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# Brugada syndrome associated with supraventricular tachycardia

## Diagnostic and therapeutic strategies

### Introduction

Brugada syndrome is an arrhythmogenic disease characterized by ST segment elevation in the right precordial leads. The syndrome is rarely associated with supraventricular tachycardias (SVT) and most commonly with ventricular tachyarrythmias leading to sudden cardiac death [1, 2, 3].

We report the case of a patient with Brugada syndrome and a history of palpitations who presented with syncope and developed atrioventricular nodal reentrant tachycardia (AVNRT) at the electrophysiological study. We describe and discuss the diagnostic and therapeutic strategies which may prove useful for clinicians.

### Case report

A 38-year-old man was admitted to our department due to an episode of syncope. The preceding symptoms included tachycardia and palpitations. The patient reported several asymptomatic and short-lasting, self-terminated episodes of tachycardia. Since his adolescence none of these episodes had been documented by ECG. Of note, the frequency of the episodes had increased remarkably during the preceding year. The patient was under no medication and had no family history of sudden cardiac death or any other cardiac disease.

The ECG on admission demonstrated sinus rhythm with mild saddle-back-like ST elevation, mostly evident in lead $V_2$ (**Fig. 1**). Physical examination and laboratory studies were unremarkable. The echocardiogram showed a structurally and functionally normal heart, ECG Holter monitoring demonstrated no evidence of arrhythmia or conduction disturbances, the tilt test was negative and coronary angiography revealed normal coronary arteries. The neurological examination and brain imaging with computed tomography were also normal. The episode of syncope along with the presence of saddle-back-like ST elevations in right precordial leads led us to suspect Brugada syndrome. To confirm diagnosis of the syndrome, the patient underwent a procainamide test which transformed the ST elevations in precordial leads to a pattern that typically occurs in type-I (coved-type) Brugada syndrome (**Fig. 2**).

On the basis of the history of tachycardia the patient underwent an electrophysiological study in an attempt to identify the type of tachycardia. A well-tolerated atrioventricular nodal reentrant tachycardia (AVNRT) was induced (**Fig. 3 and Fig. 4**), but no episode of ventricular tachycardia or fibrillation was induced up to three ventricular extras under basic condition and isoproterenal infusion. The AVNRT was successfully treated with ablation of the slow pathway. Since the patient had a history of syncope and Brugada syndrome the implantation of a cardioverter defibrillator (ICD) was decided upon for the prevention of potentially lethal ventricular tachyarrhythmias.

The patient was discharged in a good clinical condition. At 18 months following ICD implantation an electrical storm with three consecutive ventricular fibrillation episodes occurred followed by appropriate ICD discharges (**Fig. 5**). These episodes were triggered by a febrile status due to pneumonia.

### Discussion

In the current report, we present the case of a patient with Brugada syndrome and a history of tachycardia who presented with syncope and developed atrioventricular nodal reentrant tachycardia (AVNRT) at electrophysiological study [1, 2, 3].

The association of Brugada syndrome with SVT was first described in 2001 [3]. A recent multicentre study demonstrated that 23% of Brugada patients present with supraventricular tachycardias, including AVNRT (7%), atrioventricular reentrant tachycardia (2%), atrial tachycardia (3%) and atrial fibrillation or flutter (11%) [4]. Although the pathophysiological basis of the occurrence of SVT in Brugada patients is not known, one could speculate that the heterogeneity

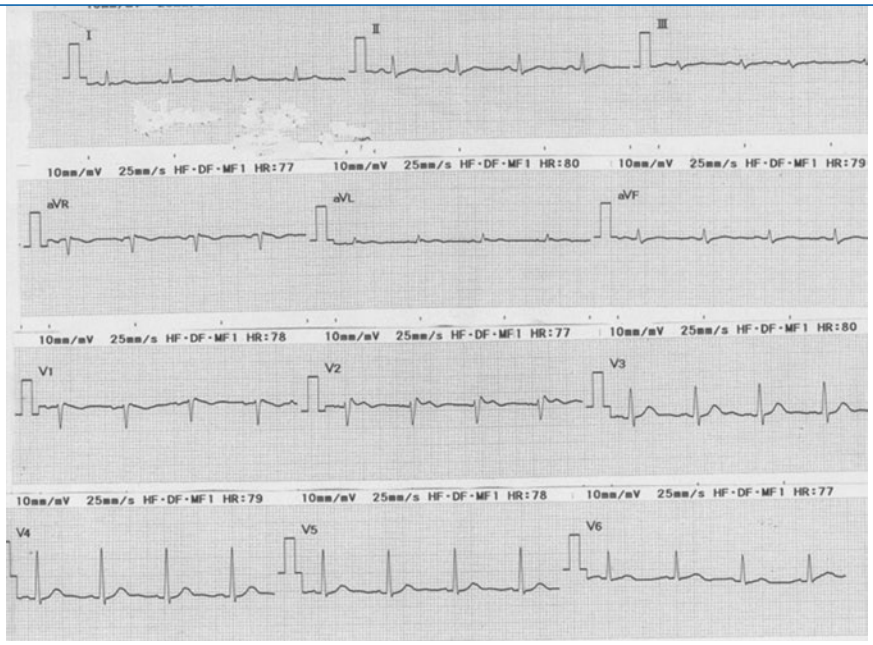


**Fig. 1** ▲ ECG on admission showing a type-II pattern of Brugada syndrome in lead $V_2$

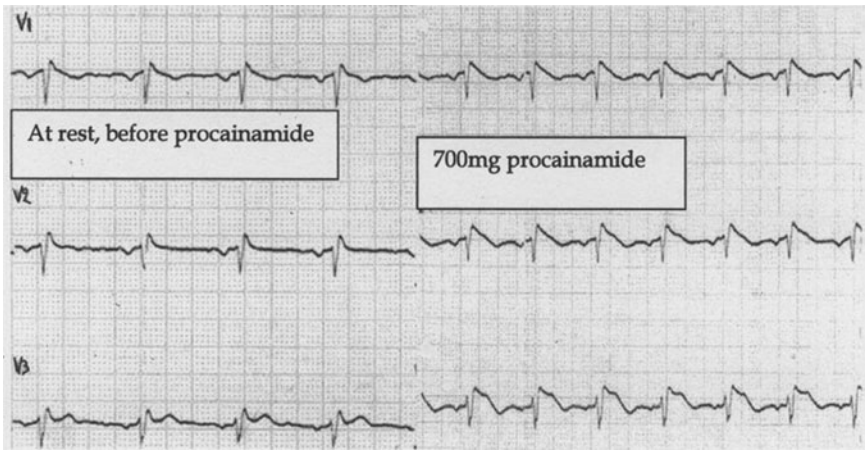


**Fig. 2** ▲ ECG after administration of 700 mg procainamide revealing a type-I pattern of Brugada syndrome in leads $V_{1-3}$

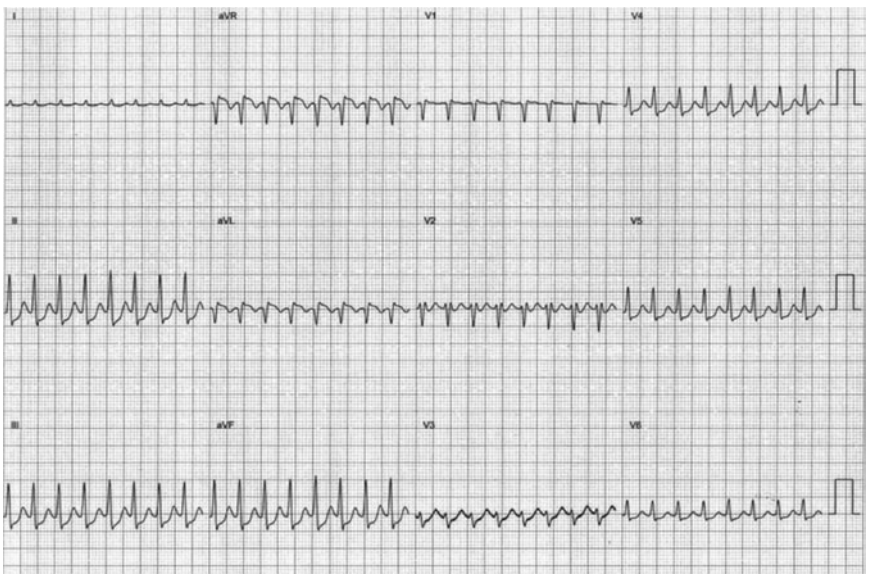

**Fig. 3** ▲ ECG of the atrioventricular nodal reentrant tachycardia (AVNRT)

## Abstract · Zusammenfassung

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### Brugada syndrome associated with supraventricular tachycardia. Diagnostic and therapeutic strategies

**Abstract**
We report the case of a patient with Brugada syndrome and a history of palpitations who presented with an episode of syncope and developed supraventricular tachycardia in the electrophysiological study. The patient was treated with radiofrequency ablation for the supraventricular tachycardia and an implantable cardioverter defibrillator for the Brugada syndrome. At 18 months following implantation of the defibrillator an electrical storm with ventricular fibrillation episodes occurred followed by appropriate discharges of the defibrillator.

**Keywords**
Brugada syndrome · Tachycardia, supraventricular · Catheter ablation, radiofrequency · Implantable cardioverter-defibrillators · Electrophysiologic techniques, cardiac

### Brugada-Syndrom in Verbindung mit supraventrikulärer Tachykardie. Strategien für Diagnose und Therapie

**Zusammenfassung**
Wir berichten über einen Patienten mit Brugada-Syndrom und anamnestisch bekannten Palpitationen, der sich nach einem synkopalen Ereignis vorstellte. In der elektrophysiologischen Untersuchung entwickelte sich eine supraventrikuläre Tachykardie. Wir führten eine Radiofrequenzkatheterablation durch wegen der supraventrikulären Tachykardie; wegen des Brugada-Syndroms implantierten wir einen Kardioverter-Defibrillator (ICD). Achtzehn Monate nach ICD-Implantation kam es zu einem elektrischen Sturm mit Kammerflimmerepisoden, die von konsekutiven Entladungen des Defibrillators terminiert wurden.

**Schlüsselwörter**
Brugada-Syndrom · Supraventrikuläre Tachykardie · Radiofrequenzkatheterablation · Implantierbarer Kardioverter-Defibrillator · Elektrophysiologische Techniken

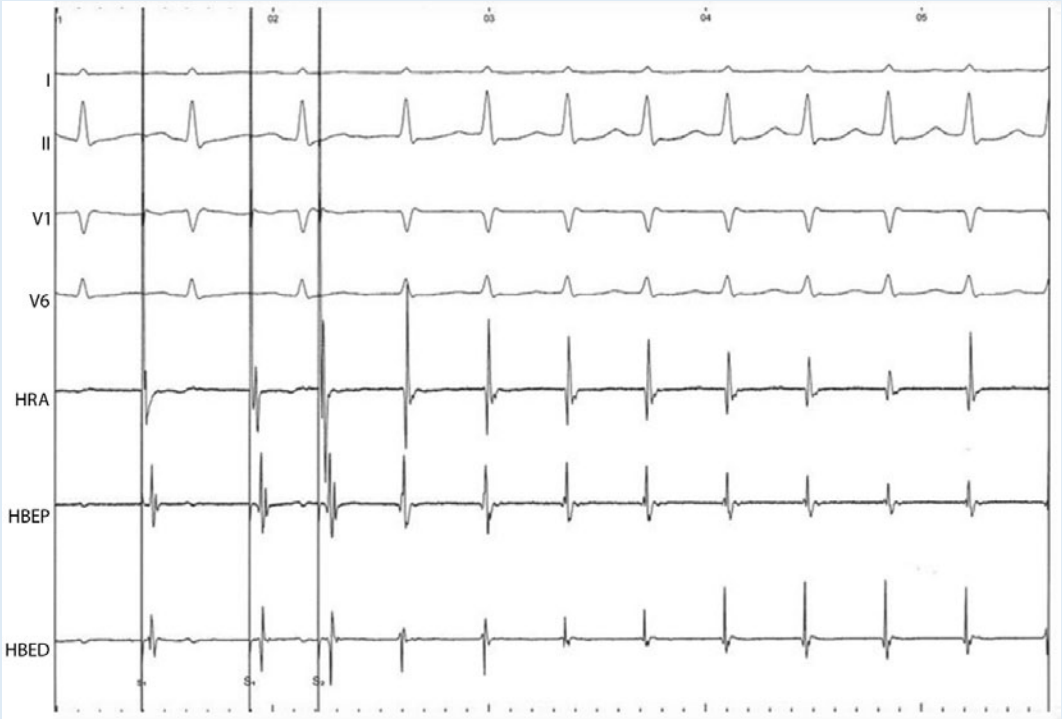


**Fig. 4** ◀ Electrophysiological study showing induction of atrioventricular nodal reentrant tachycardia (AVNRT) by an atrial extrasystole. *HRA* High right atrium electrogram, *HBEP* His bundle electrogram proximal, *HBED* His bundle electrogram distal

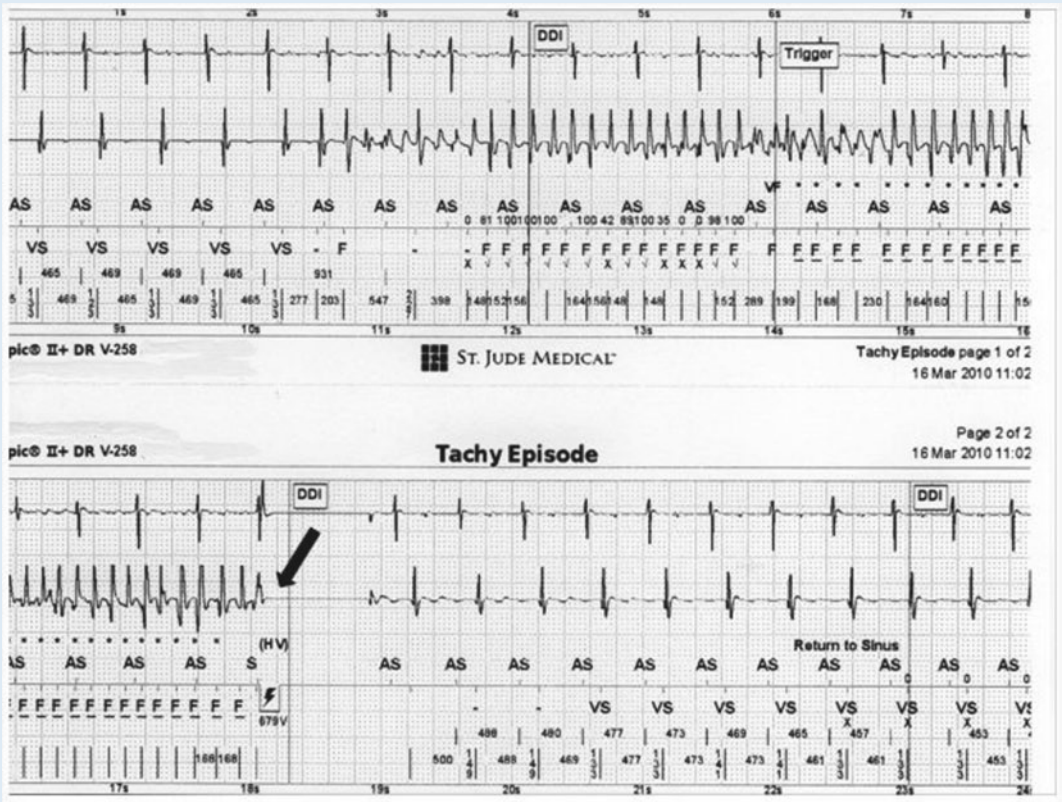


**Fig. 5** ◀ ECG taken from ICD recordings showing an episode of ventricular fibrillation successfully terminated (*black arrow*) by an appropriate shock from the ICD (*first row* atrial electrograms, *second row* ventricular electrograms, *third row* event markers)

in ventricular repolarization that occurs in Brugada syndrome is also extended to the atria and atrioventricular node [3]. Further experimental studies are warranted to elucidate the mechanisms responsible for the development of SVT in Brugada syndrome.

Intriguingly, our patient's history of tachycardia is most likely attributed to self-terminated episodes of AVNRT, which were also reproduced at electrophysiological study. However, it seems that the AVNRT was not responsible for the episode of syncope since the inducible AVNRT was very well-tolerated during the electrophysiological study. It is very likely that the syncope occurred as a result of a ventricular tachyarrhythmia in the setting of Brugada syndrome. Therefore, Brugada syndrome should always be considered in young patients with structurally normal hearts reporting palpitations or syncope [3].

According to the report of the Second Consensus Conference on Brugada syndrome and the guidelines for device-based therapy, ICD implantation is the most appropriate therapeutic strategy in patients with Brugada syndrome and a history of syncope (class IIa, level of evidence C) [1, 5, 6]. The rationale for ICD implantation in our patient was justified at follow-up when, in the setting of a febrile status due to pneumonia, the patient experienced multiple ventricular fibrillation episodes provoking appropriate ICD discharges. Febrile state is a well-known trigger that unmasks Brugada syndrome [7, 8].

The performance of an electrophysiological study in Brugada syndrome remains controversial since it does not contribute to the diagnosis or risk stratification of the syndrome (class IIb). We would like to emphasize that an electrophysiological study is recommended whenever SVT is suspected [5, 9]. The suspicion of potential SVT was considered in our patient due to a long history of short-lasting episodes of tachycardia without ECG recording confirmation. Upon coexistence of SVT in Brugada patients who are candidates for ICD, the SVT should be treated before ICD implantation, since inappropriate shocks due to SVT are a common complication of ICD therapy [10]. Radiofrequency ablation is the only therapeutic option for SVT in Brugada patients as antiarrhythmic drugs such as β-blockers, verapamil and propafenone are relatively contraindicated in such patients since they may unmask an underlying Brugada syndrome [4].

In conclusion, we report a case of a young patient with a structurally normal heart and a history of tachycardia who presented with an episode of syncope. On the basis of the medical history and ECG findings the diagnosis of Brugada syndrome was established. Prior to ICD implantation an electrophysiological study was performed which revealed an inducible SVT that was treated successfully with ablation.

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**Conflict of interest.** The corresponding author states that there are no conflicts of interest.

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