Hypokalemic Quadriplegia Secondary to Abuse of Cocaine and Heroin

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ABSTRACT

Low plasma potassium level can cause muscle weakness, lassitude, constipation as well as rhabdomyolysis and arrhythmias, when severe. In muscle, low plasma potassium increases resting membrane potential (hyperpolarization) of myocytes that tend to make muscle more refractory to excitation, leading to muscle weakness. Hypokalemia can be associated with a myriad of causes including drugs of abuse. We present a case of hypokalemia and muscle weakness following use of cocaine and heroin.

KEYWORDS: cocaine, hypokalemia, muscle weakness, quadriplegia

CASE REPORT

A 33-year-old woman presented with 5-day history of painful weakness of all four extremities. The weakness started in the legs and ascended to the arms. Past history was significant for untreated hypertension, and recent use of heroin, cocaine and marijuana. There was no family history of periodic paralysis. Her physical examination was remarkable for systolic hypertension, exquisitely tender limbs and flaccid paresis of all limbs with a normal sensory examination and preserved deep tendon reflexes. Investigation revealed sodium of 138 meq/L, severe hypokalemia (potassium of <2 meq/L), chloride of 97 meq/L, bicarbonate of 28 meq/L, and elevated creatinine phosphokinase (CPK of 5838 IU/L, [normal 38-234 IU/L]). Serum cortisol, aldosterone, magnesium, renin activity, thyroid stimulating hormone and arterial blood gas analysis were normal. Urine toxicology was positive for opioid, cocaine and tetrahydrocannabinol [THC]. The electrocardiography (EKG) revealed a prolonged QTc interval of 598 milliseconds, and ST segment depression. With a diagnosis of hypokalemia-induced quadriplegia secondary to cocaine and heroin abuse, the patient was admitted for telemetry monitoring and was started on potassium supplementation. With improvement in serum potassium, she had resolution of her symptoms and was discharged home.

DISCUSSION

The etiologies of young adults with hypokalemia and weakness can be categorized into two groups: one due to transcellular shift of potassium and the other due to loss of potassium from body. Young patients who present with recurrent weakness and hypokalemia can be due to hypokalemic periodic paralysis (HPP) due to mutations of ion channels in the muscle sarcolemma.1 HPP includes thyrotoxic periodic paralysis that is associated with hyperthyroidism, familial periodic paralysis and sporadic periodic paralysis that is not associated with family history or thyrotoxicosis. Drugs that promote transcellular shift of potassium include β2-agonists, nasal decongestants and insulin.2 Muscle weakness due to hypokalemia can also occur after profuse diarrhea, excessive vomiting, apparent mineralocorticoid excess due to licorice ingestion.1,3 Cocaine and heroin are widely abused substances. Cocaine can affect multiple organ systems and may cause cardiac arrhythmias, seizures and intense sympathetic stimulation causing tachycardia, hypertension, dilated pupils, and increased psychomotor activity.4,5 Increased sympathetic stimulation leads to increased sodium-potassium ATPase activity that causes an increased shift of potassium into intracellular space resulting in hypokalemia.2 Cocaine’s potential effect on potassium channels has also been sought as alternate mechanism. Various case reports have shown that cocaine use can result in hypokalemia and muscle weakness.6-8 Patients with potassium level between 3.0-3.5 mEq/L are often asymptomatic but patients with potassium level below 3.0 mEq/L can present with malaise, muscular weakness, restless leg syndrome and myalgia.9 When potassium level is lower than 2mEq/L, frank rhabdomyolysis can occur with markedly elevated serum creatinine kinase and myoglobinuria.9 Rhabdomyolysis has been reported to occur with cocaine and may lead to renal failure. Transient weakness with elevated creatinine kinase following use of cocaine in the absence of hypokalemia has also been reported with postulated mechanisms.8,10-13 Cocaine can block transient inward flux of sodium across the cell membrane during the depolarization, increasing intracellular calcium, resulting in

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tetany. Although the exact mechanism is not clear, rhabdomyolysis in patients taking cocaine has been suggested due to cocaine-induced vasospasm leading to muscle ischemia, direct toxic effects of cocaine on myocytes and hypokalemia associated with cocaine use.14 Contracting skeletal muscle increases concentration of potassium in interstitial fluid which leads to vasodilation of arterioles supplying the skeletal muscle and hence increases in blood flow. This increase in blood flow may not be adequate when concentration of potassium is subnormal.15-17

Hypokalemia and muscle weakness has been reported in few cases of heroin abuse.18 Clenbuterol [a drug used to treat airway obstruction in horses] was found as an adulterant in heroin that caused transcellular shifts of potassium leading to muscle weakness.3 Heroin overdose was seen to be associated with hypokalemia, and hypokalemia was more severe in patients who required higher dose of naloxone to reverse the effect of heroin. In an animal study, opiate overdose was associated with hypokalemia and sympathoadrenal stimulus to acute hypoxia after opiate overdose was suggested as possible mechanism.19,20 In our patient, we speculate that both cocaine and heroin contributed to hypokalemia. Substance abuse should always be considered as a potential cause of hypokalemia in adolescent and young adults. Cocaine and heroin may cause hypokalemia in otherwise healthy young adults. Toxicological screening can spare a patient from unnecessary testing for alternate conditions.

References

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