# An increase in the number of admitted patients with exercise-induced rhabdomyolysis

**BACKGROUND** Rhabdomyolysis may lead to serious complications, and treatment is both time-consuming and costly. The condition can be caused by many factors, including intense exercise. The purpose of this study was to investigate whether the number of hospitalisations due to exercise-induced rhabdomyolysis has changed in recent years. We describe the disease course in hospitalised patients, and compare disease course in individuals with exercise-induced rhabdomyolysis and rhabdomyolysis due to other causes.

**MATERIAL AND METHOD** The study is a systematic review of medical records from Akershus University Hospital for the years 2008 and 2011–14. All hospitalised patients with diagnostic codes M62.8, M62.9 and T79.6 and creatine kinase levels > 5 000 IU/l were included. The cause of the rhabdomyolysis was recorded in addition to patient characteristics and the results of various laboratory tests.

**RESULTS** Of 161 patients who were hospitalised with rhabdomyolysis during the study period, 44 cases (27 %) were classified as exercise-induced. In 2008 there were no admissions due to exercise-induced rhabdomyolysis; in 2011 and 2012 there were six and four admissions respectively, while in 2014 there were 22. This gives an estimated incidence of 0.8/100 000 in 2012 and 4.6/100 000 in 2014. Strength-training was the cause of hospitalisation in 35 patients (80 % of the exercise-induced cases). Three patients (7 % of the exercise-induced cases) had transient stage 1 kidney injury, but there were no cases with stage 2 or stage 3 injury. By comparison, 52 % of patients with rhabdomyolysis due to another cause had kidney injury, of which 28 % was stage 2 or 3.

**INTERPRETATION** The number of persons hospitalised with exercise-induced rhabdomyolysis has increased four-fold from 2011 to 2014, possibly due to changes in exercise habits in the population. None of the patients with exercise-induced rhabdomyolysis had serological signs of kidney injury upon hospital discharge.

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## MAIN POINTS

**There has been an increase in the number of patients admitted to hospital with exercise-induced (exertional) rhabdomyolysis**

**Altered exercise habits and increased media attention may have contributed to the increased number of diagnoses**

**Patients with exercise-induced rhabdomyolysis appear to have better renal prognosis than patients with rhabdomyolysis due to other causes**

Rhabdomyolysis is a clinical syndrome in which injury to striated muscle leads to the release of intracellular components by myocytes. The classic symptoms are myalgia, muscle weakness and dark urine. Severity ranges from an asymptomatic elevation of enzyme levels to a life-threatening condition with anuric renal failure, severe electrolyte imbalance and arrhythmias.

The condition was originally described in the context of patients with major muscle trauma who died as a result of renal failure (crush syndrome) (1). Rhabdomyolysis can occur after traumatic muscle injury, for example when the inability to get up after a fall leads to prolonged strain/pressure on the muscles. It can also occur after seizures, as a complication of surgery, and after non-traumatic muscle injury, for example in the course of infectious diseases or poisonings (2).

The diagnosis is usually based on an elevated blood level of the muscle enzyme creatine kinase (CK), which is released upon damage to muscle cells. The underlying pathological mechanisms are not fully understood; however, a decrease in intracellular adenosine triphosphate (ATP) with a subsequent increase in intracellular free calcium, which activates proteases and free radicals, may be the mechanism leading to cell death (3, 4).

Damaged muscle cells also release myoglobin, which may cause kidney injury. While it is possible to measure myoglobin levels, the shorter half-life of myoglobin means that this test is less sensitive than measurement of creatine kinase. Rhabdomyolysis is often diagnosed a few days after injury/trauma, at which point serum myoglobin levels may be low. Myoglobin will cause urine dipsticks to test positive for blood; a positive result in the absence of erythrocytes may therefore indicate a risk of kidney injury. Rhabdomyolysis is usually diagnosed when creatine kinase levels are 5–50 times the upper limit of normal (5, 6). Levels > 5 000 IU/l have been proposed as an indication for treatment.

Treatment should involve removing the precipitating factor if possible. In addition to the treatment of complications, early and aggressive fluid resuscitation is recommended to increase renal perfusion and thereby promote the excretion of harmful substances. To prevent the deposition of myoglobin in renal tubules, it is also common practice to alkalise the urine and thereby reduce the precipitation of haem pigment, which binds



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to Tamm-Horsfall protein. This is a glycoprotein that is secreted by renal tubule cells and contributes to the formation of urinary casts, which may obstruct the tubules. The efficacy of alkalinisation is subject to debate (7, 8).

Exercise-induced rhabdomyolysis occurs after physical exertion. An increase in creatine kinase levels is to be expected after strenuous exercise, but there are large individual variations dependent on factors including gender, ethnicity and muscle mass. Men have higher levels than women, and African men have the highest of all (9). It is unclear where the boundary lies between a physiological increase in creatine kinase level and the pathological state of rhabdomyolysis. Previous studies of exercise-induced rhabdomyolysis show that those who develop renal failure have higher average creatine kinase levels than those with an uncomplicated disease course, but the creatinine kinase level alone cannot predict the risk of renal failure (10, 11).

Recent years have seen the widespread adoption of new forms of exercise. Attendance at fitness centres, participation in various kinds of organised exercise classes and the use of a personal trainer have become commonplace. The purpose of this study was to determine whether the number of hospitalisations due to exercise-induced rhabdomyolysis has changed in recent years, to describe the patients who are admitted to hospital, to examine the occurrence of complications related to the disorder or its management, and to compare exercise-induced rhabdomyolysis with rhabdomyolysis due to other causes.

## Material and method

This retrospective study was considered a quality assurance study by the Regional Committee for Medical and Health Research Ethics (REC South East). It was approved by the Data Protection Officer at Akershus University Hospital as an internal quality assurance project.

The medical records of all patients aged 18 years and above who were admitted to hospital in 2008, 2011, 2012, 2013 or 2014 with a recorded primary or secondary diagnosis code of M62.8 (other specified disorders of muscle), M62.9 (disorder of muscle, unspecified) or T79.6 (traumatic ischaemia of muscle) were reviewed. All patients with creatine kinase levels > 5 000 IU/l were included.

We recorded gender, age, reason for admission, drug and alcohol use, comorbidity, regular medications and a number of serological test results. Exercise was categorised as either endurance or strength training. The latter includes bodyweight exercises (e.g., «pull-ups», where individuals pull themselves up with their arms). CrossFit, which is a form of exercise with constantly varied functional movements performed at high intensity, was recorded as strength training. Some patients had engaged in multiple types of exercise.

We recorded the muscle group(s) affected as revealed by pain, weakness and swelling. The number of days spent on standard wards and in the intensive care unit, the type of treatment and any readmissions for rhabdomyolysis in the same year were also noted. In addition, we recorded complications related to the disorder and/or its management.

Changes in renal function were assessed using creatinine levels. Kidney injury was defined as follows: stage 1: peak creatinine level > 26.5 µmol/l or 150–200 % of the level at discharge; stage 2: peak creatinine 200–300 % of that at discharge; stage 3: peak creatinine > 44.2 µmol/l higher than the level at discharge if creatinine was > 356 µmol/l at discharge or peak creatine was > 300 % of the level at discharge.

The staging definitions for acute kidney injury (AKI) in the modified version of «Kidney Disease Improving Global Outcomes» (KDIGO) from 2012 were applied (12). These definitions are based on increases relative to baseline creatinine level. We had no premorbid baseline values and therefore used the level at discharge as a baseline.

### *Statistics*

Statistical analysis was performed using SPSS version 21.0 (IBM SPSS Inc., Chicago, IL). Between-group comparisons (exercise-induced rhabdomyolysis versus rhabdomyolysis due to other causes) were conducted using a t-test or chi-square test/Fisher's exact test with associated p-values. Statistical significance was defined as $p < 0.05$.

A chi-square test was used to examine the change in the number of admissions, while ANOVA was used for trend analysis to explore how the number of admissions for rhabdomyolysis changed over time. The calculation of population incidence of exercise-induced rhabdomyolysis was based on the number of individuals living in the catchment area of Akershus University Hospital, which in 2008 was approximately 320 000 persons and from 2010, approximately 480 000.

## Results

In all, 161 patients with rhabdomyolysis were admitted during the five-year observational period, of which 44 cases (27 %) were exercise-induced (Table 1, Table 2). Of these, 35 patients (80 % of the exercise-induced cases) had engaged in strength training and four (9 %) in endurance exercise. Table 3 shows that patients with exercise-induced rhabdomyolysis were more likely to be female, had less comorbidity and were less likely to overuse drugs or alcohol than patients with rhabdomyolysis due to other causes. Patients with exercise-induced rhabdomyolysis were hospitalised for an average of 4.5 days, compared with 9.9 days for the other patients.

The number of hospitalisations due to exercise-induced rhabdomyolysis increased steadily – from none in 2008 to 22 in 2014 (trend analysis: $p = 0.01$) (Fig. 1). The estimated incidence of hospitalisations due to rhabdomyolysis rose from 0 in 2008, via approximately 1/100 000 population in 2010 and 2011, to 4.5/100 000 in 2014.

**Table 1** Patients admitted to Akershus University Hospital in 2008 and 2011–14 with exercise-induced rhabdomyolysis, classified by presumed trigger (n = 44, 27 % of all admissions)

| | |
|---|---|
| Type of exercise | |
| Endurance (running, cycling, walking) | 4 |
| Strength training | 35 |
| Weights | 28 |
| CrossFit[1] | 4 |
| Various types/unspecified | 5 |

[1] CrossFit is exercise with constantly varied functional movements performed at high intensity

**Table 2** Patients (n = 117) admitted to Akershus University Hospital in 2008 and 2011–14 with non-exercise-induced rhabdomyolysis, classified by presumed trigger (n = 117, 73 % of all admissions)[1]

| | |
|---|---|
| Prolonged contact with hard surface | 29 |
| Muscle trauma | 6 |
| Poisoning | 49 |
| Alcohol | 18 |
| Opiates | 11 |
| Mixed – legal agents | 10 |
| Mixed – illegal agents | 8 |
| Other | 2 |
| Epileptic seizures | 10 |
| Postoperative complication | 7 |
| Other specific disease | 12 |
| Known muscle disease | 1 |
| Statin use | 2 |
| Unknown | 1 |

[1] More than one stated cause

**Table 3** Patients (n = 161) admitted to Akershus University Hospital in 2008 and 2010–14 with exercise-induced rhabdomyolysis or rhabdomyolysis due to other causes. Number and percentage (%) unless otherwise stated

| | Exercise-induced (n = 44) | Other cause (n = 117) | P-value |
|---|---|---|---|
| Age (years) – median (min–max) | 28 (18–87) | 56 (21–90) | < 0.01 |
| Women | 20 (45) | 25 (21) | < 0.01 |
| Tobacco use | | | |
| Smoker | 8 (18) | 49 (42) | 0.01 |
| Snus user | 3 (7) | 0 | < 0.01 |
| Alcohol | | | |
| Known overuse/dependency | 2 (5) | 32 (27) | 0.01 |
| Abstinence | 5 (11) | 14 (12) | 0.53 |
| Other substances | | | |
| Known user | 4 (9) | 35 (30) | < 0.01 |
| Readmitted in the course of a year | 4 (9) | 2 (2) | 0.05 |
| Comorbidity (before admission) | | | |
| Previously healthy | 34 (77) | 42 (36) | < 0.01 |
| Kidney disease | 0 | 2 (2) | 0.38 |
| Pulmonary disease | 2 (5) | 12 (10) | 0.25 |
| Cardiac disease | 4 (9) | 20 (17) | 0.20 |
| Hypertension | 1 (2) | 26 (22) | < 0.01 |
| Diabetes mellitus | 0 | 8 (7) | 0.075 |

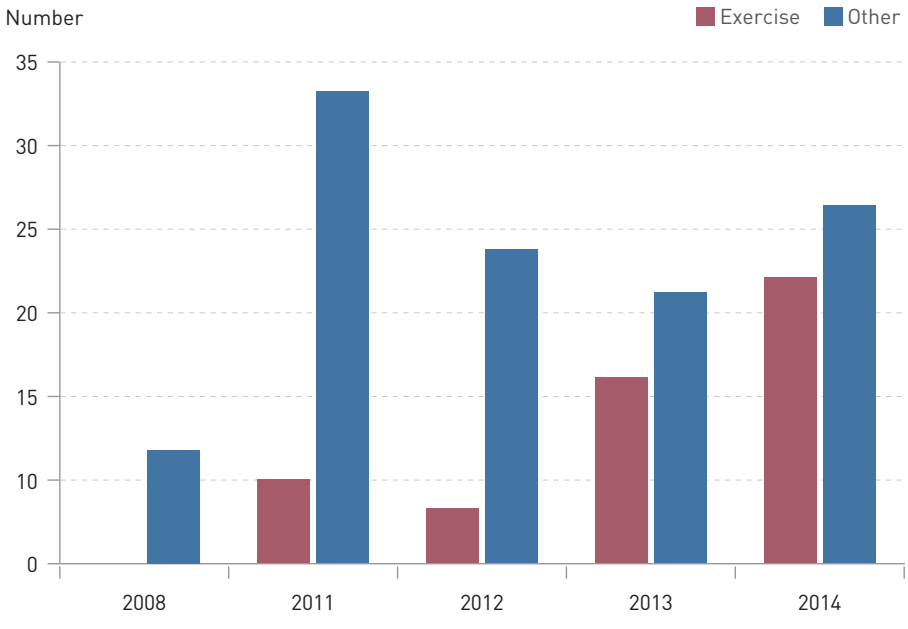


**Figure 1** *Patients (n = 161) admitted to Akershus University Hospital in 2008 and 2011–14 with exercise-induced rhabdomyolysis or rhabdomyolysis due to other causes*

Strength training was the causal factor in 35 of 44 cases (80 %) of exercise-induced rhabdomyolysis, with 28 patients having engaged in weight training and four in CrossFit. Muscles in the arms were affected in 24 patients (69 % of those who had practised strength training).

Maximum creatine kinase levels did not differ between the groups with exercise-induced rhabdomyolysis and rhabdomyolysis due to other causes. As shown in Table 4, 7 % of hospitalised patients with exercise-induced rhabdomyolysis had acute kidney injury (all at stage 1), compared with 52 % of patients with rhabdomyolysis due to other causes (Table 4).

None of the patients with exercise-induced rhabdomyolysis needed dialysis, whereas seven patients in the other group (6 %) required intermittent dialysis ($p < 0.01$). Forced alkaline diuresis was used to treat 40 patients with exercise-induced rhabdomyolysis (91 %) and 85 (77 %) patients with rhabdomyolysis due to other causes ($p = 0.018$).

## Discussion

This study has revealed a significant increase in the number of hospitalisations due to exercise-induced rhabdomyolysis since 2008. From 2011 to 2014, the number of admissions almost quadrupled. One reason for this may be the increasing popularity of new forms of exercise, as confirmed by the fact that 80 % of patients had engaged in strength training at fitness centres. Most of those who had practised weight training had symptoms in the arm muscles, but other major muscle groups may also be affected.

While earlier studies most commonly described exercise-induced rhabdomyolysis after endurance exercise, such as long-distance running, recent studies have shown that strength training is more likely to trigger the condition. The increase in the incidence of exercise-induced rhabdomyolysis was also confirmed by a US military study, which recorded a 30 % increase in exercise-induced cases from 2008 to 2012 (13).

Exercise-induced rhabdomyolysis has been described in connection with various forms of endurance and strength training (11). Eccentric training, as seen in CrossFit, is the form of exercise most strongly associated with the development of muscular injuries and rhabdomyolysis (14, 15). Poor fitness is a risk factor, but physically fit individuals can also be affected (16). Low fluid intake and high temperatures are considered precipitating factors (17).

Exercise combined with the use of narcotics and banned muscle-building substances, or with the use of non-steroidal anti-inflammatory drugs, statins or dietary supplements such as creatine and ephedra/ephedrine, has

been associated with rhabdomyolysis (16, 18). Genetic polymorphisms may also increase risk (9). Information came to light on the use of various narcotic and performance-enhancing drugs by five persons in this study, but the relationship between rhabdomyolysis and drug use was not examined systematically and the numbers are therefore very uncertain.

In recent years there has been extensive media coverage of exercise-induced rhabdomyolysis, with stories about how the condition may be highly dangerous accompanied by information on symptoms and advice on when to seek medical attention. This may lead to more frequent diagnoses as patients visit their doctor with symptoms that they would not previously have sought advice over. On the other hand, there are probably many others who do not consult their doctor and therefore many cases that go unreported. In this study we tentatively estimated the incidence of rhabdomyolysis to be 4.5 per 100 000 population in 2014. We were unable to find any other such calculations, but a study in military personnel measured the incidence at 29.9 per 100 000 person-years (19).

It is common for some muscle damage to occur with intense physical activity. A study of 499 US military recruits revealed that after seven days of basic training, 88.5 % had elevated creatine kinase levels and 11.3 % had levels > 10 times the upper reference limit. Levels ranged from 34 IU/l to 35 056 IU/l, and the average was 1 226 IU/l after seven days. None of the recruits had symptoms or signs of rhabdomyolysis (6). Another study showed that 25 of 44 participants who completed an ultramarathon of 99 km had markedly elevated serum creatine kinase and that five of these developed myoglobinuria, although none developed renal failure (20).

The aim of rhabdomyolysis treatment is to prevent kidney injury. A retrospective analysis found that 19 % of those with exercise-induced rhabdomyolysis developed acute kidney injury, defined as a creatinine level >114.9 µmol/l, as did 34.2 % of those with rhabdomyolysis due to other causes (21). The proportion with acute kidney injury requiring dialysis was 1.6 % and 9.7 % respectively. In another study of 35 patients with exercise-induced rhabdomyolysis, none of those affected developed acute kidney injury (22).

However, serious complications of exercise-induced rhabdomyolysis have been described in several case reports (23, 24). In our study, 6 % of patients with exercise-induced disease had transient mild kidney injury but none required dialysis. Fewer of those with exercise-induced rhabdomyolysis developed renal failure compared to those with other forms of rhabdomyolysis. This may be because the causal factors in the latter group can themselves give rise to kidney injury (e.g., hypovolaemia and sepsis) and also because these patients often have considerable comorbidity, which again increases the risk of kidney injury.

**Table 4** Patients (n = 161) admitted to Akershus University Hospital in 2008 and 2011–14 with exercise-induced rhabdomyolysis or rhabdomyolysis due to other causes. Treatment and laboratory test results. Average (SD) unless otherwise specified

| | Exercise-induced (n = 44) | Other cause (n = 117) | P-value |
|---|---|---|---|
| Forced alkaline diuresis (FAD) – number (%) | 40 (91) | 85 (73) | 0.018 |
| Max creatinine kinase (CK) (x · $10^3$) (IU/l) | 57.6 (91.1) | 42.5 (39.2) | 0.16 |
| Max creatinine (µmol/l) | 81.2 (19.0) | 171.0 (166.2) | < 0.01 |
| Creatinine clearance (µmol/l) | 70.1 (13.9) | 88.1 (85.3) | 0.17 |
| Kidney injury | | | |
| Stage 1 – number (%) | 3 (7) | 28 (24) | 0.014 |
| Stage 2 – number (%) | 0 | 12 (10) | 0.027 |
| Stage 3 – number (%) | 0 | 21 (18) | 0.03 |
| Electrolyte levels during hospital stay (mmol/l) | | | |
| Phosphate peak – average (SD) | 1.41 (0.40) | 1.47 (0.71) | 0.61 |
| Phosphate nadir – average (SD) | 0.87 (0.28) | 0.79 (0.44) | 0.30 |
| Potassium peak – average (SD) | 4.37 (0.38) | 4.93 (1.02) | < 0.01 |
| Potassium nadir – average (SD) | 3.48 (0.34) | 3.33 (0.51) | 0.028 |
| Calcium peak – average (SD) | 2.35 (0.12) | 2.25 (0.20) | <0.10 |
| Calcium nadir – average (SD) | 2.03 (0.20) | 1.84 (0.34) | <0.01 |
| Days in intensive care | 1.9 (1.4) | 2.6 (3.0) | 0.01 |
| Days in standard ward | 2.6 (3.0) | 7.3 (8.9) | <0.01 |

Previous studies have shown that 10–50 % of patients with rhabdomyolysis develop renal failure (25). Although the definition of acute kidney injury varies between studies, this is reasonably consistent with our figure for the non-exercise-induced rhabdomyolysis group. A relatively large proportion of patients had electrolyte imbalance, either as a result of the rhabdomyolysis itself or its treatment. The clinical significance of this is uncertain, especially in patients with exercise-induced rhabdomyolysis.

Patients with exercise-induced rhabdomyolysis probably have a better renal prognosis than others with the same disorder, and should perhaps be treated as a separate group with distinct therapeutic criteria. Rhabdomyolysis treatment is intensive and relatively prolonged.

There have been no randomised studies comparing treatment regimes. The efficacy of urinary alkalinisation is subject to debate (7, 8, 26). The treatment involves administering large volumes of fluid intravenously, which entails a risk of complications due to overhydration. In this study none of those with exercise-induced rhabdomyolysis developed symptoms of severe hypervolaemia, whereas some of those with rhabdomyolysis due to other causes displayed symptomatic fluid retention. We did not systematically record the volume of fluid administered and the amount excreted by patients, but we did observe major electrolyte imbalance in some cases, with much of this probably due to treatment.

The current study used serum creatinine alone as an index of kidney injury and renal function. However, creatinine is an imprecise index, especially for low-grade kidney injury. Additional measures of kidney injury and previous creatinine levels were not used because patients were hospitalised with the disease. We therefore used the creatinine level at discharge as an index of kidney injury instead. However, patients with exercise-induced rhabdomyolysis had normal creatinine levels at discharge and it is thus

unlikely that premorbid measurements would have revealed those with kidney injury, especially in view of the limitations related to creatinine quantification.

This study is based on a retrospective review of medical records. Patients who fulfilled the diagnostic criteria for rhabdomyolysis but who were assigned the wrong diagnostic code may have been omitted. This is most likely to apply to rhabdomyolysis as a complication of surgery. In patients with symptoms suggesting overuse of muscles in the shoulders and upper arms, the condition may for example have been coded as tendonitis.

This study is not compatible with comparing treatment strategies, but the evidence base for existing treatments is relatively limited. There is a need for studies comparing different forms of treatment, particularly for exercise-induced rhabdomyolysis, where patients have normal creatinine levels upon admission to hospital. Serum creatinine kinase levels increase approximately 12 hours after an injury and remain elevated for 2–3 days before gradually decreasing.

Myoglobin has a short half-life (10–15 min. in plasma). It may be possible to use urinary myoglobin to guide treatment: when urine dipsticks test negative for blood, treatment can be scaled down. This algorithm requires validation in a clinical trial. There is also a need for long term follow-up of rhabdomyolysis patients to determine whether they are at increased risk of renal failure in the future.

This study has revealed a clear increase in the number of hospitalisations due to exercise-induced rhabdomyolysis. We believe that new types of exercise and increased societal pressure to exercise may be contributing to the increased incidence of the condition. Renal prognosis is good, but whether the disorder leads to continued muscle weakness is unknown. Treatment is extensive and demanding, with a risk of complications, and comparative efficacy studies are required.

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**References**

1. Bywaters EG. 50 years on: the crush syndrome. BMJ 1990; 301: 1412–5.
2. Zimmerman JL. Shen MC. Rhabdomyolysis. Chest 2013; 144: 1058–65.
3. Giannoglou GD. Chatzizisis YS. Misirli G. The syndrome of rhabdomyolysis: Pathophysiology and diagnosis. Eur J Intern Med 2007; 18: 90–100.
4. Khan FY. Rhabdomyolysis: a review of the literature. Neth J Med 2009; 67: 272–83.
5. Melli G. Chaudhry V. Cornblath DR. Rhabdomyolysis: an evaluation of 475 hospitalized patients. Medicine (Baltimore) 2005; 84: 377–85.
6. Kenney K. Landau ME. Gonzalez RS et al. Serum creatine kinase after exercise: drawing the line between physiological response and exertional rhabdomyolysis. Muscle Nerve 2012; 45: 356–62.
7. Homsi E. Barreiro MF. Orlando JM et al. Prophylaxis of acute renal failure in patients with rhabdomyolysis. Ren Fail 1997; 19: 283–8.
8. Ozgüç H. Kahveci N. Akköse S et al. Effects of different resuscitation fluids on tissue blood flow and oxidant injury in experimental rhabdomyolysis. Crit Care Med 2005; 33: 2579–86.
9. Landau ME. Kenney K. Deuster P et al. Exertional rhabdomyolysis: a clinical review with a focus on genetic influences. J Clin Neuromuscul Dis 2012; 13: 122–36.
10. de Meijer AR. Fikkers BG. de Keijzer MH et al. Serum creatine kinase as predictor of clinical course in rhabdomyolysis: a 5-year intensive care survey. Intensive Care Med 2003; 29: 1121–5.
11. Oh RC. Arter JL. Tiglao SM et al. Exertional rhabdomyolysis: a case series of 30 hospitalized patients. Mil Med 2015; 180: 201–7.
12. KDIGO Clinical Practice Guideline for Acute Kidney Injury. Section 2: AKI Definition. Kidney Int Suppl (2011) 2012; 2: 19–36.
13. O'Connor FG. Deuster PA. Rhabdomyolysis. I: Goldman L. Schafer AI. red. Goldman's Cecil Medicine. 24. utg. Kap. 115. Philadelphia. PA: Saunders Elsevier. 2011.
14. Heled Y. Zarian A. Moran D et al. Exercise induced rhabdomyolysis–characteristics. mechanisms and treatment. Harefuah 2005; 144: 34–8. 70.
15. Armstrong RB. Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. Med Sci Sports Exerc 1984; 16: 529–38.
16. Furman J. When exercise causes exertional rhabdomyolysis. JAAPA 2015; 28: 38–43.
17. Zimmerman JL. Shen MC. Rhabdomyolysis. Chest 2013; 144: 1058–65.
18. Clarkson PM. Exertional rhabdomyolysis and acute renal failure in marathon runners. Sports Med 2007; 37: 361–3.
19. Armed Forces Health Surveillance Center. Update: excertional rhabdomyolysis. active component. U.S. Armed forces. 2011. MSMR 2012; 19: 17–9.
20. Schiff HB. MacSearraigh ET. Kallmeyer JC. Myoglobinuria. rhabdomyolysis and marathon running. Q J Med 1978; 47: 463–72.
21. Alpers JP. Jones LK Jr. Natural history of exertional rhabdomyolysis: a population-based analysis. Muscle Nerve 2010; 42: 487–91.
22. Sinert R. Kohl L. Rainone T et al. Exercise-induced rhabdomyolysis. Ann Emerg Med 1994; 23: 1301–6.
23. DeFilippis EM. Kleiman DA. Derman PB et al. Spinning-induced rhabdomyolysis and the risk of compartment syndrome and acute kidney injury: Two cases and a review of the literature. Sports Health 2014; 6: 333–5.
24. Oh JY. Laidler M. Fiala SC et al. Acute exertional rhabdomyolysis and triceps compartment syndrome during a high school football cAMP. Sports Health 2012; 4: 57–62.
25. Bosch X. Poch E. Grau JM. Rhabdomyolysis and acute kidney injury. N Engl J Med 2009; 361: 62–72.
26. Holt SG. Moore KP. Pathogenesis and treatment of renal dysfunction in rhabdomyolysis. Intensive Care Med 2001; 27: 803–11.

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