Journal of Electrocardiology 51 (2018) 658–662

Contents lists available at ScienceDirect

Journal of Electrocardiology

journal homepage: www.jecgonline.com

Serial observation of electrocardiographic responses to corticosteroid therapy in a patient with right ventricular-predominant cardiac sarcoidosis

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ARTICLE INFO

Keywords:
Cardiac sarcoidosis
Corticosteroid therapy
Electrocardiogram
Right ventricle

ABSTRACT

Predominant or isolated right ventricular involvement in cardiac sarcoidosis is uncommon, but should always be considered in a case of right ventricular hypertrophy combined with ventricular arrhythmia and/or conduction disturbance. Although improvement in right ventricular hypertrophy and atrioventricular conduction disturbance following corticosteroid therapy has been reported, the detailed serial electrocardiographic responses during corticosteroid therapy, as well as temporal changes in the electrocardiographic, biochemical, and morphological responses, have not been reported. We describe the clinical course and supportive imaging findings of reversible right ventricular hypertrophy and cardiac conduction disturbance in a case of right ventricular-predominant cardiac sarcoidosis.

Introduction

Predominant or isolated involvement of the right ventricle in cardiac sarcoidosis is uncommon, but should always be considered in a case of right ventricular hypertrophy combined with ventricular arrhythmia and/or conduction disturbance [1–4]. Although corticosteroid therapy was reportedly effective for right ventricular-predominant cardiac sarcoidosis [3–5], the detailed serial electrocardiographic responses during corticosteroid therapy have not been reported. We describe the clinical course and supportive imaging findings of reversible right ventricular hypertrophy and cardiac conduction disturbance in a case of right ventricular-predominant cardiac sarcoidosis.

Case report

A 54-year-old man without a history of cardiovascular disease was admitted to our hospital for the management of sustained ventricular tachycardia possibly originating from the inferior right ventricular outflow tract or mid-anterior right ventricle (Fig. 1). He was hemodynamically stable, and his only symptom was palpitation with sudden onset and cessation. The electrocardiogram following cardioversion showed sinus rhythm with complete right bundle branch block and first-degree atrioventricular block. The right bundle branch block had first been noted on a routine annual medical checkup 2 months before admission. At the end of the QRS-wave, a tiny delayed deflection was observed in the inferior leads (Fig. 2A). Clinical examination and chest radiography showed bilateral hilar lymphadenopathy without cardiomegaly. Laboratory tests showed an elevated high-sensitivity troponin I level of 0.05 ng/mL and a C-reactive protein level of 1.72 mg/dL. Transthoracic echocardiography showed right ventricular hypertrophy. Cardiac magnetic resonance imaging confirmed right ventricular hypertrophy with prominent late gadolinium enhancement in the right ventricular free wall and trabeculations and basal inferoseptal-inferior left ventricle (Fig. 3A). ^{18}F-fluorodeoxyglucose positron emission tomography/computed tomography showed an intense uptake in multiple mediastinal lymph nodes, the right ventricular free wall and trabeculations, and the basal inferoseptal-inferior left ventricle (Fig. 4A). Right ventricular-predominant cardiac sarcoidosis was diagnosed, and 30 mg of prednisolone daily was initiated. Fig. 5 shows temporal improvements in the troponin I level, PR interval, QRS width, and right ventricular free-wall thickness measured by transthoracic echocardiography during the course of treatment. The conduction

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https://doi.org/10.1016/j.jelectrocard.2018.04.008
0022-0736/

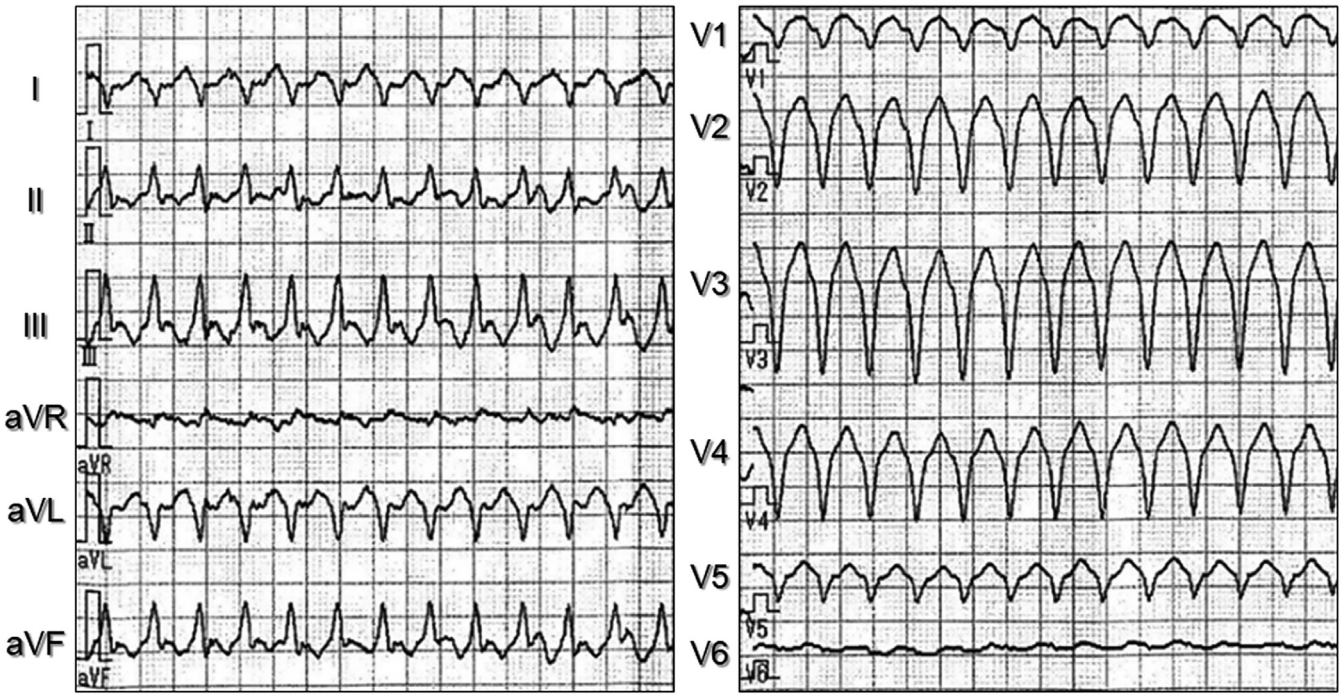

Fig. 1. Sustained ventricular tachycardia originating from the inferior right ventricular outflow tract or mid-anterior right ventricle. The R-waves in the inferior leads were relatively small, and no r-waves were observed in the precordial leads. The QS-waves were observed in leads V1-V5, which were most prominent in lead V3 rather than in lead V2. These findings indicate a more inferiorly and apically located focus/exit than that in typical ventricular tachycardias originating from the right ventricular outflow tract.

disturbance and right ventricular hypertrophy gradually improved over the course of a month, following a prompt improvement in the troponin I level. Although the electrocardiogram still demonstrated right bundle branch block, the QRS width was narrowed with an improvement in the late R-wave in lead V1 and disappearance of the tiny delayed deflection in the inferior leads (Fig. 2B). Bilateral hilar lymphadenopathy, as well as the right ventricular hypertrophy and late right ventricular gadolinium enhancement, improved (Fig. 3B). The left and right ventricular ejection fractions were 65.2% and 44.8%, respectively. Fluorodeoxyglucose uptake was absent (Fig. 4B). Subsequent

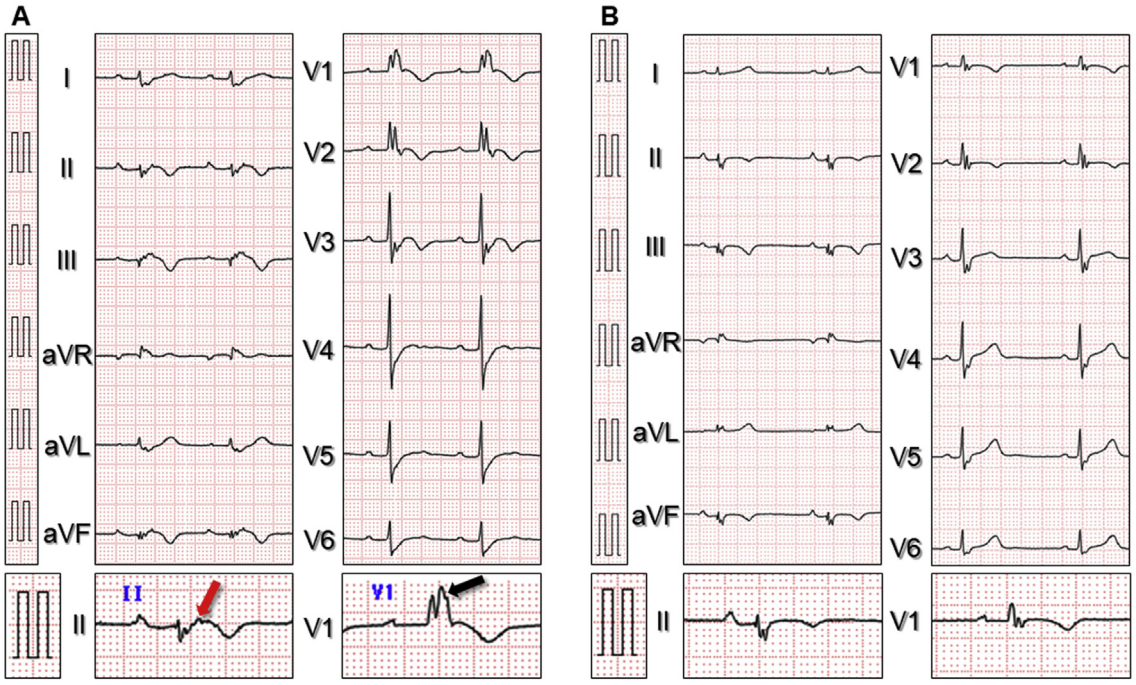

Fig. 2. Electrocardiogram before and after corticosteroid therapy. Sinus rhythm with complete right bundle branch block and first-degree atrioventricular block (PR interval, 288 ms) was observed (A). After corticosteroid therapy, the PR interval improved from 288 ms to 198 ms, as did the QRS width and morphology (B). There were improvements in the late R-wave in lead V1 (black arrow) and disappearance of the tiny delayed deflection in lead II (red arrow).