Differential Expression of Cardiac Troponin T and I in a Patient with Isolated Skeletal Muscular Sarcoidosis

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Abstract

A 49-year-old female was referred to our hospital due to high serum creatine kinase (CK) (2,605 IU/L) and serum cardiac troponin T (cTnT) (0.342 ng/mL) levels. She had no other complaints and further examinations suggested no signs of cardiac disease. Additionally, the serum cardiac troponin I (cTnI) levels were normal. She reported having gradually felt difficulty in walking upstairs. A biopsy indicated skeletal muscle sarcoidosis with positive staining for cTnT. Steroid therapy immediately resolved her muscular symptoms with normalization of the serum CK levels. Since the serum levels of cTnI were normal, the concomitant measurement of cTnT/cTnI might be useful to diagnose skeletal muscular disease biochemically in such cases.

Key words: cardiac troponin T, cardiac troponin I, sarcoidosis, skeletal muscle

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Introduction

Sarcoidosis is an inflammatory disease characterized by the presence of noncaseating granulomas in multiple organs, including the lung, skin, eye, liver, and heart (1). Although muscular involvement is relatively common, isolated skeletal muscular sarcoidosis is reported to be rare (2). Both cardiac troponin T (cTnT) and I (cTnI) are specific markers of cardiac injury (3). Recent papers, however, have demonstrated that cTnT is induced in diseased skeletal muscle with Becker muscular dystrophy, Duchenne muscular dystrophy, Friedreich’s ataxia, inclusion body myositis, limb girdle muscular dystrophy, myasthenia gravis and myotonic dystrophy (4, 5). In the present paper, we report a case of sarcoidosis with the induction of cTnT in the diseased skeletal muscle without any involvement of the heart. Because several samplings of serum cTnI were within the normal ranges in this case, the concomitant measurement of cTnT and cTnI might therefore be useful to diagnose skeletal muscular sarcoidosis biochemically in such cases.

Case Report

A 49-year-old female was referred to our hospital due to high levels of serum creatine kinase (CK) (2,605 IU/L; normal range: 41-153). She had been treated with ferric medicine for anemia due to uterine fibroids. She had no episodes of chest discomfort, and her electrocardiogram and echocardiogram findings revealed no abnormalities. Although her serum levels of cTnT were elevated (0.342 ng/mL; normal range <0.014), as confirmed by two independent assays, the CK-MB and cTnI levels were within the normal limits without any renal dysfunction, hypothyroidism or other specific findings. Computed tomography and magnetic resonance imaging showed no evidence of either coronary arterial lesions, myocardial injury or skeletal muscle injury.

Twenty weeks after her first visit, she gradually began to...
feel difficulty in walking upstairs. Her manual muscle test (MMT) score was 4/5 in the proximal limb muscles at this time. An electromyogram on her right bicep showed low voltage and short duration motor unit potentials. Therefore, a biopsy of her bicep was performed and revealed non-casaeating epithelioid granuloma (Fig. 1A), suggesting sarcoidosis. Immunohistochemical staining for cTnT was positive in the degenerated skeletal muscle (Fig. 1B and D). No evidence of lung, eye or skin lesions was observed by routine physical and radiographic examinations. Contrast cardiac MRI showed no abnormality in the heart. Whole body positron emission tomography using fluorodeoxyglucose (FDG-PET) detected an uptake only in the gastrocnemius muscles and no abnormal uptake in the heart or any other organs. The serum levels of soluble interleukin-2 receptors were slightly elevated, while the angiotensin-converting enzyme (ACE) activity and lysozyme levels were within the normal limits. Because the patient exhibited symptoms of weakened skeletal muscle and abnormal histological findings, after excluding other types of skeletal muscle diseases, she was finally diagnosed with isolated skeletal muscular sarcoidosis, chronic myopathy type.

Her muscular weakness was accompanied by progressively increasing CK (5,372 IU/L) and cTnT (0.45 ng/mL; Roche, Mannheim, Germany) levels. Therefore, steroid pulse therapy (intravenous methylprednisolone 1,000 mg/day for 3 days) was administered twice, followed by oral prednisolone (40 mg/day) two weeks later with gradual tapering. Her muscular symptoms subsequently improved, and the CK (131 IU/L) and cTnT (0.026 ng/mL; Roche) levels also normalized (Fig. 2).

Discussion

In the present case, the diagnosis of sarcoidosis was histologically established based on the presence of noncaseating granulomas in the biopsied skeletal muscle. No evidence of any cardiac involvement was demonstrated by electrocardiogram, echocardiography, cardiac CT, cardiac contrast MRI, or FDG-PET examinations. Because further examination revealed no sarcoidosis lesions in the lung, skin, eye or liver, the patient was diagnosed with isolated skeletal muscular sarcoidosis.

The elevation of the serum cTnT levels was confirmed by two independent assays in the present patient. The value of cTnT was 0.257 IU/L according to an electrochemilumines-
Figure 2. Time course of creatine kinase (CK) and cardiac troponin T (cTnT) in response to steroid therapy.

cTnT might therefore be useful to discriminate cardiac and muscular diseases biochemically.

The authors state that they have no Conflict of Interest (COI).

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