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Case Report

Unaccustomed exertion and opiate consumption leading to acute renal failure in a healthy male

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ABSTRACT

A 38 year old male presented with stupor and acute renal failure (ARF) following opium consumption and a protracted excursion on foot to a holy shrine situated at altitude of 4329 m above sea level. The finding of dark colored urine, myoglobinuria and a markedly elevated serum creatinine phosphokinase level supported the diagnosis of rhabdomyolysis. The patient had a rapid and complete recovery following fluid resuscitation and haemodialysis.

Keywords: Rhabdomyolysis, Acute renal failure, Opium consumption

INTRODUCTION

Rhabdomyolysis is a clinical syndrome wherein contents of injured muscles cells escape into circulation resulting in electrolyte imbalance, acidosis, clotting disorders, hypovolemia, ARF. This may result in visible myoglobinuria, i.e. red or brown urine. Incidence is higher in males, adults, inherited enzyme deficiencies of carbohydrate or lipid metabolism, and myopathies. In US, rhabdomyolysis accounts for an estimated 8-15% cases of acute renal failure. No Indian data is available. Mortality in rhabdomyolysis is roughly 5%.

Rhabdomyolysis following severe physical exertion with or without heat stress resulting in ARF is rare. We report a rare case of exertional rhabdomyolysis under the effect of dehydration, opium overdose, complicated by acute renal failure (ARF) in a previously healthy individual.

CASE REPORT

A 38 year old male, previously in good health went on excursion to a holy shrine situated at altitude of 4329 m above sea level in Uttaranchal, India. After continuous walking for 21 kilometres in the hot, humid hilly terrain, he collapsed. He had illicitly consumed opium (as raw opium) before starting the trek and also during the trek in significant amount along with tablets of proxyvon (Paracetamol 400 mg, dextropropoxyphene napsilate 100 mg). On admission, he was drowsy (GCS 9/15) and dehydrated. There was no pallor, icterus, cyanosis or edema. The pupils were small and sluggishly reactive, blood pressure was 98 mmHg systolic, pulse 98/min and respiration was laboured. Chest, cardiovascular and abdominal examinations were unremarkable. Plantars were flexor bilaterally and there were no meningeal signs. Catheterization yielded 100ml of reddish-brown urine.

Investigations at admission revealed hemoglobin of 13 gm/dl, hematocrit 42 (normal range 38.8-46), total leucocyte count 10700/cumm and platelet count of 1.5 lacs/cumm. Prothrombin time was 13 sec (normal range 12.7-15.4 s), urine analysis showed pH 5.0, albumin 1+, 5-6 red blood cells/HPF and muddy casts. Urine for hemoglobin (by multistix) tested positive (normally undetectable). Urine spot Na was 88 mEq/L (normal

range 100-260 meq/L) and urine myoglobin was 157 IU (normally undetectable). Toxicology screen for opioids tested positive.

The renal function tests showed blood urea to be 206 mg/dl and serum creatinine to be 7.5 mg/dl which increased to 9.5 mg/dl on the second day. Other lab parameters were serum calcium 7.45 meq/l, serum phosphate 6.35 meq/l, serum albumin 4.2, uric acid 7.85/ml, Serum sodium 143 meq/l, serum potassium 5.5 meq/l, serum chloride 100 meq/l. ABG (on supplemental oxygen) analysis revealed pH-7.21, pCO_2-45, pO_2-89, HCO_3-16.7, O_2 saturation 96% (acute respiratory and metabolic acidosis). The creatinine phosphokinase showed a value of 867 U/L (normal range 51-294 U/L), lactate dehydrogenase 980 U/L (normal range 115-221 U/L), alanine aminotransferase 85 IU/l (normal range 7-41 U/L) and aspartate aminotransferase 77 IU/l (normal range 12-38 U/L). ECG showed no hyperkalemic changes. Ultrasound abdomen showed normal sized kidneys.

He was resuscitated with intravenous fluids under Central Venous Pressure (CVP) monitoring. After a bolus dose of 0.4 mg, 6 mg of naloxone was given as a slow infusion over 3 hours, during which his papillary reaction and GCS improved. He was infused with saline containing sodium bicarbonate followed by intravenous diuretics. However his overnight urine output was only 600 ml. Due to worsening of azotemia; he was hemodialysed the next day and subsequently required two more sessions of haemodialysis. After three sessions of haemodialysis, the patient started showing improvement, entered diuretic phase and his renal functions improved with serum creatinine falling to 2.1mg/dl after one week. Subsequently the patient was reviewed in the out-patient clinic after two weeks; his urine output was normal and serum creatinine had come down to 1.1 mg/dl, transaminases had normalized and he had no myalgias.

DISCUSSION

Rhabdomyolysis is caused by injury to skeletal muscles resulting in release of intracellular muscle constituents. It was first described in the victims of crush injury during Second World War in 1940-1941 and was known as "crush injury syndrome".[1] Since then, several non-traumatic conditions leading to rhabdomyolysis and myoglobinuric renal failure have been described.[2,3]

Excessive muscular activity especially in unconditioned men (so called white collar rhabdomyolysis), physical injury from compression, ischemia, hyperthermia, electrical injury, all can result in hyperkalemia, metabolic acidosis and rhabdomyolysis.[4] Non-traumatic rhabdomyolysis is usually caused by toxic reaction to drugs, common compounds associated with it being opium, alcohol, cocaine, amphetamines and ecstasy. Prolonged hypoxic coma following opiate overdose can lead to intracapillary myohypoxia causing rhabdomyolysis.[5] A positive ortho-toluidine test (less sensitive) or preferably spectrophotometry in the supernatant urine, an elevated CPK and myoglobin in serum helps clinch the diagnosis of rhabdomyolysis.

Ramamoorthy et al. described myoglobinuria with ARF in a nineteen year old boy who performed three hours of continuous dance programme on a hot humid summer afternoon.[6] Uberoi et al. reported seven cases over a period of six years with ARF due to exercise induced myoglobinuria in the absence of heat stress.[7] Similarly another series of eight cases was reported from a naval officers training institute.[8]

In a retrospective study of 181 patients of rhabdomyolysis from poisoning centre of Loghman-Hakim hospital in Tehran during September 2004 to September 2005, opium overdose was found to be most commonly associated with rhabdomyolysis with ARF.[9] Melandri et al. reported a case of myocardial damage along with rhabdomyolysis due to prolonged hypoxic coma following opiate overdose.[5]

The major life threatening complication of myoglobinuria is acute tubular necrosis, as occurred in our case. The exact mechanism of ARF is not well understood. It is postulated that direct tubulo-toxic effect of ferrihemate or myoglobin, obstruction to tubular lumen by myoglobin casts, back diffusion of glomerular filtrate through a break in the epithelium and decreased glomerular filtration rate, leads to ARF.

Dehydration, heat stress, hypovolemia and acidification of urine are crucial precipitating factors. Renal involvement is characterized by oliguria, exceptionally high creatinine levels, hyperkalemia, hyperphosphatemia and hyperuricemia. Serum calcium may be low in the oliguric phase and later in diuretic phase patients may develop hypercalcemia.

Our patient had hypocalcemia, hyperphosphatemia, hyperkalemia and hyperuricemia. Predictors for development of renal failure are CPK levels more than 6000 IU/L, dehydration, hematocrit >50, serum sodium >150 meq/L, orthostasis and Pulmonary Capillary Wedge Pressure (PCWP) <5 mmHg.

Treatment of ARF due to myoglobinuria is by volume replacement, forced diuresis, hemodialysis and supportive measures. ARF should be suspected in patients with CPK levels in excess of 2-3 times the reference range, in the presence of risk factors for rhabdomyolysis. Vigorous hydration with isotonic crystalloid supports the intravascular volume, increases the Glomerular Filtration Rate (GFR) and oxygen delivery, and dilutes myoglobin and other renal tubular toxins. If initiated early, alkaline solute diuresis and infusion of mannitol or sodium bicarbonate can improve renal function.

CONCLUSION

Unintentional or intentional overdose with illicit drugs like opium or cocaine should always be investigated in cases of unexplained rhabdomyolysis and acute renal failure. Sporadic strenuous and prolonged exercise along with poor intake of fluids and heat stress can be potentially life threatening.

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