ORIGINAL ARTICLE

https://doi.org/10.1590/1806-9282.20230322

Electrophysiological study in chagasics with syncope and conduction disorder

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SUMMARY

BACKGROUND: Investigation of syncope involves the use of electrophysiological study, particularly in patients with cardiac conduction disorder. There is conflicting evidence about the role of electrophysiological study in patients with Chagas disease.

OBJECTIVE: The objective of this study was to evaluate the electrophysiological study findings in patients with Chagas disease and bundle branch block and/or divisional block presenting with syncope.

METHODS: This is a retrospective study of patients with Chagas disease and cardiac conduction disorder who underwent electrophysiological study from 2017 to 2021 for the investigation of syncope in a tertiary hospital in São Paulo, Brazil. Those with non-interpretable ECG, known coronary artery disease, and/or other cardiomyopathies were excluded. HV interval and electrophysiological study-induced malignant ventricular arrhythmias data were analyzed.

RESULTS: A total of 45 patients (60.2±11.29 years, 57.8% males) were included. The mean HV interval was 58.37 ms±10.68; 22.2% of the studied population presented an HV interval of ≥70 ms; and malignant ventricular arrhythmias were induced in 57.8% patients. The use of beta-blockers and amiodarone (p=0.002 and 0.036, respectively), NYHA functional class≥II (p=0.013), wide QRS (p=0.047), increased HV interval (p=0.02), Rassi score >6.5 (p=0.003), and reduced left ventricular ejection fraction (p=0.031) were associated with increased risk of inducible malignant ventricular arrhythmias.

CONCLUSION: More than half of the patients with Chagas disease, syncope, and cardiac conduction disorder have inducible malignant ventricular arrhythmias. Prolonged HV interval was observed in only 20% of population. Wide QRS, prolonged HV, reduced ejection fraction, and higher Rassi score were associated with increased risk of malignant ventricular arrhythmias.

KEYWORDS: Chagas disease. Electrophysiological study. Syncope. Bundle branch block.

INTRODUCTION

Chagas disease (CD) is an endemic disease in Latin America. It affects approximately 18–20 million individuals and is responsible for high rates of morbidity and early mortality[1]. Approximately 30–40% of the infected population develop the cardiac form, with a worse prognosis, which may manifest with heart failure (HF) symptoms, cardiac arrhythmias, or thromboembolism[2].

The chronic inflammatory disease caused by the presence of parasite may result in sinus dysfunction and cardiac conduction system abnormalities. Myocardial fibrosis is the substrate for reentrant circuits and the main mechanism of malignant ventricular arrhythmias (MVA) and sudden death (SD) even in patients without HF or severe left ventricular dysfunction. In patients with chagasic cardiomyopathy, syncope may be a consequence of ventricular tachycardia (VT), ventricular fibrillation (VF), atrioventricular block (AVB), sinus node dysfunction, or neuromodulated mechanisms[3].

In patients with unexplained syncope and bifascicular branch block, a permanent pacemaker (PM) is indicated in cases of HV interval≥70 ms and/or second- or third-degree AVB during atrial stimulation or pharmacological testing[4].

Investigation of syncope involves the use of electrophysiological study (EPS), particularly in patients with cardiac conduction disorder (CCD). There is conflicting evidence about EPS in patients with CD. The purpose of this study was to evaluate the EPS findings in patients with CD and bundle branch block and/or divisional block presenting with syncope.

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Conflicts of interest: the authors declare there is no conflicts of interest. Funding: none.
Received on August 21, 2023. Accepted on August 22, 2023.

METHODS

All the participants signed a written informed consent form. In November 2021, the protocol number 4893/2018 was approved by the local ethics committee.

This retrospective study included consecutive patients with CD and CCD who underwent EPS for the investigation of syncope in a tertiary hospital in São Paulo, Brazil, from 2017 to 2021. HV interval and EPS-induced MVA. Clinical data, electrocardiographic, echocardiographic, and 24-h Holter findings were obtained.

Inclusion and exclusion criteria

Patients with CD at any age, syncope, and bundle branch block and/or divisional block who underwent EPS were included in the analysis. Those with non-interpretable ECG and/or other cardiomyopathies such as hypertrophic, valvopathy, and right ventricular dysplasia were excluded. Patients with coronary artery disease defined by either symptoms of angina pectoris and/or dyspnea on exertion associated with ≥50% obstruction of the vascular lumen of epicardial coronary arteries on cineangiocoronariography or myocardial Ischemia on non-invasive exam as well as patients with previous MI with reduced LV function/scar were also excluded.

For EPS, the following protocol was used: ventricular stimulation with two basic cycles and up to three extrastimuli as well as rapid ventricular stimulation (up to 250 ms or 2:1 ventricular capture) in both apex and right ventricular outflow tract. VMA was classified into sustained VT and VF according to the definitions of current guidelines[5]. HV interval was measured from the beginning of the His bundle potential deflection (H) to the earliest onset of ventricular activity (V).

Statistical analysis

Quantitative variables were presented by means, standard deviations, and minimum and maximum values, and categorical variables were presented by frequencies and percentages.

For univariate analysis, Fisher's exact test or chi-square test was used for categorical variables, and for those with a quantitative character, Student's t-test for independent samples or Mann-Whitney's non-parametric test was used. The normal condition of the quantitative variables was assessed using Kolmogorov-Smirnov's test.

As for the multivariate analysis, a logistic regression model was adjusted as explanatory variables showed significance in the univariate analysis. The stepwise backward method was used to reduce the model. For model adjustment, Hosmer-Lemeshow's test was applied and the value of the area under the C-statistic [receiver operating characteristic (ROC) curve] was estimated. $p<0.05$ was considered statistically significant. Data were analyzed using IBM SPSS Statistics for Windows, Version 19.0 (Released 2010, Armonk, NY: IBM Corp.) and R Core v3.6.3.

RESULTS

A total of 62 patients with syncope, Chagas disease, and CCD undergoing EPS were evaluated, of whom 17 were excluded due to incomplete records. Therefore, 45 patients were included in the analysis.

The mean age was 60.2±11.29 years, and 26 (57.8%) were males. Approximately 70% of patients had hypertension and 13.3% were diabetic. Most patients were in NYHA functional class I. The mean Rassi score was 8.53±5.86. The clinical characteristics of the studied population are presented in Table 1.

Most patients were in sinus rhythm, of whom 13.3% were in atrial fibrillation, and 4.4% had paced rhythm on ECG. Almost half of the patients had both right bundle branch block (RBBB) and left anterior fascicular block (LAFB). Left bundle branch block (LBBB) was observed in 20% of patients. The mean QRS duration was 144.8 ms (ranging from 80 to 210 ms). Notably, 51.1% of patients had frequent PVCs (>30/h), 40% had non-sustained VT, and 17.7% sustained VT on 24-h Holter monitoring (Table 1).

The mean LVEF was 45±15.7%, being 40% lower than 40%. Echocardiographic findings are presented in Table 1. The mean LVEF was 51.9 and 39.8% in the groups without and with MVA induction, respectively (p=0.013) (Table 2 and Figure 1).

The mean HV interval was 58.37±10.68 ms. In only 22.2% of patients, HV was ≥70 ms. Ventricular arrhythmias were induced in 57.8% of the sample.

In the univariate analysis, MVA predictors were use of beta-blockers and amiodarone (p=0.002 and 0.036, respectively), HV>70 ms (p=0.02), Rassi score >6.5 (p=0.003), and low LVEF.

For each 10-ms increase in the HV interval, there was a 51% increase in MVA inducibility (p=0.19).

For each 10-ms increase in QRS duration, there was a 29% increase in MVA inducibility (p=0.19). An ROC curve was performed to determine the cutoff point of the QRS interval associated with MVA induction. The value of 127 ms showed a sensitivity of 80.8% and a specificity of 36.8% [area under the curve (AUC) of 0.67 (p=0.04)] (Figure 1).

For each 10-unit decrease in LVEF, an increased risk of 75% in MVA was observed (p=0.01). An ROC curve was performed to determine the cutoff point of LVEF associated with VMA induction. The value of 48% showed a sensitivity of 73.1% and a specificity of 68.4% [AUC of 0.72 (p=0.01)] (Figure 1).

Table 1. Demographic characteristics.

Variable		Mean and N	%
Age (years)		60.27±11.29	
Male		26	57.8
Dyslipidemia		20	44.4
Systemic hypertension		32	71.1
Previous stroke		3	6.7
Diabetes mellitus		6	13.3
Coronary artery disease*		5	11.1
NYHA	I	28	62.2
	II	11	24.4
	III	6	13.3
	IV	0	0.0
Smoking	No	32	71.1
	Former smoker	12	26.7
	Yes	1	2.2
Rassi score		8.53±5.86	
ACEI/ARB		37	82.2
Beta-blockers		26	57.8
Amiodarone		27	60.0
Diuretic		29	64.4
Estatin		23	51.1
AAS		12	26.7
Warfarin		11	24.4
DOAC		3	6.7
Electrocardiographic findings			
Sinus rhythm		37	82.2
Atrial fibrillation		6	13.3
Pacemaker		2	4.4
Right bundle branch block		9	20.0
Left anterior fascicular block		5	11.1
RBBB+LAFB		22	48.8
LBBB		9	20
First-degree AV block		15	33.3
Second-degree AV block		2	4.4
Holter findings			
PVC>30/h		23	51.1
Non-sustained VT		18	40.0
Sustained VT		8	17.7
Echocardiogram findings			
LVEF mean (SD)		45%	15.7
Left ventricular thrombus		0	0
Left ventricular aneurysm		7	15.6
Left atrial volume≥32 mL/m²		43	95.5

ACEI/ARB: angiotensin-converting enzyme inhibitor/angiotensin receptor block; AV: atrioventricular; DOAC: direct oral anticoagulant; LAFB: left anterior fascicular block; LBBB: left bundle branch block; LVEF: left ventricular ejection fraction; PVC: premature ventricular contraction; RBBB: right bundle branch block; SD: standard deviation; VT: ventricular tachycardia. *This includes mild CAD patients (<50% obstruction of the vascular lumen of epicardial coronary arteries on cineangiocoronariography) or previous MI without compromised LV function/scar.

Finally, a Rassi score of 6.5 presented a sensitivity of 75.9% and a specificity of 75%, for VMA induction [AUC of 0.77 (p=0.003)] (Figure 1).

DISCUSSION

In this study of patients with Chagas disease and conduction disorder, MVA induction on EPS was the main factor associated with the occurrence of syncope. This finding might help physicians in the decision-making by indicating an ICD instead of a PM for this specific population.

Syncope in patients with CD and cardiac involvement is an alert situation, assuming that the main etiology is ventricular arrhythmia[6]. However, other causes such as paroxysmal AVB should be considered, with a more favorable prognosis[7].

Unlike other conditions, vasovagal syncope in patients with CD is not always benign, once cardiac dysautonomia is related to reduced baroreflex sensitivity and the occurrence of complex ventricular arrhythmias[8]. Autonomic dysfunction may occur before ventricular dysfunction in CD, and this was demonstrated by myocardial scintigraphy with Iodine-123-labeled metaiodobenzylguanidine (123I-MIBG). In a study of patients with CD and normal or slightly reduced LV function, the presence of ventricular arrhythmias was associated with more extensive areas of viable and denervated myocardium identified by 123I-MIBG[9].

The Rassi score is widely used for mortality prediction in CD. Its elaboration was based on a systematic review of 12 studies that did not include syncope[10]. Syncope inclusion as a risk factor would probably increase the score sensitivity without changing its simplicity. In this study, a Rassi score of 6.5 was associated with 75.9% sensitivity and 75% specificity for MVA induction.

Few studies have assessed the value of EPS in patients with CD. Leite et al.[11] analyzed chagasics with spontaneous sustained VT despite the use of class III antiarrhythmics. Those who presented unstable VT had a worse prognosis compared with patients in whom VT was either hemodynamically tolerated or not induced. In most individuals with preserved LVEF and either no spontaneous arrhythmias or NSVT on 24-h Holter monitoring, EPS does not provide relevant prognostic information[12,13]. In this study, patients underwent EPS for syncope investigation according to the recommendations of current guidelines[14], after inconclusive non-invasive evaluation.

In the study published by Silva et al., EPS-induced VMA was a predictor of arrhythmogenic death and all-cause mortality[15]. The pathophysiological mechanism involves the presence of regional fibrosis, particularly in the left ventricular posterior-lateral wall, and results in reentrant circuits[16-19]. In our study,

Table 2. Clinical characteristics and VMA induction.

	VMA induction		p-value
	No	Yes	
Male (26)	9 (34.6%)	17 (65.4%)	0.28
Smoking (13)	6 (46.2%)	7 (53.8%)	0.41
Dyslipidemia (20)	11 (55%)	9 (45%)	0.12
Hypertension (32)	15 (46.9%)	17 (53.1%)	0.32
Stroke (3)	0	3 (100%)	0.12
Diabetes mellitus (6)	2 (33.3%)	4 (66.7%)	0.64
CAD (5)*	1 (20%)	4 (80%)	0.29
NYHA			0.034
I (28)	16 (57.1%)	12 (42.9%)	0.013
II and III (17)	3 (17.6%)	14 (82.4%)	0.013
ACEI/ARB (37)	17 (45.9%)	20 (54.1%)	0.277
Beta-blockers (26)	6 (23.1%)	20 (76.9%)	0.002
Amiodarone (27)	8 (29.6%)	19 (70.4%)	0.036
Diuretic (29)	11 (37.9%)	18 (62.1%)	0.433
Statin (23)	11 (47.8%)	12 (52.2%)	0.436
Aspirin (12)	4 (33.3%)	8 (66.7%)	0.467
Warfarin (11)	6 (54.5%)	5 (45.5%)	0.341
DOAC (3)	2 (66.7%)	1 (33.3%)	0.375
Sinus rhythm (37)	14 (37.8%)	23 (62.2%)	0.4
Atrial fibrillation (6)	4 (66.7%)	2 (33.3%)	0.4
RBBB (31)	16 (51.6%)	15 (48.4%)	0.58
LBBB (9)	2 (22.2%)	7 (77.8%)	0.17
LAFB (27)	13 (48.1%)	14 (51,9%)	0.32
First-degree AV block (15)	5 (33.3%)	10 (66.7%)	0.39
Second-degree AV block (2)	1 (50%)	1 (50%)	0.82
PVC>30/H (23)	7 (30.4%)	16 (69.6%)	0.1
NSVT (18)	7 (38.9%)	11 (61.1%)	0.71
HV			0.02
<70 ms (31)	16 (51.6%)	15 (48.4%)	0.02
≥70 ms (10)	1 (10%)	9 (90%)	0.02
Left ventricular aneurysm (7)	2 (28.6%)	5 (71.4%)	0.43
Mean LVEF (%) SD	51.9% (±14.7)	39.8% (±14.6)	0.013
Left atrial volume			
≥32 (43)	19 (44.1%)	24 (55.8%)	0.747

ACEI/ARB: angiotensin-converting enzyme inhibitor/angiotensin receptor block; AV: atrioventricular; CAD: coronary arterial disease; DOAC: direct oral anticoagulant; LAFB: left anterior fascicular block; LBBB: left bundle branch block; LVEF: left ventricular ejection fraction; NSVT: non-sustained ventricular tachycardia; PVC: premature ventricular contraction; RBBB: right bundle branch block; SD: standard deviation; VT: ventricular tachycardia. *This includes mild CAD patients (<50% obstruction of the vascular lumen of epicardial coronary arteries on cineangiocoronariography) or previous MI without compromised LV function/scar.

more than half of the patients had VMA induction. This finding is in accordance with the data published by Martinelli et al., in which the most prevalent cause of syncope in chagasics was VMA (43%) followed by paroxysmal AVB (21%)[7].

In this study, the use of amiodarone and beta-blockers, NYHA functional class>I, reduced LVEF (<50%), QRS duration, and prolonged HV interval (>70 ms) were found to be predictors of VMA.

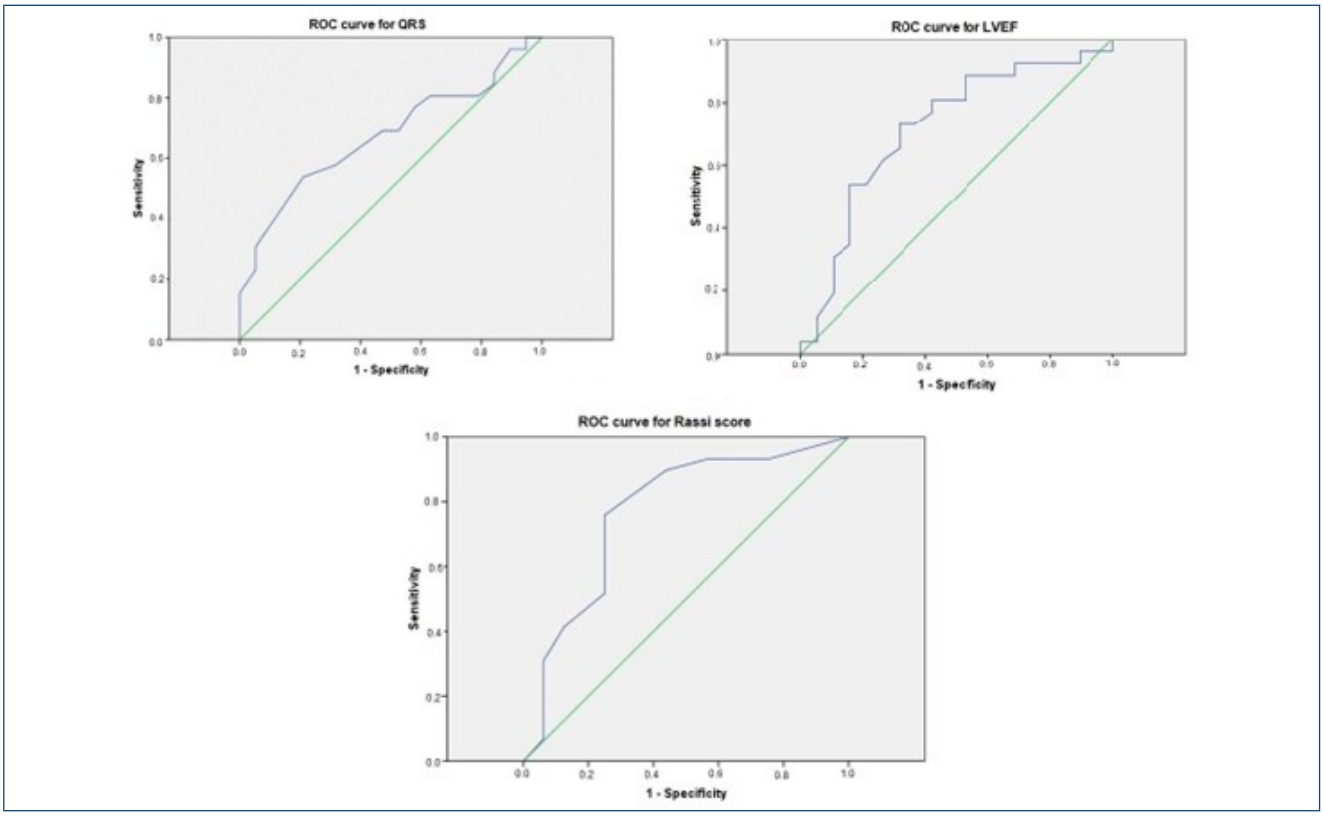

Figure 1. Receiver operating characteristic curve for QRS, left ventricular ejection fraction, and Rassi score values in the prediction of VMA.

The higher occurrence of VMA in patients under amiodarone treatment may reflect the previous diagnosis of ventricular arrhythmias and, consequently, the greater severity of these patients. The same finding was observed in the study of Cardinalli et al.[20], in which amiodarone therapy was an independent risk of VMA.

Ventricular dysfunction is also a predictor of ventricular arrhythmias in patients with CD. For each 10-unit decrease in LVEF, we found a 75% increase in the risk of VMA induction. LVEF of 48% had a sensitivity of 73% and a specificity of 68.4% for VMA induction. On the contrary, there was no association between the density of ventricular arrhythmias on 24-h Holter and VMA induction, which is different from the publication of Souza et al.[6], in which the presence of syncope, QT interval, ventricular dysfunction, and ventricular ectopies were the predictors of sudden cardiac death in patients with CD.

Wide QRS complex and prolonged HV interval are the markers of structural heart disease (fibrosis and/or ventricular dysfunction), which reflect slower and nonsynchronized ventricular depolarization, a substrate for reentrant circuits. For each 10-ms increase in QRS duration, we showed a 29% increase in VMA inducibility. QRS interval >127 ms was associated with a sensitivity of 80.8% of VMA induction. Although not statistically significant, for each 10-ms increase in the HV interval, there was a 51% increase in VMA inducibility.

Prolonged HV interval is a controversial risk factor for the development of AV block. Studies have shown that HV>70 ms is associated with a higher risk of AV block, especially in symptomatic patients. HV>100 ms identifies a group of very high risk of AV block (25% in 22 months)[21]. In the presence of RBBB with or without fascicular block, HV is normal as long as the conduction through the left bundle branch is unchanged. However, 50% of patients with RBBB are combined with anterior superior divisional block and 75% with LBBB have HV interval prolongation[22]. Although our sample was composed of patients with CCD, only 22.2% had prolonged HV interval (11% of patients with LBBB and 34.8% with RBBB). In these cases, the etiology of syncope is multifactorial and may be secondary to paroxysms of AVBs, sinus node disease, dysautonomia, or VMA.

The main limitations of this study are the inclusion of a single center, the retrospective nature, and the small sample. Despite these, we were able to demonstrate that, similar to previous publications, even in the presence of intraventricular conduction abnormalities, VMA is the main cause of syncope in patients with Chagas disease.

CONCLUSION

More than half of patients with Chagas disease, syncope, and CCD have inducible VMA. Prolonged HV interval was observed in only 20% of the population. Wide QRS, prolonged HV, reduced ejection fraction, and higher Rassi score were associated with an increased risk of VMA. Larger studies are needed to confirm the findings.

AUTHORS' CONTRIBUTIONS

AHD: Data curation, Investigation, Project administration, Writing – original draft. **LA:** Investigation, Project administration, Writing – review & editing. **DARM:** Writing – review & editing. **RDL:** Writing – review & editing. **BPV:** Writing – review & editing.

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