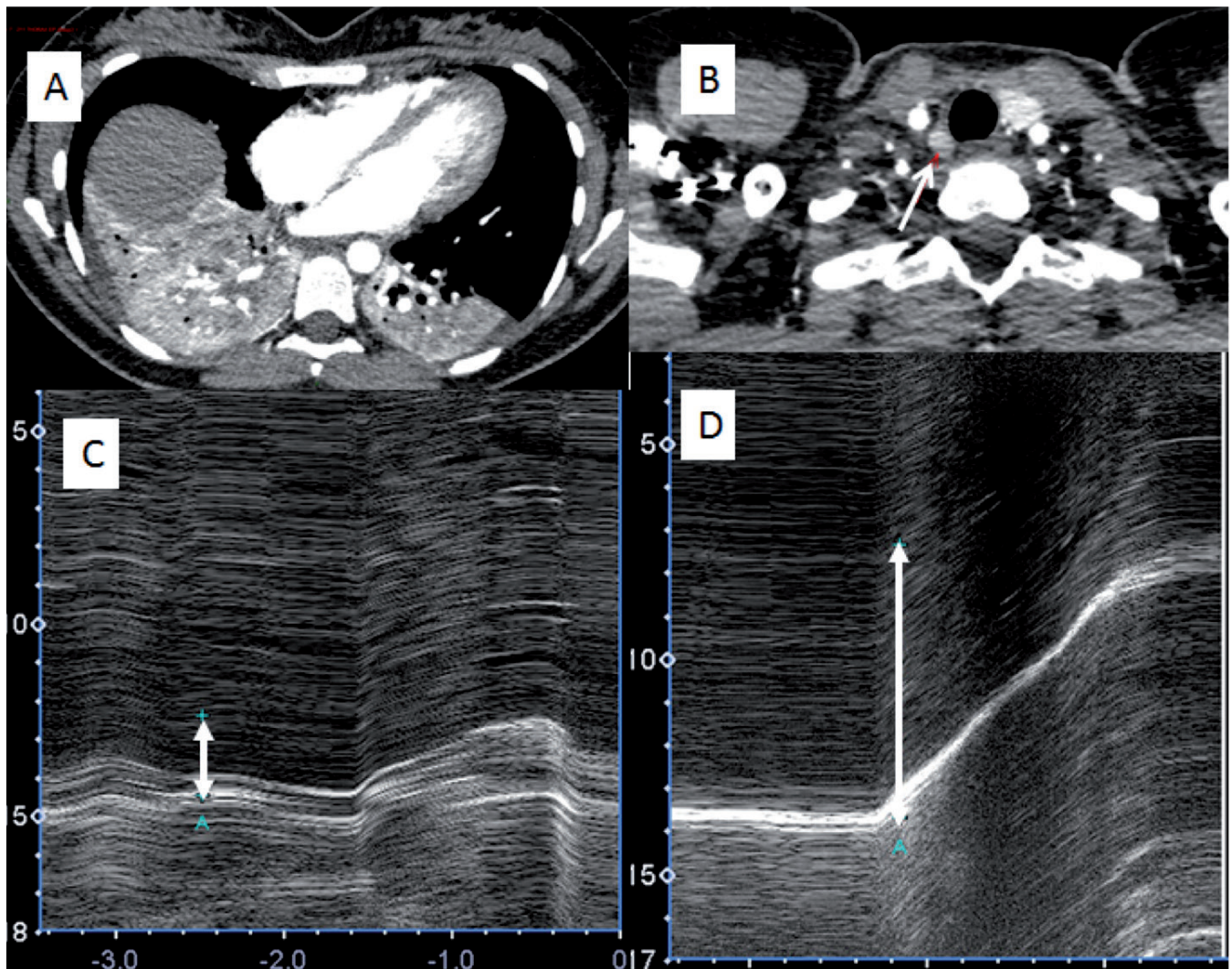
A chest CT revealed bilateral posterior condensations suggestive of bilateral diaphragmatic dysfunction (Fig. 1A) that

was subsequently confirmed by ultrasonography, showing the altered diaphragm course, 16 mm (Fig 1C).

An ultrasound and scintigraphy of the parathyroid glands revealed localised P3 adenoma which was visible on the chest CT (Fig. 1B). This diaphragmatic dysfunction was responsible for pulmonary atelectasia, which in turn was secondary to hypophosphatemia in a context of PHP. No other endocrine disease was found.

The patient was treated with intravenous phosphate and cinacalcet. Her serum calcium levels normalised and serum phosphate levels rose to 0.8 mmoles/l at four days. Her symptoms improved dramatically within a few days.

Resection of the parathyroid adenoma enabled a full recovery of diaphragm ampliation, 63 mm (Fig. 1D). Muscle thickening was respectively 2.5 mm and 1.5 mm in inspiration and in expiration



**Fig. 1.** A: Bilateral basal pulmonary condensation. B: Right para-tracheal nodule, corresponding to a parathyroid adenoma (arrow). C: Ultrasound of the diaphragm showing the altered diaphragm course at diagnosis, 16 mm; D: its normalisation after parathyroid surgery, 63 mm.

without significant difference between before and after surgery.

After one year of follow-up, the patient's serum calcium and phosphate levels have remained normal.

Neuromuscular involvement is a rare complication of PHP (1, 2). The myopathy is related to hypercalcaemia and hypophosphatemia (3). In our case, the suspected myopathy was localised to the diaphragm and regressed rapidly after serum calcium and phosphate levels returned to normal.

Diaphragmatic dysfunction has not previously been described in a context of PHP. It was suspected in a study by Giles *et al.*, who showed that pulmonary function improved after the surgical treatment of PHP (4).

Diaphragmatic dysfunction is uncommon and probably an underdiagnosed cause of dyspnoea. The methods used at present to diagnose diaphragmatic weakness or paralysis are either invasive or inaccurate. The ultrasound diagnosis of diaphragmatic dysfunction is primarily based on measuring the diaphragmatic range of motion (less than 2 cm), muscle thickness (less than 1.5 mm) and an absence

of thickening between inspiration and expiration (5).

In PHP, the exploration of neuromuscular disorders could be supplemented systematically by ultrasonography of the diaphragm.

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