A Runners Nightmare: Rhabdomyolysis strikes, Kidneys suffer: A Case Report

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Abstract

Exercise Induced Rhabdomyolysis (EIR) refers to breakdown of striated myocytes and release of their contents into circulation after prolonged intense physical activity. The presentation is variable from asymptomatic to symptoms like intense muscle pain, weakness and dark tea-coloured urine. Potential complications like volume depletion, metabolic and electrolyte abnormalities, acute kidney injury (AKI), multi organ failure and death can occur if early diagnosis and treatment is missed. A high index of suspicion is important to avoid this. We report a case of EIR causing AKI in a 44-year-old male who presented with lower limb muscle pain for 5 days after running 70 km marathon followed by symptoms of fluid overload requiring haemodialysis.

Key words: Exercise Induced Rhabdomyolysis (EIR), Acute Kidney Injury (AKI), Haemodialysis (HD).

Introduction

Rhabdomyolysis refers to injury to striated muscle leading to the release of its intracellular contents (myoglobin, electrolytes, creatine kinase, aldolase, lactate dehydrogenase, ALT, AST) into the systemic circulation (1). It can occur due to many causes, exercise being one of them.

Most of the time, this damage is reversed without any adverse effects. It is not common for patients to seek medical attention on developing any clinical symptoms after exercise.

The exact global incidence of EIR thus remains unknown due to underreporting of cases and lack of prospective studies. EIR is common amongst males especially African-Americans, age <10 or > 60 years and morbidly obese BMI>40 (2).

Rhabdomyolysis can complicate with AKI, metabolic and electrolyte derangements (hyperuricemia, hyperkalaemia, hyperphosphatemia), disseminated intravascular coagulation, arrythmias, in severe cases, multi-organ failure and death. About 10–40% of patients with rhabdomyolysis develop AKI. AKI increases mortality rate up to 80% (3).

Case report

A 44-year male, known to have type 2 diabetes, well controlled on lifestyle modification, presented with persistent bilateral lower limb muscle pain for 5 days, progressive bilateral ankle/leg swelling and morning facial puffiness for 3 days. He also complained of shortness of breath on moderate exertion, dark coloration of urine and reduced urine output during the same period.

The above symptoms developed after running a fundraising marathon for 70km in hot and low humid weather without prior preparation and adequate hydration. He also reported use of Non-Steroidal Anti-inflammatory Drug (NSAIDS) - eterocoxib for pain relief. He had no history of herbal medicine use or substance abuse.

Physical examination

General exam: On assessment we had a sick looking young male, who was tachypnoeic at a rate of 26 breaths/min and mildly dehydrated. He had moderate pitting bilateral pedal oedema with mild facial puffiness.

Vitals: BP 132/86mmHg, pulse 58b/min (regular, normal volume), SPO2 93% (room air), temp- 36.8° Celsius.

Systemic exam

Respiratory: He was not in respiratory distress. On auscultation, vesicular breath sounds heard bilaterally, reduced in the bases.

Abdominal: He had mild abdominal distension, soft and non-tender on palpation, liver span- 12cm, spleen not palpable, kidneys not ballotable. Puddle sign- positive.

Other systems: No significant finding.

Investigations

Laboratory Investigations (Table 1) showed high anion gap metabolic acidosis with elevated urea and creatinine, high Creatine Kinase (CK), myoglobin, Alt and AST levels. Urine analysis showed protein and blood with no RBCs. Imaging (Table 2) showed features of mild fluid overload (pleural effusion and ascites). Based on his clinical presentation and investigations, diagnosis of EIR with AKI requiring haemodialysis (HD) was made.

Table 1: Initial laboratory investigations

CBC: Wbc	10.0×10^9/l (4.0-10.0)
Hb	14.4g/dl (14-18)
Plt	150×10^9/l (150-450)
UECs: Na+	122 mmol/l (135-150)
K+	**4.7 mmol/l** (3.5-5.5)
Urea	**44.6 mmol/l** (1.7-8.3)
Creat	1459 umol/l (53-106)
Calcium	2.02mmol/l (2.02-2.65)
Magnesium	**1.01mmol/l** (0.60-1.06)
Phosphate	**3.92mmol/l** (0.81-1.62)
Uric acid	**0.603mmol/l** (0.21-0.42)
Urine analysis (dipstick)	**Brownish colour**
	Protein ++
	Blood +
	P.H 6.0
	RBCs- Nil
Creatinine Kinase	**15420 U/l** (0-190)
LFTs: T.bil	16 umol/l (3-23)
D.bil	5.8 umol/l (0-6.8)
ALT	1239 u/l (5-40)
AST	420 u/l (5-40)
GGT	188 u/l (0-50)
ALP	80 u/l (30-120)
T. protein	69 g/l (60-87)
Albumin	38.2 g/l (34-52)
HbA1c	6.4%
CK-MB	36.7 ng/ml (0-4.3)
Myoglobin	**>500 ng/ml** (0-107)
Troponin I	<0.05 ng/ml (0.0.4)
Pro-BNP	440 ng/ml (0-100)
D-dimers	1420 ng/ml (0-500)
UACR	285.6 mg/mmol (<15)
PTH	230pg/ml (15-65)

BGA: PH	**7.28** (7.35-7.45)
HC03-	**13.4mmol/l** (22-28)
PC02	22mmHg (35-45)
(HAGMA)	
HIV, HAV, HBV, HCV	Negative
ANA/ENA	Negative
Coag. profile	Normal
Lipid profile	Normal
TFTs	Normal

Table 2: Imaging results

ECG	Sinus bradycardia of 58bpm
Echocardiogram	Normal cardiac chambers with preserved diastolic and systolic function. Normal pulmonary pressures. No pericardial effusion
CXR	Features of mild bilateral pleural effusion
Abdominal U/S	Liver-normal Kidneys- normal size with reduced parenchymal echogenicity Mild ascites

Initial management and progression

He was admitted to HDU for observation, counselled on HD and consent taken for the same. Right internal jugular vein catheter was fixed and HD with ultrafiltration initiated. Initial treatment included oral tramadol for pain control, SC enoxaparin 20mg OD for thromboprophylaxis (renal-dosed), sevelamer 800mg TDS for phosphate binding and febuxostat 40mg OD for lowering urate. Daily fluid input-urine output charting and blood glucose monitoring was done.

His symptoms improved gradually with twice weekly haemodialysis. His BPs were noted to be elevated and he was started on amlodipine 5mg OD and hydralazine 25mg BD. After 2 weeks, he was discharged home on treatment and follow up in renal unit. His urine output improved significantly though renal functions remained high. He continued HD twice weekly for almost three months at the renal unit with serial reviews of his renal functions. Table 3 shows the evolution of laboratory results.

Table 3: Evolution of laboratory results

	Inpatient					Outpatient		
	D2	D4	D8	D12	D14	D20	D30	D60
Urea	32.7	17.2	14.3	21.1	19.6	21.3	18.9	14.7
Creatinine	1278	989	943	888	720	530	385	218
Phosphate	-	-	-	-	1.5			
ALT	-	-	635	-	219			
AST	-	-	180	-	36			
CK	-	-	1343	-	140			
Uric acid	-	-	0.233	-	0.132			
UACR	-	-	7.29	-	0.55			

Discussion

Prolonged intense physical activity causes mechanical and thermal injury to striated myocytes. Pre-disposing factors include hypokalaemia, extreme heat and low humidity, sickle-cell trait, exercise-induced asthma or pre-exertion fatigue (3). Clinical presentation varies from asymptomatic to intense muscle pain, weakness and dark tea-coloured urine with volume depletion, metabolic and electrolyte abnormalities and AKI (2, 3). Diagnosis is confirmed by elevation of serum CK levels >1000 U/L or at least 5x the upper limit of normal (3). Urine analysis shows positive blood on dipstick with absent or <5 RBCs per high-power field on microscopy (4).

AKI in EIR is attributed to myoglobin induced capillary damage, vasoconstriction and renal ischemia, direct myoglobin toxicity on proximal tubules, and formation of intra-tubular casts (4,5). Additional factors like nephrotoxic drugs e.g. NSAIDs for pain relief can worsen the AKI.

McMahon score can be used as a risk prediction tool in patients with rhabdomyolysis and AKI to assess risk of RRT or in-hospital mortality. Variables used in the scoring include age, gender, underlying aetiology, and initial laboratory values (6) (Table 4).

Management includes IV fluid repletion with crystalloids, volume assessment and target urine output of ≥ 300ml/h, electrolyte replacement, sodium bicarbonate administration for acidosis, target urine PH >6.5, and haemodialysis if necessary (3, 6).

Table 4: McMahon risk prediction

Variable	Score
Age (years)	
>50 to ≤70	1.5
>70 to ≤80	2.5
≥80	3.0
Female sex	1
Initial creatinine	
1.4–2.2 mg/dL	1.5
>2.2 mg/dL	3.0
Initial calcium <7.5 mg/dL	2.0
Inital CK >40,000 U/L	2.0
Initial phosphate	
4–5.4 mg/dL	1.5
>5.4 mg/dL	2.0
Underlying cause other than seizures, syncope, exercise, statins, or myositis	3.0
Initial serum bicarbonate <19 mEq/L	2.0

Score ≤5: Low risk of AKI requiring RRT or mortality
Score ≥6: High risk of AKI requiring RRT or mortality (86% sensitive, 68% specific)

Conclusion

Due to increasing awareness about positive effects of exercise on physical and mental health, more people are participating in physical activities. It is important to educate general population on measures to prevent exercise induced myocyte injury and AKI. These include warm-up exercises prior to intense activity, avoiding intense exercise for prolonged duration, adequate hydration and avoiding excess use of over the counter drugs like NSAIDs. Prompt medical care should be sought on occurrence of any symptoms post intense exercise as timely diagnosis and management of rhabdomyolysis can prevent AKI.

Declaration

Conflict of interest: None to disclose.

Consent: Consent to document the case was obtained from the patient

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