

Brugada-Like Early Repolarization Pattern Misdiagnosed as Acute Anterior Myocardial Infarction in a Patient with Myocardial Bridging of the Left Anterior Descending Artery

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Abstract

The diagnosis of acute coronary syndrome in patients presenting to the emergency department with chest pain is still challenging. Since the symptoms and electrocardiographic abnormalities of patients with acute myocardial infarction can be indistinguishable from those of patients with other conditions that lead to ST-segment elevation, a high clinical index of suspicion is needed to avoid an incorrect diagnosis and subjecting the patient to unwarranted thrombolytic therapy. Our report concerns a 53-year-old male with myocardial bridging of the left anterior descending artery. He presented with the combined electrocardiographic abnormality of the Brugada-like or early repolarization pattern, which was misdiagnosed as acute anterior myocardial infarction.

Key Words: Brugada-like electrocardiogram, early repolarization, myocardial bridging.

Introduction

THE EVALUATION OF ACUTE CHEST PAIN remains challenging, despite many insights and innovations over the past two decades (1). Since “time is myocardium,” it is important to make the diagnosis as quickly as possible. It is equally important, however, not to confuse other causes of ST-segment elevation with acute myocardial infarction (MI) (2, 3). The electrocardiogram (ECG) remains a crucial tool in the identification of acute chest pain with a detailed analysis of patterns of ST-segment elevation (4–6). We present a case and discussion about a patient admitted to the emergency department with chest pain and ECG changes consistent with an acute MI, Brugada syndrome, or early repolarization; the patient subsequently was found to have myocardial bridging (MB).

Case Presentation

A 53-year-old man was admitted to the emergency department with burning, nonexertional chest pain that persisted for 1 hour. The pain had not occurred before and was associated with nausea and vomiting. He was free from any conventional coronary risk factors, including cigarette smoking, diabetes mellitus, hyperlipidemia, hypertension and a family history of coronary artery disease. His medical history was unremarkable, and he was not taking any prescribed medications. He had no family history of sudden death, nor had he experienced any episodes of syncope. At the time of the patient’s admission, his temperature was 36.2°C, his pulse was 75 beats per minute, his respiratory rate was 20 breaths per minute, and his blood pressure was 130/85 mm Hg. Auscultation revealed no cardiac murmurs or rales. Upon examination, his other systems were also found to be normal. ECG showed incomplete right bundle branch block (RBBB) with abnormal ST segments in V1–4, consistent with acute anterior MI (Fig. 1). The levels of glucose, urea nitrogen, creatinine, aspartate aminotransferase, alanine aminotransferase, troponin T, and the creatine kinase MB

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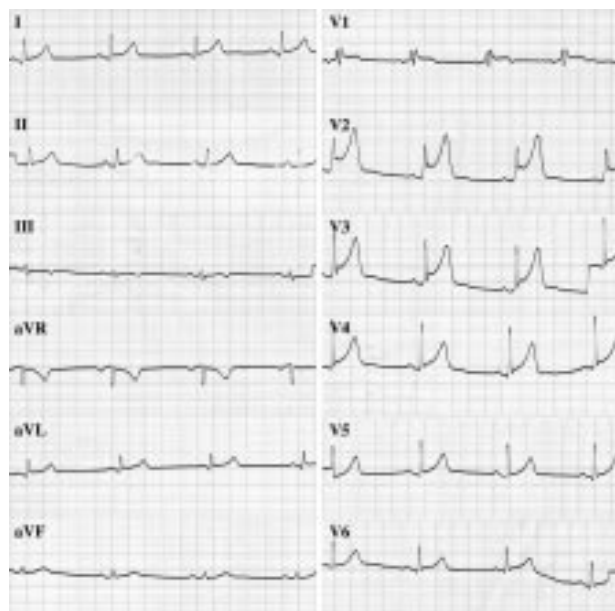


Fig. 1. 12-lead electrocardiogram shows incomplete right bundle branch block with ST-segment (J-point) elevation in leads V1–V4.

isoenzyme were normal (Table). Based on the clinical and electrocardiographic findings, an acute anterior MI was diagnosed, and the patient was treated with an intravenous infusion of streptokinase as a thrombolytic agent. But serial cardiac enzyme values were normal, and his ECG did not evolve as an MI pattern; it remained unchanged. Two days later, his chest pain resolved gradually. Echocardiographic examination revealed no obvious heart diseases, wall motion abnormalities or pericardial effusion.

The patient was referred for coronary angiography and was found to have a significant coronary artery systolic luminal narrowing in the mid segment of the left anterior descending coronary artery at left anterior oblique cranial position (Fig. 2). The right coronary artery and left ventriculo-

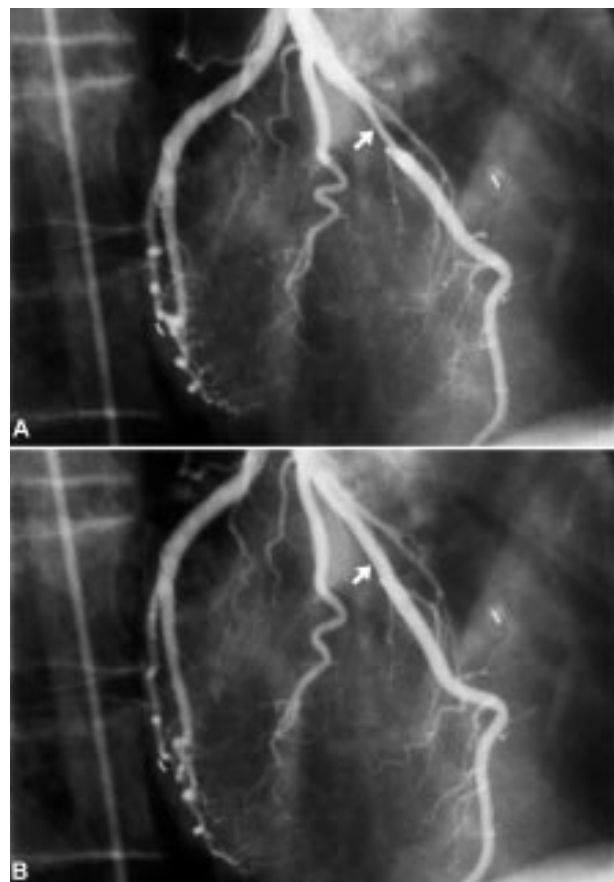


Fig. 2. The coronary angiography shows the myocardial bridging of the left anterior descending artery (arrows) in systole (A) and no coronary obstruction in diastole (B).

gram were normal. Since the patient was free of any personal or family arrhythmic history, a drug challenge test with antiarrhythmic agents was not performed. Beta-blocker therapy was prescribed and the patient was discharged without any problems. At 6-month follow-up, the patient had no cardiac symptoms, and his ECG was unchanged.

TABLE
Laboratory Data

	On Admission	6 h Later	12 h Later	24 h Later
Glucose (mg/dL)	108	98	100	96
AST (IU/L)	32	32	30	32
CK-MB (IU/L)	10	16	14	14
LDH (IU/L)	274	268	266	270
Troponin t (ng/mL)	<0.01	<0.01	-	<0.01
Total Chol (mg/dL)	-	169	-	-
HDL-Chol (mg/dL)	-	47	-	-
LDL-Chol (mg/dL)	-	90	-	-
Triglyceride (mg/dL)	-	161	-	-

AST=aspartate aminotransferase, CK-MB=creatinase MB isoenzyme, LDH=lactate dehydrogenase, Chol=cholesterol.

Discussion

ST elevation is typically seen in acute pericarditis, acute and subacute MI, Prinzmetal angina, old infarction with aneurysm, mirror image of left ventricular systolic overload and normal variants (including early repolarization). It is rarely encountered in hyperkalemia, hypercalcemia, cerebrovascular accidents, hypothermia, pneumothorax and hypertrophic obstructive cardiomyopathy (7). It is well known that the benefit of thrombolytic therapy is greatest when it is given soon after the onset of symptoms of acute ST-segment elevation MI (8). However, the symptoms and electrocardiographic abnormalities of patients with acute myocardial infarction can be indistinguishable from those of patients with other conditions that lead to ST-segment elevation, such as early repolarization, acute pericarditis, hyperkalemia, Brugada syndrome, ventricular aneurysm. Therefore, a high clinical index of suspicion is needed to avoid missing the diagnosis (9), resulting in unwarranted thrombolytic therapy or emergency angiography (2, 3).

“Early repolarization” is a rare normal variant, characterized by a marked, constant elevation of the J point and the ST segment of 2–4 mm, emerging directly from the R wave downstroke, in the anterior precordial leads (accentuated more septally or more laterally) and/or the inferior leads DIII and aVF. The mechanism is not clear (10). Ginzton and Laks studied 19 patients with acute pericarditis and 20 healthy individuals with ST elevation as a normal variant (early repolarization included). They observed that an ST/T ratio of greater than or equal to 0.25 in lead V6 identified the patients with pericarditis and excluded normal variants (11).

Brugada syndrome, first described as a new clinical entity by Pedro and Josep Brugada in 1992, is an inherited cardiac disease causing life-threatening ventricular tachyarrhythmias in individuals with structurally normal hearts and characteristic electrocardiograms showing a pattern of RBBB and ST segment elevation in right precordial leads V1 to V3 (12, 13). Nevertheless, diagnosis and risk stratification are not always clear. The ECG pattern may be dynamic over time and may include transient normalization. In some patients, complete or incomplete RBBB is present. In others, the high-takeoff ST segment mimics the pattern of RBBB, but the wide S waves in leads I, aVL, and V6 that are typically seen in RBBB are missing. The ST-segment elevation is primarily limited to leads V1 and V2 and can have a saddle-back shape, but in typical cases the ST segment

begins from the top of the R' wave, is downsloping, and ends with an inverted T wave. This pattern is so distinctive that it should not be mistaken for evidence of acute MI. In anteroseptal MI complicated by RBBB, the downstroke of the R' wave and the beginning of the ST segment have a distinct transition, and the ST segment is horizontal or upsloping, not downsloping (3, 6). In the present case, the electrocardiographic appearance was similar to that seen in Brugada syndrome, but it also resembled early repolarization pattern. But since the patient was asymptomatic for arrhythmic family and personal history, drug challenge was not performed; therefore, the term “Brugada-like” was used.

Acute MI or ischemia from vasospasm involving the right ventricular outflow tract can mimic ST-segment elevation similar to that in Brugada syndrome. This effect is probably the result of a depression of calcium channel current and the activation of ATP-sensitive potassium channel current during ischemia (14). MB causes the systolic compression of a coronary artery by overlying myocardial tissue and usually has a benign prognosis, but in some patients there may be myocardial ischemia, infarction, myocardial stunning, and/or sudden death. Brugada syndromes associated with MB (15) and early repolarization (16) have also been reported in the literature. Significant differences exist between athletes with early repolarization and patients with the Brugada syndrome with regard to the amplitude of ST elevation and QRS duration. The maximum ST elevation is greater in the athletes than in patients with the Brugada syndrome, and Brugada syndrome patients have a longer QRS duration than athletes with early repolarization (16).

The present case is unique in that the combined electrocardiographic abnormality of the Brugada-like and early repolarization pattern coexist with the bridged left anterior descending (LAD) artery. Considering the anamnesis and clinical findings, the condition underlying the ST elevation can be determined in most cases, but, as with this patient, it can sometimes be difficult. It is not clear whether the electrocardiographic appearance of this patient was due to transient ischemia caused by a bridged LAD artery, or if it was due to Brugada syndrome or early repolarization. However, since the ECG remained the same over time, a diagnosis of ischemia related to the bridged LAD artery was unlikely.

In this case report, we presented an interesting ECG sample, but there was no conclusive proof of its etiology. However, this case reminds us that a high clinical index of suspicion is needed to avoid an incorrect diagnosis of acute coronary syndrome.

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