CASE REPORT

A Case of Brugada Pattern Migrant from Right Precordial Leads to Peripheral Leads

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Since the first report in 1992, Brugada pattern (BP) diagnosis is mainly based on analysis of the precordial leads. In cases with no clear BP evidence in the conventional right precordial leads (4th intercostal space), limb leads analysis resulted helpful in suspecting BP. Fluctuations within right precordial leads between the diagnostic ECG pattern and nondiagnostic ECGs are well known. For the first time, in the patient herewith reported, the transformation of BP phenotype involves both precordial and peripheral leads, confirming that the analysis of all the 12 leads has a key role in BP diagnosis.

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Brugada pattern; ST segment elevation; 12-lead 24-hour Holter; inferior leads; ST segment depression

Since the first report in 1992,[1] but particularly in the last years, Brugada pattern (BP) has been searched for due to the potential link of this electrocardiogram (ECG) with sudden cardiac death. The diagnosis is mainly based on analysis of the precordial leads.[2,3] In cases with no clear BP evidence in the conventional right precordial leads (4th intercostal spaces [ics]), ST depression in the inferior leads resulted helpful in suspecting BP, as recently reported.[4] We describe a patient whose ECG showed an unexpected change from ST elevation in the right precordial leads to ST depression in the inferior leads.

A 72-year-old female was admitted for asthenia and nausea. She had neither common cardiovascular risk factors nor history of cardiac arrest or syncope. She was assuming levothyroxine for hypothyroidism. Physical examination was unremarkable apart from a 2/6 aortic systolic murmur. The 12-lead ECG at rest showed sinus rhythm, normal atrioventricular conduction, normal QRS axis, and a spontaneous type 1 BP (coved ST segment elevation, negative T wave; Fig. 1). No ST segment abnormality was recognizable in the inferior leads. Moving V_1 and V_2 electrodes at the 3rd and 2nd ics type 1 BP disappeared. The echocardiogram showed slight interventricular septum thickening (12 mm), normal ejection fraction (60%), mild mitral and tricuspid regurgitation, and normal right and left ventricular chamber size. A 12-lead Holter recording, as part of a research protocol, was performed, the day after, with the precordial electrodes in the conventional position (V_1 and V_2 electrodes at the 4th ics, where type 1 BP was present at the rest ECG). It showed no type 1 BP whereas persistent ST segment upsloping depression was observed in the inferior leads. ST segment elevation with coved pattern was observed only in lead aVL. In addition, only a few atrial and ventricular extrasystoles were recorded (Fig. 2). Serum electrolyte and concentration was normal and necrosis markers were negative during the hospitalization.

Not rarely, the diagnosis of BP is challenging since the typical signs are intermittent or

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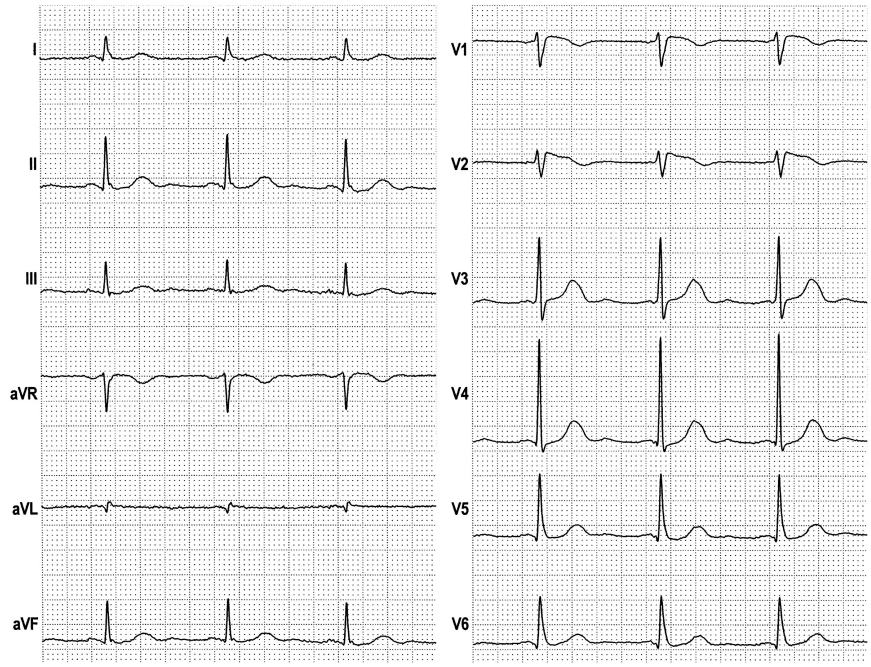

Figure 1. ECG at rest.

recognizable only in the "high" right precordial leads. Holter monitoring can be useful to reveal, in selected cases, an otherwise concealed BP, as recently demonstrated by Cerrato et al.[5] A recent article has pointed out that ST segment depression in the inferior leads can be helpful in suspecting BP, suggesting the need for right precordial electrodes displacement 1 or 2 spaces above.[4] This holds true especially in patients whose ECG is quite normal in leads V_1 and V_2 recorded at the 4^{th} ics. Whenever no common causes of ST segment abnormality are found, such as ischemia, electrolyte imbalance, intraventricular conduction disturbance, left or right ventricular hypertrophy, etc., an unexplained ST segment depression in the inferior leads should be considered as a possible pointer to BP, suggesting the need for right precordial electrodes displacement 1 or 2 spaces above.

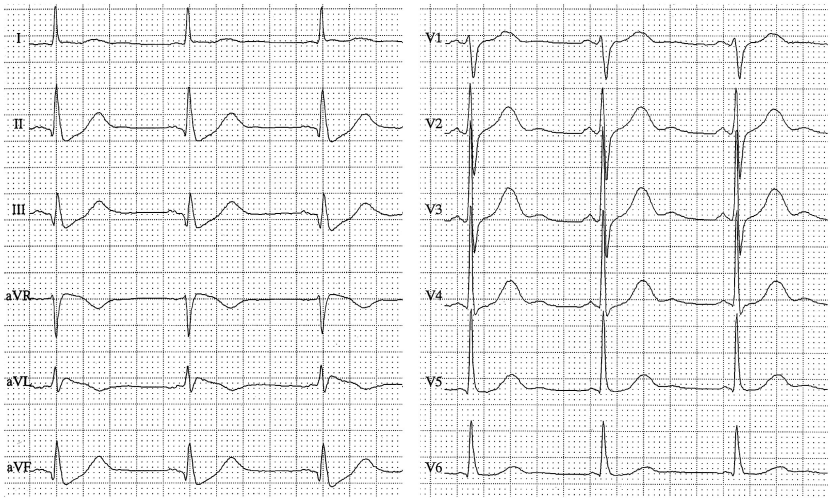

Figure 2. Twelve-lead ECG recorded during Holter monitoring.

In the patient herewith reported, the standard ECG did not show any ST segment abnormality in the peripheral leads, whereas a type 1 BP appeared in the right precordial leads. The 12-lead Holter recording, in contrast, did not show spontaneous type 1 BP in leads V_1 and V_2, but revealed persistent ST segment depression in the inferior leads and a coved ST segment elevation in lead aVL. A persistent 24-hour ascending ST depression, in the absence of typical symptoms or positivity of myocardial necrosis markers, excluded acute coronary artery disease. A sodium channel blocker test would have been helpful in order to further confirm the diagnosis of BP but it could not be performed. On the 3rd day from admission the patient refused any other diagnostic exams and was discharged from hospital. We recommended avoiding drugs reported on the Web site www.brugadadrugs.org, to treat fever promptly and to perform an ECG in first-degree relatives.

Fluctuations within right precordial leads between the diagnostic ECG pattern and nondiagnostic ECGs are well known. For the first time, in this case, the transformation of BP phenotype involves both precordial and peripheral leads, confirming that the analysis of all the 12 leads has a key role in BP diagnosis.

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