Takotsubo Syndrome in Metabolic Myopathy Unmasked by Statin-triggered Rhabdomyolysis

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To the Editor We read with interest the article by Kamada et al. about a 73-year-old man in whom rhabdomyolysis under long-term statin treatment was suggested to be a trigger of Takotsubo Syndrome (TTS) (1). We have the following comments and concerns.

We do not agree that TTS is a “reversible disorder” in each case. Classical, as well as inverted TTS is associated with a fatal outcome in some patients (2).

Was paraparesis already present prior to the initiation of statins? What was the degree of muscle weakness on the Medical Research Council scale? Infection was suspected to have been causative (1). Since no reference limits are provided in the Table, it is unclear if the infection parameters were abnormal. In case there was no “infection, injury, or mental stressor”, what was the cause of paraparesis? Was it due to rhabdomyolysis, or statin-myopathy, or another peripheral nervous system disease? It is also unclear whether the patient’s muscle weakness completely resolved after discontinuation of the statin.

Rhabdomyolysis was attributed to rosuvastatin, which has not been reported as a trigger of rhabdomyolysis (1). Since the patient has taken rosuvastatin for at least four years, it is rather unlikely to have caused hyper-creatine-kinase-emia. The authors themselves state that administration of statins for longer than three months is associated with a reduced risk for developing myopathy (1). Furthermore, the statin was given at a low dosage, which is less likely to cause myopathy than high-dose statin treatment (3). Did they also consider pioglitazone or the combination with the statin or other antidiabetic drugs as triggers of rhabdomyolysis, as has been reported (4)?

It is suspected that patients who develop statin-myopathy actually suffer from subclinical metabolic myopathy, which may be disclosed by various triggers, such as muscle-toxic medication, infection, or metabolic stress (5). Indications for a systemic metabolic defect were the presence of diabetes, hyperlipidemia and the history of Leriche Syndrome. Were creatine-kinase values also elevated prior to the initiation of statins or after the resolution of rhabdomyolysis? Did the patient report typical muscle symptoms prior to initiating statins? Was the family history positive for mitochondrial Multi Organ Disorder Syndrome (MIMODS) including myopathy? Did the patient have a history that was positive for problems during or after general anesthesia? An argument for primary myopathy underlying rhabdomyolysis is that metabolic myopathy has been reported in association with TTS.

Overall, this interesting case could profit from a more extensive work-up of suspected myopathy in the patient and his first-degree relatives. Subclinical cardiac involvement in myopathy may promote the development of TTS, in case classical TTS triggers suddenly appear. Subclinical myopathy favouring the development of TTS specifically include metabolic disorders due to defects in fatty acid or oxidative metabolism.

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References


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