An unusual case of "renal failure"

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ABSTRACT

Myopathy is common in hypothyroidism, but a very high creatinine kinase (CK) level in the range commonly seen with inflammatory myopathy is rare. Reversible elevation of creatinine is known to occur in hypothyroidism due to a decrease in the glomerular filtration rate, but it can also occur rarely due to enhanced creatinine production. We present a case of severe hypothyroidism with massively elevated CK levels and high creatinine levels, both of which reversed on treatment of hypothyroidism.

Key words: Creatinine, creatinine kinase, hypothyroidism

Introduction

Raised creatinine is known to occur in hypothyroidism, and there are different mechanisms for this. Cases of hypothyroidism with severe myopathy, rhabdomyolysis, and acute kidney injury are rare and are limited to case reports.[1-3] We present one such patient who was directly referred to a nephrologist by the general practitioner suspecting renal pathology, and discuss the mechanisms of such a presentation and the importance of looking for alternative causes in a patient presenting with isolated elevation of creatinine.

Case Report

A 30-year-old male presented with a history of generalized edema and significant weight gain of 20 kg in 6 months. He had tiredness, significant myalgia, and slurring of speech. The creatinine was elevated at 2.12 mg/dL, which was the reason for referral to the nephrologist. Routine investigations revealed a completely normal hemogram and urine analysis, elevated creatinine at 2.17 mg/dL but normal blood urea at 15 mg/dL. Hypothyroidism was suspected, which was confirmed with a T3 of 0.19 ng/mL, T4 of 0.4 µg/mL, and a thyroid stimulating hormone (TSH) of 780 µIU/mL. Creatinine kinase (CK) was 11,365 U/L. He also had abnormal lipid profile and abnormal liver enzymes. Abdominal ultrasound showed completely normal kidneys and mild fatty changes in the liver. Echocardiography was normal. A diagnosis of severe hypothyroidism with myopathy was made and given the normal blood urea, normal urine analysis, and renal ultrasound, the raised creatinine was thought to be secondary to excessive production rather than impaired renal function. He was commenced on thyroxine replacement therapy and a review in 2 weeks revealed a CK of 2,228 U/L and a creatinine of 1.5 mg/dL, both of which had normalized during the subsequent visit.

Discussion

Serum CK levels are often modestly elevated in hypothyroidism, but elevations in the levels usually seen in inflammatory myopathies or dystrophies are rare. There are case reports of extremely high elevations of serum CK with or without rhabdomyolysis associated with hypothyroidism.[1-3] If such a patient has significantly elevated creatinine, it may either be due to excessive production or acute kidney injury (AKI) caused by the toxicity of the non-protein heme pigment that is released from myoglobin. Tests for myoglobinuria are insensitive and should not be used for screening.[4] A urine analysis that shows microhematuria in the absence of visible red cells by urine microscopy in the appropriate clinical setting is sufficient to diagnose AKI due to rhabdomyolysis. Our patient was non-oliguric and had completely normal urine analysis, which ruled out rhabdomyolysis as the cause of

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his elevated serum creatinine.

An elevation in the serum creatinine concentration usually means a reduction in the glomerular filtration rate (GFR), and is associated with a concomitant rise in the blood urea. However, there are a variety of settings in which the creatinine can increase in isolation, independent of the GFR, and therefore there is no true change in the overall kidney function. This may be due to one of the three factors: Decreased creatinine secretion, interference with the serum assay, or enhanced creatinine production. Our case is one such example of a mechanism where there is likely to be a release of pre-formed creatinine from injured muscle and/or release of creatine that is then converted into creatinine in the extracellular fluid. It is important to look for such unusual causes in someone with unexplained isolated elevation in serum creatinine, especially in the absence of other markers of renal disease like abnormal urine analysis or ultrasonological abnormality of the kidneys.

Consistent and reversible elevation of serum creatinine in the hypothyroid state has been described in a study of 24 consecutive patients of iatrogenic hypothyroidism.[5] In this study, the rise in creatinine levels did not relate to abnormalities in CK levels or abnormal urine analysis, suggesting that neither hypothyroid myopathy nor intrinsic renal disease contributed to the changes seen. Another study has shown significantly elevated serum urea and creatinine in patients with overt and sub-clinical hypothyroidism, which correlated positively with TSH levels.[6] In our patient, both the CK and the serum creatinine levels were significantly elevated and blood urea was consistently normal with unremarkable urine analysis and renal ultrasound. Both creatinine and CK levels improved together, suggesting that the elevated creatinine was a reflection of enhanced creatinine production in the muscle rather than decrease in the GFR. Proving this theory will involve measuring the GFR with radionuclide study or inulin clearance, which has not been carried out in this case. Serum cystatin C level has also not been measured in our patient; however, the cystatin level showed no superiority when compared with serum creatinine estimation in the assessment of renal function in the setting of hypothyroidism.[7]

To conclude, our patient had an unusually high level of CK, which resulted in elevation in his serum creatinine level, both of which normalized with the treatment of hypothyroidism. Elevation in serum creatinine levels can occur even in the absence of decline in the GFR, and one should look hard for unusual causes, especially in a patient with absence of concomitant elevation of blood urea.

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