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Other full case

Recurrent exercise-induced rhabdomyolysis due to low intensity fitness exercise in a healthy young patient

Premnath Reddy Karre,[1] Jeetinder Gujral[2]

[1]Department of Hospitalist Medicine, Christus St Michael Health System, Texarkana, Texas, USA;
[2]Department of Hospitalist Medicine, NSLIJ-Southside Hospital, Bay shore, New York, USA

Correspondence to Dr Premnath Reddy Karre, karreprn@gmail.com

Summary

Rhabdomyolysis is an uncommon but life threatening condition that develops due to breakdown of muscle and release of intracellular components into the circulation. A 24-year-old man otherwise healthy was admitted to our hospital because of muscle aches and weakness as well as cola coloured urine developed 3 days after carrying out the low intensity exercise. Diagnosis of rhabdomyolysis was made with creatine kinase (CK) levels of 214 356 U/l. He was treated for a similar condition at age 21. A muscle biopsy was done and the findings were normal. Rhabdomyolysis can develop with low intensity exercise; thus, it be considered in healthy young people. Young people with recurrent rhabdomyolysis due to low intensity exercise, in the absence of obvious medical and physical causes, should be evaluated further to rule out uncommon metabolic diseases. Our case demonstrates that complications especially renal failure in patients with rhabdomyolysis do not correspond to CK levels.

BACKGROUND

Rhabdomyolysis is an uncommon but life threatening condition that develops due to breakdown of muscle and release of intracellular components into the circulation. A 24-year-old man otherwise healthy was admitted to our hospital because of muscle aches and weakness as well as cola coloured urine developed 3 days after carrying out the low intensity exercise. Diagnosis of rhabdomyolysis was made with creatine kinase (CK) levels of 214 356 U/l. He was treated for a similar condition at age 21. A muscle biopsy was done and the findings were normal. Rhabdomyolysis can develop with low intensity exercise; thus, it be considered in healthy young people. Young people with recurrent rhabdomyolysis due to low intensity exercise, in the absence of obvious medical and physical causes, should be evaluated further to rule out uncommon metabolic diseases. Our case demonstrates that complications especially renal failure in patients with rhabdomyolysis do not correspond to CK levels.

CASE PRESENTATION

A 24-year-old Indian male referred from his primary medical doctor (PMD) for admission because of rhabdomyolysis had presented to his PMD with muscle aches, joint pains and cola-coloured urine. Three days before his primary care visit, the patient had performed exercises such as squatting, push ups and running on a treadmill for 1 h with brief interruptions. He was apparently healthy until this episode happened. At age 21, however, he had been treated without complications in our hospital with rhabdomyolysis that developed after low intensity exercise.

He did not have any other significant medical and surgical history. He denied taking any drugs, prescribed medications, over-the-counter and herbal products as well as smoking tobacco and ingesting alcohol. His family history was negative for metabolic, neuromuscular and autoimmune diseases. He also denied any relative who had experienced a similar problem with exercise. Review of the systems was unremarkable.

Vital signs were stable on admission. He had a normal respiratory, cardiovascular, neurological and musculoskeletal examination except for diffuse muscle tenderness on palpation. Psychiatric mental status examination was normal.

INVESTIGATIONS

Initial blood tests revealed total CK 214356 U/l, alanine aminotransferase 252 U/l, aspartate aminotransferase 1112 U/l, alkaline phosphatase 73 U/l, creatinine 0.8 mg/dl, blood urea nitrogen 11 mg/dl. Plasma myoglobin measured 1347 ng/ml. Urine and blood toxin screen results were normal. Urine analysis was positive for blood but without red blood cells. Urinary levels of serum lactate, ammonia, carnitine, acylcarnitine, amino acids and organic acids were normal. Evaluations for autoimmune diseases were negative as were hepatitis viruses and Lyme disease serologies. Thyroid stimulating hormone was 0.66 µ/l. Testing for sickle cell trait was negative.

Leucocyte DNA testing for carnitine palmitoyltransferase II gene and muscle glycogen phosphorylase (PYGM) gene did not reveal any mutations.

We did not perform ischaemic forearm test but a muscle biopsy report was normal (table 1). Echocardiogram, ECG and chest radiograph findings were normal. On discharge, a complete metabolic profile was normal.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis considered in our patient is given below.

- Physical overexertion
- Drugs and alcohol

Table 1 Studies on muscle biopsy

Type of test	Test results
Staining	Normal. No evidence of abnormal storage of any material
H&E stain	
Gomori trichrome	
Periodic acid-Schiff (PAS)	
Sudan Black	
NADH satin	
HLA class I antigen	
Myosin heavy chain I and II	
Microscopy	Normal. No necrosis/inflammation
Histochemical studies	Normal
Myophosphorylase	
Phosphofructokinase	
Myoadenylate deaminase	
Succinic dehydronase	
Cytochrome oxidase	
Electron microscopy	Normal

- Autoimmune disease
  - Polymyositis
- Endocrinologic causes
  - Electrolyte imbalances
  - Hypothyroidism
  - Thyrotoxicosis
  - Diabetes ketoacidosis
- Lipid metabolism disorders
  - Carnitine palmitoyltransferase II (CPT II) deficiency
- Glycogen storage disorders
  - Myophosphorylase deficiency (McArdle's disease)
  - Phosphofructokinase deficiency
- Purine metabolism
  - Myoadenylate deaminase deficiency.

TREATMENT

Our patient was treated with vigorous intravenous hydration with addition of sodium bicarbonate throughout his stay in hospital. We titrated intravenous fluids to maintain the pH of urine greater than 6.5. CK levels declined as showed in figure 1. The patient did not develop any complications.

OUTCOME AND FOLLOW-UP

We collaborated with his PMD and followed the patient. Laboratory tests were repeated and continued to be normal at his primary care physician. We are working on exercise regimen for our patient through physical therapy.

DISCUSSION

Cases of recurrent exercise induced rhabdomyolysis associated with low intensity fitness exercise are rarely reported. Case reports of recurrent rhabdomyolysis in patients with various metabolic diseases[1–3] were reviewed. Our patient performed mostly aerobic exercise, that is, treadmill, but also partially anaerobic exercises, that is, push-ups and squatting. We did not find studies describing any correlation between type of exercise and frequency and severity of rhabdomyolysis in patients with similar presentation. Currently, our patient has been working with physical

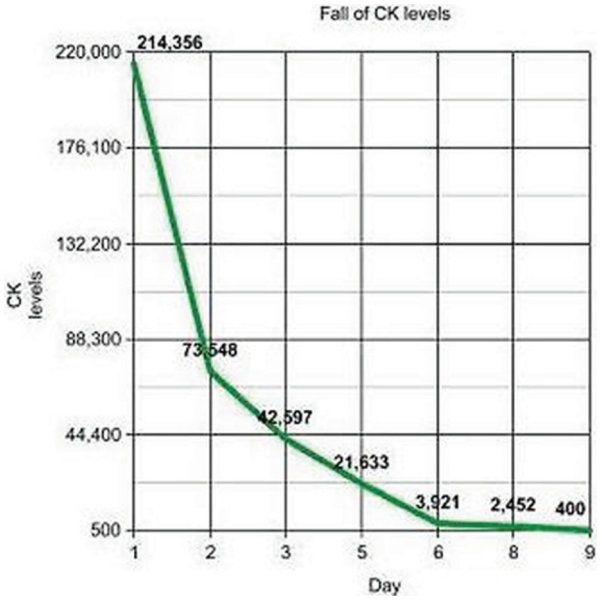

Figure 1 Fall of CK levels.

therapy to develop an exercise regimen to prevent episodes of rhabdomyolysis. Cases of rhabdomyolysis with extremely high levels of CK due to severe exertion,[4] trauma or heat stroke have been reported.[5] We initially thought levels of CK were disproportional to intensity of exercise and considered metabolic disorders but all test results were normal. The mechanism of rhabdomyolysis occurring after low intensity fitness exercise and elevated CK levels in our patient remains a mystery. Only one other case report was published[5] with higher levels of CK than those of our patient.

Learning points

- Rhabdomyolysis can develop with low intensity fitness exercise and should be considered in healthy young people.
- Young people with recurrent rhabdomyolysis due to low intensity exercise, in the absence of obvious medical and physical causes, should be evaluated further to rule out any uncommon metabolic diseases.
- We believe proper work-up is necessary in these patients to counsel and direct appropriate course of management especially regarding exercise regimen. Eliminating exercise in these patients' lives would be detrimental to their mental and physical health.
- Our case demonstrates as well as re-emphasises[6] that complications especially renal failure in patients with rhabdomyolysis are not always associated with elevated CK levels.

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Competing interests None.

Patient consent Obtained.

REFERENCES

1. **Aoyama T,** Yazawa I, Sugie H, *et al*. A case of skeletal muscle type very-long-chain-acyl CoA dehydrogenase (VLCAD) deficiency with repeated rhabdomyolysis. *No To Shinkei* 2004;**56**:64–8.
2. **Löfberg M,** Jänkälä H, Paetau A, *et al*. Metabolic causes of recurrent rhabdomyolysis. *Acta Neurol Scand* 1998;**98**:268–75.
3. **Krivickas LS**. Recurrent rhabdomyolysis in a collegiate athlete: a case report. *Med Sci Sports Exerc* 2006;**38**:407–10.
4. **Hamer R**. When exercise goes awry: exertional rhabdomyolysis. *South Med J* 1997;**90**:548–51.
5. **Pattison ME,** Logan JL, Lee SM, *et al*. Exertional heat stroke and acute renal failure in a young woman. *Am J Kidney Dis* 1988;**11**:184–7.
6. **Karre PR,** Gujral J, Rordiguez E. Rhabdomyolysis with erratic serum creatinin phosphokinase levels: intramuscular injection of haloperidol decanoate. *BMJ Case Rep.* Published Online First: 17 August 2009. doi:10.1136/bcr.06.2009.1931.