Rhabdomyolysis Following Severe Hypokalemia Caused by Familiar Hypokalemic Periodic Paralysis

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Nontraumatic rhabdomyolysis is a polyetiologicaldisease being recognized with increasing frequency. One of the most interesting causes of rhabdomyolysis is pottasium deficiency. Potassium plays a major role in regulating the skeletal muscle blood flow, and hypokalemic periodic paralysis is one form of periodic muscle weakness, a group of disorders that can cause of sudden onset weakness associated with low serum potassium levels. Familial hypokalemic periodic paralysis is rare genetic disease characterized by periodic attacks of muscle weakness associated with a decrease in serum potassium without other detectable causes. Rhabdomyolysis following severe hypokalemia as the manifestation of familiar hpokalemic periodic paralysis is extremely rare. Here, we report an unusual case of rhabdomyolysis caused bysevere hypokalemia, which in turn was the result of familiar hypokalemic periodic paralysis. An 30-year-old woman had 3 episodic attacks of suddenly muscle weakness with hypokalemia after excessive intake of carbohydrate. She had no history of hypertension, drug intake including of diuretics, fever and rash. After receiving potassium supply, muscle weakness was recovered, but the patient complained a severe ache in both calves without corresponding physical exertion. Laboratory test showed elevated creatinine phosphokinase (> 15,000 IU/L) and extremely low serum potassium (1.9 mmol/L). The patient described in the case report had the characteristic clinical features of rhabdomyolysis caused by profound potassium deficiency associated with familial hypokalemic periodic paralysis.

A case of type 4 renal tubular acidosis after kidney transplantation

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Type 4 renal tubular acidosis is a normal anion gap metabolic acidosis and shows hyperkalemia and positive urine anion gap. Renal tubular acidosis is commonly caused in the early post-transplant period, due to calcineurin inhibitor (CNI) nephrotoxicity, acute rejection suboptimal allograft function, and ischemic tubular dysfunction. We report on a case of type 4 renal tubular acidosis after kidney transplantation in a 29-year-old man. He had a history of IgA nephropathy, and chronic kidney disease with continuous ambulatory peritoneal dialysis. After kidney transplantation, His examinations showed a serum potassium of 7.4 mEq/L, blood pH 7.27, serum bicarbonate 20 mmol/L and serum creatinine 1.83 mg/dL. Subsequent investigation showed normal anion gap metabolic acidosis, urinary pH 5.0, positive urinary anion gap, and low levels of aldosterone (12 pg/mL), suggesting the presence of type 4 renal tubular acidosis (RTA). Mineralocorticoid was initiated and we reduced tacrolimus dosage. And after 2 weeks, hyperkalemia was corrected. In our case, type 4 renal tubular acidosis was reversible and responded to mineralocorticoid and a reduction of tacrolimus dosage.