Published in final edited form as:

Glob Heart. 2015 September ; 10(3): 145–150. doi:10.1016/j.ghheart.2015.07.002.

The Prevalence of Chagas Heart Disease in a Central Bolivian Community Endemic for *Trypanosoma Cruzi*

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Abstract

Background—Though the incidence of new *Trypanosoma cruzi* infections has decreased significantly in endemic regions in the Americas, medical professionals continue to encounter a high burden of resulting Chagas disease among infected adults. The current prevalence of Chagas heart disease in a community setting is not known; nor is it known how recent insecticide vector control measures may have impacted the progression of cardiac disease in an infected population.

Objectives and Methods—Nested within a community serosurvey in rural and periurban communities in central Bolivia, we performed a cross-sectional cardiac substudy to evaluate adults for historical, clinical, and electrocardiographic evidence of cardiac disease. All adults between the ages of 20 and 60 years old with *T. cruzi* infection and those with a clinical history, physical exam, or ECG consistent with cardiac abnormalities were also scheduled for echocardiography.

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Results and conclusions—Of the 604 cardiac substudy participants with definitive serology results, 183 were seropositive for infection with *T. cruzi* (30.3%). Participants who were seropositive for *T. cruzi* infection were more likely to have conduction system defects (1.6% versus 0 for complete right bundle branch block and 10.4% versus 1.9% for any bundle branch block; $p=0.008$ and $p<0.001$, respectively). However, there was no statistically significant difference in the prevalence of bradycardia among seropositive versus seronegative participants. Echocardiogram findings were not consistent with a high burden of Chagas cardiomyopathy: valvulopathies were the most common abnormality, and few participants were found to have low ejection fraction or left ventricular dilatation. No participants had significant heart failure. Though almost one third of adults in the community were seropositive for *T. cruzi* infection, few had evidence of Chagas heart disease.

Keywords

Chagas disease; Chagas heart disease; *Trypanosoma cruzi*; Community prevalence; Bolivia

Introduction

Chagas disease, caused by the parasite *Trypanosoma cruzi*, remains an important cause of morbidity and mortality in Central and South America [1-3]. Vector-borne transmission occurs when the feces from the infected triatomine insect is inoculated through the bite wound or intact mucosa of the mammalian host. Since 1991, affected countries in the Southern Cone have implemented insecticide campaigns with the goal of reducing the vector burden and interrupting transmission [4]. Nevertheless, an estimated 8-10 million people are currently living with Chagas disease [2, 5, 6].

Infection with *T. cruzi* often goes undetected. The acute phase lasts four to eight weeks and generally results in only non-specific signs and symptoms. Infection persists in the absence of effective treatment, and untreated patients enter the chronic phase of infection. Approximately 20-30% of those infected subsequently develop cardiac or, less commonly, digestive disease years or decades after infection. Common manifestations of Chagas cardiomyopathy include atrioventricular and bundle branch blocks, arrhythmias, and dilated cardiomyopathy eventually leading to congestive heart failure [1, 7, 8].

In central Bolivia, an area that remains endemic for *T. cruzi*, the primary livelihood is subsistence farming. Most farmers continue to live in adobe houses with mud floors, structures that easily accommodate triatomine bugs. In 2003, the Bolivian national control program instituted large-scale insecticide campaigns to reduce triatomine infestation. We conducted a serosurvey of all community residents older than two years to determine the community-wide prevalence of *T. cruzi* infection, and a nested study of cardiac disease in adults 20-60 years old.

Materials and Methods

Ethics statement

The protocol was approved by the institutional review boards of CEADES, Asociación Benéfica PRISMA, and Johns Hopkins Bloomberg School of Public Health. All adult participants provided written informed consent, with separate forms for the serosurvey and the cardiac substudy. Written informed consent was obtained from the parent or guardian on behalf of children <18 years old, and children aged 7 to 17 years provided written assent.

Study setting and serosurvey participants

The study was conducted in Punata, a province of Cochabamba Department with a population of 47,735 inhabitants at the time of the 2001 census. Participants were recruited between January 2009 and May 2010. Communities were selected to include rural and peri-urban populations living in the catchment area of Punata Hospital, located in the central city (also called Punata). Field workers invited all residents 2 years or older to participate. All participating individuals provided a blood sample for serologic testing. Testing was also offered for children younger than 2 years whose mothers were found to have *T. cruzi* infection, based on the risk of congenital transmission.

Adult cardiac substudy

All permanent community residents in the age range 20-60 years who participated in the serosurvey were asked to participate in the nested cardiac substudy. Adults older than 60 were excluded based on prior evidence that rates of Chagas heart disease peaked in adults ages 30 to 50 years old [9-12]. They were also excluded to reduce the number of age-related non-specific cardiac findings on electrocardiogram and echocardiogram.

Laboratory Methods

Serum samples were evaluated for antibodies to *T. cruzi* using at least two of three commercially available assays: a rapid immunochromatographic (IC) screening test based on recombinant *T. cruzi* antigens (STAT-PAK, Chembio Diagnostic Systems, Inc., Medford, New York), an epimastigote lysate enzyme-linked immunosorbent assay (ELISA) (Chagatek, bioMérieux, Buenos Aires, Argentina), and an indirect hemoagglutination assay (HAI) (IHA - Chagatest, Wiener Laboratorios, Rosario, Argentina). All tests were performed and interpreted following the manufacturers guidelines. Individuals were considered to have *T. cruzi* infection if they had two positive serologic tests. Twelve individuals participating in the nested cardiac substudy had inconclusive serologic results based on conflicting or indeterminate assay results; these individuals were excluded from analysis.

Cardiac study procedures

All substudy participants underwent a focused history and physical exam and an electrocardiogram (ECG), and gave one blood sample. All individuals who were positive for infection with *T. cruzi* and all those with any evidence of cardiac abnormalities by history, physical exam, or ECG (including bradycardia with a heart rate less than 60 beats per

minute) were scheduled to undergo echocardiogram. Echocardiograms were performed using a portable Esaote Caris machine. A cardiologist read all electrocardiograms and performed and interpreted all echocardiograms. Electrocardiograms were coded and interpreted according to established criteria [13]. The cardiologist was blinded for all ECG interpretation; study participants occasionally revealed their *T. cruzi* infection status to the cardiologist during their echocardiogram rendering echocardiogram blinding incomplete.

Statistical Analysis

We evaluated associations between infection with *T. cruzi* and cardiac lesions using odds ratios with 95% confidence intervals (CI) and two-tailed Fisher's exact test with an alpha of 0.05, as well as multivariate logistic regression models. Logistic regression models included age and serostatus as predictor covariates. Because incomplete right bundle branch block has been shown to be a highly non-specific finding and can be a normal variant in healthy adults [14], all analyses evaluating combined conduction system defects excluded incomplete right bundle branch blocks. Data were collected and managed using Microsoft Access; statistical analysis was performed using Stata 11.0.

Results

Study Population

A total of 1,380 individuals participated in the *T. cruzi* serosurvey; 36 (2.6%) individuals were excluded due to inconclusive serology results, yielding a survey population of 1344 (Table 1). The mean age of all participants in the community serosurvey was 31.29 years (standard deviation 21.83). The mean age of the participants in the cardiac substudy was 39.19 years (standard deviation 12.16), and among those who received an echocardiogram was 39.89 years (standard deviation 12.26). Seroprevalence increased with increasing age in all decades until the seventh decade, with a maximum seroprevalence of 56.88% among study participants between the ages of 60 and 69 years. Rates were lower among study participants in their 70s and 80s but with confidence intervals that overlapped with those in younger age groups. Twenty-one of 52 participants between the ages of 70 and 79 years were positive for *T. cruzi* infection, yielding a seroprevalence of 40.38% (95% confidence interval 27.31-54.87). Of those participants between the ages of 80 and 89 years, seven of 18 were positive for *T. cruzi* infection, yielding a seroprevalence of 38.89% (95% confidence interval 18.26-63.86) (Figure 1).

Cardiac Disease According to *T. cruzi* Infection

Of 632 eligible adults, 604 (95.6%) participated in the cardiac substudy; 183 (30.3%) had confirmed *T. cruzi* infection. Three (1.6%) seropositive participants had complete right bundle branch block (RBBB) on ECG (1.6%) compared to none of the seronegative participants ($p=0.008$) (Table 2). Nineteen *T. cruzi*-infected participants had any conduction system defect, compared with eight seronegative participants (10.4% vs. 1.9%, $p<0.001$). The percentages with bradycardia were similar among those with and without *T. cruzi* infection (3.8 versus 4.7% respectively for heart rate less than 50 beats per minute).

The prevalence of right bundle branch block was highest in seropositive individuals older than 40 years, whereas bifasicular blocks were read more commonly in younger seropositive participants (Figure 2). The prevalence of any conduction system defect among both seropositive and seronegative individuals was highest among those between the ages of 20 and 29 years: 23.1% and 2.6% among seropositive and seronegative individuals, respectively. In multivariable logistic regression models adjusted for sex and age (by decade), bifasicular block and any conduction system defect were significantly associated with *T. cruzi* infection (odds ratio for bifasicular block 8.19 (95% CI 1.55-43.17), $p=0.02$; any conduction system defect 7.36 (2.97-18.6), $p<0.001$).

Per study protocol, 434 participants were scheduled for echocardiograms, but only 201 (46%) participants (78 seropositive, 123 seronegative) underwent the examination (Table 4). Participants who missed an echocardiogram were contacted by the study team by phone or by home visit to reschedule the appointment. Those who missed repeat appointments often cited the need to work and the lack of any symptomatic illness as the reasons for skipping the echocardiogram. One seronegative participant had an uninterpretable study due to body habitus; the echocardiogram was excluded from analysis. There was no sex difference between seropositive participants who did and did not present for echocardiogram; seronegative women were more likely to miss their appointment than seronegative men. Left ventricular dilatation (defined as LV end diastolic diameter $> 57\text{mm}$) was found in four seropositive and three seronegative participants (Table 3). Seven seropositive participants had left atrial dilatation (defined as LA $>40\text{mm}$) compared with only two uninfected participants (9.0% vs. 1.6%, $p=0.014$). One seronegative participant had an ejection fraction of 42%; two seropositive and two seronegative participants had slightly depressed ejection fractions between 51 and 54%. There was no difference in the distribution of ejection fractions by infection status (mean 68% [standard deviation 0.16] for seropositives vs 68% [0.16] for seronegatives; $p=0.959$ by t-test). Sixteen participants had valvulopathies not related to Chagas disease: 12 aortic sclerosis, two mitral prolapse, one mitral stenosis, and one mitral insufficiency. There was no significant difference in rates of valvular disease among participants with and without *T. cruzi* infection. None of the participants had an apical aneurysm or an intracavitary thrombus.

Discussion

Our study was one of the first in decades to evaluate the community burden of *T. cruzi* infection and Chagas heart disease in the inter-Andean valleys of Bolivia. Housing improvement programs began in Cochabamba in the 1990s and household insecticide application was instituted on a large scale by the Bolivian National Chagas Disease Control Program between 2000 and 2004 [15; personal communication, F. Torrico, 2009]. Our data support the success of these efforts. We found a much lower prevalence of *T. cruzi* infection than a study conducted in Cochabamba Department in 1988 (74% in 1988 vs 23% in our data) and a strikingly lower prevalence in children (38% in 1988 vs 5.6% in our data) [12].

Although our age range and case definitions varied from those used in the 1988 study, it is interesting to note that we also found a lower rate of bundle branch blocks among infected participants than in the 1988 study (20% in 1988 vs 10.4% in our data), and found no

participants with clinically evident congestive heart failure (compared to 9% in the 1988 data) [12]. A similar study restricted to Bolivian women of childbearing age in the 1980s also showed higher rates of conduction system abnormalities than we found [11]. Nevertheless, *T. cruzi* infection is still a significant risk factor for conduction system defects in our data. These findings provide an optimistic picture for the future, since bundle branch blocks [10], decreased ejection fraction [16, 17], left and right ventricular dysfunction [18, 19], and abnormal diastolic function [20, 21] have all been associated with increased mortality in individuals with Chagas disease. Evidence from mouse models [22, 23] and communities [24] suggest that continued vector-borne exposure to *T. cruzi* may increase the risk of Chagas cardiomyopathy. Failure to down-regulate the anti-*T. cruzi* inflammatory immune response is widely accepted as an important driver of Chagas cardiomyopathy development and progression [25, 26]. Vector-borne parasite exposure is hypothesized to maintain sustained antigen exposure and consequent higher chronic inflammatory immune responses, contributing to Chagas cardiomyopathy. The lower rate of clinical disease in our study may reflect the better vector control over the previous decades and decreased exposure to the parasite. However, further data are needed to better understand this possible association.

Our observation of an association between *T. cruzi* infection and left atrial dilatation is intriguing but its clinical significance is unclear. Previous studies have found left atrial dilatation associated with right bundle branch block in infected individuals, perhaps indicating early manifestations of cardiac involvement [27-29]. Furthermore, left atrial dilatation has been linked to increased risk of sudden cardiac death in those with known Chagas cardiomyopathy [20, 21]. The most common abnormalities found on echocardiogram were valvulopathies, most commonly isolated aortic sclerosis in participants between 50 and 60 years old. This finding was consistent with prior studies, and most consistent with age-related changes [30]. We anticipated seeing a greater burden of valvular disease associated with known or suspected rheumatic heart disease, which most commonly causes mitral stenosis; it is not common to have aortic disease resulting from rheumatic heart disease without concomitant mitral involvement [31, 32]. We found no association between valvulopathies and *T. cruzi* infection.

Our study had several limitations. First, it is possible that those individuals who were in poor health did not present to our mobile clinic to participate in the study and we therefore underestimated the cardiac disease burden. Second, we excluded adults over age 60 to minimize non-specific cardiac disease such as paroxysmal atrial fibrillation. While the work of Lima-Costa and colleagues indicates that Chagas may be a greater problem in elderly patients than was previously recognized [33, 34], prior studies have found the majority of cardiac disease developing in adults between 30 and 50 years old [9-12]. It is possible, however, that our exclusion of adults over age 60 reduced the amount of Chagas heart disease detected. Finally, our echocardiogram findings may have been affected by the low rates of echocardiogram completion. However, consensus among Chagas experts indicates that the rate of clinically-significant Chagas heart disease is rare in the absence of ECG abnormalities [35].

Conclusion

Our study found low rates of Chagas cardiomyopathy in a community endemic for *T. cruzi*, possibly related to the effects of widespread vector control efforts over the previous 20 years. More research is needed to better understand who, among those infected with *T. cruzi*, is at increased risk of progression to associated disease.

Acknowledgments

We would like to thank Drs. René Ugarte, Marco Solano Mercado, and Gimena Rojas Delgadillo for their assistance in the laboratory and in the field. We would additionally like to thank all the members of the field team who collected data and specimens for the community study, and the members of CEADES Salud y Medio Ambiente who assisted with data collection and management. In addition, the authors would like to thank the Fogarty International Clinical Research Fellows Program for funding support for Drs. Yager and Lozano Beltran.

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Highlights

- We conducted a community survey of *Trypanosoma cruzi* infection in central Bolivia.
- We evaluated adults aged 20-60 years old for evidence of Chagas heart disease.
- 30.3% of adults were seropositive for *T. cruzi* infection.
- Bundle branch blocks were more common among participants with *T. cruzi* infection.
- Overall rates of Chagas cardiomyopathy were lower than in prior community studies.

Figure 1. Prevalence of *T. cruzi* infection in Punata, Bolivia community serosurvey, by age
N represents total number of individuals in each age group.

Figure 2. ECG Findings by Age and *T. cruzi* serostatus

Table 1
Baseline characteristics of participants in community serosurvey with conclusive serologic data

Characteristics	Community (N=1344) ^a		Cardiology substudy (N=604)		Completed Echocardiogram (N= 200)	
	N	Seropositive n (%)	N	Seropositive n (%)	N	Seropositive n (%)
Age (years)						
<20	540	30 (5.6)	--	--	--	--
20-29	189	28 (14.8)	179	26 (14.5)	53	10 (18.9)
30-39	126	39 (31.0)	118	39 (33.1)	43	15 (34.9)
40-49	159	52 (32.7)	152	47 (30.9)	47	22 (46.8)
50-60	158	73 (46.2)	155	71 (45.8)	57	31 (54.4)
>60	172	87 (50.6)	--	--	--	--
Female	818	187 (22.9)	387	107 (27.6)	112	46 (41.1)
Male	526	122 (23.2)	217	76 (35)	88	32 (36.4)
Rural	966	251 (25.1)	456	146 (32.0)	137	58 (42.3)
Periurban	378	58 (15.3)	148	37 (25.0)	63	20 (31.8)

^aThirty-six individuals were excluded due to inconclusive serology results.

Table 2

Electrocardiography findings in individuals with and without *T. cruzi* infection

ECG Findings ^a	Seropositive (N=183)	Seronegative (N=421)	Odds Ratio	95% Confidence Interval	P value
Heart rate and rhythm					
Heart Rate <60	63 (34.4)	128 (30.4)	1.202	0.815-1.764	0.329
Heart Rate <50	7 (3.8)	20 (4.8)	0.797	0.280-2.009	0.613
Mean Heart Rate (SD)	62.65 (9.4)	62.66 (10.6)			
Atrial Fibrillation/Flutter	0	1 (0.2)	0.0		1.000 ^b
Ventricular Arrhythmia (PVC)	1 (0.5)	0	0.0		0.303 ^b
Bundle branch blocks (BBB)					
RBBB (complete)	3 (1.6)	0	0.0		0.027 ^b
LAFB	7 (3.8)	5 (1.2)	3.309	0.888-13.378	0.052 ^b
LPFB	3 (1.6)	1 (0.2)	7.000	0.556-368.280	0.085 ^b
Bifascicular block	6 (3.3)	2 (0.5)	7.102	1.250-72.337	0.006
Any BBB ^c	19 (10.4)	8 (1.9)	5.981	2.432-16.061	<0.001
Incomplete RBBB	13 (7.1)	29 (6.9)	1.034	0.481-2.112	0.924
Other abnormalities					
1° AV block	4 (2.2)	4 (1.0)	2.330	0.428-12.633	0.253 ^b
ST-T changes	13 (7.1)	13 (3.09)	2.400	1.001-5.739	0.025
Low Voltage QRS	0	1 (0.2)	0.0		1.000 ^b
Normal ECG	88 (48.1)	228 (54.2)			0.171

Values are n (%). AV: atrioventricular; BBB: bundle branch block; LAFB: left anterior fascicular block; LPFB: left posterior hemiblock; PVC: premature ventricular contraction; RBBB: right bundle branch block.

^aNo participant was found to have LVH or an abnormal initial portion of their QRS complex on ECG.

^bBy 2-tailed Fisher exact test

^cAny complete bundle branch block (RBBB, LAFB, LPFB, bifascicular block)

Table 3Echocardiography findings in individuals with and without *T. cruzi* infection

Echo Findings ^a	Seropositive N=78	Seronegative N=122	Odds Ratio	95% Confidence Interval	P value
	Mean (SD)	Mean (SD)			
LV End Diastolic Diameter	4.58 (0.94)	4.42 (0.79)			0.207
LV Ejection Fraction	0.68 (0.16)	0.68 (0.12)			0.959
LA diameter	3.00 (0.84)	2.84 (0.68)			0.149
	n (%)	n (%)			
LV dilatation	4 (5.1)	3 (2.4)	2.144	0.351-14.989	0.435 ^b
LAD	7 (9.0)	2 (1.6)	5.915	1.077-59.355	0.030 ^b
EF<55%	2 (2.6)	3 (2.5)	1.044	0.853-9.329	1.000 ^b
Any Valvulopathy	8 (10.3)	8 (6.5)	1.629	0.506-5.215	
Early systolic dysfunction	2 (2.6)	1 (0.8)	3.184	0.162-189.36	0.562 ^b
L VH	62 (79.5)	96 (78.7)	1.049	0.495-2.274	0.892

Values are n (%). EF: ejection fraction; LA: left atrium; LAD: left atrial dilatation; LV: left ventricle; LVH: left ventricular hypertrophy.

^aNo participant was found to have an apical aneurysm, an intracavitary thrombus, or diffuse wall motion abnormalities on echocardiogram.^bBy 2-tailed Fisher exact test

Table 4

Comparing those who did and did not complete echocardiograms, by serostatus, in those who were scheduled for study per protocol

	Chagas seropositive		Chagas seronegative	
	Received echo (N=78)	No echo (N=105)	Received echo (N=123)	No echo (N=128)
Mean age, years	44.0 ± 11.2	43.4 ± 11.6	37.3 ± 12.2	36.6 ± 11.9
Female	46 (59.0)	61 (58.1)	67 (54.5)	85 (66.4)
Heart rate <60	28 (35.9)	35 (33.3)	67 (54.5)	60 (46.9)
Heart rate <50	4 (5.1)	3 (2.9)	7 (5.7)	13 (10.2)
Bifascicular block	3 (3.9)	3 (2.9)	2 (1.6)	--
Any conduction system defect	6 (7.7)	13 (12.4)	6 (4.9)	2 (1.6)

Values are mean ± SD or n (%).