Global burden of cardiovascular disease

Heart failure, ventricular dysfunction and risk factor prevalence in Australian Aboriginal peoples: the Heart of the Heart Study

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ABSTRACT

Background Limited strategies have been developed to evaluate and address the alarming discrepancy in early mortality between Indigenous and non-Indigenous populations.

Objective To assess heart failure (HF), HF risk factors and document cardiac characteristics in an Australian Aboriginal population.

Design, setting, participants Adults were enrolled across six Aboriginal communities in Central Australia. They undertook comprehensive cardiovascular assessments, including echocardiography, to determine HF status, asymptomatic ventricular dysfunction and underlying risk factor profile.

Results Of 436 participants (mean age 44 ± 14 years; 64% women) enrolled, 5.3% (95% CI 3.2% to 7.5%) were diagnosed with HF, only 35% of whom had a pre-existing HF diagnosis. Asymptomatic left ventricular dysfunction (ALVD) was seen in 13% (95% CI 9.4% to 15.7%) of the population. Estimates of HF risk factor prevalence were as follows: body mass index (BMI) ≥ 30 kg/m^2 42%, hypertension 41%, diabetes mellitus 40%, coronary artery disease (CAD) 7% and history of acute rheumatic fever or rheumatic heart disease 7%. In logistic regression analysis (after adjustment for age and gender), HF was associated with CAD (OR=9.6, p<0.001), diabetes (OR=5.4, p=0.002), hypertension (OR=4.8, p=0.006), BMI ≥ 30 kg/m^2 (OR=2.9, p=0.02), acute rheumatic fever or rheumatic heart disease (OR=5.6, p=0.001) and B-type natriuretic peptide (OR=1.02, p<0.001).

Conclusion The burden of HF, ALVD and risk factors in this population was extremely high. This study highlights potentially modifiable targets on which to focus resources and screening strategies to prevent HF in this high-risk Indigenous population.

INTRODUCTION

The world is home to over 370 million Indigenous peoples[1 2] who, despite recent improvements in health from declining infectious disease rates, have increased mortality from lifestyle factors and non-communicable disease.[3] Poorer health and reduced life expectancies remain the norm.[1]

Australian Aboriginal peoples have the poorest life expectancy of any Indigenous peoples living in high-income countries.[3] They have a life expectancy that is over a decade less than that of the general Australian population.[4] Recent data suggest that 80% of this gap in life expectancy in Australia's Indigenous population is attributable to non-communicable disease, almost half of which is due to cardiovascular disease (CVD) and diabetes.[5]

Heart failure (HF), a common outcome of CVD, has a poor prognosis and its prevalence is increasing. Based on risk factor profiles,[4 6] HF prevalence in Indigenous populations is likely to be high,[4 7 8] but few data are available. Once a HF diagnosis has been made, mortality rates are higher in Indigenous peoples than in non-Indigenous populations, particularly in New Zealand Maori, American Indian and Australian Aboriginal populations.[4 7 8]

In many populations around the globe the epidemiology of HF risk factors is changing. For example, diabetes and obesity are both strong risk factors and are reaching epidemic proportions.[9] Australian Aboriginal peoples have diabetes rates 3—10 times, and obesity rates twice, those of the non-Indigenous population.[4 6] Indigenous Australians also have among the highest rates of acute rheumatic fever (ARF) and rheumatic heart disease (RHD) in the world and are 20 times more likely to die from one of these conditions.[4] However, in this group, little is known about the burden of HF or its antecedents.

The Heart of the Heart Study sought to undertake a comprehensive assessment of CVD and risk factors from a representative cross-section of Aboriginal Australian adults living in Central Australia. This first phase of the Heart of the Heart Study has two main objectives: (1) to document HF prevalence and left ventricular characteristics; and (2) to identify their association with key HF risk factors.

METHODS

Heart of the Heart Study population and setting

Participants were Aboriginal adults (>18 years) residing in one of the participating communities. Enrolment took place between May 2008 and November 2009. Six communities participated, including Alice Springs town (estimated Aboriginal population 5000), Town Camps (18 small communities of family members or members of the same language group on the outskirts of Alice Springs; estimated population 2000[10]) and four remote communities (three centralised communities and one decentralised community, approximately 10 km, 100 km, 300 km and 400 km from Alice Springs and with estimated Aboriginal

populations of 100, 560, 240 and 770 individuals, respectively). Before the study, consultation took place with communities and Aboriginal medical services.

The Aboriginal population is dynamic, travelling and often without a single primary residence; there are no records that reflect the current resident population. Thus in order to obtain a representative sample, recruitment was tailored to each location. In Town Camps and the three centralised remote communities, the study team visited each dwelling to discuss the study and invite participation. Where possible, a community member or Aboriginal health worker used the local Indigenous language. The fourth remote community was decentralised and comprised 16 outstations (approximately five houses at each) dispersed across $10\,000\,km^2$. In this community, meetings were held at each outstation to discuss the study. Volunteers who wished to participate put their name forward to the health clinic staff who visited each outstation weekly. In Alice Springs town (as opposed to Town Camps) the study was advertised on local radio, in newspapers and through mailings.

Assessment

After providing informed consent in the nominated language, participants undertook a researcher-administered structured questionnaire, to collect information on medical history, drugs, smoking, alcohol intake and HF symptoms. Medical records were reviewed. Anthropomorphic measures were taken according to WHO methods[11] (waist, height (Seca stadiometer 213, Germany) and weight (Tanita: Wedderburn scales, Japan)). Body mass index (BMI) was calculated as weight $(kg)/height^2$ (m^2). Blood pressure was taken after the participant had been seated for 5 min (digital blood pressure monitor; IntelliSense: Omron HEM-907, Japan). Clinical assessment included cardiac and lung auscultation as well as fluid status assessment by a study cardiologist (MM).

Venous blood was collected for assessment of renal function, random non-fasting blood glucose level, glycosylated haemoglobin (HbA1c) and B-type natriuretic peptide (BNP). BNP was measured, from EDTA plasma, using a point of care Biosite Triage meter (Inverness Medical, San Diego, USA). All other bloods were analysed according to one of two accredited commercial pathology providers.

Echocardiography

Echocardiography was undertaken to a standard protocol using a GE Vingmed Vivid I portable ultrasound machine (GE Medical Systems, Milwaukee, Wisconsin, USA). Scans were obtained by a single cardiologist (MM), digitally recorded and measurements and grading made offline by a single cardiologist (MM) blinded to the clinical details using GE EchoPAC software version 6.0 (GE Medical Systems). Measurements were taken according to the American Society of Echocardiography,[12] and were the average of three cardiac cycles (five if an arrhythmia was

present). Left ventricular function was quantified using Simpson's rule or visual estimation, if the endocardium was not well defined. Ten per cent of studies were randomly selected and reported independently at a core laboratory blinded to all other data.

Heart failure, ventricular and valvular dysfunction

HF diagnosis was independently adjudicated by two doctors (for participants with ventricular dysfunction on echocardiography) according to HF guidelines.[13] Where there was disagreement (<10% of cases), a third doctor reviewer adjudicated. Left ventricular dysfunction threshold was defined as greater than two standard deviations below the mean of a healthy reference sample. Mild dysfunction was defined as ejection fraction 40—50% and moderate to severe <40%. Diastolic function was assessed using Doppler evaluation of mitral valve and pulmonary vein inflow and tissue Doppler evaluation of the lateral mitral annulus[14] and graded into four categories (table 1).[15 16] Valvular heart disease was defined as moderate mitral or aortic stenosis or regurgitation or RHD.[17—19]

Statistical methods

Data analysis was undertaken using Stata SE V.11.0. Variables not normally distributed were expressed as medians and interquartile ranges. Normally distributed variables were expressed as means and standard deviations. Univariate and multivariate logistic regression analysis was used to investigate the relationship between risk factors and ventricular function. Interobserver reproducibility was summarised with concordance correlation coefficients.[20]

RESULTS

Demographics and heart failure risk factors

Four hundred and thirty six participants enrolled in the study. Participants had a mean age of 44 years (18—80 years), 8% were over the age of 65 years and 64% were female (table 2). The prevalence of key HF risk factors was as follows (table 2): BMI $\geq 30\,kg/m^2$ 42%, hypertension 41%, diabetes mellitus 40%, coronary artery disease (CAD) 7%, history of ARF or RHD 7% and atrial fibrillation 2.5%. Older age was associated with increased prevalence of CAD, diabetes, hypertension and atrial fibrillation, but not with obesity, RHD or ARF. Smoking was, however, less common in the older age groups.

On average, women had higher BMI than men (mean BMI $30.9\pm 7.1\,kg/m^2$ vs $28.3\pm 6.1\,kg/m^2$; p<0.001) and were more likely to be obese (50.2% vs 27.7%; p<0.001). Other HF risk factors, however, including diabetes and hypertension, were not associated with female gender (table 3). Two hundred and forty-nine (57%) participants lived in an urban environment (Alice Springs or within a 10 km radius) and 187 (43%) resided in remote communities. Participants living in an urban compared with a remote location (table 3) were older (44.9 ± 14.4 years vs

Table 1 Doppler criteria for the classification of left ventricular diastolic function

Doppler measures	Diastolic function			
	Normal	Mild dysfunction	Moderate dysfunction	Severe dysfunction
Mitral inflow	0.75<E/A<1.5 DT>160 ms	E/A≤0.75	0.75<E/A<1.5 DT>160 ms	E/A>1.5 DT<160 ms
Pulmonary venous inflow	S≥D MV Adur>PV Adur	S≥D MV Adur>PV Adur	S<D MV Adur+30<PV Adur	S<D MV Adur+30<PV Adur
Mitral annular TDI	E/e′<10	E/e′<10	E/e′≥10	E/e′≥10

Minimum of two Doppler criteria required to be diagnosed as moderate or severe diastolic dysfunction.

A, peak mitral filling velocity at atrial contraction; D, peak velocity of the pulmonary venous forward flow during diastole; DT, deceleration time of the mitral E wave; e′, peak velocity of the mitral annulus motion during early diastole; E, peak early mitral inflow velocity; MV Adur, duration of the mitral A wave; PV Adur, duration of the pulmonary venous reversal wave during atrial contraction; S, peak velocity of the pulmonary venous forward flow during systole; TDI, tissue Doppler imaging.

Table 2 Characteristics of the Heart of Heart Study population

| Characteristics | Age in years | | | | |
	All (n=436)	<40 (n=175)	40–55 (n=165)	>55 (n=96)	p Value
Demographics					
Age in years	44 (14)				N/A
Female, n (%)	281 (64)	113 (65)	107 (65)	61 (64)	0.898
Urban dwelling, n (%)	249 (57)	93 (53)	93 (56)	63 (66)	0.065
Remote dwelling, n (%)	187 (43)	82 (47)	72 (44)	33 (34)	0.065
Risk factor profile—n (%)					
CAD	31 (7)	3 (2)	10 (6)	18 (19)	<0.001
Vascular disease	43 (10)	4 (2)	13 (8)	26 (27)	<0.001
Cardiac valve disease	15 (3)	6 (3)	4 (2)	5 (5)	0.558
ARF/RHD	32 (7)	14 (8)	12 (7)	6 (6)	0.503
Diabetes	174 (40)	38 (22)	82 (50)	54 (56)	<0.001
Hypertension	177 (41)	26 (15)	83 (50)	68 (71)	<0.001
Atrial fibrillation	11 (2.5)	2 (1)	1 (0.5)	8 (8)	0.002
Current smoker	166 (38)	93 (53)	52 (32)	21 (22)	<0.001
BMI ≥30 kg/m^2	184 (42)	77 (44)	73 (44)	34 (35)	0.254
Anthropomorphic data and blood pressure					
Waist (cm)	99.0 (16.1)	98.8 (16.9)	101.7 (15.8)	98.1 (14.3)	0.168
Body mass index (kg/m^2)	30 (7)	30 (7)	31 (7)	29 (6)	0.342
Systolic BP (mm Hg)	129 (19)	121 (16)	130 (19)	141 (20)	<0.001
Diastolic BP (mm Hg)	81 (13)	80 (12)	83 (14)	82 (12)	0.061
Biochemical and lipid profile					
Creatinine* (μmol/l)	66 (56–80)	61 (53–76)	67 (57–79)	76 (59–95)	0.013
Glucose* (mmol/l)	5.5 (4.7–7.5)	5.2 (4.4–6.1)	5.8 (4.8–9.3)	6.0 (5.2–8.1)	0.002
HbA1c* (%)	6.1 (5.7–7.4)	5.8 (5.5–6.3)	6.4 (5.9–8.4)	6.4 (5.9–8.1)	<0.001
Total cholesterol (mmol/l)	4.7 (1.2)	4.7 (1.2)	4.8 (1.2)	4.4 (1.0)	0.112
Triglycerides (mmol/l)	2.5 (1.8)	2.2 (1.6)	2.8 (2.2)	2.3 (1.4)	0.221
BNP* (pg/ml)	7.2 (5.0–15.9)	5.1 (5.0–10.8)	6.8 (5.0–12.9)	16.8 (7.5–44.3)	0.001

Data are number (%), mean (SD) if normally distributed, or *median (IQR) if not normally distributed and p value is from ordered logistic regression analyses across age groups.
CAD, coronary artery disease was defined as a history of angina or myocardial infarction or revascularisation procedure documented in medical records; Vascular disease, defined as history of CAD, stroke or peripheral vascular disease documented in the medical records; ARF/RHD, defined as acute rheumatic fever or rheumatic heart disease documented in the medical records; Atrial fibrillation, defined as atrial fibrillation documented in medical records. Diabetes, defined as diabetes documented in medical records or HbA1c>6.5% (on study blood test); Hypertension, defined as hypertension documented in medical records; Obesity—BMI ≥30 in kg/m^2.
BMI, body mass index; BNP, B-type natriuretic peptide; BP, blood pressure; HbA1c, glycosylated haemoglobin; N/A, not applicable.

42.2±13.8 years; p=0.05) and more likely to have a history of CAD (9.2% vs 4.3%; p=0.05). HF or presence of other HF risk factors, however, including diabetes, hypertension and obesity, were not associated with place of dwelling.

Echocardiography
Echocardiography was completed by 430 participants. Ejection fraction was measurable using Simpson's rule in 94% of participants (biplane 88%, single plane 6% and estimated in 6%).

Visual estimation was used more often in participants with BMI ≥30 kg/m^2 (9% vs 3%; p=0.007). Diastolic function could be assessed in 409 participants, was indeterminate on echocardiography in 14 participants and could not be assessed in seven participants owing to rhythm (atrial fibrillation, etc) or valve pathology. A core echocardiography laboratory (TM, BH) reported (while blinded to all other data) 10% of randomly selected echocardiograms for diagnostic reproducibility. Interobserver reproducibility was good, as follows (concordance

Table 3 Association of heart failure and risk factors prevalence with gender and place of dwelling

| Characteristics | Gender | | | Primary place of residence | | |
	Male (n=155)	Female (n=281)	p Value	'Urban' (n=249)	'Remote' (n=187)	p Value
Age in years*	43.7 (14.1)	43.8 (14.3)	0.913	44.9 (14.4)	42.2 (13.8)	0.049
Female	N/A	N/A		152 (61)	129 (69)	0.086
CAD	14 (9)	17 (6)	0.218	23 (9)	8 (4)	0.049
Diabetes	58 (37)	116 (41)	0.431	94 (38)	80 (43)	0.289
Hypertension	71 (46)	106 (38)	0.108	105 (42)	72 (39)	0.547
Obesity	43 (28)	141 (50)	<0.001	100 (40)	84 (45)	0.319
Heart failure	11 (7)	12 (4)	0.198	15 (6)	8 (4)	0.399
ALVD	14 (9)	40 (14)	0.121	30 (12)	24 (13)	0.850
ARF/RHD	8 (5)	24 (9)	0.195	21 (8)	11 (6)	0.312

Data are number (%) unless otherwise indicated.
*Mean (SD).
'Urban', includes participants living in Alice Springs town, Town Camps and the community 10 km from Alice Springs; 'Remote', includes participants from the communities located 11–400 km from Alice Springs town.
ALVD, asymptomatic left ventricular dysfunction; ARF/RHD, history of acute rheumatic fever or rheumatic heart disease; CAD, coronary artery disease; Obesity, body mass index ≥30 kg/m^2.

correlation coefficient[20] (95% CI)): left ventricular ejection fraction 0.89 (0.83 to 0.95); Doppler of the mitral valve inflow, E wave 0.82 (0.71 to 0.92), A wave 0.76 (0.65 to 0.88) and for diastolic function grading κ was 0.87 (SE 0.11).

Left ventricular dysfunction

Left ventricular dysfunction was present in 17% of participants, 6% with systolic dysfunction (left ventricular ejection fraction <50%) and 13% with diastolic dysfunction (seven had both systolic and at least moderate diastolic dysfunction). Of the participants with left ventricular systolic impairment, 42% were asymptomatic and of those with at least moderate diastolic dysfunction 75% were asymptomatic.

Heart failure

Twenty-three (5.3%) of 430 participants had HF, three with acute decompensated HF and 20 with chronic HF, and 65% of these cases were previously undiagnosed. Two of the three participants with decompensated HF required hospital admission; only one had a pre-existing HF diagnosis. Dilated cardiomyopathy with reduced systolic function of unknown cause was present in two cases and the third had HF with preserved ejection fraction. Overall, in those with acute or chronic HF, 61% had impaired left ventricular systolic dysfunction (ejection fraction < 50%) and 39% had HF with preserved ejection fraction.

Diabetes, hypertension, CAD, obesity, ARF or RHD and raised BNP were associated with HF after adjusting for age and gender (table 4). There was no association between alcohol intake or the frequency of drinking alcohol with left ventricular dysfunction or HF (data not presented). Diabetes, hypertension and obesity (individually or in combination) were considered a major contributing factor in almost half (48%) of HF cases. Other conditions associated with HF were CAD in 26% of cases, dilated cardiomyopathy in 9%, RHD in 4% and primary pulmonary hypertension in 4%. For 9% of HF cases there was no clearly identifiable contributory risk factor. Similarly, for those with asymptomatic left ventricular dysfunction (ALVD), three-quarters (79%) were associated with diabetes, hypertension, obesity, or a combination of these factors. Other associations included RHD (8%) and in 13% of cases no contributory risk factor was clearly identified.

Left ventricular dysfunction (systolic and diastolic) was associated with age, gender, CAD, hypertension and increasing BNP after adjusting for age and gender. In addition moderate to severe diastolic dysfunction was also associated with diabetes and obesity after adjusting for age and gender (table 5).

DISCUSSION

To the best of our knowledge, the Heart of the Heart Study is the first comprehensive assessment of the prevalence of HF and associated risk factors in Australian Aboriginal peoples. We observed an HF prevalence of 5%, which is five times higher than the previously reported national HF prevalence for Australia Aboriginal peoples.[4] Moreover, only 1.8% of participants had a prior HF diagnosis. This suggests that there is a large, as yet unidentified, burden of HF in the Australian Aboriginal population. These are important findings, which highlight one of the major contributors to the large disparity in life expectancy between Indigenous and non-Indigenous Australians.[6]

Comparisons of HF and ventricular dysfunction among studies are limited by differences in population characteristics and research methodology, including end-point definitions. We used rigorous HF adjudication criteria and comprehensive echocardiogram measures, with independent specialist and echocardiogram laboratory review and documented good concordance. Despite this we found an HF prevalence similar to those reported in populations with similar risk factor profiles. For example, the Strong Heart Study investigators (American Indian cohort) reported a 3% prevalence of HF.[21] While their finding of 3% HF is less than the 5% we observed, the definition of HF in the Strong Heart Study included a previous HF hospital admission. Therefore, mild cases of HF managed in the community would not have been included and thus the burden of HF in this group is probably underestimated.

In comparison with a number of non-Indigenous cohorts, prevalent HF in this study was higher than in other studies (3.2% in the ECHO study[22] and 1.8% in the Glasgow MONICA study[23]). This may in part be explained by the differences in underlying HF risk factor profiles, which in the main were higher in our study.

One Australian study, The Canberra Heart Study (CHS), reported a higher HF prevalence (6.3%) than we observed.[24] The CHS is a non-Indigenous population and had a more favourable risk factor profile than the Heart of the Heart participants, but they were also considerably older (mean age 69 years vs 44 years). The Heart of the Heart cohort is relatively young and, unfortunately, there are no comparable age-matched cohorts in the non-Indigenous Australian population. Importantly, HF incidence is reported to increase exponentially with age,[25] with rates doubling in the elderly for every decade over 65 years,[25] and thus the burden of HF we observed is very high in this relatively young population. Incident HF also varies between populations. In the CARDIA Study, for instance, HF was more common in African Americans than in Caucasian Americans

Table 4 Association of heart failure with clinical characteristics

Characteristics	Heart failure			Risk factor adjusted for age and sex	
	Present (n=23)	Absent (n=413)	p Value	OR (95% CI)	p Value
Age (years)*	49.5 (12.3)	43.6 (14.2)	0.053	1.0 (1.0 to 1.1)	0.055
Female	12 (52)	266 (64)	0.198	0.57 (0.2 to 1.3)	0.198
CAD	9 (39)	21 (5)	<0.001	9.6 (3.4 to 27.3)	<0.001
Diabetes	18 (78)	155 (38)	<0.001	5.4 (1.9 to 15.7)	0.002
Hypertension	18 (78)	157 (38)	<0.001	4.8 (1.6 to 14.8)	0.006
Obesity	14 (61)	168 (41)	0.064	2.9 (1.2 to 7.1)	0.022
ARF/RHD	6 (26)	26 (6)	0.001	5.6 (2.0 to 15.9)	0.001
BNP pg/ml†	81 (25–325)	7 (3–14)	<0.001	1.02 (1.01 to 1.03)	<0.001

Data are number (%) unless otherwise indicated.
*Mean (SD);
†median (IQR).
ARF/RHD, history of acute rheumatic fever or rheumatic heart disease; BNP, B-type natriuretic peptide; CAD, coronary artery disease; Obesity, body mass index ≥30 kg/m².

Table 5 Association of left ventricular systolic and diastolic dysfunction with clinical characteristics (adjusted for age and sex)

Characteristics	Systolic dysfunction (LVEF<50%)		Diastolic dysfunction (moderate to severe)	
	OR (95% CI)	p Value	OR (95% CI)	p Value
Age	1.06 (1.03 to 1.09)	<0.001	1.04 (1.02 to 1.06)	<0.001
Female gender	0.36 (0.16 to 0.81)	0.01	2.00 (1.03 to 3.89)	0.042
CAD	5.21 (1.94 to 14.0)	0.001	3.14 (1.30 to 7.62)	0.011
Diabetes	1.77 (0.75 to 4.17)	0.19	2.58 (1.39 to 4.78)	0.003
Hypertension	2.87 (1.03 to 7.96)	0.04	3.94 (1.83 to 8.47)	<0.001
Obesity	1.22 (0.51 to 2.91)	0.65	2.25 (1.23 to 4.13)	0.009
ARF/RHD	5.24 (1.77 to 15.50)	0.003	5.28 (2.35 to 11.89)	<0.001
BNP	1.01 (1.01 to 1.02)	<0.001	1.00 (1.000 to 1.004)	0.04

ARF/RHD, history of acute rheumatic fever or rheumatic heart disease; BNP, B-type natriuretic peptide; CAD, coronary artery disease; LVEF, left ventricular ejection fraction; Obesity, body mass index $\geq$30 kg/m^2.

before the age of 50 years,[26] and in the MESA study, incident HF was highest among African Americans and lowest in Chinese Americans.[27] In the MESA study the increased HF risk reflected differences in the prevalence of hypertension, diabetes together with socioeconomic factors.[27] These factors might also contribute to the difference in HF between Australian Indigenous and non-Indigenous populations.

Differentiation between undiagnosed and diagnosed HF is infrequently reported. However, the proportion of undiagnosed HF cases in the Heart of the Heart Study was high (64% of cases) compared with, for example, the CHS in which undiagnosed HF prevalence was approximately 10% of the HF cases.[24] This is an important finding as undiagnosed HF is potentially untreated and this may explain the higher mortality in the Indigenous population. Almost 80% of those with undiagnosed HF had multiple risk factors, including diabetes, obesity and hypertension, which might help to identify this group as high risk in the future and aid early diagnosis. Early diagnosis is the recommendation of HF guidelines.[13] Detection of asymptomatic disease and early HF is particularly important in remote areas because access to end-stage care and treatments such as defibrillators and cardiac resynchronisation therapy are limited by distance to tertiary centres.

Reported left ventricular systolic dysfunction prevalence also varies depending upon the population and their risk factor profile. However, approximately equal numbers of symptomatic and asymptomatic systolic dysfunction are reported in any given population.[15 22 23] Overall, in the Heart of the Heart Study, we observed left ventricular systolic dysfunction in 6% of cases—approximately half were asymptomatic. The Strong Heart Study investigators,[8] examining a relatively younger American Indian cohort with similar risk factor profile, reported a greater proportion of participants with systolic dysfunction (14% mild and 2.7% severe systolic impairment) than in our study. However, when we defined systolic impairment with the same cut-off points as the Strong Heart Study (ejection fraction <54% mild and <40% severe impairment) we observed very similar proportions of systolic impairment.

We also observed a higher burden of HF risk factors than found in nationally reported Australian data (eg, CAD 7% vs 1.2%, diabetes 40% vs 11%[4]). National data are self-reported, however, and other researchers have noted similar rates of disease in Indigenous populations. For example, Peiris et al observed 9% CAD in 1200 Indigenous primary care patients,[28] and 5% CAD was reported in the Strong Heart Study.[21] Likewise, similar high diabetes rates were reported by O'Dea et al (52.4% in an older Indigenous population[29]) and Devereux et al (51% in an American Indian population[21]).

Our study highlights the high burden of HF and HF risk factors in the Indigenous population of Central Australia and also potential targets to prevent future HF. Diabetes, hypertension and obesity were the most common antecedent risk factors in both HF and ALVD and often all three conditions coexisted. Diabetes and obesity rates, in particular, were extremely high. Previous work has described cardiac structural and functional changes within a couple of years of onset of diabetes or obesity in adolescents.[30] Hypertension and obesity, particularly early onset (<35 years), have also been identified as important antecedents for incident HF in young African Americans.[26] These modifiable antecedents provide potential targets to develop interventions and screening strategies to prevent HF in populations at high risk.

Limitations

Records do not reflect the current population in any community. It is not therefore possible to determine accurately a target population at one point in time. We used the best local information available, advertised the study broadly and visited individual dwellings in communities and Town Camps. Traditional mailing to target populations is not possible or effective. To overcome this, we advertised the study widely through local Indigenous radio, newspaper and mail. We estimated participation at 10% of the population, less for men, but it is not possible to determine how representative of the broader population this sample is and hence how generalisable the findings are. Nevertheless, the age profile of the population closely reflects that of the overall Indigenous population in Central Australia. Likewise, as noted earlier, our results for the prevalence of CAD and diabetes are similar to findings of other studies in Indigenous Australian communities where medical record review was used for the whole community.[28 29] Other comorbidities may have contributed to the development of HF (eg, sleep apnoea) but these are difficult to assess in Central Australia and are not included in this work. Rheumatic heart disease was present in a single HF case and thus we are limited in the ability to assess the role of RHD in HF. Notwithstanding these difficulties, this is the most comprehensive cardiovascular assessment conducted in this population to date.

CONCLUSION

Heart failure and ALVD were common in this relatively young Indigenous population and closely associated with 'traditional' risk factors for HF. The large proportion of subjects with previously undiagnosed and untreated HF may be contributing to the excess HF mortality experienced by this group. These findings support the need to target resources for the prevention

and early detection of underlying risk factors, asymptomatic ventricular dysfunction and HF in high-risk populations.

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