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Subcutaneous Implantable Cardioverter-Defibrillator in an Asymptomatic Young Woman With Flecainide-Induced Brugada Syndrome Identified Through Family Screening: A Case Report

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Abstract

Brugada syndrome is an inherited channelopathy predisposing to ventricular fibrillation and sudden cardiac death in structurally normal hearts. Family screening increasingly identifies asymptomatic individuals with drug-induced type 1 patterns, in whom risk stratification and the decision to implant a prophylactic defibrillator remain challenging.

A 21-year-old woman with no significant medical history was referred for familial screening because her father had a confirmed diagnosis of Brugada syndrome. She had no history of aborted sudden cardiac death, documented ventricular arrhythmias, arrhythmic syncope, nocturnal agonal respiration, or palpitations, reporting only a single episode of vasovagal malaise with dizziness. The baseline 12-lead electrocardiogram (ECG) did not demonstrate a spontaneous type 1 Brugada pattern. Given the positive first-degree family history, a flecainide challenge test was performed and unmasked a diagnostic type 1 Brugada ECG pattern, confirming the diagnosis. Transthoracic echocardiography was normal, and genetic investigation of the father and both daughters was planned. Following multidisciplinary evaluation and shared decision-making, a subcutaneous implantable cardioverter-defibrillator (S-ICD) was implanted, favored because of her young age, the absence of pacing or anti-tachycardia pacing indications, and the objective of avoiding long-term transvenous lead-related complications. Intraoperative defibrillation testing successfully induced, detected, and terminated ventricular fibrillation with a single shock. During follow-up, the patient remained completely asymptomatic, with normal device function, stable sensing vectors, and no arrhythmic events or delivered therapies.

This case highlights the diagnostic value of sodium-channel blocker testing in family screening for Brugada syndrome and the difficult issue of prophylactic defibrillator implantation in asymptomatic individuals. The S-ICD represents an attractive option in young patients with inherited arrhythmia syndromes, offering protection against sudden cardiac death while avoiding transvenous lead complications.

Categories: Therapeutics, Cardiology, Genetics
Keywords: brugada syndrome, family screening, flecainide challenge, subcutaneous implantable cardioverter-defibrillator, sudden cardiac death

Introduction

Brugada syndrome is an inherited primary arrhythmia syndrome characterized by a coved-type ST-segment elevation in the right precordial leads (type 1 pattern) and an increased risk of polymorphic ventricular tachycardia, ventricular fibrillation (VF), and sudden cardiac death (SCD) in the absence of overt structural heart disease [1].

The diagnosis rests on the demonstration of a spontaneous type 1 electrocardiogram (ECG) pattern or, in appropriately selected patients, a type 1 pattern unmasked by a sodium-channel blocker challenge such as ajmaline or flecainide [2,3]. Because the syndrome is transmitted as an autosomal dominant trait with incomplete penetrance, cascade family screening of first-degree relatives of probands is recommended and frequently identifies asymptomatic individuals [4].

In this population, the appropriateness of pharmacological unmasking and the subsequent management of a drug-induced type 1 pattern remain debated, since the prognosis of drug-induced Brugada syndrome in asymptomatic subjects is considerably more benign than that of patients with a spontaneous type 1 pattern or prior arrhythmic events [5,6]. When an implantable cardioverter-defibrillator (ICD) is deemed appropriate, the subcutaneous ICD (S-ICD) has emerged as an alternative to transvenous systems,

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particularly attractive in young patients with channelopathies and structurally normal hearts, in whom decades of device therapy are anticipated and neither bradycardia pacing nor anti-tachycardia pacing (ATP) is required [7,8].

A spontaneous type 1 pattern is documented on the baseline ECG without pharmacological provocation, whereas a drug-induced type 1 pattern becomes apparent only after administration of a sodium-channel blocker in patients whose baseline ECG is non-diagnostic. This distinction has important prognostic implications, as asymptomatic patients with a drug-induced pattern generally have a lower risk of ventricular arrhythmic events than those with a spontaneous type 1 pattern, particularly when the latter is associated with arrhythmic symptoms [4-6].

Despite advances in risk stratification, the management of asymptomatic individuals with a drug-induced type 1 Brugada pattern remains particularly challenging. Current guidelines do not recommend routine prophylactic ICD implantation in this population [4]. Nevertheless, individual clinical circumstances, family context, and patient preferences may complicate decision-making, creating a clinical dilemma, particularly in young individuals facing the prospect of lifelong device-related risks and the potential consequences of SCD. Furthermore, when ICD implantation is considered, selecting the most appropriate device requires balancing long-term transvenous lead-related complications against the risk of inappropriate shocks associated with subcutaneous systems [7-9].

We report a case of an asymptomatic young woman diagnosed with Brugada syndrome exclusively through family screening after a positive flecainide challenge, who underwent prophylactic S-ICD implantation following multidisciplinary evaluation and shared decision-making.

Case Presentation

Patient information and presentation

A 21-year-old woman with no significant personal medical history was referred to our electrophysiology department for familial screening because her father had a confirmed diagnosis of Brugada syndrome. She had no history of sudden cardiac arrest, documented ventricular arrhythmias, unexplained syncope, nocturnal agonal respiration, or palpitations. She reported only a single episode of vasovagal malaise associated with dizziness, without any clinical feature suggestive of arrhythmic syncope. There was no evidence of structural heart disease.

Regarding the family history, the patient's father had a confirmed diagnosis of Brugada syndrome. No additional family history of sudden cardiac death or documented ventricular arrhythmias was reported.

Diagnostic assessment

The baseline 12-lead ECG was normal and did not demonstrate a spontaneous type 1 Brugada pattern. Considering the positive first-degree family history, a flecainide challenge test was performed using intravenous flecainide at a dose of 2 mg/kg administered over 10 minutes, according to the institutional protocol. During the test, high right precordial leads (V1 and V2) were also recorded. The challenge unmasked a diagnostic type 1 Brugada ECG pattern, confirming the diagnosis of Brugada syndrome. Transthoracic echocardiography was normal, showing no structural cardiac abnormalities. An electrophysiological study was also performed; however, its results were not available for inclusion in this report. Genetic investigation was planned for the father and both daughters, but genetic testing results were not available at the time of manuscript preparation.

Therapeutic decision

Following multidisciplinary evaluation and shared decision-making with the patient and her family, prophylactic S-ICD implantation was performed. An electrophysiological study had been undertaken as part of the clinical assessment; however, its results were not available for review. The decision was individualized, taking into consideration the patient's young age, her first-degree family history of Brugada syndrome, and her preferences after discussion of the potential benefits and risks of ICD therapy. We acknowledge that prophylactic ICD implantation in an asymptomatic patient with an exclusively drug-induced type 1 pattern goes beyond current guideline-based recommendations and should not be interpreted as a routine management strategy for this population [4].

The choice of a subcutaneous rather than a transvenous system was favored because of: (i) her young age; (ii) the absence of any pacing indication; (iii) the absence of a need for anti-tachycardia pacing; (iv) the expected long lifetime of device therapy, with the objective of avoiding transvenous lead-related complications; and (v) the presence of an inherited arrhythmia syndrome in an otherwise structurally normal heart [7-9].

Procedure

A Boston Scientific EMBLEM MRI S-ICD (Marlborough, MA) was successfully implanted. Defibrillation testing was performed intraoperatively: VF was successfully induced, appropriately detected by the device, and effectively terminated by a single shock, confirming appropriate sensing and satisfactory defibrillation efficacy (Figure *1*). Chest radiography (Figure *2*) confirmed satisfactory generator and parasternal lead positioning.

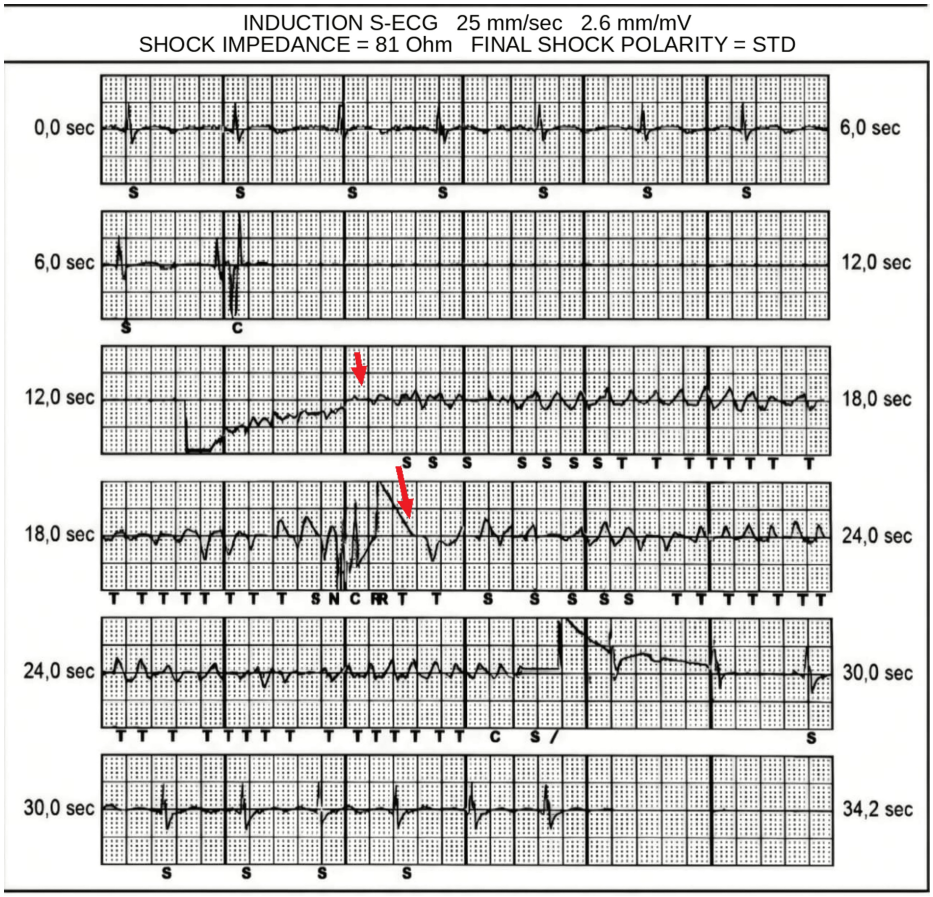


FIGURE 1: Intraoperative subcutaneous implantable cardioverter-defibrillator (S-ICD) electrogram recorded during defibrillation threshold testing. Ventricular fibrillation was successfully induced (first red arrow) and promptly detected by the device, followed by capacitor charging (second red arrow) and delivery of a single shock, resulting in successful termination of ventricular fibrillation and restoration of sinus rhythm. The measured shock impedance was 81 Ω.

Recording speed: 25 mm/s; gain: 2.6 mm/mV.

Event markers: S = sensed event; R = pacing; N = noise; T = tachycardia detection; C = charge start; A = anti-tachycardia pacing request; ● = reject; "/" = shock; ↓ = end of episode.

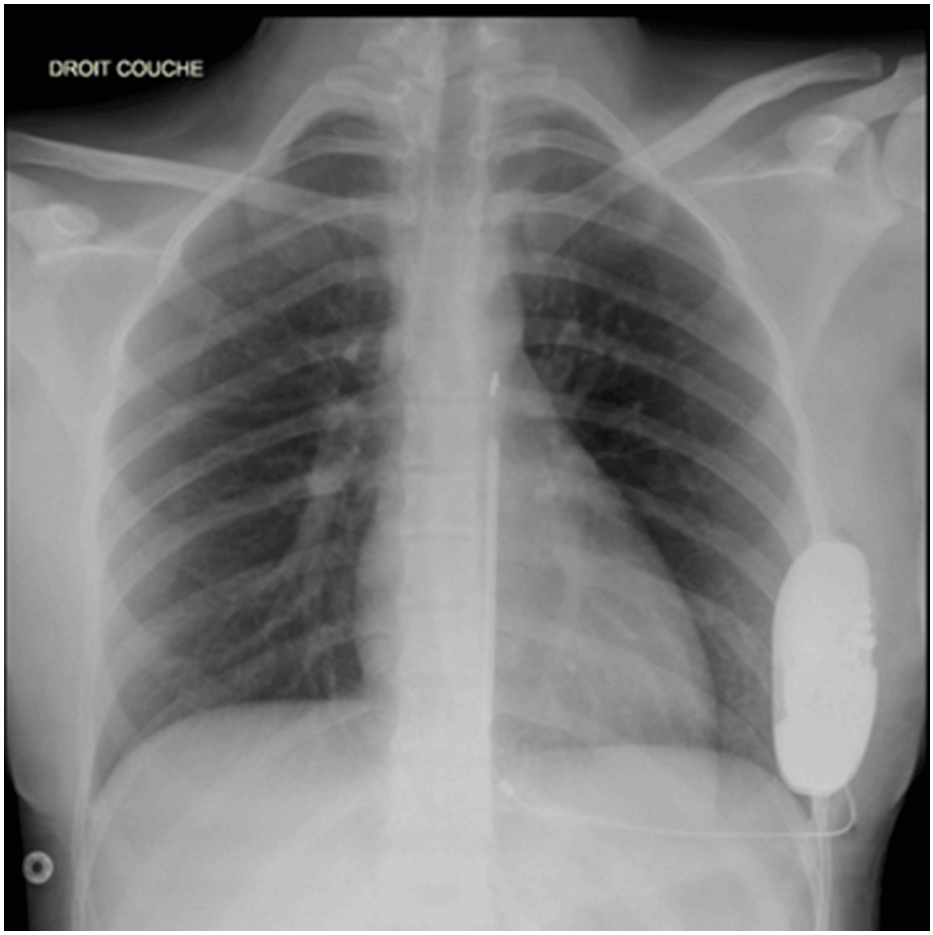


FIGURE 2: Postoperative chest radiograph after subcutaneous implantable cardioverter-defibrillator implantation.

Follow-up and outcomes

The patient was followed up at three, six, and 12 months after S-ICD implantation. Throughout this period, she remained completely asymptomatic, without syncope, presyncope, palpitations, or any other cardiac symptoms. Device interrogation demonstrated normal S-ICD function, stable sensing vectors, normal lead parameters, and a shock impedance of 81 Ω, with no recorded arrhythmic events and no appropriate or inappropriate therapies delivered. The postoperative 12-lead ECG is shown in Figure 3. Follow-up transthoracic echocardiography remained normal.

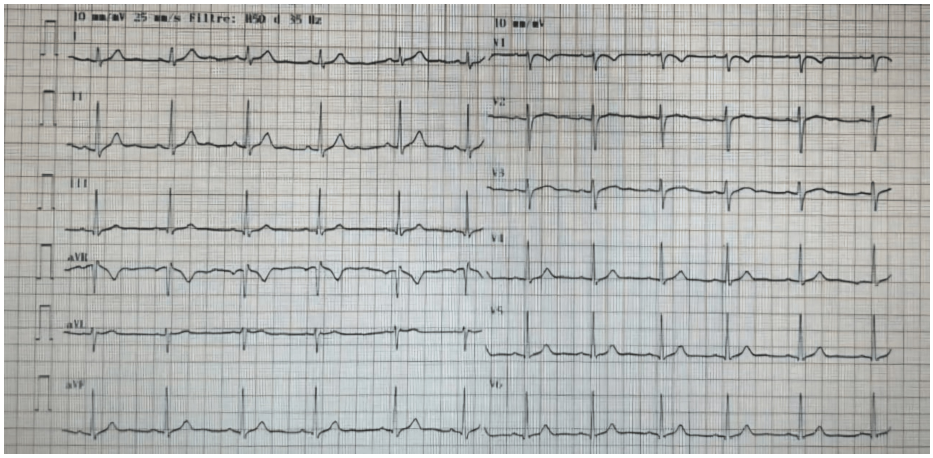


FIGURE 3: Twelve-lead electrocardiogram after subcutaneous implantable cardioverter-defibrillator implantation.

Discussion

This case raises several issues that lie at the heart of contemporary management of Brugada syndrome: the role of family screening, the value and limitations of sodium-channel blocker testing, risk stratification in asymptomatic drug-induced disease, and device selection when a prophylactic ICD is chosen.

Family screening and sodium-channel blocker testing

Cascade screening of first-degree relatives of probands with Brugada syndrome is recommended by current guidelines and consensus documents, combining clinical evaluation, resting and high precordial lead ECG, and, where appropriate, pharmacological provocation [2,4]. Sodium-channel blocker challenge with ajmaline or flecainide is the most widely used method to unmask a concealed type 1 pattern. However, the test is not without limitations: its specificity is imperfect, false-positive responses may occur, and a positive result carries important consequences for the patient in terms of lifestyle recommendations, drug avoidance, fever management, insurability, and psychological burden. Viskin and colleagues have recently emphasized that the decision to perform a sodium-channel blocker test should be individualized, and that relatives of probands represent one of the groups in whom testing is most defensible, while indiscriminate testing of low-pretest-probability individuals should be avoided [5]. In our patient, the first-degree family history justified provocation testing in accordance with these principles.

Risk stratification in asymptomatic drug-induced Brugada syndrome

Risk stratification remains the principal unresolved challenge in Brugada syndrome [1,10]. The strongest predictors of future arrhythmic events are a history of aborted cardiac arrest or arrhythmic syncope and a spontaneous type 1 ECG pattern. In the FINGER registry, the annual event rate in asymptomatic patients was low (approximately 0.5%), and symptoms together with a spontaneous type 1 pattern were the main predictors of events [6]. In the PRELUDE registry, VF inducibility at programmed electrical stimulation did not predict outcome, whereas a spontaneous type 1 pattern, ventricular refractoriness, and QRS fragmentation were associated with events [11]. Asymptomatic patients with a purely drug-induced type 1 pattern, such as our patient, constitute the lowest-risk stratum of the syndrome, and systematic prophylactic ICD implantation is not recommended in this group by the 2022 ESC guidelines, which reserve ICD therapy for survivors of cardiac arrest, patients with documented ventricular arrhythmias, and selected patients with spontaneous type 1 pattern and arrhythmic syncope [4].

The Shanghai score system integrates ECG findings, clinical history, family history, and genetics into a probabilistic diagnostic framework and illustrates the graded nature of diagnostic certainty and risk in this syndrome [3]. In our patient, the decision to implant a prophylactic S-ICD followed a multidisciplinary discussion and an explicit shared decision-making process with the patient and her family, taking into account the family context, patient values and preferences, and local follow-up conditions. We acknowledge that this decision is more aggressive than what current European recommendations propose for asymptomatic drug-induced disease, and this tension is precisely one of the instructive aspects of the case: it illustrates the real-world difficulty of translating population-level risk estimates into individual decisions when a young person and her family are confronted with a disease whose first manifestation may be sudden death.

Sex-related differences

Brugada syndrome is markedly more prevalent and more malignant in men, whereas women represent a minority of clinically affected patients and generally display a lower arrhythmic risk profile [12]. Milman and colleagues reported that women with Brugada syndrome were less likely to present with a spontaneous type 1 pattern and experienced fewer arrhythmic events than men [12]. These sex-related differences further lower the expected event rate in our asymptomatic young female patient and should be explicitly integrated into risk discussions in similar cases.

Subcutaneous versus transvenous ICD in inherited channelopathies

When lifelong protection against SCD is chosen in a young patient with a structurally normal heart, the S-ICD offers an appealing risk profile. The pivotal experience of Bardy and colleagues established the feasibility and efficacy of an entirely subcutaneous defibrillation system [7], and the IDE study and EFFORTLESS registry subsequently confirmed high conversion efficacy for induced and spontaneous ventricular arrhythmias with acceptable complication rates and, importantly, the absence of lead-related endovascular complications [8,9]. In patients with Brugada syndrome specifically, the main limitation of the S-ICD is the risk of inappropriate shocks, driven largely by T-wave oversensing of the dynamic repolarization patterns typical of the disease; recent data suggest a numerically higher rate of nonatrial-tachyarrhythmia-related inappropriate shocks with S-ICD than with transvenous devices in this population, underlining the importance of rigorous pre-implantation screening in multiple postures, careful vector selection, activation of high-pass filtering algorithms, and conditional zone programming [13]. In our patient, intraoperative testing confirmed reliable sensing and first-shock efficacy, and no inappropriate therapy occurred during follow-up.

Remaining controversies

Important knowledge gaps persist regarding the management of asymptomatic Brugada syndrome, including the optimal use and interpretation of programmed stimulation, the incremental value of genetics for individual risk prediction, the long-term psychological and device-related burden of prophylactic ICD therapy in young patients, and the place of emerging alternatives such as quinidine therapy and epicardial substrate ablation [1,10]. Prospective registries dedicated to drug-induced, asymptomatic disease and to S-ICD outcomes in channelopathies will be essential to refine future recommendations.

Conclusions

This case highlights the importance of family screening in identifying asymptomatic individuals with Brugada syndrome and emphasizes the need for individualized risk assessment and shared decision-making. Although S-ICD implantation was performed in this patient, this single case does not establish a benefit of prophylactic ICD therapy in asymptomatic individuals with an exclusively drug-induced type 1 Brugada pattern. Such decisions should be approached cautiously, considering current guideline recommendations and the individual clinical context.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ayoub Laaboussi, Wassim Beladel, Mohamed El Minaoui

Acquisition, analysis, or interpretation of data: Ayoub Laaboussi, Wassim Beladel

Drafting of the manuscript: Ayoub Laaboussi

Critical review of the manuscript for important intellectual content: Ayoub Laaboussi, Wassim Beladel, Mohamed El Minaoui

Supervision: Wassim Beladel, Mohamed El Minaoui

Disclosures

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