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Case Report

# Acute myocarditis in bodybuilder from coxsackievirus and thyrotoxicosis

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ABSTRACT

A 25-year-old male with no past medical history presented with 1 day of chest pain. The patient had exercised with high intensity for a bodybuilding competition. He had fever, malaise, sore throat, and cough 1 week before presentation. He was tachycardic and tachypneic. Cardiac examination was unremarkable. Electrocardiogram showed diffuse ST segment elevation. Laboratory results showed leukocytosis, creatinine kinase 3078 unit/L, and troponin I 78.06 ng/mL. Coronary angiography revealed no occlusion. Echocardiogram showed ejection fraction of 45% with global hypokinesis. The next day, the patient became dyspneic, hypoxic, and hypotensive. Chest X-ray showed pulmonary edema requiring intubation for respiratory failure. Inotropic support and intra-aortic balloon pump were started. A viral panel was ordered and antibody titer of coxsackievirus B type 4 was ≥1:640. On obtaining further history, it was found that he took liothyronine 75 mcg daily for 3 weeks. Thyroid-stimulating hormone was 0.015 U/mL and free T3 was 4.4 ng/mL. Burch–Wartofsky score was 75. Methimazole and hydrocortisone were started. Cardiac magnetic resonance imaging showed diffuse myocardial inflammation and edema. There was multifocal dense epicardial and midmyocardial necrosis in all segments. The patient was discharged on metoprolol and enalapril. The patient was instructed to refrain from supplements.

<**Learning objective:** Exogenous thyroid hormone abuse may be an unusual cause of acute myocarditis in young healthy individuals. Physicians should emphasize to athletes to avoid overtraining and to minimize exposure to infection. Athletes with a clinical diagnosis of viral myocarditis should be temporarily excluded from competitive physical activity. Physicians should check whether athletes' immunizations are up-to-date and advise athletes against the use of thyroid hormone.>



## Introduction

Athletes experience significant intrinsic and extrinsic pressures motivating them to set high performance outcomes, especially before competitions. Athletes often turn to several dietary supplements to increase strength and muscle mass or to decrease body weight and fat. However, these supplements may exert adverse effects on various organs and tissues, including the cardiovascular system. Overtraining athletes have increased susceptibility to viral infection and exercise in early phase of myocarditis enhances viral replication in myocardium [1,2]. We report a case of thyroid hormone-associated acute myocarditis complicated by coxsackievirus infection.

## Case report

A 25-year-old male with no past medical history presented with one day of chest pain. The patient had exercised with high intensity for a bodybuilding competition. He had fever, malaise, sore throat, and cough 1 week before presentation but he continued rigorous daily exercise. He was tachycardic and tachypneic. Cardiac examination was unremarkable. Electrocardiography showed diffuse ST segment elevation (Fig. 1). Laboratory results showed

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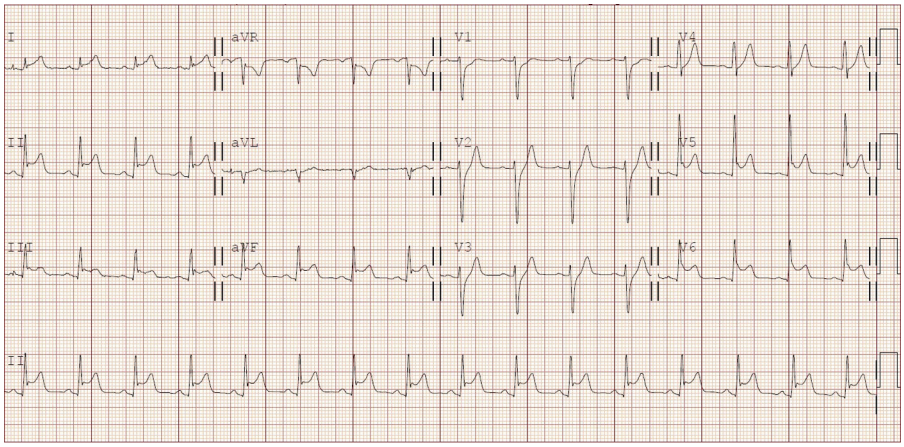


**Fig. 1.** Electrocardiogram on day 1 showing diffuse ST segment elevation.

leukocytosis, creatinine kinase 3078 unit/L, and troponin I 78.06 ng/mL. Coronary angiography revealed no occlusion (Fig. 2). Echocardiogram showed left ventricular ejection fraction (LVEF) of 45% with global hypokinesis. The next day, the patient became dyspneic, hypoxic, and hypotensive. Chest X-ray showed pulmonary edema requiring intubation for respiratory failure. Inotropic support and intra-aortic balloon pump were started. A viral panel was ordered and antibody titer of coxsackievirus B type 4 was $\geq$1:640. On obtaining further history, it was found that he took liothyronine 75 mcg daily for 3 weeks. Thyroid-stimulating hormone was 0.015 U/mL and free T3 was 4.4 ng/mL. Burch–Wartofsky score was 75 and a diagnosis of thyrotoxicosis was made [3]. Methimazole and hydrocortisone were started. Cardiac magnetic resonance imaging (MRI) showed diffuse myocardial inflammation and edema. There was multifocal dense epicardial and mid-myocardial necrosis in all segments (Fig. 3). The patient was discharged on metoprolol succinate 50 mg once daily and enalapril 5 mg once daily. The patient was instructed to refrain from supplements. The patient resumed gentle exercise two months after discharge and tolerated it well. Follow-up cardiac MRI two months after discharge showed residual myocardial scar with LVEF 55%.

## Discussion

Thyrotoxicosis, coxsackievirus infection, and overtraining may have caused this patient's acute myocarditis. About 6% of thyrotoxic patients develop heart failure and less than 1% develop dilated cardiomyopathy with impaired left ventricular systolic dysfunction, due to a tachycardia-mediated mechanism leading to an increased level of cytosolic calcium during diastole with reduced contractility of the ventricle and diastolic dysfunction [4].

Bodybuilders often use thyroid hormones to increase metabolic rate. However, accelerated oxygen consumption by thyroid hormone leads to enhanced generation of reactive oxygen species (ROS) in myocardium, with a higher consumption of antioxidants and inactivation of antioxidant enzymes, thus inducing oxidative stress [5]. These cellular events play an important role in the development and progression of myocardial injury and remodeling [5]. Thus, hyperthyroidism can trigger acute decompensated heart failure or complicate pre-existing cardiac disease because of increased myocardial oxygen demand and increased contractility and heart rate [6]. Treatment with beta-blockers reduces ROS by activation of Jun kinases and stimulation of apoptosis in addition to their antioxidant properties [7,8].

Our patient had neutralizing antibody titer of coxsackievirus B type 4 $\geq$1:640. A fourfold increase in titer in acute and convalescent serum, or a single high titer of $>$1:320 confirms an acute or recent infection [9]. Overtraining athletes have increased susceptibility to upper respiratory infections via a depressant effect on T cell, interleukin, and natural killer cell systems [1]. Research has confirmed earlier clinical and animal studies in showing that either a single bout of exhausting exercise or persistent overtraining can increase susceptibility to upper respiratory and other viral infections, although resistance to bacterial infections is apparently unaltered [1]. In contrast, moderate training enhances immune defenses [1]. Murine experiments showed that exercise during early phase of myocarditis may increase viral replication rate in myocytes, resulting in enhanced cytolysis and immune response leading to

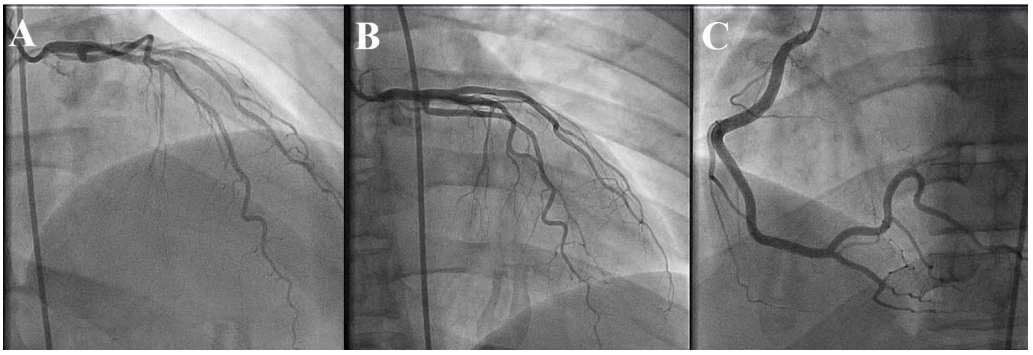


**Fig. 2.** Coronary angiography with normal coronary vessels. (A) Left anterior descending artery in right anterior oblique (RAO) cranial view; (B) left circumflex artery in RAO caudal view; (C) right coronary artery in left anterior oblique cranial view.

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