One Brugada Syndrome Patient with Ventricular Fibrillation Misdiagnosed as Secondary Epilepsy

Bingsheng Huang, Ying Cheng, Qiang Xie, Yuncheng Zeng, Kuixiong Lin, Yanlin Feng, Yuyan Wu and Weijian Xie

The patient was a 47-year-old male who had 3 syncope and convulsion episodes over a 1-month period. The patient experienced right temporal bone fracture 3 years previous to this admission. At the first admission period, his interictal electroencephalogram (EEG) was normal, but he was misdiagnosed as secondary epilepsy because of the history of temporal bone fracture. In second admission, his electrocardiogram (ECG) revealed atrial fibrillation, partial right bundle branch block (RBBB), and J-point and ST-segment elevation in right precordial leads. The ST-segment was downward elevation on lead v1. But the ST-segment showed typical coved-pattern ECG of Brugada syndrome on lead v2. While the patient was in syncope, the ECG monitor revealed ventricular fibrillation. The ST-segment fell to the basic level on lead v1 and the coved-pattern ECG disappeared on lead v2 after administration of intravenous isoprenaline (0.25 μg/kg·min) for 2 hours. The patient was free of syncope and convulsion after implanted implantable cardioverter defibrillator (ICD) and administration of amiodarone to prevent atrial fibrillation.

At first admission, the patient was diagnosed secondary epilepsy because of the history of temporal bone fracture, syncope and convulsion, although there was the normal interictal electroencephalogram. It was regretted that we didn’t follow him up further.

**Key Words:** Brugada syndrome • Epilepsy • ICD • Ventricular fibrillation

Brugada syndrome is a sudden cardiac death syndrome that has the specific electrocardiogram (ECG) features of J-point elevation and ST-segment elevation.\(^1\,^2\,^3\) The disease is genetically determined, with an autosomal dominant pattern of transmission. Three different mutations that affect the structure and function of the cardiac sodium channel gene \(SCN5A\) have been identified. Some studies showed there was cardiac autonomic dysfunction in Brugada syndrome.\(^4\,^5\,^6\) Also atrial vulnerability is increased in patients with Brugada syndrome.\(^7\) The association of Brugada syndrome with supraventricular tachycardia has also been reported. In treatment, long-term beneficial action of quinidine has been described in a population of Brugada syndrome. In high-risk patients with Brugada syndrome, primary prophylactic ICD therapy is an effective treatment.\(^8\)

We describe a case of Brugada syndrome with ventricular fibrillation misdiagnosed as secondary epilepsy.

**CASE REPORT**

A 47-year-old male was admitted to our department of neurology with 3 syncope and convulsion episodes in the preceding one month in September, 2005. One month before, the patient experienced syncope and convulsion...
without any causes. The symptoms stopped without treatment after continuing for 10s seconds. Then he was admitted to the department of neurology with secondary epilepsy. In 2002, his right temporal bone had been fractured because of accident. Hypertension, diabetes mellitus, coronary heart disease, rheumatic heart disease, myocarditis, cardiomyopathy arrhythmias, congenital heart disease and cerebral disease were not shown in his medical record. At the first admission, the physical examination was normal. The patient had no history of donating blood recently. The electrocardiogram (ECG), interictal electroencephalogram (EEG) and brain CT were normal. Blood-RT, plasma electrolytes, cardiac markers, liver and renal function was also normal. The patient was free of syncope and convulsion after anti-epileptic treatment. One week later, the patient was discharged.

In October, 2005, the patient was admitted to the department of neurology with similar symptoms of syncope and convulsions again. The physical examination was normal. Echocardiography and the general biochemistry were also normal. But his ECG revealed (Figure 1): atrial fibrillation; partial right bundle branch block (RBBB); the J-point and ST-segment elevated 2-3 mv on leads v1-2. The ST-segment was downward elevation on lead v1. But the ST-segment showed typical coved pattern ECG of Brugada syndrome on leads v2. After admission, the patient experienced the same symptoms as above, and the ECG monitor revealed ventricular fibrillation (Figure 2). The ventricular fibrillation converted to sinus rhythm without RBBB by immediate electric defibrillation. So, the cause of the syncope experienced by the patient was suspected to be associated with Brugada syndrome. After administration of intravenous isoprenaline (0.25 μg/kg·min) for 2 hours, the ST-segment didn’t elevate on lead v1 and the coved-pattern ECG disappeared on lead v2. But the ST-segment was elevated 2mv on lead v2 still. After stopping intravenous isoprenaline, the ST-segment on leads v1-2 elevated again (Figure 3). Thus Brugada syndrome was diagnosed. But the patient hadn’t undergone provocative test with class I antiarrhythmia drug. After an implantable cardioverter defibrillator (ICD) was implanted and amiodarone was prescribed, the patient was free of syncope and convulsion.

ECGs of his family members didn’t reveal similar electrocardiogram, and his family members didn’t experience syncpe or convolution episodes.

**DISCUSSION**

At present, ventricular fibrillation is without clearly understood precipitating factors. But hypokalemia and the physical and emotional stress caused by the repeated shocks maybe contribute to the episodes of ventricular fibrillation. Cerebral and heart disease were not shown in our patient’s medical record. At his first admission, the physical examination was normal. The patient had no history of donating blood recently. The ECG, interictal EEG and brain CT were normal. Blood-RT, plasma elec-
trolytes, cardiac markers, liver and renal function were also normal. So the cause of the syncope experienced by the present patient was suspected to be associated with epilepsy because of the history of right temporal bone fracture, syncope and convulsion.

On second admission, ECG revealed that atrial fibrillation, partial RBBB, and J-point and ST-segment elevation were all in right precordial leads. The ST-segment was downward elevation on lead v1, but the ST-segment showed typical coved-pattern ECG of Brugada syndrome on lead v2. While the patient was in syncope, the ECG monitor revealed ventricular fibrillation. After intravenous isoprenaline (0.25 μg/kg·min), J-point and ST-segment in right precordial leads turned to normal or slightly elevated. After stopping intravenous isoprenaline, the ST-segment on leads v1-2 elevated again, showing that autonomic nerve participated in the ST-segment elevation on leads v1-2. Brugada syndrome was strongly supported.

Studies have shown there is impact of myocardial autonomic dysfunction in patients with Brugada syndrome. In Brugada syndrome, spontaneous augmentation of ST elevation in daily life occurs along with an increase in vagal activity. Patients with a Brugada-syndrome ECG pattern had lower HRV and QT/RR slopes than control subjects during nighttime. High-risk patients with spontaneous Brugada syndrome-ECG patterns had the lowest nocturnal QT/RR slopes. These unique repolarization dynamics might be related to the frequent nocturnal occurrence of ventricular tachycardia in Brugada syndrome. At present, results of study suggest that the shape of ST-segment elevation may be associated with myocardial autonomic nervous function. In addition, the electric heterogeneity of the action potential in the right ventricular epicardial myocardiun, which is frequently influenced by autonomic nervous activity, is closely associated with the development of Brugada syndrome.

Atrial vulnerability is increased in patients with Brugada syndrome. Abnormal atrial conduction may be an electrophysiologic basis for induction of atrial fibrillation in patients with Brugada syndrome. Study showed that atrial flutter-fibrillation incidence was 20% in Brugada syndrome patients. In patients with indication for implantable cardioverter defibrillator (ICD), the incidence of atrial flutter-fibrillation reached 27% vs 13% in patients with Brugada syndrome but without ICD indication. It strongly suggests a more advanced disease process in Brugada syndrome patients with spontaneous atrial flutter-fibrillation.

At present, long-term beneficial action of quinidine has been described in a population of Brugada syndrome. Primary prophylactic ICD therapy is an effective treatment. Antiarrhythmic drugs like amiodarone and beta-blockers do not prevent sudden death in symptomatic or asymptomatic individuals. Quinidine is not prescribed to patients because there is not quinidine in many places in China, including Hongkong. Amiodarone prescribed because the ECG revealed atrial fibrillation when our pa-
tient had an episode and due to the association between Brugada syndrome and atrial fibrillation. So the episodes of syncope and convulsion might be decreased by amiodarone preventing atrial fibrillation.

This patient was misdiagnosed as secondary epilepsy because of the history of temporal bone fracture, syncope and convulsion, although there was normal interictal electroencephalogram (EEG). At the first admission period, he was free of syncope and convulsion after administration of anti-epilepsy medication, and cerebral and heart diseases were not shown in his medical records. His family members didn’t experience syncope or convulsion episodes. With all these features, we believed that he had epilepsy and didn’t pay attention to other diseases that caused syncope and convulsion. It was regretted that we didn’t follow him up further.

REFERENCES


誤診為繼發性癲癇的 Brugada 綜合症併心室顫動

黃冰生  程穎  解強  曾寶澄  林桂雄  馮燕玲  吳鈺燕  謝偉堅  
廣東省  廣州市番禺何賢紀念醫院  心內科

該患者為 47 歲的男性，在入院前的 1 月裡共有 3 次神志不清和抽搐發作。3 年前因車禍導致右顳骨骨折。1 月前，患者因神志不清和抽搐發作入院，行心電圖未見異常，診斷為繼發性癲癇。第二次入院后心電圖顯示心房纖顫，部分右室胸前導聯 J 点和 ST 段抬高。當患者神志不清和抽搐再次發作時，心電監護提示心室顫動。該患者植入埋藏式心臟轉複除顫器后未再有神志不清和抽搐的發作。此病例提示診斷為癲癇的患者應排除 Brugada 綜合征的可能。

關鍵詞：Brugada 綜合症、癲癇、埋藏式心臟轉複除顫器、心室顫動。