DOI: 10.7860/JCDR/2015/10275.6507

Case Report

Anaesthesia Section

Sports Induced Cardiac Arrest: A Case of Missed Rhabdomyolysis

PARSHOTAM L GAUTAM[1], NEERU LUTHRA[2], PRABHDEEP SINGH NAIN[3]

ABSTRACT

Exercise induced rhabdomyolysis although uncommon, is well known in strenuous and exhaustive sports like marathons, cycling and wrestlers. But it is not known in Kabaddi players. We report a case of nearly fatal rhabdomyolysis which was missed during early resuscitation in emergency room and lead to cardiac arrest due to catastrophic metabolic acidosis and severe -hyperkalemia. After high quality cardiopulmonary resuscitation and return of spontaneous rhythm, emergency resuscitative exploratory laparotomy was performed for suspected bladder injury which was negative. He had remarkable recovery over 24 h following diagnosis and aggressive supportive management including peritoneal dialysis. Heat stroke and rhabdomyolysis should be suspected early in players playing strenuous sports in tropical countries even during winter. High degree of suspicion and early aggressive general support is the key to success for unusual clinical presentation of any such clinical entity.

Keywords: Acute renal failure, Creatine kinase, Hyperkalemia

CASE REPORT

An 18-year-old, 64 kg boy with body mass index 19 Kg/m^2 was brought to emergency room of tertiary care hospital after 10 h of alleged history of lower abdominal trauma while playing Kabaddi tournament. Kabbadi is an Asian game which involves wrestling, running with multiple players. He was restless and irritable on admission with cold clammy peripheries. There was no history of any drug intake, abuse or doping. His vitals were: pulse 129/min, blood pressure 100/70 mmHg, and respiratory rate 30/min. He was afebrile. His systemic examinations were inconclusive. Abdomen was not distended. There was no limb injury. Two litres of normal saline was rushed intravenously and oxygen was supplemented. Urethral catheterization drained 50 ml of dark red colored urine. Portable abdominal ultrasound showed moderate amount of free fluid in peritoneal cavity with empty bladder. In view of alleged abdominal trauma it was thought that he had hematuria secondary to bladder injury and he was taken up for surgery immediately keeping a provisional diagnosis of intestinal perforation. Investigations at admission are as shown in [Table/Fig-1] with ABG showing severe metabolic acidosis [Table/Fig-1].

He had cardiac arrest in preoperative area. With immediate high quality cardiopulmonary resuscitation for 20 min, normal sinus rhythm returned. He was given 50 ml of $NaHCO_3$ 8.4% during resuscitation. Emergency resuscitative exploratory laparotomy was done in view of deteriorating hemodynamics and free fluid in peritoneal cavity which was negative for haemoperitonium. He was shifted to ICU on ventilator with 100% oxygen and started with dopamine 15 µg/kg/hr, norepinepherine 0.5µg/kg/min and epinephrine 2 µg/min. He had pink froth from tracheal tube suggesting pulmonary edema. Repeat serum potassium level was 8mEq/L along with electrocardiographic (ECG) changes of hyperkalemia and associated severe metabolic acidosis (ABG in [Table/Fig-1]). Hyperkalemia was aggressively treated with injection calcium gluconate 10% 10 ml, sodium bicarbonate 8.4% 100 ml and dextrose 25% 100 ml with 10 units regular insulin, injection furosemide 40 mg intravenously followed by five cycles of peritoneal dialysis through abdominal drain. Prolonged QT syndrome which was present in the first ECG was now corrected. Subsequent EKG was normal. Patient's hemodynamics was stabilized over few hours with correction of hyperkalemia and metabolic acidosis. Further investigations like MRI, electromyography and musle biopsy were not done as the patient improved clinically. He gained consciousness and trachea was extubated next day. Transthoracic echocardiography done later in ICU was normal. He was discharged from ICU on the third day.

DISCUSSION

No comprehensive data on the overall prevalence of rhabdomyolysis are available because occurrence is highly dependent on the underlying cause [1]. Rhabdomyolysis has been reported in marathon runners, cyclists and rarely with unaccustomed mild exertion [2-4]. However, it has been not reported in Kabbadi game. Few authors have explored the issue but largely question remains unanswered that why some of these subjects fail to cope up the exertional stress. Regular training leads to cellular adaptation at molecular level with repeated exposure to hypoxia. Prolonged hypoxia causes the degradation of plasma membrane Na-K-ATPase as a mechanism of cellular adaptation. This occurs with specific molecular pathways requiring enzymes which can be deficient or absent in some individuals. Genetically, absence of these enzymes in some individuals may be cause of early rhabdomyolysis with mild exertion and its complications [5]. Kabaddi (sometimes transliterated Kabbadi or Kabadi) is a South Asian team sport. It is a combative, strenuous sport like basketball or soccer. Kabaddi has received international recognition during the 1936 Berlin Olympics. As in other combative sports, kabaddi players are prone to concussions, distortions and other orthopaedic injuries.

Patients with acute rhabdomyolysis classically present with the triad of muscle weakness, muscle pain, dark urine and associated evidence of physiological or direct insult to muscles. The clinical profile might suggest rhabdomyolysis, but definitive diagnosis can only be confirmed through laboratory investigations. In the absence of cerebral or myocardial infarction, it is generally agreed that creatine kinase (CK) levels five times the normal concentration (approximately 1000 IU/L) are highly suggestive of rhabdomyolysis and values higher than 5000 IU/L warn about against development of acute renal failure. CK levels initially rise within the first 12 hours of muscle destruction, peak after one to three days and decline three to five days after muscle injury ends [6-8]. Myoglobin, a direct product of muscle damage may be assessed in serum or urine but is unreliable due to its unpredictable metabolism and short half life. Normally urine is assessed for myoglobinuria [6,7]. Some of the well known complications of rhabdomyolysis are acute kidney injury, disseminated intravascular coagulation (DIC), compartment syndrome and severe electrolyte abnormalities.

Our patient had unique presentation with history of abdominal trauma which delayed the diagnosis. He never had any fever during this episode. He had biochemical features of hemoconcentration and signs of renal failure. ECG showed prolonged QT but recovered

Investigations	At the time admission		After 12 Hours	Day2	Day3
Haemoglobin (g/L)	186		171	133	136
Hct%	54		51	38.8	39.8
TLC (cells x 10^9/L)	21.2		15.4	10.0	8.0
DLC (polymorphs/ lymphocytes/ Eosinophil/ monocytes / basophil)	69/24/3.1/3.2 /0.7		91/4. 7/3.8/ 0/0	85/8. 3/6.0/ 0.1/0	74/13/6. 8/5.4
Platelets (cells x 10^9/L)	199		198	151	122
INR	1.27				
Glucose (mmol/L)	6.5				
Urea (mmol/L)	11.07		16.78	24.28	10.36
Creatinine (µmol/L)	212.1		194.5	167.96	92.8
Na (mmol/L)	143		140	143	145
K (mmol/L)	6.67		8.7	3.7	3.8
Cl (mmol/L)	95		100	108	109
Ca (mmol/L)			3.1		
Phosphorus (mmol/L)			3.8		
Bilirubin Total (conjugated) mmol/L			3.1		21.38 (3.93)
AST/SGOT (U/L)			3.8		178
ALT/SGPT (U/L)			368		208
Alkaline phosphatise (U/L)			167		70
Total serum proteins (g/L)			68		48
Serum Albumin (g/L)			40		30
Albumin / globulin (A/G) ratio			1.4		1.3
Creatine kinase (IU/L)			3849		2532
Creatine phosphokinase myocardial band (IU/L)			105		
Urine for myoglobin			Negative		
Arterial blood gas analysis	Before cardiac arrest (at admission)	After cardiac arrest			
pH	7.05	7.03	7.23	7.39	7.36
$PaCO_2$mmHg	32	96	55	45	36
PaO_2 mmHg	134	74	56	136	123
HCO_3 mmol/L	9.2	22.8	22.8	26.6	26.0
BE_ECF mmol/L	-20.	-14	-6.0	+1.6	2.3
Lactate mmol/L	4	5			

[Table/Fig-1]: Investigations

with correction of hyperkalemia. An important finding in repeated blood gases was hypercarbia. It was probably due to dead space ventilation associated with poor circulation. Possibility of malignant hyperthermia is unlikely as rhabdomyolysis occurred without fever. The prevailing climate does not support diagnosis of heat exhaustion but possibility cannot be ruled out as he was playing an aggressive outdoor game. Rhabdomyolysis is certain as CK level were raised (3849 IU/L) but it is difficult to ascertain it as we do not have histopathological evidence. Whether it was due to afebrile malignant hyperthermia or underlying genetic propensity to rhabdomyolysis remains unclear. Negative myoglobinuria report may be spurious as bladder was flushed during surgery. The long list of known causes for rhabdomyolysis includes trauma, drug intoxication, alcoholism, hyperpyrexia, vascular occlusion, infections, insect bite, electrolyte imbalances, heat intolerance, seizures, severe exertion, substance abuse and pure vegetarian diet in athletes. None of the above mentioned risk factors was present in our case. Incidence of acute renal failure in rhabdomyolysis has been reported up to 40%. Ward in a historical cohort study reported acute renal failure in 16.5% of 157 patients with rhabdomyolysis over a two-year period. He suggested that predictors for the development of renal failure include peak CK level more than 6000 IU/L, dehydration (hematocrit >50, serum sodium level >150 mEq/L, orthostasis, pulmonary wedge pressure < 5 mm Hg, urinary fractional excretion of sodium<1%), sepsis, hyperkalemia or hyperphosphatemia on admission, and the presence of hypoalbuminemia [9]. In our case, there was exertional rhabdomyolysis with low creatine phosphokinase levels which has not been reported earlier. It highlights the difficulties faced by surgeons, anaesthetists and emergency teams in distinguishing abdominal pain from exercise induced rhabdomyolysis, which can occur at any level of exercise intensity even in healthy individuals in a comfortable weather. Lack of awareness of this phenomenon may lead to delay and inappropriate management with catastrophic outcome. Although it is single case report, but the response of our patient to peritoneal dialysis suggests that early institution of peritoneal dialysis may prevent evolving acute renal failure and cardiovascular shock in rhabdomyolysis. Due to renal shutdown forced bicarbonate diuresis was not done. It is important for sports, emergency, critical care physicians, anaesthesiologists and surgeons handling players for emergency management of injuries to have high suspicion about association of heat exhaustion, trauma and coacine like drugs with rhabdomyolysis [10].

CONCLUSION

To conclude, rhabdomyolysis can occur after high strenuous exercise or sports with variable clinical presentation. All red coloured urine is not haemturia and can be haemoglobinuria or myoglobinuria. High degree of suspicion can ascertain the diagnosis for definite care and good general support is paramount to tide over crisis and buy time to reach diagnosis.

REFERENCES

[1] Criddle LM. Rhabdomyolysis Pathophysiology, Recognition and Management. *American Association of Critical-Care Nurses*. 2003;23:14-30.
[2] Tunstall PDS. Marathon cardiac deaths: the London experience. *Sports Med.* 2007;37(4-5):448-50.
[3] Rae DE, Knobel GJ, Mann T, Swart J, Tuckerand R and Noakes T D. Heatstroke During Endurance Exercise: Is There Evidence for Excessive Endothermy? *Medicine and Science in Sports and Exercise®*. 2008;40(7):1193-204.
[4] Senert R, Kohl L, Rainone T and Scalea T. Exercise-induced rhabdomyolysis. *Ann Emerg Med*. 1994;23:1301-06.
[5] Carpenter D, Robinson RL, Quinnell RJ, Ringrose C, Hogg M, Casson F. Genetic variation in RYR1 and malignant hyperthermia phenotypes. *Br J Anaesth*. 2009;103:538–48.
[6] Huerta-Alardín AL, Varon J, Marik PE. Bench-to-bedside review: rhabdomyolysis—an overview for clinicians. *Crit Care*. 2005;9(2):158–69.
[7] Cervellin G, Comelli I and Lippi G. Rhabdomyolysis: historical background, clinical, diagnostic and therapeutic features. *Clin Chem Lab Med*. 2010;48(6):749–56.
[8] Bosch X, Poch E, Grau JM. Rhabdomyolysis and acute kidney injury. *N Engl J Med*.2009;361(1):62–72. Erratum in: *N Engl J Med*. 2011;364(20):1982.
[9] Ward MM. Factors predictive of acute renal failure in rhabdomyolysis. *Arch Intern Med*. 1988;148(7):1553-57.
[10] Nemiroff L, Cormier S. Don't you forget about me: Considering acute rhabdomyolysis in ED patients with cocaine ingestion. *Can Fam Physician*. 2012;58(7):750–54.

PARTICULARS OF CONTRIBUTORS:
1. Professor and Head, Critical Care Division, Department of Anaesthesiology, Dayanand Medical College & Hospital, Ludhiana-141 001, Punjab, India.
2. Assistant Professor, Department of Anaesthesiology, Dayanand Medical College & Hospital, Ludhiana-141 001, Punjab, India.
3. Professor, Department of General Surgery, Dayanand Medical College & Hospital, Ludhiana-141 001, Punjab, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Neeru Luthra,
9225/1, Street no.1, Joshi Nagar, Ludhiana-141001, Punjab, India.
E-mail : drneeru1977@yahoo.co.in

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Jun 10, 2014**
Date of Peer Review: **Jul 28, 2014**
Date of Acceptance: **Mar 02, 2015**
Date of Publishing: **Sep 01, 2015**