Brugada Syndrome in the Elderly in Taiwan — Report of Two Cases

Jyh-Ming Juang, Ling-Ping Lai, Jiunn-Lee Lin and Fu-Tien Chiang

Brugada syndrome is a potentially lethal ventricular arrhythmia disease with an ECG pattern of ST-segment elevation in leads V1-3 and normal QT interval without structural heart disease. Clinical presentation of Brugada syndrome is characterized by a male predominance with syncope or sudden death, mostly at an average age of 40 years. We report 2 male patients, in whom the diagnosis was made at the ages of 71 and 74 years, respectively. They presented with aborted sudden death or repeated syncope. They had coved-type Brugada ECG, and one of them had positive procainamide provocative test. Both received implantation of a cardioverter defibrillator. One had recurrent events and received ICD discharge within 4 weeks after implantation. Our study showed that Brugada syndrome could occur in the elderly, and its clinical presentations were as severe as those in younger patients.

Key Words: Brugada syndrome • Elder population • Ventricular fibrillation • Sudden cardiac death

INTRODUCTION

In 1992, Brugada and Brugada reported a group of patients with recurrent polymorphic ventricular tachycardia (VT) leading to cardiac arrest. They had an electrocardiographically (ECG) specific pattern of right bundle branch block (RBBB) and ST segment elevation in leads V1-3 with normal QT interval and no structural heart disease.1 Brugada syndrome is a major cause of sudden cardiac death due to ventricular arrhythmia in young men with no evidence of structural heart disease and more commonly observed in Asian countries than in the United States and European countries.2,3 The manifestation of Brugada syndrome ranges widely. Patients may be asymptomatic but may also die suddenly. Sudden death is the first symptom of the disease in some cases, and rapid polymorphic VT may be seen during monitoring. Self–terminating episodes of ventricular tachyarrhythmias typically lead to repeated episodes of syncope. The mean age of the first appearance of arrhythmic event is at the age of 40 years (1-77 years).1,4 Subjects in many published studies or patients in clinical observation were mostly around 30 to 50 years. However, little information was available on the geriatric population. In 2004, we found 2 patients who were older than 70 years old when they were identified as Brugada syndrome. The clinical characteristics and follow-up information in these 2 symptomatic patients are described in this report.

CASE REPORT

We summarized the clinical characteristics of these 2 patients in Table 1. The average ages at the time of diagnosis were 71 and 74 years, respectively. They were both old men who had been apparently healthy before the index event. There were no similar episodes of syncope or sudden cardiac death recognized in their family members. Patient 1 had experienced repeated syncope up to
3–5 times per year. Patient 2 presented with seizure and circulatory arrest requiring cardiopulmonary resuscitation during the first event, which attacked in the early morning. After detailed medical history review, no particular predisposing factors or a history of special drug abuse or medication such as tricyclic antidepressants or cocaine could be identified at the time of the first cardiac arrest or syncope. Physical examinations were all unremarkable. During hospitalization after resuscitation, neither patient had significant biochemical abnormalities that could be associated with the arrhythmic event.

Both of them showed a coved-type ST segment elevation in leads V1–V2 on the 12-lead ECG, which was not taken immediately after the index event (Figure 1). They were in sinus rhythm before onset of the index arrhythmia, but did not have prolonged PR interval (0.160–0.168 sec). Neither patient had a right axis QRS deviation in the frontal plane (+96 and +63, respectively). Repolarization was abnormal, as expressed by ST segment elevation (at least 0.2 mV) in leads V1 to V2 or V3. The QT interval and the rate-corrected QT (QTc) interval were within normal limits (397 ms and 375 ms, respectively). The 2 patients received signal-averaged ECG, and one of them had positive results for late potentials. A 24-hour Holter recording was obtained, and showed no significant tachy- or brady-arrhythmias recorded other than isolated rare premature atrial complexes in patient 1. Echocardiography and MRI did not

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**Figure 1.** Resting standard 12-lead ECG showed a type I (coved-type) Brugada ECG in patients 1(A) and 2(B), respectively.

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**Table 1.** Clinical profiles of the 2 patients with Brugada syndrome

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr) at onset</th>
<th>Gender</th>
<th>Circumstance</th>
<th>Presenting symptoms</th>
<th>Documented arrhythmias</th>
<th>Family history</th>
<th>EP study</th>
<th>SAECG</th>
<th>MRI</th>
<th>Provocative test</th>
<th>24-hr Holter</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>71</td>
<td>M</td>
<td>Rest</td>
<td>Repeated syncope</td>
<td>VT</td>
<td>None</td>
<td>Short run VT</td>
<td>Positive</td>
<td>Negative</td>
<td>Positive</td>
<td>Rare SVEs</td>
</tr>
<tr>
<td>2</td>
<td>74</td>
<td>M</td>
<td>Asleep</td>
<td>Seizure/SCD</td>
<td>Nil</td>
<td>None</td>
<td>Nil</td>
<td>Nil</td>
<td>Negative</td>
<td>Negative</td>
<td>Nil</td>
</tr>
</tbody>
</table>

EP study = electrophysiologic study; M = male; MRI = magnetic resonance image; NA = not available; SAECG = signal-averaged electrocardiography; SCD = sudden cardiac death; SVE = supraventricular event; VT = ventricular tachycardia.
disclose structural heart disease such as ventricular dysplasia, hypertrophic or dilated cardiomyopathy or other forms of abnormal heart disease.

Cardiac catheterization did not reveal any coronary artery disease in the 2 patients. Patient 1 underwent a complete electrophysiologic study. However, only non-sustained polymorphic VT was induced during programmed ventricular stimulation. The coved-type Brugada ECG in patient 1 disappeared several days later. He received a pharmacological provocative test, which showed a positive result with prominent J wave after intravenous injection of 1 gm of procainamide (Figure 2).

Both patients had been implanted with an implantable cardioverter defibrillator (ICD). Patient 1 had first time of ICD discharge within 4 weeks after implantation. Several episodes of VT (cycle length 320–380 ms) were recorded and successfully converted by intracardiac defibrillation of ICD (Figure 3). Those episodes seemed to be subsequently controlled by quinidine (100 mg) 1#

Figure 2. Pharmacological provocative test in patient 1. Note J wave, ST segment and T-wave change in leads V1-3 (arrows) before and after intravenous procainamide 1000 mg.

Figure 3. ICD recorded one episode of recurrent ventricular arrhythmias followed by successful DC shock in patient 1.
bid. Patient 2 received ICD implantation and has been asymptomatic without taking antiarrhythmic drugs during follow-up.

**DISCUSSION**

The clinical presentation of Brugada syndrome is characterized by a male predominance and the appearance of arrhythmic events at an average age of 40 years (1-77). Since June 1997, we have identified 10 male patients with the diagnosis of Brugada syndrome and mean age of 46 ± 7 years (36-61) from 6 hospitals in Taiwan. In April 2004, we identified 2 elderly patients (71 and 74 years) with Brugada syndrome, who had been apparently healthy before the index event. This report demonstrated that Brugada syndrome could occur in the elderly patient even older than 70 years, although most studies reported an average age of 40 years at the time of index event. Thus, we have learned that the diagnosis of Brugada syndrome should still be considered in patients older than 70 years, who present with sudden death or syncope.

The clinical presentations of Brugada syndrome vary widely from sudden cardiac death, seizure, recurrent syncope to an isolated ECG abnormality in asymptomatic individuals. In this report, the clinical presentations of the 2 patients were as severe as those in younger patients. One had aborted cardiac arrest and required cardiopulmonary resuscitation when the first event attacked, and the other presented with frequent attacks of syncope. Transient or non-sustained VT/VF may be the reason for presentation with syncope. Moreover, the patient with syncope had recurrent episodes of index event just within 1 month after diagnosis was confirmed. These events were successfully corrected by ICD discharges. Atarashi et al. have reported that younger patients with Brugada syndrome are at a higher risk of sudden death while those aged above 50 years tend to remain asymptomatic. However, the 2 cases demonstrate that ventricular arrhythmia can occur in the elderly as well, and hence the risk stratification must not be done solely based on age. Although there are limited case numbers in this report, the frightening presentation may also imply that the severity of manifestation of Brugada syndrome is not less severe as the age of onset increases.

The ECG can normalize intermittently in up to 40% of cases, a feature that can make the diagnosis of Brugada syndrome difficult. Sodium channel blocking agents such as ajmaline, procainamide, flecainide, and propafenone can accentuate the changes and can be used as a diagnostic test. A positive pharmacological provocative test was found in patient 1.

A positive signal-averaged electrocardiogram has been reported to be useful for screening patients who are at a high risk for the induction of sustained VT/VF by programmed electrical stimulation. However, patient 1, who had a positive signal-averaged ECG, had only inducible non-sustained VT.

The prognosis of patients with symptomatic Brugada syndrome is poor unless they are treated. Mortality in symptomatic patients is thought to be up to 10% per year. Patient 1 had recurrent episodes that were successfully converted by ICD discharge 4 weeks after implantation. So far, drug treatment is not effective, but the implantation of an ICD has been shown to prevent sudden death.

**CONCLUSIONS**

To our best knowledge, this is the first case report of Brugada syndrome and its characteristics in the elderly population in Taiwan. This report disclosed that Brugada syndrome could be a disease in elderly patients older than 70 years, although most studies reported an average age of 40 years at the time of index event. The 2 cases demonstrated that ventricular arrhythmia could occur in the elderly patient as well, and hence the risk stratification must not be done solely based on age.

**REFERENCES**


台灣老人的 Brugada 症候群 — 兩病例報告

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Brugada 症候群是一種可能致死的心室性心律不整疾病。它在心電圖 V1-3 上會有特殊的 ST 節段上升, 但 QT 間距正常且無任何心臟結構問題。臨床表現可突然暈厥或猝死，平均發病年齡在四十歲左右，以男性為多。在這裡，我們報告了兩個 Brugada 症候群的老人案例，分別是 71 歲及 74 歲。一個是突然心臟停止，另一個是反覆暈厥。兩個都帶有 Coved 型 Brugada 心電圖，其中一個接受 Procainamide 誘發試驗呈陽性反應。他們都有裝置體內去顫器，其中一個在裝置後四個禮拜內復發，但都經體內去顫器成功救治。從我們的報告可以看出 Brugada 症候群即使在大於 70 歲的老人仍可出現，且臨床表現和年輕人的 Brugada 症候群一樣嚴重。

關鍵詞：Brugada 症候群、老年人族群、心室顫動、突發性心因性猝死。