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Acute alcoholic myopathy, rhabdomyolysis and acute renal failure : a case report.

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Abstract

A case of middle aged male who developed swelling and weakness of muscles in the lower limbs following a heavy binge of alcohol is being reported. He had myoglobinuria and developed acute renal failure for which he was dialyzed. Acute alcoholic myopathy is not a well recognized condition and should be considered in any intoxicated patient who presents with muscle tenderness and weakness.

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Full Text

:: Introduction



Trauma is the commonest cause of rhabdomyolysis. Rarely non-traumatic causes such as seizures, physical exercise, vascular occlusions, drugs, toxins, infections and extremes of temperature can also be responsible.[1] Ethanol generally causes chronic myopathy which occurs after its prolonged use.[2] However, rarely acute muscle injury can occur following binge drinking. It is often asymptomatic with only elevation of muscle enzymes in serum, but can lead to painful muscle swelling, myoglobinuria and acute renal failure.[3-7] We report a middle aged male, a chronic alcohol abuser, who developed acute rhabdomyolysis, myoglobinuria and acute renal failure, following a heavy bout of drinking.

:: Case report



A 48 years old male was admitted to Nehru hospital attached to the Postgraduate Institute of Medical Education and Research, Chandigarh with history of painful and tender lower limb muscles. He had been a chronic alcohol abuser for the last 20 years and was consuming about 50-100 gm of ethanol 3-4 times per week. Two days prior to the admission he had an alcoholic binge and had consumed about 400 gm of ethanol (1 litre of 80 proof whiskey) following which he slept for 9-10 hr. On getting up in the morning, he found that he could not walk on his own because of marked pain in the muscles of the lower limbs and weakness. There was no history of trauma, convulsions or coma. Two days later, his urine output decreased and he was brought to the hospital. There was no history of passing cola colored urine or haematuria.

At admission, he was conscious and afebrile. There was no pallor, icterus or cyanosis. The pulse was 100/min.

and BP 130/80mm Hg. Bilateral pitting oedema was present over legs. Cardiovascular and abdominal examination was normal. Examination of chest revealed bilateral crepitations. The neurological examination revealed swollen and tender thigh and gluteal muscles and the muscle power was 2-3/5. The upper limbs were normal. There was no sensory loss. On investigations, haemoglobin was 13.8 gm/dl; total leucocyte count 11000/cumm with normal differential count. Blood biochemistry revealed urea 287 mg/dl; creatinine 10.5 mg/dl; bilirubin 0.7 mg/dl; SGOT 72 IU/1 (normal 2-20); SGPT 70 I.U/1 (normal 2-15); proteins 6.2 gm/dl with albumin 3.5 gm/dl; calcium 6.5 mg/dl; inorganic phosphate 11.4 mg/dl and creatine kinase (MM) 19565 U/1. The urine was positive for myoglobin. The arterial blood gases revealed pH 7.45; PaO₂ 80 mm Hg; PaCO₂ 20 mm

Hg; HCO₃ 14 m mol/1 and SaO₂ 96.5%. The sonological examination of abdomen revealed normal kidneys and there was no evidence of cirrhosis. He was first given peritoneal dialysis which was followed by haemodialysis on alternate days, till his urine output became normal in 4 weeks time. At discharge, his power in the lower limbs was normal with no muscle swelling or tenderness. The urine was negative for myoglobin and his enzymes i.e. SGOT, SGPT and CK (MM) were normal. On follow up at 8 weeks, he was abstinent from alcohol and his muscle power in lower limbs was normal.

:: Discussion

Ethanol is not a well recognized cause of acute nontraumatic rhabdomyolysis. However, it is well known to cause chronic myopathy.[2] Experiments in human volunteers have shown that it is toxic to the striated muscles.[8] However in acute muscle injury, other mechanisms may also be playing a role i.e. the patient may be comatosed and lying in particular position, over long periods of time, causing continuous pressure on certain parts of body. This would result in muscle compression and capillary occlusion, leading to ischaemia and subsequent rhabdomyolysis.[9] Our patient was never unconscious, though he did sleep for 9-10 hours after binge of alcohol and it is quite possible that some muscle groups were compressed. When the muscles in tight compartments are involved, muscle swelling can lead to compression of nerves with resultant neuropathy. However, our patient did not have any neuropathy.

The usual complication of acute rhabdomyolysis is acute renal failure. When more groups of muscles are injured, a number of substances such as myoglobin, creatine kinase, urate etc. are released. Though myoglobin is more likely to cause acute renal failure, other substances may also be responsible. The present case had myoglobinuria and very high creatine kinase (MM) levels. A good relationship has been observed between CK levels and serum myoglobin levels. Though CK concentrations are commonly elevated myoglobinuria is observed in 37% cases only.[10] However, serum myoglobinaemia is more common as myoglobin does not get bound to any serum protein. Possibility of rhabdomyolysis should be considered in any intoxicated patient with acute muscle paralysis.

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