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Thyrotoxic-Related Myocarditis: A Case Report

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Introduction

The manifestations of hyperthyroidism-related myocardial damage are multitudinous, including arrhythmia, dilated cardiomyopathy, valvular diseases, and even cardiogenic shock. Acute myocarditis induced by thyrotoxicosis had been reported in a few studies. However, attention on its prevalence and underlying mechanisms is sorely lacking. Its long-term harm is often ignored, and it may eventually develop into dilated cardiomyopathy and heart failure.

Case Report

We report the case of a 34-year-old male with a history of smoking and hyperthyroidism receiving only propranolol alongside the discontinuation of his anti-thyroid drugs. Consulting his primary care physician for acute constrictive chest pain radiating to his left arm. A 12 lead ECG was performed demonstrating a non-persistent anterior ST segment elevation associated with a very high ultra-sensitive troponin. The patient was respiratory and hemodynamically stable, so the diagnosis of acute coronary syndrome was retained, and the patient was transported to the cardiac catheterization room where the coronagraphy showed no significant lesions and the rate of the troponin was declining. A meticulous interrogation was made without any notion of flu syndrome; the cardiac transthoracic ultrasound showed a normal left ventricular function with homogeneous kinetic and dilated right cavity and a dry pericard. The diagnosis was revised, and acute myocarditis was confirmed by cardiac MRI: Localized inflammatory edema (enhanced signals under T2 mapping), myocardial necrosis (enhanced signals under T1 late gadolinium enhancement) in the inferior epicardial wall, with a preserved left ventricular systolic function (68%). The patient was then given an optimized treatment of heart failure with a favorable evolution.

Discussion

However, the available evidence regarding the occurrence of myocarditis induced by thyrotoxicosis appears to be limited. Only a few scattered reports and a small number of studies have investigated the connection between hyperthyroidism and myocarditis [1]. Based on the positive outcome observed in our patient's case, we strongly believe that it is crucial and valuable to extensively explore this condition and consolidate the existing research.

In hyperthyroidism-induced heart dysfunction, inflammatory cell infiltration and the release of inflammatory factors contribute o myocyte necrosis [2]. Different types of infiltrated lymphocytes, including CD4+ and CD8+ T cells, B cells, and natural killer cells, play a role in the progression of myocarditis [3]. Imbalances in T helper cell subtypes and regulatory T cells also contribute to the pathological process. Thyroxine alteration affects T lymphocyte recruitment and activity, promoting lymphocyte infiltration into affected organs [4]. Auto-antibodies targeting cardiomyocytes are implicated in the inflammatory disorders observed in autoimmune myocarditis. Autoantibodies against self-myosin may be produced due to exposure of myosin antigens during T cell selection [5].

Innate immune cells, such as macrophages, killer cells, and dendritic cells, also play a role in autoimmune myocarditis. Inflammatory monocytes are recruited early in response to cardiac injury, and their differentiation into pro-inflammatory macrophages is influenced by Th1 cells [6]. Excessive thyroxine levels enhance the proliferation and pro-inflammatory function of various cells in the innate immune system [7].

Conclusions

Acute myocarditis and hyperthyroidism are common diseases that often present in young healthy patients. Autoimmunity is central to the pathogenesis of both. Physicians should consider screening for myocarditis in patients with hyperthyroidism and persistent cardiac symptoms.

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