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Case Report

Pulse steroids serve in rhabdomyolysis secondary to strenuous exercise in a 17-year-old male

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ABSTRACT

Rhabdomyolysis is a potentially life-threatening condition characterized by the breakdown of skeletal muscle fibers and the subsequent release of intracellular components, such as myoglobin and creatine kinase (CK), into the bloodstream. This case report describes a 17-year-old male who developed rhabdomyolysis secondary to strenuous exercise, specifically after performing 200 sit-ups. The patient presented with typical symptoms, including muscle pain, swelling, dark urine, and decreased urine output. Initial laboratory investigations revealed significantly elevated CK levels (193,500 U/L), indicating severe muscle damage. Prompt management included aggressive hydration, electrolyte monitoring, and supportive care. Notably, corticosteroid therapy (methylprednisolone) was initiated on day 3 based on consensus opinion and literature review, although the use of corticosteroids in rhabdomyolysis remains controversial. The patient's condition improved with treatment, as evidenced by a gradual decline in CK levels and normalization of liver enzymes. Renal function remained stable, and the patient did not develop acute kidney injury, a potentially severe complication of rhabdomyolysis. This case highlights the importance of prompt recognition and aggressive management of exertional-induced rhabdomyolysis, particularly in young individuals engaging in strenuous physical activity. Early intervention, including corticosteroid therapy in selected cases, can help prevent potentially life-threatening complications and facilitate a successful outcome.

Keywords: Exertional rhabdomyolysis, Creatine phosphokinase, Acute kidney injury, Myoglobinuria, Strenuous exercise, Compartment syndrome, High-dose corticosteroids, N-acetylcysteine infusion

INTRODUCTION

The word rhabdomyolysis is derived from the Greek words rhabdos (rod-like/striated), mus (muscle), and Lucis (breakdown). Rhabdomyolysis means dissolution of skeletal muscle, and it is characterized by leakage of muscle cell contents, myoglobin, sarcoplasmic proteins (creatine kinase, lactate dehydrogenase, aldolase, alanine, and aspartate aminotransferase), and electrolytes into the extracellular fluid and the circulation.[1] In a 10-year review of cases of rhabdomyolysis at a regional tertiary care center, 631 patients were identified. 383 patients met the definition of rhabdomyolysis and were included in the analysis. 220 were males and 163 were females, with a median age of 71 years. The leading causes of rhabdomyolysis were prolonged immobility (71%), infection (6%), decreased blood supply (5%) and physical exertion (4.2%).

AKI was present in 229 patients (59.8%) on presentation and 38 patients (9.9%) required renal replacement therapy. The mean peak serum creatinine was 212 µmol/l. Death resulted in 7.6% of patients during their admission.[2] Acute kidney injury (AKI) is a life-threatening complication of rhabdomyolysis. A recent

Chinese epidemiological survey found that the overall incidence of acute kidney injury (AKI) is 11.6%, with in-hospital mortality of AKI being 8.8%.[3] Patient outcomes are worse in RM patients with AKI, which increases the medical burden. Therefore, prevention and early diagnosis of AKI improve the prognosis for these patients.[4]

Although direct muscle injury remains the most common cause in RM, additional causes include hereditary enzyme disorders, drugs, toxins, endocrinopathies, malignant hyperthermia, neuroleptic malignant syndrome, heatstroke, hypothermia, electrolyte alterations, diabetic ketoacidosis and non-ketotic hyperosmolar coma, severe hypo- or hyperthyroidism and bacterial or viral infections.[5]

The earliest experimental studies that described the involvement of myoglobin in the development of rhabdomyolysis were conducted in the early 1940s. Bywaters and Stead administered myoglobin injections to rabbits and found that this "toxin" was the cause of acute respiratory failure (ARF) after muscle damage.[6] Massive necrosis, expressed as limb weakness, myalgia, edema, and, frequently, extensive pigmenturia without hematuria, is the common denominator of traumatic and nontraumatic rhabdomyolysis.[7]

The clinical presentation of rhabdomyolysis may vary, ranging from an asymptomatic increase in serum levels of enzymes released from damaged muscles to worrisome conditions such as volume depletion, metabolic and electrolyte abnormalities, and acute kidney injury (AKI). The diagnosis is confirmed when the serum creatine kinase (CK) level is > 1000 U/L or at least 5x the upper limit of normal. Other important tests to request include serum myoglobin, urinalysis (to check for myoglobinuria), and a full metabolic panel including serum creatinine and electrolytes.[8]

Compartmental syndrome is not a rare finding in rhabdomyolysis.[2,9] Most skeletal muscles are enfolded in confined, inflexible compartments created by bones, fascia, and other structures.

Compartmental syndrome develops more often in the compartments of the extremities, such as the anterior compartment (containing the biceps-brachialis muscle) and the posterior compartment (containing the triceps muscle) of the upper arm, the volar compartment (wrist and finger flexors) and dorsal compartment (wrist and finger extensors) of the forearm, the three gluteal compartments, the anterior and posterior compartments of the thigh, and the four compartments of the lower leg.[9]

Signs and symptoms of compartment syndrome (CS) include the “6 P’s”, of which pain out of proportion to the injury and increased analgesic requirements should raise suspicion of developing compartment syndrome,22 other signs appear late and indicate delayed diagnosis, including pallor, paralysis, and absence of pulses.[10]

CASE REPORT

A 17-year-old male with no comorbidities presented with symptoms suggestive of rhabdomyolysis following a strenuous exercise routine involving 200 sit-ups. He experienced burning micturition, fever with chills, pedal edema, decreased urine output, dark urine, and muscle stiffness and pain over both limbs. Upon examination, cardiovascular findings were normal, but respiratory examination revealed bilateral adventitious breath sounds. The abdomen was soft, and abdominal ultrasound findings were unremarkable.

Investigations

Laboratory investigations on day 1 revealed elevated levels of creatine phosphokinase (CPK) and liver enzymes, with normal renal function. Subsequent investigations on days 2 and 3 showed a declining trend in CPK levels, along with normalizing renal parameters (Table 01). Day 4 investigations demonstrated further improvement in CPK levels and liver enzymes. By day 8, CPK levels had significantly decreased, indicating the resolution of muscle damage.

Treatment during hospitalization included N-acetylcysteine infusion, cholecalciferol supplementation, pantoprazole, and mucolytic agents. Corticosteroid therapy (Injection Methylprednisolone Sodium succinate 500mg) was initiated on day 3 based on consensus opinion and literature review.

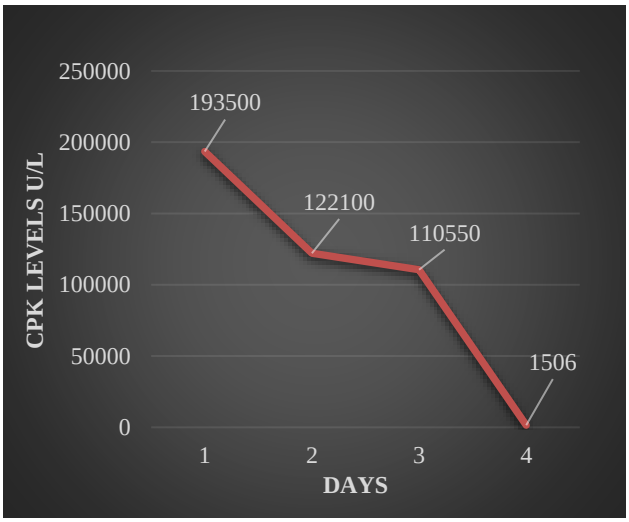

Figure 1: Graphical representation of gradual decrease in CPK levels.

Diagnosis and management

Based on the clinical presentation and laboratory findings, a diagnosis of rhabdomyolysis secondary to

intense exercise was made. The patient was immediately started on aggressive hydration therapy to prevent acute kidney injury and facilitate the clearance of myoglobin. Supportive measures, including electrolyte monitoring and pain management, were instituted. The patient was advised to refrain from strenuous physical activity until complete recovery.

Follow-up

The patient's condition was closely monitored over subsequent days to assess renal function, electrolyte balance, and resolution of symptoms. Repeat laboratory investigations and clinical assessment were performed to guide ongoing management and ensure a favourable outcome.

Treatment and discharge

The patient showed clinical improvement with treatment and was discharged in a hemodynamically stable condition on day 4.

Discharge medications included mucolytic agents, pantoprazole, and analgesics as needed, along with cholecalciferol supplementation. The patient was advised to maintain adequate oral fluid intake and follow-up after three days for review.

Table 1: Laboratory investigations.

Parameters	Day	Value	Biological reference interval
Complete blood picture			
Hb	3	14.7	13.0- 17.0 gm%
	4	14.9	
	8	14.7	
WBC	8	11500	4000- 10000 cells/cumm
RBC	8	5.58	4.5- 5.5 millions/cumm
Platelets	8	3.17	1.5-4.1 lakhs/cumm
Liver function test			
Total bilirubin	1	0.62	0.3- 1.2 mg/dl
	4	0.54	
Direct bilirubin	1	0.13	<0.2 mg/dl
	4	0.15	
Indirect bilirubin	1	0.49	T.B-D.B
	4	0.39	
Alkaline phosphatase	1	69	54-369 U/l
	4	80	
SGPT/ALT	1	608	<45.0 IU/l
	4	661	
SGOT/ AST	1	2434	<50.0 IU/l
	4	1400	
Total protein	1	6	6.4- 8.3 mg/dl
	4	6.8	
Albumin	1	3.8	3.5-5.0 gm/dl
	4	4.2	
Globulin	1	2.2	2- 3.5 gm/dl
	4	2.6	
Renal function test			
Creatinine	1	0.98	Male-0.67- 1.17 mg/dl
	3	0.92	
	4	0.78	
	8	0.79	
Uric acid	1	3.7	Male-3.5- 7.2 mg/dl
Urine myoglobin	4	Negative	
Biochemistry			
CPK	1	193500	Male- <171.0 U/l
	2	122100	
	3	110550	
	8	1506	

DISCUSSION

The case presented describes a 17-year-old male with symptoms suggestive of rhabdomyolysis following an intense exercise routine. Rhabdomyolysis is a syndrome characterized by muscle breakdown and the release of intracellular contents, including myoglobin, into the bloodstream.

It typically occurs as a result of muscle injury or damage, which can be caused by various factors such as trauma, drug toxicity, infections, metabolic disorders, or extreme physical exertion, as seen in this case. Most strenuous exercise results in an increase in blood CK activity because of the stress placed on the contracting muscle fibers. Peaks of up to 80,000 U/l have been reported after exercise with no clinical consequence.[11]

Currently, there are no set guidelines for the diagnosis of rhabdomyolysis. CK level greater than five times the upper limit is a common finding in rhabdomyolysis.[12]

The initiation of corticosteroid therapy on day 3 raises some considerations. While corticosteroids have been suggested in some cases of severe rhabdomyolysis to mitigate inflammation and tissue damage, the decision to use them should be based on a careful assessment of individual risk factors and benefits. It's noteworthy that corticosteroids were initiated based on consensus opinion and literature review, indicating a lack of clear-cut guidelines for their use in rhabdomyolysis.

The muscle weakness and respiratory failure gradually improved after intravenous methylprednisolone administration, and the serum CK level was normalized in several days.[13] The patient's favorable response to treatment, as evidenced by clinical improvement and declining CPK levels, underscores the importance of prompt recognition and aggressive management of rhabdomyolysis.

Close monitoring during hospitalization and follow-up assessments are essential to track renal function, electrolyte balance, and overall recovery.

CONCLUSION

This case highlights the importance of recognizing exertional-induced rhabdomyolysis and its potential complication, compartment syndrome, particularly in young individuals engaging in strenuous physical activity. Prompt diagnosis and appropriate management, including High-dose corticosteroids with hydration, supplementation, and supportive care in the short duration, the patient achieved a favorable outcome and prevented long-term complications.

Throughout the treatment, the patient's condition improved, with a gradual decline in CPK levels and normalization of liver enzymes, indicating the resolution of muscle damage (Fig.1). Renal function remained stable, and the patient did not develop acute kidney injury, which is a common and potentially life-threatening complication of rhabdomyolysis. Education regarding safe exercise practices and monitoring for warning signs of muscle injury are crucial in preventing recurrence.

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