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Utility of Cardiac Implantable Electronic Devices in Patients with Chagas Disease and Systolic Heart Failure

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Abstract

Chagas disease (CD) is the principal cause of congestive heart failure (CHF) in areas where the disease is endemic and migration has increased the likelihood of these diseases being the probable cause of CHF in other countries of the world. Sudden cardiac death (SCD) is the most common cause of death in CD (55–65%). Implantable cardioverter defibrillator (ICD) is useful in the secondary prevention of SCD, but there is less information regarding primary prevention. The evidence supporting the use of cardiac resynchronization therapy (CRT) in CHF of chagasic etiology is poor; however, one should apply current guidelines regarding the insertion of these devices in patients with Chagas disease and CHF.

Keywords: Chagas disease, congestive heart failure, sudden cardiac death, implantable cardioverter defibrillator, cardiac resynchronization therapy

1. Introduction

Chagas disease (CD), also known as American trypanosomiasis, was discovered by Carlos Chagas in 1909, is caused by infection with the protozoa *Trypanosoma cruzi* (*T. cruzi*). CD had become widely recognized by the World Health Organization as a neglected tropical disease [1]. *T. cruzi* may be transmitted through blood transfusion, organ transplantation, congenital transmission, or ingestion of contaminated food [2, 3]. However, *T. cruzi* infection most often occurs via vectorial transmission by a type of reduviid bug called a triatomine. *T. cruzi* is excreted in the feces of an infected triatomine bug onto human skin or near mucous membranes. The parasites breach the dermis through excoriations in the skin and gain systemic access [4]. Inoculation is followed by an incubation period of 1 to 2 weeks; it is characterized by parasitemia and subsequent immune response; and 10–30% of infected individuals will begin to

exhibit nonspecific symptoms of acute CD, including abdominal pain, anorexia, fever, lymphadenopathy, rash, malaise and localized swelling around the site of infection [5]. However, the majority of individuals become asymptomatic carriers of *T. cruzi* or indeterminate phase.

Approximately one-third of patients progress to the determinate phase in which cardiac symptoms and signs arise from progressive myofibril fibrosis and conduction system injury [6]. This phase begins after several decades during which there are no clinically overt symptoms of organ damage or abnormal electrocardiographic results; 30–40% of asymptomatic carriers will develop chronic CD characterized by dilated cardiomyopathy leading to congestive heart failure (CHF) and/or by development of gastrointestinal disorders [7].

The Pan American Health Organization (PAHO) estimates between 8 and 12 million people seropositive [8]. Annual deaths are of less variable, ranging from 10,600 to 12,500 [9]. In Latin American countries, 100 million people are at risk of infection and 300,000 new cases are reported each year [10].

The United States (US) Centers for Disease Control and Prevention have reported more than 300,000 immigrants living in the US infected with *T. cruzi* [11]. In 2010 CD was responsible for 550,000 (274,000–1,069,000) disability-adjusted life years (DALYs), a measure that captures both premature mortality and nonfatal health loss [12].

The diagnosis of chronic CD should be suspected in patients from endemic areas (Central and South America) with dilated cardiomyopathy or electrocardiographic abnormalities like right bundle-branch block associated or not with left anterior hemiblock (LAHB). Definitive diagnosis is based on serology to detect immunoglobulin G antibodies to *T. cruzi*, using at least two serological tests of different principles. The most commonly used are enzyme-linked immunosorbent assay (ELISA), indirect immunofluorescence (IIF) and indirect hemagglutination (IHA).

2. Sudden cardiac death and Chagas disease

Sudden cardiac death (SCD) is the most common cause of death in CD (55–65%), followed by congestive heart failure (CHF) (25–30%) and cerebral or pulmonary embolism (10–15%) [13]. Although SCD may affect asymptomatic patients, it affects patients with evidence of chronic heart disease, particularly those with CHF in the majority of cases [14]. The prevalence of SCD in CD patients with CHF is about 46% [15], whereas in a general unselected CD population is 29% [16]. The prevalence also changes in different areas, varying from 29% in non-endemic to 37% in endemic areas [16, 17]. Most SCD cases are in patients with manifest chagasic cardiomyopathy and mainly between 30 and 50 years of age, being rare after the sixth decade of life [14]. On the other hand, up to 20% of patients who die suddenly do not report previous symptoms [14]. SCD may exceptionally occur as a result of rupturing of the left ventricular apical aneurysm, massive cardioembolic stroke, or pulmonary embolism [18, 19]. However, in the overwhelming majority of cases, it is essentially an arrhythmic phenomenon.

In a study of ten chagasic patients who died suddenly with an ambulatory Holter, bradyarrhythmias were the final event in one patient. Ventricular fibrillation (VF) was the final arrhyth-

mia in nine patients; torsades de pointes was the precursor in six and sustained ventricular tachycardia (VT) in three patients [20]. From the studies carried out in Chagas' disease patients receiving implantable cardioverter defibrillator (ICD) therapy, it has become clear that sustained VT is the most frequently observed life-threatening ventricular arrhythmia, although in about 30% of patients who develop VF without having sustained TV as a precursor [21].

The mechanism underlying the tachyarrhythmia episodes in Chagas' disease patients is micro-reentry. There is impressive reparative confluent fibrosis intermingled with normal myocardium. In addition, there is also a diffuse mononuclear cell infiltrate. The association of these two myocardial abnormalities can provoke the appearance of multiple areas of slow conduction in the vicinity of scars, forming foci of reentry disseminated throughout the heart, mainly in the epicardial areas [22, 23]. Another point that deserves further consideration is the autonomic dysfunction; studies in patients with chronic CD have clearly demonstrated parasympathetic derangement in patients with chronic CD [24]. In a study of 52 patients with Chagas cardiomyopathy with pacemaker or ICD, we found more positive serological response against 2e-m2MAChR (antibody that recognizes the muscarinic acetyl choline receptor type II) than in patients with pacemaker or ICD without CD (32.7 vs 3.8% $p < 0.01$) [25].

The risk of SCD is not similar for every patient. Rassi et al. [26] developed and validated a risk score for predicting death in 424 patients followed for a mean of 7.9 years. They identified six independent prognostic factors: New York Heart Association (NYHA) class III or IV (5 points), cardiomegaly on chest radiography (5 points), segmental or global wall motion abnormality on echocardiogram (3 points), non-sustained VT on Holter monitoring (3 points), low QRS voltage (2 points) and male sex (2 points). This score classifies in three groups of risk for 10 years mortality: low risk (0–6 points, 10%), intermediate risk (7–11 points, 44%) and high risk (12–20 points, 84%). In this study the rate of SCD was 2.4% for a year. Although the score risk was development for total mortality, all of the variables were also strong predictors for SCD, except low QRS voltage.

Other risk factors include syncope, spontaneous sustained VT, abnormalities on the 12-lead electrocardiogram or echocardiogram, or sustained TV induced by programmed ventricular stimulation (PVS). In 28 chagasic patients with sustained TV over a mean follow-up of 3816 months, deaths occurred in 13 patients (46.4%) and resulted from SCD in seven subjects [27]. Interestingly, in this study the prognosis was similar with non-sustained VT. The presence on echocardiogram of left ventricular dilatation and left apical ventricular aneurysm was associated with SCD [15]. In the same way, the presence of Q waves, frequent premature ventricular contractions, left anterior fascicular block (LAFB), or QT interval dispersion has been established predictors of SCD [28]. In 78 patients with CD and non-sustained VT, PVS was carried out and sustained monomorphic VT was induced in 25 patients (32%) and VF in four (5.1%). Induction of sustained ventricular arrhythmias was the independent and main variable that predicted cardiac death (OR 2.17 CI 95% 1.23–3.83) [29].

In summary CD is associated with SCD, most commonly through VF, often preceded by sustained VT. There is a higher risk group that may be established by clinical criteria and invasive or noninvasive procedures (**Table 1**).

• Syncope

• NYHA class III–IV

• Male sex

• Spontaneous sustained VT

• Q wave

• Left anterior fascicular block

• QT interval dispersion

• Non-sustained VT

• Frequent premature ventricular contractions

• Cardiomegaly on chest radiography

• Left ventricular dilatation

• Left apical ventricular aneurysm

• Segmental motion abnormality

• Sustained VT induced with PVS

NYHA, New York Heart Association; VT, ventricular tachycardia; PVS, programmed ventricular stimulation.

Table 1. Risk factors associated with sudden cardiac death in chagasic patients.

3. ICD in Chagas disease

3.1. Secondary prevention of SCD

The use of ICD has become a main therapeutic strategy for prevention of sudden death.

A meta-analysis of AVID (Antiarrhythmics versus implantable defibrillators), CIDS (Canadian Implantable Defibrillator Study) and CASH (Cardiac Arrest Study Hamburg) that evaluated utility of ICD in secondary prevention of SCD demonstrated that ICD therapy was associated with 50% ($p = 0.0001$) reduction in arrhythmic mortality and a 28% ($p = 0.006$) reduction in total mortality [30]. Today, ICD is considered class I A recommendation in patients recovered of SCD [31]. However, in these studies there is no information about chagasic patients.

In a study of secondary prevention, 65 chagasic patients were compared with 70 non-chagasic patients and were followed for the median time of 266 days. Appropriate ICD therapy occurred in 32 (49.2%) chagasic patients and in 19 (27.1%) of the control group (p = 0.005). There was a statistically significant difference in event-free survival in the Chagas group. Finally, CD doubles the risk of the patient to have appropriate therapy (HR = 2.2) and appropriate therapy or death (HR 2.2). The annual mortality rate was 17% [32].

Some authors have proposed the use of amiodarone alone for secondary prevention in chagasic patients. The limited evidence available does not favor this hypothesis. A study compared the outcomes of Chagas' heart disease patients with life-threatening ventricular arrhythmias, who were treated with ICD with a historical group treated with amiodarone alone. The ICD

group (76 patients) had higher use of beta-blocker ($p < 0.0001$). Amiodarone was also used in 90% of the ICD group. Therapy with ICD plus amiodarone produced 72% reduced risk of all-cause mortality (p = 0.007) and a 95% reduced risk of sudden death (p = 0.007) compared with amiodarone-only therapy. The follow-up was 36 ± 16 months for the ICD group and 35 ± 17 months for the control group. There are ten deaths (4.7% per year) in the ICD group and nine deaths (11% per year) in the control group [33].

Several studies have evaluated over a long-term follow-up period the efficacy of ICD. A study included 116 consecutive patients with CD and an ICD implanted for secondary prevention. The average follow-up was 45 months. In this survey 58 (50%) patients had appropriate shocks. A total of 31 patients died (7.1% annual mortality rate) [34]. Another study assessed a cohort with 65 patients (51 in secondary prevention) with median follow-up of 40 ± 26.8 months. Among the patients 23 (36.5%) had appropriate shocks. A total of 13 (20%) patients died (6.1% of annual mortality rate) and there was no sudden death [35]. A survey with 90 patients with ICD for secondary prevention found, with median follow-up of 756 ± 581 days, 31 (34%) deaths (16.4% of annual mortality rate) [36]. Maybe the largest study in secondary prevention with chagasic patients evaluated 148 subjects with mean follow-up was 12 ± 7 months. During the follow-up 15 patients died (10.2%) [37].

Patients with chronic CD with life-threatening ventricular arrhythmias have an annual mortality rate between 8.6% and 11% when they are treated with amiodarone [33, 38]. However, survival probability at 3 years of follow-up is 30% in patients with no treatment and 20% treated with quinidine or procainamide [39].

No randomized clinical trial has assessed the effect of ICD therapy on outcome of Chagas' disease patients with malignant ventricular arrhythmias thus far. However, with available information it is obvious that patients without treatment (mortality 70% at 3 years) or with class IA antiarrhythmic (mortality 80% at 3 years, possibly due to antiarrhythmics) have high mortality [39] and need other options. Amiodarone has been used for a long term in these patients, with an annual mortality rate between 8.6 and 11% [33, 38].

ICD is associated with an annual mortality rate between 6.1 and 17% [32–37]. Thus, the impact of ICD implantation on all-cause mortality has shown inhomogeneous results, possibly related to differences among populations and treatments in the studies. The only study that compared ICD plus amiodarone against amiodarone alone demonstrated reduction of the risk of all-cause mortality and sudden death. Patients with left ventricular ejection fraction (LVEF) < 40% derived more survival benefit [33].

Although some authors have suggested the need of a randomized clinical trial to demonstrate the usefulness of ICD in this population [40], most groups working with chagasic patients are extrapolating the secondary prevention ICD indications proposed by international guidelines [31] as reflected by the I Latin American guidelines for the diagnosis and treatment of Chagas heart disease [41].

On the other hand, these patients have differences with other patients with non-Chagas cardiomyopathy. In the meta-analysis of the ICD secondary prevention trials, the authors found that the mean LVEF was 34 ± 15% [30]. Conversely, the mean LVEF among the studies with chagasic patients is higher (37–47%); it is suggesting that CD is more arrhythmogenic heart

disease or less dependent of the ventricular damage [32–37]. Moreover, a substantial number of the included patients had no left ventricular dysfunction.

Another difference between chagasic or non-chagasic patients with ICD for secondary prevention is the age at the time of implantation. In the meta-analysis was 63 ± 11 years, while in chagasic patients was lower (54–59 years) [32–37].

There are differences with life-threatening ventricular arrhythmias; Chagas' disease patients tend to experience more shocks. In a study during the first 6 months of follow-up, 17 of the 20 (85%) chagasic patients received at least one appropriate therapy. In the control group (ischemic patients), 18 of the 35 (51%) received one ICD shock (RR 1.6; $p < 0.02$) [42]. In other studies, the frequency of appropriate shock was 36–64% in the follow-up for 1–2 years [32–37].

Otherwise, several groups have shown predictors of all-cause mortality in patients with Chagas heart disease receiving ICD. The most common predictors of mortality were NYHA class III (HR 3.09 95% CI 1.37-6.98, p = 0.0064), LVEF (HR 0.97 95% CI 0.94-0.99, p = 0.04) and low cumulative right ventricular pacing < 40% (HR 0.23 95% CI 0.11-0.49, p = 0.0001) [34].

In another study the only predictor was the number of shocks; probability of survival for patients receiving more than four shocks by day 30 post-implant was 75% at 30 days and 19% at 60 days, whereas probability of survival for patients receiving up to four shocks by day 30 were 97% at 30 days, 96% at 120 days, 94% at 270 days and 89% at at 360 days of follow-up (p = 0.00005). Mean life expectancy was 2.1 months (95% CI 0.79–3.4) in patients receiving more than four shocks by day 30 and 46.5 months in patients receiving up to four shocks by day 30 (p = 0.0005) [36]. Pereira et al. found predictors of poor prognosis: a LVEF < 30% and low education [35] and another group showed that patients older than 65 years of age and LVEF < 30% were independent predictors of all-cause 1 year mortality [37]. It is important to note that in the studies of secondary prevention, the medical treatment was incomplete for the use of beta-blocker, angiotensin-converting enzyme inhibitors (ACEI)/Angiotensin II receptor blockers (ARB) or spironolactone.

Although several authors have suggested that amiodarone is the initial management in chagasic patients with VT without hemodynamic instability and ICD in other patients [43], most

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Secondary prevention of SCD

• ICD therapy + amiodarone (class I)

Primary prevention of SCD

• ICD therapy in chagasic patients with LVEF < 40% (class I)

Treatment of CHF

• CRT in patients with LVEF < 35% + OMT + LBBB + NYHA class II–IV (class I)

• CRT in patients with LVEF < 35% + OMT + RBBB + NYHA class III–IV (class II B)

SCD, sudden cardiac death; ICD, implantable cardioverter defibrillator; LVEF, left ventricular ejection fraction; OMT, optimal medical treatment; LBBB, left bundle branch block; NYHA, New York Heart Association; RBBB, right bundle branch block.

Table 2. Indications of ICD and CRT in chagasic patients.

groups recommend ICD therapy as an initial approach possibly associated with the use of amiodarone (**Table 2**) [31].

3.2. Primary prevention of SCD

If the evidence, in the management of patients with life-threatening ventricular arrhythmias, is not of great quality, the information is even poor in primary prevention of SCD. As expected, no randomized controlled study has been carried out.

In the only study available, Cardinalli-Neto et al. included 32 patients with a LVEF < 35%, receiving standard therapy for chronic systolic heart failure and to have a reasonable expectation of 1-year survival after device implantation; all patients were on NYHA class II and none had syncope. Nineteen (59%) patients had a positive serology for CD. Notably, 87% of patients were on beta-blocker and 100% were on ACEI/ARB.

Sustained VT was detected in four (21%) patients with Chagas heart disease and in two (15%) patients with non-Chagas (p = NS). VF was observed in four (21%) patients with Chagas cardiomyopathy and in two (15%) with non-Chagas patients (p = NS). Median time to first event was 78 (34–151) days in Chagas and 173 (71–593) days in non-Chagas patients (p = 0.005). Median follow-up was 292 (78–845) days in Chagas and 654 (159–987) days in non-Chagas (p = 0.005) here was no difference in mortality) [44].

Non-sustained VT is an independent predictor of all-cause mortality and SCD in patients with Chagas cardiomyopathy with LVEF from 30 to 50% [45]. In these patients the prognosis role of PVS has been studied. In 78 chagasic patients with mean LVEF 48% and non-sustained VT, electrophysiologic testing was realized. In 25 (32%) out of 78 patients, sustained VT was induced. During a mean follow-up of 56 ± 38 months, 22 (28%) patients died, SCD affecting 16 (73%) of them. A significant association between inducible sustained VT and SCD was found (p < 0.05). All patients induced with sustained VT received amiodarone [29].

Actually, European guidelines recommend using ICD in chagasic patients with LVEF < 40% [31]. However, a significant number of SCD occur in patients with LVEF > 40% and in these subjects, there is no adequate way to establish their risk. PVS might be an option, but new and larger studies are necessary (**Table 2**).

4. Heart failure in Chagas disease

After the acute phase of the disease, most patients enter a clinically asymptomatic chronic phase without electrocardiographic or radiological abnormalities in the heart, which has been described as the indeterminate chronic form. Nevertheless, when these individuals are subjected to echocardiogram or radionuclide, ventriculography is common to find abnormalities; endomyocardial biopsy shows abnormalities in 60% of the cases [46]. Every year 2.5% would evolve into cardiac or digestive symptomatic forms [47]. CHF affects approximately 5% of a general unselected CD population and up to 76% of patients followed at outpatient services in tertiary referral centers [48].

Chagasic cardiomyopathy is a chronic myocarditis that affects all chambers, the parasympathetic cardiac nerves and all levels of the system [49]. Four possible mechanisms have been suggested: cardiac parasympathetic neuronal depopulation, immune-mediated myocardial injury, parasite persistence in cardiac tissue with secondary antigenic stimulation and coronary microvascular abnormalities [25, 49].

CD is the principal cause of CHF in areas where the disease is endemic [50]. Mortality is still high even in the current era of heart failure therapy (around 20% annual) [51]. When compared with other etiologies, outcome is poorer [52].

CD is associated with ventricular conduction delay. In a large population date base on primary case patients, 7590 had CD. The electrocardiogram showed right bundle branch block (RBBB) in 22.7%, left anterior hemiblock (LAHB) in 22.5%, RBBB + LAH in 13,74% and left bundle branch block (LBBB) in 3.07% of patients [53]. In chagasic patients an increase in QRS duration correlated with a decrease in LVEF and increase in left ventricular diastolic diameter [54]. On echocardiographic evaluation, the presence of apical or inferobasal aneurysms in the left ventricle is common (**Figures 1** and **2**).

The beneficial effects on survival and morbidities of drugs observed in non-Chagas disease heart failure are extrapolated to Chagas' disease patients. Medical treatment of CD heart failure is not supported by strong evidence. Small studies have shown that neurohormonal inhibition can improve both symptoms and left ventricular function. However, a systematic review of Cochrane found very low-quality evidence for the effects of carvedilol compared with placebo for treating heart failure in people with CD [55]. On the other hand, chagasic
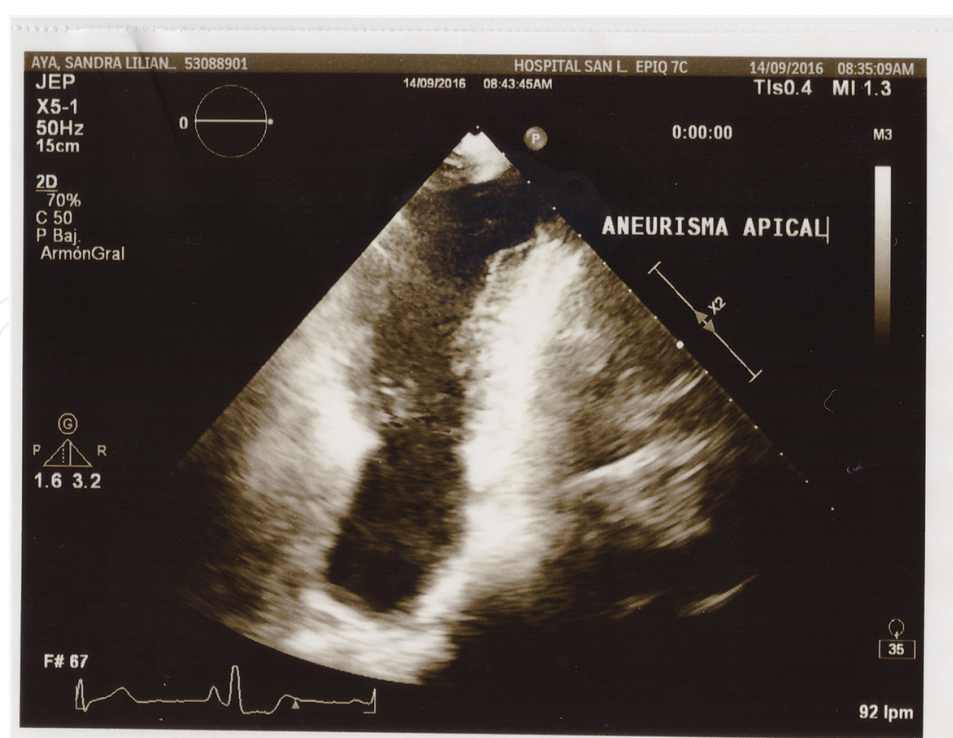

Figure 1. Echocardiogram with apical aneurysm in the left ventricle in a patient with Chagas cardiomyopathy.
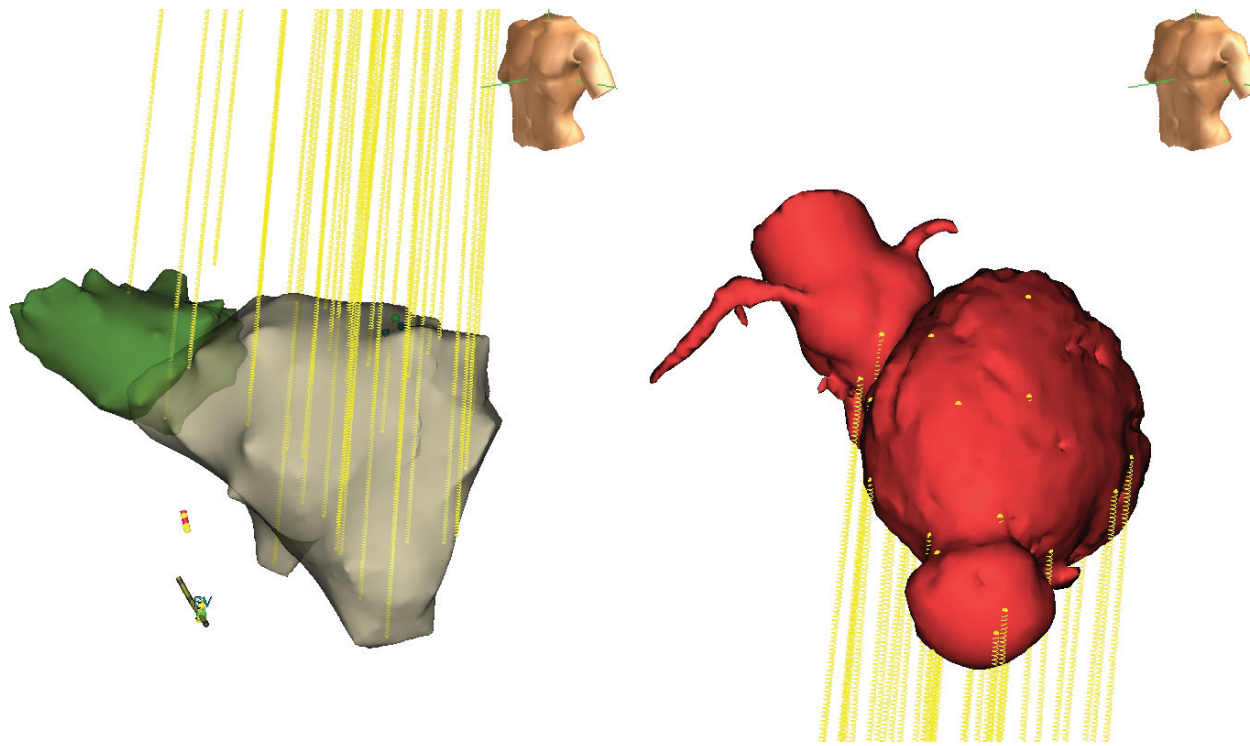
Figure 2. Real-time image integration of the left ventricle with computed tomography and electroanatomical map with EnSite NavX. Image of the left ventricle with apical and inferobasal aneurysm.

patients frequently have lower blood pressure and a higher incidence of bradyarrhythmias and may not tolerate target doses of angiotensin-converting enzyme inhibitors (ACEI) and beta-blockers.

In conclusion CHF secondary to CD has higher mortality and problems with the neurohormonal blockade, related to difficulty in reaching optimum doses of beta-blockers and ACEI.

5. Cardiac resynchronization therapy

Cardiac resynchronization therapy (CRT) is an established therapeutic modality for patients with non-Chagas heart disease with LVEF < 35%, in appropriate medical management and LBBB with a QRS 150 mseg (class I). In patients with non-LBBB pattern, the recommendation is class II [56].

Evidence of the usefulness of CRT in CD is scarce. Araujo et al. analyzed 72 chagasic patients in NYHA class III or IV, who underwent CRT. The average clinical follow-up was 46.6 month (4–79). At the end of the evaluation, 87.4% of patients were in NYHA class I or II and they had increase of the LVEF. There was an overall mortality of 34.7% of the patients underwent implantation of the electrode of the left ventricle through a left anterior mini-thoracotomy. All patients were on beta-blocker therapy and 70% on ACEI. Mean QRS duration was 148.1 17 mseg, 47% was with LBBB, 15% on permanent right ventricular paging and mean LVEF was 27.3 ± 7.7% [57].

In another study 30 chagasic patients in NYHA class III or IV undergone to right ventricular bifocal pacing. No change in the LVEF after 6 months of follow-up. However, a marked increase in arrhythmic episodes was observed.. The mortality rate was 43% in the first year of follow-up.

In a small study, 29 patients (52% chagasic) with conventional pacemakers implanted in the right ventricular apical area, in NYHA class III/IV refractory to drug therapy and LVEF < 35%, underwent CRT. During the follow-up of 22.7 ± 13 months, 86.2% of the patients benefited from CRT. However, there was no differential analysis of the chagasic patients [58].

There is low frequency of complete LBBB (16%) in chagasic patients with CHF. The most frequently found intraventricular conduction dysfunction is RBBB alone or with LAFB [51]. Nowadays, there is controversial evidence to support the use of CRT in patients without LBBB. Recently, a small study with 78 patients with RBBB showed that single-site pacing of the right ventricular septum near the proximal right bundle resulted in a marked decrease in QRS duration and often normalized the ECG [59], so that there may be new alternatives of stimulation in this patient group.

In conclusion the evidence of CRT in CHF of chagasic etiology is poor; however, in patients with LBBB, LVEF < 35% with adequate medical therapy CRT is indicated. In patients with RBBB, CRT is probably unhelpful. If RBBB is attached to LAFB, some authors consider useful biventricular pacing, but studies proving this theory are necessary.

6. Other treatments

Heart transplantation is an option in patients with refractory CHF. There are many concerns with regard to the usefulness in chagasic patients because of *T. cruzi* infection reactivation, the adequate immunosuppresive protocol and long-term results. A systematic review found that survival probability at 1 month, 1 year, 4 years and 10 years of follow-up was 83, 71, 57 and 46%, respectively. Such an outcome was better than that seen in non-Chagas patients [60]. Later, a study with 107 chagasic patients with follow-up between 30 and 168 months found the highest mortality (42.9%) [61]. Transplantation in Chagas' disease has several problems that differ from other etiologies due to the possibility of disease reactivation and the increased possibility of emergence of cancers. However, transplantation is the only treatment able to modify the natural progression of the disease in its terminal phase.

The utility of the left ventricular circulatory support as bridge to heart transplantation has been little studied. A study with 6 patients with chagasic cardiomyopathy, the mean time of circulatory support was 27 days, authors found that 2 patients were bridged to heart transplantation successfully and other four patients died.

Sustained VT is common in chagasic patients. The most frequent mechanism is scar-related reentry; the circuit may be subepicardial, intramyocardial, or subendocardial (**Figure 3**). Reports have described a higher prevalence of epicardial VT (37%). The electrophysiological signs show delayed potentials predominantly in the target area during mapping in sinus

rhythm and presystolic activity. Meso-diastolic and continuous activities are also frequent in the original place of VT. The critical isthmus of the reentrant circuit may be confirmed by entrainment maneuvers or interruption of VT during the application of RF in these places. In general, the site of origin of well-tolerated recurring VT can be identified and the VT inter-rupted in 60–80% of the patients, but rapid and poorly tolerated SVT is frequently induced in the final assessment of the procedure. During long-term follow-up, at least 50% of the patients have clinical relapse [41]. The main indication for catheter ablation is ICD shocks despite antiarrhythmic therapy.
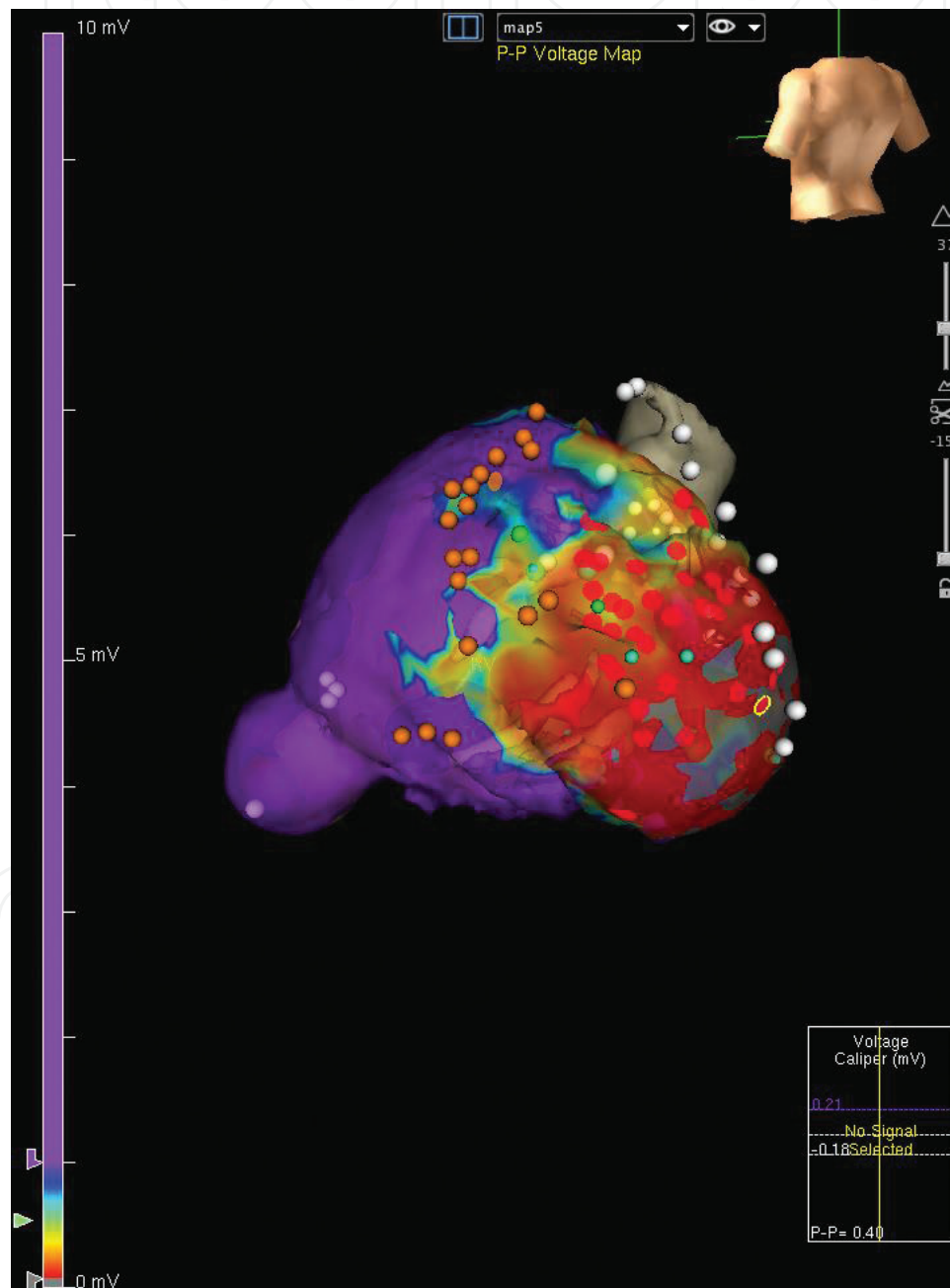

Figure 3. Image of **Figure 2** with voltage sinus rhythm map showing extensive scar inferobasal.

7. Conclusions

CD is an important cause of CHF in Latin America; migration has become this disease in a probable cause of CHF in other countries of the world. CD is associated with high prevalence of SCD; ICD is indicated as therapy class I in secondary prevention of SCD. ICD is also indicated as therapy class I in primary prevention of SCD in chagasic patients with LVEF < 40% with optimal medical treatment (OMT). The utility of PVS should be investigated in patients with non-sustained VT and LVEF > 40%.

CHF is very common in patients with CD. CRT is indicated as therapy class I in patients with LBBB + LVEF < 35% + OMT. Indication in patients with non-LBBB is controversial and new and large studies are necessary.

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