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ORIGINAL ARTICLE

A Comparison of Clinical Characteristics and Outcomes Between Indigenous and Non-Indigenous Patients Presenting to Townsville Hospital Emergency Department With Chest Pain

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Background Indigenous Australians have a high rate of ischaemic heart disease (IHD). There is a paucity of local data for North Queensland regarding the clinical characteristics of Indigenous people who present to the emergency department (ED) with chest pain. The aim of the study is to compare the cardiovascular risk factors, social characteristics, and the clinical outcomes between Indigenous and non-Indigenous patients who presented with cardiac-related chest pain.

Methods This is a retrospective single-centre audit. The data was collected through chart reviews of chest pain presentations to the Townsville University Hospital Emergency Department, Queensland, Australia, from January to December 2017. We categorised the patients into Indigenous and non-Indigenous groups and compared their cardiac risk factors and social characteristics. We further classified the patients into three diagnosis groups and we measured the clinical outcomes in the patients with a diagnosis of cardiac-related chest pain. We used a data linkage to the Registry of Births, Deaths and Marriages for the death outcomes. A multivariable analysis was done to determine the risk of major adverse cardiac event (MACE) for Indigenous vs non-Indigenous patients.

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Results Indigenous patients were over-represented making up 19.1% of the total cohort (compared with 11.1% of the North Queensland Indigenous population) and presented at a younger age (median age: 45 vs 52, p<0.005). Traditional cardiovascular risk factors were significantly higher in Indigenous patients. The incidence of discharge against medical advice was also higher (6.5% vs 2.7%, p<0.005). There was an underutilisation of the local chest pain pathway amongst the Indigenous group (35.8% vs 44.7%, p<0.005). In patients with a diagnosis of cardiac-related chest pain, the rate of receiving invasive coronary angiogram procedures was similar in both cohorts (44.5% vs 43.7%, p=0.836).
With regards to outcomes, Indigenous patients suffered from acute coronary syndrome (ACS) at a younger median age (51 vs 64, p<0.005) and were more likely to have severe three vessel disease (17% vs 6%, p<0.005) leading to coronary bypass graft surgery (CABG) (19% vs 6%, p<0.005). When adjusted for age, gender, and comorbidities, Indigenous patients were more likely to have MACE within 1 year of their chest pain presentation, compared with non-Indigenous patients with the same diagnosis (adjusted odds ration [AOR]=2.0, 95% CI [1.1, 3.8], p=0.03).

Conclusion In our study, Indigenous patients carried a heavier burden of cardiovascular risk factors, presented at a younger age, with more severe coronary disease and had a higher rate of CABG. We found an under-utilisation of the local chest pain protocol amongst the Indigenous cohort, which suggests a need to improve support structures in the ED. In our multivariable analysis, Indigenous patients suffered from a significantly higher MACE compared to non-Indigenous patients which indicates that more collaborative efforts are needed to improve the cardiovascular health of local Aboriginal and Torres Strait Islander people.

Keywords Indigenous • Chest pain • Ischaemic heart disease • Closing the gap • Cardiovascular risk factors

Introduction

Circulatory diseases such as ischaemic heart disease contributed to 23% of Aboriginal and Torres Strait Islander mortality in 2019 [1]. Ischaemic heart disease (IHD) accounts for 11.7 % of all deaths amongst this group, making it the leading cause of death [1].

In 2019, ischaemic heart disease accounted for 405 deaths amongst Aboriginal and Torres Strait Islander people, which translated to a death rate of 113.1. There were 12,182 deaths attributed to ischaemic heart disease amongst non-Indigenous people, which translated to a death rate of 114.322. The death rate amongst Indigenous people that was attributable to ischaemic heart disease was approximately twice that of their non-Indigenous counterparts [2]. Indigenous patients also develop the disease at a younger median age, suffer higher rates of recurrent myocardial infarction and therefore suffer a greater degree of morbidity and mortality [3–8].

A recent meta-analysis revealed that the disproportionate burden of ischaemic heart disease in the Indigenous Australian population is due to a complex interplay of multiple factors. These range from psychosocial disadvantage, a heavier burden of risk factors for coronary artery disease, remoteness and inequitable access to specialist services [9].

Aboriginal and Torres Strait Islanders account for 11.1% of the total population of North Queensland, which is 38.1% of the total Indigenous population in Queensland. The Townsville University Hospital is the tertiary referral centre for North Queensland, treating a high volume of Indigenous patients [10].

Chest pain is one of the most common presenting complaints to emergency departments (ED) across Australia [11–13]. A single centre prospective study showed that 11.1% of 926 patients who presented with cardiac sounding chest pain were diagnosed with acute coronary syndrome (ACS) which was further categorised into unstable angina (UA), non–ST-elevation myocardial infarction (NSTEMI) and ST-elevation MI (STEMI) [13–15]. Many hospitals in Australia have developed tools and pathways to assess patients presenting with chest pain to ED to facilitate diagnosing acute coronary syndrome. The Townsville University Hospital uses the Accelerated Chest Pain Risk Evaluation Protocol (ACRE) [16].

Aims

The aim of this study is to compare the cardiovascular risk profiles, social indicators and clinical outcomes of both Indigenous and non-Indigenous patients who presented to the emergency department with chest pain.

Methods

This study is a retrospective single-centre audit. Data was collected from patients 18 years and older (both Indigenous and non-Indigenous) who presented to the emergency department at the Townsville University Hospital with the

initial complaint of chest pain. The study period for the first ED visit was from January 2017 to December 2017. The outcomes and follow-up data for these presentations were taken 1 year after the first visit. We also collected data for recurrent presentations with chest pain which was defined as re-presentations of five times or more within the study period.

The data was reviewed through hospital records and electronic charts in consultation with the Indigenous Health Service Group at the Townsville Hospital. Basic demographics such as age, gender, postcode and living status were collected. We categorised the living status into a regular home, homeless, nursing home and other living arrangements. Postcodes of the patients in Group 1 were analysed with the Modified Monash Model as a reference to assess remoteness from our regional centre (Supplementary Table 1). Data for social and traditional cardiac risk factors such as smoking and alcohol status, hypertension, diabetes, chronic kidney disease, hypercholesterolaemia, previous ischaemic heart disease, family history of ischaemic heart disease and stroke were extracted from medical records and clinical notes. We followed the NHMRC classification for alcohol excess which is more than 10 standard drinks per week and more than 4 standard drinks on any one day [17]. The alcohol status was self-reported. The medical diagnoses such as hypertension and diabetes were a mixture of self-reporting and clinical documentation.

Patients were grouped according to discharge and final diagnoses. The diagnosis groups were classified as 1) Acute coronary syndromes and/or cardiac chest pain, 2) Non-cardiac chest pain, and 3) Chest pain from other cardiovascular conditions. Group 1 includes diagnoses of Unstable Angina, STEMI, NSTEMI, chest pain for investigation, cardiac chest pain, and cardiac arrest. Group 2 comprises diagnoses other than cardiac causes such as pulmonary embolism, pneumonia, atelectasis, pleurisy, musculoskeletal and chest wall pain, epigastric pain, cholecystitis, gall stone, pancreatitis, gastritis, gastro-oesophageal reflux and undifferentiated chest pain (full list of diagnoses in Supplementary Table 2). Group 3 includes pericarditis, atrial fibrillation, atrial flutter, arrhythmia, valvular heart diseases, heart failure, acute pulmonary oedema, pericardial effusion, and infective endocarditis.

Postcodes of the patients in Group 1 were analysed with the Modified Monash Model as a reference (Supplementary Table 1) [17]. These classifications are based on the Australian Statistical Geography Standard – Remoteness Area (ASGS-RA). The model measures remoteness and population size on a scale of Monash Model categories MM 1 to MM 7. MM1 is a major city. MM2 are areas categorised ASGS-RA 2 and ASGS-RA 3 that are in, or within 20 km road distance of a town with a population greater than 50,000 (Townsville city and its surrounding regions are classed as MM2). MM3 are areas categorised ASGS-RA 2 and ASGS-RA 3 that are not in MM2 and are in or within 15 km road distance of a town with a population between 15,000 and 50,000. MM4 are areas categorised ASGS-RA 2 and ASGS-RA 3 that are not in MM2 or MM3 that are within 10 km road distance of a town with a population between 5,000 and 15,000. MM5 are all other areas in ASGS-RA 2 and 3. MM6 are all areas categorised as ASGS-RA 4 that are not on a populated island that is separated from the mainland by more than more than 5 km offshore or islands that have an MM5 classification with a population of less than 1,000. MM7 are all other areas that are ASGS-RA 5 and on a populated island that is separated from the mainland by more than 5 km.

The outcomes of the Group 1 in terms of treatment such as percutaneous coronary intervention, coronary bypass graft surgery, conservative/medical management, deaths and discharge against medical advice were assessed. Details such as results of the invasive coronary angiogram, echocardiogram, and non-invasive cardiac investigations were also collected.

Major adverse cardiac events (MACE) were defined as cardiovascular deaths, non-fatal MI, non-fatal stroke and admission for heart failure. It was calculated at 12 months after the initial presentation. We carried out a data linkage to the Registry of Births, Deaths and Marriages to have a more robust assessment of deaths. We requested the assistance of the Statistical Services Branch (SSB) at Queensland Health for the data linkage. The probabilistic linkage was done to the Queensland state registry. The identification process was strictly handled by Queensland Health staff members and identifiers such as full names, dates of birth, gender and addresses were used for the linkage. The resulting data was then de-identified before being fed back into the main data set. The SSB used mature linkage algorithms reinforced with thorough manual review by experienced data linkage professionals. Therefore, we have high confidence in the outcomes of the probabilistic linkage and we assess error rates to be very low.

Statistical Analysis

Data analysis was performed in STATA (StataCorp. 2017. Stata Statistical Software, College Station, TX, USA) and SPSS (IBM Corp. Version 27.0. Armonk, NY, USA). Descriptive summaries for continuous variables were computed as median and interquartile range (IQR) and counts and percentages for categorical variables. The Mann-Whitney U test was used to compare age and number of hospital presentations between Indigenous and non-Indigenous patients overall and those with Group One diagnosis. Chi-square test was used to compare participant's baseline demographic and clinical characteristics. The association between Indigenous status and incidence of MACE at 1 year was assessed adjusting for the variables including ethnicity, gender, age groups, alcohol consumption, and smoking status. The model was adjusted for explanatory variables with p-value ≤ 0.2 in preliminary univariate analysis. Logistic regression was used to investigate the magnitude of associations between the incidence of MACE at 1 year and covariates including indigenous status. Odds ratio (OR) estimates and 95% confidence interval were presented

for categorical variables. Inference was based on 5% level of significance.

Ethics Approval

The study was approved by the Townsville University Human ethics committee (Project ID number: LNR/2018/QTHS/47408). The data used in this study were fully anonymised before the authors had access to them.

Results

Patient Characteristics (Table 1)

A total of 4,087 patients presented to the Townsville emergency department with possible cardiac related chest pain between January 2017 and December 2017. Indigenous (Aboriginal or Torres Strait Islanders or both) patients comprised 19.3% (789) of the total whereas non-Indigenous patients made up the rest (81%, 3,298). Indigenous patients were over-represented in the study cohort when it is considered that 11.1% of the total population of North Queensland is Indigenous and that the hospitalisation rate for Indigenous Queenslanders was 5% according to the 2013–2014 data [18].

An analysis of the postcodes of the patients in Group 1 using Modified Monash (MM) model demonstrated that the majority of patients 68.97% vs 82.29% (Indigenous vs non-Indigenous) lived in MM2 which is Townsville and its surrounding regions. A significant number of Indigenous people (21.7%) lived in very remote regions (MM7) compared to the non-Indigenous people (2.7%) in this cohort. The rest of the population were evenly distributed in all other areas among Indigenous and non-Indigenous people (1.5% vs 2.7% in MM1, 1% vs 0.4% in MM3, 2% vs 5% in MM4 and MM5, and 3% vs 2.9% in MM6 respectively).

Men and women were equally distributed throughout the cohort. The Indigenous group 46.5% men to 53.5 % women. The non-Indigenous group had 50.9 % to 49.1 % (men to women).

Overall, age ranged from 16 to 102. Median age was 50 (36–64). Both men and women have similar median ages (52 vs 49 respectively). Median age at presentation was significantly younger in the Indigenous group than the non-Indigenous group (45 vs 52, $p<0.005$).

Indigenous patient's presentations ranged from 0–51, while non-Indigenous patient's presentations ranged from 0 to 25. The median number of presentations was 1. They were similar by gender. Indigenous patients had more presentations than non-Indigenous people assessed using the Mann-Whitney test.

A subgroup analysis was carried out for recurrent presentations with chest pain. Seventy (70) patients with a total of 778 presentations were recurrent. It was found that Indigenous patients made up a significant proportion of the group (52.8%, 37 [n], $p<0.005$).

Indigenous patients had significantly higher rate of alcohol excess (29.2% vs 8%, $p<0.005$), and more likely to be homeless than the non-Indigenous counterparts (2.4% vs 0.2%, $p<0.005$). The rate of discharge against medical advice was significantly higher among Indigenous patients (6.5% vs 2.7%, $p<0.005$).

The Indigenous cohort had a significantly higher prevalence of all the cardiovascular risk factors investigated in the study when compared to the non-Indigenous patients. They had a higher prevalence of patients who had ever smoked (75.9% vs 47.1 %, $p<0.005$) and had higher prevalence of having hypertension (42.9% vs 34.9%, $p<0.005$), diabetes (31.5% vs 14.2%, $p<0.005$), chronic kidney disease including patients with end-stage renal disease (13.6% vs 5%, CI 95%, $p<0.005$), hypercholesterolaemia (30.1 % vs 25.6%, $p=0.012$), previous history of ischaemic heart disease (23.6% vs 15.3%, $p<0.005$) and family history of premature ischaemic heart disease (22.9% vs 15.2%, $p<0.005$).

Indigenous patients were less likely to be referred to the cardiology service using the ACRE chest pain pathway (36% vs 45%, $p<0.005$) even though they had a similar rate of troponin elevation (14.4% vs 13.2%, $p=0.437$). Non-invasive cardiac investigations were done for both Indigenous and non-Indigenous patients and the data can be reviewed in Supplementary Table 3.

Outcomes

A total of 1,258 patients were admitted to the wards through the emergency department (295 Indigenous patients and 963 non-Indigenous patients). 2,217 patients were discharged from ED.

In terms of primary diagnoses for the presentations, there were 835 patients in diagnosis Group 1. Their age ranged from 19 to 99 years, with a mean age of 60.3 ($\pm$0.1). There were 320 patients diagnosed as having acute coronary syndrome (7.8% of total presentations), of whom 73 patients were Indigenous (32.2%) and, 247 patients were non-Indigenous (40.2%). Five hundred and fifteen (515) patients were diagnosed as chest pain for investigation (10%), cardiac chest pain (2.2%) or cardiac arrest (0.1%). The remaining patients (3,252 [n]) were diagnosed as non-cardiac chest pain and were categorised into Groups 2 and 3 (Table 2).

In diagnosis group 1, the mean age for Indigenous patients (51.7$\pm$0.8) was significantly lower than for non-Indigenous patients (63.4$\pm$0.6), a mean difference of 11.6 years, $t(833) = 11.21$, $p<0.001$, 95% CI (9.6 to 13.7).

A total of 367 patients (27% Indigenous and 73% non-Indigenous) underwent invasive coronary angiogram either as an inpatient or outpatient. A comparison of the results of the coronary angiograms revealed a significantly higher proportion of Indigenous patients had severe three vessel disease compared to their non-Indigenous counterparts (17% vs 6%, $p<0.005$). A further 514 patients (28% Indigenous and 72% non-Indigenous) underwent non-invasive cardiac investigations to risk stratify the chest pain either as an inpatient or outpatient. The incidence of having moderate to severe left ventricular dysfunction on echocardiogram was similar among Indigenous and non-Indigenous patients.

Table 1 Overall Patient demographics and clinical variables comparing Indigenous and non-Indigenous patients presenting to the Emergency Department with chest pain.

Variables	All Patients Median (*IQR*) or n (%)	Indigenous Patients Median (*IQR*) or n (%)	Non-Indigenous Patients Median (*IQR*) or n (%)	P-value
Total No. of patients	4,087 (100%)	789 (19.3)	3,298 (80.7)	NA
Age				
15-34 yr	906 (22.2)	214 (27.1)	692 (21.0)	
35-54 yr	1,478 (36.2)	372 (47.2)	1,106 (33.5)	p<0.005
55-74 yr	1,258 (30.8)	180 (22.8)	1,078 (32.7)	
75 yr plus	445 (10.9)	23 (2.9)	422 (12.8)	
Number of presentations per each patient in a year	1 (1-1)	1 (1-2)	1 (1-1)	p<0.005[a]
Number of patients with presentations more than 5 times in one year	70 (1.7)	37 (4.6)	33 (1)	p<0.005
Gender (male)	2,046 (50.1)	367 (46.5)	1,679 (50.9)	p=0.027
Social Living				
Regular home	3,507 (96.1)	678 (90.5)	2,829 (97.5)	
Homeless	27 (0.7)	19 (2.5)	8 (0.3)	p<0.005
Nursing home	29 (0.8)	<5 (<1)[b]	26 (0.9)	
Others	88 (2.4)	49 (6.5)	39 (1.3)	
Smoking				
Never	1,761 (47.3)	174 (24.1)	1,587 (52.9)	
Ever smoked	1,961 (52.7)	547 (75.9)	1,414 (47.1)	p<0.005
Alcohol				
No	2,049 (60.2)	323 (48.3)	1,726 (63.2)	
Yes	940 (27.6)	151 (22.6)	789 (28.9)	p<0.005
Excess	413 (12.1)	195 (29.2)	218 (8.0)	
Hypertension				
Yes	1,429 (36.5)	324 (42.9)	1,105 (34.9)	p<0.005
Diabetes				
Yes	686 (17.6)	240 (31.5)	446 (14.2)	p<0.005
Chronic Kidney Disease				
Yes	262 (6.6)	104 (13.6)	158 (5.0)	p<0.005
Hypercholesterolaemia				
Yes	1,024 (26.5)	228 (30.1)	796 (25.6)	p=0.012
Family history of premature ischaemic heart disease				
Yes	453 (16.7)	126 (22.9)	327 (15.2)	p<0.005
Previous history of ischaemic heart disease				
Yes	675 (16.9)	183 (23.6)	492 (15.3)	p<0.005
Previous stroke				
Yes	92 (2.3)	20 (2.6)	72 (2.3)	p=0.586
Chest pain pathway referral				
Yes	1,740 (43.0)	277 (35.8)	1,463 (44.7)	p<0.005
No	2,306 (57.0)	497 (64.2)	1,809 (55.3)	
Invasive angiogram				
Yes	444 (10.8)	123 (15.5)	321 (9.7)	p<0.005
Non-invasive cardiac investigations				
Yes	859 (21.0)	225 (28.5)	634 (19.2)	p<0.005
Troponin elevation				
Yes (≥0.04 ug/L)	391 (13.5)	92 (14.4)	299 (13.2)	p=0.437
No (<0.04 ug/L)	2,507 (86.5)	546 (85.6)	1,961 (86.8)	

Table 1. (continued).

Variables	All Patients Median (*IQR*) or n (%)	Indigenous Patients Median (*IQR*) or n (%)	Non-Indigenous Patients Median (*IQR*) or n (%)	P-value
Departure Status				
➢ Admitted to inpatient ward	1,258 (30.8)	295 (37.4)	963 (29.2)	
➢ Died in ED	2 (0.0)	0 (0.0)	2 (0.1)	
➢ Admitted to ED short stay	431 (10.6)	65 (8.2)	366 (11.1)	p<0.005
➢ Home/usual residence	2,217 (54.3)	377 (47.8)	1,840 (55.9)	
➢ Discharged against medical advice	139 (3.4)	51 (6.5)	88 (2.7)	
➢ Transfer to other hospital	32 (0.8)	1 (0.1)	31 (0.9)	

Abbreviations: CABG, coronary artery bypass graft surgery; ED, emergency department.
[a]Mann-Whitney U test, others are Chi-Square test.
[b]<5= (actual value range between 0.1 and 4.99).

Table 2 Final diagnoses of the chest pain presentations.

Diagnosis Groups	All Patients n (%)	Indigenous Patients n (%)	Non-Indigenous Patients n (%)	P-value
Group 1				
Acute coronary syndromes	320 (7.8)	73 (9.5)	247 (7.5)	
• Unstable angina	55 (1.3)	11 (1.4)	44 (1.3)	
• Non-ST elevation myocardial infarction	196 (4.8)	42 (5.4)	154 (4.7)	
• ST elevation myocardial infarction	69 (1.7)	20 (2.3)	49 (1.5)	
Other chest pains	515 (12.6)	147 (19)	368 (11.2)	p=0.068
• Chest pain for investigation	414 (10.1)	98 (12.7)	316 (9.6)	
• Cardiac chest pain	88 (2.2)	44 (5.7)	44 (1.5)	
• Cardiac arrest	6 (0.1)	<5 (<1)[b]	<5 (<1)[b]	
• Others	7 (0.2)	<5 (<1)[b]	<5 (<1)[b]	
Group 2[a]	2,949 (72.1)	488 (63.2)	2,461 (74.9)	p<0.005
Group 3[a]	274 (6.7)	64 (8.3)	211 (6.4)	p=0.054

Note: There are discrepancies in the overall total number of patients compared with Table 1 due to some patients not getting a diagnosis.
[a]The diagnoses for group 2 and 3 are provided in Supplementary Table 2.
[b]<5= (actual value range between 0.1 and 4.99).

In terms of treatment outcomes of patients in group 1 (Table 3), a higher proportion of Indigenous patients received coronary artery bypass graft (19% vs 6%, p<0.005) whereas Indigenous patients were less likely to receive percutaneous coronary interventions (13% vs 20%, p<0.005). A similar proportion of Indigenous and non-Indigenous patients were treated conservatively (16% vs 16.6%). However, Indigenous patients were treated conservatively primarily due to non-adherence to medications (33% vs 1%, p<0.005).

Major adverse cardiac events (MACE) were defined as cardiovascular deaths, non-fatal MI, non-fatal stroke and admission for heart failure. The difference in MACE on univariate analysis was not considered statistically significant at 1 year. The MACE at 1 year was 10.9% in the Indigenous group while in the non-Indigenous group was 7.5% with a p-value of 0.08. At 1 year the cardiovascular deaths were 6 (2.7%) in the Indigenous group and 24 (3.9%) in the non-Indigenous group. There were 24 (2.9%) for non-fatal MI, 14 (6.4%) within Indigenous group and 10 (1.6%) in the non-Indigenous group. There were 16 (1.9%) admissions for heart failure, 4 (1.8%) for Indigenous and 12 (2%) for the non-Indigenous groups. There were only two non-fatal stroke which occurred in non-Indigenous group.

Multivariable Analysis

Multivariable logistic regression was performed to examine the risk of MACE for Indigenous versus non-Indigenous

Table 3 Comparison of outcomes in diagnosis Group 1.

Variables	All Patients Median (*IQR*) or n (%)	Indigenous Patients Median (*IQR*) or n (%)	Non-Indigenous Patients Median (*IQR*) or n (%)	P-value
Total no. of patients	835 (100%)	220 (26.3)	615 (73.7)	NA
Age				
18-34 yr	25 (3.0)	17 (7.7)	8 (1.3)	
35-54 yr	280 (33.5)	120 (54.6)	160 (26.0)	p<0.005
55-74 yr	380 (45.5)	76 (34.6)	304 (49.4)	
75 yr plus	150 (18.0)	7 (3.2)	143 (23.3)	
Gender (male)	485 (58.1)	104 (47.3)	381 (62.0)	p<0.005
Invasive Coronary Angiogram				
Done	367 (44.0)	98 (44.5)	269 (43.7)	p=0.836
Invasive Coronary Angiogram Results				
• Normal angiogram	38 (4.6)	8 (3.6)	30 (4.9)	
• Mild non-obstructive coronary artery disease	56 (6.7)	10 (4.5)	46 (7.5)	
• Moderate non-obstructive coronary artery disease	32 (3.8)	8 (3.6)	24 (3.9)	
• Severe single vessel disease	102 (12.2)	15 (6.8)	87 (14.1)	
• Severe two vessel disease	46 (5.5)	14 (6.4)	32 (5.2)	
• Severe three vessel disease	76 (9.1)	38 (17.3)	38 (6.2)	
• Severe left main disease	<5 (<0.6)[a]	<5 (<2.3)[a]	<5 (<0.8)[a]	
• Severe graft disease	13 (1.6)	<5 (<2.3)[a]	9 (1.5)	
Non-Invasive Cardiac Investigations				
Done	514 (61.6)	142 (64.5)	372 (60.5)	p=0.288
Echocardiogram				p=0.876
Normal left ventricular (LV) function	157 (57.1)	47 (57.3)	110 (57.0)	
Mildly reduced LV function	72 (26.2)	22 (26.8)	50 (25.9)	
Moderately reduced LV function	24 (8.7)	8 (9.8)	16 (8.3)	
Severely reduced LV function	22 (8.0)	5 (6.1)	17 (8.8)	
Treatment Outcomes				p<0.005
• Percutaneous intervention	138 (17.9)	27 (13.1)	111 (19.6)	
• Coronary artery bypass surgery	76 (9.8)	40 (19.4)	36 (6.4)	
• Medical/conservative management	127 (16.5)	33 (16.0)	94 (16.6)	
• Cardiac investigations normal/unchanged	162 (21.0)	44 (21.4)	118 (20.8)	
• Referred for cardiac clinic	171 (22.2)	36 (17.5)	135 (23.9)	
• Discharged from ED	38 (4.9)	5 (2.4)	33 (5.8)	
• Discharged against medical advice	25 (3.2)	15 (7.3)	10 (1.8)	
• Not available	18 (2.3)	<5 (<2.3)[a]	16 (2.8)	
Reasons for Conservative Management for IHD				p<0.005
• Non-obstructive CAD	69 (8.2)	16 (7.3)	53 (8.6)	
• Significant co-morbidities	45 (5.4)	6 (2.7)	39 (6.3)	
• Non-adherence to treatment	12 (1.4)	11 (5)	<5 (<0.8)[a]	
MACE at 1 yr				
• No MACE	763 (91.6)	196 (89.1)	567 (92.5)	
• MACE	70 (8.4)	24 (10.9)	46 (7.5)	p=0.08
➢ Cardiovascular death	30 (3.6)	6 (2.7)	24 (3.9)	
➢ Non-fatal MI	24 (2.9)	14 (6.4)	10 (1.6)	
➢ Admission for heart failure	16 (1.9)	4 (1.8)	12 (2.0)	

Abbreviations: MACE, major adverse cardiac events; CAD, coronary artery disease; IHD, ischaemic heart disease; ED, emergency department; LV, left ventricular; MI, myocardial infarction.

[a]< 5= (actual value range between 0.1 and 4.99).

Table 4 Multivariable robust logistic regression of factors associated with MACE at 1 year.

Risk Factors	Major Adverse Cardiac Event (MACE) at 1 Year		
	Adjusted Odds Ratio	95% Confidence Interval	P-value
Indigenous status	2.0	1.1-3.8	0.03
Gender, male	2.2	1.2-4.0	0.01
Age, yr[a]			
35-54	0.7	0.2-3.0	0.66
55-74	0.7	0.2-3.1	0.67
75 plus	1.8	0.4-8.4	0.47
Smoking, Ever smoked	1.6	0.9-3.1	0.14
Presence of previous history of ischaemic heart disease	2.3	1.3-4.1	0.01

[a]Reference age = 15-34 year old.

patients, taking into account the confounding factors. All variables with p≤0.2 in the univariate analysis were included in the multivariable analysis. At the end, the explanatory variables that were found to influence the incidence of MACE at 1 year were: Indigenous status, gender, age categories, smoking and previous history of ischaemic heart disease (Table 4).

Using the most parsimonious model with a log-pseudo-likelihood of -233.80 and an acceptable goodness of fit test [Table 4]. Indigenous patients were more likely to suffer from MACE at 1 year (adjusted odds ratio [AOR]=2.0, 95% CI [1.1, 3.8], p=0.03). The odds of MACE at 1 year is 2.2 among males than their female counterparts, (AOR=2.2, 95% CI [1.2, 4.0], p=0.01), this means that the likelihood of males having MACE at 1 year is significantly higher than that of females. The odds of MACE at 1 year also increased with a previous history of IHD (AOR=2.3, 95% CI [1.3, 4.1], p=0.005), with smoking and age above 75. These were, however, not statistically significant.

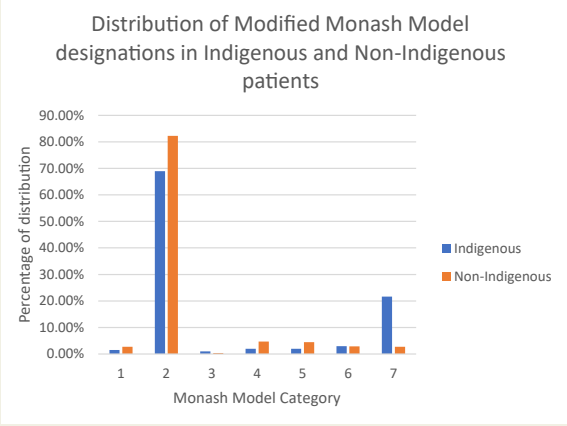

Figure 1 Distribution of both Indigenous and non-Indigenous cohorts, by area of remoteness using Modified Monash Model.

Discussion

The audit is a snapshot of chest pain presentations to the Townsville University Hospital (TUH), which is a major cardiac tertiary referral centre in North Queensland that treats a large number of Aboriginal and Torres Strait Islander patients. It shows that Indigenous patients who presented with cardiac-related chest pain have a higher burden of cardiovascular risk factors, present at a younger median age, have a higher prevalence of three vessel disease that could require surgical revascularisation. The multivariable analysis demonstrated that Indigenous patients have a higher risk of major adverse cardiovascular events (MACE) at 1 year when compared to their non-Indigenous counterparts.

The literature clearly describes the disparity between Indigenous and non-Indigenous patients in relation to ischaemic heart disease at every stage of the disease process. It begins with an excess and disproportionate effect of cardiovascular risk factors, poor socioeconomics and a younger median age of presentation. This results in a younger age of presentation for a coronary artery bypass graft (CABG), higher rates of preoperative left ventricular dysfunction, higher rates of midterm morbidity, reduced long-term survival and a higher rate of MACEs [9,19,20].

The audit confirmed that with regard to chest pain presentations, the Indigenous cohort carried a disproportionately higher burden of cardiovascular risk factors when compared to the non-Indigenous cohort. Indigenous patients had a greater degree of social disadvantage especially with regard to the higher rate of homelessness and rurality. A significant proportion lived in areas classed as very remote (MM7) when compared to non-Indigenous patients (21.7% vs 2.7%) (Figure 1). The Indigenous cohort were also more likely to consume an excessive amount of alcohol. These social factors could have a detrimental effect on adherence to treatment, long-term follow-up and outcomes.

A closer analysis of the emergency department presentations suggested that there was an underutilisation of the

local chest pain pathway amongst the Indigenous group (35.8% vs 44.7%). The reasons for underutilisation of the clinical pathway in the ED was not clear from the data and was beyond the scope of the study. However, this is a significant issue, which would need to be addressed with the aim of improving adherence to the pathway and improving the outcomes for Indigenous patients presenting the cardiac chest pain. A 2016 single centre audit in Western Australia showed that a quantifiable improvement in the approach to chest pain assessment is achievable with improvements to support structures in ED [21].

The rate of discharge against medical advice was also significantly higher (6.5% vs 2.7% $p<0.005$). A comparison of the treatment outcomes showed that the rate of medical or conservative management was similar between the two groups (16% vs 16.6 %) (Table 3). A further analysis into the reasons for conservative management revealed that Indigenous patients had a lower rate of non-obstructive disease and significant co-morbidities. However, they were managed conservatively primarily due to non-adherence to treatment. The data pertaining to non-adherence in our study is limited because it was taken at face value during data collection. This is a complex matter that was likely driven by several factors such as cultural misunderstanding, waiting times, transport issues, financial constraints and poor health literacy [22]. Future studies into this issue would need a culturally sensitive standardised measure of reporting adherence in order to avoid biases.

In relation to patients in Group 1, which were patients with acute coronary syndrome, chest pain for investigation and cardiac chest pain, Indigenous patients presented 11.6 years younger than the non-Indigenous cohort and such findings are consistent with current metanalyses [9]. The literature thus far shows Indigenous patients receiving coronary angiography at a lower rate when compared to non-Indigenous patients, which was inconsistent with our study population where equal proportions of Indigenous and non-Indigenous patients received an invasive coronary angiogram [23,24]. A Newcastle analysis of coronary angiograms comparing the Indigenous and non-Indigenous patients found that Indigenous patients had more significant coronary artery lesions, which were less amenable to percutaneous coronary intervention (PCI), although there was no excess in multivessel disease [25]. On the contrary, our findings suggested that Indigenous patients had a higher rate of severe three vessel disease that could lead to higher rates of coronary artery bypass graft surgery. One study comparatively analysed invasive coronary angiogram findings and demonstrated that Indigenous patients had more prevalent severe three vessel disease compared to non-Indigenous patients. There was also a strong association between Indigenous ethnicity and significant coronary artery disease (AOR 2.7, 95% CI [1.38, 5.39]), independent of the disproportionate burden of traditional cardiovascular risk factors in the Indigenous cohort [26]. A recent prospective longitudinal study in regional Australia has also been able to show that Indigenous Australians were 2.8 times more likely to have a higher burden of coronary artery disease, independent of the higher rate of cardiovascular risk factors [27]. This association is suggestive of a distinct predisposition to coronary artery disease amongst Indigenous Australians that would require further study.

Finally, the key primary outcome in the study was MACE at 1 year. The univariate analysis for MACE did not demonstrate a statistically significant difference between the two groups. However, the multivariable analysis showed that Indigenous patients had twice the odds of MACE in 1 year compared to the non-Indigenous patients of the same age group, gender and risk factor profiles. The literature also suggests that the outcomes measured in the MACE occurs at a disproportionately higher rate in the Indigenous patients [28]. In our study, a higher risk of MACE in Indigenous patients occurred despite a living in relative proximity to a regional centre (69% resided in MM2) and having similar access to a coronary angiogram to non-Indigenous patients. We noted that the risk of MACE was not statistically different in regards to age over 75 and a history of smoking despite the fact that the odd ratios were significantly higher for them (AOR 1.8, 95% CI [0.4–8.4] and AOR 1.6, 95% CI [0.9–3.1] respectively). We believe that was due to a smaller sample size in this cohort.

These findings suggest that there are improvements that can be made at a local level to better manage cardiovascular disease in Indigenous patients. It would be prudent to establish robust local programs with ongoing secondary prevention strategies for coronary artery disease, improvement with cardiac rehabilitation programs and effective outreach services in very remote areas. Improving health literacy in the community by targeting a younger age group can also be effective, considering the younger age of onset for coronary heart disease in the Indigenous population.

At a national level, it would be advisable to revise the current 10-year absolute cardiovascular risk score calculation since the current risk calculator may under-estimate the risk for Indigenous people [29]. A more accurate cardiovascular disease risk assessment for Indigenous people would increase the disease surveillance among health care providers and improve awareness within the community itself.

Limitations

One limitation of the study is collecting the documentation of patient's accurate medical history on the initial presentation to ED. An effort is made to review through the documentation of past and subsequent encounters through electronic medical records (eMR) and discharge summaries to improve the accuracy of the health information of each patient. Another factor is an interpretation bias when reviewing the medical records. We made an independent analysis of medical history, investigation findings and 1-year MACE outcome to minimise this bias. Thirdly, the study may not represent the true volume of Indigenous and non-Indigenous patients suffering from ischaemic heart disease in North Queensland. We focussed on analysing the patient cohort presenting with chest pain suspicious of IHD through the

ED. It is common knowledge that coronary heart disease can present with symptoms other than chest pain. In addition, there was missing information for 0.8% (n 32) of patients (majority were non-Indigenous patients) who were transferred to another hospital because we were unable to access their records.

Conclusion

The study suggests that the local population of Aboriginal and Torres Islander patients who present to the emergency department with chest pain have a higher burden of cardiovascular risk factors, have a younger median age of presentation, have a higher prevalence of severe three vessel disease that could likely lead to surgical revascularisation. We found an underutilisation of the local chest pain protocol amongst the Indigenous cohort, which suggests a need to improve support structures in the ED. In our multivariable analysis, Indigenous patients had a higher risk of MACE at 1 year compared to the non-Indigenous patients. This indicates that more collaborative efforts are needed to improve the cardiovascular health of local Aboriginal and Torres Islander people.

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Competing Interest

We report no competing interest associated with the work reported in this manuscript.

Conflict of Interest Statement

There are no conflicts of interest to disclose.

Authors Contributions

Conceptualisation, KTHW, BT, TIE, RY; methodology, KTHW, TIE, RY; software, TIE; validation, KTHW, BT, TIE, RY formal analysis, TIE; investigation, KTHW, BT, TIE, RY and all other authors; resources, KTHW, BT, TIE, RY and all other authors; data curation, all the others; writing—original draft preparation, KTHW, BT, TIE, RY; writing—review and editing, KTHW, BT, TIE, RY, all others; funding acquisition, KTHW. All authors have read and agreed to the published version of the manuscript.

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Appendices. Supplementary Data

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