CASE REPORTS

# Reversible myopathy and renal impairment

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In a patient with myopathy and renal impairment, a cause to be excluded is thyroid dysfunction.

## CASE HISTORY

A man aged 39 was referred in March 1998 to our otolaryngology service with a new symptom of snoring, which was treated unsuccessfully by uvulopalatopharyngoplasty a year later. Subsequently, in December 2000, he was referred to the regional renal unit with anorexia and lethargy associated with renal impairment. He was slightly short of breath and had experienced a change in his vision, necessitating glasses. On examination, he was normotensive. Creatinine was 189 μmol/L, urea 7.5 mmol/L, cholesterol 8.4 mmol/L. Other blood and urine tests, renal tract ultrasound and renal biopsy were normal. By May 2001, his creatinine had increased to 220 and his cholesterol to 10. A renal scan and doppler study were unrevealing but a glomerular filtration rate of 55 mL/min confirmed that renal function was subnormal. He was started on atorvastatin but within a week experienced new-onset myalgia and weakness. His creatine phosphokinase (CPK) was 3700 IU/L (normal range 24–195), so the atorvastatin was stopped. The CPK continued to increase, reaching 4200 a month later. Renal function was unchanged.

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With a possible diagnosis of polymyositis in mind, the patient, now 43, was referred to the rheumatology service. On examination, proximal and distal muscle power seemed normal but periorbital oedema was noted. Thyroid function tests indicated hypothyroidism and treatment was started with levothyroxine 50 μg daily. By March 2002, at which time the levothyroxine dose was 100 μg daily, indices were all normal (Table 1) and the patient had returned to his premorbid state.

*Table 1* **Laboratory indices before and after levothyroxine treatment**

| | *Free T4* | *TSH* | *Creatinine* | *Creatinine clearance* | *Cholesterol* | *CPK* |
|---|---|---|---|---|---|---|
| | *pmol/L (NR 10–27)* | *mIU/L (NR 0.3–4.1)* | *μmol/L (NR 60–125)* | *mL/min (NR 90–120)* | *mmol/L (NR 3.5–6.5)* | *IU/L (NR 24–195)* |
| Before treatment | <2.5 | >75 | 155 | 55 | 10.5 | 4200 |
| After 1 month treatment | 16.5 | 31.1 | 145 | 74 | 6.2 | 225 |
| After 5 months treatment | 14.6 | 4.3 | 121 | 94 | Not done | 176 |

NR=normal range; CPK=creatine phosphokinase; TSH=thyroid-stimulating hormone

## COMMENT

In a Medline review from 1979 to the present we found three cases of renal impairment and primary hypothyroidism associated with a raised CPK. In all three patients the raised CPK was associated with myopathy on electromyography or muscle histology, and two had rhabdomyolysis.[1–3] All three responded dramatically to thyroid replacement therapy with improvement in renal function and a decline in CPK. Our patient in addition had lipid abnormalities (a feature of dysthyroid states[4]) and these too were reversed by treatment of the hypothyroidism. Muscle enzymes in serum seem to correlate with the severity of the hypothyroidism rather than the extent of the myopathy.[2] Electromyographic abnormalities and muscle biopsy histological changes are usually minor and non-specific, making the condition hard to differentiate from polymyositis. Progression to rhabdomyolysis in hypothyroidism is very rare, and most previously reported cases have been associated with precipitating factors such as trauma, exercise and lipid-lowering medication.[1]

The presentation of hypothyroid-related myopathy can be further masked by other co-existent autoimmune disease such as Addison's disease. Interestingly, in such cases, the myalgic symptoms did not improve until levothyroxine was added to the steroid replacement therapy.[5]

Hypothyroidism is not only a recognized cause of *de novo* renal failure but can also precipitate deterioration in patients

with stable chronic renal failure, with levothyroxine replacement resulting in up to 50% improvement in renal function. It is important to detect, especially in the elderly, since hypothyroid symptoms may be masked in patients with known renal failure and the condition is easily treatable.[6,7]

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# Unusual presentations of acute lymphoid malignancy in children


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Acute paraplegia in childhood requires immediate diagnostic evaluation and treatment. Lymphoblastic leukaemia/lymphoma is a cause easily missed if signs of bone marrow dysfunction are absent.


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## CASE HISTORIES

### Case 1

A 4-year-old boy complained of knee pain and had increasing difficulty with weight-bearing for five weeks. A week before admission he stopped walking completely and the pain spread to involve most of his bones, especially his spine and ribs. He also described numbness over his thighs and strained to pass urine and stool. At this time an antero-posterior X-ray of his spine showed no abnormality. Intermittent spinal traction for five days was of no benefit, and on the day before admission an MRI of his spine was done. This showed a paraspinal mass from T2 to T8 passing through the intervertebral foramina into the spinal epidural space and compressing the cord (Figure 1). The radiological differential diagnosis was neuroblastoma or non-Hodgkin lymphoma. After starting dexamethasone for tumour-related cord compression he was transferred to our centre for further management. The relevant physical findings were hepatomegaly, decreased power and sensation in the lower limbs, and bilateral calf wasting more pronounced on the left. Though his full blood count was normal, leukaemic blast cells were seen on a peripheral blood film. The bone marrow contained 61% blast cells and immunophenotyping was positive for CD10, CD19 and CD79a. Acute lymphoblastic leukaemia was diagnosed and he was started on treatment according to the UK ALL 2003 protocol. Within a week his lower limb function was improving.

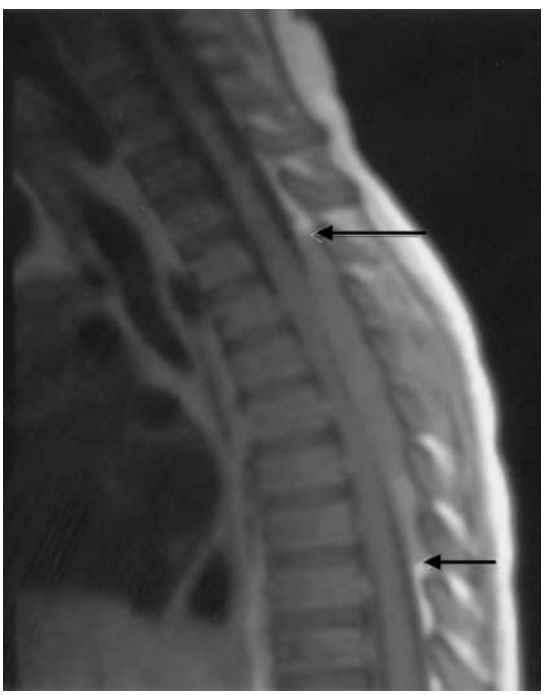

*Figure 1* **Sagittal T1 weighted MR image showing epidural mass in the thoracic spinal canal (between arrows)**