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Heart failure among Indigenous and non-Indigenous Australians in remote Central Australia

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Key words
Indigenous health, heart failure, left ventricular systolic dysfunction, disadvantage, risk factors, aetiology.

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Abstract

Background: There is a paucity of data on the burden of heart failure (HF) in Central Australia, the most populous Indigenous region in the country.
Aims: To characterize Indigenous and non-Indigenous Australians with HF in Central Australia.
Methods: Consecutive patients with HF and reduced ejection fraction <50% were included for the period 2019 to 2021. Clinical, echocardiographic and major adverse cardiovascular events (MACE) data were collected.
Results: Four hundred twenty-four patients with HF were included (70% Indigenous, 59% male; follow-up 2.2 ± 0.5 years). Indigenous Australians were younger (53 ± 15 vs 68 ± 13 years, $P < 0.001$) with higher rates of rheumatic heart disease (18% vs 1%, $P < 0.001$), diabetes (63% vs 33%, $P < 0.001$) and severe chronic kidney disease (CKD; 32% vs 7%, $P < 0.001$). HF was more prevalent among Indigenous (138 [95% confidence interval (CI), 123–155] per 10 000) compared with non-Indigenous Australians (53 [95% CI, 44–63] per 10 000), particularly among younger individuals and females. There were similar HF aetiologies between groups. Guideline-directed medical therapy (GDMT) was suboptimal and similar between the groups: angiotensin-converting enzyme inhibitor/angiotensin receptor blocker (64% vs 67%, $P = 0.47$) and β-blockers (68% vs 71%, $P = 0.47$). Indigenous Australians had a significantly higher rate of MACE (54% vs 28%, $P < 0.001$) and death from any cause (24% vs 13%, $P = 0.013$).
Conclusions: HF is more than two times as prevalent among Indigenous Central Australians, particularly among younger individuals and females. Despite similar HF aetiologies and GDMT, MACE and mortality outcomes are higher in Indigenous individuals with HF. These data have implications for efforts to close the Indigenous gap in morbidity and mortality.

Introduction

Heart failure (HF) continues to present a significant health care burden within Australia with high rates of morbidity and mortality.[1] Estimates of HF prevalence within Australia range from 2% to 5%; however, these numbers may be increased twofold to threefold in Indigenous Australian and nonurban communities.[2,3] These patient populations exhibit higher rates of HF risk factors, including ischaemic heart disease, hypertension and rheumatic heart disease (RHD) and associated comorbidities such as diabetes and severe chronic kidney disease (CKD),[4–6] whereas lower rates of both prescription and adherence to guideline-directed HF therapy have been well documented.[7–9] Previous studies exploring echocardiographic data specific to Indigenous Australians with HF have demonstrated incomplete data capture or included small patient numbers.[3,10] Worse cardiovascular outcomes seen in Indigenous Australians are multifactorial and stem not only from geographic and socioeconomic barriers to accessing health care but also a

Conflict of interest: None.

mistrust of institutions and providers reflecting the 'intergenerational trauma' felt by many First Nations peoples.[11]

In this study, we thus sought to characterise the contemporary prevalence, echocardiographic parameters, aetiology, comorbidities, use of guideline-directed medical therapy (GDMT) and outcomes associated with HF among Indigenous and non-Indigenous Australians living in remote Central Australia.

Methods

Study setting and design

Central Australia is well suited for the study of cardiovascular epidemiology because of several unique features. It is a vast remote geographic region spanning some 830 000 km^2 with a population of approximately 40 000 people, of which Indigenous Australians make up 44% – the largest proportionally in the country.[12] Alice Springs Hospital is the only secondary health care facility and sole provider of cardiac care in the region for all elective, emergency, inpatient and outpatient management. Furthermore, the remoteness of the region, high prevalence of disease, geographic distance and limited community health care facilities contribute to a low threshold for transfer to Alice Springs Hospital for evaluation. Even when residents undergo cardiovascular care in the community or subsequently at tertiary hospitals in South Australia and the Northern Territory, this is coordinated and catalogued by Alice Springs Hospital cardiovascular services.

Patient population

We included consecutive Indigenous and non-Indigenous Australians with HF and left ventricular systolic dysfunction (LVSD), identified by left ventricular ejection fraction (LVEF) on echocardiography <50%, coordinated by Alice Springs Hospital over a 2-year period from 2019 to 2021. Self-identification as an Aboriginal or Torres Strait Islander person was accepted as confirmation of Indigenous Australian ethnicity for this study. Clinical information pertaining to demographics, clinical characteristics, aetiology of HF, comorbidities and prescription of guideline-directed HF therapy and other medications were collected from paper and electronic medical records. Echocardiographic data were collected from imaging databases, including serial studies.

Long-term follow-up

Patients were followed up for a composite of myocardial infarction, cardiac hospitalisation, stroke, revascularisation with coronary artery bypass grafting or percutaneous coronary intervention, or all-cause death. Outcome data were determined using Central Australian cardiac databases, hospital admission records and death registries. Study approval was obtained from the Central Australia Human Research Ethics Committee.

Statistical analysis

Demographics and comorbidities were analysed using descriptive statistics, including mean and standard deviation for continuous variables. Baseline characteristics were compared using a *t* test. HF prevalence in Central Australia was estimated using classical methods for period prevalence, with the case period being 2006 to 2016 and the 2021 Australian Bureau of Statistics Census population as the estimated standard population denominator in this period. Age- and sex-specific prevalence of HF and 95% confidence intervals (CIs) were calculated for four age categories (<45, 45–54, 55–64 and ≥65 years). Indigenous to non-Indigenous age-specific prevalence ratios and CIs were then calculated. Kaplan–Meier methods were used to determine the associations of a diagnosis of HF with LVSD with outcomes, with interaction testing according to Indigenous status. Cox regression analysis was used to determine associations between clinical characteristics and comorbidities with outcomes. Analyses were undertaken using SPSS version 28.0 (IBM) and statistical significance was set at $P < 0.05$.

Results

Study population

A total of 424 patients were included (Table 1). Of these, Indigenous Australians accounted for 70% and were significantly younger (53 ± 15 years vs 68 ± 13 years, $P < 0.001$) compared with non-Indigenous individuals. There was a greater predominance of males in the non-Indigenous group compared with the Indigenous group (73% vs 53%, $P < 0.001$).

HF prevalence

The overall HF prevalence in Central Australia during the study period was 93 (95% CI, 85–102) per 10 000 people (Table 2). The prevalence of HF was significantly greater in Indigenous compared with non-Indigenous Australians. By ethnicity, the overall crude prevalence was 138 (95%

Table 1 Population characteristics for Indigenous and non-Indigenous Australians in Central Australia

	Total ($N = 424$)	Indigenous Australians ($n = 296$)	Non-Indigenous Australians ($n = 128$)	*P* value
Demographics				
Age, years	57 ± 16	53 ± 15	68 ± 13	<0.001
Male sex, %	250 (59)	156 (53)	94 (73)	<0.001
Body mass index, kg/m^2	29.1 ± 6.8	28.7 ± 6.5	30.2 ± 7.3	0.038
Comorbidities				
AMI, *n* (%)	164 (39)	122 (41)	42 (33)	0.103
PCI, *n* (%)	72 (17)	54 (18)	18 (14)	0.294
CABG, *n* (%)	57 (13)	40 (14)	17 (13)	0.949
AF, *n* (%)	104 (25)	60 (20)	44 (34)	0.002
RHD, *n* (%)	54 (13)	53 (18)	1 (1)	<0.001
Hypertension, *n* (%)	232 (55)	165 (56)	67 (52)	0.52
Hyperlipidaemia, *n* (%)	171 (40)	127 (43)	44 (34)	0.101
Diabetes, *n* (%)	227 (54)	185 (63)	42 (33)	<0.001
Smoker, *n* (%)	48 (11)	36 (12)	12 (9)	0.407
Severe CKD, *n* (%)	105 (25)	96 (32)	9 (7)	<0.001
Stroke, *n* (%)	40 (9)	20 (7)	20 (16)	0.004
Heavy alcohol use, *n* (%)	60 (14)	47 (16)	13 (10)	0.121
Echo				
LVEF (%)	38.9 ± 9.0	39.1 ± 9.2	38.3 ± 8.4	0.438
Severe LVSD (EF < 30%), *n* (%)	64 (15)	44 (15)	20 (16)	
LVEDVi (mL/m^2)	62.6 ± 21.3	60.5 ± 19.8	67.2 ± 23.5	0.005
LVESVi (mL/m^2)	39.6 ± 19	38.4 ± 18.6	42.0 ± 19.6	0.089
LVEDD (mm)	52.7 ± 8.2	51.7 ± 8.0	55.0 ± 8.0	<0.001
LVESD (mm)	41.7 ± 8.6	40.8 ± 8.2	43.6 ± 9.3	0.004
LAESVi (mL/m^2)	39.3 ± 18.1	38.2 ± 16.3	41.8 ± 21.2	0.085
Mean time to follow-up echo (years)	0.9 ± 0.5			
Follow-up LVEF (%)	42.8 ± 13.0	43.1 ± 13.4	42.4 ± 12.1	0.707
Change in LVEF (%)	5.0 ± 12.0	4.9 ± 12.1	5.1 ± 11.7	0.903
HF aetiology				
Idiopathic, *n* (%)	61 (14)	37 (13)	24 (19)	0.093
Ischaemic, *n* (%)	162 (38)	116 (39)	46 (36)	0.528
Valvular, *n* (%)	48 (11)	37 (13)	11 (9)	0.245
Hypertensive, *n* (%)	12 (3)	5 (2)	7 (6)	0.031
Alcoholic, *n* (%)	19 (5)	14 (5)	5 (4)	0.708
Tachycardia-mediated, *n* (%)	12 (3)	5 (2)	7 (6)	0.031
Other, *n* (%)	24 (6)	17 (6)	7 (6)	0.911
Unknown, *n* (%)	77 (18)	56 (19)	21 (16)	0.539
Medications				
ACEi/ARB, *n* (%)	274 (65)	188 (64)	86 (67)	0.469
β-Blockers, *n* (%)	291 (69)	200 (68)	91 (71)	0.474
MRA, *n* (%)	132 (31)	88 (30)	44 (34)	0.344
Neprilysin Inhibitor, *n* (%)	10 (2)	5 (2)	5 (4)	0.168
Aspirin, *n* (%)	201 (47)	156 (53)	45 (35)	<0.001
P2Y12 Inhibitor, *n* (%)	52 (12)	38 (13)	14 (11)	0.585
Warfarin, *n* (%)	26 (6)	13 (4)	13 (10)	0.023
NOAC, *n* (%)	64 (15)	33 (11)	31 (24)	<0.001
Loop diuretic, *n* (%)	121 (29)	82 (28)	39 (31)	0.564
Digoxin, *n* (%)	27 (6)	15 (5)	12 (9)	0.096
Antiarrhythmics, *n* (%)	9 (2)	3 (1)	6 (5)	0.016
Metformin, *n* (%)	86 (20)	68 (23)	18 (14)	0.036
SGLT2 inhibitor, *n* (%)	28 (7)	20 (7)	8 (6)	0.847
Insulin, *n* (%)	65 (15)	56 (19)	9 (7)	0.002
Ivabradine, *n* (%)	3 (1)	3 (1)	0 (0)	0.254
Baseline blood values				
HbA1c (%)	8.1 ± 2.8	8.5 ± 2.9	7.1 ± 2.1	<0.001
eGFR (mL/min)	52.1 ± 33.5	46.8 ± 35.2	64.2 ± 25.4	<0.001

ACEi, angiotensin-converting enzyme inhibitor; AF, atrial fibrillation; ARB, angiotensin receptor blocker; AMI, acute myocardial infarction; CABG, coronary artery bypass grafting; CKD, chronic kidney disease; EF, ejection fraction; eGFR, estimated glomerular filtration rate; HbA1c, glycated haemoglobin; HF, heart failure; LAESVi, left atrial end-systolic volume index; LVEDD left ventricular end-diastolic diameter; LVEDVi, left ventricular end-diastolic volume index; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic diameter; LVESVi, left ventricular end-systolic volume indexed; LVSD, left ventricular systolic dysfunction; MRA, mineralocorticoid receptor antagonist; NOAC, novel oral anticoagulant; PCI, percutaneous coronary intervention; RHD, rheumatic heart disease; SGLT2, sodium-glucose cotransporter 2.

Table 2 Age- and sex-specific HF prevalence for Indigenous and non-Indigenous Australians in Central Australia

	All (95% CI)	Indigenous Australians (95% CI)	Non-Indigenous Australians (95% CI)	Prevalence ratio (95% CI)	*P* value
Age					
<45	28 (23–35)	51 (41–63)	4 (1–9)	12.71 (5.55–29.10)	<0.001
45–54	154 (125–188)	325 (259–402)	38 (21–63)	8.60 (4.89–15.13)	<0.001
55–64	207 (169–252)	458 (358–577)	94 (64–132)	4.90 (3.22–7.46)	<0.001
65+	457 (386–538)	610 (471–776)	380 (300–473)	1.61 (1.16–2.23)	0.004
All (crude prevalence)	93 (85–102)	138 (123–155)	53 (44–63)	2.62 (2.13–3.22)	<0.001
Males					
<45	25 (17–34)	42 (29–58)	5 (1–14)	7.83 (2.78–22.02)	<0.001
45–54	187 (142–242)	370 (270–494)	70 (37–120)	5.28 (2.85–9.76)	<0.001
55–64	278 (216–353)	681 (499–904)	127 (80–192)	5.35 (3.23–8.85)	<0.001
65+	615 (495–753)	897 (625–1236)	516 (390–668)	1.74 (1.15–2.64)	0.009
All (crude prevalence)	110 (96–124)	147 (125–172)	77 (62–94)	1.92 (1.49–2.48)	<0.001
Females					
<45	32 (24–43)	60 (45–80)	3 (0.3–10)	22.66 (5.51–93.19)	<0.001
45–54	123 (87–167)	285 (203–389)	5 (0.1–30)	52.74 (7.25–383.63)	<0.0001
55–64	139 (96–194)	291 (189–427)	57 (26–107)	5.14 (2.41–10.95)	<0.001
65+	318 (238–416)	448 (302–636)	231 (145–347)	1.94 (1.13–3.35)	0.015
All (crude prevalence)	77 (66–89)	130 (109–153)	29 (20–40)	4.54 (3.12–6.60)	<0.001

CI, 123–155) per 10 000 Indigenous Australians and 53 (95% CI, 44–63) per 10 000 non-Indigenous Australians (ratio, 2.62 [95% CI, 2.13–3.22] $P < 0.001$). Although present among all categories, this differential varied considerably according to both age and sex. HF prevalence was particularly greater in Indigenous compared with non-Indigenous individuals at younger ages, with prevalence ratios decreasing with age (Table 2 and Fig. 1). There also appeared to be sex-specific differences, with Indigenous to non-Indigenous prevalence ratios significantly greater in young females compared with males. In Indigenous and non-Indigenous Australians younger than 45 years, the age-specific HF prevalence for males was 42 (95% CI, 29–58) and 5 (95% CI, 1–14) per 10 000 (ratio, 7.83 [95% CI, 2.78–22.02]; $P < 0.001$) and for females was 60 (95% CI, 45–80) and 3 (95% CI, 0.3–10) per 10 000 respectively (ratio 22.66 [95% CI, 5.51–93.19]; $P < 0.001$). In contrast, for Indigenous and non-Indigenous Australians 65 years and older, the age-specific HF prevalence for males was 897 (95% CI, 625–1236) and 516 (95% CI, 390–668) per 10 000 (ratio, 1.74 [95% CI, 1.15–2.64], $P = 0.009$) and for females was 448 (95% CI, 302–636) and 231 (95% CI, 145–347) per 10 000 respectively (ratio, 1.94 [95% CI, 1.13–3.35], $P = 0.015$).

Comorbidity profile

Compared with the non-Indigenous group, Indigenous Australians had higher rates of RHD (18% vs 1%, $P < 0.001$), diabetes (63% vs 33%, $P < 0.001$) and severe CKD with estimated glomerular filtration rate (eGFR) < 30 mL per 1.73 m^2 (32% vs 7%, $P < 0.001$), but lower rates of atrial fibrillation (AF) (20% vs 34%, $P = 0.002$), stroke (7% vs 16%, $P = 0.004$) and obesity (body mass index 28.7 ± 6.5 vs 30.2 ± 7.3, $P = 0.038$) (Table 1). Indigenous Australians also had significantly higher glycated haemoglobin (HbA1c) (8.5 ± 2.9% vs 7.1 ± 2.1%, $P < 0.001$) and lower eGFR (46.8 ± 35.2 vs 64.2 ± 25.4 mL/1.73 m^2, $P < 0.001$), in keeping with comorbid rates of diabetes and severe CKD described above. There were no significant differences in baseline rates of acute myocardial infarction (AMI), revascularisation with percutaneous coronary intervention (PCI) or coronary bypass grafting (CABG), hypertension (HTN) or heavy use of alcohol between groups.

Aetiology

HF aetiology was similar in Indigenous and non-Indigenous Australians and most commonly was ischaemic (39% vs 36%, $P = 0.53$), dilated idiopathic (13% vs 19%, $P = 0.09$), valvular (13% vs 9%, $P = 0.24$) or of unknown aetiology (19% vs 16%, $P = 0.54$).

Echocardiography

Non-Indigenous individuals had larger left ventricular end-diastolic diameter (55.0 ± 8.0 vs 51.7 ± 8.0,

$P < 0.001$) and left ventricular end-diastolic volume index (LVEDVi) (67.2 ± 23.5 vs 60.5 ± 19.8, $P = 0.005$) (Table 1) compared with Indigenous individuals. However, there was no difference in mean initial LVEF (38.3 ± 8.4% vs 39.1 ± 9.2%, $P = 0.44$). Two hundred forty (57%) patients had serial imaging performed over a mean interval period of 0.9 ± 0.5 years. There was no significant difference in LVEF change among non-Indigenous and Indigenous Australians (5.1% ± 11.7% vs 4.9% ± 12.1%, $P = 0.90$).

Prescription of GDMT and other medications

GDMT was suboptimal in both Indigenous and non-Indigenous groups with similar rates of prescription of angiotensin-converting enzyme inhibitors (ACEis) and angiotensin receptor blockers (64% vs 67%, $P = 0.47$), β-blockers (68% vs 71%, $P = 0.47$), mineralocorticoid receptor antagonists (30% vs 34%, $P = 0.34$) and angiotensin receptor neprilysin inhibitors (2% vs 4%, $P = 0.17$).

Major adverse cardiac events

During a mean follow-up of 2.2 ± 0.5 years, Indigenous Australians experienced a higher rate of major adverse cardiovascular events (MACEs) (54% vs 28%, $P < 0.001$) and all-cause death (24% vs 13%, $P = 0.013$).

As shown in Figure 2, the Indigenous group had significantly lower MACE-free survival ($P < 0.001$) and overall survival ($P = 0.027$) compared with the non-Indigenous group.

Prediction of mortality

The univariate and multivariate predictors of MACE and all-cause death for those with HF are reported in Table 3 and Table 4 respectively. Increasing age and Indigenous ethnicity were predictive of MACE and all-cause death in both univariate and multivariate analyses, whereas ACEi use was protective. History of AMI was predictive of MACE only. Although diabetes and CKD were predictive of outcomes in univariate analyses, there was no significant association found in multivariate models for MACE and all-cause death.

Discussion

In this study, we sought to characterise the contemporary prevalence, comorbid associations, aetiology, echocardiographic parameters, management trends and clinical outcomes associated with HF and LVSD among Indigenous and non-Indigenous Australians in Central Australia. To our knowledge, this is the largest study to examine these factors in this region to date.

The major findings in our study are as follows:

1 Indigenous Australians had a higher prevalence of HF compared with non-Indigenous Australians across all age brackets and in both sexes.
2 The burden of HF was particularly greater among young Indigenous Australians and females compared with their non-Indigenous counterparts.
3 Comorbid rates of diabetes, severe CKD and RHD were significantly higher in Indigenous Australians.
4 Non-indigenous Australians had larger left ventricular cavity size but otherwise there was no significant difference in LVEF or HF aetiology.
5 Rates of GDMT were low in both groups.

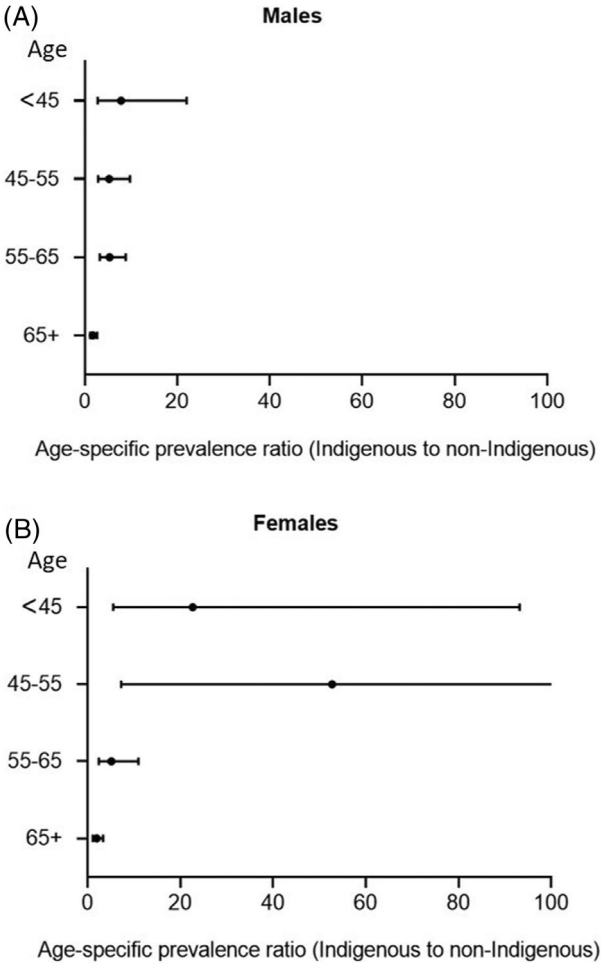

Figure 1 Indigenous to non-Indigenous Australian age-specific prevalence ratios for heart failure in Central Australia for: (A) males and (B) females.

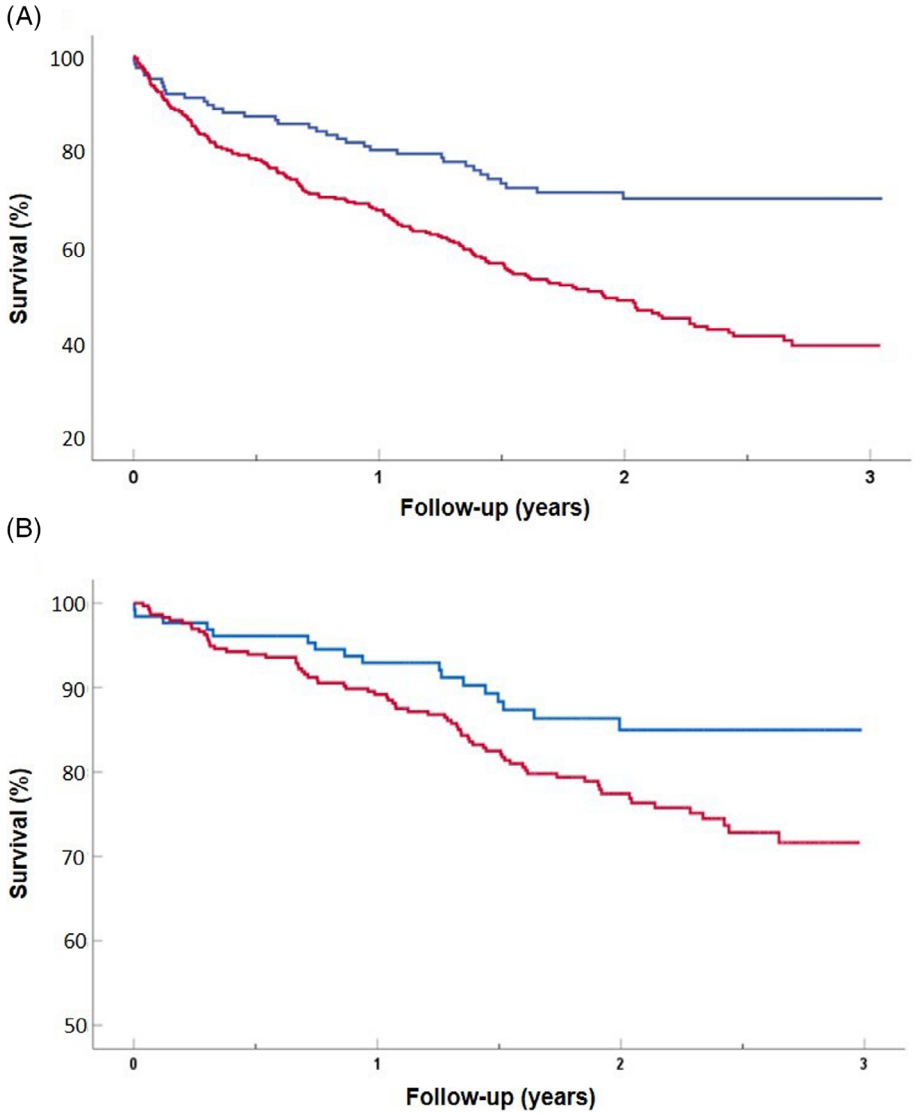

Figure 2 Kaplan–Meier curves showing (A) survival free from major adverse cardiac events ($P < 0.001$) and (B) overall survival ($P = 0.027$). (■) Indigenous; (■) Non-Indigenous.

6 Indigenous Australians with HF had lower overall survival and lower MACE-free survival compared with non-Indigenous Australians.
7 Even after accounting for their greater rate of comorbidities, Indigenous ethnicity remained a strong, independent predictor of mortality in patients with HF in multivariate models.

Over a decade ago, McGrady *et al.* performed the first comprehensive Indigenous HF study in the region examining prevalence, risk factors and echocardiographic parameters.[3] The observed HF prevalence was 5% and significantly higher than previously reported estimates of 1–2%.[1,2] However, this study used an 'all-comers' approach and, therefore, absolute HF case numbers were low. Rates of cardiovascular and HF risk factors, including diabetes, HTN, severe CKD and RHD, were high in our study and reflect similar studies around Australia.[11,13]

Ischaemic aetiology of HF with LVSD is the most common worldwide,[14] and this is reflected in our study. Interestingly, despite the higher burden of HF seen in younger Indigenous Australians, the rates of AMI and revascularisation with PCI or CABG were similar to non-Indigenous individuals in our study. This highlights the disparity in access to advanced cardiac imaging techniques and in-hospital interventions, including invasive coronary angiography.[15] There was also no significant difference in valvular HF in Indigenous compared with non-Indigenous Australians, despite a higher rate of RHD. Underdiagnosis and underreporting of acute rheumatic fever and RHD to state and nationwide registers

Table 3 Univariate and multivariate predictors of MACE

	Univariate HR (95% CI)	*P* value	Multivariate HR (95% CI)	*P* value
Demographics				
Age	1.011 (1.002–1.020)	0.015*	1.017 (1.007–1.028)	0.001*
Indigenous Australian	2.231 (1.554–3.203)	<0.001*	2.642 (1.742–4.008)	<0.001*
Male sex	0.859 (0.648–1.139)	0.291		
Echo				
LVEF	0.989 (0.973–1.004)	0.143		
Comorbidities				
AMI	2.252 (1.701–2.983)	<0.001*	1.820 (1.346–2.462)	<0.001*
AF	1.255 (0.921–1.710)	0.151		
RHD	0.980 (0.649–1.481)	0.925		
HTN	1.147 (0.751–1.753)	0.525		
Diabetes	1.518 (1.138–2.025)	0.005*	1.006 (0.724–1.397)	0.974
CKD	1.536 (1.138–2.074)	0.005*	0.965 (0.687–1.356)	0.838
Medications				
ACEi	0.668 (0.503–0.886)	0.005*	0.699 (0.520–0.940)	0.018*

ACEi, angiotensin-converting enzyme inhibitor; AF, atrial fibrillation; AMI, acute myocardial infarction; CI, confidence interval; CKD, chronic kidney disease; HR, hazard ratio; HTN, hypertension; LVEF, left ventricular ejection fraction; MACE, major adverse cardiac events; RHD, rheumatic heart disease.
*denotes statistical significance $p < 0.05$.

Table 4 Univariate and multivariate predictors of all-cause death

	Univariate HR (95% CI)	*P* value	Multivariate HR (95% CI)	*P* value
Demographics				
Age	1.038 (1.024–1.053)	<0.001*	1.049 (1.031–1.067)	<0.001*
Indigenous Australian	1.800 (1.060–3.056)	0.029*	2.586 (1.402–4.772)	0.002*
Male sex	0.838 (0.550–1.276)	0.409		
Echo				
LVEF	0.995 (0.973–1.017)	0.638		
Comorbidities				
AMI	1.753 (1.153–2.665)	0.009*	0.935 (0.532–1.642)	0.814
AF	1.460 (0.932–2.286)	0.099	1.143 (0.715–1.826)	0.576
RHD	0.638 (0.309–1.320)	0.226		
HTN	1.147 (0.751–1.753)	0.525		
Diabetes	2.405 (1.504–3.845)	<0.001*	1.507 (0.883–2.572)	0.133
CKD	2.637 (1.729–4.024)	<0.001*	1.611 (0.989–2.623)	0.055
Medications				
ACEi	0.340 (0.222–0.519)	<0.001*	0.432 (0.277–0.673)	<0.001*

ACEi, angiotensin-converting enzyme inhibitor; AF, atrial fibrillation; AMI, acute myocardial infarction; CI, confidence interval; CKD, chronic kidney disease; HR, hazard ratio; HTN, hypertension; LVEF, left ventricular ejection fraction; RHD, rheumatic heart disease.
*denotes statistical significance $p < 0.05$.

means many patients are identified very late and usually with decompensated HF.[16] AF as a cause or complication of HF was also likely underrecognised in our cohort when compared with other studies in the region.[17] Although not examined in our study, diabetic cardiomyopathy and type 4 cardiorenal syndrome warrant further research as potential HF aetiology, given the high prevalence of diabetes and CKD in First Nations peoples respectively.[18,19]

Echocardiographic data pertaining to HF in Indigenous Australians is limited; therefore, we mandated echocardiography-proven LVSD as a prerequisite for inclusion in our study. Our mean LVEF of 38.9% was similar to a Hunter New England region study[10]; however, echocardiographic data were only available for half of the cohort in that study. McGrady *et al*. reported LVSD in 6% of their cohort; however, specific LVEF figures were not reported.[3] Interestingly, in our study, non-Indigenous patients had larger LVEDVi (67.2 ± 23.5 vs 60.5 ± 19.8, $P = 0.005$) compared with Indigenous individuals, which may suggest an ethnic difference that has not been previously reported to our knowledge.

Previous studies have shown lower prescription rates of HF medications in Indigenous patients compared with non-Indigenous patients.[7,8,20] Although we found no significant difference between both groups, prescription

of HF medical therapy in general was well below expected rates set out in HF guidelines[4] and highlights the disadvantage experienced by all individuals residing in remote communities regardless of ethnicity. It has been well established that patients in rural and remote settings experience disproportionately high rates of cardiovascular disease, hospitalisation and death.[21] In the context of Central Australia, this is further exacerbated by specific factors, including Indigenous belief paradigms in illness that preference traditional understandings of cause and treatment, a hot desert climate and poor housing standards complicating the storage of food and safe use of HF medicines such as diuretics.[22,23]

Studies have shown that Indigenous Australians have disproportionately worse cardiovascular outcomes than their non-Indigenous counterparts.[24,25] Rates of MACE and all-cause death observed in our study were high and significantly worse for Indigenous compared with non-Indigenous individuals. Multivariate analysis suggested that age, Indigenous ethnicity and history of AMI were responsible for the outcome disparities. Although comorbidities, such as diabetes and severe CKD, were predictive of mortality on univariate analysis, this did persist in the multivariate analysis model.

Finally, there was a significantly lower rate of MACE-free and overall survival in Indigenous Australians in the present study, despite the younger age of the cohort and similar LV function, HF aetiology and management trends compared with non-Indigenous Australians. This finding is emblematic of the stark inequality of health and life expectancy experienced by First Nations peoples, termed the 'Indigenous Gap'.[26] The 'gap' with respect to HF is partially explained by higher prevalence of comorbidities and risk factors; however, there is also a complex interplay of Indigenous ethnicity and underlying sociocultural, language, financial and geographic barriers to health care that need to be addressed to allow meaningful change for the future.[13]

Limitations

The major limitation of our study is the single-centre and retrospective nature of our analysis, which limits the generalisability of our results. Given we focused on patients with a diagnosis of HF and LVSD, we did not account for patients with HF with preserved ejection fraction or those with asymptomatic LVSD. Larger prospective studies are needed to further elucidate the heavy burden of HF in First Nations peoples and those living in remote communities.

Conclusion

HF is more than two times as prevalent among remote Indigenous Australians, particularly among younger individuals and females. Despite similar HF aetiologies and GDMT, MACE and mortality outcomes are higher in Indigenous Australians with HF. These data have implications for efforts to close the First Nations gap in morbidity and mortality.

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