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CASE REPORT

A rare case of brucine poisoning complicated by rhabdomyolysis and acute renal failure

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Abstract

Brucine is the predominant alkaloid present in the bark of the tree *Strychnos nux vomica* and is a weaker alkaloid when compared to strychnine. However, its toxicological property is akin to strychnine. We report a rare case of brucine poisoning complicated by acute renal failure and rhabdomyolysis. A 24-year-old male presented with a history of consumption of a decoction made from the bark of the *Strychnos nux vomica* tree. Soon after, he developed widespread muscle spasms and convulsions, which were promptly treated. On the fifth day of admission, he developed features of rhabdomyolysis and acute renal failure. Investigations revealed elevated creatine phosphokinase levels and elevated blood urea and serum creatinine. The patient was managed with hemodialysis and recovered gradually. There are many reports of strychnine poisoning producing rhabdomyolysis and renal failure. In this case report, attention is drawn to the fact that brucine, although a weaker alkaloid, can also produce life threatening complications like rhabdomyolysis and acute renal failure.

Keywords: brucine, strychnine, poisoning, spasms, convulsions, rhabdomyolysis, acute renal failure

INTRODUCTION

Brucine is one of the alkaloids present in *Strychnos nux vomica*, a tree predominantly seen in India, Sri Lanka, Southeast Asia and northern Australia. Strychnine is the other alkaloid and is 10 to 20 times more poisonous than brucine. However, brucine is allied to strychnine in composition and action. Though all the parts of the tree are toxic, the seeds are the most toxic as they contain about 1.5% of strychnine. The bark of the tree contains only brucine and no strychnine.[1,2, 3] We report rhabdomyolysis and acute renal failure (ARF) resulting from brucine poisoning in a patient who consumed a decoction made from the bark of the *strychnos nux vomica* tree. There are many reports of strychnine poisoning induced rhabdomyolysis and ARF[4,5,6] but such complications from brucine poisoning are poorly documented.

CASE REPORT

A 24-year-old male presented with convulsions and a history of consumption of a decoction made from the bark of the *Strychnos nux vomica* tree. He drank the preparation along with six other members of the family as a religious ritual.[7] Within a few minutes of consumption, he developed convulsions and spasm of various muscles. All the six members of the family were poisoned. Two young children died on the way to the hospital. Three other adult members of the family had mild muscular spasms and stiffness.

On examination, the patient was fully conscious, but exhibited frequent convulsive movements of the extremities. His vital signs were within normal limits. He was given a stomach washout using activated charcoal. His muscle spasms and convulsions were controlled with parenteral diazepam. He made a smooth recovery, until the 5th day of admission, when he complained of weakness of the lower limbs along with abdominal pain and vomiting. There was no history of decreased urine output. The patient was transferred to the nephrology unit with the diagnosis of rhabdomyolysis and acute renal failure. He was managed with regular

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hemodialysis and made a full recovery in two weeks. The patient was closely followed up for three months after that and his renal functions returned to normality.

Investigations:
Blood counts, blood urea, serum creatinine and urine analysis were normal on admission. However, on the 5th admission day, the repeated parameters were as follows: Hb 12 gm/dl; total leucocyte counts 10200/dl, differential count: polymorphs 79%, eosinophils 1%, lymphocytes 19%, monocytes 1%, ESR 40mm/1st hour, blood urea 154mg/dl, serum creatinine 10.3mg/dl, sodium 128mEq/L, potassium 5.4 Eq/L, chlorides 88Eq/L, calcium 9.0mg/dl, serum amylase 48 IU/L, HCO3 14.0 mEq/L, RBS 84mg/dl, CPK 7182 IU/L, Urine: myoglobin absent. Ultrasound abdomen: normal kidney size and texture.

DISCUSSION

Brucine, a less potent alkaloid of Strychnos nux vomica, is allied to strychnine in composition and action. Malone *et al* found Brucine [2, 3- dimethoxy strychnidin-10-one] to possess strychnine-like pharmacological and toxicological properties in their mice experiments.[8] In this case we believe brucine consumption led to convulsions and rhabdomyolysis, leading to acute renal failure. Extensive review of literature did not reveal any cases of brucine poisoning producing rhabdomyolysis and renal failure. Strychnine is very well known to produce rhabdomyolysis[4,5,6] and acute renal failure.[4,5,6] Hence, we presume that the same can be produced by brucine, a less potent but similar alkaloid.

Strychnine competes with the inhibitory neurotransmitter, glycine at the post synaptic receptor site in the motor neurons of anterior horn cells of the spinal cord. This produces excitatory effect on the anterior horn cells leading to hyperreflexia, severe muscle spasms and convulsions.[3] Strychnine action is at the level of the spinal cord, producing convulsions in fully conscious patients without a post ictal phase.[9] In view of the hyperexcitability state of the anterior horn cells, even the slightest stimulation can trigger diffuse muscle contraction.[5] Prolonged muscle spasms and convulsions lead to a potentially life threating complication, namely rhabdomyolysis, due to the damage to skeletal muscle, as indicated by elevated creatine phosphokinase levels as in our patient.

In rhabdomyolysis, skeletal muscle breakdown products like myoglobin are released into the circulation. Subsequent myoglobinuria results in characteristic tea colored urine. The identification of components of the muscle cell like myoglobin or creatine kinase is the definite tool for the diagnosis of rhabdomyolysis.[10,11] Early diagnosis is crucial for the successful management of this condition. The severity of rhabdomyolysis varies widely from asymptomatic elevation of muscle enzymes to the life threatening complications of acute renal failure and severe electrolyte abnormalities. Myoglobin binds loosely to plasma proteins and finds its way to the urine easily and in the early stage of the illness. Because of this early clearance, myoglobin may not be detected in the urine in the later stages of the illness, as in our case.[12]

Acute renal failure (ARF) is a serious and late complication of this syndrome.[10,11] Approximately, 15-30% of rhabdomyolysis cases advance to ARF and rhabdomyolysis features in 5-9% of all causes of ARF. In the early phase of ARF with myoglobinuria, the pathogenesis is mainly on account of vasoconstriction and decreased perfusion at the glomerular level. Later intraluminal cast formation and direct cytotoxicity due to heme in the myoblobin lead to tubular damage. Added problems like dehydration, hypovolemia and aciduria are important factors which push the patient into myoglobinuric ARF. Management of fluid and electrolyte balance using saline, bicarbonate along with mannitol will definitely help in preventing myoglobinuric ARF.[11,13] Proximal tubular cytotoxicity is predominantly due to the free iron released from the heme protein and the oxidant stress. [14]

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