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Letter to the Editor

An Initial Episode of Rhabdomyolysis Following Excessive Physical Exertion Requires a Thorough Investigation of the Underlying Cause

Josef Finsterer
Neurology & Neurophysiology Center, Vienna, Austria

Corresponding Author: **Josef Finsterer**

Letter to the Editor

We read with interest the article by Baig *et al*. regarding a previously healthy 26-year-old man who developed severe rhabdomyolysis (initial creatine kinase [CK] level of 260,100 U/L) and myoglobinuria following a session of high-intensity functional training after a prolonged period of inactivity [1]. Surprisingly, renal function parameters and serum electrolytes remained within the normal range throughout the period of massive CK elevation [1]. The patient was treated with intravenous fluids, and the hyper-CK-emia resolved within four days [1]. The study is informative, yet certain aspects warrant discussion.

First, the absence of renal impairment in patients with rhabdomyolysis is not unusual and has been described previously [2]. Whether or not renal dysfunction develops depends on numerous factors, such as the timing of treatment initiation, baseline renal function, age, hydration status, acidic urine, concomitant nephrotoxic medication, underlying conditions (e.g., diabetes, sepsis, pre-existing kidney disease, electrolyte disturbances), and acidosis [3]. Were any of these factors present in the patient described here at the time of the rhabdomyolysis?

A second point is that the cause of the rhabdomyolysis was not determined [1]. Although excessive physical exertion can trigger rhabdomyolysis in isolated cases [4], extremely high CK levels attributable solely to physical activity, as seen in the index patient, are unusual. It is therefore conceivable that the index patient actually suffered from an underlying subclinical condition that became symptomatic due to the excessive physical strain.

A third point is that there is no information regarding the patient's diet [1]. Since certain foods can also trigger rhabdomyolysis, it is crucial to know what the patient regularly consumed. Was there any evidence of regular consumption of quail contaminated with hemlock, poisonous mushrooms (e.g., Russula subnigricans, Tricholoma equestre), certain fish species (Haff disease), licorice, energy drinks or caffeine, dietary supplements (such as creatine, Hydroxycut, synephrine, or ephedrine), herbs (such as Ephedra or Tribulus terrestris), or of excessive sodium intake, a high-protein diet, electrolyte imbalances, grapefruit, or red yeast rice [5]?

A fourth point is that there were no reports on whether the patient had engaged in excessive exercise prior to the current event [1]. If he had repeatedly exercised without developing rhabdomyolysis, it is rather unlikely that physical exertion was the trigger for the current event. Alternatively, it is conceivable that he had previously experienced rhabdomyolysis, but that it had run a subclinical course. If a patient does not notice symptoms such as altered urine color, myalgia, muscle weakness, fatigue, nausea, fever, and cardiac arrhythmias, it is quite likely that such episodes of rhabdomyolysis remain subclinical.

Another point is that, following the resolution of hyper-CK-emia, the patient was not thoroughly evaluated for alternative causes of rhabdomyolysis. Rhabdomyolysis can also stem from genetic disorders that often run a subclinical course, such as a predisposition to malignant hyperthermia. Has the patient ever undergone general anesthesia, and did any complications arise? Furthermore, rhabdomyolysis can be triggered by the use of illicit drugs (e.g., cocaine, heroin, amphetamines), infections, hypokalemia, hypocalcemia, hyperthermia, or even hypothermia [6]. Were these potential triggers for rhabdomyolysis considered and carefully ruled out?

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