Myoclonus in Exertional Rhabdomyolysis Without Renal Failure

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Myoclonus can present as a secondary movement disorder to various conditions such as hypoxia or metabolic disorders including renal failure. Rhabdomyolysis is not infrequently complicated with renal failure and myoclonus. In this case, myoclonus is diagnosed as a secondary manifestation of renal failure.

Herein, we report a patient presenting with rhabdomyolysis and myoclonus without renal failure.

CASE REPORT

A 66-year-old man was admitted to our institute due to sudden-onset involuntary movements. He had been healthy with unremarkable medical or family history. He was not a regular exerciser and had started a very intensive fitness program three days before admission. The program did not include dietary restriction. In particular, the program included repeated squatting sessions, which was not typical in the patient's daily activities. The session left him nearly exhausted. He did not take any medication including recreation drugs and diet pills before the onset of his symptom. He did not drink alcohol before and during the fitness program.

On presentation, he was alert and well-oriented. His neurologic examination was normal with normal reflexes. The involuntary movements were irregular, fast, jerky, and continuous, affecting mainly the upper extremities and trunk and could not be voluntarily suppressed, which was compatible with myoclonus.

Laboratory tests showed increased levels of creatine kinase (CK, 1906 U/L; normal range = 60 ~ 220 U/L) and lactate dehydrogenase (LDH, 574 U/L; normal range = 263 ~ 450 U/L) and normal levels of T3 (92 ng/dL), free T4 (1.14 ng/dL), thyroid-stimulating hormone (0.79 uU/mL), blood urea nitrogen (BUN, 15 mg/dL) and creatinine (Cr, 0.8 mg/dL) and a normal BUN/Cr ratio (18.75). His serum glucose mildly increased (113 mg/dL, hemoglobin A1c = 6.4%). Oral glucose intolerance test was compatible with mild diabetes mellitus. Liver enzymes were mildly elevated (aspartate aminotransferase/alanine aminotransferase = 66/43 U/L), which was normalized two days later (aspartate aminotransferase/alanine aminotransferase = 33/33 U/L). The serum lactic acid level was not elevated (0.48 mmol/L). The levels of electrolytes were within normal limits (sodium = 137 mEq/L, potassium = 4.3 mEq/L, total calcium = 8.1 mg/dL). Arterial blood gas analysis showed normal results. His brain magnetic resonance imaging was unremarkable except incidental meningioma (24 × 18 × 13 mm) in the right frontal convexity. His electroencephalography was normal.

No specific medication for myoclonus was administered. His myoclonus gradually improved and completely disappeared on the third morning after admission, with resolution of rhabdomyolysis (CK = 184 U/L). He remains symptom free for more than one year.

DISCUSSION

Rhabdomyolysis can be caused by various disorders ranging from direct muscle injury to genetic problems. Some cases of rhabdomyolysis after strenuous physical activity have been reported and are classified as exertional rhabdomyolysis (ER). The mean serum level of CK was variable among the reported cases (3812 to 7713 IU/L).[1]

Patients with various medical problems such as fasting, hypokalemia, hypovolemia, and inherited metabolic myopathies are vulnerable to ER.[1] Our patient presented without significant medical problems related to the rhabdomyolysis except excessive physical activity a few days prior to symptom onset. The close temporal relationship between the exercise and the development and spontaneous resolution of rhabdomyolysis without recurrence was compatible with ER.

Myoclonus is one of the clinical manifestations of uremic encephalopathy. In cases of uremic encephalopathy, the accumulation of various toxins, caused by impaired renal function, is considered as a possible pathomechanism.[2] In this case, there was no evidence of renal failure. Thus, alternative causes such as the accumulation of LDH or CK could be suspected.

Physiologically, LDH can remove increased lactic acid after exercise, thereby reducing muscle fatigue. However, no direct toxicity has been ascribed to LDH. In some patients, CK increased without cause (idiopathic hyperCKemia, IHCK), in which direct role of CK could be considered. Fatigue and muscle pain were reported in 10-30 % of patients with IHCK in a previous study.[3] Most of the reported patients with IHCK had a benign course, while a few patients suffered neurologic disorders such as ominous neuromuscular disease.[3,4] However, there was no previous report of myoclonus associated with IHCK.

Hyperexcitability was reported to be important in the pathogenesis of myoclonus. Elevated blood lactate could increase neuronal excitability, but serum lactate level was normal in

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our patient.[5] However, although serum level of creatinine or lactate was normal, derangement of local milieu inside the brain by CK or LDH cannot be completely excluded.

To our knowledge, symptomatic myoclonus secondary to ER without renal failure has never been reported. Further studies are needed to confirm possible direct roles of these enzymes on myoclonus.

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