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Reply to Letter to the Editor: "Spontaneous Coronary Artery Dissection in the Setting of Duchenne Muscular Dystrophy: More Questions Than Answers"

LETTER TO THE EDITOR
REPLY

To the Editors,

Thank you for your attention and interest in our manuscript.[1]

Duchenne muscular dystrophy (DMD) is a severe, progressive, muscle-wasting disease that leads to difficulties with movement and eventually to the need for assisted ventilation and premature death. The disease is caused by mutations encoding dystrophin that abolish the production of dystrophin in muscle. Dystrophin is a cytoskeleton protein. That is why DMD primarily affects muscle cells and it does not affect vascular structures. Therefore, no association of spontaneous coronary artery dissection (SCAD) has been reported.

In some previous studies, factors such as depression, anxiety and stress are common in these patients. It has been suggested that emotional and physical stress may trigger SCAD in those with underlying arteriopathy. These factors can increase catecholamine levels and intra-thoracoabdominal pressures, which can increase arterial vascular stress.[2-4]

In our opinion, SCAD is not related to dystrophin deficiency in patients with DMD, but it may be related to endogenous stress and side effects of drugs rather than progressive vasculopathy.

SCAD in patients with Duchene muscular dystrophy may occur due to the drugs used and exposure to various stressors as previously mentioned. Steroids have many side effects, especially those that have potentially serious fragile side effects on vascular structures. Ataluren is an investigational and orally administered nonantibiotic drug that appears to promote ribosomal read-through of nonsense (stop) mutations to allow bypass of the pathogenic variant and continuation of the translation process to the production of a functioning dystrophin protein. Ataluren have potential side effects like vomiting, constipation, susceptibility to infections, headache, cough but spontaneous coronary artery dissection has not been reported. In addition, potential side effects of ataluren mentioned before might have triggered SCAD in our patient.

The assisted walking patient was admitted to our hospital with the complaint of chest pain; the patient was evaluated urgently upon the detection of significant myocardial infarction findings on the patient's electrocardiography (ECG), coronary angiography was performed under emergency conditions, and a stent was placed in the patient with dissection in the coronary artery and the stability of the patient was ensured. From his history, we learned that he did not experience any of the known side effects of ataluren such as vomiting, headache, weakness, and hypotension.

Our patient follows up in another center due to DMD. That is why, we do not know the ECG changes of the patient before he applied to us and whether the Holter examination was performed and how often it was done.

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The patient did not have cardiac magnetic resonance imaging taken beforehand. Therefore, the changes that may exist are unknown. However, our ECG examination did not show myocardial changes suggestive of cardiomyopathy.

Thank you for these constructive suggestions and comments.

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