Ethanol-Associated Prolonged Immobilisation as a Cause of Rhabdomyolysis

Sir,

Rhabdomyolysis is the release of toxic muscle contents into the circulation because of damage to the striated muscles due to traumatic or non-traumatic causes. The release of these toxic substances can cause crush syndrome and acute kidney injury, which is one of the most important clinical problems related to this condition.[1]

Rhabdomyolysis was first described in the 1940s in patients exposed to trauma in the form of crushing under destroyed houses during the Second World War. As a result of muscle ischemia, cells cannot be supplied with oxygen and production of ATP is interrupted. This situation leads to necrosis of muscle cells if it continues for a long time. Compression of the blood vessels locally or due to thrombosis, embolism, compartment syndrome, or systemic causes such as hypotension and shock may impair the blood supply of the skeletal muscle.[2]

Trauma, toxins, and drugs are the most common causes in adults, while infections and congenital disorders predominate in the paediatric population. The most common toxins are ethanol, heroin, methadone, barbiturates, cocaine, caffeine, amphetamine, and benzodiazepines.

Muscle breakdown may be observed due to intense sports activity, status epilepticus, or severe dystonia.[3]

Electrolyte problems such as severe hyponatremia, hypernatremia, hypokalemia, and hypophosphatemia can cause rhabdomyolysis by causing loss of function of the cell membrane.[4]

Prolonged immobilisation due to anaesthesia, coma, or alcohol-induced loss of consciousness can also cause rhabdomyolysis. Prolonged immobilisation due to alcohol is a rare cause of rhabdomyolysis in our country and more rarely, it may cause acute kidney injury.[5]

A 61-year-old male presented to the emergency department with pain in both legs. It was learned from the anamnesis that the patient fell asleep with his feet folded due to the effect of alcohol at night as he was addicted to alcohol. In history, it was learned that he had liver cirrhosis secondary to ethanol. He was conscious, cooperative and oriented at the time of the presentation. Vital signs were stable. Physical examination revealed ecchymoses on both legs. Laboratory results showed serum creatinine of 1.08 mg/dL, creatine phosphokinase of 45,035 U/L, potassium, 5.5 mEq/L, aspartate aminotransferase, 1,018 U/L, and alanine aminotransferase of 160 U/L. Urine analysis showed macroscopic hematuria, but no erythrocytes were detected in microscopic examination. Hydration was started, and the patient was hospitalised. The serum creatinine levels were 2.61 mg/dL, 2.84 mg/dL, 3.01 mg/dL, 3.18 mg/dL, 2.97 mg/dL 2.88 mg/dL, and 2.66 mg/dL for the first seven days, respectively. The patient, who bled from gastroesophageal varices on the 15th day of hospitalisation, died due to haemorrhagic shock.

In conclusion, ethanol-induced prolonged immobilisation should be considered in the aetiology of acute kidney injury in alcohol-addicted individuals.

COMPETING INTEREST:
The authors declared no competing interest.

AUTHORS' CONTRIBUTION:
SO, AO: Study concept and design, initial draft writing, literature search, and final approval.

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