Case Report

PANHYPOPITUITARISM PRESENTATION WITH ACUTE RENAL FAILURE ASSOCIATED WITH RHABDOMYOLYSIS

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SUMMARY

A 30-year woman developed rhabdomyolysis and acute renal failure. She had history of amenorrhea after her last delivery. On admission, she was pale, afebrile and ill, had slight tenderness of the muscles. Her muscle enzyme was high and her renal function decreased rapidly, suggesting rhabdomyolysis. Her laboratory investigation was compatible with panhypopituitarism. Treatment was started with levothyroxine and Hydrocortisone. Her renal function recovered completely with conservative treatment without need of dialysis. The present case suggests that rhabdomyolysis could occur in patient with central hypothyroidism.

KEY WORDS: Panhypopituitarism, Rhabdomyolysis, Renal failure.

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INTRODUCTION

We describe a patient of rhabdomyolysis associated with panhypopituitarism. Primary hypothyroidism frequently leads to myalgias, muscle stiffness, cramps and sometimes elevated levels of muscle enzymes,[1] but rhabdomyolysis as a first presentation of central hypothyroidism is quite rare.[2]

CASE REPORT

A 30-year-old woman was admitted in our hospital with complaints of nausea, vomiting for four days. She had experienced fever and chill for three days prior to her admission. She had history of seven time delivery and was amenorrhic with no breast feeding after last twin complicated delivery, four years ago. She had no regular medication history. On examination, she was pale, afebrile and ill, had periorbital puffiness but no goiter. Her pulse was 80 beats/min regular, blood pressure 90/60mm/Hg. Neurological examination revealed marked proximal-dominant weakness and slight tenderness of the muscles. No sensory disturbance was observed. Her skin was dry, axillaries hair was scattered and breast was atrophic. Other systemic examinations were normal. First investigation revealed: haemoglobin 13.6g/dl, total leucocyte count 8×10^3/l with 90% polymorphs, serum Na 137mEq/l, K 3.6mEq/l, Blood urea nitrogen 25mmol/l, creatinine 1.5 mg/dl, random blood glucose 47mg/dl, stool smear & culture and urine culture were negative. Her serum free T4 was 0.72ng/ml(normal 0.9-1.6), TSH 0.5µIU/ml and (normal, cortisol at 08.00 h 4µg/dl. (Normal 8-19), FSH 1.65 IU (normal 5-10), LH 1.90 IU(Normal 5-10), Estradiol 22.9pg/dl (normal 40-150) and prolactin 1.88 (normal 5-20). She received interavenous dextrose-saline, hydrocortisone

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and 0.1mg of L-thyroxine daily. On day two of admission, she became oliguric and serum creatinine raised rapidly to 4.3mg/dl and then to 7.1mg/dl the next day. Muscle weakness progressed and finally she was unable to walk and became disoriented. Her laboratory tests revealed: serum aspartate transaminase 190IU/l (normal 20-40), alanine transaminase 77IU/l (normal 20-45), creatinekinase-MM (CK-MM) 50000 IU/l (normal<397) and dehydrogenate 1542IU/l (normal 98-192) and serum myoglubolin >100ng/ml (normal <50ng/ml). Her urinary myoglubin was positive. Her brain CT scan showed empty sella. She was managed as an acute renal failure case and her intravenous intake was adjusted according to her intake and output. After several days, her urine output gradually increased, she regained conscious and fortunately recovered without resorting to dialysis. The serum creatinin decreased to 1.0mg/dl and patient's muscle power improved gradually so, she could walk at the time of discharge. She was finally discharged and was put on with 0.1mg levothyroxine and 10mg prednisolone. Her serum muscle enzyme normalized over a 3-week period.

DISCUSSION

According to her past history, she is a case of panhypopituitarism (Sheehan syndrome) who presented with rhabdomyolysis and acute renal failure. Diagnosis of panhypopituitarism was confirmed with hormonal assays. Diagnosis of rhabdomyolysis was made on the severe muscle weakness and marked elevation of Ck-MM, as well as remarkably high levels of urinary and serum myoglubin. Disorders such as trauma, collagen disease, digestion of alcohol or other agents and systemic infection,[3] as a cause of rhabdomyolysis were excluded. Several cases of rhabdomyolysis due to hypothyroidism have been reported in the literature,[4,5] but in Sheehan syndrome, rhahabdomyolysis as a first presentation of central hypothyroidism is quiet rare.[2] The exact cause of rhabdomyolysis in hypothyroidism remains unclear. Usually an aggravating factor such as use of lipid-lowering drugs, Alcohol, exercise or chronic renal failure has been identified.[6-8] In our case, we didn't find any precipitating factors. Hence we suggest that, panhypopituitarism must be considered in patients presenting with acute renal failure and elevated muscle enzymes.

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