

LETTER TO THE EDITOR

DON'T FORGET ABOUT CONCOMITANT MEDICATIONS AND COMORBIDITIES AS CAUSES OF RHABDOMYOLYSIS

Dear Editor:

We read with interest the article by Wen et al. (1) about a 69-year-old multimorbid woman who, 4 months after implantation of an intrathecal morphine pump, developed weakness of the proximal muscles of the lower extremities and elevated serum creatine kinase (CK), mild hypokalemia and a serum lactate level of 2.5 mmol/L. The patient was diagnosed with rhabdomyolysis (RM) after a fall and the morphine dose was reduced by 50% (1). However, the muscle weakness did not disappear (1). The study is interesting, but some points require discussion.

The first point is that we disagree with the diagnosis of RM (1). The patient had a maximum serum CK value of 1687 U/L, but had been lying on the floor for three days when she was discovered. Patients who are found immobile on the floor often have elevated serum CK levels because certain muscle groups are under constant pressure during immobility. Another strong argument against RM is that the patient had hypokalemia (1). RM is associated with hyperkalemia in most cases, but not usually with hypokalemia. Only in cases where hypokalemia is the cause of RM can low serum potassium levels be detected. Another argument against RM is that CK levels were low (1). CK levels above 5000 U/L are generally considered to be RM.

The second problem is that alternative causes of hyper-CKemia or contributing factors were not sufficiently considered and discussed (1). The patient was taking atorvastatin. Statins are known to cause RM under certain contributing circumstances, such as fever or concomitant medications (2). Albuterol is also known to cause RM in rare cases (3). The patient was also taking furosemide, which is known to cause hypokalemia, which in turn can lead to RM (4). Overdoses of gabapentin and lamotrigine have also been reported to cause RM (5,6). There are also reports of semaglutide causing RM in rare cases.

The third problem is that there was no history of epilepsy, but the patient was receiving lamotrigine (1). Lamotrigine is usually administered as an antiepileptic drug, but is also used in bipolar disorder. We should know the indication and dosage for which the patient received lamotrigine and what her serum levels were. Was the hyper-CKemia actually due to an epileptic seizure?

The fourth point is that we disagree with the statement in the introduction that a muscle biopsy should be considered when a genetic muscle disease is suspected (1). If a genetic cause is suspected, a detailed medical history of the patient and their family must be taken, differential diagnoses of genetic causes of RM must be ruled out, and genetic testing should be performed. A muscle biopsy shortly after RM is not meaningful, as most likely only necrotic muscle fibers will be visible. If at all, a biopsy should be performed at the earliest six months after the RM event.

Finally, no long-term results on muscle weakness and hyper-CKemia have been reported (1). Long-term follow-up data are essential to assess whether the 50% reduction in the opiate dose actually led to a complete recovery from muscle weakness. Were CK and lactate levels still above normal 6 months after the fall? We should know whether values higher than the only value given in Table 1 were ever measured and when serum CK returned to normal. The course of serum CK over time should be specified.

In summary, hyper-CKemia in a multimorbid patient receiving multiple medications that can trigger RM cannot be attributed to an opioid overdose until all other possible causes have been sufficiently ruled out.

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