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Sick sinus syndrome and orthostatic hypotension in Parkinson's disease

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Abstract

We present a case of Parkinson's disease patient whose initial symptoms were sick sinus syndrome and orthostatic hypotension. Our case illustrates difficulties in distinguishing syncope of primary cardiac or neurological origin and highlights the importance of a diagnostic workup including neurological examination.

Key words: Parkinson's disease, sick sinus, orthostatic hypotension.

Introduction

Autonomic dysfunction can be prominent in Parkinson's disease (PD) and can be the presenting symptom of the disease [1]. There has been only one case report of sick sinus syndrome caused by autonomic failure in a patient with PD [2].
We report a PD patient who presented with sick sinus syndrome and orthostatic hypotension as leading symptoms.

Case report

A 60-year-old patient was admitted to cardiology department complaining of shortness of breath and dizziness. He had intermittent fainting episodes, especially while standing for longer periods of time, several times daily for the last year. He underwent a thorough diagnostic work up. Electrocardiogram (ECG) revealed bradycardia of 42 beats per minute with PR interval of 0.18 second. 24-hour ambulatory ECG recording demonstrated sinus rhythm with ventricular frequency from 33-77 per minute, with an average of 47 per minute. Echocardiography showed mild left ventricular hypertrophy with an ejection fraction of 78% and preserved systolic and diastolic function. He had degenerative changes of mitral valve with no mitral regurgitation. Coronary angiography was normal.
Chronotropic incompetence was demonstrated by exercise ECG with his maximum exercise achieved heart rate of 63 % of his maximum predicted heart rate.
Sick sinus syndrome was diagnosed and permanent cardiac pacemaker DDD Sigma SD 303 “fixed rate” was implanted.
Six months later he was readmitted to cardiology department because of persistence of fainting episodes. Tilt-table test was performed revealing orthostatic hypotension. His blood pressure (BP) of 132/80 mmHg in the supine position declined to 106/70 mmHg on passive tilting. 24-h ambulatory BP monitor disclosed systolic pressure ranging from 74 to 152 mmHg and diastolic pressure from 42 to 100 mmHg. His cardiac pacemaker rate modulation was adjusted and he was prescribed amiodarone.
Another six months later, he was admitted to our neurology department complaining of frequent fainting episodes, walking difficulties and urinary retention.

Neurological examination revealed bradykinesia and rigidity of all extremities, tremor at rest of the left fist, hypomimia, hypokinetic speech and severe postural instability. Family history disclosed that his mother's brother had Parkinson's disease. Laboratory tests were normal, including complete blood count and thyroid function tests. CT scan showed mild cortical atrophy. MRI was contraindicated because of cardiac pacemaker. 6-[^{18}F]fluorodopa uptake on positron emission tomography was reduced in corpus striatum bilaterally, more on the right side. Metaiodobenzylguanidine (MIBG) myocardial scintigraphy demonstrated decreased myocardial uptake of MIBG with a decreased heart-to-mediastinum count (HM) ratio (less then 1.2) (Fig. 1).

The patient was diagnosed with Parkinson's disease and was treated with levodopa/benserazide. There was a substantial improvement of motor symptoms which was registered using Unified Parkinson disease rating scale (UPDRS). UPDRS motor examination section score was 48 points prior to treatment and a month after introduction of levodopa the score was 39. Rigidity of extremities and bradykinesia were greatly reduced but postural instability still limited the patient in daily activities. He was administered pressor drug ethylnorphenylephrine and volume expander fludrocortisone which led to a substantial improvement of orthostatic hypotension leaving only occasional dizziness on standing up from a supine position. No further autonomic dysfunction was noted in one-year follow-up period and motor symptoms were under control with the given treatment.

Discussion

Autonomic failure can lead to various symptoms such as orthostatic hypotension, sphincter dysfunction, erectile dysfunction, and disorders of sweating. Almost half of PD patients have orthostatic hypotension. Symptoms include light-headedness and syncope upon rising from a supine position or standing still for prolonged periods. Our patient had a BP decrease of 26 mmHg systolic and 10 mmHg diastolic on passive upright tilting confirming orthostatic hypotension.

Sick sinus syndrome as a result of autonomic failure has been described in only two case reports [2]. Yamamoto et al described the first PD patient who was diagnosed with sick sinus syndrome associated with autonomic failure [2].

Orthostatic hypotension as well as sick sinus syndrome result from sympathetic denervation of the cardiovascular target organs [2, 3]. Sympathetic hypoactivity was demonstrated in our patient by decreased myocardial uptake of MIBG and low HM. The reported patient had a cardiac pacemaker implanted because his syncopes were identified as being of cardiac origin supported by his low heart rate and prolonged PR intervals. Nevertheless, he continued to have syncopes.

When first presented to a neurologist his examination revealed four cardinal features of PD: bradikinesia, rigor, tremor at rest and postural instability [1]. Multiple system atrophy (MSA) was suspected because of prominent autonomic failure. However the results of tilt table testing did not show a significant enough decrease of BP that is required for the diagnosis of probable MSA (30 mm Hg systolic or 15 mmHg diastolic). Since the patient was unable to perform brain MRI due to implanted cardiac pacemaker we were unable to make a differential diagnosis based on these findings. The results of MIBG myocardial scintigraphy showed low HM which is characteristic for PD as opposed to MSA patient who tend to have normal HM ratio [4]. Positive family history for PD is another non-supporting feature for the diagnosis of MSA.

Cardiovascular autonomic dysfunction is a common symptom in patients with PD and heart ischemia causes significant mortality in this population [5]. Thus, a substantial number of PD patients will be examined by a cardiologist and a part of them with cardiovascular difficulties as their leading symptom. Our case illustrates the difficulties in distinguishing syncope of primary cardiac or neurological origin and highlights the importance of a full diagnostic workup including neurological examination, even in the absence of more suggestive neurological symptoms.

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Fig.1 Metaiodobenzylguanidine myocardial scintigraphy demonstrating decreased myocardial uptake of MIBG with a decreased heart-to-mediastinum count ratio.