ORIGINAL RESEARCH PAPER

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RHABDOMYOLYSIS SECONDARY TO ALCOHOL INTOXICATION

KEY WORDS:

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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.

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. Ethanol generally causes chronic myopathy which occurs after its prolonged use. 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.

Case Report

37yrs old male was admitted in D Y Patil hospital with complaints of Decreased urine output since 4-5 days, he also c/o episodes of vomiting since 4-5 days (3-5 episodes per day, non-billous, non projectile, non blood stained), he also c/o Watery loose stools since 3-4 days (3-4 episodes per day, watery in nature, non foul smelling , non bloody). Patient had no other comorbidities.He gave history of consuming country liquor for 3-4 days. He consumed 350 ml alcohol/day .

On examination Pulse 110 bpm ,BP 100/70 mmhg , SpO2-99% on Room air, Afebrile.

No pallor or icterus. He had B/L pitting oedema. There were no other signs of chronic liver disease.

On S/E abdomen was soft non tender, no organomegaly, no fee fluids noted.

Respiratory examination air entry was equal and vesicular there were present no crepitations, On CNS examination he was conscious and oriented in time place and person. There was no neck stiffness or asterixis.

A clinical diagnosis of acute gastroenteritis with alcohol binge drinking was managed accordingly.

While in hospital his urine was noted to be cola coloured He was investigated for the same CBC-Hb-12.1, WBC-9.2, platelets-164 ELECTROLYTES- Na-117, K-4.1, Cl-87 LFT-Bili(total)-1.2, Direct bili-0.6, Indirect bili-0.6

SGOT-3994, SGPT-1093, Albumin-2.7 PT-INR-14.0/0.98 RFT-BUN-100.92, Creat-8.75, uric acid-15.6, Urea-216.2

Urinr R/M-colour-black, blood-+++++, High urine myoglobinuria +++++ Serum Amylase-393

Serul lipase-517 CPK(total)-22392.7 USG ABDOMEN-B/L kidneys bulky in size with altered cortico medullary differentiation.

Treatment-

He was treated with HEMODIALYSIS

DISCUSSION

Ethanol is not a well recognized cause of acute nontraumatic rhabdomyolysis. However, it is well known to cause chronic myopathy. Experiments in human volunteers have shown that it is toxic to the striated muscles. However in acute muscle injury, other mechanisms may also be playing a role i.e. the patient may becomatosed 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. 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. 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.

CONCLUSION

The recommendations for the prevention of ARF are: Fluid administration at a rate that maintains an urine output of at least 300 mL/h as soon as possible (preferably within the rst 6 h of muscle injury) and for at least the first 24 h; Intravenous sodium bicarbonate should preferably be administered only if necessary to correct systemic acidosis although some experts recommend its administration for achieving a urine target PH of 6.5 which promotes myoglobin washout; and Mannitol that should be administered only if needed to maintain desired urine outpu. Urine alkalization has been

shown to inhibit the myoglobin-induced lipid peroxidation which has a causative role for oxidative injury in the renal failure of rhabdomyolysis. Mannitol must be given after volume replacement and must be avoided in patients with oliguria. Systemic corticosteroids may have a role as second line treatment in cases of resistant alcohol-induced rhabdomyolysis co-existing with polymyositis. Intravenous fluids should be continued until CPK levels have declinedpreferably to 1000 IU/L or below 4. The administration of bicarbonate and mannitol may not prevent renal failure, dialysis, or mortality in trauma- induced rhabdomyolysis and CPK levels less than 30,000 IU/L and may be beneficial in patients with higher levels.

In conclusion, clinicians should be aware of alcohol-induced rhabdomyolysis as early recognition and treatment of the disease can prevent life- threatening conditions such as ARF.

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