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Let's not blame cocaine as a cause of rhabdomyolysis until all other causes have been ruled out☆,☆☆

Letter to the editor,

We read with interest the article by Lugo-Fagundo et al. about a 56-year-old male with rhabdomyolysis in whom the condition was diagnosed by muscle computed tomography (CT) [1]. His previous history was positive for polytoxicomania [1]. The study is appealing but raises concerns that should be discussed.

Rhabdomyolysis is usually diagnosed upon the clinical presentation and blood chemical values. Patients complain about tiredness, myalgia, cola-colored urine, and urinary retention. Clinical exam may reveal muscle weakness, reduced tendon reflexes, sore muscles, swelling, and stiffness. Blood tests may show elevated muscle enzymes, particularly creatine-kinase (CK) of five times the upper limit, myoglobinemia, myoglobinuria, hypocalcemia, hyperkaliemia, and renal insufficiency.

We disagree with the statement in the introduction that non-traumatic causes of rhabdomyolysis only include alcohol abuse, illicit drugs, and pharmaceutical agents [1]. The spectrum of causalities of non-traumatic rhabdomyolysis is much broader and additionally includes bacterial or viral infections, including sepsis, seizures, primary or secondary myopathy, snake or insect bites, hyperthermia, or ketoacidosis.

We disagree with the notion that the patient had myositis [1]. CT clearly showed "decreased enhancement" suggesting that rather myocyte necrosis or connective tissue replacement rather than inflammation was responsible for the findings. Myositis usually presents with muscle enhancement on muscle CT [2]. In case of uncertainty about the diagnosis myositis, a muscle biopsy should be carried out.

Initially absent blood flow in the left tibial and peroneal vessels may not only be due to compression by edematous surrounding muscles but could be also due to vasospasm. Because it is known that cocaine leads to vasospasms not only of the cerebral arteries but also of arteries in other regions [3,4], it is conceivable that absent blood flow was due to hypercontraction of these arteries.

Because the patient regularly took cocaine, it is quite surprising that he developed rhabdomyolysis this time. It should be discussed if this was due to cocaine overdose, low-quality cocaine, or a co-factor triggering muscle necrosis. Was there any evidence for a seizure or status epilepticus, such as tongue biting or urine loss, during the night prior to admission? Were infections parameters elevated? Did the patient report fever?

Missing is the individual history for previous episodes of rhabdomyolysis, complications during general anesthesia, and previous secondary myopathy. Missing is also the family history for primary muscle disease.

Missing are the results of work-up for primary and secondary myopathy during follow-up. Patients with unknown cause of rhabdomyolysis should undergo thorough neurological investigations, including needle electromyography, muscle biopsy, and biochemical investigations of the muscle homogenate.

Overall, the interesting study has limitations that call the results into question and their interpretation. Clarifying these weaknesses would strengthen the conclusions and could improve the study. Although regular cocaine use suggests cocaine-induced rhabdomyolysis, all other differentials should be thoroughly ruled out.

Ethics approval

Ethics approval was in accordance with ethical guidelines. The study was approved by the institutional review board.

Patient consent

Consent to participate: was obtained from the patient.
Consent for publication: was obtained from the patient.

Abbreviations: CT, computed tomography; CK, creatine kinase.

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Availability of data

All data are available from the corresponding author.

Code availability

Not applicable.

Author contribution

JF: design, literature search, discussion, first draft, critical comments, final approval.
SM: literature search, discussion, critical comments, final approval.

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