Indian Pacing and Electrophysiology Journal 17 (2017) 10–15

Contents lists available at ScienceDirect

Indian Pacing and Electrophysiology Journal

journal homepage: www.elsevier.com/locate/IPEJ

Inappropriate implantable cardioverter-defibrillator shocks in Brugada syndrome: Pattern in primary and secondary prevention

Aimé Bonny [a, *], Mohammed A. Talle [b], Thibaut Vaugrenard [c], Jérôme Taieb [d], Marcus Ngantcha [e]

[a] Clinique Paul Picquet, Service de cardiologie, Sens, France
[b] Department of Internal Medicine, University of Maiduguri Teaching Hospital, Maiduguri, Nigeria
[c] Centre Hospitalier d'Aix en Provence, Service de cardiologie, Aix en Provence, France
[d] Centre Hospitalier d'Aix en Provence, Service de cardiologie, France
[e] Statprest, Biostatistics Unit, Paris, France

ARTICLE INFO

Article history:
Received 25 July 2016
Accepted 21 October 2016
Available online 24 October 2016

Keywords:
Implantable cardioverter-defibrillator
Inappropriate shock
Brugada syndrome

ABSTRACT

Background: Inappropriate implantable cardioverter-defibrillator (ICD) shocks is a common complication in Brugada syndrome. However, the incidence in recipients of ICD for primary and secondary prevention is unknown.

Method and results: We compared the rate of inappropriate shocks in patients with Brugada syndrome that had an ICD for primary and secondary prevention. We studied 51 patients, 86.5% of whom were males. Their mean age at diagnosis was 47 ± 11 years. Eighteen (35%) were asymptomatic, while 25 (49%) experienced syncope prior to implantation. Eight (16%) patients were resuscitated from ventricular fibrillation before implantation. During a mean follow-up of 78 ± 46 months, none of the asymptomatic patients experienced appropriate therapy, whereas 21.6% of symptomatic patients had ≥1 shock. Inappropriate shock occurred in 7 (13.7%) patients, with a mean IS of 6.57 ± 6.94 shocks per patient occurring 16.14 ± 10.38 months after implantation. There was a trend towards higher incidence of inappropriate shock in the asymptomatic group ($p = 0.09$). The interval from implantation to inappropriate shock occurrence was 13.91 ± 12.98 months. The risk of IS at 3 years was 13.7%, which eventually plateaued over the time.

Conclusion: Inappropriate shock is common in Brugada syndrome during the early periods after an ICD implantation, and seems to be more likely in asymptomatic patients. This finding may warrant a review of the indications for ICD implantation, especially in the young and apparently healthy population of patients with Brugada syndrome.

1. Introduction

Brugada syndrome (BrS) is a life-threatening arrhythmogenic disorder requiring implantation of cardioverter-defibrillator (ICD) in some patients to prevent sudden cardiac death (SCD) [1,2]. From the discovery of the disease until recent years, ICD has been implanted almost systematically in asymptomatic BrS carriers with ≥1 risk factors such as spontaneous type 1 ECG (Fig. 1), inducible ventricular fibrillation (VF) during programmed electrical stimulation (PES), and family history of premature death without strong evidence of the rationale for this preventive approach. Because of the low event rates observed on ICD-stored electrograms over a long period of follow-up after implantation, two contending views with regard to appropriateness or otherwise of primary prevention by ICD in BrS have emerged [3–10]. The aim of our study was to compare the rates of inappropriate shock (IS) in the setting of primary and secondary prevention in BrS recipients of ICD.

2. Methods

2.1. Study population

Consecutive patients diagnosed with BrS who underwent ICD

* Corresponding author. Clinique Paul Picquet, service de cardiologie, 12 rue Pierre Castet, 89100, Sens, France.
E-mail address: aimebonny@yahoo.fr (A. Bonny).
Peer review under responsibility of Indian Heart Rhythm Society.

http://dx.doi.org/10.1016/j.ipej.2016.10.010

implantation from 1999 to 2013 in four centers were followed-up and ICD therapy, whether appropriate shock (AS) or IS documented. The patients were divided into 2 categories of asymptomatic (group A), and symptomatic (syncope or cardiac arrest, group B) subjects prior to implantation. Diagnosis of BrS was made based on an episode of aborted sudden cardiac arrest (SCA) or during evaluation of syncope, occurrence of spontaneous ECG pattern consistent with BrS in asymptomatice subjects undergoing routine evaluation, or during screening of kindred of patient diagnosed with BrS. This registry was approved by institutional review committees, and the subjects gave informed consent.

2.2. Diagnosis, clinical data, and diagnostic workup

The following clinical data were collected in the participating centers: circumstances and age at the time of diagnosis, gender, family history of SCD before the age of 45 years, presence of atrial fibrillation (AF) and ventricular tachycardia (VT) before ICD implantation, results of pharmacological testing for unmasking the characteristic coved-type ECG pattern, results of invasive electrophysiological study (EPS), and indication for ICD implantation.

Diagnosis of BrS was made in accordance with the recommendations of the second consensus conference [11]. Patients had to have a prominent coved-type ST-segment elevation (Fig. 1) displaying J-wave amplitude or ST-segment elevation$\geq$0.2 mV at its peak, followed by a negative T wave [11]. In subjects with type 2 or type 3 ECG pattern and other reasons for suspecting the BrS, 1.0 mg/kg of ajmaline (a class I antiarrhythmic drug) was administered intravenously at a rate of 10 mg per minute, and a 12-lead ECG monitored for ST-segment changes of the coved-type BrS pattern. Patients with a history of presumed arrhythmic syncope, documented sustained VT, or aborted SCA were considered symptomatic.

We excluded conditions mimicking BrS by undertaking the following investigations: laboratory tests to exclude acute cardiac ischemia as well as metabolic and electrolyte disturbances; echocardiography and stress testing where indicated; coronary and/or right ventricular angiography, radionuclide ventriculography and cardiac magnetic resonance imaging to rule out structural heart disease. The decision to implant an ICD or not was made at the behest of an experienced electrophysiologist. Until 2010, ICD was implanted in asymptomatic patients in whom sustained VF was induced by any level of programmed stimulation.

2.3. ICD follow-up

In the absence of symptoms or device therapy, patients were routinely seen for ICD interrogation every 3–6 months (depending of physician's protocol). ICD programming consisted of a single detection (VF) zone above 200 to 220 bpm. Appropriate therapies were defined as shocks or antitachycardia pacing delivered for VT or VF. Inappropriate shock was defined as shock delivered in the absence of documented ventricular arrhythmias. We first counted overall occurrence of arrhythmic events (self-terminating ventricular arrhythmias, anti-tachycardia pacing and appropriate shocks) and IS. Afterwards, we compared event rates between both groups. Quinidine hydrochloride (300 mg twice daily) was added for supraventricular tachyarrhythmia or electrical storm during follow-up.

2.4. Statistical analysis

Continuous variables were expressed as the mean $\pm$ SD or the median and interquartile range for non-normally distributed data. Categorical variables were expressed in percentage and compared using the Chi-square test while continuous variables were compared using the Student's t-test. Conditions of validity of tests were checked and in case they were not verified we performed non-parametric tests (Fisher exact, and Mann-Whitney). The event-rate curve was determined using the Kaplan-Meier method. The differences in the rate of appropriate therapy and IS-free survival were analysed with the Log-rank test. To assess the contribution of baseline patient characteristics to the prediction of the likelihood of first IS during follow-up, multivariable Cox proportional hazard regression analysis was used. Adjustment variables were age at diagnosis, the coved type ECG pattern, the history ofAF, and the treatment with quinidine which was analysed in intention-to-treat fashion. Given the low incidence and prevalence of BrS in the general population [12], we assumed the choice of 10% α level for

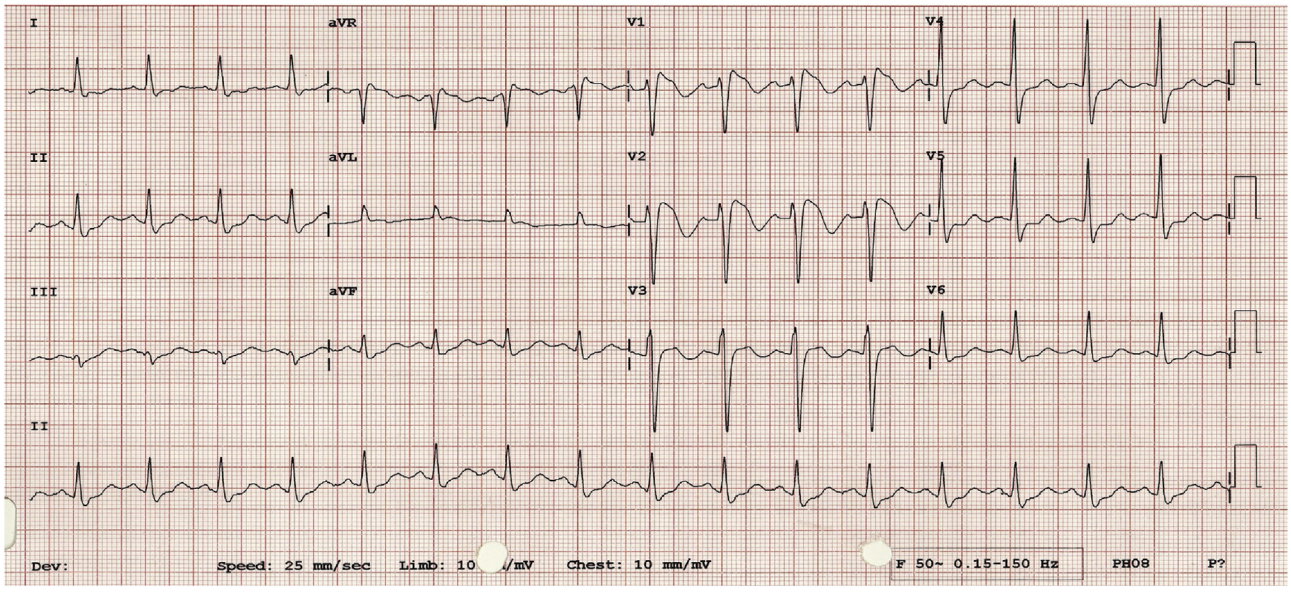

Fig. 1. Diagnostic ECG pattern of Brugada syndrome. Spontaneous coved-type S-T elevation in right precordial leads (mostly in V_1 and V_2 rather than V_3) is the hallmark of Brugada syndrome.

the two-sided *t*-test. Analyses were performed in SAS 9.3 (SAS Institute, Cary NC).

3. Results

3.1. Clinical characteristics and ICD indications

The study population characteristics are summarized in Table 1. Fifty-one patients (male = 86.5%) with a mean age at diagnosis of 47 ± 11 years (20–70 years) were studied. Three patients were lost to follow-up at 13, 14 and 23 months respectively. The vast majority of patients were white (94%), with people of Asian and African decent constituting 4% and 2% respectively. Spontaneous type 1 ECG pattern was found in 40 (78.4%) patients at baseline, whereas 11 (21.6%) were challenged with Ajmaline to unmask the coved type ECG pattern. EPS was performed in 39 (76.5%) patients, 34 (88%) of whom had it before the FINGER publication [4]. Age at diagnosis, gender, a spontaneous type 1 ECG, history of AF and/or non-sustained VT, induced VF/polymorphic VT during EPS, SCN5A gene mutation, and need for quidinine medication were similar in symptomatic and asymptomatic individuals (Table 1).

Eighteen (35%) patients were asymptomatic at the time of diagnosis, while 25 (49%) had previously had at least 1 episode of syncope with no clear extracardiac cause. Eight (16%) patients had been resuscitated from VF prior to diagnosis of BrS. Indication for primary ICD prevention was based on the presence of a type 1 ECG pattern (either spontaneous or induced by drug challenge) in conjunction with (1) inducible ventricular arrhythmias (n = 9), and/or (2) a family history of SCD (n = 9). A family history of SCD was found in 21 (41%) patients. Atrial Fibrillation was diagnosed in 7 (13.7%) patients prior to ICD implantation. A single-chamber ICD was implanted in all patients, but one was upgraded to dual-chamber implant for inappropriate therapies due to AF. Implantation data (DFT, pacing threshold, and R-wave amplitude) were available in all patients.

3.2. Outcomes

During a mean follow-up of 78 ± 46 months (median 76 months; range 1–192 months) after ICD implantation, 1 extracardiac death due to haematological disorder occurred. Therapies (either AS or IS) and ICD-related complications are listed in Table 2.

Asymptomatic patients were free from any life-threatening arrhythmias as detected by ICD, and appropriate ICD shocks were documented only in symptomatic patients.

Inappropriate therapies: IS occurred in 7 patients (13.7%; mean 6.57 ± 6.94 shocks per patient) after a mean follow-up of 16.14 ± 10.38 months from ICD implantation, reaching its peak of 13.7% at 3 years, where it remained over the ensuing 7 years (Table 3).

Compared to symptomatic patients, asymptomatic BrS carriers experienced IS more frequently (27.8% versus 6.1%, p = 0.08, Table 2). The freedom from IS tends to diverge between both groups if we consider the risk α of 10% (p = 0.09, Fig. 2). Reasons for IS were lead dysfunction (n = 4), supraventricular arrhythmias (n = 2), or T-wave oversensing (n = 1). One asymptomatic BrS carrier experienced 10 IS for T wave oversensing. Another asymptomatic subject experienced 17 IS due to a Sprint Fidelis (Medtronic, Mineapolis, MN) lead failure (Fig. 3) implanted for near-syncope, and ajmaline-induced type 1 ST-segment elevation.

Other complications: Pocket infection was diagnosed in 2 (3.9%), and endocarditis in 1 (2%). Treatment for depression as a result of frequent inappropriate shocks was reported in 1 (5.6%) asymptomatic patient, while 1 (3%) symptomatic patient received antidepressants due to chronic pocket chest pain.

4. Discussion

4.1. Main findings

The results of this study shows a trend towards higher incidence of IS in patients with BrS who did not experience syncope nor cardiac arrest before ICD implantation, particularly during the first 3 years of the device. The outcome of this cohort also confirmed the assertion regarding the usefulness of ICD in preventing sudden death in patients with previous arrhythmic events [13], and that asymptomatic BrS-carrier patients have a good long-term prognosis [14].

4.2. Inappropriate shocks

Inappropriate shock is a common adverse effect occurring in recipients of ICD, regardless of the underlying disease or indication [15]. In asymptomatic BrS population with several risk factors for sudden death, Sarkozy et al. [6] registered 17 IS in 47 patients after 47.5 months of ICD implantation. Sacher et al. [4] found the prevalence of 24% after 6 years with a risk at 10 years of 34%, a prevalence similar to that reported by Miyazaki et al. [13] 7 years of follow-up. In a high-risk pediatric hypertrophic cardiomyopathy cohort, Maron et al., found that ICD-related complications, particularly IS, occurred more commonly in asymptomatic patients than recipients for secondary prevention [16]. This has not been studied in the BrS population yet. Our data showed a trend towards higher propensity for unnecessary therapies particularly in asymptomatic BrS carriers (Fig. 2), and the first 3 years was marked by increased incidence of IS, after which the patients remain free of IS. The incidence was highest (8.3%) in the first year following implantation, increasing to 12% in the second before reaching the peak of 13.7% by the end of the third year, remaining static over the ensuing 7 years of follow-up. Sacher et al. [3] reported a different figure, but the trend towards increased occurrence of IS among our cohort is attenuated over time. Given the high rate of lead fracture in association with IS, one can hypothetically attribute the very high occurrence of IS in the aerly post-implantation period to procedure-related lead damage.

In contrast to previous studies [4,5,17], our study specified the occurrence of IS in recipients of ICD for primary and secondary prevention. This have provided an insight on the differential burden of IS in asymptomatic and symptomatic patients with BrS, a fact that previously unknown. Inappropriate shock is associated with poor prognosis in ICD recipients [18,19], and the findings of our study underscores the complexity associated with managing asymptomatic patients with BrS [18,20]. This calls for a better system of risk classification to better guide the indications of ICD implantation in this population of patients.

The mechanism underlying the delivery of IS in by an ICD in the setting of primary and secondary prevention have not been elucidated. Factors identified to be associated with increased risk of IS include age less than 50 years. Younger patients have a higher propensity for electrode failure due to physical activity, T wave oversensing, and faster rates of atrial tachyarrhythmias [4–6,17]. However, these factors are equally distributed in our sample, and thus cannot explain the somewhat higher susceptibility of asymptomatic individuals. We speculate that younger asymptomatic individuals are more likely to engage in physical activities that may uncover a hitherto concealed lead fracture to generate noise, and consequently inappropriate therapies.

One issue that makes our findings rather intriguing is that the risk of electrode fractures increases with time, especially in young active male patients [4,6]. It has been demonstrated that the hazard of Sprint Fidelis lead fracture increases exponentially over time

Table 1
Baseline characteristics of the subjects.

	Asymptomatic (n = 18)	Symptomatic (n = 33)	Total (n = 51)	p-value
Age at diagnosis (mean + SD)	44.6 ± 12.5	48.1 ± 9.8		0.55
Male[a]	15(83.3)	30(90.9)	45(88.2)	0.18
Race[a]				
White	16(88.9)	32(97)	48(94.1)	0.12
Black	0	1(3)	1(2)	–
Asian	2(11.1)	0	2(3.9)	–
Spontaneous type 1 ECG[a]	12(66.7)	28(84.8)	40(78.3)	0.16
AF before ICD[a]	3(16.7)	4(12.1)	7(13.7)	0.42
NSVT before ICD[a]	2(11.1)	7(21.2)	9(17.7)	0.67
Patients with EPS[a]	14(77.8)	18(54.6)	32(62.8)	0.22
SCN_5A[a,b]	0	1(3.0)	1(2.0)	–
Family history of SCD[a]	9(50.0)	12(36.4)	21(41.2)	0.36
Quinidine therapy[a,c]	4(22.2)	12(36.4)	16(31.4)	0.36

ECG = Electrocardiogram, AF = Atrial fibrillation, NSVT = Non-sustained ventricular tachycardia, EPS = Electrophysiological study, SCN_5A = gene mutation encoding sodium channel, SCD = Sudden cardiac death.
[a] Expressed as number (%).
[b] Only 15 of the 51 patients had screening for SCN_5A. mutation.
[c] Intention-to-treat analysis was considered to assess the effect of Quinidine therapy given for all indications (electrical storm or AF) after ICD.

Table 2
Implantable cardioverter-defibrillator therapies and mechanisms of inappropriate shocks after 78 ± 46 months of follow-up.

	Primary prevention (n = 18)	Secondary prevention (n = 33)	Total (n = 51)	p-value[a]
Appropriate shocks	0(0)	11(33.3)	11(21.6)	0.006
Inappropriate shocks[b]				
Lead malfunction[c]	2(11.1)	2(11.1)	4(7.8)	0.522
Atrial fibrillation	2(11.1)	0(0)	2(3.9)	0.0511
T-wave over-sensing	1(5.6)	0(0)	1(2)	0.707
Total with IS	5(27.8)	2(6.1)	7(13.7)	0.032

IS = Inappropriate shock.
[a] p value for test of difference between proportions.
[b] Inappropriate shock presented by mechanism and total in the last row.
[c] Lead malfunction includes displacement, insulation erosion and conductor fracture.

Table 3
Rate of inappropriate shocks after ICD implantation.

Years	Inappropriate shocks, n(%)		
	Primary prevention	Secondary prevention	Total
1	3(18.7)	1(3.1)	4(8.3)
2	4(23.5)	2(6.1)	6(12)
3	5(27.8)	2(6.1)	7(13.7)
4	5(27.8)	2(6.1)	7(13.7)
5	5(27.8)	2(6.1)	7(13.7)
10	5(27.8)	2(6.1)	7(13.7)

ICD=Implantable cardioverter-defibrillator.

[21,22]. Moreover, the peculiarity of the patient's lead as depicted in Fig. 2 is that insulation breach with low-voltage, non-physiological signals are an uncommon pattern of failure with Sprint Fidelis leads, which are prone to conductor fracture rather than insulation abrasion [23]. Thus, the trend towards higher lead complication in the early period after implantation in this population might not be explained by the specific lead model. Why IS declined in the 4th year onward, after the high incidence that characterizes the immediate post-implantation period remained unexplained. Early tracking of subclinical lead failure by means of lead integrity alerts (LIA, Medtronic, Mineapolis, MN) or conventional impedance monitoring may provide an insight [23].

Finally, the psychological state of ICD recipients is often neglected [17,20]. In our study, 2 patients suffered severe depression and had been on medication. One of them received an ICD for primary prevention, and did not experience any arrhythmic event after 14 years of follow-up. In this regard, social impacts and consequences for employment are important and should be considered when dealing with young adults [20]. A reappraisal of the benefits and potential hazards of ICD insertions in the young population of patients with Brugada syndrome will enable physicians to have a more mutually informed and balanced dialogue with their patients and relatives [18].

4.3. Study limitations

This is a retrospective study with a potential bias in the data collection such as the adherence to quinidine, and the extent of documentation of lead failures. Secondly, the number of patients is small to conclusively interpret the results. It will be imperative to carry out a similar study involving larger cohorts.

5. Conclusion

Brugada syndrome is characterized by young age of ICD carriers with a high likelihood of experiencing inappropriate shocks. This study showed that the highest risk of inappropriate therapy is in the early post-implantation period, and mostly occurred in asymptomatic younger patients receiving ICD for primary prevention. In addition, the absence of arrhythmic events in our recipients of ICD for primary prevention as well as reports of other registries after a long term follow-up warrants a more careful evaluation of these young and otherwise “healthy” individuals before implanting an ICD. This approach can minimize the incidence of iatrogenic depression and other complications. A study involving larger cohorts of patient will be required to adjudicate our findings, and possibly elucidate the mechanisms underlying the increased rate of

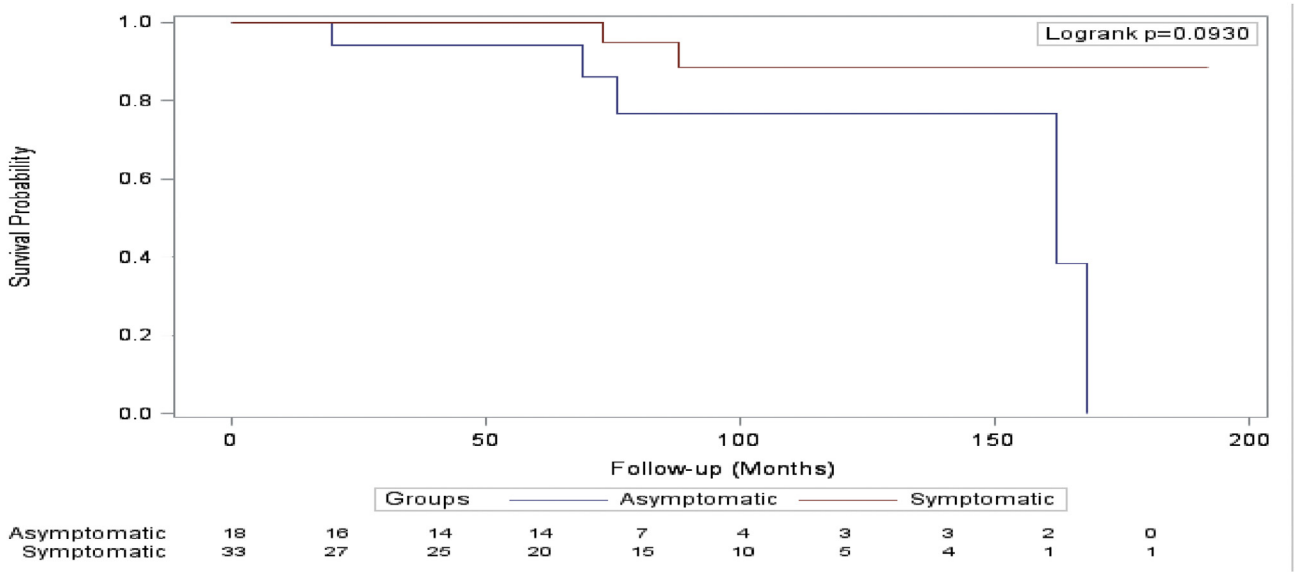

Fig. 2. Kaplan-Meier curve of inappropriate shocks in BrS patients who underwent ICD implantation for primary and secondary prevention. Given the low prevalence of Brugada syndrome in the general population, the difference between both groups was calculated using two-sided *t*-test α-level of 10%.

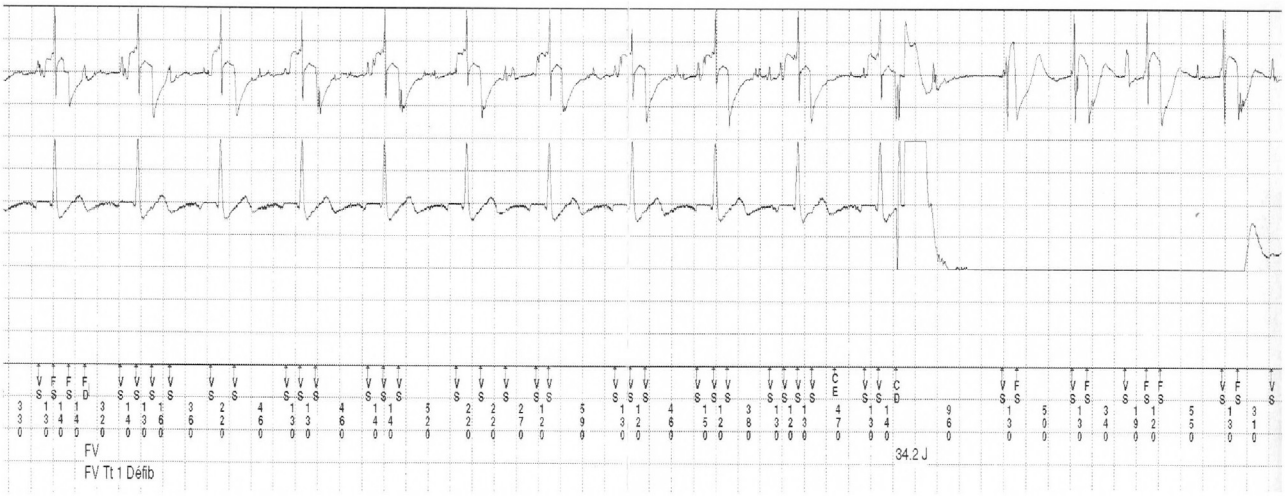

Fig. 3. Pace-sense failure related to inappropriate shock. A 54-year old asymptomatic female in whom an implantable cardioverter-defibrillator was implanted have experienced 17 inappropriate shocks. Pace-sense insulation breach of a Sprint Fidelis lead (Medtronic, Mineapolis, MN) led to oversensing low-voltage nonphysiological signals.

IS in this population.

Contributors

AB, TV, JT, and MN conceived and designed the study, collected the data and provided the first draft. AB, MN and MAT analysed the data, while MAT provided the second draft. AB, MN and MAT provided critical review of the manuscript, and all authors read and approved the final version of the manuscript.

Funding

None.

Disclosures

None.

Acknowledgments

None.

References

[1] Priori SG, Wilde AA, Horie M, Cho Y, Behr ER, Berul C, et al. Executive summary: HRS/EHRA/APHRS expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes. Europace 2013;15:1389–406.
[2] Priori SG, Gasparini M, Napolitano C, Della Bella P, Ottonelli AG, Sassone B, et al. Risk stratification in Brugada syndrome: results of the PRELUDE (PRogrammed ELectrical stimUlation preDictive valuE) registry. J Am Coll Cardiol 2012;59:37–45.
[3] Sacher F, Probst V, Maury P, Babuty D, Mansourati J, Komatsu Y, et al. Outcome after implantation of a cardioverter-defibrillator in patients with Brugada syndrome: a multicenter study-part 2. Circulation 2013;128:1739–47.
[4] Probst V, Veltmann C, Eckardt L, Meregalli PG, Gaita F, Tan HL, et al. Long-term prognosis of patients diagnosed with Brugada syndrome: results from the FINGER Brugada Syndrome Registry. Circulation 2010;121:635–43.
[5] Sarkozy A, Boussy T, Kourgiannides G, Chierchia GB, Richter S, De Potter T,

et al. Long-term follow-up of primary prophylactic implantable cardioverter-defibrillator therapy in Brugada syndrome. Eur Heart J 2007;28:334–44.
[6] Brugada J, Brugada R, Brugada P. Determinants of sudden cardiac death in individuals with the electrocardiographic pattern of Brugada syndrome and no previous cardiac arrest. Circulation 2003;108:3092–6.
[7] Viskin S, Rogowski O. Asymptomatic Brugada syndrome: a cardiac ticking time-bomb? Europace 2007;9:707–10.
[8] Antzelevitch C, Brugada P, Borggrefe M, Brugada J, Brugada P, Corrado D, et al. Brugada syndrome: report of the second consensus conference: endorsed by the heart rhythm society and the European heart rhythm association. Circulation 2005;111:659–70.
[9] Gallagher MM, Forleo GB, Behr ER, Magliano G, De Luca L, Morgia V, et al. Prevalence and significance of Brugada-type ECG in 12,012 apparently healthy European subjects. Int J Cardiol 2008;130:44–8.
[10] Takagi M, Sekiguchi Y, Yokoyama Y, Aihara N, Hiraoka M, Aonuma K, et al. Long-term prognosis in patients with Brugada syndrome based on class II indication for implantable cardioverter-defibrillator in the HRS/EHRA/APHRS expert consensus statement: multicenter study in Japan. Japan idiopathic ventricular fibrillation study (J-IVFS) investigators. Heart Rhythm 2014;11:1716–20.
[11] Bouzeman A, Traulle S, Messali A, Extramiana F, Denjoy I, Narayanan K, et al. Long-term follow-up of asymptomatic Brugada patients with inducible ventricular fibrillation under hydroquinidine. Europace 2014;16:572–7.
[12] Tan VH, Wilton SB, Kuriachan V, Sumner GL, Exner DV. Impact of programming strategies aimed at reducing nonessential implantable cardioverter defibrillator therapies on mortality: a systematic review and meta-analysis. Circ Arrhythm Electrophysiol 2014;7:164–70.
[13] Miyazaki S, Uchiyama T, Komatsu Y, Taniguchi H, Kusa S, Nakamura H, et al. Long-term complications of implantable defibrillator therapy in Brugada syndrome. Am J Cardiol 2013;111:1448–51.
[14] Maron BJ, Spirito P, Ackerman MJ, Casey SA, Semsarian C, Estes NA, et al. Prevention of sudden cardiac death with implantable cardioverter-defibrillators in children and adolescents with hypertrophic cardiomyopathy. J Am Coll Cardiol 2013;61:1527–35.
[15] Moss AJ, Schuger C, Beck CA, Brown MW, Cannom DS, Daubert JP, et al. MADIT-RIT Trial Investigators. Reduction in inappropriate therapy and mortality through ICD programming. N Engl J Med 2012;367:2275–83.
[16] Veltmann C, Kuschyk J, Schimpf R, Streitner F, Schoene N, Borggrefe M, et al. Prevention of inappropriate ICD shocks in patients with Brugada syndrome. Clin Res Cardiol 2010;99:37–44.
[17] Probst V, Plassard-Kerdoncuf D, Mansourati J, Mabo P, Sacher F, Fruchet C, et al. The psychological impact of implantable cardioverter defibrillator implantation on Brugada syndrome patients. Europace 2011;13:1034–9.
[18] Tung R, Zimetbaum P, Josephson ME. A critical appraisal of implantable cardioverter-defibrillator therapy for the prevention of sudden cardiac death. J Am Coll Cardiol 2008;52:1111–21.
[19] Swerdlow CD, Ellenbogen KA. Implantable cardioverter-defibrillator leads: design, diagnostics, and management. Circulation 2013;128:2062–71.
[20] Sears Jr SF, Conti JB. Quality of life and psychological functioning of ICD patients. Heart 2002;87:488–93.
[21] Faulknier BA, Traub DM, Aktas MK, Aguila A, Rosero S, Daubert JP, et al. Time-dependent risk of Fidelis lead failure. Am J Cardiol 2010;105:95–9.
[22] Hauser RG, Hayes DL. Increasing hazard of Sprint Fidelis implantable cardioverter-defibrillator lead failure. Heart Rhythm 2009;6:605–10.
[23] Ellenbogen KA, Gunderson BD, Stromberg KD, Swerdlow CD. Defibrillator leads cardioverter defibrillator lead failures: analysis of different implantable cardioverter performance of lead integrity alert to assist in the clinical diagnosis of implantable. Circ Arrhythm Electrophysiol 2013;6:1169–77.