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Teaching Point
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An unusual cause of acute renal failure: hypothyroidism

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Introduction

Hypothyroidism is a systemic disorder with myopathic features including myalgia, muscle stiffness and cramp, and occasional moderately elevated levels of muscle enzymes [1]. Overt rhabdomyolysis and renal failure have been reported in only a few cases [1-3]. Here, we report a case of hypothyroidism in an elderly diabetic patient with acute renal failure (ARF) and rhabdomyolysis.

Case Report

A 69-year-old man presented with generalized body swelling and complaints of increasing stiffness. He had experienced severe myalgia, paraesthesia in both hands and lower limb weakness which had caused a reduction in everyday activity. He had a known medical history of diabetes mellitus but had neither familial nor prior personal history of thyroid disease and muscle disorders. Upon physical examination, the patient was afebrile, with normal heart rate (72 beats/min), periorbital puffiness, lip swelling, diffuse goitre and generalized non-pitting oedema. The remainder of the physical examination was normal. There were no signs suggesting an associated systemic inflammatory disease. The patient's laboratory findings were as follows: serum urea 9.99 mmol/L; creatinine 159.1 μmol/L; creatinine phosphokinase (CPK) 3332 U/L (normal 0–170); lactate dehydrogenase (LDH) 885 U/L (normal 0–248); and myoglobin 357 ng/mL (normal 0–38.5). Thyroid function tests confirmed the diagnosis of hypothyroidism: free T3 1.7 pg/mL (normal 2.3–4.2); free T4 0.43 ng/dL (normal 0.7–1.76); thyroid-stimulating hormone (TSH) >100 IU/mL (normal 0.35–5.5); anti-microsomal antibody 236 IU/mL (normal <60); and anti-thyroglobulin antibody 2128 IU/mL (normal <60). Urine dipstick chemical analysis suggested moderate blood presence, but there were no erythrocytes identified by microscopic examination, suggesting myoglobinuria. Other laboratory tests were normal. The complete panel of findings were compatible with autoimmune thyroid disorder, primary hypothyroidism and rhabdomyolysis. Treatment was initiated immediately by means of intravenous fluid, rapidly increasing doses of L-thyroxine (25 μg incrementally increased to 100 μg daily within 5 days and sustained over a 2-week period). On discharge, his muscular symptomatology and laboratory parameters (CPK, LDH, myoglobin, urea, creatinine) returned to normal. Moreover, TSH, free T3 and free T4 levels were detected at normal levels (6 weeks after treatment was initiated).

Discussion

Hypothyroidism is frequently accompanied by asymptomatic, mild to moderate (usually <10 times the upper normal limit), CPK elevations. To our knowledge, obvious rhabdomyolysis and ARF have rarely been associated with hypothyroidism alone [1-3]. The exact cause of rhabdomyolysis in hypothyroidism remains unclear, but impaired glycogenolysis and mitochondrial oxidative metabolism could be possible [4,5].

The present case describes an elderly diabetic patient, subsiding after thyroxine replacement, who had suffered from ARF and rhabdomyolysis due to hypothyroidism. In our patient, hypothyroidism was determined to be a principal cause of rhabdomyolysis. However, we consider the pre-existing diabetes mellitus and advanced age likely to be the precipitating factors, despite the two conditions not being defined for the classical cause of rhabdomyolysis.

In conclusion, although hypothyroidism is an uncommon cause of rhabdomyolysis, we suggest for it to be suspected in elderly diabetic patients presenting with ARF and, especially, those cases in which muscle pain is accompanied by elevated muscle enzymes. Thyroid hormone replacement therapy improved the thyroid and renal function of our patient and resulted in involution of rhabdomyolysis.

Conflict of interest statement. None declared.

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