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Brugada sign unmasking the location of an acute myocardial infarction

Objaw Brugadów ujawniający lokalizację ostrego zawału serca

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Abstract

Two patients with acute myocardial infarction were admitted with unremarkable electrocardiograms, which did not reveal the location of the damage. A review of these electrocardiograms led to a suspicion of the presence of Brugada sign (BRSG), something subsequently confirmed by the administration of a sodium channel blocker. The unmasking of BRSG was unexpectedly accompanied by repolarisation abnormalities, showing ischaemia in the lateral wall, concordant with the distribution of the culprit vessels in the coronary angiogram.

Key words: Brugada sign, electrocardiogram, acute myocardial infarction, procainamide

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INTRODUCTION

In 1992, three types of Brugada sign (BRSG) were first described in the electrocardiogram (ECG) of patients with malignant ventricular arrhythmias and cardiac sodium channelopathy [1]. Type 1 is the prototype of BRSG, consisting of a characteristic coved ST segment elevation with negative T waves in leads V1–V3. Types 2 and 3 display a saddle-back ST segment elevation in the same leads. To be of use in diagnosis, they must be converted into Type 1, usually under a procainamide or other sodium channel blocker challenge [2]. In the absence of an arrhythmogenic substrate in the familial or individual history, discovering BRSG is considered a fortuitous finding, although its potential meaning remains the subject of debate [3].

Although leads V1–V3 of the ECG are used primarily for diagnosing BRSG, other leads should not be overlooked. In the cases we report here, lateral leads acquired a particular significance in two patients with acute myocardial infarction (AMI) and pharmaceutically unmasked BRSG.

CASE 1

A 54 year-old previously healthy man was admitted with persistent retrosternal pain. Family history and clinical examination were unremarkable, and the ECG (Fig. 1A) was not compatible with an AMI. Echocardiography revealed a normal left ventricle and pericardium, while computerised chest tomography excluded pulmonary embolism and aortic events. When the elevated values of cardiac enzymes and troponin I became available, the diagnosis of non-ST elevation AMI was obvious, although its anatomical location was uncertain. A second interrogation of the ECG generated a suspicion of a Type 3 BRSG, and procainamide was administered to the patient, in accordance with the guidelines [2]. A typical Type 1 BRSG appeared in leads V1–V3, as well as an intriguing ST segment depression with negative T waves in inferolateral leads (Fig. 1B). This last finding was explained by a coronary angiogram, which showed an 85% stenosis in the circumflex artery. The rest of the coronary tree was free of significant disease. Angioplasty and stenting of the culprit lesion led to an uneventful recovery. The discharge ECG was similar to that done on admission. Eight months later, myocardial perfusion imaging with Thallium-201 was normal, and a new procainamide test reproduced the Type 1 BRSG without on this occasion influencing the inferolateral leads (Fig. 1C). An electrophysiological study (EPS) did not induce any ventricular arrhythmias. The patient was characterised as a fortuitous carrier of BRSG, and has successfully completed a six year follow-up.

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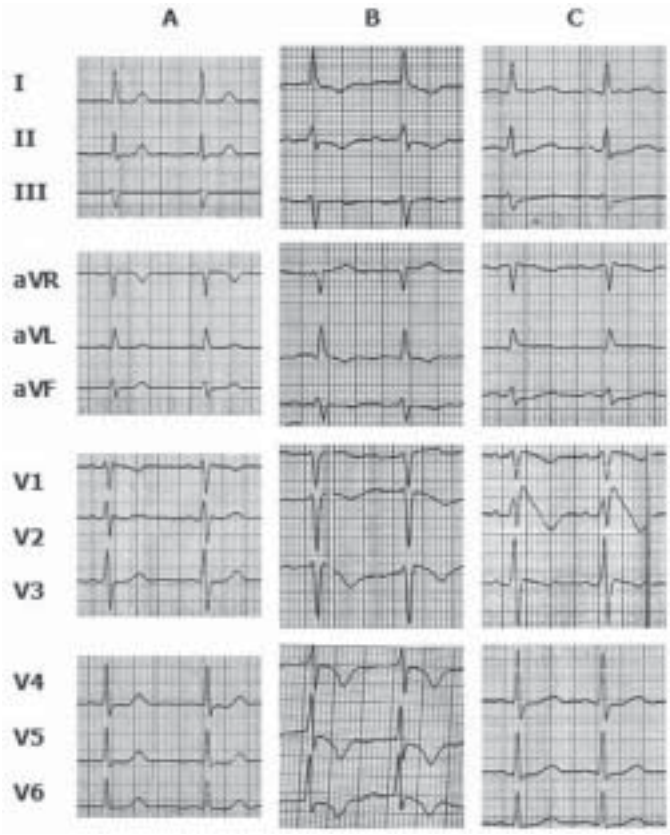

Figure 1. Electrocardiogram on admission (**A**), and after procainamide infusion (**B**). A repeat procainamide administration, eight months later (**C**)

CASE 2

A 62 year-old woman with a negative past medical history and no family history of cardiac disease was referred to us because of AMI. The diagnosis was based on a two hour history of chest pain and elevated cardiac enzymes and troponin I, while other aetiologies were excluded. Neither the echocardiogram nor the ECG (Fig. 2A) could locate the anatomy of the AMI. However, a Type 3 BRSG revealed in the ECG led us to administer procainamide, which unmasked a Type 1 BRSG in leads V1-V3 and, unexpectedly, deep negative T waves in lateral leads (Fig. 2B). A coronary angiogram was performed, showing a 75% stenosis of a great diagonal branch, which was treated with angioplasty and stenting. No other significant lesion was found. The discharge ECG was similar to the admission one. One year later, her exercise tolerance test was normal, and EPS did not induce ventricular ectopics. A new procainamide infusion accentuated the baseline Type 3 BRSG, leaving unaltered the lateral leads (Fig. 2C). The patient is doing well and has completed an uneventful four year follow-up.

Pharmaceutical challenging in these two cases performed a dual function. It unmasked a concealed Type 1 BRSG, and at the same time suggested the distribution of an AMI, by provoking unexpected ECG modifications in leads other than V1–V3.

DISCUSSION

The two cases we described share some common characteristics. Both had an AMI of uncertain anatomical location according to their admission ECG. When a pharmacological challenge, with a sodium channel blocker, was deemed necessary, a typical Type 1 BRSG was unmasked in leads V1––V3, with concomitant negative T waves in leads V4–V6. The changes in the lateral precordial leads were found to be concordant with the distribution of the culprit vessels in the coronary angiogram.

When a spontaneous or induced BRSG is expressed, it usually involves leads V1–V3, or less frequently the inferior leads [4]. A typical BRSG consists of a coved ST segment elevation with negative T waves in the aforementioned leads. An extension of the repolarisation abnormalities in the lateral precordial leads is extremely rare [5].

In the cases presented here, two hypotheses concerning the negative T waves in leads V1–V6 can be postulated. According to the first, these negative T waves could be attributed to an endogenous effect of the sodium channel blocker we administered. Contradicting this hypothesis is the fact that following the correction of ischaemia, repeating the pharmacological challenge with the same drug, at the same dose, left the leads V4–V6 unaffected, whereas BRSG reappeared in

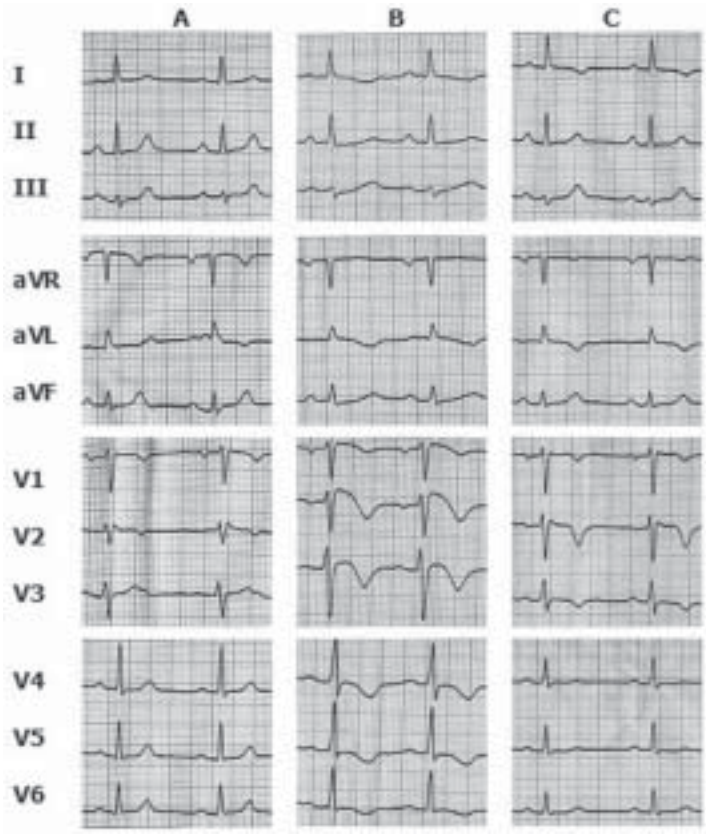

Figure 2. Admission electrocardiogram, baseline (**A**) and after procainamide challenge (**B**). One year later, in a new pharmacological challenge, the lateral wall remained unaffected (**C**)

the right precordial leads. The second hypothesis appears more plausible and is the one we propose. Negative T waves in leads V4–V6 were indicative of ischaemia. To enhance this theory, we have two facts: the coronary anatomy of the culprit vessels correspond to the lateral wall perfusion, and the inability to reproduce negative T waves in the same leads when a repetitive pharmaceutical challenge was performed after relieving ischaemia. BRSG in the two presented cases was not part of a Brugada syndrome. Neither the family and personal history, nor the electrophysiological study (EPS), confirmed the existence of such a syndrome. Thus, according to the latest guidelines, the two patients were placed under surveillance follow-up, during which they both had an uneventful course [6].

Several articles have been published, where the ECG findings of an AMI interact with BRSG and vice versa, leading to misinterpretations. There are reports of BRSG being mistaken for an AMI, and AMI which presented with BRSG [7, 8]. In our case, BRSG not only did not obscure the diagnosis, but it elucidated a previously unremarkable ECG, suggesting the presence of underlying ischaemia. Is there any clinical implication for the cases described? When anteroseptal lead BRSG extends to other leads in the form of negative T waves, the suspicion of myocardial ischaemia should be raised.

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