Case Report

Sudden failure of ventricular pacing and recovery in a patient with cardiac sarcoidosis

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Article info

Article history:
Received 14 December 2016
Received in revised form 16 May 2017
Accepted 13 June 2017
Available online 26 July 2017

Keywords:
Sarcoidosis
Pacing failure
Steroid therapy
Pacing threshold

Abstract

A 76-year-old woman with sarcoidosis who had an implantable pacemaker for complete atrioventricular block was admitted with syncope. Electrocardiogram revealed ventricular pacing failure, and a marked rise in the ventricular pacing threshold. 18F-Fluorodeoxyglucose positron emission tomography (FDG-PET) indicated increased uptake of FDG in the ventricular septum. Three days after steroid therapy, the ventricular pacing threshold reverted to normal, and FDG-PET showed decreased FDG uptake in the ventricular septum. In this case report, we demonstrate that a sudden deterioration in the ventricular pacing threshold due to worsening cardiac sarcoidosis can be reversed with early steroid therapy.

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1. Introduction

Cardiac sarcoidosis is an inflammatory heart disease and may present with electrical abnormalities including atrioventricular (AV) block and ventricular arrhythmias [1]. Although corticosteroids have been recommended as the first-line treatment for inflammation, their efficacy remains unclear.

In the present report, we describe a case of cardiac sarcoidosis with an implanted pacemaker for complete AV block in which we observed a sudden reversal in pacing failure immediately after the administration of prednisolone, an oral steroid.

2. Case report

A 76-year-old woman was hospitalized because of syncope. She had received a dual-chamber pacemaker implant for complete AV block at the age of 73 years. The initial ventricular pacing threshold was 0.5 V at a pulse width of 0.4 ms. Whole-body computed tomography showed enlargement of the lymph nodes in multiple organs, including the bilateral hilar lymph nodes. Serum levels of soluble interleukin-2 receptor were elevated, and histological examination of an axillary lymph node biopsy specimen revealed noncaseating granulomas. The patient was diagnosed with cardiac sarcoidosis with associated complete AV block at the age of 73 years, and was started on steroid therapy. However, after 3 months of treatment, she discontinued steroid treatment owing to the potential worsening of her diabetes.

A 12-lead electrocardiogram on admission revealed complete AV block and ventricular pacing failure. Fig. 1 shows the pacemaker interrogation summary. The ventricular pacing threshold showed a marked rise in voltage (4.8 V at a pulse width of 0.4 ms) (Fig. 1, asterisk). The pacemaker lead impedence and the position remained unchanged. The patient did not have any clinical conditions such as hyperkalemia or heart failure that could induce increases in the ventricular pacing threshold. 18F-Fluorodeoxyglucose positron emission tomography (FDG-PET) demonstrated abnormal FDG accumulation in the ventricular septum. We suspected worsening of cardiac sarcoidosis and initiated oral prednisolone therapy at 30 mg/day. After 3 days of steroid therapy, we observed improvements in the ventricular pacing and normalization of the threshold (0.8 V at a pulse width of 0.4 ms) (Fig. 1, dagger). After 132 days of steroid therapy, FDG accumulation almost disappeared (Fig. 2). Prednisolone dosage was tapered to 15 mg/day by reducing the daily dosage by 5 mg once every fortnight over a 6-week period, without increases in the ventricular pacing threshold. The patient was being treated with 10 mg of oral prednisolone at the time of writing this manuscript. She had been

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http://dx.doi.org/10.1016/j.joa.2017.06.004
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followed up for 1 year, during which no recurrence of ventricular pacing failure was noted.

3. Discussion

This report demonstrates that cardiac sarcoidosis associated with ventricular pacing failure can be successfully treated with steroid therapy. Using FDG-PET images, we observed changes in the inflammatory lesion before and after the administration of prednisolone. Similarly, Takasugi et al. reported marked deterioration in the ventricular pacing threshold with a sudden reversal in the threshold after steroid treatment [2].

Immunosuppressive therapy, such as the use of steroids, has been recommended as the first-line treatment for cardiac sarcoidosis. However, the effectiveness of steroids for the treatment of cardiac sarcoidosis has not been fully elucidated. Although steroid therapy was reported to be less effective in patients with advanced left ventricular (LV) dysfunction (≤ 35% LV ejection fraction) [3], echocardiographic observations showed that steroid therapy could reduce LV volume and improve LV ejection fraction in the early or middle stages, but not in the late stage (≤ 30% LV ejection fraction), of cardiac sarcoidosis [4]. Early steroid therapy was not effective in approximately half of the patients with complete AV block, in whom pacemaker implantation was required [5]; it is possible that active inflammation induced ventricular pacing failure after pacemaker implantation. This case suggests that the complications associated with ventricular pacing failure may be reversed with early steroid therapy. It also highlights the significance of continuous steroid therapy in cardiac sarcoidosis.

We performed FDG-PET to assess the severity of cardiac sarcoidosis, and observed high FDG uptake with a marked decrease in the anterior-to-septal LV wall uptake of FDG before and after steroid therapy, together with a reversal in ventricular pacemaker failure. Therefore, we propose that steroid therapy may be effective when initiated during the early stage of inflammation in cardiac sarcoidosis. As FDG-PET was reported to be more sensitive in assessing for inflammation in cardiac sarcoidosis [6], we did not perform Gallium scintigraphy.

In conclusion, we encountered a patient with cardiac sarcoidosis associated with ventricular pacing failure, who was successfully treated with steroid therapy. Early administration of immunosuppressive agents such as corticosteroids may be effective in patients with cardiac sarcoidosis who experience pacing failure after pacemaker implantation.

Conflict of interest

All authors declare that they have no conflict of interest related to this study.

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