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Localized basal left ventricular dyssynchrony induced by manifest accessory pathway: Successful differentiation from cardiac involvement of sarcoidosis with administration of flecainide acetate☆

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ABSTRACT

We present a patient with non-cardiac sarcoidosis complicated with manifest ventricular preexcitation. Initially, cardiac involvement of sarcoidosis was suspected from the echocardiographic findings showing localized hypokinesia at the left ventricular basal inferior wall. We, however, considered that the hypokinesia was a preexcitation-induced mechanical dyssynchrony rather than cardiac sarcoidosis, because polarities of the delta-waves indicated a left ventricular inferior accessory pathway. Temporal administration of oral flecainide acetate eliminated the basal left ventricular motion abnormality. Accordingly, we could successfully differentiate the mechanism of hypokinesia. In this context, we could rule out cardiac sarcoidosis, and initiation of glucocorticoid therapy was reasonably withheld.

Introduction

In patients with non-cardiac sarcoidosis, careful and periodic monitoring of conduction disturbance as well as cardiac function using both electrocardiography and transthoracic echocardiography is required to detect early signs of cardiac involvement. Herein, we present a patient with non-cardiac sarcoidosis complicated with ventricular preexcitation. Temporal administration of oral flecainide acetate was useful to elucidate the etiology of basal left ventricular hypokinesia detected during routine echocardiographic monitoring of this patient.

Case report

A 32-year-old man with a diagnosis of pulmonary and ocular sarcoidosis was referred to our institution in order to investigate cardiac involvement. He had no cardiac symptoms including dyspnea and palpitation. Electrocardiography revealed a normal sinus rhythm with heart rate of 73 bpm. A manifest atrioventricular accessory pathway was suspected due to the apparent delta-wave with right bundle branch block QRS-wave morphology (Fig. 1). Polarities of the delta-waves were positive in leads I, aVL, and V1–V6 and negative in the inferior leads, indicating a left ventricular inferior accessory pathway. Laboratory data did not show elevation in the serum levels of high-sensitivity troponin I (<0.01 ng/mL), C-reactive protein (0.02 mg/dL), or angiotensin-converting enzyme (18.6 U/L). Transthoracic echocardiography (iE33, Philips, Philips Medical Systems, Andover, MA, USA) performed with a frame rate of 58 Hz detected a localized hypokinesia at the basal inferior wall of the left ventricle (Supplementary Movie 1). The left ventricular ejection fraction was maintained (50%). The local hypokinesia was also confirmed by cardiac magnetic resonance imaging. Late gadolinium enhancement, however, was not observed in any regions of the left ventricle. According to close frame by frame observation of the local hypokinesia detected by echocardiography and cine-magnetic resonance imaging, we considered that the hypokinesia was a dyssynchrony presumably caused by preexcitation of the basal inferior left ventricle via the accessory pathway rather than cardiac sarcoidosis. It is, however, clinically important to confirm this speculation by ruling out cardiac involvement of sarcoidosis. We therefore re-examined the transthoracic echocardiography. This time, 200 mg of daily oral flecainide acetate was administered 3 days before the study to block the antegrade conduction of the atrioventricular accessory pathway. Along with the disappearance of the delta-wave (Fig. 1), transthoracic echocardiography confirmed complete disappearance of initial local dyssynchrony (Supplementary Movie 2). The dyssynchrony, therefore, was diagnosed to

☆ Conflicts of interest: None.

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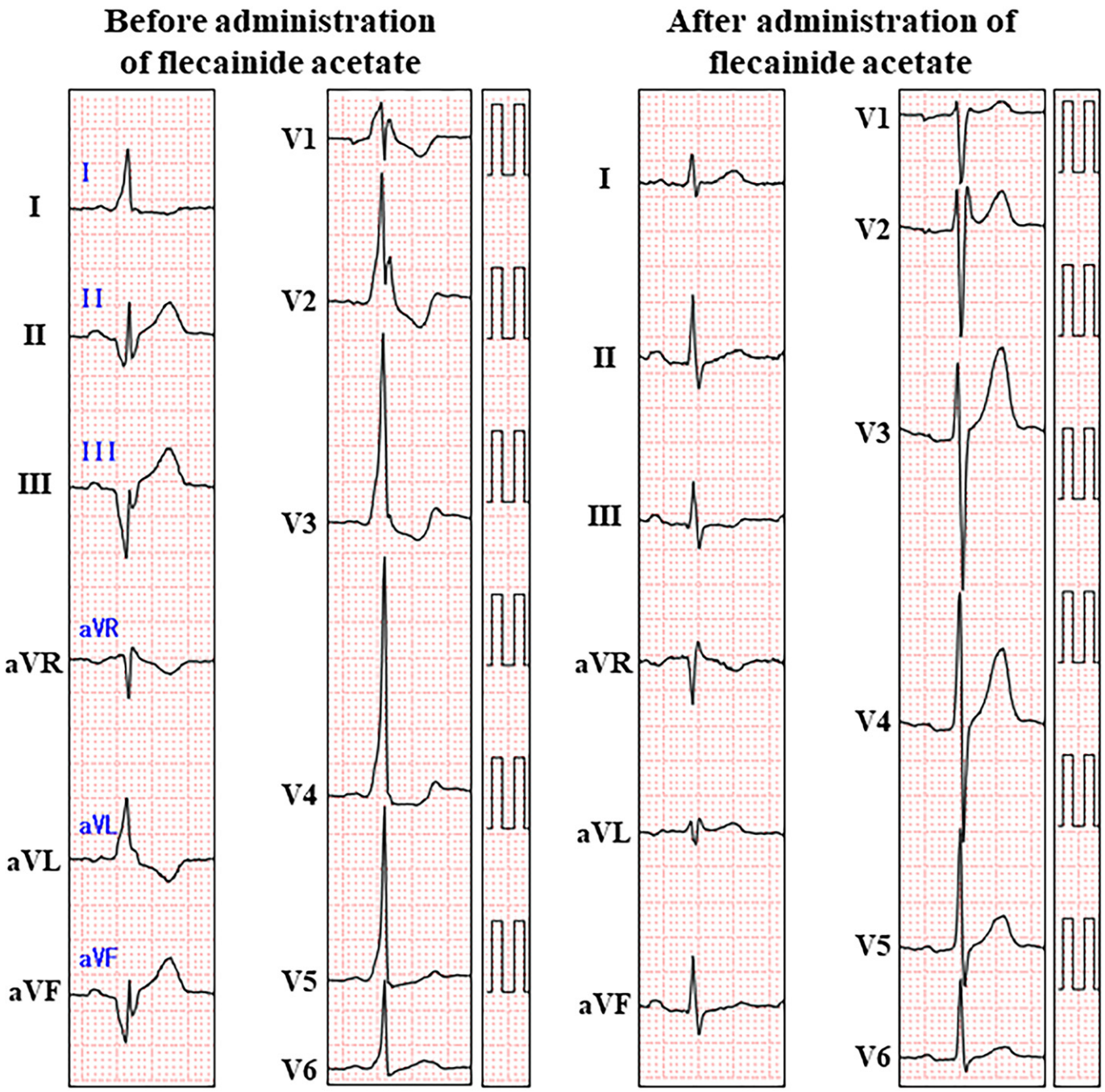

Fig. 1. Electrocardiography before and after administration of flecainide acetate.

be an accessory pathway-related preexcitation of the basal inferior left ventricle. In this context, we could rule out cardiac sarcoidosis, and initiation of glucocorticoid therapy was reasonably withheld. Flecainide acetate was discontinued after the second echocardiography. The patient was scheduled to visit our outpatient clinic to undergo annual echocardiographic follow-up.

Discussion

Sarcoidosis is a systemic inflammatory disease of unknown etiology, characterized by the presence of noncaseating granulomas. Specific diagnosis often requires the involvement in 2 or more organs. Cardiac involvement is not infrequent and is influenced by race. Over a quarter of Japanese sarcoidosis patients develop cardiac involvement, whereas only 5% of sarcoidosis patients in United States and Europe develop cardiac sarcoidosis. Cardiac involvement usually presents as either left or right ventricular dysfunction or cardiac arrhythmias, including bradycardia due to conduction disturbance and/or ventricular tachycardia. In cases of sarcoidosis, focal hypokinesia of the left or right ventricle can indicate the diagnosis of new onset cardiac sarcoidosis, in addition to the detection of cardiac arrhythmias [1]. Usually, cardiac involvement requires glucocorticoid administration, especially in patients with active inflammation. Accurate differential diagnosis of the hypokinesia, as detected in the present case, is important in order to avoid unnecessary glucocorticoid therapy.

Manifest ventricular preexcitation pattern electrocardiography is characterized by the delta-wave under the presence of a manifest accessory pathway between the atrium and ventricle. Manifest atrioventricular accessory pathways commonly induce preexcitation of the basal left ventricular myocardium located at the end of ventricular

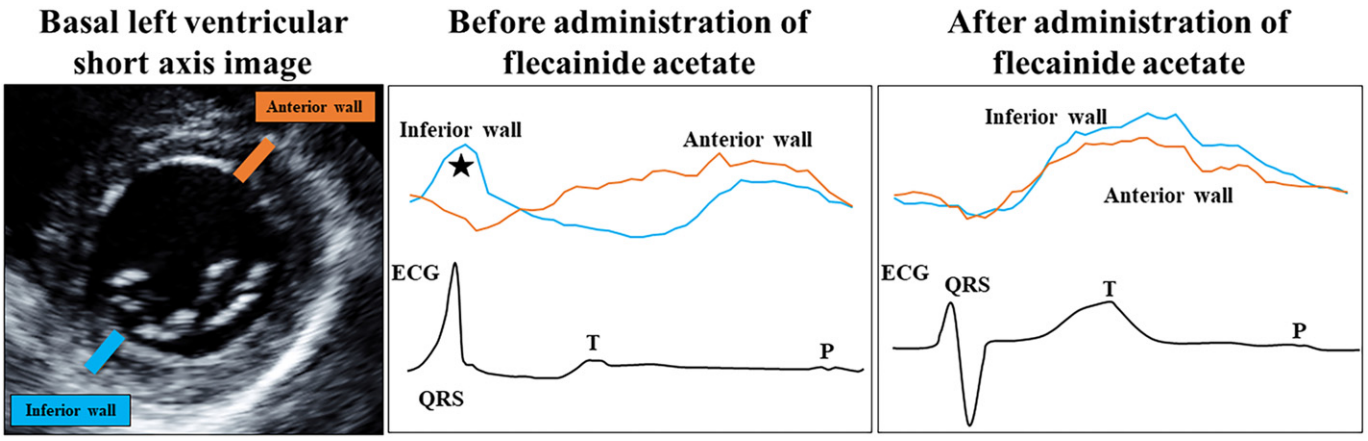

Fig. 2. The speckle tracking echocardiography clearly detected the preexcitation of the basal inferior wall (black star) accompanied with delta-wave and resultant left ventricular dyssynchrony. The preexcitation as well as dyssynchrony disappeared after oral administration of flecainide acetate along with disappearance of the delta-wave in electrocardiography (ECG).

attachment of the accessory pathway. Accordingly, local ventricular mechanical preexcitation can be observed as regional wall motion abnormality and/or ventricular dyssynchrony [2–4]. Echocardiography focused on the left ventricle basal level is useful for visualizing accessory pathway-related regional wall motion abnormalities. In contrast to the present case, accessory pathway-related mechanical preexcitation is frequently detected in patients with right or septal accessory pathways compared to those with left accessory pathways [5–7]. In this patient, the site of the accessory pathway presumed by the electrocardiography [8] was identical to the region of focal hypokinesia detected by transthoracic echocardiography and cardiac magnetic resonance imaging. This observation led us to suspect accessory pathway-related preexcitation rather than newly developed cardiac sarcoidosis. Although administration of flecainide acetate is not necessary for isolated cases with asymptomatic manifest ventricular preexcitation, it is useful in this particular patient to rule out cardiac involvement of sarcoidosis. When "hypokinesia" is detected in cases with manifest ventricular preexcitation, active searching of the pre-systolic shortening around the atrioventricular valve annulus using frame-by-frame B-mode echocardiography, M-Mode echocardiography, tissue-Doppler echocardiography, and/or speckle tracking echocardiography is useful (Fig. 2).

In some cases of manifest atrioventricular accessory pathway, left ventricular systolic dysfunction can be induced along with left ventricular dyssynchrony, which can be cured by radiofrequency catheter ablation [9–11]. As the present case was asymptomatic with preserved left ventricular ejection fraction, we selected annual echocardiographic observation without any interventions, including continuous oral administration of flecainide acetate and radiofrequency catheter ablation.

Conclusion

This case highlights the importance to appreciate the mechanical left ventricular preexcitation due to manifest accessory pathway, which could be eliminated by temporal administration of flecainide acetate. Accordingly, we could successfully differentiate the mechanism of hypokinesia.

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