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# Syncope in a Young Man

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**ABSTRACT**

**Introduction:** Syncope is a common complaint in the emergency department, accounting for 1%–3% of all visits and hospital admissions. Most causes are often benign and self-limited allowing safe discharge. However, others, particularly those resulting from cardiac syncope, are associated with significant morbidity and mortality. An electrocardiogram (ECG) is essential in addition to clinical history and physical examination for risk stratification. It is useful for identifying patients with inherited syndromes that predispose them to cardiac arrhythmias.

**Case Report:** We report a case of a young man who presented with syncope to the emergency department. He was asymptomatic on arrival, but subsequently developed seizures secondary to ventricular fibrillation that required defibrillation. His initial ECG revealed characteristics of type 1 Brugada syndrome. The patient was fitted with an internal cardiac defibrillator and was discharged. We discuss the presentation, pathophysiology, and management of patients with Brugada syndrome.

**Conclusion:** In patients presenting with syncope, ECG is essential for risk stratification and identifying patients with possible inherited syndromes. A high index of suspicion is necessary for diagnosing

**Keywords:** Syncope, Brugada syndrome, electrocardiogram

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## Introduction

Syncope is a common presenting complaint in the emergency department (ED), accounting for 1%–3% of all hospital admissions. Although most causes are often benign and self-limiting, allowing for safe discharge, others particularly those resulting from cardiac syncope are associated with significant morbidity and mortality. An electrocardiogram (ECG) is an essential component of a clinical workup for syncope as it aids in risk stratification. It is useful in identifying patients with inherited syndromes that predispose them to cardiac arrhythmias. Here we report a case of a young man who presented with syncope and was subsequently diagnosed with Brugada syndrome. Consent was not obtained as there was no patient identifier.

## Case Report

A 31-year-old Bangladeshi man with no medical history of note was brought into the ED by his friends. He had an episode of syncope while driving. His vital signs on arrival were blood pressure, 116/70 mmHg; respiratory rate, 16/min; oxygen saturation, 100% on room air, and temperature, 36°C. The patient was slightly drowsy but arousable. Physical examination was unremarkable. His blood glucose level was 8.9 mmol/L. ECG was performed on arrival (Figure 1).

Shortly after arrival, the patient developed two episodes of tonic-clonic seizures that stopped spontaneously. He was immediately intubated for airway protection. Repeat ECG showed atrial fibrillation with ST depression in anterolateral leads (Figure 2).

A few minutes post-intubation, he developed two episodes of ventricular fibrillation (VF), both of which responded to biphasic defibrillation of 150 J. Slow intravenous bolus of 150 mg amiodarone (cordarone; Wyeth Pharmaceuticals Inc.) was given fol-

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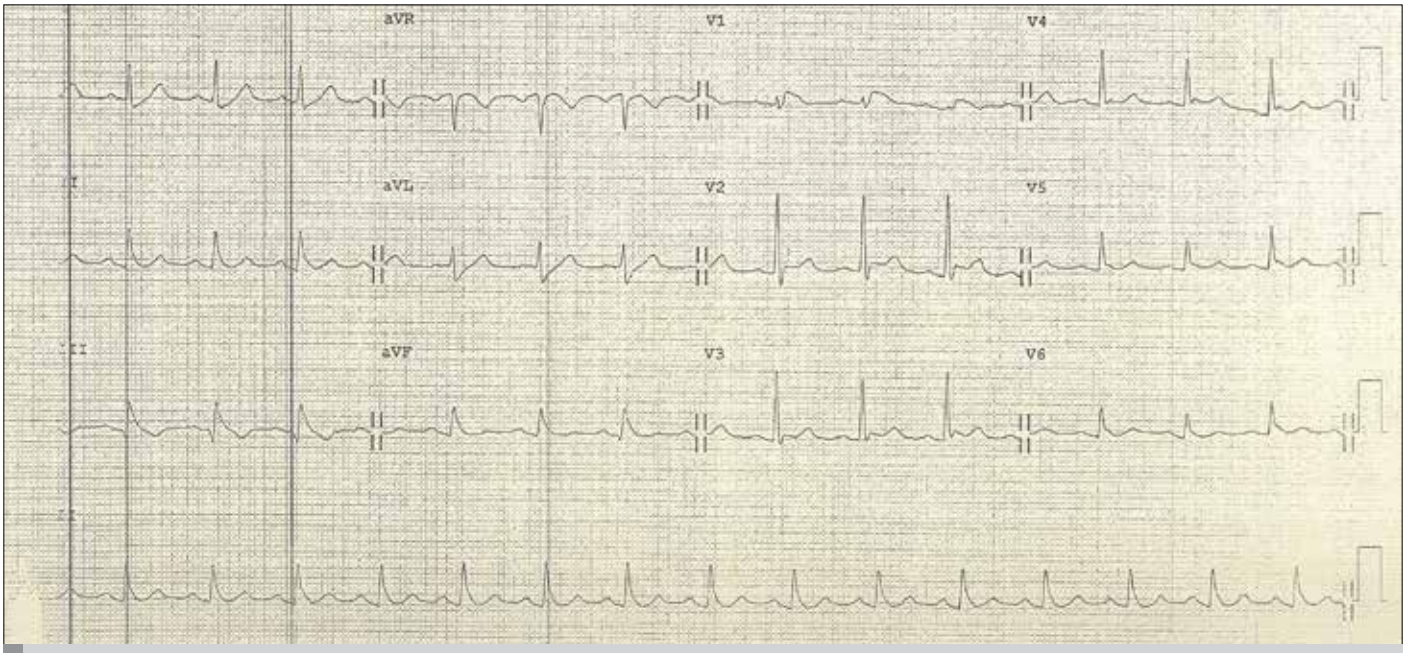


FIGURE 1. Initial ECG on arrival

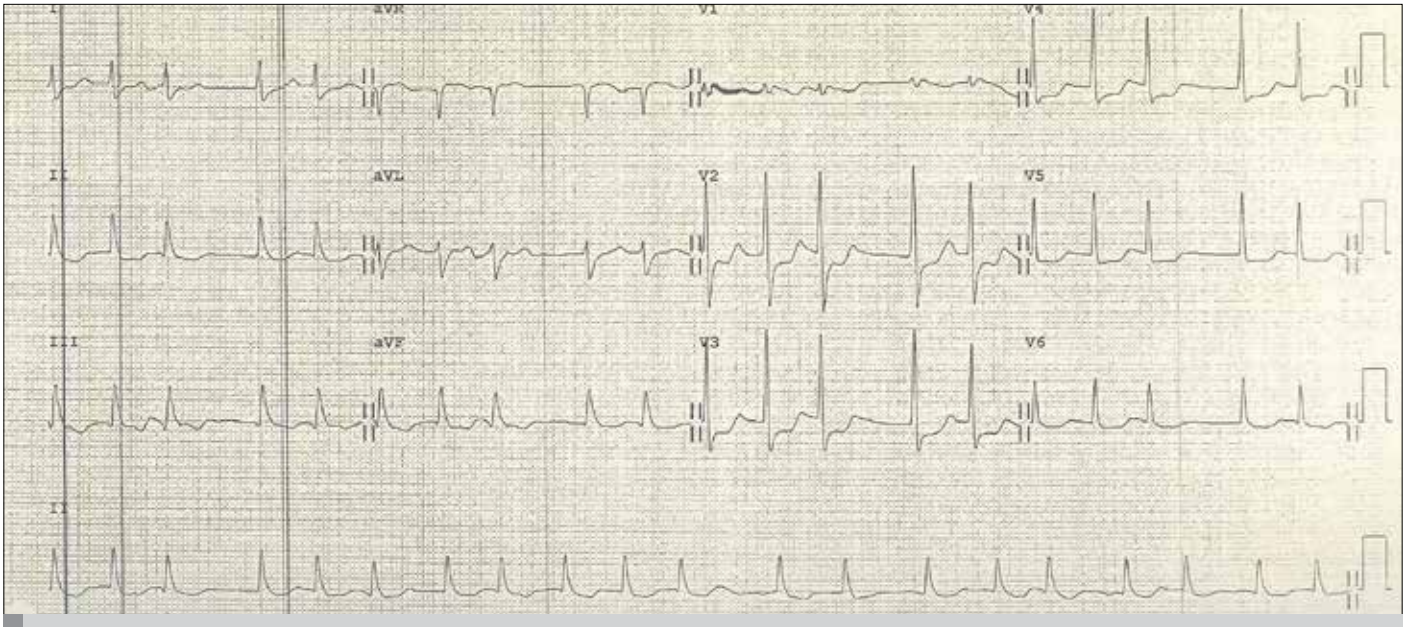


FIGURE 2. Atrial fibrillation with ST depression in anterolateral leads

lowed by infusion of a further 300 mg over 8 h. Laboratory investigations including full blood count, renal panel, cardiac enzyme levels, and arterial blood gas levels were unremarkable. Once stabilized, the patient was sent for emergent computed tomography of the brain which was also normal. He was successfully extubated the following day. An echocardiogram done later did not reveal any abnormalities. The first ECG demonstrated an RsR' pattern in V1. This is typical of a type 1 Brugada pattern. We presumed that his initial seizure-like activity was likely due to self-terminating ventricular tachyarrhythmia leading to cerebral hypoperfusion. We did not capture this as the patient did not undergo cardiac monitoring. The presence of ventricular tachyarrhythmia and type 1 Brugada pattern on ECG in the absence of structural heart disease establishes the diagnosis of Brugada syndrome. The patient was fitted with an internal cardiac defibrillator (ICD) and was discharged thereafter.

## Discussion

Brugada syndrome was first recognized as a clinical entity in 1992 by Brugada et al (1). It is inherited in an autosomal dominant manner with variable expression. In 1998, the SCN5A gene was identified in patients with the Brugada syndrome and further genetic testing has indicated the presence of this gene in 18%–30% of these (2).

It is more prevalent in Asian populations with a typical patient being a male of Asian descent in his early 40s (3, 4). The syndrome is characterized by ECG changes, a predisposition to VF, and sudden cardiac death (SCD) in the absence of underlying structural abnormalities. The underlying pathophysiology is a channelopathy that involves ionic imbalances between inward and outward currents of the phase 1 of the action potential (5). Abnormal prolongation or shortening of the action potential predisposes the patients to reentry phenomena and arrhythmias.

ECG changes are characterized by ST segment elevation and T-wave inversion in the right precordial leads in the presence of a structurally normal heart and a normal QT interval. Three distinct ECG patterns have been identified.

Type 1 is characterized by "coved" ST elevation in the right precordial leads. Type 1 ECGs in themselves are described as an idiopathic Brugada ECG pattern. To diagnose type 1 Brugada syndrome, an ECG pattern in addition to one of the criteria listed below is needed:

1. Documented ventricular tachycardia (VT)
2. Self-terminating polymorphic VT
3. Electrophysiological inducibility of VT
4. Family history of SCD at <45years
5. Type 1 coved ST segment elevation in family members
6. Unexplained syncope suggestive of a tachyarrhythmia
7. Nocturnal agonal respiration

Type 2 and type 3 patterns involve the same right precordial leads with the main distinction being a slightly different morphological appearance. Type 2 and 3 are described as "saddle-back" because of an upright T-wave. Types 2 and 3 have ST segment elevations of >1 and <1 mm, respectively. However, both type 1 and type 2 ECG pattern changes are insufficient, by themselves, for diagnosing Brugada syndrome. When a high degree of suspicion is present (patients fulfilling at least one of the features described above), a test is performed with a sodium channel blocker to see if the ST segment changes to a type 1 morphology. ST segment changes must be present in at least one lead (6).

Most patients remain asymptomatic until an arrhythmic event, although 23% patients have a history of syncope preceding their diagnosis (7). If episodes of VT or VF are self-terminating, they may have a history of palpitations and dizziness or present to the hospital with syncope or a seizure; as in the case of our patient.

Typical ECG ST segment elevations vary in amplitude and morphology and they have been known to be transient, making the diagnosis more difficult. ECGs may normalize or fluctuate between the different types (8, 9). Physicians should maintain a high index of suspicion, carefully examine ECG, monitor for dynamic changes, and check for the presence of other diagnostic criteria. Atrial fibrillation is known to affect up to 20% patients and has been associated with a higher risk of VF in certain cases (10).

Fever, excessive alcohol intake, and large meals may unmask a type 1 ECG pattern and predispose the patient to VF. Certain drugs including anti-arrhythmics, psychotropics, and cocaine that may induce ST elevation in right precordial leads should be avoided in patients with known Brugada syndrome (11).

Brugada syndrome is a potentially fatal condition. In total, 62% patients who survive an arrhythmic event will have another event in the following 54 months (12). The 2005 Brugada consensus report recommends risk-stratifying asymptomatic patients for determining who need to be fitted with ICD. These include those with spontaneous type 1 ECGs who have a positive electrophysiology study (EPS) and those with drug-induced type 1 ECGs with a positive EPS and a family history of SCD (6).

Internal cardiac defibrillator implantation in symptomatic patients remains the only effective treatment for reducing the risk of SCD (13). It is recommended in patients with documented VF or VT and in patients who present with a spontaneous type 1 ECG and a history of syncope (14). No single pharmacological treatment exists as yet. Antiarrhythmic drugs may be used as adjunctive therapy in those with frequent ICD discharges. Isoproterenol or quinidine should be considered for electrical storms associated with Brugada syndromes, though beta-adrenergics and amiodarone have also been used (14, 15).

Finally, given the recognized genetic component of Brugada syndrome, family members of asymptomatic patients with Brugada pattern on their ECGs should also be screened.

## Conclusion

This case highlights the importance of considering consider malignant cardiac arrhythmias in patients presenting with syncope and seizures. ECG remains essential in the initial evaluation for identifying potential and specific causes of syncope (16). Emergency physicians must maintain a high index of suspicion to identify inherited conditions when patients present to the ED. Early diagnosis and appropriate management of these potentially fatal conditions reduces morbidity and mortality.

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