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Case Report

Exertion induced rhabdomyolysis in both triceps muscles in a 36-year old woman: A case report [☆]

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ABSTRACT

Rhabdomyolysis is a condition, often caused by strenuous exercise, which can lead to acute kidney injury, severe electrolyte imbalances, coagulopathies, compartment syndromes, and even have a fatal outcome in a few cases.

Recognition and management of fluid and electrolyte abnormalities is one of the first steps of treatment and key to a good outcome.

We report a case of a 36-year old woman who was referred to the ER by her general practitioner with severe muscle tenderness to the upper arms and highly elevated creatine kinase (CK) serum levels.

Initial ultrasound imagery showed a patent venous system but demonstrated a moderate edematous infiltration of the muscle bellies of both m. triceps. Additional magnetic resonance imagery showed a hyperintense signal in T2 in both triceps' muscles. Given the clinical presentation, the MRI-findings were consistent with a form of exertion-induced rhabdomyolysis of both triceps' muscles.

The patient was admitted for administration of IV-fluids to prevent acute kidney injury. Symptoms resolved in a few days and new magnetic resonance imagery showed a regression of the subcutaneous soft tissue infiltration.

The aim of this paper is to raise awareness about this diagnosis. If overlooked, severe complications as mentioned above can occur.

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Introduction

Exercise induced rhabdomyolysis, or exertion-induced rhabdomyolysis, is a condition where a patient presents with muscle stiffness, pain, and swelling out of proportion to the expected effect and fatigue post-exercise. The diagnosis is based on clinical presentation, the presence of myoglobin in the urine and elevated serum creatine kinase levels, usually up to more than 10 times the normal upper limit.

Considering there is often elevated serum creatine kinase (CK) levels after strenuous exercise, there is no official cut-off value [1–3].

Rhabdomyolysis is a condition where muscle tissue is damaged and released into the blood stream. Damage to muscles results in elevated intracellular calcium levels which activates proteases and causes necrosis. Other proteins are cleared by the reticulo-endothelial system. Myoglobin, a protein located in the striated muscles is cleared by the kidneys. Myoglobinuria occurs when kidneys are unable to fully clear myoglobin out and it spills over into the urine resulting in a dark color. This is an important sign meaning one should seek medical help [2].

Exertion-induced rhabdomyolysis can evolve into compartment syndrome, intravascular fluid depletion, disseminated intravascular coagulation, pigment-induced acute kidney injury or even cardiac arrhythmia leading to a cardiac arrest. [2] The pathophysiology of this clinical entity is further described in Fig. 1.

Upon initial presentation based on clinical findings alone it can be difficult to differentiate with delayed-onset muscle soreness (DOMS) where a patient experiences myalgia the days after a strenuous unaccustomed exercise. DOMS is a very mild form of rhabdomyolysis. This becomes clinically relevant when there is extensive damage to the muscle fibers, the pain is severe, and large amounts of muscle proteins are released into the blood stream (e.g., creatine kinase [CK], lactate dehydrogenase [LDH], and myoglobin) [5].

Diagnostic imagery

While contributory, magnetic resonance imaging (MRI) findings are often non-specific. Clinical history is therefore essential in making the diagnosis. MRI images usually show

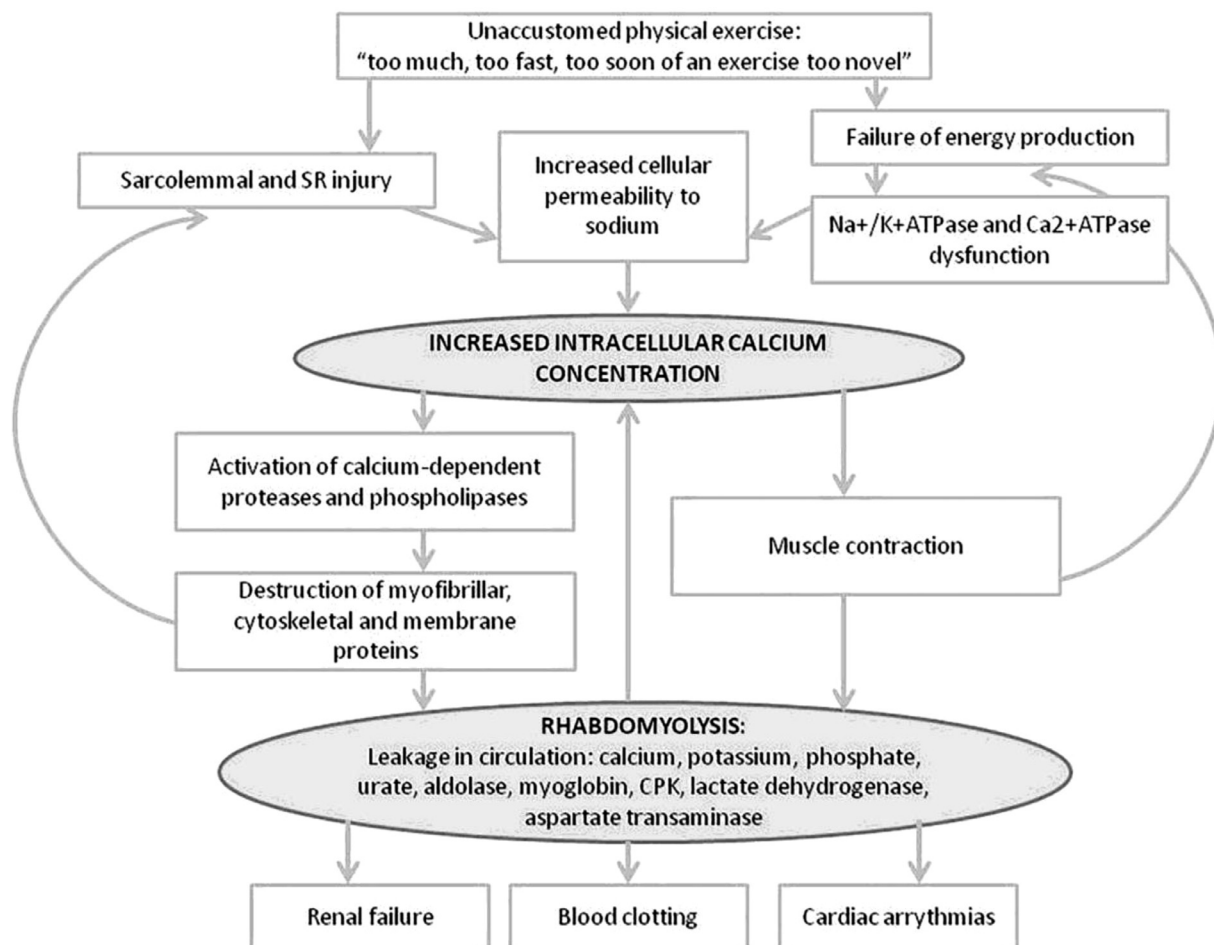


Fig. 1 – Pathophysiology of rhabdomyolysis. The pathophysiological events in rhabdomyolysis follow a common pathway, irrespective of its cause. CK, creatine kinase; SR, sarcoplasmic reticulum [4].

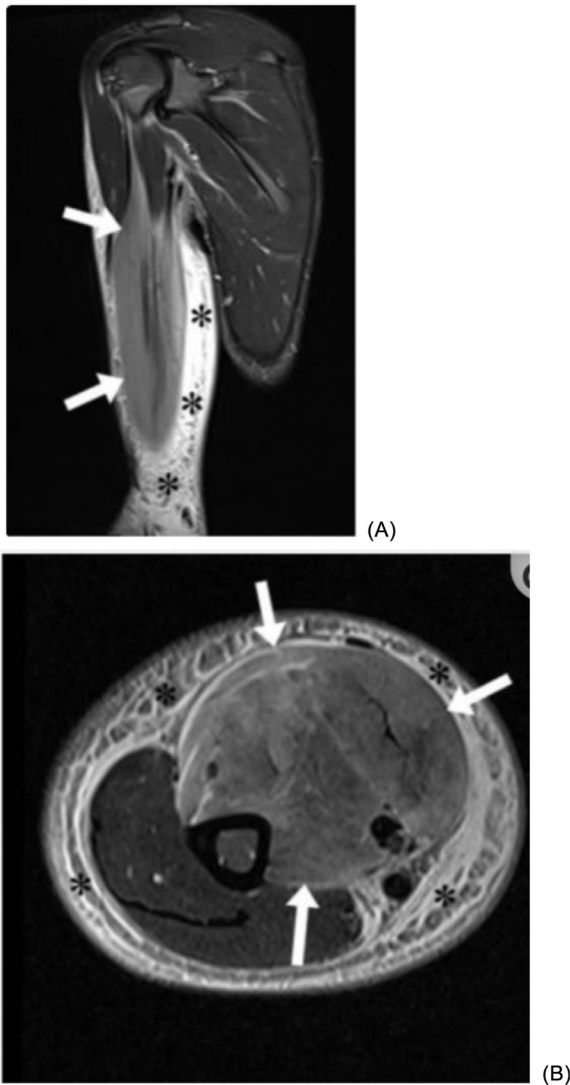


Fig. 2 – Coronal (A) and axial (B) inversion recovery images demonstrate hyperintensity in the biceps and brachialis muscles (white arrows) and subcutaneous edema (black asterisks) [9].

non-specific intramuscular hyperintensity on T2 weighed-sequences without appreciable fluid collection or intramuscular signal abnormality on T1-weighted images [6]. Still, MRI yields increased sensitivity when compared to computed tomography (CT) or ultrasound [7]. According to Robert et al. gradient echo imaging may be helpful in the late phase of the disease to distinguish hemosiderin seen in hemorrhagic transformation. Signal abnormalities within the affected muscle on MRI sequences may persist longer than abnormal serum CK values [7]. Based on imaging alone, differential considerations include cellulitis, necrotizing fasciitis, compartment syndrome, inflammatory processes, and diabetic myonecrosis [8]. Usually differentiation can be made based on anamnestic features and clinical history. **Figs. 2 and 3** display images of exertion induced rhabdomyolysis of the biceps, brachialis and triceps muscles on MRI.

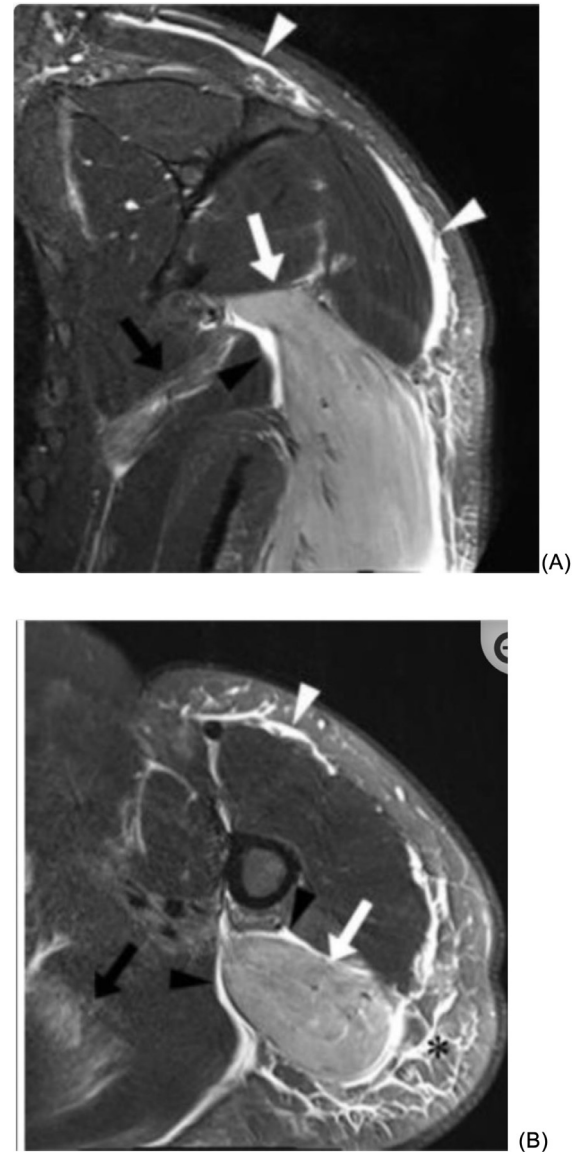


Fig. 3 – Coronal (A) and axial (B) inversion recovery images demonstrate hyperintensity in the long head of the triceps muscle (white arrows) and to a lesser extent teres major muscle (black arrows), superficial fascial signal abnormality (white arrowheads), deep fascial signal abnormality (black arrowheads), and subcutaneous edema (black asterisks) [9].

Case report

A 36-year old woman presents to the ER by referral of the general practitioner (GP) because of elevated CK-levels in a recent blood draw.

She complained of painful swelling in both upper arms for 3 days. An initial blood draw showed a total serum creatine kinase of 25,000 IU/L.

The patient was otherwise healthy, and showed no symptoms of nausea, vomiting or fever.

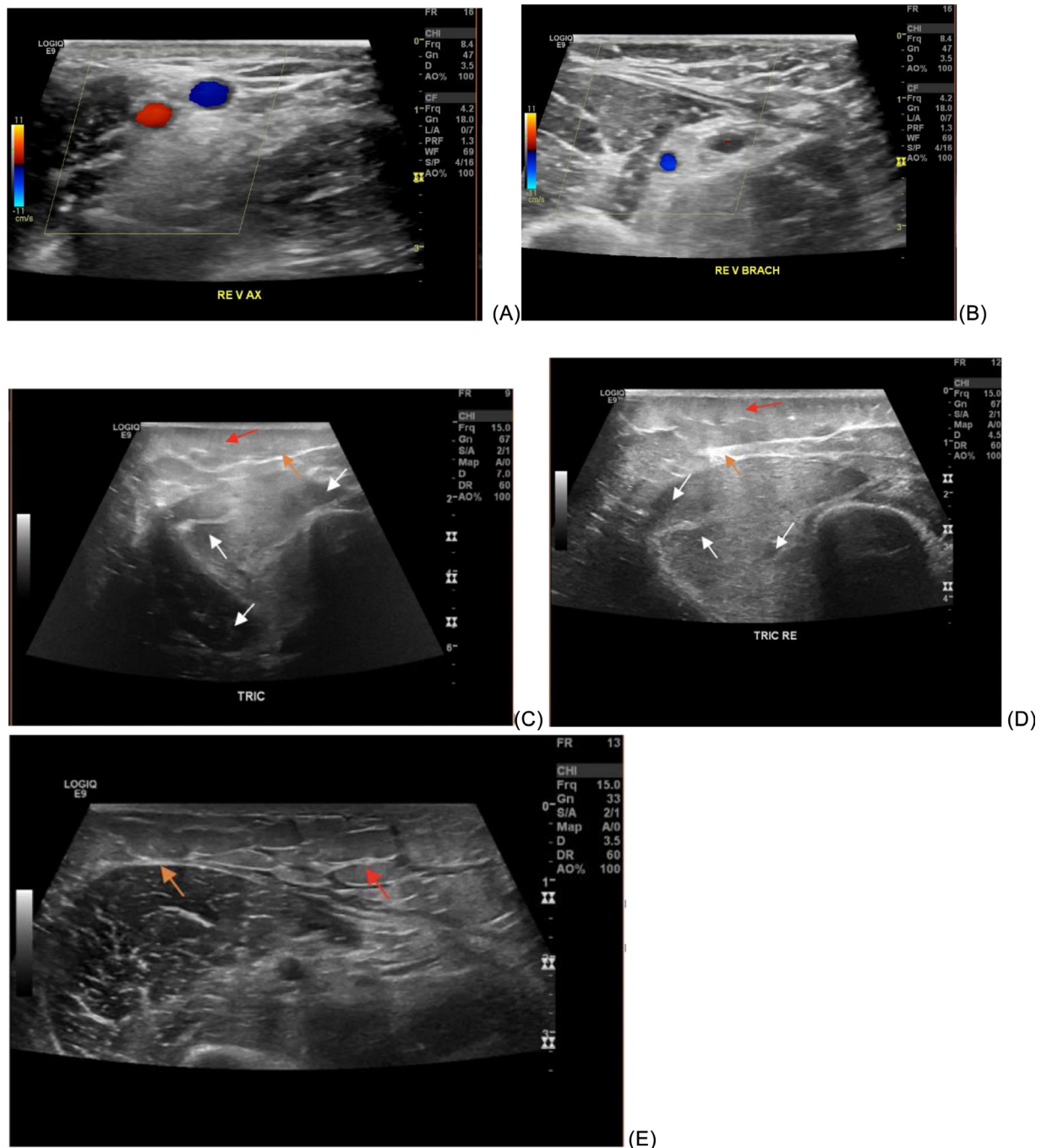


Fig. 4 – Color-doppler ultrasound images of patent right vena axillaris and vena brachiocephalica (image A and B). Ultrasound images of the triceps muscles in longitudinal section showing anechoic areas compatible with oedema (white arrows), increased muscle fascia (orange arrows) and infiltration of adjacent subcutaneous adipose tissue (red arrows) (image C, D, and E).

Patient noted a painful mobilization of the upper arms with limited painful extension of the forearms.

The clinical neurological examination was normal. Increased diameter of both arms could be observed, up to 28.5 cm on the left and 29.5 cm on the right. Both arms felt warm and hard, the left upper arm showed a hematoma.

An initial ultrasound (Fig 4) showed the veins of both arms to be patent but demonstrated a moderate edematous infiltra-

tion of the muscle bellies, more pronounced on the right side than the left and most pronounced in the musculus triceps muscle belly as well as associated edematous infiltration of adjacent subcutaneous adipose tissue.

She later on disclosed that she participated in a gym boot camp activity consisting of doing a lot of push-ups. She was otherwise a fit and athletic woman in her day to day life.

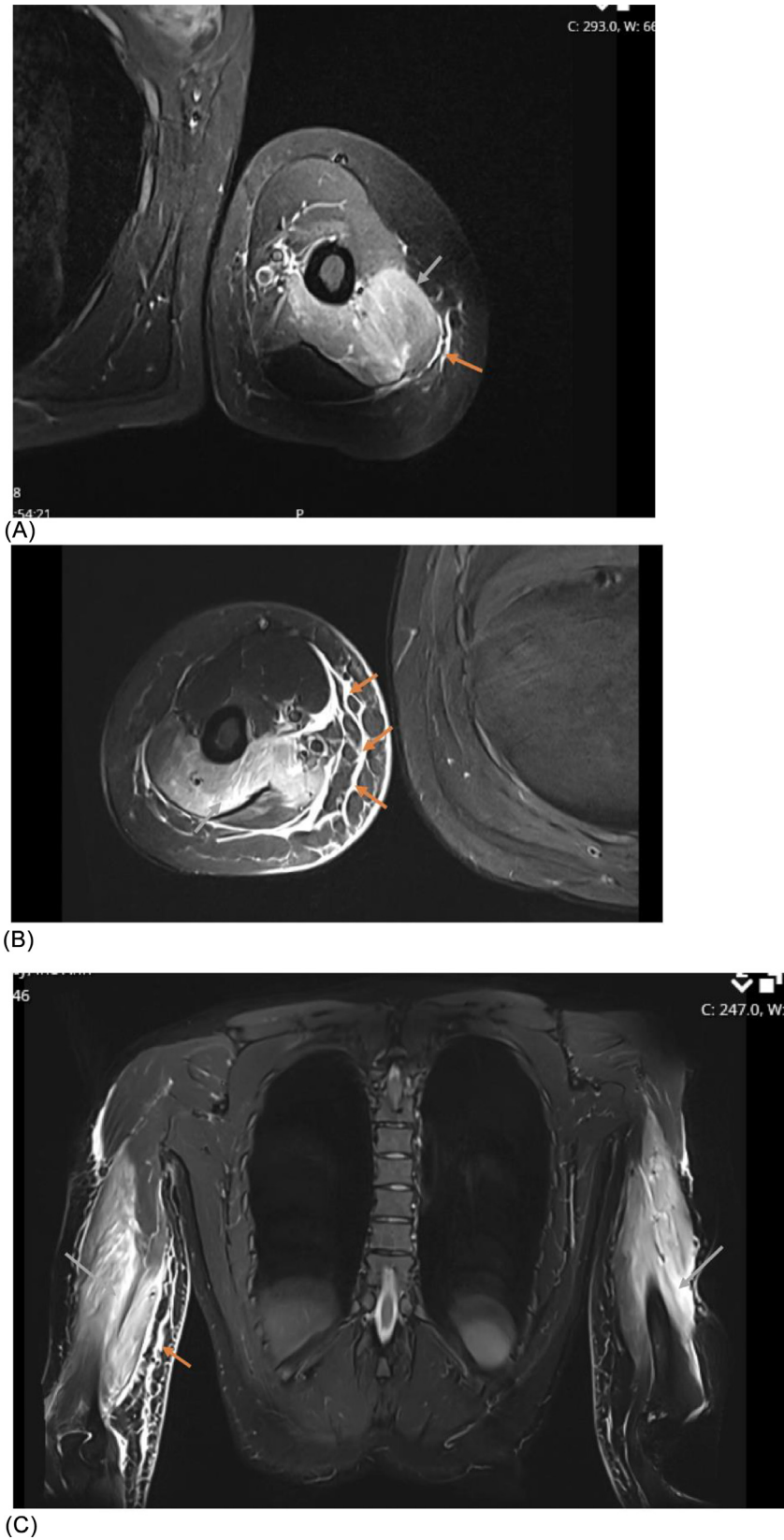


Fig. 5 – Axial (image A and B) and coronal (C) inversion recovery images demonstrate hyperintensity in the long head of the triceps right more than left (grey arrows), superficial fascial signal abnormality and subcutaneous oedema.(orange arrows).

Table 1 – Summarizing table of underlying causes of exertion-induced rhabdomyolysis.

Acquired causes	Congenital causes
Medication <ul style="list-style-type: none"> • Statins [19,20], isotretinoin [21,22], anti-psychotics, sedative hypnotics, antihistamines [1] 	Genetic disorders <ul style="list-style-type: none"> • McArdle disease • Carnitine palmitoyl transferase deficiency 2 • Myoadenylate deaminase deficiency • Malignant hyperthermia [24–26] • Ryanodine receptor-1 mutation [27] • Sickle-cell trait [28,29] • Duchenne Muscular Dystrophy [30,31]
Illicit drugs <ul style="list-style-type: none"> • Heroin, cocaine, amphetamine, methadone, LSD [1] 	
Infection <ul style="list-style-type: none"> • Influenza, coxsackie virus, malaria, herpes, HIV, legionella, salmonella [1] 	
Metabolic deficiencies <ul style="list-style-type: none"> • 25-OH-vitamin D deficiency [23] 	

Her upper-arms started feeling more tender the days after with painful flexion and extension of the elbows leading to her presentation at her GP's office. No other systemic symptoms were mentioned. Upon further questioning there was no use of illicit drugs in the past few days.

A number of differential diagnoses were considered such as a muscle tear, compartment syndrome, myositis, DOMS.

An MRI of both upper arms was performed showing a hyperintense signal in T2 in both triceps' muscles. Given the clinical presentation, the MRI-findings were consistent with a form of exertion-induced rhabdomyolysis of both triceps' muscles (Fig. 5).

She was admitted into the hospital to be administered IV-fluids (3-4 L during 24 h) to prevent acute kidney injury. During the hospital admission there was a preserved kidney function with progressive decline in CK serum values and myoglobinuria.

Patient was allowed to leave the hospital with a scheduled check-up MRI scan and a check-up blood draw at the GP. One week later her CK-serum levels had dropped to 393 U/L. The check-up MRI showed persistent increased signal alterations intramuscularly in the M. triceps, right side more than the left side, decreased compared to previous MRI examination with regression of the subcutaneous soft tissue infiltration. No necrosis foci were to be withheld. Following check-up showed a decrease of the diameter of both arms (right 26 cm, left 25.5 cm) with regain of muscle strength on both sides. A complete rheumatologic work-up with myositis serum anti-body testing remained negative.

This case report highlights a condition of exertion-induced rhabdomyolysis in a boot camp workout that does often go underdiagnosed or underreported.

Discussion

Exertion-induced rhabdomyolysis occurs most often in young male patients, at a mean age of 28 years [10].

Running, including marathons, and weightlifting are most likely to cause this condition. Other sports like spinning are

also reported as being a risky sport for developing it [11–13]. Backer et al.'s systematic review states that at the time of presentation the patients mean creatine kinase serum level was 31 481 IU/L (range 164-106,488 IU/L) [10].

Risk factors for developing exercise rhabdomyolysis include younger age, male sex, lower educational level and a lower chronic level of physical activity [14].

Many other causes of rhabdomyolysis have been described such as direct muscle injury from trauma, ischemia, extreme temperatures, chronic electrolyte disorders, endocrinological conditions (hypothyroid or hyperthyroid states), genetic disorders, auto-immune disorders, infections, drugs, toxins, venoms, immobility, and some congenital muscular dystrophy [5,15].

The classic characteristic triad comprises of myalgia, muscle weakness and dark-brown colored urine. However the full triad is often only seen in 1 to 10% of patients, making it even more difficult to diagnose [1,16,17]. Exertion-induced rhabdomyolysis is considered when one presents with muscle stiffness, swelling and pain out of proportion to the expected fatigue for one particular work-out. Usually an elevated serum-CK level helps diagnosing this condition. One of the major challenges in diagnosing it lies in the fact that CK serum levels often rise after a strenuous physical activity in almost all people up to 10 times the upper normal limit [18].

• Underlying causes

Underlying causes of exertion-induced rhabdomyolysis can usually be categorized in 2 groups, the acquired causes versus the congenital causes summarized in Table 1.

In general treatment involves aggressive fluid resuscitation, mostly with a saline solution at a rate of approximately 400 mL/hour, adjustments ranging between 200 and 1000 mL/hour depending on clinical presentation and severity [32].

Diagnosing this condition remains difficult as not all patients present with the classic triad of symptoms mentioned before, and there is no official cut-off value for serum CK-levels. Exercise induced compartment syndrome is a rare diagnosis with a high rate of poor outcomes. Therefore, it is important to consider it during work-up for rhabdomyolysis [33].

Though magnetic resonance imaging sequences remain non-specific in ER, in combination with the correct clinical information and diagnostic query it remains the exam by choice.

Patient consent

The authors obtained from the patient written informed consent for the publication of this case report and images.

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