Ventricular fibrillation/Brugada-like ST segment elevation: First presentation of subarachnoid hemorrhage

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ABSTRACT

Brugada syndrome is an autosomal dominantly inherited channelopathy estimated to be responsible for 4—12% of all sudden deaths, particularly among middle-aged men. It is characterized by ST-segment elevation in the right precordial leads, in the absence of acute coronary syndrome. This report discuses a patient with subarachonoid hemorrhage who developed the characteristic electrocardiographic features of Brugada syndrome.

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1. Introduction

Brugada syndrome (BS) was first described in 1992 as a new autosomal dominantly inherited channelopathy caused by a defective gene located on chromosome 3 in a structurally normal heart. It is characterized by ST-segment elevation in the right pre-cordial leads and increases susceptibility to ventricular tachyar-rhythmias. Importantly, fluctuations in ECG patterns seem to be common among BS patients. For instance, vagotonic agents, α-adrenergic agonists, fever and bradycardia are some of the conditions that may reveal the abnormality on a surface ECG. Sub-arachonoid hemorrhage results in repolarisation abnormalities and changes in autonomic nervous system activity. In this report, we present the case of a patient with subarachnoid hemorrhage who was admitted to the emergency department after suffering an out-of-hospital cardiac arrest with typical ST-segment elevation and an ECG pattern characteristic of BS.

2. Case report

A 48-year-old man with no past medical history was presented to our emergency department after collapsing suddenly while shopping. A store employee trained in basic life support was the first to assist the patient and started to cardiopulmonary resusci-tation (CPR). Upon arrival to the local emergency medical service, the paramedics commenced ventricular fibrillation (VF) and an automated external defibrillator was used to defibrillate the pa-tient. After two separate defibrillations and 5 min of CPR the patient regained spontaneous circulation, but remained unconscious. The patient was then intubated and transferred to the hospital. At the emergency department he was flaccid, non-febrile and comatose (Glasgow Coma Scale of 4), with a heart rate of 75 bpm and blood pressure of 110/75 mmHg. Cardiovascular and respiratory exami-nation findings were unremarkable. Meanwhile, VF was performed several times and the patient was defibrillated successfully to a normal sinus rhythm. Electrocardiography revealed bizarre coved-type ST-segment elevation in leads V1 through V3, with T wave inversion consistent with the type 1 Brugada pattern (Fig. 1) and reciprocal ST depression in inferior and lateral leads. Emergent cardiac catheterization was performed, which revealed no stenosis or flow abnormalities in his coronary arteries. After contrast in-jection into those coronary arteries, ST-segment elevation was resolved and did not recur (Fig. 2). Echocardiography showed normal left ventricular ejection fraction and no segmental wall motion abnormality. Laboratory tests, including cardiac biomarkers and coagulation studies, produced results that were within normal reference limits. Since the patient was not responding, a non-contrasted brain computed tomography scan was performed and revealed a diffuse subarachnoid hemorrhage. He was admitted to

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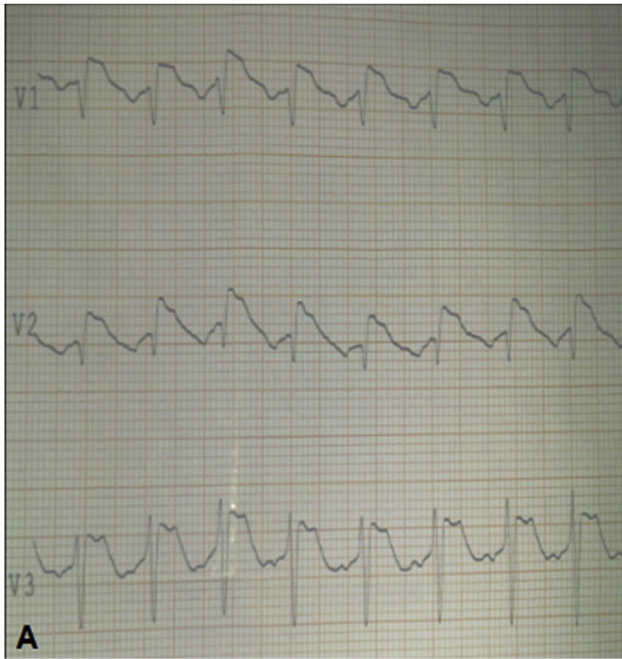 E-mail address: alosk@hotmail.com (A. Ösken).

Fig. 1. Coved-type ST elevation in leads V1 through V3.

the intensive care unit and determined to be brain dead after further neurological testing.

3. Discussion

Brugada syndrome is characterized by ST-segment elevation in the right precordial ECG leads, specifically V1-V3, and a high incidence of sudden death due to ventricular tachyarrhythmias in patients with structurally normal hearts. The syndrome is generally thought to be responsible for 4–12% of all sudden deaths and at least 20% of deaths in patients with structurally normal hearts; the most common presentation is syncope. The syndrome is inherited and auotosomally dominant, and in 25% of the cases the responsible mutation is found on the cardiac sodium transport gene (SCN5A), located on chromosome 3. Because the ECGs of patients with Brugada syndrome are complex and often conceal the condition, it is difficult to estimate the true incidence of the disease in the general population [1]. However, three types of repolarisation patterns in the right precordial leads are currently recognized. Type 1 is diagnostic of BS and is characterized by a coved ST-segment elevation ≥2mm (0.2mV), followed by a negative T wave. Type 2 has a saddleback appearance with a high take-off ST-segment elevation of ≥2mm, followed by a trough displaying ≥1mm ST-segment elevation and either a positive or biphasic T wave. Type 3 has either a saddleback or coved appearance with an ST-segment elevation of <1mm [2]. Sodium-channel blockers, vagotonic agents, α-adrenergic agonists, β-adrenergic blockers, tricyclic antidepressants, glucose-induced insulin secretion, fever, bradycardia, ischemia, hypothermia, hyperkalemia, hypokalemia, hypercalcemia, alcohol and cocaine can expose or induce these ECG patterns. Additionally, ST-segment elevation in the Brugada patients is significantly influenced by heart rate and autonomic tone.

The ventricular myocardium is composed of at least three electrophysiologically distinct cell types: epicardial, endocardial and M cells. The ST-segment elevation and T wave inversions seen in the right precordial leads of those with BS are thought to be due to an alteration in the action potential of the epicardial and possibly the M cells, but not the endocardial cells. The resulting dispersion of repolarisation across the ventricular wall, which is most pronounced in the right ventricle, results in a transmural voltage gradient that is manifested in the electrocardiogram as ST-segment elevation. This dispersion of repolarisation facilitates the development of phase 2 reentry, which generates a phase 2 reentrant extrasystole that takes advantage of the window of opportunity to precipitate the ventricular tachycardia and/or fibrillation that often results in sudden cardiac death.

A BS diagnosis is confirmed when a type 1 ST-segment elevation is observed in more than one right precordial lead (V1-V3), in the presence or absence of a sodium channel block, and in conjunction
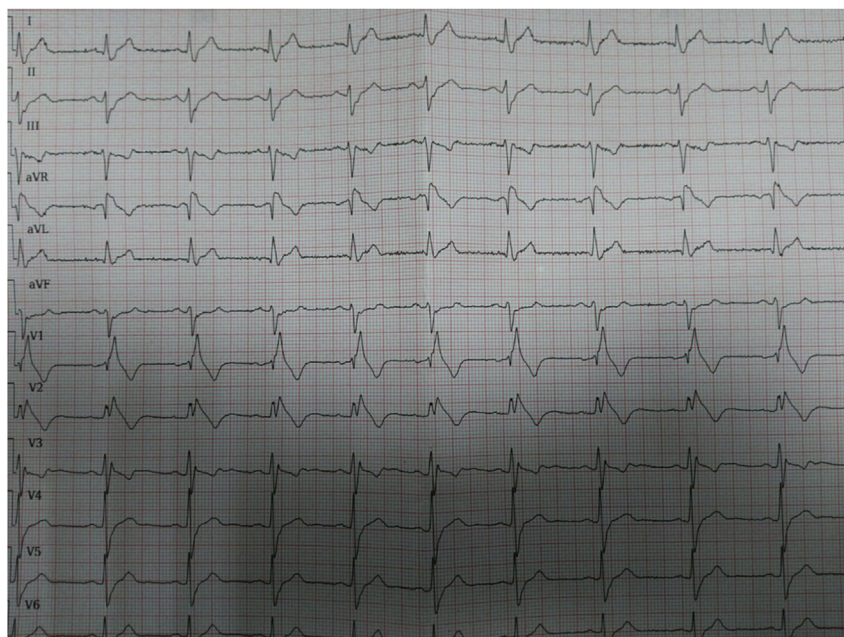

Fig. 2. Resolution of ST elevation in V1-3 leads.

with one of the following: documented ventricular fibrillation, self-terminating polymorphic ventricular tachycardia, a family history of SCD (<45years), coved type ECGs in family members, inducibility of VT with programmed electrical stimulation, syncope or nocturnal agonal respiration [2].

Cardiac arrhythmias or repolarisation abnormalities occur after a stroke in approximately 60–70% of patients and may have important prognostic implications. In one report, Mitsuma et al. investigated the circulatory collapse mechanisms in patients who experienced out-of-hospital cardiac arrest (OHCA) due to subarachnoid hemorrhage [3]. Pulseless electrical activity and asystole were common among study patients, but no ventricular fibrillation was observed. Mitsuma et al. found that the return of spontaneous circulation rate was higher than other etiologies of OHCA, but long-term survival was poor. A rate of VF similar to the initial cardiac rhythm was observed in a study by Inamasu et al. Researchers concluded that the prognosis was more favourable if the initial rhythm causing circulatory collapse was VF, but not brainstem damage [4]. In another study, Brouwers et al. demonstrated that ECG changes were most prominent 24–72 hours after subarachnoid hemorrhage [5]. Specifically, this event leads to repolarisation and several electrocardiographic abnormalities, including QT segment prolongation, aberrant Q waves, non-specific ST-segment changes, T wave abnormalities and the appearance of U waves [6]. Evidence of subendocardial infarction or anterolateral ischemia is common after a stroke, especially after subarachnoid hemorrhage [7]. Myocardial injury during a stroke is most likely the result of a centrally mediated release of catecholamines, due to hypoperfusion of the posterior hypothalamus. Transient coronary vasospasm secondary to increased sympathetic tone during a stroke has also been described [8]. The depression of ICA and activation of IK-ATP during ischemia associated with vasospasm involving right ventricular outflow tract results in ST-segment elevation similar to that observed in cases of BS [9].

In our patient, subarachnoid hemorrhage created a transmural voltage gradient due to its effect on repolarisation or increased α-adrenergic activity and resulted in a BS-like ECG pattern. After coronary angiography, ST-segment elevation was reversed. Subarachnoid hemorrhage may be one of the factors predisposing the patient to the electrocardiographic and arrhythmic manifestations of BS.

To conclude, when a patient without known structural heart disease presents with ventricular tachyarrhythmia and BS-like ST-segment elevation, subarachnoid hemorrhage should be kept in mind, since its prompt and accurate treatment could be life saving.

Conflict of interest

None.

References

[1] 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.

[2] Wilde AA, Antzelevitch C, Borggrefe M, Brugada J, Brugada R, Brugada P, et al. Proposed diagnostic criteria for the Brugada syndrome: consensus report. Circulation 2002;106:2514–9.

[3] Inamasu J, Nakagawa Y, Kuramae T, Nakatsukasa M, Miyatake S. Subarachnoid hemorrhage causing cardiopulmonary arrest: resuscitation profiles and outcomes. Neurol Medico Chirurgica 2011;51:619–23.

[4] Mitsuma W, Ito M, Kodama M, Takano H, Tomita M, Saito N, et al. Clinical and cardiac features of patients with subarachnoid haemorrhage presenting with out-of-hospital cardiac arrest. Resuscitation 2011;82:1294–7.

[5] Brouwers PJ, Wijdicks EF, Hasan D, Vermeulen M, Wever EF, Frericks H, et al. Serial electrocardiographic recording in aneurysmal subarachnoid hemorrhage. Stroke 1989;20:1162–7.

[6] Goldstein DS. The electrocardiogram in stroke: relationship to pathophysiological type and comparison with prior tracings. Stroke 1979;10:253–9.

[7] Chua HC, Sen S, Cosgriff RF, Gerstenblith G, Beauchamp NJ, Oppenheimer SM. Neurogenic ST depression in stroke. Clin Neurol Neurosurg 1999;101:44–8.

[8] Wang TD, Wu CC, Lee YT. Myocardial stunning after cerebral infarction. Int J Cardiol 1997;58:308–11.

[9] Noda T, Shimizu W, Taguchi A, Satomi K, Suyama K, Kurita T, et al. ST-segment elevation and ventricular fibrillation without coronary spasm by intracoronary injection of acetylcholine and/or ergonovine maleate in patients with Brugada syndrome. J Am Coll Cardiol 2002;40:1841–7.