Clinical Case Report

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A case report of Brugada-like ST-segment elevation probably due to coronary vasospasm

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Abstract

Rationale: Vasospastic angina is caused by sudden occlusive vasoconstriction of a segment of an epicardial artery, with transient ST-segment elevation on electrocardiography. Brugada Syndrome is an inherited arrhythmogenic cardiac disorder with a diagnostic electrocardiography characterized by coved-type ST-segment elevation in right precordial leads (V1-V3). Those two diseases usually have no correlation. In this report, we discuss an interesting case of a patient who was diagnosed as vasospastic angina according to his coronary angiography, but his electrocardiography showed a Brugada-like ST-segment elevation.

Patient concerns: Our patient had a 9-month history of temporary but progressive substernal burning sensation with acid bilges of shoulders and arms, as well as profuse sweating at night.

Diagnoses: Although he had no abnormal laboratory test result, no dysfunctional recorded echocardiogram or documented arrhythmia after being admitted to the hospital, his electrocardiography showed a Brugada-like ST-segment elevation. The coronary angiography result confirmed a diagnosis of vasospastic angina.

Interventions: The patient was prescribed diltiazem, aspirin, isosorbide mononitrate and rosuvastatin and was strongly advised to quit cigarettes and alcohol.

Outcomes: Follow-up at half a year turned out well.

Lessons: This case links Brugada syndrome to coronary vasospasm. They may share similar mechanisms. Provocation test and gene test needs to be ran to distinguish both. Long-term follow-up is essential for it may bring a warning sign for life threatening ventricular arrhythmias.

Abbreviations: BrS = Brugada syndrome, ECG = electrocardiography, RCA = right coronary artery, RVOT = right ventricular outflow tract, SCD = sudden cardiac death, SCN5A = sodium voltage-gated channel alpha subunit 5, VF = ventricular fibrillation, VSA = vasospastic angina, VT = ventricular tachycardia.

Keywords: artery, Brugada syndrome, coronary, threatening ventricular arrhythmias, vasospasm

1. Introduction

Coronary artery vasospasm is an established cause for angina pectoris with transient ST-segment elevation on electrocardiography (ECG), which can be diagnosed as vasospastic angina (VSA). The electrocardiographic characteristic of Brugada syndrome (BrS) is spontaneous or induced coved-type ST-segment elevation in right precordial leads V1–V3.[1] Herein, we report an interesting case presenting with coronary artery vasospasm and a Brugada-like ST-segment elevation, which may reveal some mechanisms.

2. Case report

A 65-year-old Chinese male, who worked as an aid man before retirement, had a 9-month history of temporary but progressive substernal burning sensation with acid bilges of shoulders and arms, and profuse sweating at night. Each episode lasted for 1 h and recovered spontaneously, without any triggers. Previously, he was diagnosed with reflux esophagitis for which he was prescribed some acid-inhibitory medicines which was, however, not effective. He suffered the above-mentioned symptoms again 1 night with higher severity than before with palpitation and weakness. His blood pressure (130/110 mm Hg) and heart rate (55 beats/min) were measured, was accordingly prescribed 3 grain of traditional Chinese pills, fosinopril 5 mg, and metoprolol 25 mg and was taken to the emergency room. His personal history revealed a 25 pack-year smoking history, a 40-year drinking history, and a snoring habit at night. However, his medical, family, and psychosocial histories were normal. Interestingly, his ECG showed coved ST-segment elevation of approximately 0.3 to 0.8 mV in precordial leads V1–V3 and 0.5 to 0.7 mV in standard limb leads II, III, aVF, and "mirror change"

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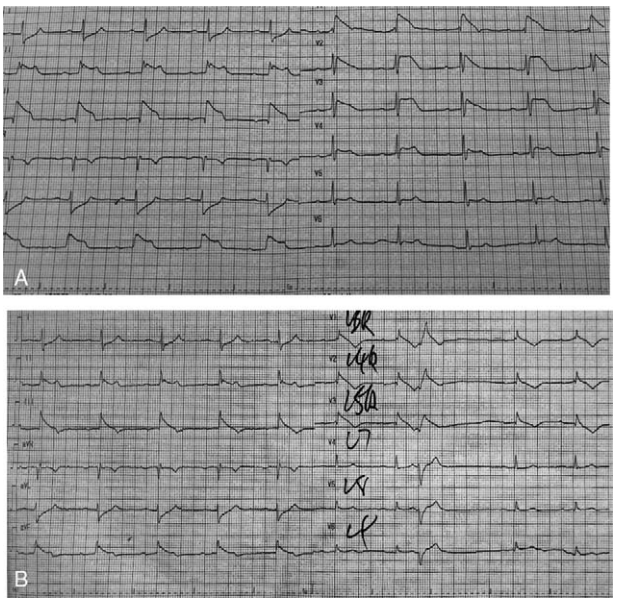

Figure 1. (A) Coved ST-segment elevation of approximately 0.3 to 0.8 mV in V1–V3 precordial leads and 0.5 to 0.7 mV in II, III, aVF limb leads, and "mirror change" in I, aVL. (B) Right bundle branch block (RBBB) in V3R–V5R, a ventricular premature contraction with long pause.

in leads I, aVL (Fig. 1A and B). Unfortunately, the echocardiogram was not taken immediately due to occasional reason. No arrhythmia was recorded. Laboratory tests showed no abnormalities. He was treated with intravenous nitroglycerin injection and exhibited dim consciousness with sweating, pallor, and cold limbs after that. In about 5 min after the treatment, the patient gained consciousness. The ECG, at this stage, was unremarkable (Fig. 2A and B). Coronary angiography demonstrated moderate stenosis of the left anterior descending branch (Fig. 3A and D). An echocardiogram taken after that was normal. VSA was diagnosed. He also underwent sleep monitoring with a result of moderate obstructive sleep apnea. The patient was treated with diltiazem, aspirin, isosorbide mononitrate, and rosuvastatin and counseled extensively on quitting cigarettes and alcohol. We followed up the patient at half a year after his being discharged. He was compliant of taking the prescribed pills on schedule and there was no reoccurrence of the symptoms. Written informed consent for publication was obtained from the patient.

3. Discussion

3.1. Brugada syndrome

Since being described for the first in 1992, BrS has been defined as an inherited arrhythmogenic cardiac disorder generally lacking of structural abnormalities.[2] This disorder can increase the risk of ventricular fibrillation (VF) especially in young adults. It is responsible for at least 4% to 12% of cases of sudden cardiac death (SCD) in the general population and 20% in patients with a

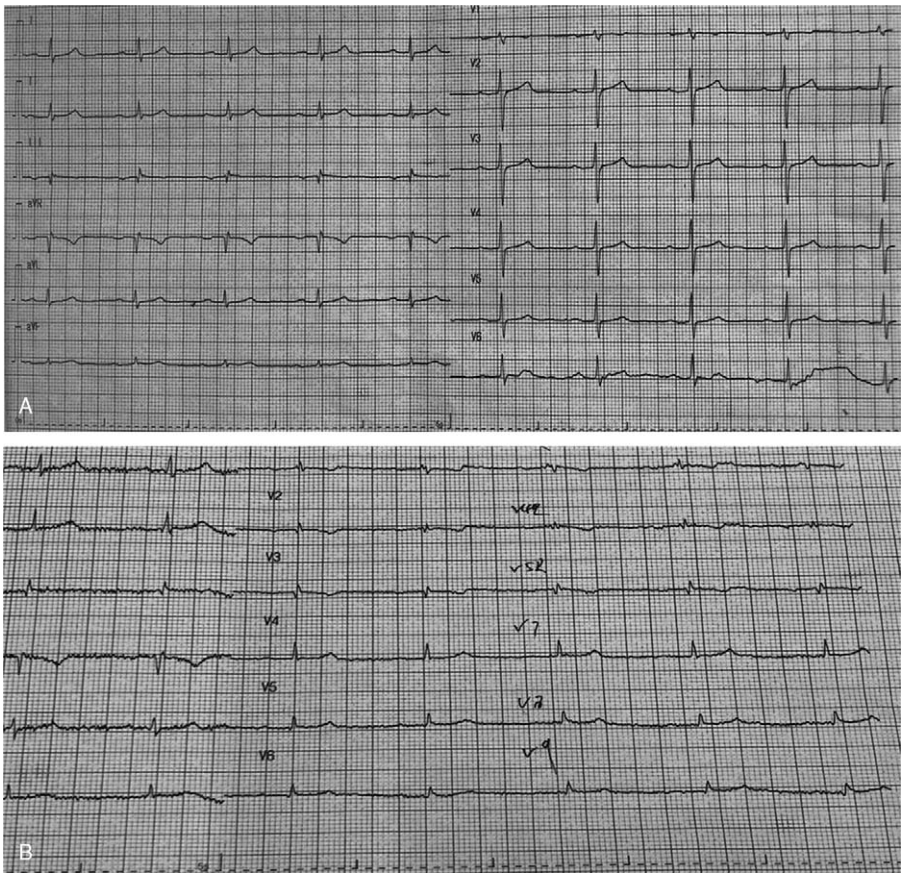

Figure 2. (A) Roughly normal electrocardiogram. (B) Roughly normal electrocardiogram.

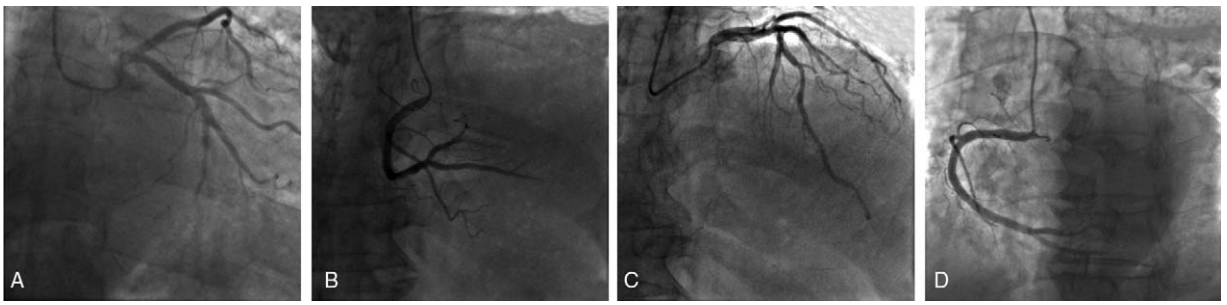

Figure 3. Coronary angiography was done after intravenous nitroglycerin and normalization of electrocardiogram. (A) Left coronary artery (straight caudal). (B) Right coronary artery (straight cranial). (C) Left coronary artery (right cranial), the red arrow shows a moderate stenosis of the middle of anterior descending branch. (D) Right coronary artery (left anterior oblique), the red arrow indicating conus branch.

structurally normal heart.[3] A diagnostic patent type 1 ECG of BrS is characterized by a coved ST-segment elevation of ≥2 mm followed by a negative T wave in >1 right precordial leads (V1–V3) in the presence or absence of a sodium channel-blocking agent, and in conjunction with one of the following: documented VF, polymorphic ventricular tachycardia (VT), a family history of SCD at <45 years of age, coved-type ECGs in family members, inducibility of VT/VF with programmed electrical stimulation, syncope, or nocturnal agonal respiration.[4] As a channelopathy, BrS is mainly caused by mutation of genes coding in sodium channel, calcium channel, potassium channel, or channel-interacting proteins.[5] The most common is the loss-of-function mutation affecting the sodium voltage-gated channel alpha subunit 5 (SCN5A) gene,[6] which accounts for <30% of clinically diagnosed BrS patients.[7] Genetic testing may be useful otherwise and is recommended for family members of a successfully genotyped proband.[7] In our case, the ECG pattern of the patient shows coved ST-segment elevation of approximately 0.3 to 0.8 mV in precordial leads V1–V3, which is similar to BrS, but there was no family history of SCD at early age, no coved-type ECGs in his 2 daughters and other relatives, no syncope or nocturnal agonal respiration in the past. We ran a gene test of the patient, the result showed no mutation of any exons or single nucleotide polymorphism loci in SCN5A, so we basically could exclude BrS.

3.2. Coronary artery vasospasm

Coronary artery vasospasm is defined as a situation in which a main epicardial coronary artery suddenly exhibits abnormal contraction. The diagnosis of VSA can be arrived at as per the documentation of transient ST-segment elevation during resting angina episodes and normal or nonobstructive plaques on coronary angiogram, without any triggers.[8] Our patient presented with both rest chest pain and transient ST-segment elevation with moderate stenosis of left anterior descending branch on coronary angiogram. The ECG pattern showed a Brugada-like ST-segment elevation, but there was also ST-segment elevation in leads II, III, aVF, and "mirror change" in limb leads I, aVL. Since we had excluded BrS as mentioned above, our patient was most likely to suffer from coronary vasospasm.

The interesting part of the case is the bizarre ECG pattern which looks like BrS, why did we get such a pattern? We think this may be attributed to vasospasm of the conus artery. The conus artery, usually the first branch of the right coronary artery (RCA), supplies blood to the right ventricular outflow tract (RVOT).[9] Blockage or narrowing of the spastic conus branch may lead to ventricular conduction delay and dispersion of conduction velocity in the RVOT, which may cause ECG abnormalities and initiation of life-threatening ventricular arrhythmias mimicking BrS.[10] While the ST-segment elevation in leads II, III, aVF may not have made the situation simple, vasospasm of only 1 branch would not cause extensive myocardial injury influencing inferior wall, let alone his unusual little conus artery according to the coronary angiography. Hence, we thought total RCA spasm or spasm at the RCA ostium before conus branch might be a possibility. Unfortunately, we did not perform the provocation test using acetylcholine to confirm this.

Otherwise, we could recognize significant right ventricular ischemia through the ECG, which showed the distribution of ST-segment elevation in the anterior leads is greater in leads V1–V2 and decreased toward V3–V4 compared with myocardial damage of left ventricular.[11] As we all know, opening of the cardiac voltage-gated sodium channel initiates cellular depolarization.[12] Accelerated inactivation of sodium channels and predominance of transient outward potassium current creates a gradient of the action potential in different layers of RVOT at the beginning of repolarization, imbalanced endocardial and epicardial action potentials explain the underlying mechanism of BrS.[1] Transient ischemia of large area affects the activities of inactivation particles, which promote changes in sodium channel structure and instability of the inactivated state of the channel.[12] Different inactivated states of sodium channel lead to different amplitude of transient outward potassium, which could explain the Brugada-like ST elevation and alternans of ST-T waves in the ECG (Fig. 1A).

Several limitations associated with the present report warrant mention. First, and most important, we did not perform provocation test. Gene test of SCN5A may not be sufficient as mutations in about 20 other genes have already been identified in patient with BrS.[13] So, it is too early to totally rule out BrS. Irrespective, whether a pattern of BrS-like ECG may bring a warning sign for life-threatening ventricular arrhythmias, and the prognosis of our patient still needs to be followed up further. Last but not the least, our patient had moderate obstructive sleep apnea and had ischemia attack at night almost all the time, so further treatment of sleep apnea may be necessary except for the calcium channel blockers for VSA.

References

[1] Rudic B, Chaykovskaya M, Tsyganov A, et al. Simultaneous non-invasive epicardial and endocardial mapping in patients with Brugada syndrome: new insights into arrhythmia mechanisms. J Am Heart Assoc 2016;5:e003122.

[2] Brugada P, Brugada J. Right bundle branch block, persistent ST segment elevation and sudden cardiac death: a distinct clinical and electrocardiographic syndrome: a multicenter report. J Am Coll Cardiol 1992;20:1391–6.
[3] Papadakis M, Raju H, Behr ER, et al. Sudden cardiac death with autopsy findings of uncertain significance: potential for erroneous interpretation. Circ Arrhythm Electrophysiol 2013;6:588–96.
[4] Bayes de Luna A, Brugada J, Baranchuk A, et al. Current electrocardiographic criteria for diagnosis of Brugada pattern: a consensus report. J Electrocardiol 2012;45:433–42.
[5] Corrado D, Zorzi A, Cerrone M, et al. Relationship between arrhythmogenic right ventricular cardiomyopathy and Brugada syndrome: new insights from molecular biology and clinical implications. Circ Arrhythm Electrophysiol 2016;9:e003631.
[6] Curcio A, Santarpia G, Indolfi C. The Brugada syndrome—from gene to therapy. Circ J 2017;81:290–7.
[7] Priori SG, Wilde AA, Horie M, et al. HRS/EHRA/APHRS expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes: document endorsed by HRS, EHRA, and APHRS in May 2013 and by ACCF, AHA, PACES, and AEPC in June 2013. Heart Rhythm 2013;10:1932–63.
[8] JCS Joint Working GroupGuidelines for diagnosis and treatment of patients with vasospastic angina (Coronary Spastic Angina) (JCS 2013). Circ J 2014;78:2779–801.
[9] Levin DC, Beckmann CF, Garnic JD, et al. Frequency and clinical significance of failure to visualize the conus artery during coronary arteriography. Circulation 1981;63:833–7.
[10] Wilde AA, Postema PG, Di Diego JM, et al. The pathophysiological mechanism underlying Brugada syndrome: depolarization versus repolarization. J Mol Cell Cardiol 2010;49:543–53.
[11] Namana V, Gupta SS, Abbasi AA, et al. Right ventricular infarction. Cardiovasc Revasc Med 2017;19:43–50.
[12] DeMarco KR, Clancy CE. Cardiac Na channels: structure to function. Curr Top Membr 2016;78:287–311.
[13] Portero V, Le Scouarnec S, Es-Salah-Lamoureux Z, et al. Dysfunction of the voltage-gated K+ channel beta2 subunit in a familial case of Brugada syndrome. J Am Heart Assoc 2016;5:e003122.