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Brugada Pattern Type II: A Case Report Highlighting Clinical Challenge in the Early Recognition of Brugada Syndrome

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ABSTRACT
Background: Brugada syndrome (BrS) is a genetic cardiac channelopathy associated with an increased risk of malignant arrhythmias and sudden cardiac death (SCD), particularly in individuals without structural heart disease. The condition is characterized by specific electrocardiographic (ECG) patterns, with type I showing coved-type ST-segment elevation and type II showing saddleback morphology. Differentiating BrS from Brugada phenocopy (BrP), which is induced by reversible conditions such as electrolyte disturbances, remains a clinical challenge, especially in patients presenting with atypical features. Objective: This case report aims to describe the clinical presentation, diagnosis, and management of a patient with a type II Brugada ECG pattern. Method: A 38-year-old male presented with chest pain, diaphoresis, and syncope. Electrocardiography, laboratory tests, cardiac monitoring, and echocardiography were performed. Results: ECG revealed a saddleback ST-segment elevation ≥2 mm in lead V2, consistent with the type II Brugada pattern. Laboratory findings, including electrolytes and troponin I, were normal. Cardiac monitoring recorded premature ventricular contractions. Echocardiography showed normal cardiac function. The Brugada pattern persisted on follow-up ECGs. Conclusion: Early recognition of the type II Brugada pattern is crucial in patients with syncope, even with normal electrolytes. Referral for electrophysiological studies and consideration of an ICD is essential to prevent SCD.

Keyword: *Brugada syndrome; implantable cardioverter-defibrillator (ICD); saddleback ST-segment; sudden cardiac death; Brugada phenocopy*

INTRODUCTION

The electrocardiogram (ECG) is one of the most frequently utilized diagnostic tools by general practitioners in both hospital and outpatient clinic settings. The presence of an ST-segment elevation on an ECG may raise suspicion of myocardial infarction (MI) (Alrawashdeh et al., 2025; Gao, Wang, & Yang, 2025; Ricci et al., 2025). However, other potential etiologies must be considered, including pericarditis, early repolarization, left bundle branch block, ventricular aneurysm, and Takotsubo cardiomyopathy. An additional, critical cause of ST-segment elevation is Brugada syndrome (BrS).

Brugada syndrome (BrS) is a cardiac arrhythmia arising from a sodium channel disorder (channelopathy) that manifests in characteristic electrocardiographic (ECG) findings (Ciabatti et al., 2025; Narasimhan et al., 2025; Xie, Chen, Li, Sun, & Chen, 2025). Specifically, type 1 BrS is characterized by an ST-segment elevation with a coved morphology, while type 2 BrS is marked by a saddleback morphology. BrS is classified as an autosomal dominant disorder and has been shown to carry an elevated risk of sudden cardiac death (SCD) in the absence of structural heart disease (Abdelaal, Shehata, Ali, & Abdel Moawed, 2025; Lovrić Benčić & Levicki, 2025; Montuoro et al., 2026). This condition is of particular concern due to its association with malignant arrhythmias. In contrast, Brugada phenocopy (BrP) is an electrocardiographic manifestation that mimics hereditary BrS but is elicited by reversible clinical conditions, such as metabolic disturbances, including hypokalemia (Alnajjar, Ibrahim, & Ellebedy, 2025). The hallmark of BrP is the restoration of the ECG pattern to its baseline

state following the correction of the underlying cause. Therefore, early diagnosis and differentiation are crucial strategies for preventing mortality (Albinni et al., 2025; Owusu et al., 2025).

Globally, the prevalence of BrS is estimated at 1–5 per 10,000 individuals, with the highest prevalence observed in Southeast Asia at ≥ 3.5 per 1,000 (Khawaja et al., 2023; Vutthikraivit et al., 2018). The prevalence of BrS in the male population is 8–10 times higher than in the female population, with initial symptoms typically presenting in the third or fourth decade of life (Jespersen et al., 2023). However, cases have been documented in children and elderly individuals; the youngest reported case was two days old, and the oldest was 84 years old. This condition accounts for more than 10% of sudden death cases and more than 20% of sudden death cases in individuals with structurally normal hearts.

This report describes a rare type II Brugada ECG pattern in a 38-year-old male who exhibited symptoms of sharp left-sided chest pain, accompanied by diaphoresis and syncope, in the absence of electrolyte imbalances (Lowenstein, 2018). The case is particularly noteworthy as it occurred in a patient with normal electrolyte levels, whereas Brugada patterns are frequently associated with severe electrolyte disturbances (e.g., K+ <2.6 mmol/L), as reported in the literature (Amusina, Mehta, & Nelson, 2022; Edi, Aminanto, & Paranita, 2025; Imowanto, Abdullah, & Wahyuni, 2026). The authors present this case to raise awareness among primary care physicians, highlighting the importance of accurate ECG interpretation and the distinction between Brugada syndrome and Brugada phenocopies to mitigate the risk of sudden cardiac death.7

The primary objective of this case report is to describe the clinical presentation, diagnostic process, and management of a patient with a type II Brugada ECG pattern, emphasizing the challenges in differentiating Brugada syndrome from Brugada phenocopy in the absence of electrolyte disturbances. This report aims to contribute to the limited body of literature documenting type II Brugada patterns with atypical clinical presentations, particularly in patients with normal electrolyte levels. The benefits of this study include enhancing clinical awareness among healthcare providers, especially in primary care and emergency settings, about the importance of recognizing subtle ECG findings that may indicate underlying Brugada syndrome. Additionally, this case report underscores the necessity of comprehensive diagnostic evaluation, including consideration of provocative testing and electrophysiological studies, to guide appropriate management and prevent life-threatening outcomes such as sudden cardiac death. By documenting this rare presentation, the authors hope to support clinical education and inform future research on optimal diagnostic and therapeutic strategies for Brugada syndrome.

METHOD

Case Report

A 38-year-old male was admitted to the emergency department with complaints of sharp, stabbing, and burning chest pain (VAS 8) radiating to the jaw and left shoulder. These symptoms were accompanied by dyspnea and diaphoresis, without palpitations. The onset of symptoms occurred one hour prior to admission. The patient reported experiencing milder, intermittent chest pain (VAS 3) over the past week, which was exacerbated by physical activity

and relieved by rest. His medical history included gastroesophageal reflux disease (GERD); however, he did not take medication regularly as symptoms were infrequent. The patient also had a history of hypertension and hypercholesterolemia but was non-compliant with regular treatment. He denied a personal history of heart disease or a family history of sudden cardiac death. The patient is a current smoker, consuming one pack of cigarettes every 2–3 days, and reported engaging in irregular daily jogging. While en route to the hospital, the patient experienced a brief episode of syncope but regained consciousness following painful stimuli.

Physical examination revealed a blood pressure of 125/64 mmHg, a heart rate (HR) of 55 bpm, a respiratory rate (RR) of 16 breaths per minute, an oxygen saturation (SpO_2) of 97% on room air, and a body temperature of 36.8°C; extremities were warm and well-perfused. Anthropometric measurements included a weight of 70 kg and a height of 175 cm, resulting in a body mass index (BMI) of 22.9 kg/m^2 (within the normal range). Cardiopulmonary examination was unremarkable upon inspection, auscultation, and palpation. Abdominal examination revealed minimal tenderness in the epigastric region. The initial ECG demonstrated sinus bradycardia (HR 52 bpm), a saddleback ST-segment elevation ≥ 2 mm in lead V2, T-wave inversion in lead V1, and evidence of left ventricular hypertrophy (LVH) based on the Sokolow–Lyon criteria. Several artifacts were noted, likely secondary to patient movement due to pain. A repeat ECG, performed 15 minutes after the administration of 5 mg sublingual isosorbide dinitrate (ISDN), showed a persistent saddleback pattern in lead V2, T-wave inversion in lead V1, and LVH. Chest radiography revealed mild cardiomegaly.

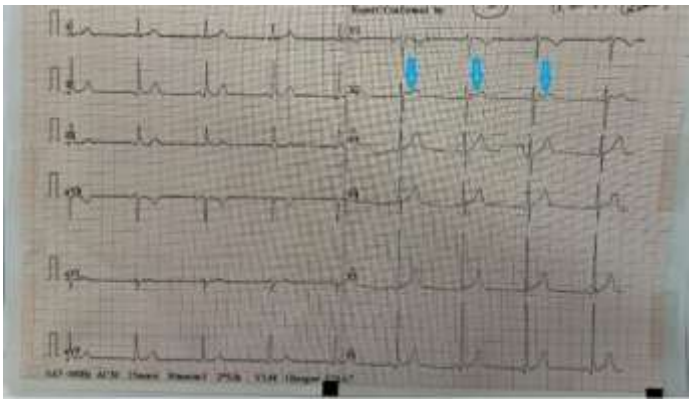

Figure 1. The initial electrocardiogram (ECG) reveals sinus bradycardia, with a heart rate of 52 beats per minute and a saddleback ST-segment elevation ≥ 2 mm in lead V2, characteristic of a type 2 Brugada pattern.
Source: Patient's medical record (2025)

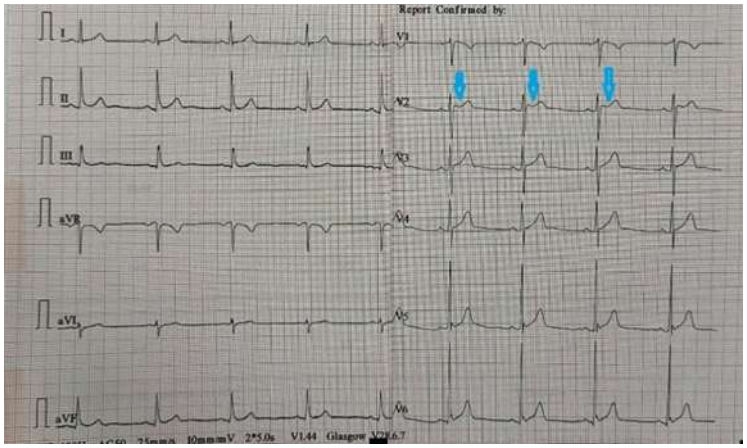

Figure 2. The evaluation ECG reveals a saddleback ST-segment elevation ≥ 2 mm in lead V2

Source: Patient's medical record (2025)

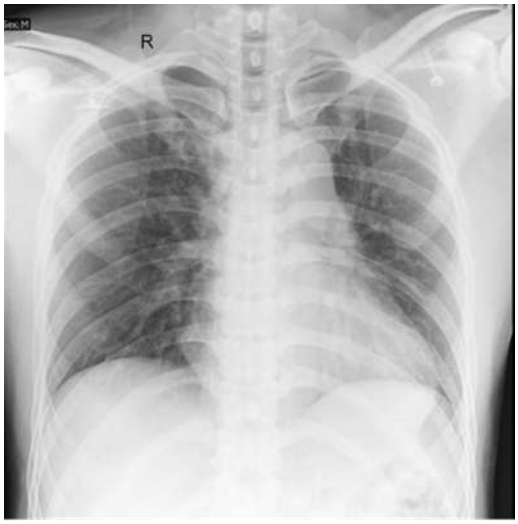

Figure 3. Chest X-ray reveals cardiomegaly

Source: Patient's medical record (2025)

Laboratory investigations revealed the following results: hemoglobin (Hb) 15.9 g/dL, hematocrit (Ht) 43.7%, 8,760/µL, and platelets: 225,000/µL. Renal function and electrolytes were within normal limits: urea 33.2 mg/dL, creatinine 0.94 mg/dL, sodium (Na^+): 138 mmol/L, potassium (K^+) 3.62 mmol/L, and chloride (Cl^-) 105.6 mmol/L. The initial cardiac biomarker, Troponin I, was 1.4 pg/mL.

In the emergency department, the patient received intravenous (IV) fluids containing mineral sorbitol at 20 gtt/min, sublingual isosorbide dinitrate (ISDN) 5 mg, IV pantoprazole, and morphine 2 mg. The patient was admitted to the Intensive Coronary Care Unit (ICCU) with a diagnosis of chest pain secondary to a type II Brugada pattern and gastroesophageal reflux disease (GERD). Follow-up NT-proBNP and Troponin I levels measured in the ICCU remained within normal limits. Additional pharmacological management included a fixed-dose combination of metamizole and diazepam, trimetazidine 35 mg, atorvastatin 10 mg, omeprazole 10 mg, and chlordiazepoxide-clidinium.

During the patient's stay in the ICCU, continuous cardiac monitoring recorded several episodes of ventricular extrasystole (VES) rhythm (Figure 4). Upon further clinical interview, the patient reported no palpitations, and his chest pain had significantly improved, with a recorded VAS score of 1. The follow-up ECG in the ICCU persistently demonstrated an ST-segment elevation with a saddleback pattern ≥ 2 mm in lead V2 and T-wave inversion in lead V1 (Figure 5).

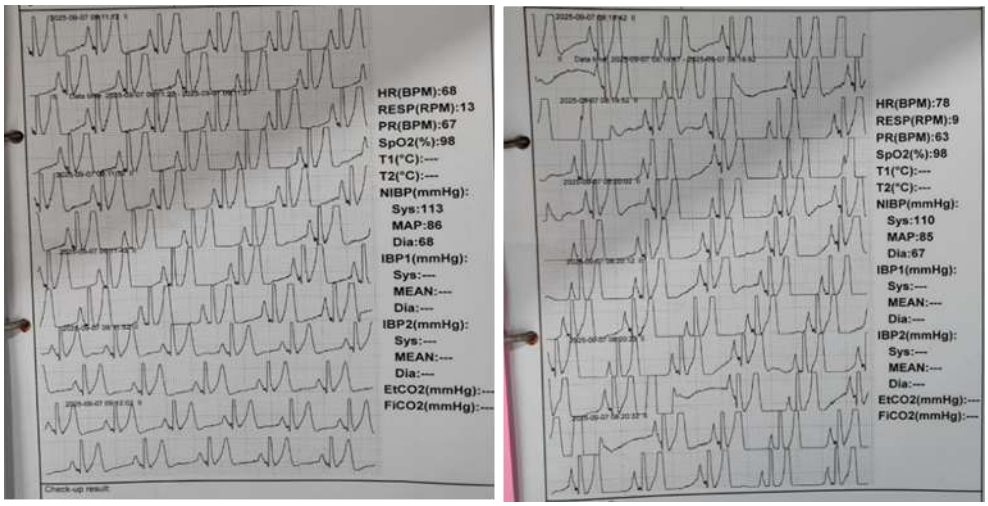

Figure 4. Cardiac monitor recording reveals the presence of premature ventricular contractions (PVCs)

Source: Patient's medical record (2025)

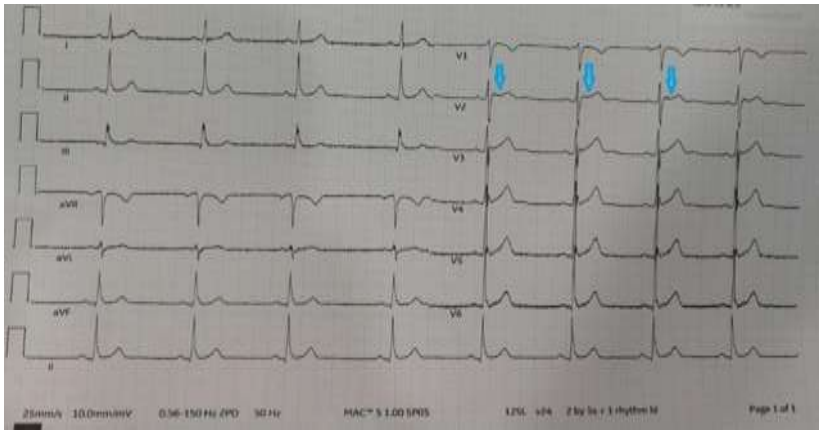

Figure 5. The ECG obtained in the ICCU reveals a type 2 Brugada pattern.

Source: Patient's medical record (2025)

On the second day of hospitalization, the patient reported complete resolution of chest pain. A repeat ECG demonstrated persistent findings consistent with previous traces. As the patient's clinical condition and vital signs remained stable, he was transferred to the general ward. Transthoracic echocardiography revealed normal cardiac chamber dimensions, preserved left ventricular (LV) contractility with an ejection fraction (EF) of 64%, normal right ventricular (RV) function, and global normokinesia. On the third day of treatment, follow-up ECG monitoring continued to show a type II Brugada pattern. Subsequently, a treadmill

exercise test was performed; the results demonstrated normal functional capacity, with appropriate heart rate and blood pressure responses to exercise.

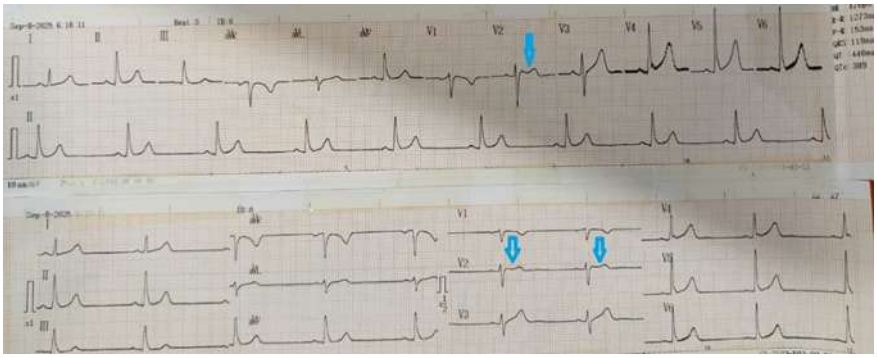

Figure 6. ECG in the general ward
Source: Patient's medical record (2025)

The patient was scheduled for discharge and advised to return for a follow-up outpatient visit in three days. The follow-up plan included electrophysiological (EP) testing and the consideration of an implantable cardioverter-defibrillator (ICD) placement. Upon discharge, nebivolol 2.5 mg was added to the patient's medical regimen.

RESULTS AND DISCUSSION

This case illustrates a type II Brugada pattern in a patient presenting with sharp left-sided chest pain, accompanied by diaphoresis and syncope, despite no prior history of heart disease or sudden cardiac death (SCD) in the family. Laboratory investigations revealed serum electrolytes within the reference range, notably without the hypokalemia often associated with Brugada phenocopies. To date, three ECG patterns have been described for Brugada syndrome (BrS). Type I BrS is characterized by a coved-type ST-segment elevation ≥ 2 mm, followed by a negative T-wave with a slight separation or absence of an isoelectric line in the right precordial leads (V1 and V2). Type II is defined by a saddleback ST-segment elevation ≥ 2 mm, followed by a positive or biphasic T-wave. Type III presents with either a coved-type or saddleback ST-segment elevation of < 1 mm (Figure 7).[8,13] It is important to note that Type 2 and Type 3 are considered non-diagnostic of Brugada syndrome. These three patterns may be observed spontaneously in serial ECG recordings from the same patient or following the administration of certain pharmacological agents.

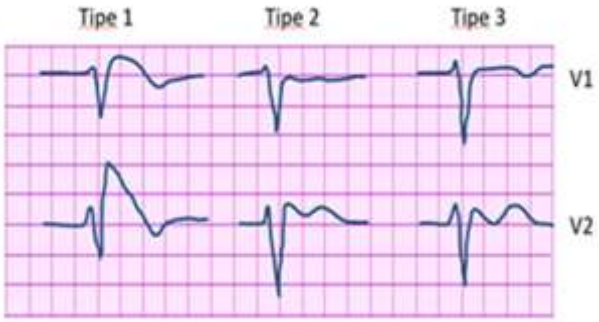

Figure 7. The ECG pattern associated with Brugada syndrome is characterized by specific abnormalities.

Source: Adapted from Wilde et al. (2022)

Brugada syndrome (BrS) is associated with a significantly elevated risk of sudden cardiac death (SCD). The majority of cases involve young males, with a mean age of 41 years at diagnosis. However, the clinical spectrum is broad, with the youngest documented case being two days old and the oldest being 84 years old. Pathophysiologically, BrS is a cardiac ion channelopathy frequently linked to mutations in the SCN5A gene, which encodes the α-subunit of the cardiac sodium channel.

Recent studies have reported cases of Brugada patterns on ECG induced by various underlying pathologies, with subsequent resolution of the ECG findings once the primary condition is treated. These cases are classified as Brugada Phenocopy (BrP).[12] BrP is categorized based on its etiology, which includes electrolyte disturbances, drug toxicity, and other metabolic diseases. The ECG patterns in BrP are identical to those of Type I and Type II Brugada syndrome (BrS), with the following distinguishing features:[12,13]

1. A clear underlying condition is identified.
2. The ECG pattern resolves following the treatment or resolution of the underlying condition.
3. There is a low pre-test probability for BrS, supported by the absence of symptoms and a negative family history of sudden cardiac death.
4. Provocation testing with sodium channel blockers yields negative results.
5. No *SCN5A* gene mutations are detected.

In patients with suspected Brugada syndrome (BrS), provocative testing using sodium channel blockers, such as flecainide, ajmaline, or procainamide, may be performed. The presence of delayed cardiac conduction during these tests further supports a diagnosis of BrS. Specific ECG findings observed following ajmaline administration may include an R^1 wave in lead aVR > 3 mm, fractionated QRS complexes, T-wave alternans, and evidence of early repolarization in the inferior leads.[14]

The diagnosis of a Brugada phenocopy (BrP) is established only when the first four aforementioned criteria are met. In the present case, only one criterion was fulfilled: the absence of a family history of Brugada syndrome (BrS). The persistence of a type II Brugada pattern, accompanied by anginal symptoms, documented arrhythmias, and syncope, necessitates that BrS remain a primary differential diagnosis. A primary limitation of this case is the absence of a provocation test with sodium channel blockers, which would be required to definitively confirm or exclude hereditary BrS. Nevertheless, the clinical presentation of arrhythmias and syncope strongly supports a diagnosis of Brugada syndrome in this patient. Furthermore, this report is inherently limited by its retrospective nature and single-patient scope, which restricts the generalizability of the findings.

In 2015, the European Society of Cardiology (ESC) published guidelines for the management of Brugada syndrome (BrS). These guidelines recommend comprehensive lifestyle modifications, including the avoidance of agents known to induce ST-segment elevation, such as Class I antiarrhythmics, tricyclic antidepressants, propofol, bupivacaine, alcohol, and ergometrine. Additionally, patients are advised to avoid large meals to prevent

potential vagal stimulation, which may trigger arrhythmic events.[10] During hospitalization, the patient was administered trimetazidine (35 mg, twice daily) to mitigate anginal symptoms, alongside proton pump inhibitors (PPIs) for the management of gastroesophageal reflux disease (GERD). Trimetazidine is a cytoprotective agent with antianginal and anti-ischemic properties; it functions by modulating cardiac metabolism without altering systemic hemodynamics. Upon discharge, the patient was prescribed nebivolol (2.5 mg, once daily), a beta-blocker with Class II antiarrhythmic properties. In vivo studies using murine models have demonstrated that nebivolol significantly reduces the incidence of ventricular tachycardia and ventricular fibrillation (VT/VF). These findings suggests that nebivolol may contribute to reducing the overall risk of arrhythmias and sudden cardiac death (SCD).

The management of Brugada syndrome (BrS) has historically relied on device-based therapy, specifically the use of an implantable cardioverter-defibrillator (ICD), which remains the only proven effective treatment for preventing sudden cardiac death.[6] Recommendations for ICD implantation, as established by the second consensus conference, are summarized in Figure 8. Briefly, ICD implantation is recommended for patients diagnosed with Brugada syndrome; however, asymptomatic patients should undergo further risk stratification to determine the necessity of the procedure. Recent therapeutic advances include pericardial catheter ablation, which may assist in preventing electrical storms in BrS patients. Nevertheless, further research is required to definitively confirm the long-term efficacy of this approach.[6,7,8]

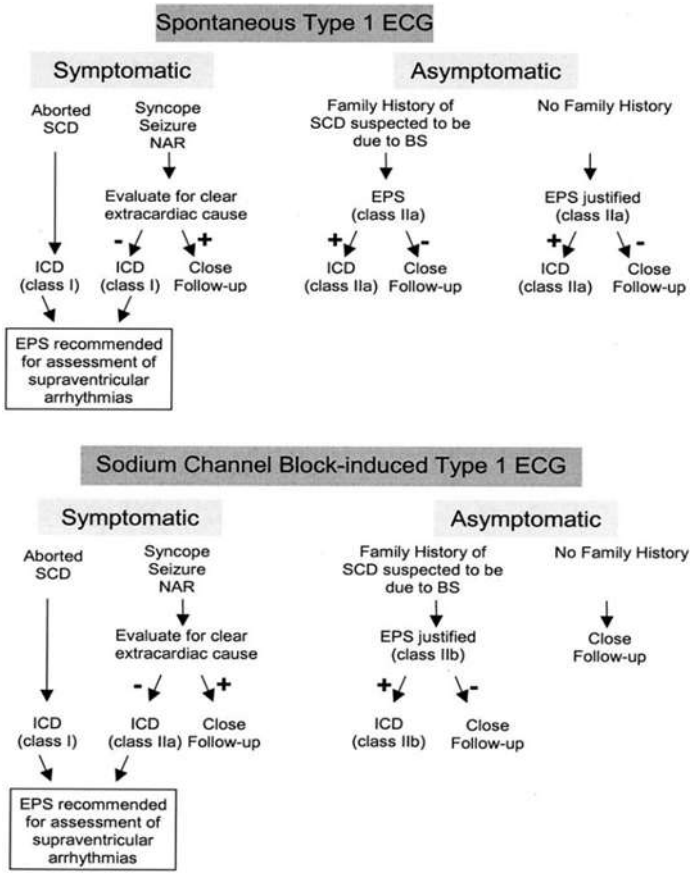

Figure 8. Schematic representation of ICD implantation in Brugada syndrome
Source: Adapted from Priori et al. (2015)

CONCLUSION

Brugada syndrome (BrS) is an autosomal dominant cardiac ion channelopathy associated with mutations in the α-subunit of the sodium channel ($SCN5A$). This condition is clinically significant as it predisposes patients to malignant arrhythmias, including ventricular tachycardia (VT), ventricular fibrillation (VF), and sudden cardiac death (SCD). Accurate ECG identification is paramount, particularly in high-risk patients presenting with syncope, even in the absence of structural heart disease and with normal laboratory findings. Currently, the only proven effective intervention for the prevention of SCD in Brugada syndrome is the implantation of an implantable cardioverter-defibrillator (ICD).

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