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Original Article

Cardiovascular outcomes of chagas-induced non-ischemic cardiomyopathy versus other nonischemic cardiomyopathies: a regression matched national cohort analysis

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Abstract: Objectives: Chagas disease, caused by Trypanosoma cruzi, is a parasitic infection endemic to Latin America and is increasingly prevalent in the United States. This study examines mortality, heart failure, arrhythmias, cardiogenic shock, and the need for heart transplantation in Chagas patients over five years in the United States. Methods: We selected all non-ischemic cardiomyopathy (NICM) patients from the National Inpatient Sample Database from 2016 to 2020 and compared them to Chagas-induced NICM. Results: A total of 783,535 patients had non-ischemic cardiomyopathy (NICM), with 250 cases being secondary to Chagas disease. Chagas NICM was predominantly seen in the Hispanic population. Patients with Chagas NICM have significantly higher odds of receiving a heart transplant (OR 15.48; P<0.05), particularly in the context of a high incidence of cardiogenic shock due to end-stage heart failure or severe myocarditis (OR 2.7; P<0.05). Furthermore, these patients demonstrate a higher incidence of ventricular fibrillation (OR 4.87; P<0.05) and pericardial effusion (OR 3.75; P<0.05) compared to other forms of NICM. They are frequently associated with the need for pacemaker placement (OR 2.80; P<0.05), likely due to ventricular fibrillation and conduction blocks. The odds of in-hospital mortality were similar between patients with Chagas NICM and those with other NICM patients. Conclusion: Patients with Chagas cardiomyopathy are more likely to experience cardiogenic shock, ventricular fibrillation, and pericardial effusion. They also face an increased risk of needing an ICD and heart transplant. Further research is necessary on this subject.

Keywords: Chagas myocarditis, chagas cardiomyopathy, non-ischemic cardiomyopathy, national inpatient sample analysis

Introduction

The protozoan Trypanosoma cruzi is responsible for causing Chagas disease and American trypanosomiasis [1]. This parasitic infection can be life-threatening. According to the World Health Organization, Chagas disease negatively impacts healthcare due to migration, affecting approximately 6 to 7 million people globally [2]. In non-endemic countries like the United States, it poses a significant threat to immigrant populations, with an estimated 300,000 individuals affected [3]. The infection can be acquired through contaminated food and beverages, blood transfusions, or organ transplants [4]. During the acute phase, patients

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may experience mild or nonspecific symptoms. About half of the patients remain complication-free during the intermediate phase, while 20% to 30% eventually progress to the chronic stage, where they face severe cardiovascular complications such as Chagas cardiomyopathy. This condition can present as conduction abnormalities, myocarditis, cardioembolic events, heart failure, and sudden death [5-9]. The final stages of the disease are marked by left ventricular enlargement and a decline in systolic function [9]. Several mechanisms contribute to the manifestations and severity of this polymorphic disease in individuals, most of which relate to immune-mediated responses. These responses are linked to the release of inflammatory cytokines that harm myocardial fibers, resulting in fibrosis and cellular death [10, 11]. Predicting disease progression in individuals is a challenging and pressing area of focus in clinical practice. According to the American Heart Association, the rate of progression from the intermediate chronic form to Chagas cardiomyopathy ranges from 1.85% to 7% [5].

The diagnosis of Chagas cardiomyopathy is advancing. Patients typically exhibit electrocardiographic changes, including bundle branch blocks and arrhythmias [12, 13]. Measurements of plasma BNP (B-type natriuretic peptide) levels can benefit patients, though they lack clinical relevance in diagnosis [14]. Additional tests such as echocardiography, Holter monitoring, and stress tests can help identify changes in wall motion and conduction abnormalities linked to ventricular arrhythmias [15, 16]. Cardiac magnetic resonance (CMR) provides advantages over echocardiograms in assessing the extent of cardiac fibrosis and left ventricular aneurysms [17, 18]. Currently, there are no studies on the prevalence and mortality rates of Chagas cardiomyopathy in the United States. This retrospective study examined cardiovascular outcomes and mortality in patients diagnosed with Chagas disease. It assessed the prevalence of mortality, heart failure, arrhythmias, cardiogenic shock, and the necessity for heart transplants related to Chagas over five years in the United States.

Methods

Study design

An analysis utilized data from the National Inpatient Sample (NIS) database from 2016 to 2020. The NIS is part of the Healthcare Cost and Utilization Project (HCUP) databases and is funded by the Agency for Healthcare Research and Quality (AHRQ). It includes information on in-hospital outcomes, various procedures, and discharge-related details. This publicly available resource features de-identified data, eliminating the need for Institutional Review Board (IRB) approval. As the largest all-payer inpatient healthcare database in the U.S. with de-identified information, the NIS compiles billing data from approximately 1,000 hospitals, accounting for about 20% of all hospitalizations in the country. Over 7 million unweighted hospitalizations are recorded annually, translating to a million when weighted. The database includes patient and hospital-level data, encompassing as many as 40 discharge diagnoses and 25 procedures per patient, categorized using the International Classification of Diseases, Tenth Revision (ICD-10) codes [19]. **Table 1** displays the ICD-10 codes for cohort identification and outcomes.

Inclusion and exclusion criteria

We included patients from all age groups aged 18 years and older diagnosed with nonischemic cardiomyopathy (NICM), identified using ICD-10-CM diagnosis codes for index admission as provided by HCUP and the ICD-10-CM codes [19].

Inclusion criteria: Our primary inclusion criteria are adult patients aged 18 years or older hospitalized with a primary or secondary diagnosis of non-ischemic cardiomyopathy (NICM), as identified by ICD-10 codes. Patients with a confirmed diagnosis of Chagas disease (ICD-10 code B57.2) for the subgroup designated with Chagas-induced NICM.

Hospitalization records must be from January 1, 2016, to December 31, 2020.

Exclusion criteria: Patients diagnosed with ischemic cardiomyopathy (ICM) or those with a documented history of coronary artery disease, those who had prior percutaneous interventions or acute coronary syndromes, or those who underwent bypass surgeries. Individuals with incomplete or missing data regarding critical clinical outcomes or demographic information. Patients with co-existing diagnoses of other forms of cardiomyopathy (e.g., hypertro-

Table 1. ICD-10 codes for cohort identification and the outcomes

Condition	ICD-10 code
Acute coronary syndrome	I21.4, I22.2, I20.0, I21.01, I21.02, I21.09, I21.11, I21.19, I21.21, I21.29, I21.29, I22.0, I22.1, I22.8, I22.9
Coronary artery disease	I25.10
Percutaneous intervention	027.23ZZ, 027.03D6, 027.03DZ, 027.03EZ, 027.03FZ, 027.03TZ, 027.03Z6, 02H03DZ, 027.13D6, 027.13DZ, 027.13EZ, 027.13FZ, 027.13TZ, 027.13Z6, 027.23D6, 027.23DZ, 027.23EZ, 027.23FZ, 027.23TZ, 027.23Z6, 027.33DZ, 027.33Z6, 02H13DZ, 02H13YZ, 027.33ZZ, 02H23DZ, 02H33DZ, 027.0346, 027.034Z, 027.0356, 027.035Z, 027.0366, 027.036Z, 027.0376, 027.037Z, 027.03E6, 027.03F6, 027.03G6, 027.03GZ, 027.03T6, 027.04D6, 027.1346, 027.134Z, 027.1356, 027.135Z, 027.1366, 027.136Z, 027.1376, 027.137Z, 027.13E6, 027.13F6, 027.13G6, 027.13GZ, 027.13T6, 027.2346, 027.234Z, 027.2356, 027.235Z, 027.2366, 027.236Z, 027.2376, 027.237Z, 027.23E6, 027.23F6, 027.23G6, 027.23GZ, 027.23T6, 027.334Z, 027.3356, 027.335Z, 027.3366, 027.336Z, 027.3376, 027.337Z, 027.33D6, 027.33E6, 027.33EZ, 027.33F6, 027.33FZ, 027.33G6, 027.33GZ, 027.33T6, 027.33TZ
Cardiomyopathies	I42.0, I42.4, I42.5, I42.1, I42.2
Chagas disease	B57.2
Atrial fibrillation	I48.0, I48.1, I48.2, I48.9
Atrial flutter	I48.3, I48.4, I48.92
Ventricular tachycardia	I47.2, I47.20, I47.21, I47.29
Valvular disease	I26.x, I27.x, I28.0, I28.8, I28.9
Peripheral vascular disease	I70.x, I71.x, I73.1, I73.8, I73.9, I77.1, I79.0, I79.2, K55.1, K55.8, K55.9, Z95.8, Z95.9
Previous PCI	Z95.5
Previous stroke	Z86.73
Hypertension, uncomplicated	I10.x
Chronic pulmonary disease	I27.8, I27.9, J40.x - J47.x, J60.x - J67.x, J68.4, J70.1, J70.3
Diabetes	E10.0, E10.1, E10.9, E11.0, E11.1, E11.9, E12.0, E12.1, E12.9, E13.0, E13.1, E13.9, E14.0, E14.1, E14.9
Liver disease	B18.x, I85.x, I86.4, I98.2, K70.x, K71.1, K71.3 - K71.5, K71.7, K72.x - K74.x, K76.0, K76.2 - K76.9, Z94.4
Heart failure with reduced ejection fraction	I50.2, I50.20, I50.21, I50.22, I50.23
Heart failure with preserved ejection fraction	I50.30, I50.31, I50.32, I50.33
AV block 1st degree	I44.0
AV block 2nd degree	I44.1
AV block 3rd degree	I.442
Left bundle branch block	I44.7
Right bundle branch block	I45/1, I45.10, I45.19
Pacemaker placement	0JH604Z, 0JH634Z, 0JH606Z, 0JH636Z, 0JH804Z, 0JH806Z, 0JH834Z, 0JH836Z, 0JH635Z, 0JH805Z, 0JH835Z, 02H40NZ, 02H43JZ, 02H60JZ, 02H60NZ, 02H63JZ, 02H63NZ, 02H70JZ, 02H73JZ, 02H73NZ, 02HL0JZ, 02HL0NZ, 02HL3JZ, 02HL3NZ, 02HN0JZ, 02HN4JZ, 0JH606Z, 0JH634Z, 0JH636Z, 0JH804Z, 0JH806Z, 02H44NZ, 02H64JZ, 02H64NZ, 02H74NZ, 02HK4JZ, 02HK4NZ, 0JH607Z, 0JH635Z, 0JH637Z, 0JH805Z, 0JH807Z, 0JH835Z, 0JH837Z, 0JH605Z
ICD placement	02HK4KZ, 02HL3KZ, 02HL4KZ, 0JH60FZ, 0JH63FZ, 02H40KZ, 02H43KZ, 02H60KZ, 02H63KZ, 02H70KZ, 02H73KZ, 02H44KZ, 02H64KZ, 02H74KZ, 0JH609Z, 0JH639Z, 0JH809Z, 02HA3QZ, 0WHC0GZ, 0WHC3GZ, 0WHC4GZ, 02HN0KZ, 02HN3KZ, 02HN4KZ, 02HK0KZ, Z95810
Cardiac transplant	02YA0Z0, 02YA0Z1, 02YA0Z2
Cardiogenic shock	R570
Stroke	I63.0, I63.1, I63.2, I63.3, I63.4, I63.5, I63.6, I63.8, I63.9
Left ventricular thrombus	I513

Table 2. Baseline characteristics and outcomes stratified according to the presence of chagas induced NICM

		Chagas NICM	Other NICM	*P* value
Age		55.0	62.1	0.000
Female sex		38.0%	41.4%	0.623
Race	White	14.3%	57.7%	0.000
	Black	6.1%	28.6%	
	Hispanic	73.5%	8.2%	
	Asian or pacific islander	2.0%	2.5%	
	Native American	0%	0.6%	
	Others	4.0%	2.2%	
Median household income	0-25th percentile	31.2%	33.3%	0.486
	26th-50th percentile (median)	18.7%	25.5%	
	51st-75th percentile	31.2%	22.7%	
	76th-100th percentile	18.7%	18.4%	
Valvular disease		28.0%	26.3%	0.790
Pulmonary circulation disorder		26.0%	18.3%	0.163
Chronic pulmonary disease		10.0%	29.7%	0.002
Peripheral vascular disease		50.0%	25.7%	0.000
Hypertension		6.0%	16.3%	0.047
Diabetes mellitus		2.0%	11.3%	0.037
Renal failure		28.0%	37.0%	0.187
Liver disease		16.0%	9.0%	0.086
Obesity		14.0%	24.0%	0.097
HIV/AIDS		0.0	0.5%	

phic or restrictive cardiomyopathy) to ensure a distinct and well-defined cohort of NICM patients.

Study outcomes

The study primarily aims to examine cardiovascular outcomes by analyzing the prevalence of various conditions, including atrial fibrillation, ventricular tachycardia, ventricular fibrillation, heart failure with reduced ejection fraction (HFrEF), right bundle branch block (RBBB), left bundle branch block (LBBB), type 1 and type 2 AV node blocks, and complete AV node block. It will also assess the need for permanent pacemakers (PPM) and implantable cardioverter-defibrillators (ICD), cardiogenic shock, and heart transplants in patients with and without Chagas disease. The secondary objective is to evaluate in-hospital mortality, length of stay, and total costs adjusted for inflation.

Statistical analysis

A descriptive analysis was conducted for baseline demographics and comorbidities. We confirmed the statistical strategy to use the median with interquartile ranges (IQR 25-75) or the mean with standard deviation based on the normality of the histogram and kurtosis. The histogram of the age variable indicated a non-Gaussian distribution, lacking the bell-shaped curve and exhibiting negative skewness. Statistical analysis involved a student's t-test for continuous variables and a chi-squared test for categorical variables. Due to the negatively skewed data from the small sample size of Chagas NICM, we reported medians with IQR for the continuous variables. To minimize confounding factors, we performed a multivariate polynomial logistic regression using the melogit module to obtain adjusted odds ratios (aOR). The confounding factors included in the regression were age, sex, median household income based on patients' ZIP codes, and relevant comorbidities (see **Table 2**). We defined statistical significance as a 95% confidence interval, not including "1" and *P* values less than 0.05. The statistical analysis used STATA version 17 (Stata Corp LLC, College Station, Texas, US).

Results

Demographics and baseline characteristics

We analyzed 783,535 hospitalizations associated with nonischemic cardiomyopathy, including 250 patients with Chagas disease. Patients with Chagas disease were more likely to be younger (55 vs. 62, p: 0.000), identified as Hispanic (73.5% vs. 8.2%), and had a higher prevalence of peripheral vascular disease (50.0% vs. 25.7%, p: 0.000). The nonischemic cardiomyopathy group without Chagas disease was more likely to be white (14.3% vs. 57.7%) and showed higher rates of chronic pulmonary disease (10.0% vs. 29.7%, p: 0.0002), hypertension (6.0% vs. 16.3%, p: 0.047), and diabetes mellitus (2.0% vs. 11.3%, p: 0.037). There were no disparities in median household income, valvular disease, pulmonary circulation disorders, liver disease, or obesity (**Table 2**). The central illustration describes the baseline characteristics and results presented in **Figure 1**.

Primary outcomes

In terms of arrhythmias, ventricular tachycardia (VT) was more prevalent in chronic Chagas disease compared to those without it (20.0% vs. 10.3%, p: 0.025), followed by ventricular fibrillation (V. Fib) (6.0% vs. 1.1%). Chagas patients exhibited higher rates of PPM and ICD placements (58.0% vs. 27.0%, p: 0.000). Hospitalizations due to chronic Chagas disease were linked to increased rates of HFrEF (52.0% vs. 37.6%, p: 0.036), cardiogenic shock (16.0% vs. 5.0%, p: 0.000), and a higher requirement for heart transplants (12.0% vs. 0.3%, p: 0.000). There were no significant differences in AV node blocks.

Adjusted outcomes showed that patients with chronic Chagas disease were more closely linked to ventricular fibrillation (aOR: 4.87, 95% CI: [1.84-12.8], P: 0.001), the need for PPM/ICD (aOR: 2.8, 95% CI: [1.65-4.74], P: 0.000), cardiogenic shock (aOR: 2.7, 95% CI: [1.39-5.28], P: 0.003), and heart transplant (aOR: 15.48, 95% CI: [5.82-41.0], P: 0.000). Additionally, this group had a stronger association with left ventricular thrombus (aOR: 3.5, 95% CI: [1.02-11.8], P: 0.04). No difference was found in LBBB; however, Chagas disease was more strongly associated with RBBB (aOR: 4.24, 95% CI: [1.51-11.63], P: 0.006), and no statistically significant difference was noted in atrial fibrillation or ventricular tachycardia (**Figures 1**, **2**).

Secondary outcomes

Adjusted outcomes indicated no statistically significant differences in mortality (adjusted odds ratio [aOR]: 1.45, 95% confidence interval [CI]: (0.47-4.24), P: 0.513), length of stay (adjusted β: 3.0 days, P: 0.189), or total costs (adjusted β: $42,874, P: 0.165).

Discussion

This study aims to evaluate the outcomes associated with Chagas cardiomyopathy over five years in the United States by analyzing a comprehensive national database. It examines the prevalence of ventricular arrhythmias, cardiogenic shock, pacemaker requirements, and heart transplants in patients with Chagas cardiomyopathy in the United States. Clinical findings from this extensive observational study indicate that patients with Chagas cardiomyopathy are more likely to be male (62% vs. 58.6%), younger (55 vs. 62 years old), and predominantly Hispanic (73.5% vs. 8.2%) compared to patients without Chagas cardiomyopathy. Ventricular arrhythmias are more prevalent in chronic Chagas disease patients, with VT followed by V. Fib, and they experience higher rates of PPM and ICD placement. Adjusted outcomes reveal that patients with chronic Chagas face increased risks of V. Fib (aOR: 4.87), cardiogenic shock (aOR: 2.7), LV thrombus (aOR: 3.5), and heart transplant (aOR: 15.48) when compared to patients without the condition.

This study shows that Chagas cardiomyopathy is a prevalent form of non-ischemic cardiomyopathy (NICM) among young Hispanic individuals. Out of 783,535 NICM cases, 250 were identified as Chagas cardiomyopathy, with an average patient age of 55 years, and 73.5% of those cases were Hispanic. The research revealed a notable difference in the prevalence of peripheral vascular disease between patients with and without Chagas cardiomyopathy, likely linked to the disease's associated vascular inflammation. In contrast, NICM cases not related to Chagas were primarily found in the white population and were significantly associated with chronic pulmonary disease

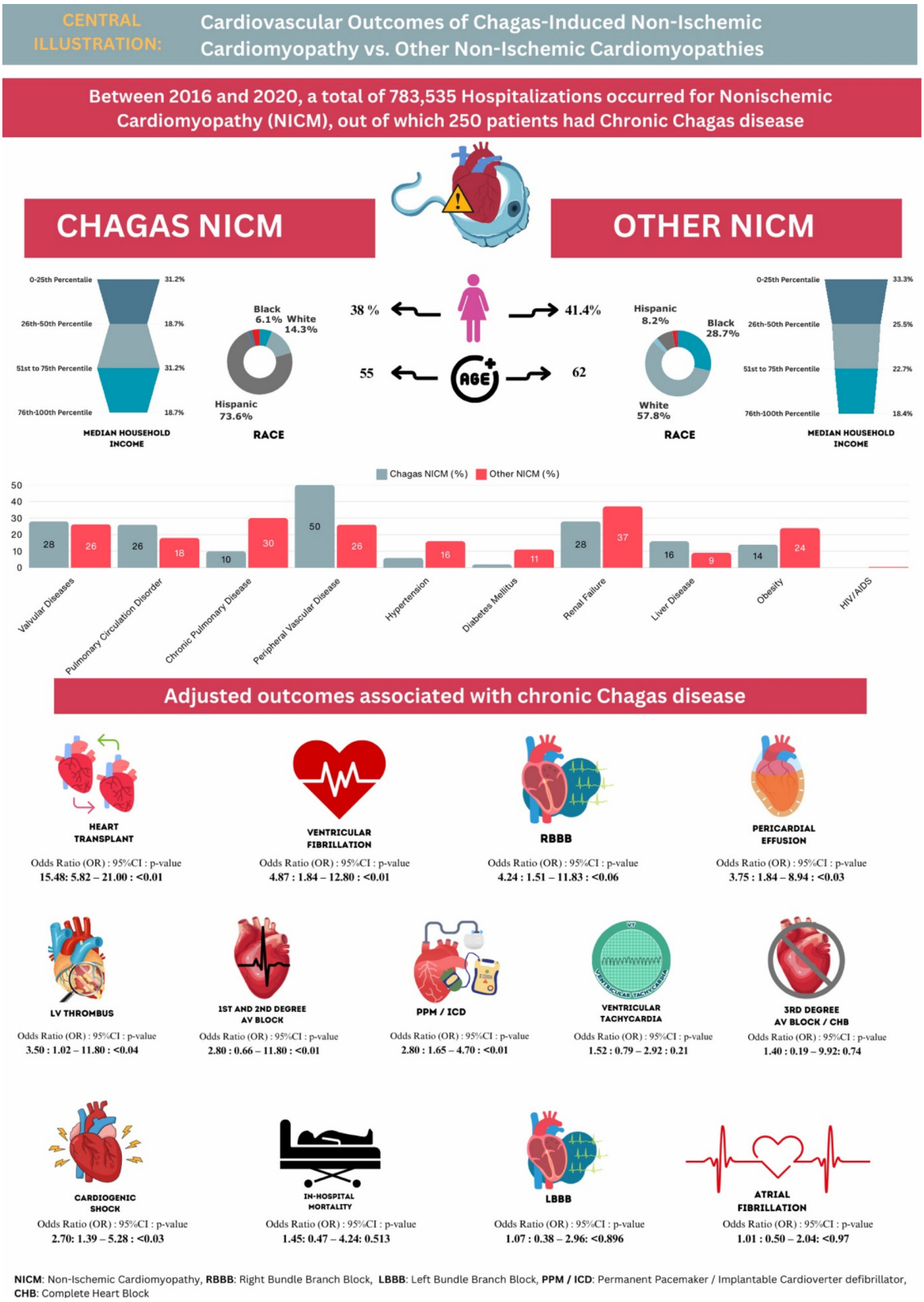

Figure 1. Central illustration showing the baseline characteristics and study outcomes.

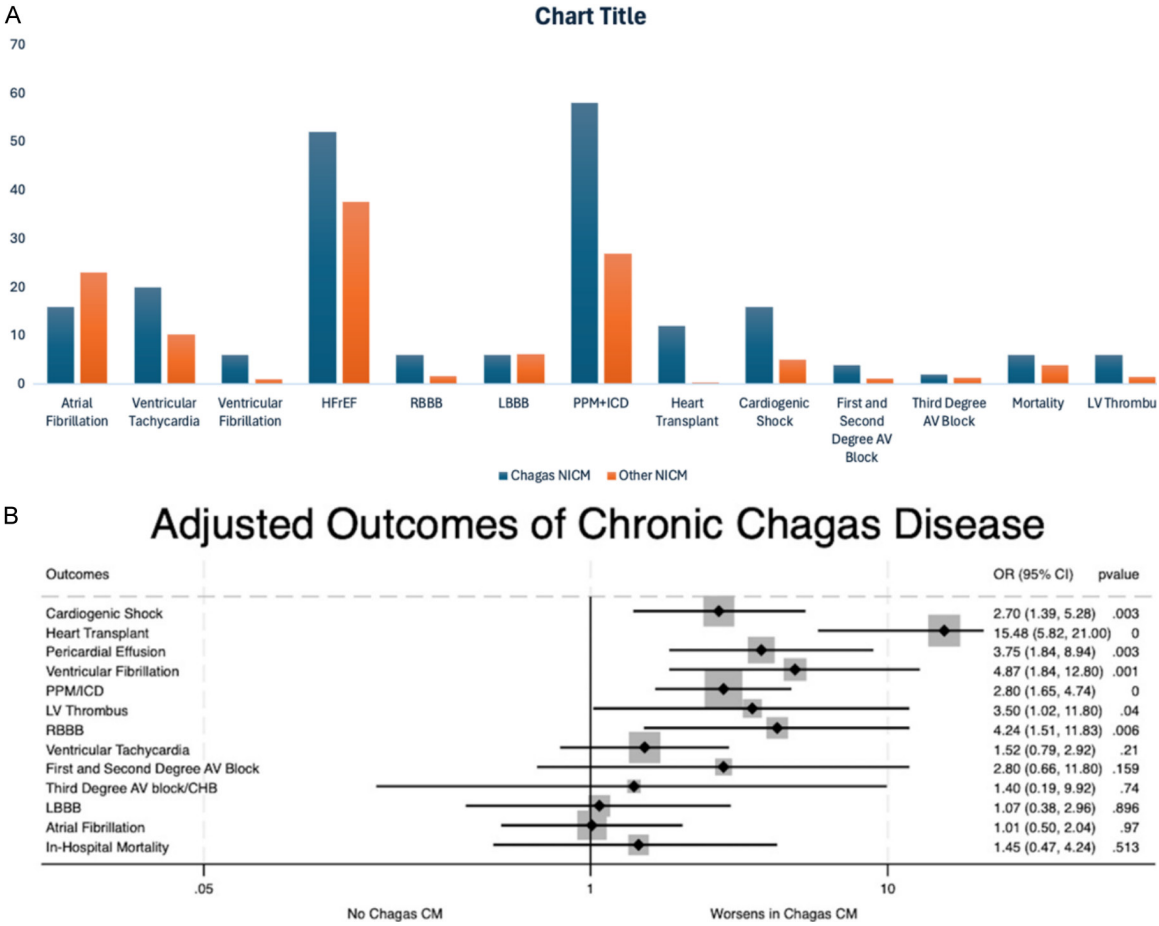

Figure 2. A: Bar chart showing Percentage of Complications/Outcomes in inpatients with and without Chagas non-ischemic cardiomyopathy. B: Adjusted odds ratio of primary and secondary outcomes comparing patients between changas NICM and other NICM.

and hypertension. Diabetes may contribute to diastolic dysfunction and subsequent heart failure.

Chagas cardiomyopathy is characterized by extensive myocarditis accompanied by localized fibrosis in the apical and basal regions of the posterior and inferior walls [20]. This area, recognized for its high arrhythmogenic potential, results in a significant incidence of tachyarrhythmias, particularly ventricular tachycardia and ventricular fibrillation, which are twice as common in individuals with Chagas disease compared to those with other non-ischemic cardiomyopathies [21]. Consequently, there is a higher rate of interventions with pacemakers (PPM) and implantable cardioverter-defibrillators (ICD) among patients suffering from Chagas cardiomyopathy. Right bundle branch block (RBBB) and atrioventricular (AV) nodal dysfunction were more often associated with both non-ischemic cardiomyopathy (NICM) and Chagas disease. However, no statistically significant differences were found between atrial fibrillation and the left bundle branch block (LBBB) study.

Severe parasitemia and direct tissue invasion cause inflammation and fibrosis, leading to dilated cardiomyopathy characterized by a typical pattern of left ventricular (LV) enlargement and impairment in global and segmental systolic function, irrespective of EKG results [5]. This condition results in increased cardiac mass, wall thinning, and dilation of both the left and right ventricles [22]. While the exact mechanism through which Chagas disease induces fibrosis and tissue damage remains unclear, it is likely linked to chronic immune responses.

Patients with chronic Chagas disease and dilated cardiomyopathy are frequently diagnosed

with acute decompensated heart failure. Previous studies, along with our findings [23], indicate that these patients are more prone to experience hospitalizations due to heart failure and cardiogenic shock. When compared to other non-ischemic cardiomyopathies, Chagas disease is associated with statistically higher rates of HFrEF, left ventricular thrombus, cardiogenic shock, and the necessity for cardiac transplantation. Nonetheless, there are no significant differences in mortality, length of stay, or total costs. Thus, early identification and management of Chagas cardiomyopathy are essential for preventing severe cardiac complications. Early detection through serological testing and vigilant monitoring of high-risk populations allows for prompt intervention.

Implementing comprehensive management strategies that incorporate antiparasitic therapy, lifestyle modifications, and cardiovascular medications during the early stages of the disease is essential for slowing or preventing declines in cardiac function. Regular cardiac monitoring and timely interventions enhance outcomes and improve the quality of life for individuals affected by Chagas cardiomyopathy [24]. Treatment with Benznidazole during the acute phase is associated with a reduced progression of chronic Chagas disease, achieving eradication in 60-90% of cases. However, according to the recent BENEFIT trial [25], Benznidazole treatment in chronic cardiomyopathy did not produce better clinical outcomes. The management of heart failure, arrhythmias, and heart transplant eligibility resembles that of other non-ischemic cardiomyopathies (NICM).

Predicting the progression of Chagas heart disease remains difficult, with electrocardiographic changes serving as standard monitoring parameters. Common abnormalities, such as right bundle branch block (RBBB), often indicate the transition from indeterminate to chronic cardiac forms, suggesting an increased risk of severe cardiomyopathy [26]. While early serum biomarkers like NT-proBNP (N-terminal pro-b-type natriuretic peptide) show potential, they lack consistency and established clinical value [27]. Imaging methods such as echocardiography and stress testing may uncover cardiac findings in seemingly asymptomatic patients, facilitating early detection and management of Chagas heart disease [15]. Further research is necessary to evaluate risk factors that can predict progression to chronic Chagas cardiomyopathy, identify biomarkers, and analyze outcomes of Chagas cardiomyopathy.

These findings highlight the notably aggressive nature of Chagas-related NICM and its significant clinical ramifications. The markedly elevated odds of heart transplantation (OR 15.48; $P<0.05$) indicate that Chagas NICM frequently advances to end-stage heart failure or severe myocarditis, thereby requiring advanced therapeutic interventions. This is further corroborated by the increased incidence of cardiogenic shock (OR 2.7; $P<0.05$), likely resulting from the chronic myocardial inflammation, fibrosis, and autonomic dysfunction characteristic of Chagas cardiomyopathy. The elevated risk of ventricular fibrillation (OR 4.87; $P<0.05$) and pericardial effusion (OR 3.75; $P<0.05$) underscores the arrhythmogenic and inflammatory burden of the disease, complicating its clinical management. Additionally, the increased necessity for pacemaker placement (OR 2.80; $P<0.05$) underscores the prevalence of conduction system disease, emphasizing the importance of rigorous rhythm monitoring and timely interventions. Despite these severe complications, the comparable in-hospital mortality rates between patients with Chagas NICM and those with other forms of NICM may reflect advancements in contemporary management practices, including early detection, optimized medical therapy, and access to advanced heart failure treatments. Given the predominance of Chagas NICM within the Hispanic population, these findings underscore the urgent need for targeted screening, prompt intervention, and enhanced access to specialized care in endemic regions to mitigate disease progression and improve long-term outcomes.

Limitations

While our study has certain limitations, we have taken every possible step to ensure the reliability and quality of our findings. As a retrospective study, we recognize the inherent limitations of this design. However, we utilized the National Inpatient Sample (NIS) from 2016 to 2020 and followed the established practices for the NIS to yield trustworthy results. Additionally, we

conducted comprehensive multivariable regression analyses to minimize potential selection bias and unadjusted confounders. Although our sample size for Chagas disease is limited, we used adjusted outcomes to produce reliable findings. Despite the risks of coding errors and underreported comorbidities in both groups, we have carefully verified our data to ensure the accuracy of our results. We believe our study provides valuable insights into the clinical and procedural outcomes of interest, and we encourage clinicians to consider sex differences in renal artery stenting outcomes when determining suitable treatment options for their patients.

Conclusion

In summary, our research highlights the distinctive clinical profile of patients with Chagas disease-related non-ischemic cardiomyopathy, which is predominantly prevalent among the Hispanic population. These patients face significantly higher odds of requiring heart transplantation due to increased risks of severe myocarditis, cardiogenic shock, and end-stage ACC-Stage D heart failure. Furthermore, Chagas NICM is associated with a higher incidence of ventricular fibrillation, pericardial effusion, and the need for pacemakers. Notably, the risk of in-hospital mortality is comparable to other forms of non-ischemic cardiomyopathy. These findings emphasize the urgent need for early detection and targeted management of Chagas NICM to improve patient outcomes.

Disclosure of conflict of interest

None.

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