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Multiple Sclerosis with Idiopathic Dilated Cardiomyopathy: A Case Report

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Introduction

Multiple sclerosis and idiopathic dilated cardiomyopathy are two conditions in which an autoimmune process is implicated in the pathogenesis. There is evidence to support clustering of autoimmune diseases in patients with multiple sclerosis and their families [1, 2]. To our knowledge, this is the first report of idiopathic dilated cardiomyopathy occurring in a patient with multiple sclerosis.

Clinical History

A 48-year-old Caucasian woman was diagnosed with multiple sclerosis in 1988. She had an episode of left optic neuritis in 1984 which was treated with a course of oral prednisolone and resolved over a period of 3 months. In 1988, she developed right-sided paraesthesia and weakness. This was followed several months later by deterioration in her mobility with an ataxic gait and impaired co-ordination in the right upper limb. The diagnosis of multiple sclerosis was supported by the presence of oligoclonal immunoglobulin G bands in the cerebrospinal fluid but not the serum and by prolonged latencies of the visual evoked responses. A magnetic resonance imaging scan of her brain showed multiple cerebral white matter lesions including periventricular lesions, typical of multiple sclerosis. The patient met the criteria of McDonald et al. [3] for a diagnosis of multiple sclerosis.

After 1988, the patient had multiple exacerbations requiring high-dose intravenous corticosteroid therapy. She was treated with interferon β for 5 months in 2000, but this was ceased because of sinusitis and depression. She was also treated with glatiramer acetate for 2 years; however, she ceased this in 2003 because she disliked injections. In August 2004, she was able to walk 200 m without aid or rest and had a Kurtzke Expanded Disability Status Scale score of 5.0 [4].

In 2002, at the age of 62 years, she developed fatigue, a dry cough, exertional dyspnoea and ankle swelling. Examination of the cardiovascular system revealed pedal oedema; however, there were no other signs of congestive cardiac failure and no heart murmurs. Her chest X-ray showed an enlarged cardiothoracic ratio, and an electrocardiogram demonstrated a left bundle branch block. A transthoracic echocardiogram revealed a mildly dilated left ventricle with severe global left ventricular dysfunction and an ejection fraction of 25% (normal >50%). Coronary angiography revealed normal coronary arteries and confirmed the presence of severe dilated global cardiac hypokinesia. A diagnosis of dilated cardiomyopathy was made and she was commenced on carvedilol, candesartan, frusemide and aspirin.

Her past history included a 15-year period of smoking approximately 20 cigarettes per day. She ceased smoking at the age of 40 years. During her only pregnancy, she was hypertensive and developed pre-eclampsia at 36 weeks of gestation. Following the pregnancy, her blood pressure became normal and remained so. She had no history of elevated cholesterol or diabetes. Her family history included heart disease in both parents, who had onset of disease in their seventies. There was no family history of autoimmune disease.

Fasting lipids revealed a total cholesterol of 5.1 mmol/l (reference range 3.9–5.5 mmol/l); triglycerides 1.8 mmol/l (0.6– 2.0 mmol/l); high-density lipoprotein 1.3 mmol/l (1.1–1.9 mmol/l)

and low-density lipoprotein 3.0 mmol/l (0–4.0 mmol/l). Iron studies, serum protein electrophoresis, serum immunoglobulins and cardiac enzymes were normal.

Thyroid function tests were normal; however, antithyroid peroxidase antibodies and antithyroglobulin antibodies were elevated to 445 IU/ml (normal <50 IU/ml) and 135 IU/ml (normal <100 IU/ml), respectively. She was positive for antismooth muscle V antibody with a titre of 160. Her antinuclear antibody was elevated with a titre of 40 in a speckled pattern.

Her human leukocyte antigen (HLA) type was DRB1*1303,1501 – DQA1*0102,0501 – DQB1*0301,0602. Thus, she carried the HLA haplotype DRB1*1501 – DQA1*0102 – DQB1*0602 that occurs most commonly in Caucasians with multiple sclerosis.

Discussion

Multiple sclerosis is a chronic inflammatory demyelinating disease of the central nervous system which most commonly presents in early adulthood. There is mounting evidence for an autoimmune pathogenesis of multiple sclerosis and for the role of genetic factors [5]. There is also evidence for an increased occurrence of other autoimmune diseases in patients with multiple sclerosis and their first-degree relatives [1, 2].

Dilated cardiomyopathy is a disease of the myocardium resulting in cardiac enlargement and impaired systolic function. Typically it affects adults between the ages of 20 and 60; however, it can occur at any age. The presentation can be with symptoms of cardiac failure, complications of arrhythmia and conduction disturbances or as an incidental finding. The causes are varied and include ischaemia, infection, drugs and toxins, familial and inherited disorders and arrhythmia. However, in a significant proportion, no specific aetiology can be identified. This group is labelled idiopathic [6]. Autoimmunity has been proposed as a mechanism in the pathogenesis of idiopathic dilated cardiomyopathy [7].

Myocarditis refers to inflammation of the cardiac muscle and can result in dilated cardiomyopathy. It is often asymptomatic and tends to affect younger people. There are many postulated causes including viral infection, toxins and systemic autoimmune disease. The development of autoimmunity to cardiac muscle in response to infectious or other stimuli has been proposed as a mechanism by which myocarditis leads to dilated cardiomyopathy. Furthermore, it is possible that this progression of disease occurs in a subgroup of people with an appropriate genetic background [8]. As with multiple sclerosis, there is an increased occurrence of other autoimmune diseases in the first-degree relatives of patients with dilated cardiomyopathy [9].

Our patient clearly fulfils the criteria for a diagnosis of multiple sclerosis. She also developed a dilated cardiomyopathy for which there was no clearly attributable cause. Glatiramer acetate has not been associated with cardiomyopathy to the best of our knowledge. Cardiomyopathy is a rare complication of interferon (mainly interferon α therapy, and usually improves after cessation of this therapy [10].

We could find no previous report of the concurrence of dilated cardiomyopathy and multiple sclerosis, but there are reports of myocarditis and dilated cardiomyopathy associated with autoimmune thyroid disease [11–13]. Although our patient had normal thyroid function tests, the elevated antithyroid antibodies suggest the presence of subclinical autoimmune thyroiditis. Furthermore, there is additional evidence of autoimmunity in our patient as shown by a weakly positive antinuclear antibody titre and the presence of antismooth muscle V antibodies. This is consistent with an underlying genetic predisposition to autoimmunity.

In conclusion, we have reported a patient with multiple sclerosis and idiopathic dilated cardiomyopathy. We suggest that these are clinically manifest autoimmune conditions in an individual with a genetic susceptibility to autoimmunity.

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