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Dr. Sabriye ERCAN
Dr. Ersin Arslan Education and
Research Hospital, Department
of Sports Medicine, Gaziantep,
Turkey

Correspondence
Dr. Sabriye ERCAN
Dr. Ersin Arslan Education and
Research Hospital, Department
of Sports Medicine, Gaziantep,
Turkey

Perfect storm' induced acute kidney failure in an elite sprinter: A case report Sabriye ERCAN

Dr. Sabriye ERCAN

Abstract

Exercise is required for the physical and mental health of individuals. In these days, rates of sport participation have increased. However, doing sport more than the optimal level or the presence of various intrinsic/extrinsic risk factors may lead to the occurrence of uncommon diseases. The occurrence of the exercise-induced acute kidney failure is very uncommon. Some nephrotoxic drugs that are prescribed because of inaccurate diagnosis, any impairment in the liquid electrolyte balance during Ramadan or 'perfect storm' may have some effects in the development of this disease.

This case presents an acute kidney failure triggered by 'perfect storm' in an elite sprinter after Ramadan.

Keywords: Acute kidney failure; dehydration; exercise-induced rhabdomyolysis; non-steroid anti-inflammatory drug; athlete

1. Introduction

Nowadays, increased rates of sport participation lead to some positive developments in physical and mental health by increasing the level of physical activity. On the contrary, extremely intensive and/or severe exercises that exceed one's physical limits cause some musculoskeletal system injuries, which include exercise-induced rhabdomyolysis^[1].

Exercise-induced rhabdomyolysis is caused by damage to skeletal muscle cells. An intensive and/or severe exercise increases the levels of creatinine phosphokinase (CPK) or myoglobin, which in turn damages skeletal muscle cells^[1, 2]. This condition usually occurs in the presence of several risk factors, such as exercises of high-severity, long-term or sudden and intense muscle contractions, and so on^[1]. Type, duration, severity, intensity of the exercise constitute primary risk factors; whereas, warm weather, latent myopathy, lasting dehydration, electrolyte imbalance, sex, nutritional issues, use of nephrotoxic drugs [such as non-steroid anti-inflammatory drug (NSAID)], creatinine supplementation or alcohol use, and presence of viral/bacterial infection constitute secondary risk factors^[1, 3].

Running that exceeds physical limits in a warm and humid environment causes temperature stress and dehydration. However, even if CPK or myoglobin levels increase at the end of the race, the development of complications is rare because dehydration is relieved^[3]. If exercise-induced rhabdomyolysis is not diagnosed and treated correctly, some rare but severe complications may occur such as acute kidney failure, liver dysfunction, compartmentalization syndrome, heart failure, dysrhythmia, electrolyte imbalance, and death^[1, 4].

Causes of Acute kidney failure are well-defined. However some special situation may trigger the development of this disease. Muslim athletes should be careful to nutrition and hydration during Ramadan. This case presents an acute kidney failure triggered by 'perfect storm' in an elite sprinter after the exercise-induced rhabdomyolysis following Ramadan.

2. Case Report

A 24-years old male (height: 173 cm, weight: 60 kg) visited Sport Medicine Clinic with complaints of pain in his bilateral thigh muscles and not being able to fold down his knees. The patient was a 100-meter elite sprint athlete. He revealed no known illness or no constant medication/supplement use in his medical history. Since he was fasting during Ramadan, he gave a 1-month break to his training. His liquid intake during Ramadan was 1.5 L/day.

After Ramadan, the patient started his training at the same level as he was doing it before. His training program constituted 75 minutes of running and 15 minutes of jumping exercise. He carried out his training outside at a temperature of 30 °C. He had no liquid intake in training session.

The patient stated that he felt bilateral thigh weakness, the range of motion in his bilateral knee joint got restricted, and he had difficulty to step up the pavement. The patient's discomfort aggravated 48 hours after the training, muscle swelling and a feeling of tightness occurred in the anterior compartment of the bilateral thigh, the range of motion in his bilateral knee joint got further restricted, and the patient could not climb the stairs. He visited another emergency service, and he had NSAID (75 mg diclofenac) and myorelaxant (4 mg thiocolchicoside) injections intramuscularly before he was discharged.

The patient's muscle pain increased gradually 24 hours after admission to the emergency service. He felt his muscles "as stiff as a wire". As the discomfort increased, acetaminophen (10 mg/mL, 100 mL) was administered intravenously to the patient in the emergency service of another hospital. Also, he was prescribed NSAID (indomethacin) and myorelaxant (thiocolchicoside) (orally and locally) and discharged.

The patient could continue only to use local NSAID but not myorelaxant because of nausea and vomiting sensation. Nausea and vomiting increased the severity of dehydration, and the urine output decreased. The last urination was 24 hours before his visit to Sport Medicine Clinic, and the color of the last urine was brownish. During the examination performed in the Sport Medicine Clinic, the patient was conscious, co-operated, and oriented. His pulse was 76 per minute; his arterial blood pressure was 110/80 mmHg, his respiratory rate was 20 per minute, and finally his body temperature was 36 °C.

His bilateral thigh anterior compartment muscles were extremely stiff, and the pain with palpation increased as a strain was applied to the anterior compartment. Weakness was observed in the bilateral frontal compartment muscles (muscle force on the right extremity: +4/5, left extremity: +3/5). The bilateral knee flexion of the patient was 30°.

There was no ecchymosis, paleness, coldness, loss of sensation, or sensation impairment in the bilateral lower extremity of the patient. Pulses belong to bilateral femoral artery, tibialis posterior artery, dorsalis pedis artery were pulsatile.

The laboratory analyses of the patient revealed that the numbers of white blood cell, hemoglobin, hematocrit, platelet, and coagulation parameters were in a normal range. Other parameters were as follows: sedimentation 51, C-reactive protein 39.5mg/L (N: 0–3mg/L), blood creatinine 12.35 mg/dL (N: 0.84–1.25 mg/L), aminotransferase (earlier known as serum glutamic oxaloacetic transaminase) 3496 U/L (N: 0–35U/L), alanine amino transferase (earlier known as serum glutamic pyruvic transaminase) 764 U/L (N: 0–45U/L), CPK 33,700 U/L (N:0–171), blood urea 174.2 mg/dL (N: 10–40mg/dL), blood urea nitrogen 81 mg/dL (N: 5–18mg/dL), lactate dehydrogenase 4246 U/L (N: 0–248U/L), uric acid 9.45 mg/dL(N: 2.6–6mg/dL), blood potassium 5.34 mmol/L (N: 3.3–5.1mmol/L), blood calcium 7.7 mmol/L (N: 8.8–10.6mmol/L), blood phosphorus 6.44 mg/dL(N: 2.5–4.5mg/dL), blood sodium 136 mmol/L (N: 136–146mmol/L), and albumin 4.2 g/dL(N: 3.5–5.2 g/dL). His urine was cloudy and brownish. Bilirubin, ketone, and urobilinogen were negative in the urine.

A small amount of glucose (mg/dL), 2+/GL leukocyte,

positive nitrite (SI), 3+/GL erythrocytes, and abundant protein were found in the urine. The pH was 6.5 (SI). In the urine microscopy, 409/high-power field (HPF) erythrocyte, 3/HPF leukocyte, 2+/HPF flat epithelial cells, and yeast were detected.

The patient was diagnosed with rhabdomyolysis-induced acute kidney failure, and an aggressive intravenous liquid treatment (1 L/hour isotonic liquid in the first hour, then 1 dose of 0.84 g/10 mL sodium bicarbonate in the 500 mL isotonic liquid) was started for the patient. The nephrology consultant physicians advised an urgent dialysis for the patient, and he underwent dialysis.

3. Discussion

A feeling of tension in the bilateral tight anterior compartment muscles, muscle stiffness, muscle pain with palpation, and a stress-induced increase in the muscle pain were detected in the present case. The inner-compartment pressure measurement was not performed for the patient because there were no compartment-syndrome signs such as paleness, no pulse, paresthesia, and paralysis.

The patient was diagnosed with acute kidney failure based on the sequence of events from his medical history, such as performing a sudden and vigorous running and jumping exercise in warm air, dehydration, increasing discomfort after NSAID use, decreased/no urination, and brownish urine color. The patient's laboratory analyses supported the diagnosis.

Exercise-induced rhabdomyolysis is a clinical condition, which is characterized by exercise-induced muscle pain, brownish urine, increase in the CPK level five times more than the normal level, and myoglobinuria [5]. Studies have shown that rhabdomyolysis occurs at various levels in 40% of the cases after 6 days of exercise [6]. The rare complications of the rhabdomyolysis may occur with secondary risk factors, resulting in the increase of CPK level [7].

It was reported that exercise-induced rhabdomyolysis is seen in people who never exercise or do little exercise, untrained soldiers, amateur athletes, weightlifting athletes who are less experienced or do overweight training, ones who do squat jumping training, ones who had eccentric muscle contraction because of prolonged and vigorous exercise, and males. Lasting dehydration after a prolonged physical activity in the warm and humid environment is a risk factor. Additionally, frequently used drugs such as creatinine supplements and NSAID and also alcohol increases the nephrotoxic effect [1, 6]. Renal vasoconstriction, ischemia, myoglobin cast formation in the distal tubule, and direct cytotoxic effect in the proximal tubule cells play a role in the pathophysiology of myoglobinuric acute kidney failure [5, 8]. The factors that decrease the renal blood flow, such as dehydration and NSAID, make a clinical condition of this disease more severe [1, 6, 9].

It was reported that acute kidney failure secondary to myoglobinuria is 10%–40% of different case series [4, 8, 10, 11]. In 80% of the cases, renal functions were expected to be repaired in 30 days. Need for acute hemodialysis occurs only in 4% of the cases [5]. Only a small proportion of the cases end up with a need for renal transplantation or death [9, 11].

4. Conclusion

Perfect storm' are several factors such as heat stress, dehydration, latent myopathy, non-steroidal anti-inflammatory or other drug/analgesic use, and viral/bacterial infection. Various combinations of these factors can lead to exercise-induced rhabdomyolysis and acute renal failure.

Awareness of athletes and coaches is important to avoid development of exercise-induced rhabdomyolysis. In particular, Muslim athletes should pay more attention to maintain the electrolyte balance of the body after prolonged dehydration processes such as Ramadan.

Physician should inform athletes so that these risks do not coexist and precautions must be taken. If the athlete has these risks, the preliminary diagnosis of acute renal failure should be considered. It should not be forgotten that NSAIDs, which are frequently used by athletes and prescribed by physicians, make this clinical status worse. It is vital to avoid nephrotoxic drugs (NSAIDs) and make an early, aggressive liquid therapy to inhibit complications^[9].

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