



Traumatic Rhabdomyolysis presenting as acute kidney Injury and acute Respiratory Distress Syndrome in young male athlete

Ayush Somani¹✉, Deep Hathi², Sourya Acharya³, Anusha Gupta⁴

¹Resident, department of Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences (Deemed to be University), Sawangi (meghe), Wardha-442001, Maharashtra, India

²Resident, department of Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences (Deemed to be University), Sawangi (meghe), Wardha-442001, Maharashtra, India

³Professor, department of Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences (Deemed to be University), Sawangi (meghe), Wardha-442001, Maharashtra, India

⁴Resident, department of Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences (Deemed to be University), Sawangi (meghe), Wardha-442001, Maharashtra, India

✉Corresponding author

Resident, department of Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences (Deemed to be University), Sawangi (meghe), Wardha-442001, Maharashtra, India

Email: dr.ayush.somani@gmail.com

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General Note



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ABSTRACT

Rhabdomyolysis is a serious, potentially life threatening condition that can develop from muscle injury to infection and toxicity to medication. The classic symptoms of rhabdomyolysis are dark reddish-brown urine, muscle pain and stiffness, muscle weakness, and overall fatigue. Diagnosis of rhabdomyolysis based on myoglobinuria, muscle pain, and extremely elevated circulating creatine kinase values. Here we present a case of exertional rhabdomyolysis with acute kidney injury with acute respiratory distress syndrome (ARDS) that occurs in a healthy, fit 24year old male athlete. Patient was managed conservatively with intravenous fluids and other supportive measures.

Keywords: Rhabdomyolysis; myoglobinuria; athlete; creatine kinase

1. INTRODUCTION

Rhabdomyolysis is a condition in which striated muscle tissue breaks down and the cellular components are released into the extracellular fluid and circulation. Rhabdomyolysis etiology is complex, ranging from severe crushing muscle injury to infection and toxicity to medication (Knochel et al., 1990; Acharya Sourya et al., 2010; Vanholder et al., 2000). Exertional rhabdomyolysis is a type of muscle damage that can be caused by over-exertion exercise. Renal (kidney) failure may result from rhabdomyolysis; it is often warranted for hospitalization to prevent it. Myoglobin released from the damaged muscle cells may precipitate in the kidneys, which may obstruct the renal tubules, resulting in tubular necrosis and ultimately renal failure (Knochel et al., 1990). If left untreated, rhabdomyolysis can be fatal (Dincer et al., 2005). The classic symptoms of exertional rhabdomyolysis were well described, including dark reddish-brown urine, muscle pain and stiffness, muscle weakness, and overall fatigue (Knochel et al., 1990; Acharya Sourya et al., 2010; Vanholder et al., 2000). The myoglobin produced by the muscle damaged will discolor the urine to a reddish brown color (Sauret et al., 2002). During rhabdomyolysis, the damaged muscle also releases massive amounts of creatine kinase (CK) and other muscle proteins into the bloodstream (Vanholder et al., 2000; Sauret et al., 2002). CK in the blood serves as a myoglobin surrogate as it is less expensive and easier to measure. Rhabdomyolysis diagnosis generally involves urine testing to detect myoglobin and blood testing to detect levels of CK. Circulating CK levels is also used to determine treatment progression, as CK levels decrease as rhabdomyolysis resolves (Vanholder et al., 2000).

Here we present a case of traumatic rhabdomyolysis with acute kidney injury with acute respiratory distress syndrome (ARDS) that occurs in a healthy, fit 24year old male following extensive workout in military training camp.

2. CASE REPORT

A 24 year old healthy male, athlete by profession went to Delhi for military training. He was supposed to undergo 15 km running early morning on next day. He started his exercise by running at a good speed, after completing his 6 km run he started experiencing excruciating cramps along left thigh for which he stopped for 5 minutes but he again continued and completed his run. After the practice, the patient reported that when he returned to his room, he noticed excruciating pain in his left quadriceps interfered with ambulation. Throughout the evening, he continued to consume water. Severe pain continued throughout the night in the quadriceps muscles, and the patient reported sleeping difficulty. The patient continued to experience such severe pain the following day that he became concerned and discussed with the assistant athletic trainer about his situation, reporting that he was experiencing dark brown urine and asking for crutches as he had difficulty walking. Since then patient had several episodes of fever with chills and rigors. As he was not able to continue practice, he returned back to his home town and was managed conservatively in local hospital. He noticed decreased in urine output and gradual onset breathlessness so he was referred to our hospital for further management.

At the time of admission, patient had a pulse of 130 beats per minute, respiratory rate of 34/min a blood pressure of 108/64 mmHg, Spo₂-86%, and a temperature of 102.6 F. pao₂/fio₂ ratio was 180 suggestive of ARDS. On respiratory system examination-bilateral crepitation was present, other systemic examination was normal. On local examination, there was local rise of temperature, redness, edematous skin and subcutaneous tissue along left thigh (fig 1).



Figure 1 gross appearance of left thigh

Subsequently swelling increased over 3-4 days and developed ulceration of about 3x2cm above iliac crest. Laboratory investigations were done and Complete blood count (CBC) showed hemoglobin of 8.6gm/dl, white blood count (WBC) - 6000, platelets 2.1 lacs. Total creatine kinase (CK) values of the patient were measured 80,899 U/L with urine positive for myoglobin. Other investigations revealed lactic dehydrogenase (LDH) 11,370 U/L, aspartate aminotransferase (AST) 2,753 U/L, and alanine aminotransferase 468 U/L. Urea was 120, Creatinine 3.2, potassium 5.8 and sodium was normal and chest x ray (fig 2) was suggestive of bilateral diffuse infiltrates. USG thigh revealed edematous swollen appearance of left vastuslateralis and gluteal muscle suggestive of rhabdomyolysis (fig 3).



Figure 2 chest x ray showing bilateral diffuse infiltrates

The patient was unable to bend his knees, and gait could not be tested due to the inability to walk. Besides the severe pain in the patient's quadriceps muscles, he was otherwise alert and healthy with no evidence of infection. The patient did not have a history of muscular dystrophy. Thus patient was diagnosed as a case of Exertional rhabdomyolysis with acute kidney injury and acute respiratory distress syndrome.

The patient was admitted in medicine ICU and was given intravenous (IV) fluids with strict intake-output monitoring. Sodium bicarbonate was added to the IV fluids to alkalinize the urine to prevent myoglobin from precipitating in the kidney tubules.

Injection calcium gluconate with glucose-insulin drip was given thrice a day in view of hyperkalemia. Higher antibiotics were started along with non-invasive ventilator support in view of acute respiratory distress syndrome. Serial levels of CK were monitored which were in falling trend. CK levels and kidney function test (KFT) came within normal limits and patient was able to maintain saturation without any oxygen support after 8 days of aggressive treatment and thus was discharged.



Figure 3 USG thigh showing swollen vastus lateralis.

3. DISCUSSION

Rhabdomyolysis occurs when the membranes of the muscle are disrupted and the cellular contents are not preserved. Myoglobin is a major protein component of the muscle and may overpower the kidneys when released from the muscle (Knochel et al., 1990; Acharya Sourya et al., 2010). Rhabdomyolysis is documented in both highly fit and novice exercisers (Springer et al., 2003; Young et al., 2004). A common thread for developing rhabdomyolysis is overexertion, whether as a result of uncommon novice exercise or a well-conditioned individual's overly vigorous and strenuous (unusual) workout (Knochel et al., 1990). Other factors can also play a role in the development of rhabdomyolysis (Walsworth et al., 2001), such as viral infections, alcohol, and drug use. In our case, we had healthy young male athlete who developed Rhabdomyolysis with acute kidney injury with acute respiratory distress syndrome following exertional exercise (long run).

Rhabdomyolysis contributes to extravasation of the fluid into the third space, decreased intravascular volume, and hypovolemia, resulting in increased sympathetic response, increased secretion of the antidiuretic hormone (ADH), activation of the pathway of renin-angiotensin-aldosterone, and release of endothelin. This results in systemic and renal vasoconstriction. Myoglobin released from muscle cell damage causes vasodilator nitrous oxide (NO) scavenging, which also contributes to vasoconstriction, renal hypoperfusion, reduced glomerular filtration rate (GFR) and eventual acute kidney failure. The increased total blood level of CK has a strong association with increased blood myoglobin. Creatine kinase levels typically rise 2 to 12 hours ahead of rhabdomyolysis and generally peak in 24 to 76 hours. Although no CK value predicts adverse outcomes in ER, a value of 20,000 U / L or 5 times the normal value is considered significant. The excess creatine released into the blood from muscle cells is converted into creatinine to increase plasma creatinine (up to 2 to 4 mg / dl) out of proportion to BUN levels, which is usually normalized within 2 to 3 days. Exact mechanism for ARDS is not understood.

Optimal hydration and alkaline diuresis decreases ischemic ATN and nephropathy of the pigments. In our case, we preserved the renal functions after assessing rhabdomyolysis by administering NaHCO₃ and sufficient fluid infusion until the serum CPK levels decreased. We supported the respiratory functions through non-invasive mechanical ventilation and administration of analgesics until arterial blood gas values and chest radiography improved.

4. CONCLUSION

Rhabdomyolysis, a potentially life-threatening condition, may occur as a result of substantial over-exertion. Though acute kidney injury is a common complication of rhabdomyolysis, ARDS is rare one. For the management of rhabdomyolysis, the early onset of alkaline diuresis and the availability of adequate medical and mechanical ventilation support for ARDS are vital in maintaining renal and respiratory functions and in preventing morbidity and mortality.

Informed Consent

Proper consent was taken from patient for writing case report.

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Conflicts of Interest: The authors declare no conflict of interest.

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