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Fatal Rhabdomyolysis with Acute Kidney Injury following A case of hanging

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Abstract

Rhabdomyolysis, an entity having multiple etiologies, is a complex medical condition characterized by the rapid dissolution of damaged or injured skeletal muscle. Regardless of the cause, the end result is the widespread disintegration of striated muscle which leads to flooding of the extracellular matrix with various ions and molecules that are normally restricted to the intracellular compartment. We present a case which highlights rhabdomyolysis as a potential complication of hanging. It points out the central role of hypoxia in the pathogenesis of rhabdomyolysis.

Keywords: Hanging, Rhabdomyolysis, Myoglobin, Acute Kidney Injury

Introduction

Rhabdomyolysis, an entity having multiple etiologies, is a complex medical condition characterized by the rapid dissolution of damaged or injured skeletal muscle[1]. Regardless of the cause, the end result is the widespread disintegration of striated muscle which leads to flooding of the extracellular matrix with various ions and molecules that are normally restricted to the intracellular compartment. The key compounds released is myoglobin, a 17,800D oxygen carrier[2]. Approximately 33%–50% of patients with rhabdomyolysis develop acute kidney injury (AKI)[3]. The prognosis for myoglobin-induced AKI is excellent and patients are expected to recover fully within 3 months in majority of the cases.

We present a case which highlights rhabdomyolysis as a potential complication of hanging. It points out the central role of hypoxia in the pathogenesis of rhabdomyolysis.

Case presentation

A 28-year-old male was found hanging from the ceiling fan of his room, with a nylon rope, with his feet touching the ground. He was immediately brought down and rushed to the hospital. At presentation, he was comatose

with a GCS of 6/15. General physical examination revealed bradycardia with normal blood pressure. A brownish ligature mark was visible over the anterior aspect of his neck, 1.6 cm wide and 7 cm long, above the level of the thyroid cartilage. Neurologic examination showed bilaterally symmetrical reactive pupils, generalised hypotonia, sluggish deep tendon reflexes and bilateral flexor plantars. Other systems were normal.

Routine laboratory tests at admission including renal function tests were normal except for neutrophilic leukocytosis (total leucocyte count: 17 880/cu.mm, 93% neutrophils). A plain CT of the head and a cervical spine x-ray was done which ruled out surgical injuries. He was diagnosed to have hypoxic ischemic encephalopathy secondary to hanging and was treated with parenteral methylprednisolone, mannitol and the application of a hard cervical collar.

The patient responded well to the therapy, and his sensorium showed steady improvement over the next 24 hours. Blood investigations were repeated serially. Despite maintaining a good urine output, renal function tests deteriorated with serum levels of urea and creatinine peaking at 132 mg/dl and 5.2 mg/dl, respectively on day 5. Patient also developed hyperkalemia with serum potassium level being >7 meq/L. The acute decompensation of renal function was thought to be due to rhabdomyolysis secondary to hypoxia. Relevant investigations were sent. Serum creatine kinase (CPK) was massively elevated (19250 U/l). Urine tested positive for presence of myoglobin. The diagnosis was revised to acute kidney injury due to rhabdomyolysis with myoglobinuria secondary to hypoxia. Nephrology opinion was taken and patient was advised hemodialysis. He underwent 2 cycles of hemodialysis following which, his renal parameters showed an improvement with serum levels of urea and creatinine at 76mg/dl and 1.9 mg/dl respectively on day 8. The patient was discharged the following day. The investigations have been summarized in the following table.

Table 1: Summary of the lab investigations

	Day 1	Day 3	Day 5	Day 8	Day 15
Haemoglobin (g/dl)	13.2	12.8	12.6	12.8	12.9
Total Count (/cu mm)	17880	16750	15430	13450	11290
Platelet Count (/cu mm)	2.48 L	2.22 L	1.90 L	1.82 L	2.03 L
Serum Urea (mg/dl)	24	62	132	76	48
Serum Creatinine (mg/dl)	1.0	1.7	5.2	3.9	1.6
Serum Sodium (mEq/dl)	132	130	125	127	130
Serum Pottasium (mEq/dl)	3.8	5.2	>7	5.4	4.1

Discussion

Speaking theoretically, any form of muscle damage and therefore, any entity that leads to or causes muscle damage, can initiate rhabdomyolysis. According to the data available, the most common causes of rhabdomyolysis include drug or alcohol abuse, trauma, neuroleptic malignant syndrome (nms), and immobility[4].

In a specific case of rhabdomyolysis, the etiology is often known is often known but the exact pathways by which the various insults ultimately lead to muscle injury and necrosis are less clear. But the ultimate result is the rapid dissolution of damaged or injured skeletal muscle which leads to the direct release of intracellular muscle components, including myoglobin, creatine

kinase (ck), aldolase, lactate dehydrogenase, and also electrolytes, into the bloodstream and interstitial space.

One of the key compounds is myoglobin. Myoglobin is usually loosely bound to plasma globulins and only small amounts reach the urine. But the presence of massive amounts of myoglobin exceeds binding capacity of plasma proteins resulting in myoglobin to filtered by glomeruli. It then reaches the tubules, where it may cause an obstruction and renal dysfunction[5]. Urinary myoglobin invokes a typical reddish-brown colour, even in the absence of hematuria. Myoglobin is rapidly eliminated through hepatic metabolism; therefore, tests for myoglobin in plasma or urine are not sensitive diagnostic markers. Elevated serum ck levels are used to establish the diagnosis of rhabdomyolysis. five times higher than the normal value of ck is confirmatory of rhabdomyolysis[6]. Myoglobin gets filtered freely by the glomeruli. In the presence of acidic urine, the concentrated myoglobin precipitates with tamm–horsfall protein to form intratubular casts leading to obstruction of the renal tubules. The heme pigment resulting from myoglobin degradation has a direct tubulotoxic effect, but other precipitating factors such as volume depletion, acidosis, and ischemia also play a role for myoglobinuria to induce aki. Ferrihemate, a degradation product of heme, is more tubulotoxic[7].

Once the diagnosis of rhabdomyolysis is made, the treatment strategy is targeted to prevent kidney injury by preventing the precipitation of myoglobin within the renal tubules. Therefore, the management is directed towards aggressive fluid therapy to maintain an hourly urine output of 150 to 300 ml, and alkalinisation of the urine to a ph beyond 6.5 to prevent dissociation of the ferrihemate complex thereby reducing oxygen free-radical generation, as well as the judicious use of mannitol and loop diuretics[8]. Early diagnosis and volume expansion are vital to reduce the risk of aki. However, renal replacement therapy may be needed in a few cases.

Conclusion

This is the third reported case of rhabdomyolysis following hanging, in medical literature. An exhaustive search for previously reported cases yielded only two others reported cases[9,10], thus making i a very rare primary cause. Nevertheless, it is pivotal for physicians to be aware of rhabdomyolysis as a potential complication of hanging and actively monitor for it. Lab parameters like renal function tests should be repeated serially so that any complication occurring can be treated early and effectively. Early intervention and appropriate management can effectively mitigate this potentially severe complication.

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