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Original Article

Cardiac amyloidosis risk across ethnoracial and clinical subgroups: a five-year national study

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Abstract: Background: Cardiac amyloidosis is underdiagnosed. Amyloid deposits cause progressive diastolic dysfunction of a nondilated ventricle. Among the main subtypes, transthyretin amyloidosis (ATTR) affects 1 in 2,000 people, while light-chain (AL) amyloidosis is increasing in prevalence. Early identification of risk factors and prompt intervention may prevent severe complications. This study aimed to identify the risk factors for developing cardiac amyloidosis and its associated morbidity and mortality. Methods: This retrospective cohort study used the National Inpatient Sample (2016-2020) to examine patients hospitalized with cardiac amyloidosis identified by ICD-10 codes. Over 50 risk factors, including hematologic, rheumatologic, and renal conditions, were evaluated as possible contributors. Predictors of cardiac amyloidosis and mortality were analyzed by demographic group. Inclusion required a primary or secondary diagnosis. Multiple logistic regression, adjusted for age, sex, and comorbidities, assessed associations. The primary outcome was cardiac amyloidosis; the secondary outcome was in-hospital mortality. Results: There were 23,119 patients with cardiac amyloidosis. The mean age was 72.23 years, and 58% were male. Hereditary transthyretin amyloidosis (OR 215.89, 95% CI: 176.17-264.56) and familial Mediterranean fever (OR 111.99, 95% CI: 72.39-173.24) showed the strongest associations, both statistically significant (P<0.001). Additional risk factors included hematologic conditions, such as multiple myeloma (OR 15.31), Waldenström's macroglobulinemia (OR 6.99), and MGUS (OR 4.45), all P<0.001. Renal conditions such as chronic kidney disease (OR 1.83), end-stage renal disease (OR 2.76), and renal transplant (OR 2.4), and rheumatologic conditions such as Sjögren's syndrome (OR 2.04) and sarcoidosis (OR 1.69) were also significant (all P<0.001). By demographics, Black race (OR 2.52, P<0.001), Asian race (OR 1.11, P = 0.034), and older age (OR 1.05, P<0.001) were associated with increased risk. Notably, asthma, diabetes, and obesity were not linked to cardiac amyloidosis. In-hospital mortality increased with ESRD (OR 2.4), multiple myeloma (OR 1.41), and older age (OR 1.015). Conclusion: Hereditary transthyretin amyloidosis, familial Mediterranean fever, and Black race were the strongest risk factors, while end-stage renal disease and multiple myeloma correlated with higher in-hospital mortality. ICD-10-based diagnoses may limit accuracy. This stresses the need for improved risk stratification and targeted screening.

Keywords: Cardiac amyloidosis, amyloidosis, heart failure, race

Introduction

Cardiac amyloidosis is an increasingly recognized but historically underdiagnosed cause of restrictive cardiomyopathy and heart failure [1]. It leads to major morbidity and mortality in systemic amyloidosis due to extracellular deposition of misfolded amyloid fibrils within the myocardium [1]. Established risk factors include advanced age, male sex, plasma cell dyscrasias (e.g., multiple myeloma), chronic renal disease, and chronic inflammatory conditions [2]. Infiltration causes wall thickening, concentric remodeling, and reduced output, leading to heart failure and arrhythmias [3]. Amyloid in other organs can cause kidney failure and neuropathy [2].

There are over 30 known amyloidogenic precursors. However, most cardiac amyloidosis involves two subtypes: light-chain (AL) amyloidosis and transthyretin (ATTR) amyloidosis [4]. AL amyloidosis comes from monoclonal immunoglobulin light chains produced by clonal plasma

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cells. ATTR amyloidosis is due to the hepatic transport protein transthyretin [4]. It is important to note that ATTR amyloidosis includes both hereditary (ATTRv) and wild-type (ATTRwt) forms [4, 5].

New imaging techniques, biomarker assays, and increased knowledge have improved the detection of cardiac amyloidosis. Nevertheless, the disease's true burden across groups remains unclear, indicating the need for further study [6].

Cardiac involvement causes most illness and death in systemic amyloidosis, regardless of type [5]. Untreated AL cardiac amyloidosis is highly aggressive, with a median survival of under one year [6]. ATTR amyloidosis has a median survival of about four years [6].

Disease progression often leads quickly to advanced heart failure [7]. As a result, early mortality remains high; up to 23% of patients die within six months, especially in certain groups [8].

Recognition of cardiac amyloidosis is rising as diagnostic resources improve. AL amyloidosis occurs at 9.7-14.0 cases per million per year. ATTR amyloidosis, now at 14-27 cases per million per year, is also increasing [9].

Cardiac amyloidosis prevalence is likely underestimated. Geographic studies show substantial differences in detection near amyloidosis centers [10]. Autopsies reveal higher rates, especially in the elderly [11]. Recent data show notable racial and ethnic differences in prevalence and outcomes. For example, the V122I transthyretin mutation, found in 3-4% of those of African ancestry in the U.S., is the most common TTR variant worldwide. This mutation increases hereditary ATTR cardiomyopathy risk, driving more aggressive disease and most ATTR cases in African Americans [12, 13]. African Americans also have higher mortality, with men's rates twice those of other groups. Some studies report underdiagnosis and health system barriers. However, outcomes and survival by race and ethnicity are poorly described, as most studies include mainly Caucasian patients [10]. Patients from underrepresented groups more often present with severe disease, more comorbidities such as diabetes and hypertension, and worse baseline cardiac function [4, 14].

Few nationwide studies have examined cardiac amyloidosis risk and mortality by race, ethnicity, and clinical factors, leaving a significant knowledge gap. To address this, we specifically examined how clinical and demographic factors affect cardiac amyloidosis and mortality across subgroups. Our main goal was to identify predictors of disease development and mortality by demographic group.

Methods

This retrospective cohort study used the National Inpatient Sample (NIS) database (2016-2020), the largest all-payer inpatient database in the U.S., which generates national estimates of hospitalizations and outcomes [15].

Study population

Adult patients (≥18 years) were included if they had a primary or secondary discharge diagnosis of cardiac amyloidosis. Diagnosis was identified using ICD-10 codes for amyloidosis (E85.1, E85.2, E85.3, E85.4, E85.8, E85.9) plus codes for cardiac involvement (e.g., heart failure, cardiomyopathy, arrhythmia). Patients with missing demographic data were excluded. Table S1 lists the cardiac codes.

Variable definitions and data extraction

The extracted demographics included age, sex, and race/ethnicity. Race/ethnicity was defined as non-Hispanic White, non-Hispanic Black, Hispanic, Asian, or Other. Clinical risk factors were identified using ICD-10 codes. Clinical subgroups included hematologic (multiple myeloma, Waldenström's macroglobulinemia, MGUS), renal (CKD, ESRD, renal transplant), and rheumatologic (Sjögren's syndrome, sarcoidosis, familial Mediterranean fever) conditions. Comorbidities analyzed were diabetes, hypertension, obesity, and asthma. The ICD-10 code list used for data extraction is presented in Table S1.

Outcomes

The primary outcome was the presence of cardiac amyloidosis. The secondary outcome was in-hospital mortality.

Statistical analysis

Descriptive statistics were used to summarize baseline characteristics. Categorical variables

are reported as frequencies and percentages, and continuous variables are reported as means with standard deviations (mean ± SD). Categorical variables were compared using chi-square tests. Continuous variables were analyzed using t-tests or Wilcoxon rank-sum tests as appropriate. Multivariable logistic regression assessed associations between demographic and clinical risk factors and cardiac amyloidosis. Predictors of in-hospital mortality were examined, adjusting for age, sex, race/ethnicity, and comorbidity burden. Odds ratios (ORs) and 95% confidence intervals (CIs) were reported. Sensitivity analyses excluded patients with incomplete data and stratified patients by amyloidosis subtype. A *p*-value <0.05 was considered to be statistically significant. Those with a *p*-value <0.2 were included in the multivariate analysis. Statistical analysis was carried out using IBM SPSS Statistics, version 28.0 (IBM Corp., Armonk, NY, USA).

Ethical considerations

The study used de-identified, publicly available data and was exempt from institutional review board approval.

Results

Demographic characteristics

A total of 23,119 adults with cardiac amyloidosis were identified and included in this study. The mean age was 72.23 ± 11.67 years, and 58% were male. **Table 1** summarizes the demographic and clinical characteristics of patients with cardiac amyloidosis.

Ethnoracial associations

Table 2 highlights the demographic, ethnoracial, and clinical risk factors associated with the development of cardiac amyloidosis and mortality. Older age, male gender, and Black race were independently associated with increased risk of developing cardiac amyloidosis. For example, African-American patients had more than twice the odds of cardiac amyloidosis compared to White patients (OR 2.52, 95% CI: 2.44-2.61). Asian patients also showed a modest but significant increased risk (OR 1.11, 95% CI: 1.02-1.21). Each additional year of age increased the risk by 5% (OR 1.05, 95% CI: 1.04-1.06).

Clinical risk factors

The strongest clinical associations were observed for hereditary transthyretin amyloidosis (OR 215.89, 95% CI: 176.17-264.56) and familial Mediterranean fever (OR 111.99, 95% CI: 72.39-173.24), both P<0.001. Hematologic disorders were highly predictive: multiple myeloma (OR 15.31, 95% CI: 14.63-16.03), Waldenström's macroglobulinemia (OR 6.99, 95% CI: 5.46-8.95), and MGUS (OR 4.45, 95% CI: 4.08-4.86), all P<0.001. Renal conditions also increased risk: chronic kidney disease (OR 1.83, 95% CI: 1.71-1.96), end-stage renal disease (OR 2.76, 95% CI: 2.41-3.16), and renal transplant (OR 2.4, 95% CI: 1.98-2.91). Rheumatologic conditions, including Sjögren's syndrome (OR 2.04, 95% CI: 1.67-2.50) and sarcoidosis (OR 1.69, 95% CI: 1.41-2.02), were also significant. Asthma, diabetes, and obesity were not associated with increased risk (**Table 2**).

In-hospital mortality and outcomes

In the overall cohort, the in-hospital mortality rate was 7.4%. Mortality increased significantly with advancing age (OR 1.015 per year, 95% CI: 1.012-1.018) and was higher among African American patients compared to White patients (OR 2.01, 95% CI: 1.88-2.15). Among comorbidities, hereditary transthyretin amyloidosis showed the strongest association with mortality (OR 5.93, 95% CI: 3.37-9.48), followed by end-stage renal disease (OR 2.4, 95% CI: 1.88-3.07), diabetes mellitus (OR 1.81, 95% CI: 1.51-2.1), and multiple myeloma (OR 1.41, 95% CI: 1.20-1.67) (**Table 2**).

Discussion

In this extensive study, key factors associated with cardiac amyloidosis and in-hospital mortality were identified. Older age and male sex were major demographic factors linked to cardiac amyloidosis. Clinical factors included familial Mediterranean fever; hematologic disorders such as multiple myeloma (MM), Waldenström's macroglobulinemia (WM), and monoclonal gammopathy of unknown significance (MGUS); renal dysfunction, particularly end-stage renal disease (ESRD); and rheumatologic conditions such as Sjögren's syndrome and sarcoidosis. Notably, older age, ESRD, and MM were associated with increased in-hospital mortality. These data demonstrate the com-

Table 1. Baseline demographic and clinical characteristics of identified patients with cardiac amyloidosis

Variables	N = 23,119
Age (mean ± SD)	72.23 ± 11.67
n (%)	
Gender	
Male	13,379 (58)
Female	9,740 (42)
Race	
White	14,209 (61.4)
Black	5,429 (23.5)
Hispanic	1,482 (6.4)
Asian	614 (2.6)
Native American	81 (0.3)
Other	616 (2.7)
Median household income	
0-25th percentile	5,286 (22.8)
26-50th percentile	5,201 (22.5)
51-75th percentile	5,685 (24.6)
76-100th percentile	6,604 (28.5)
Comorbidities	
Hereditary Transthyretin Amyloidosis	162 (0.7)
Common Comorbidities	
Smoking	5,704 (24.8)
Obesity	2,105 (9.1)
Diabetes Mellitus	6,154 (26.6)
Liver Cirrhosis	601 (2.6)
Chronic Kidney Disease Stages III-IV	5,640 (24.4)
End Stage Renal Disease	2,111 (9.1)
Renal Replacement Therapy	1,498 (6.5)
Kidney Transplant	925 (4.0)
Asthma/Seasonal Allergy	1,145 (4.9)
Hematologic Conditions	
Multiple Myeloma	2,613 (11.3)
Plasma Cell Leukemia	7 (0.03)
Extramedullary plasmacytoma	5 (0.02)
Monoclonal Gammopathy of Undetermined Significance	626 (2.7)
Waldenström's Macroglobulinemia	107 (0.5)
Leukemia	246 (1.1)
Non-Hodgkin Lymphoma	318 (1.4)
Hodgkin Lymphoma	7 (0.03)
Immune/Rheumatologic Conditions	
Crohn's Disease	81 (0.3)
Ulcerative Colitis	83 (0.4)
Chronic Osteomyelitis	119 (0.5)
Tuberculosis	10 (0.04)
Rheumatoid Arthritis	517 (2.2)
Juvenile Arthritis	6 (0.02)
Ankylosing Spondylitis	21 (0.1)
Familial Mediterranean Fever	38 (0.2)
Systemic Lupus Erythematosus	101 (0.4)
Sjögren's Syndrome	91 (0.4)
Sarcoidosis	124 (0.5)
Overlapping Rheumatologic Syndromes	12 (0.1)
Polymyalgia Rheumatica	97 (0.4)
Giant Cell Arteritis	26 (0.1)
Takayasu Arteritis	4 (0.01)
Behçet's Disease	4 (0.01)

plex, multisystem nature of cardiac amyloidosis, where both hereditary predispositions along with acquired comorbidities contribute to disease development and progression.

This study corroborates prior literature confirming that older age and male sex are major demographic factors associated with cardiac amyloidosis [16]. These results match with a nationwide retrospective study by Choi et al., which included 2,239 amyloidosis patients, of whom 758 had cardiac involvement, with a mean age of 64 years and a predominance of males [17]. The study also recorded an increase in average patient age over time, from approximately 60 years in 2009 to 68 years in 2020 [17]. Similarly, a single-center retrospective analysis of 62 patients assessing risk factors for one-year survival after cardiac amyloidosis diagnosis found a predominantly older male cohort (87.1%) with a mean age of 72 years [18]. Age-related changes may explain the increased susceptibility of older adults to cardiac amyloidosis [19]. Poor protein quality control, cumulative cellular stress, and organ susceptibility contribute to the development of this disease. In ATTR, aging affects cellular handling of misfolded proteins, leading to gradual cardiac amyloid accumulation, whereas AL amyloidosis incidence increases as age increases due to rising plasma cell disorders [20]. Regarding male predominance, sex-related differences are influential. Endogenous estrogen in women exerts cardioprotective effects, in-

Table 2. Multivariable analysis of factors associated with the risk of developing cardiac amyloidosis and mortality

Variables	Risk of Developing Cardiac Amyloidosis	Risk of Developing Cardiac Amyloidosis	Risk of Mortality	Risk of Mortality
	OR (95% CI)*	*p*-value*	OR (95% CI)*	*p*-value*
Age	**1.05 (1.04-1.06)**	**<0.001**	**1.01 (1.012-1.018)**	**<0.001**
Female	**0.63 (0.62-0.65)**	**<0.001**	0.92 (0.83-1.03)	0.166
Race				
Black	**2.52 (2.44-2.61)**	**<0.001**	**2.01 (1.88-2.15)**	**0.008**
Hispanic	1.05 (0.996-1.11)	0.068	1.01 (0.81-1.27)	0.932
Asian	**1.11 (1.02-1.21)**	**0.034**	1.16 (0.85-1.58)	0.365
Native American	1.08 (0.86-1.35)	0.528	1.84 (0.87-3.87)	0.108
Other	1.27 (0.71-1.73)	0.1	0.78 (0.54-1.13)	0.184
Comorbidities				
Smoking	**1.87 (1.52-2.22)**	**<0.001**	**1.70 (1.61-1.8)**	**<0.001**
Obesity	1.67 (0.98-2.36)	0.064	0.84 (0.68-1.05)	0.124
Diabetes Mellitus	1.65 (0.63-2.71)	0.131	**1.81 (1.51-2.1)**	**0.004**
Liver Cirrhosis	**1.17 (1.08-1.27)**	**<0.001**	1.32 (0.97-1.80)	0.074
Chronic Kidney Disease Stages III-IV	**1.83 (1.71-1.96)**	**<0.001**	0.91 (0.79-1.04)	0.155
End Stage Renal Disease	**2.76 (2.41-3.16)**	**<0.001**	**2.4 (1.88-3.07)**	**<0.001**
Renal Replacement Therapy	0.85 (0.67-1.03)	0.066	0.91 (0.75-1.04)	0.081
Kidney Transplant	**2.4 (1.98-2.91)**	**<0.001**	**0.59 (0.42-0.84)**	**0.003**
Multiple Myeloma	**15.31 (14.63-16.03)**	**<0.001**	**1.41 (1.20-1.67)**	**<0.001**
Plasma Cell Leukemia	0.51 (0.24-1.07)	0.075	1.63 (0.19-14.04)	0.655
Extramedullary Plasmacytoma	1.02 (0.38-2.77)	0.972	1.97 (0.71-1.43)	0.999
Monoclonal Gammopathy of Undetermined Significance	**4.45 (4.08-4.86)**	**<0.001**	0.93 (0.66-1.31)	0.678
Waldenström's macroglobulinemia	**6.99 (5.46-8.95)**	**<0.001**	0.92 (0.40-2.13)	0.839
Leukemia	0.88 (0.78-1.01)	0.061	1.49 (0.96-2.33)	0.079
Non-Hodgkin lymphoma	1.05 (0.91-1.20)	0.517	1.55 (0.96-2.51)	0.074
Hodgkin lymphoma	0.66 (0.32-1.39)	0.276	2.44 (0.29-20.73)	0.414
Hereditary Transthyretin Amyloidosis	**215.89 (176.17-264.56)**	**<0.001**	5.93 (3.37-9.48)	0.832
Crohn's Disease	0.99 (0.78-1.12)	0.08	0.86 (0.31-2.36)	0.764
Ulcerative Colitis	0.99 (0.80-1.24)	0.959	0.72 (0.26-1.99)	0.532
Asthma/Seasonal Allergy	1.82 (0.77-2.88)	0.07	0.70 (0.32-1.07)	0.081
Chronic Osteomyelitis	1.47 (0.39-2.56)	0.13	1.00 (0.46-2.18)	0.994
Tuberculosis	1.39 (0.75-2.59)	0.3	1.75 (0.22-14.00)	0.599
Rheumatoid Arthritis	1.04 (0.95-1.14)	0.346	0.81 (0.54-1.22)	0.314
Juvenile Arthritis	**3.00 (1.34-6.71)**	**0.008**	0.93 (0.74-1.32)	0.999
Ankylosing Spondylitis	1.37 (0.88-2.14)	0.162	0.8 (0.3-1.98)	0.998
Familial Mediterranean fever	**111.99 (72.39-173.24)**	**<0.001**	1.64 (0.56-4.75)	0.365
Systemic Lupus Erythematosus	0.97 (0.79-1.18)	0.756	0.49 (0.15-1.57)	0.228
Sjögren's Syndrome	**2.04 (1.67-2.5)**	**<0.001**	0.63 (0.20-2.00)	0.427
Sarcoidosis	**1.69 (1.41-2.02)**	**<0.001**	0.84 (0.37-1.91)	0.677
Overlapping Rheumatologic Syndromes	1.45 (0.82-2.58)	0.201	2.94 (0.63-13.70)	0.169
Polymyalgia Rheumatica	0.88 (0.72-1.08)	0.229	0.63 (0.23-1.71)	0.362
Giant Cell Arteritis	1.10 (0.74-1.63)	0.633	1.96 (0.58-6.59)	0.276
Takayasu Arteritis	1.51 (0.56-4.05)	0.413	1.3 (0.34-3.48)	0.999
Behçet's Disease	2.57 (0.82-8.00)	0.104	2.7 (0.49-5.95)	0.999

*Numbers in bold are statistically significant.

cluding attenuation of oxidative stress, inhibition of interstitial fibrosis, and modulation of inflammatory pathways, thus lessening the harmful effects of amyloid deposition on myocardial structure and function. In men, the absence of this hormonal protection, in combination with potential androgen-mediated effects on transthyretin expression and aggregation, may raise susceptibility to cardiac amyloid infiltration and dysfunction [21].

Ethnoracial disparities

The study's finding that African-Americans carried the highest odds ratio of developing cardiac amyloidosis (aOR 2.52, 95% CI: 2.44-2.61, P<0.001) and had higher in-hospital mortality (aOR 2.01, 95% CI: 1.88-2.15, P = 0.008) is consistent with the existing evidence that racial differences exist among this subpopulation [22]. The V122I transthyretin mutation, carried by approximately 3-4%, is the most common pathogenic TTR variant in the United States and involves the heart, with a worse prognosis than ATTRwt [23]. In the Transthyretin Amyloid Outcome Survey (THAOS), approximately 25% of enrolled patients in the United States were of African descent compared to 0.5% in other regions worldwide; V122I was the most prevalent pathogenic mutation in this population [24]. Additionally, a study of over 1,000 patients with ATTR cardiomyopathy at the UK National Amyloidosis Centre demonstrated that the V122I mutation was associated with shorter median survival (31 vs. 57 months for ATTRwt, P<0.001) [25]. A recent study by Shankar et al. affirmed these disparities: among 282 patients with ATTR cardiomyopathy, African-American patients (consisting 46% of the cohort) were more likely to have heart failure or die over a 5-year period (P<0.001), with the Black race being an independent risk factor for heart failure hospitalization or death (HR 1.97, 95% CI: 1.21-3.21, P = 0.007) [26]. This study also showed that poor socioeconomic status accompanied racial disparities, as Black patients with a high area deprivation index had a 2.77-fold higher hazard of heart failure hospitalization or death compared to Caucasian patients [26]. The association between biological factors (i.e., V122I mutation), underdiagnosis, and socioeconomic barriers likely explains the high risk and worse outcomes observed among African-Americans in this study.

The underdiagnosis of cardiac amyloidosis can also be attributed to geographic disparities. Spencer Bonilla et al. demonstrated that southern US states reported the lowest amyloidosis mortality, likely as a result of underdiagnosis rather than a true low disease burden [22]. This suggests that inequities in access to care and treatment, more than intrinsic disease biology, contribute to the noted survival differences.

The present study demonstrated a modest but statistically significant association between Asian race and cardiac amyloidosis (aOR 1.11, 95% CI: 1.02-1.21, P = 0.034) as well. In the Asian scientific literature, ATTR cardiac amyloidosis is not widely recognized, but emerging evidence suggests marked underdetection in this population [27]. Case reports have reported the existence of the V122I variant in Asian individuals, putting the assumption that this mutation is confined to those of African descent into question [28].

Hematologic disorders

An association between hematological disorders and cardiac amyloidosis was noted. In a retrospective study conducted by Phull et al. among patients with biopsy-proven ATTRwt and genopositive ATTR V122I amyloidosis, it was noted that 39% of the ATTRwt cohort and 49% of the ATTR V122I cohort had MGUS [1]. Additionally, evidence from Waldenström's macroglobulinemia-associated AL amyloidosis (WM-AL) supports this association. In a cohort of 49 patients evaluated between 2006 and 2022, 10 patients (20%) were diagnosed with WM and AL amyloidosis simultaneously, while 39 patients (80%) developed AL amyloidosis a median of three months after WM diagnosis [29]. Similarly, an association between cardiac amyloidosis and multiple myeloma has been described. In one study of 47 patients who developed AL amyloidosis six months following diagnosis, 62% had single-organ involvement and 23% had multiorgan disease, with cardiac involvement as a key driver of poor outcomes [2]. The present study's results show that it significantly increases mortality in patients with cardiac amyloidosis (OR 1.41; 95% CI: 1.20-1.67). A recent systematic review and meta-analysis done by Stabile et al. demonstrated that among 264 patients, 109 (41%) had multiple myeloma with co-existing cardiac AL amyloidosis [30]. At 12 months, the all-cause mortality was nearly five-fold higher in patients with cardiac involvement compared to those without (RR 4.73; 95% CI: 1.82-12.27; P = 0.001) [30]. This persisted at 24 months (RR 1.87; 95% CI: 1.17-3.00; P = 0.009) and 36 months (RR 1.82; 95% CI: 1.22-2.73; P = 0.004) [30]. This is attributed to excessive production of amyloidogenic light chains with subsequent cardiac amyloid fibril deposition and myocardial stiffness,

resulting in diastolic dysfunction and heart failure [30].

Renal conditions and end-stage renal disease

Studies directly linking end-stage renal disease (ESRD) to cardiac amyloidosis and mortality are scarce. A large retrospective registry (1963-2010) of 58,422 patients in Australia and New Zealand on renal replacement therapy showed that only 490 patients (0.8%) had ESRD due to amyloidosis. It reported that amyloidosis was associated with poor survival following dialysis and/or renal transplantation, with 10-year survival rates of 37% compared to 69% in patients with other ESRD causes (P<0.001) [31]. This is supported by a smaller cohort study of 48 patients with systemic amyloidosis and ESRD on dialysis compared to 63 nondiabetic dialysis patients without amyloidosis. Martinez-Vea et al. reported that the median survival in the amyloidosis group was 52 months, with 1-, 2-, and 6-year survival rates of 72%, 62%, and 44%, respectively, compared with 95%, 91%, and 81% in controls (P<0.001) [32]. Survival was shorter in patients who progressed to ESRD (<3 months) or experienced acute renal deterioration before dialysis, with median survivals of 4 and 1.5 months, respectively [32]. Consistent with our findings that ESRD is a risk factor for cardiac amyloidosis (aOR 2.76, 95% CI: 2.41-3.16, P<0.001) and mortality (aOR 2.4, 95% CI: 1.88-3.07, P<0.001), a recent analysis of 114,790 hospitalized amyloidosis patients found that 12.2% had ESRD, which was associated with a 37% higher risk of in-hospital mortality (OR 1.37, 95% CI: 1.17-1.62, P<0.001) and increased risk of complications (e.g., cardiac arrest, acute respiratory failure septic shock) [33]. In patients with ESRD, the increased risk of amyloidosis and mortality may be explained by uremia and fluid overload, which strain the cardiovascular system and increase the risk of heart failure and sudden cardiac death [33].

Rheumatologic and immunologic diseases

In this study, Sjögren's syndrome (OR 2.04, 95% CI: 1.67-2.5, P<0.001) and sarcoidosis (OR 1.69, 95% CI: 1.41-2.02, P<0.001) were identified as significant risk factors for cardiac amyloidosis. AA amyloidosis, resulting from chronic systemic inflammation, is a known complication of primary Sjögren's syndrome [34]. In a review and case series by Zaher et al., only 8 cases of AA amyloidosis secondary to primary Sjögren's syndrome were detected, with the kidneys as the most commonly involved organs [34]. It carries several cardiovascular implications: in a population-based cohort study (N = 4,175) conducted by Wu et al., patients with primary Sjögren's syndrome reported an adjusted hazard ratio (HR) of 1.17 (95% CI, 1.03-1.34) for coronary artery disease [35]. A separate Taiwanese cohort study noted that primary Sjögren's syndrome is an independent risk factor for heart failure, with cumulative 5- and 10-year incidences of 1.89% and 4.33%, respectively (Lin et al). Nevertheless, the risk of heart failure-related hospitalization did not differ significantly from that in the general population (HR: 0.98, 95% CI: 0.84-1.14) [36].

The association between sarcoidosis and cardiac amyloidosis observed in the present study reflects the diagnostic complexity and pathophysiologic overlap between infiltrative cardiomyopathies. Sarcoidosis and amyloidosis can both present with concentric ventricular hypertrophy, diastolic dysfunction, and conduction abnormalities. In a pathology series, Treaba et al. noted that sarcoidosis was found to coexist with AL amyloidosis in at least two autopsy cases [37].

The strong association between familial Mediterranean fever (FMF) and cardiac amyloidosis (OR 111.99; 95% CI: 72.39-173.24, P<0.001) in this study reflects the well-established relationship between FMF and AA amyloidosis [38]. FMF remains the leading cause of AA amyloidosis in endemic regions, accounting for 78.7% of cases in a recent study conducted by Bektas et al. among 174 patients with AA amyloidosis [39].

The growing literature suggests that the cardiovascular complications among patients with FMF are more prevalent than formerly recognized. In the same study completed by Bektas et al. in Turkey (2024), cardiac involvement was reported in 20.2% of AA amyloidosis patients and in 21.2% of cases with FMF [39]. They found that cardiac involvement was an independent risk factor for increased mortality, with an OR of 2.8 (95% CI: 1.07-7.3, P = 0.037) in the overall sample size analysis and an OR of 12.8 (95% CI: 2.54-64.9, P = 0.002) in the FMF-AA amyloidosis subgroup [39]. Cardiac

involvement in FMF shows in various ways, including pericarditis, diastolic dysfunction, and amyloid deposition in the heart and vessel walls [40]. It has also been associated with higher long-term prevalence of ischemic heart disease, atrial fibrillation, stroke, and heart failure [41].

Age

Older age is a known predictor of mortality among patients with cardiac amyloidosis. A large cohort study from the UK National Amyloidosis Centre involving 295 patients with AL amyloidosis (median age 78.5 years) reported poor survival. The 1-, 2-, and 5-year overall survival rates were 59%, 47%, and 26%, respectively, which were significantly worse than the median overall survival of 6.1 years observed in patients aged <75 years over the same period. The overall survival decreased with advancing age from 24.2 months in patients aged 75-80 years to 13.5 months in those aged >80 years [42]. Furthermore, data from a Korean nationwide retrospective study support the association between older age and decreased survival, where patients aged ≥70 years had significantly higher in-hospital mortality compared to those aged <70 years (P = 0.004) [17]. This is further supported by a study of 165 treatment-naïve AL cardiac amyloidosis patients presenting with symptomatic heart failure at Boston University Medical Center (mean age 61.6 ± 9.5 years), where median survival was 10.9 months (95% CI, 6.2-14.7), and older age independently predicted mortality (HR = 1.04; 95% CI, 1.01-1.06) [43]. The higher mortality observed in older patients with cardiac amyloidosis can be ascribed to multiple causes. One important factor is amyloid fibril accumulation, which increases myocardial stiffness, leading to diastolic dysfunction and raising the risk of developing heart failure with preserved ejection fraction (HFpEF), causing increased mortality [43]. In addition, frailty is common among older adults and is frequently found among patients with transthyretin cardiac amyloidosis. This group tolerates aggressive therapies poorly, experiences worse heart failure symptoms, and is more prone to therapy-related complications [43]. Frailty increases the physiologic burden of the disease by impacting multiple organ systems involved in amyloidosis, significantly increasing mortality in older adults [44].

Limitations

This study has several limitations. The NIS is an administrative database that relies on ICD-10 codes to identify diagnoses and comorbidities. These codes are prone to variation in documentation methods, coding errors, and the absence of specificity. Additionally, this database does not include unique patient identifiers. Thus, readmissions may be counted as separate encounters, potentially inflating the case count. Lastly, residual confounding from unmeasured variables (e.g., disease stage, biomarker levels, echocardiographic parameters, and administered therapies) cannot be excluded in this study.

Future directions

The results in this study carry several implications. Large geographic disparities in amyloidosis mortality suggest widespread underdiagnosis, especially in areas with high prevalence of minority populations. Future studies need to focus more on these populations. Future trial designs should prioritize recruiting participants from different ethnoracial backgrounds to ensure that therapeutic efficacy and safety are evaluated across all at-risk groups.

The significant associations between rheumatologic and autoinflammatory diseases (e.g., Sjögren's syndrome and sarcoidosis) and cardiac amyloidosis justify further investigation. Future studies need to concentrate on linking rheumatologic diseases to cardiac screening to test whether these conditions represent independent risk factors for the development of cardiac amyloidosis.

Efforts should be made to improve the specificity of ICD coding for cardiac amyloidosis subtypes to boost the reliability of future studies and ensure correct epidemiologic surveillance.

Conclusion

Cardiac amyloidosis is becoming a more common and treatable cause of heart failure, particularly among older adults, African Americans, and patients with hematologic, immunologic, and renal disorders. Risk stratification and screening should be carried out to reduce morbidity and mortality from this disease. Active research should emphasize optimizing diagnostic pathways, expanding availability to specialty

care, and handling disparities in diagnosis and management.

Disclosure of conflict of interest

None.

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Table S1. ICD-10 codes used for variables and data extraction

Variable	ICD-10 Code(s)
Heart Failure	I50.0, I50.1, I50.2, I50.3, I50.4, I50.5, I50.6, I50.7, I50.9, I50.9
Atrial Fibrillation and Flutter	I48.0, I48.1, I48.2, I48.3, I48.4, I48.91, I48.92
Paroxysmal and Other Tachycardias	I47.0, I47.1, I47.2, I47.9
Chronic Kidney Disease Stages III & IV	N18.3, N18.4
End-Stage Renal Disease (ESRD)	N18.6
Renal Replacement Therapy	Z99.2
Kidney Transplant	Z94.0
Ischemic Stroke	I63.0, I63.1, I63.2, I63.3, I63.4, I63.5, I63.6, I63.8, I63.9
Hemorrhagic Stroke	I61.0, I61.1, I61.2, I61.3, I61.4, I61.5, I61.6, I61.8, I61.9, I62.0, I62.1, I62.9
Transient Ischemic Attack (TIA)	G45.0, G45.1, G45.2, G45.3, G45.4, G45.8, G45.9
Diabetes Mellitus	E08.x, E09.x, E10.x, E11.x, E13.x
Hypertension	I10, I11.x, I12.x, I13.x, I15.x
Dyslipidemia	E78.0, E78.1, E78.2, E78.3, E78.4, E78.5, E78.6, E78.8, E78.9
Coronary Artery Disease	I20.x, I21.x, I22.x, I23.x, I24.x, I25.x
Carotid Artery Disease	I65.2, I67.2, I67.89
Peripheral Vascular Disease	I70.x, I73.9
Chronic Obstructive Pulmonary Disease	J43.x, J44.x
Obstructive Sleep Apnea	G47.30, G47.33
Morbid Obesity	E66.01
Smoking	Z72.0, F17.2
Venous Thromboembolism (DVT/PE)	I26.x, I80.x, I82.4, I82.5, I82.6, I82.7, I82.8, I82.9
Liver Cirrhosis	K74.60, K74.69
Multiple Myeloma	C90.0
Plasma Cell Leukemia	C90.1
Extramedullary Plasmacytoma	C90.2
MGUS (Monoclonal Gammopathy of Undetermined Significance)	D47.2
Waldenström's Macroglobulinemia	C88.0
Leukemia	C91.x-C95.x
Non-Hodgkin Lymphoma	C82.x-C85.x
Hodgkin Lymphoma	C81.x
Hereditary Transthyretin Amyloidosis	E85.1, E85.82
Crohn's Disease	K50.x
Ulcerative Colitis	K51.x
Asthma	J45.x
Seasonal Allergy	J30.2-J30.5
Chronic Osteomyelitis	M86.3x
Tuberculosis	A15.x-A19.x
Rheumatoid Arthritis	M05.x-M06.x
Juvenile Arthritis	M08.x, M09.x
Ankylosing Spondylitis	M45.x
Familial Mediterranean Fever	E85.0
Systemic Lupus Erythematosus	M32.x
Sjögren's Syndrome	M35.0x
Sarcoidosis	D86.x
Overlapping Rheumatologic Syndromes	M35.1
Polymyalgia Rheumatica	M35.3
Giant Cell Arteritis	M31.6
Takayasu Arteritis	M31.4
Behçet's Disease	M35.2