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Letter To The Editor

Can Statins Trigger Takotsubo Syndrome?

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LETTER TO THE EDITOR

In a recent article, Kamada *et al*. reported about a 73yo male with Takotsubo syndrome (TTS) presumably triggered by statin-triggered (rosuvastatin) rhabdomyolysis [1]. We have the following comments and concerns.

The patient is reported to had to lower limb muscle weakness [1]. Was muscle weakness associated with pain, muscle cramps, or gait disturbance? Which was the cause of lower limb muscle weakness? Were deep tendon reflexes reduced or exaggerated? Were pyramidal signs positive or negative? Was there reduction of the muscle tone or spasticity? Was muscle weakness associated with ataxia? Was the MRI of the lumbar spine normal? Which were the results of nerve conduction studies? Were there any indications for polyneuropathy? Which were the findings on needle electromyography? Was the family history positive for classical muscle manifestations of a neuromuscular disorder, such as exercise intolerance, easy fatigability, muscle weakness, cramps, stiffness, impaired relaxation, muscle hypotonia, or myalgia? Were there any indications for a beta oxidation defect?

Patients developing statin myopathy more frequently have an underlying mitochondrial disorder (MID) than those who do not suffer from a MID [2]. Were there any indications from the individual or family history suggesting a genetic mitochondrial defect in the index case? The patient complained about acute-onset general fatigue, dysbasia and had a history of hyperlipidemia, diabetes, and atherosclerosis. Which were his cardiovascular risk factors in addition to hyperlipidemia and diabetes? Did he smoke, was there arterial hypertension? Was the family history positive for premature atherosclerosis? Was renal insufficiency attributed to diabetes or due to other causes? Which were the current HbA1c values?

Since subarachnoid bleeding is the most frequent CNS cause of TTS [3], it would be interesting to know if the patient complained about headache and if the cerebral CT scan was normal. Since the second most frequent CNS trigger of TTS is epilepsy [3], we should be informed if there were any indications for a history of epilepsy in this patient.

Though it is indicated in the title that there was rhabdomyolysis from a statin therapy it is not discussed why rhabdomyolysis could trigger TTS? Was rhabdomyolysis symptomatic or asymptomatic? Did the patient undergo muscle biopsy after resolution of rhabdomyolysis? Was there a clear-cut time correlation between onset of the statin therapy and rhabdomyolysis?

Concerning the outcome of TTS, we should be informed if and after which time ECG and echocardiography were completely normal after resolution of TTS?

Overall, this interesting case could profit from provision of a more extensive family and individual history, from a more detailed description of the clinical findings, from more extensive diagnostic work-up for rhabdomyolysis, and from a broader discussion how rhabdomyolysis could trigger TTS.

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