IJC Heart & Vasculature 51 (2024) 101371

Contents lists available at ScienceDirect

IJC Heart & Vasculature

journal homepage: www.sciencedirect.com/journal/ijc-heart-and-vasculature

Implantable loop recorder in Brugada syndrome: Insights from a single-center experience

Gianmarco Arabia [a,*,1], Manuel Cerini [a,1], Angelica Cersosimo [a,1], Paolo Vinciguerra [a,1], Emiliano Calvi [a,1], Gianfranco Mitacchione [b,1], Mohamed Aboelhassan [c,1], Daniele Giacopelli [d,1], Antonio Curnis [a,1]

[a] Cardiology Department, Spedali Civili Hospital, University of Brescia, Italy
[b] Department of Cardiology, Luigi Sacco University Hospital, Milan, Italy
[c] Department of Cardiovascular Medicine, Assiut University Heart Hospital, Assiut University, Assiut, Egypt
[d] Clinical Unit, Biotronik Italia, Cologno Monzese (MI), Italy

ARTICLE INFO

Keywords:
Brugada syndrome
Implantable loop recorder
Sudden cardiac death

ABSTRACT

Background: This study aimed to investigate the characteristics and outcomes of patients diagnosed with Brugada syndrome (BrS) who underwent implantable loop recorder (ILR) insertion during routine clinical activity.
Methods: We conducted a comprehensive screening of all consecutive patients diagnosed with BrS at our institution. We analyzed baseline clinical characteristics, arrhythmic findings, and outcomes.
Results: Out of 147 BrS patients, 42 (29 %) received an ILR, 13 (9 %) underwent implantable cardioverter-defibrillator (ICD) placement, and 92 patients (63 %) continued regular cardiological follow-up. Patients who received an ILR had a higher prevalence of suspected arrhythmic syncope (43 % vs. 22 %, $p = 0.012$) and tended to be younger (median age 38 years, interquartile range 30–52, vs. 43 years, 35–55, $p = 0.044$) with a higher presence of SCN5A gene mutations (17 % vs. 6 %, $p = 0.066$) compared to those who continued regular follow-up. Additionally, compared to patients with an ICD, those with an ILR had a significantly lower frequency of positive programmed ventricular stimulation (0 % vs. 91 %, $p < 0.001$). During a median follow-up period of 14.7 months (4.7–44.8), no deaths occurred among the patients with ILR. Eight individuals (19 %) were diagnosed with arrhythmic findings through continuous ILR monitoring, primarily atrial fibrillation, and asystolic pauses. The median time from insertion to the occurrence of these events was 8.7 months (3.6–46.4). No adverse events related to ILR were reported.
Conclusion: Continuous monitoring with ILR may facilitate the timely detection of non-malignant rhythm disorders in BrS patients with risk factors but without an indication for primary prevention ICD implantation.

1. Introduction

Brugada syndrome (BrS) is characterized by 'coved' ST segment elevation of ≥2 mm in the right precordial electrocardiographic (ECG) leads, either spontaneously or drug-induced. It is associated with an increased risk of ventricular arrhythmias and sudden cardiac death (SCD) [1,2]. Despite several proposed scoring systems, risk stratification in individuals with BrS remains challenging, and the use of implantable cardioverter-defibrillators (ICDs) for primary prevention of SCD is still a topic of controversial [3–6]. Moreover, individuals with BrS often experience neurocardiogenic or unexplained syncope, palpitations secondary to atrial fibrillation (AF), and atrioventricular nodal reentrant tachycardia [7,8].

Currently, miniaturized and remotely monitored implantable loop recorders (ILRs) are available, providing physicians with a diagnostic tool that can help guide intervention or therapy decisions for these patients. The recent guidelines from the European Society of Cardiology (ESC) acknowledge the consideration of ILRs for BrS patients with episodes of unexplained syncope and 'low risk' of SCD [1,9]. However, there is still limited clinical evidence regarding the effectiveness of this approach [10]. The objective of this study was to investigate the characteristics and outcomes of patients diagnosed with BrS who underwent

* Corresponding author at: Cardiology Department, Spedali Civili Hospital, Piazzale Spedali Civili 1, 25123 Brescia, Italy.
E-mail address: gianmarcoarabia@gmail.com (G. Arabia).
[1] This author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

https://doi.org/10.1016/j.ijcha.2024.101371
Received 31 January 2024; Received in revised form 19 February 2024; Accepted 19 February 2024

ILR insertion during routine clinical activity.

2. Methods

This is a retrospective cohort study. We screened all consecutive patients who were diagnosed with BrS based on the presence of spontaneous or drug-induced type 1 ECG pattern between January 2009 and May 2023 at Spedali Civili hospital in Brescia, Italy. Baseline clinical characteristics and arrhythmic findings detected during the follow-up period were collected retrospectively from electronic clinical records. In June 2023, all patients were contacted to assess their clinical status and the occurrence of major cardiovascular events. Informed consent was obtained from all patients. This study was conducted in accordance with the principles outlined in the Helsinki Declaration on human research. The study data are available from the corresponding author upon reasonable request.

Although our approach to treating patients suspected of BrS has undergone slight changes over the years, it is primarily based on the evaluation of the ECG pattern and the assessment of the Shangai score, which incorporates clinical, familial, and genetic data [5]. An Ajmaline test is routinely conducted if the ECG does not show a spontaneous type 1 pattern and the Shangai score exceeds the 2 points threshold. For patients presenting with a spontaneous or drug-induced type 1 ECG pattern, along with suspected arrhythmic syncope, nocturnal agonal respiration, or a family history of SCD, we propose programmed ventricular stimulation as an additional step for risk stratification. The protocol consists of 2 basic drive cycles (600 and 400 ms, S1-S1) and 3 extrastimuli (S2 to S4). The coupling interval of the extrastimuli was reduced in 10 ms steps to a minimum of 200 ms or higher in case refractoriness of the right ventricle. Programmed ventricular stimulation is defined positive if it leads to the induction of either sustained or hemodynamically significant polymorphic ventricular tachycardia or ventricular fibrillation requiring DC shock. Genetic testing is always recommended for these patients. Once this comprehensive diagnostic pathway is completed, we engage in detailed counseling with patients to discuss the potential need for ICD implantation or ILR insertion, if deemed indicated. Patients who undergo cardiac device implantation are regularly followed through remote monitoring and scheduled for an in-person visit every 12 months, or more frequently in the event of arrhythmic events. Similarly, individuals with BrS who have not received a cardiac device undergo routine cardiological follow-up, involving scheduled cardiologist assessments every 12 months, or as needed in case of symptomatic occurrences.

Descriptive statistics were used to analyze the demographic and clinical data. Categorical variables were expressed as absolute numbers (percentages) and compared using the Fisher's exact test or chi-square test. Continuous variables were reported as median (interquartile range, IQR) and compared using the Mann-Whitney *U* test. To show arhythmic findings over follow-up, a Kaplan-Meier survival curve, accompanied by a 95 % confidence interval, was generated. The statistical analysis was conducted using Stata 18.0MP by StataCorp LLC (Texas, US). All p-values were two-sided, and statistical significance was accepted at $p < 0.05$, except for tests involving multiple comparisons, for which the Bonferroni correction was applied.

3. Results

3.1. Study population

Between January 2009 and May 2023, a total of 147 patients were diagnosed with BrS at our institution based on the presence of a spontaneous or drug-induced type 1 ECG pattern. These patients were included in the current study. Following the diagnosis, 42 patients (29 %) received an ILR: 24 (58 %) received Reveal Linq or XT (Medtronic), 13 (30 %) received Confirm Rx (Abbott), and 5 (12 %) received BIOMONITOR III/IIIm (Biotronik). Thirteen of the remaining patients (9 %) received an ICD, while 92 patients (63 %) continued regular cardiological follow-up without any implanted cardiac device. Table 1 provides an overview of the patients' characteristics for the entire population, as well as for each group. Compared to patients who did not receive a device, those who received an ILR had a higher prevalence of suspected arrhythmic syncope (43 % vs. 22 %, $p = 0.012$) and tended to be younger (median age 38 years, IQR 30–52, vs. 43 years, IQR 35–55, $p = 0.044$) and with a higher presence of SCN5A gene mutations (17 % vs. 6 %). On the other hand, compared to patients who received an ICD, those with an ILR underwent programmed ventricular stimulation less frequently (40 % vs. 85 %, $p = 0.010$) and, as a result, had a lower frequency of positive result of this test (0 % vs. 91 %, $p < 0.001$).

3.2. Follow-up of patients with ILR

During a median follow-up period of 14.7 months (IQR, 4.7–44.8), no deaths occurred among the patients with ILR. Eight individuals (19 %) were diagnosed with arrhythmic findings through continuous ILR monitoring (Table 2). Among these findings, three patients experienced AF with symptoms (including one case of new-onset AF), two patients manifested asystolic pauses one lasting 16 s, associated with syncope, and the other lasting 3.6 s without symptoms. Additionally, two patients had asymptomatic non-sustained ventricular tachycardias, and another patient had episodes of symptomatic paroxysmal supraventricular tachycardia (Fig. 1). The median time from implantation to the occurrence of these events was 8.7 months (IQR, 3.6–46.4). Fig. 2 depicts the Kaplan-Meier survival curve for arrhythmic findings in patients who received an ILR. Except for a history of AF, no significant differences were observed between patients with arrhythmic findings and those without any events (Table 3). Following the diagnosis, two patients underwent transcatheter ablation procedures (one undergoing pulmonary vein isolation and the other receiving nodal re-entry ablation), and one patient received an ICD. No adverse events related to the implanted device were reported. During the follow-up period, two patients who had been implanted with an ILR but did not exhibit any arrhythmic findings experienced recurrences of syncope.

3.3. Follow-up of patients implanted with ICD or with no device

In the ICD group, the median follow-up period was 32.2 months (IQR, 23.2–95.0). During this period, one death occurred due to non-cardiovascular reasons, specifically oral cavity cancer. No appropriate or inappropriate ICD therapies were delivered, although non-sustained ventricular tachycardias were recorded in three patients (23.1 %). Additionally, one patient (0.8 %) required transvenous lead extraction and re-implantation due to lead malfunction 41.8 months after ICD implantation.

Among patients without an implanted device, the median follow-up period was 81.4 months (IQR, 26.9–141). No deaths or cardiovascular hospitalizations were reported during this follow-up period.

4. Discussion

In our single-center study, approximately one-fourth of patients diagnosed with BrS underwent ILR insertion. This approach was primarily utilized in patients who presented with risk factors such as suspected arrhythmic syncope or the presence of SCN5A gene mutations, despite negative programmed ventricular stimulation. Notably, around 20 % of patients had arrhythmias recorded by the ILR within a relatively short period of time (median time from insertion to arrhythmic finding: 8.7 months), with four of them receiving medical treatment following these diagnoses. No deaths, malignant ventricular arrhythmias, or device-related complications occurred.

Continuous monitoring revealed episodes of supraventricular tachycardias, including AF, as well as non-sustained ventricular tachycardia. Additionally, two patients exhibited asystolic pauses lasting 16 s

Table 1
Study population.

Characteristic	All N = 147	ILR (a) N = 42	ICD (b) N = 13	No device (c) N = 92	P value (a) vs (b)	P value (a) vs (c)
Sex, female (n, %)	41 (28 %)	15 (36 %)	2 (15 %)	24 (26 %)	0.303	0.255
Age (years)	41 (34–55)	38 (30–52)	45 (36–54)	43 (35–55)	0.322	0.044
Height (cm)	172 (170–175)	173 (168—178)	175 (172—178)	172 (169—175)	0.236	0.440
Weight (Kg)	74 (69–78)	75 (65–79)	76 (70–80)	73 (70–78)	0.129	0.959
History of						
Cardiac arrest	1 (0.7 %)	0 (0 %)	1 (7.7 %)	0 (0 %)	0.236	–
Polymorphic VT or VF	1 (0.7 %)	0 (0 %)	1 (7.7 %)	0 (0 %)	0.236	–
Suspected arrhythmic syncope	42 (29 %)	18 (43 %)	4 (31 %)	20 (22 %)	0.528	0.012
Nocturnal agonal respiration	2 (1.4 %)	0 (0 %)	1 (7.7 %)	1 (2.4 %)	0.236	0.498
Atrial fibrillation	3 (2.0 %)	2 (4.8 %)	0 (0 %)	1 (1.1 %)	0.423	0.231
Family history of BrS	34 (23 %)	10 (24 %)	3 (23 %)	21 (23 %)	0.957	0.900
Family history of SCD	24 (16 %)	5 (12 %)	4 (31 %)	15 (16 %)	0.108	0.507
ECG						
Spontaneous type 1 ECG pattern	18 (12 %)	5 (12 %)	0 (0 %)	13 (14 %)	0.324	0.726
Rest HR (bpm)	71 (66–73)	71 (67–75.)	70 (70–72)	70 (66–73)	0.345	0.115
PR interval (ms)	157 (152–167)	157 (150–164)	164 (155–168)	156 (150–166)	0.134	0.673
QRS duration (ms)	107 (102–110)	108 (102–110)	110 (107–112)	107 (101–110)	0.041	0.499
QTc interval (ms)	411 (401–427)	409 (401–433)	423 (404–433)	410 (401–423)	0.475	0.245
Genetic test						
SCN5A	18 (12 %)	7 (17 %)	5 (38 %)	6 (6 %)	0.096	0.066
Negative	129 (88 %)	35 (83 %)	8 (62 %)	86 (93 %)	–	–
PVS						
Performed	67 (46 %)	17 (40 %)	11 (85 %)	39 (42 %)	0.010	0.835
Positive	11 (16 %)	0 (0 %)	10 (91 %)	1 (2.4 %)	<0.001	0.516

Data are shown as median (interquartile range) or as number (percentage).
BrS = Brugada syndrome; ECG = elettrocardiogram; ICD = implantable cardioverter defibrillator; ILR = implantable loop recorder; PVS = programmed ventricular stimulation; SCD = sudden cardiac death; VF = ventricular fibrillation; VT = ventricular tachycardia.

Table 2
Details on patients with arrhythmic findings detected by ILR.

Age at ILR insertion (years)	Sex	BrS type 1 ECG pattern	Risk factors	Arrhythmic finding	Medical intervention
39	Male	Drug-induced	o History of AF	AF	Transcatheter AF ablation
34	Male	Drug-induced	o Suspected arrhythmic syncope	New-onset AF	–
62	Male	Drug-induced	o Family history of BrS	Asymptomatic non-sustained VT	–
23	Male	Drug-induced	o History of AF	AF	–
52	Female	Drug-induced		Asymptomatic non-sustained VT	–
60	Male	Drug-induced	o Suspected arrhythmic syncope o Family history of SCD	Asystolic pause (16 s)	ICD implantation
63	Male	Spontaneous		Symptomatic paroxysmal SVT	Nodal re-entry transcatheter ablation
53	Female	Drug-induced	o Suspected arrhythmic syncope	Asystolic pause (3.6 s)	–

AF = atrial fibrillation; BrS = Brugada syndrome; ECG = elettrocardiogram; ICD = implantable cardioverter defibrillator; ILR = implantable loop recorder; SCD = sudden cardiac death; VT = ventricular tachycardia.

and 3.6 s, respectively, which were associated with syncope. These findings suggest that patients with BrS may be predisposed to syncope caused by vasovagal mechanisms. Several studies have demonstrated the recurrence of vasovagal syncope with sinus rhythm recorded by ILRs, while ventricular arrhythmias have been rarely reported [11–14]. Kubala et al. studied 11 BrS patients who underwent ILR monitoring and reported that 8 of them experienced recurrent symptoms. Two patients had sinus bradycardia, and two experienced pauses due to atrioventricular blocks, while the remaining 50 % had normal sinus rhythm during their symptoms. The final diagnosis revealed that 75 % of the patients suffered from vasovagal syncope, and 25 % had typical epileptic seizures [15]. Therefore, ILR implantation and monitoring contributed to distinguishing between symptoms caused by vasovagal mechanisms and ventricular arrhythmias. Giustetto et al. conducted a study involving 27 BrS patients who underwent ILR insertion [12]. Among them, 13 patients had neurally mediated syncope, while the remaining 14 had unexplained syncope. The study revealed that none of the patients in either group experienced ventricular arrhythmic events or sudden death after the ILR insertion. However, one patient with an ILR experienced a syncope episode accompanied by a 24-second asystolic pause [12]. Sakhi et al. prospectively assessed 20 BrS patients who experienced symptoms such as syncope and/or palpitations and underwent ILR insertion. During a median follow-up period of 32 months, there were no cases of sudden death or sustained ventricular arrhythmia observed among the patients. However, one patient underwent AF ablation, and another patient underwent pacemaker implantation for sinus arrest and atrioventricular block [13]. More recently, Scrocco et al. conducted a retrospective analysis of 50 BrS patients with ILR, reporting actionable arrhythmic events in 22 % of patients, which aligns with our observations. These events included sinus node defects and supraventricular tachycardias [16]. Our findings align with previous studies in this population, contributing additional data to the existing literature that evaluates the efficacy of an ILR in patients with BrS. Within our study cohort, the majority of patients did not exhibit a spontaneous type

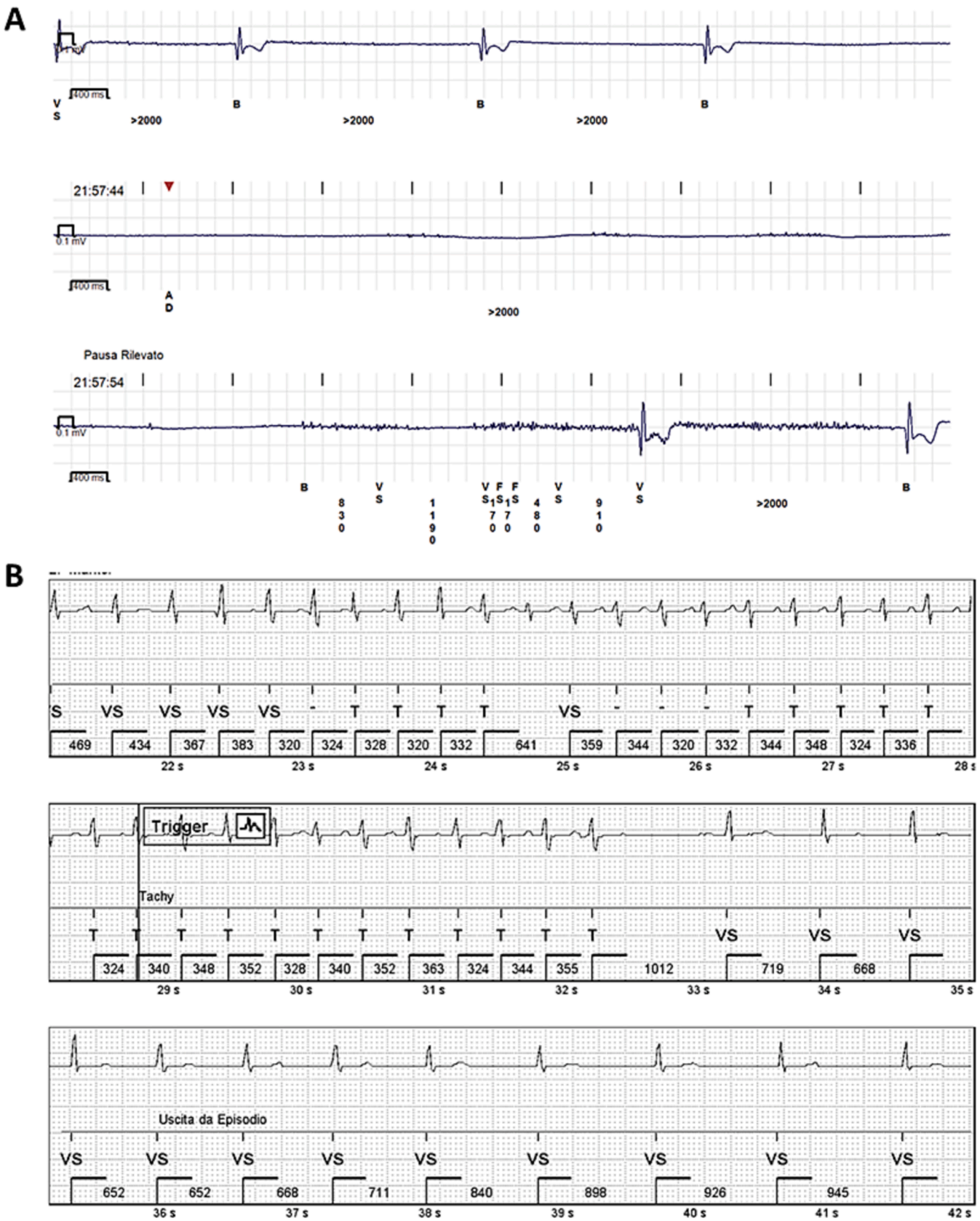

Fig. 1. Episodes of asystolic pause (panel A) and paroxysmal supraventricular tachycardia (panel B) recorded by the implanted loop recorder in two study patients.

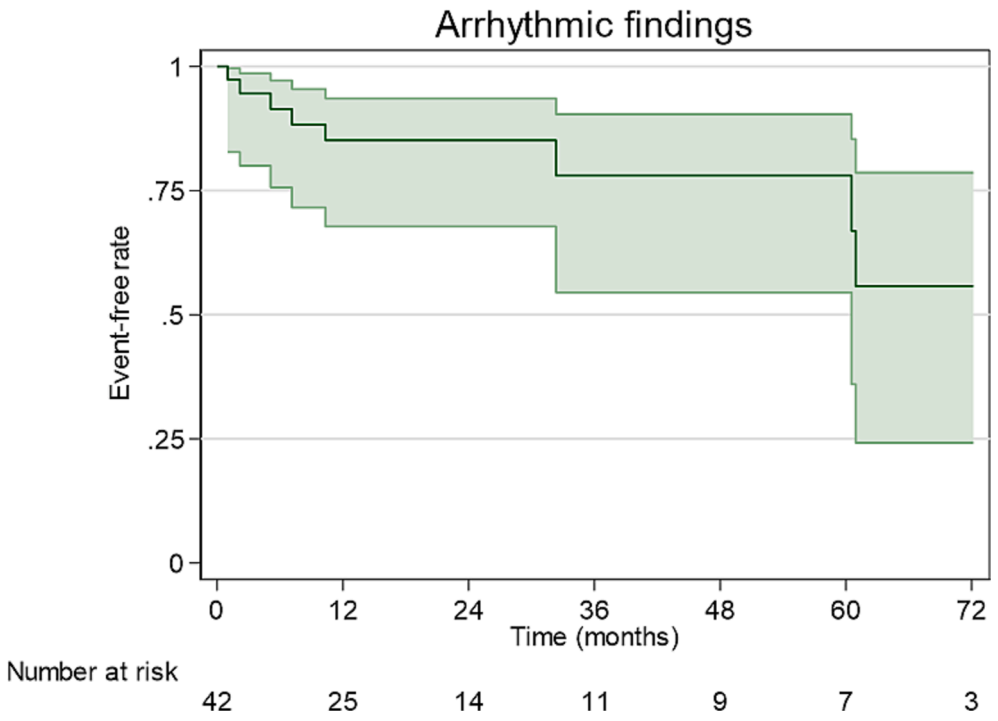

Fig. 2. Kaplan-Meier curve with 95% confidence interval for arrhythmic findings in patients who received an implantable cardiac monitor.

Table 3
Patients with ILR by arrhythmic finding.

Characteristic	Without arrhythmia N = 34	With arrhythmia N = 8	P value
Sex, female (n, %)	13 (38 %)	2 (25 %)	0.689
Age (years)	45 (36–54)	43 (35–55)	0.322
Height (cm)	175 (172—178)	172 (169—175)	0.236
Weight (Kg)	76 (70–80)	73 (70–78)	0.129
History of			
Cardiac arrest	0 (0 %)	0 (0 %)	–
Polymorphic VT or VF	0 (0 %)	0 (0 %)	–
Suspected arrhythmic syncope	15 (44 %)	3 (38 %)	0.734
Nocturnal agonal respiration	0 (0 %)	0 (0 %)	–
Atrial fibrillation	0 (0 %)	2 (25 %)	0.033
Family history of BrS	9 (27 %)	1 (13 %)	0.655
Family history of SCD	4 (12 %)	1 (13 %)	0.954
ECG			
Spontaneous type 1 ECG pattern	4 (12 %)	1 (13 %)	0.954
Rest HR (bpm)	70 (70–72)	70 (66–73)	0.345
PR interval (ms)	164 (155–168)	156 (150–166)	0.134
QRS duration (ms)	110 (107–112)	107 (101–110)	0.041
QTc interval (ms)	423 (404–433)	410 (401–423)	0.475
Genetic test			
SCN5A	7 (21 %)	0 (0 %)	0.312
Negative	27 (79 %)	8 (100 %)	–
PVS			
Performed	16 (47 %)	1 (13 %)	0.010
Positive	0 (0 %)	0 (0 %)	–

Data are shown as median (interquartile range) or as number (percentage). Abbreviations as in Table 1.

1 ECG pattern, suggesting a lower arrhythmic risk category and potentially explaining the absence of sustained ventricular events. Extrapolating clinical implications from these observations may pose challenges, as many of the detected arrhythmic events could not be directly attributed to the ILR indication, and, in some instances, their clinical impact may be limited. Hence, larger multicenter registries are required to establish the utility and benefits of ILR in this particular population.

Several limitations should be considered when interpreting the findings of this study. Firstly, the single-center design may limit the generalizability of the results, as the patient population and healthcare practices may not fully represent the diversity of the broader population or healthcare settings. Secondly, the retrospective nature of the study introduces potential bias and limits the control over data collection and patient selection. Thirdly, the small sample size might reduce the statistical power and precision of the estimates.

5. Conclusions

Continuous ECG monitoring with ILR may help in the early detection of non-malignant rhythm disorders, including sinus node dysfunction and supraventricular tachycardias, in BrS patients with risk factors but without an indication for primary prevention ICD implantation. Given the low risk of device-related complications, this approach could be an option worth considering in these patients.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

CRediT authorship contribution statement

Gianmarco Arabia: Writing – original draft, Investigation, Data curation, Conceptualization. **Manuel Cerini:** Writing – review & editing, Investigation, Data curation. **Angelica Cersosimo:** Writing – review & editing, Investigation, Data curation. **Paolo Vinciguerra:** Writing – review & editing, Investigation, Data curation. **Emiliano Calvi:** Writing – review & editing, Investigation, Data curation. **Gianfranco Mitacchione:** Writing – review & editing, Investigation, Data curation. **Mohamed Aboelhassan:** Writing – review & editing, Validation. **Daniele Giacopelli:** Writing – review & editing, Formal analysis. **Antonio Curnis:** Writing – review & editing, Supervision, Investigation.

Declaration of competing interest

D.G. is employee of Biotronik Italia. The remaining authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] K. Zeppenfeld, J. Tfelt-Hansen, M. de Riva, B.G. Winkel, E.R. Behr, N.A. Blom, P. Charron, D. Corrado, N. Dagres, C. de Chillou, L. Eckardt, T. Friede, K.H. Haugaa, M.H ocini, P.D. Lambiase, E. Marijon, J.L. Merino, P. Peichl, S.G. Priori, T. Reichlin, J. Schulz-Menger, C. Sticherling, S. Tzeis, A. Verstrael, M. Volterrani, ESC Scientific Document Group. 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death, Eur. Heart J. 2022;43(40):3997-4126.

[2] J. Brugada, O. Campuzano, E. Arbelo, G. Sarquella-Brugada, R. Brugada, Present status of brugada syndrome: JACC state-of-the-art review, J. Am. Coll Cardiol. 72 (9) (2018) 1046–1059.

[3] J. Sieira, G. Conte, G. Ciconte, G.-B. Chierchia, R. Casado-Arroyo, G. Baltogiannis, G. Di Giovanni, Y. Saitoh, J. Julia´, G. Mugnai, M. La Meir, F. Wellens, J. Czapla, G. Pappaert, C.D. Asmundis, A. Brugada, A score model to predict risk of events in patients with Brugada syndrome, Eur. Heart J. 38 (2017) 1756–1763.

[4] S. Kawada, H. Morita, C. Antzelevitch, Y. Morimoto, K. Nakagawa, A. Watanabe, N. Nishii, K. Nakamura, H. Ito, Shanghai score system for diagnosis of Brugada syndrome: validation of the score system and system and reclassification of the patients, JACC Clin. Electrophysiol. 4 (2018) 724–730.

[5] C. Antzelevitch, G.-X. Yan, M.J. Ackerman, M. Borggrefe, D. Corrado, J. Guo, I. Gussak, C. Hasdemir, M. Horie, H. Huikuri, C. Ma, H. Morita, G.-B. Nam, F. Sacher, W. Shimizu, S. Viskin, A.A.M. Wilde, J-wave syndromes expert consensus conference re- port: emerging concepts and gaps in knowledge, Heart Rhythm 13 (2016) e295–e324.

[6] S. Iacopino, G.B. Chierchia, P. Sorrenti, F. Pesce, J. Colella, G. Fabiano, G. Campagna, A. Petretta, F. Placentino, P. Filannino, P. Artale, D. Giacopelli, G. Santarpino, A. Sorgente, P. Brugada, C. de Asmundis, dST-tiso interval, a novel electrocardiographic marker of ventricular arrhythmia inducibility in individuals with Ajmaline-induced Brugada type I pattern, Am. J. Cardiol. 159 (2021) 94–99.

[7] G. Hamilton, D. O'Donnell, H.C. Han, Brugada syndrome and undifferentiated syncope: use of an implantable loop recorder to document causation, Med. J. Aust. 209 (2018) 113–114.

[8] G. Mascia, R. Della Bona, P. Ameri, M. Canepa, I. Porto, M. Brignole, Brugada syndrome and syncope: a systematic review, J. Cardiovasc. Electrophysiol. 31 (2020) 3334–3338.

[9] M. Brignole, A. Moya, F.J. de Lange, J.C. Deharo, P.M. Elliott, A. Fanciulli, A. Fedorowski, R. Furlan, R.A. Kenny, A. Martín, V. Probst, M.J. Reed, C.P. Rice, R. Sutton, A. Ungar, J.G. van Dijk, ESC Scientific Document Group. 2018 ESC Guidelines for the diagnosis and management of syncope, Eur. Heart J. 39(21) (2018) 1883-1948.

[10] C. Balfe, R. Durand, D. Crinion, D. Ward, R. Sheahan, The evidence for the implantable loop recorder in patients with inherited arrhythmia syndromes: a review of the literature, Europace. 24 (5) (2022) 706–712.

[11] J. Champagne, F. Philippon, M. Gilbert, F. Molin, L. Blier, I. Nault, J.F. Sarrazin, L. Charbonneau, L. Dufort, B. Drolet, M. Chahine, G.E. O'Hara, The Brugada syndrome in Canada: a unique French-Canadian experience, Can J. Cardiol. (2007).

[12] C. Giustetto, S. Drago, P.G. Demarchi, P. Dalmasso, F. Bianchi, A.S. Masi, P. Carvalho, E. Occhetta, G. Rossetti, R. Riccardi, R. Bertona, F. Gaita, Italian Association of Arrhythmology and Cardiostimulation (AIAC)-Piedmont section. risk stratification of the patients with Brugada type electrocardiogram: a community-based prospective study, Europace 11 (2009) 507–513.

[13] R. Sakhi, A. Assaf, D.A.M.J. Theuns, J.M.A. Verhagen, T. Szili-Torok, J.W. Roos-Hesselink, S.C. Yap, Outcome of insertable cardiac monitors in symptomatic patients with Brugada syndrome at low risk of sudden cardiac death, Cardiology 145 (2020) 413–420.

[14] M. Brignole, R. Sutton, C. Menozzi, R. Garcia-Civera, A. Moya, W. Wieling, D. Andresen, D.G. Benditt, P. Vardas, Early application of an implantable loop recorder allows effective specific therapy in patients with recurrent suspected neurally mediated syncope, Eur Heart J 27 (2006) 1085–1092.

[15] M. Kubala, L. A¨ssou, S. Traulle´, A.L. Gugenheim, J.S. Hermida, Use of implantable loop recorders in patients with Brugada syndrome suspected risk of ventricular arrhythmia. Europace 14 (2012) 898–902.

[16] C. Scrocco, Y. Ben-Haim, B. Devine, M. Tome-Esteban, M. Papadakis, S. Sharma, P. W. Macfarlane, E.R. Behr, Role of subcutaneous implantable loop recorder for the diagnosis of arrhythmias in Brugada syndrome: A United Kingdom single-center experience, Heart Rhythm. 19 (1) (2022) 70–78.