Exertional Rhabdomyolysis Causing Acute Renal Failure

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Introduction

Traumatic rhabdomyolysis causing myoglobinuria and acute renal failure (ARF) was initially described in 1941 in soldiers with multiple crush injuries during Second World War [1]. Since then several non-traumatic conditions leading to rhabdomyolysis and myoglobinuric renal failure have been recognized [2,3].

Excessive muscular activity is increasingly recognized as a common and preventable cause of rhabdomyolysis. Strenuous and exhaustive exercise, especially in unconditioned men (so called 'white collar rhabdomyolysis') can result in major morbidity from hyperkalemia, metabolic acidosis, disseminated intravascular coagulation, adult respiratory distress syndrome and rhabdomyolysis [4]. Though rhabdomyolysis following severe physical exertion with or without heat stress may result in ARF, it is rare [5]. We report a case of exertional rhabdomyolysis complicated by ARF in a healthy individual who was well acclimatized to physical exertion and with no evidence of heat stress.

Case Report

A 21 year old military recruit was brought to the hospital in the month of October, with history of having collapsed during an endurance run of 16km in a hilly terrain. He was in normal health before undertaking the exercise with no relevant past history of illness. He had completed eight months of training and never before developed similar illness. Examination showed no pallor, cyanosis or pedal edema. He was in a state of shock with dry tongue, feeble peripheral pulses and systolic blood pressure of 70mm of Hg. He was resuscitated with IV fluids (N-saline and Ringer lactate) and he improved hemodynamically. Investigations showed Hb 15gm%, urea 78mg% and creatinine 1.8mg%. On the next day he developed diffuse tender swelling of both lower limbs and hypertension and continued to be anuric. Urinary catheterization yielded 80ml urine which showed 15-20 RBCs per high power field and tested positive for myoglobin. Urine

spot sodium was 95mEq/L. Other investigations revealed serum calcium 9.2mg%, phosphorus 5.3mg%, sodium 130mEq/L, potassium 5.3mEq/L, bilirubin 1.0mg%, AST 57IU/L, ALT 48 IU/L, Alkaline phosphate 184 IU/L, creatine phosphokinase (CPK) 1122IU/L and lactate dehydrogenase (LDH) 4744IU/L. Ultrasound showed right kidney of 12.1cm and left kidney of 12.9cm increased cortical echogenicity and prominent pyramids and normal pelvicalyceal system. He was negative for HbsAg, anti-HCV and HIV. Blood and urine cultures were sterile. He showed progressive worsening of azotemia (urea 185mg%, creatinine 11.9mg%) and was taken up for haemodialysis next day and subsequently required 7 sessions of hemodialysis. He remained oliguric for 14 days and thereafter entered into a diuretic phase and made an uneventful recovery. His metabolic parameters a month later were urea 43mg% and creatinine 1.0mg%.

Discussion

Rhabdomyolysis is defined as a clinical and laboratory syndrome resulting from skeletal muscle injury with release of muscle cell content into the plasma. This may result in visible myoglobinuria, i.e. red or brown urine. Rhabdomyolysis could be traumatic or non-traumatic, trauma being the commoner. The list of causes of non-traumatic rhabdomyolysis is exhaustive and includes sports, seizures, status asthmaticus and delirium tremens; muscle ischemia due to sickle cell trait, vascular obstruction, air embolism; immunological causes like dermatomyositis and polymyositis; metabolic causes like diabetes, hypokalemia, hypothermia, myxedema; drugs, toxins and infectious diseases (influenza-like illness, sepsis and gangrene). Despite a long list of causes, rhabdomyolysis as such is not very common. The pathognomonic features of rhabdomyolysis are myoglobinuria and elevated serum aldolase and CPK. Rhabdomyolysis following severe physical exertion with or without heat stress resulting in ARF is rare. In our case ARF with rhabdomyolysis was observed in a healthy individual who had been reasonably well acclimatized

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to physical exertion, having finished 8 months of basic military training. There was no evidence of heat stress with exercise undertaken in the early hours of October at a hill station. Uberoi et al [6] reported 7 cases over a period of 6 years with ARF due to exercise induced myoglobinuria in the absence of heat stress. Another series of 8 cases was reported from a Naval Officers training institute [7]. Ramamoorthy et al [8] described myoglobinuria with ARF in a 19 year old boy, who performed a three hours continuous dance programme on a hot humid summer afternoon.

Diagnosis of myoglobinuria is made by a positive orthotoluidine test in a urine sample free of RBCs, and an elevated CPK and myoglobin in serum. The orthotoluidine test is not very sensitive and hence other tests like spectrophotometry should be done for a definite diagnosis [9]. A urine sediment with tubular epithelial cells, pigmented granular casts and occasional RBCs is the usual finding in cases of myoglobinuric renal failure. The microscopic hematuria observed in our patient was probably due to urinary catheterization. Microscopic hematuria in myoglobinuric ARF can occur due to traumatic rhabdomyolysis. Since the urinary dipstick test and orthotoluidine test do not distinguish between hemoglobin and myoglobin in the presence of RBCs in urine, diagnosis of rhabdomyolysis with myoglobinuria is made by demonstrating a positive test on the supernatant urine sample, normal colour of serum (i.e. absence of hemolysis) and elevated CPK and aldolase.

The major life threatening complication of myoglobinuria is acute tubular necrosis, as occurred in our case. The exact mechanism by which ARF results from myoglobinuria is not well understood. Postulated mechanisms are direct tubulo-toxic effect of ferrihemate or myoglobin, obstruction to tubular lumen by myoglobin casts, back diffusion of glomerular filtrate through a break in the epithelium and decreased glomerular filtration rate. Dehydration, heat stress, hypovolemia and acidification of urine are crucial precipitating factors. Our case had dehydration and hypovolemic shock at the time of presentation. Renal involvement is characterized by oliguria, exceptionally high creatinine levels, hyperkalemia, hyperphosphatemia and hyperuricemia. Serum calcium may be low in the oliguric phase and later in the diuretic phase patients may develop hypercalcemia. Our patient had hyperphosphatemia, hyperkalemia and hyperuricemia but normal serum calcium levels.

Treatment of ARF due to myoglobinuria is by volume replacement, hemodialysis and supportive measures. Alkaline solute diuresis and infusion of mannitol or sodium bicarbonate improve renal function, if initiated early. The clinical course of ARF due to rhabdomyolysis is not different from other causes of ARF and mortality has been reported to be up to 29.3%. Our case recovered completely, possibly because he was healthy, well acclimatized and had the benefit of early diagnosis and energetic management.

Cases presenting with rhabdomyolysis following strenuous exercise should be evaluated for McArdle's disease, a primary myopathy due to myophosphorylase deficiency. The disease is inherited as an autosomal recessive trait and is characterized by excessive fatigability, cramps and myoglobinuria following physical exercise. The diagnosis is confirmed by ischemic forearm exercise test and muscle biopsy. Since our patient had no symptoms of muscle cramps or pains following physical exertion or limitation of physical activity in past, McArdle's disease was not considered. There is only an occasional report of McArdle's disease presenting with acute renal failure in the absence of a past history of exercise-induced muscle pain and stiffness [10].

References

1. Bywaters EQL, Beall D. Crush injuries with impairment of renal function. *Br Med J* 1941; 1: 427-32.
2. Black C, Jick H. Etiology and frequency of rhabdomyolysis. *Pharmacotherapy* 2002; 22: 1524-6.
3. Singh S, Sharma A, Sharma S, Sud A, Wanchu A, Bamberg P. Acute alcoholic myopathy, rhabdomyolysis and acute renal failure: a case report. *Neurol India* 2000; 48: 83-5.
4. Knochel JP. Catastrophic medical events with exhaustive exercise: "White collar rhabdomyolysis". *Kidney Int* 1990; 38: 709-19.
5. Braseth NR, Allison EJ Jr, Gough JE. Exertional rhabdomyolysis in a body builder abusing anabolic androgenic steroids. *Eur J Emerg Med* 2001; 8: 155-7.
6. Uberoi HS, Dugal JS, Kasthuri AS, Kolhe VS, Sampath Kumar AK, Cruz SA. Acute Renal Failure in Severe Exertional Rhabdomyolysis. *J Assoc Physicians India* 1991; 39: 677-9.
7. Smith RF. Exertional rhabdomyolysis in naval officer candidates. *Arch Intern Med* 1968; 121: 313-9.
8. Ramamoorthy KP, Varghese AM, Ravikumar P, Naik PM, Narayan R. Myoglobinuria after exercise – a rare presentation. *Ind J Nephrol* 2002; 12: 61-2.
9. Beetham R. Biochemical investigation of suspected rhabdomyolysis. *Ann Clin Biochem* 2000; 37: 581-7.
10. McMillan MA, Hallworth MJ, Doyle D, Briggs JD, Junor BJ. Acute renal failure due to McArdle's disease. *Ren Fail* 1989; 11: 23-5.