Incidence and survival after acute myocardial infarction in Indigenous and non-Indigenous people in the Northern Territory, 1992–2004

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ABSTRACT

Objective: To estimate the incidence and survival rates of acute myocardial infarction (AMI) for Northern Territory Indigenous and non-Indigenous populations.

Design and participants: Retrospective cohort study for all new AMI cases recorded in hospital inpatient data or registered as an ischaemic heart disease (IHD) death between 1992 and 2004.

Main outcome measures: Population-based incidence and survival rates by age, sex, Indigenous status, remoteness of residence and year of diagnosis.

Results: Over the 13-year study period, the incidence of AMI increased 60% in the NT Indigenous population (incidence rate ratio [IRR], 1.04; 95% CI, 1.02–1.06), but decreased 20% in the non-Indigenous population (IRR, 0.98; 95% CI, 0.97–1.00). Over the same period, there was an improvement in all-cases survival (ie, survival with and without hospital admission) for the NT Indigenous population due to a reduction in deaths both pre-hospital and after hospital admission (death rates reduced by 56% and 50%, respectively). The non-Indigenous all-cases death rate was reduced by 29% as a consequence of improved survival after hospital admission; there was no significant change in pre-hospital survival in this population. Important factors that affected outcome in all people after AMI were sex (better survival for women), age (survival declined with increasing age), remoteness (worse outcomes for non-Indigenous residents of remote areas), year of diagnosis and Indigenous status (hazard ratio, 1.44; 95% CI, 1.21–1.70).

Conclusions: Our results show that the increasing IHD mortality in the NT Indigenous population is a consequence of a rise in AMI incidence, while at the same time there has been some improvement in Indigenous AMI survival rates. The simultaneous decrease in IHD mortality in NT non-Indigenous people was a result of reduced AMI incidence and improved survival after AMI in those admitted to hospital. Our results inform population-specific strategies for a systemwide response to AMI management.

METHODS

Ischaemic heart disease includes AMI, angina pectoris and other related conditions. However, the difficulty of obtaining reliable data on angina pectoris and related conditions means that monitoring of incidence and survival is necessarily confined to AMI events.

Identification of new AMI cases

Almost all people who suffer an AMI in Australia are admitted to hospital for treatment, with the exception of those who die suddenly. We used three sources to identify new AMI cases: hospital inpatient records and two sources of death registration data.

The five public hospitals in the NT share a single patient-information system, in which each patient is allocated a unique hospital registration number. We identified all inpatient episodes for NT residents with any AMI diagnosis code from the NT hospital separations dataset for the period 1 July 1990 to 31 December 2004. AMI diagnosis codes comprised ICD-9 (International classification of diseases, 9th revision) code 410 (for 1990–1997) and ICD-10 (ICD, 10th revision) code I21 (for 1998–2004). We collated inpatient episodes for all patients and identified the first AMI episode. Patients with an AMI episode in the 18-month clearance period before 1 January 1992 were excluded. During the study period, the NT had one private hospital; this hospital did not have coronary care facilities, and patients diagnosed with an AMI were routinely transferred to the adjoining public hospital.

We used deaths data from the Australian Bureau of Statistics (ABS) to identify all deaths of NT residents from AMI that occurred in Australia between 1 January 1992 and 31 December 2004. All deaths with an underlying cause of IHD (ICD-9 codes 410–414 for 1992–1996; ICD-10 codes I20–I25 for 1997–2004) were included. These expanded codes were necessary as the distinction between AMI and other IHD subcategories in deaths data has been shown to be unreliable. This approach was consistent with that used in previous studies of national AMI incidence.

Combining hospital inpatient and ABS death data created an overlapping list of AMI cases. To eliminate the overlap, we matched individuals identified in the hos-
pital data with the National Death Index (NDI) held at the Australian Institute of Health and Welfare using probabilistic matching on names, date of birth and sex. Potential matches were verified manually. We then separated matched cases from the IHD deaths identified from the ABS data using the death registration number, to establish a list of new AMI patients who died without hospital admission.

Matching across the three datasets created three study cohorts: a group who died without hospital admission; a second group who were admitted to hospital and died as a result of IHD; and a third group who were admitted and either survived or died of other causes.

Statistical analysis

We calculated age-specific and directly age-standardised AMI incidence rates by age group, sex, Indigenous status, year of diagnosis and residence (urban [Darwin and Alice Springs] or remote), using the NT population derived from ABS estimated resident population data. We used the 2001 Australian estimated resident population as the standard population.

Three outcome measures were investigated: AMI incidence; access to treatment (the proportion of people who died without being admitted to hospital); and survival after an AMI (cause-specific mortality for all patients and also for hospitalised patients). We used regression modelling to assess the association between each of these three outcome measures and five risk factors: sex (female versus male), Indigenous status (Indigenous versus non-Indigenous), place of residence (remote versus urban), age at diagnosis (compared with median age) and year of diagnosis (compared with 1998). Negative binomial regression was used to model AMI incidence; logistic regression to model the proportion of deaths without hospitalisation; and proportional hazards regression to model the risk of death after AMI. Results are presented as incidence rate ratio (IRR), odds ratio (OR) and hazard ratio (HR), respectively.

Interaction terms were assessed by backward selection for each outcome measure. All interaction terms in the final models included the risk factor “Indigenous status”, indicating that the association between the other risk factor in the term and the outcome measure was different for Indigenous and non-Indigenous people. In these cases, the general estimate for the population expressed the association for non-Indigenous people. The association for Indigenous people was calculated by multiplying the estimate for the non-Indigenous population by the estimate for the interaction term.

We censored data for survival analysis at 31 December 2004; patients who died from a cause other than IHD before this date were censored at the date of death. Survival time was estimated as follows: for AMI patients who were admitted to hospital and died of IHD before the censoring date, the survival time was the date of death minus the first AMI admission date; for other patients admitted to hospital with AMI, the survival time was the censoring date minus the first AMI admission date; and for those who died without being admitted to hospital, survival time was assumed to be 0.5 days.

All statistical analyses were performed using Stata, version 10 (StataCorp, College Station, Tex, USA).

Ethics approval

The study was approved by the Human Research Ethics Committee of the NT Department of Health and Families and the Menzies School of Health Research. The Human Research Ethics Committee of the Australian Institute of Health and Welfare approved access to the NDI.

RESULTS

We identified 3419 new AMI cases in NT residents between 1 January 1992 and 31 December 2004 (Box 1). Of these, 1417 people (41%) were Indigenous, and 2414 (71%) were men. Median age was older for women than for men, and also for non-Indigenous than for Indigenous people.

Incidence of AMI

The univariate analysis showed that the age-adjusted AMI incidence rate was higher for Indigenous than for non-Indigenous people, higher for men than for women, and higher in remote areas than in urban areas for non-Indigenous people, but not for Indigenous people (Box 2). Incidence increased sharply with age group.

National AMI incidence has been previously reported for the population aged 40 years or over. In this age group in the NT,
the AMI incidence rate for non-Indigenous people dropped between 1992 and 2004 by 23% (IRR, 0.98 per year; 95% CI, 0.95–1.01). In the years for which national data are available, AMI incidence rates for non-Indigenous NT men and women did not differ greatly from national rates, and decreases were at much the same rate (Box 3).

In contrast, the incidence rates among Indigenous men and women aged 40 years or over in the NT, although similar to NT non-Indigenous and national rates at the beginning of the study period, increased between 1992 and 2004 by 48% (IRR, 1.03 per year; 95% CI, 1.00–1.07). By 1998, these rates were about twice those in the corresponding national populations (Box 3).

For the 20–39-year age group, AMI incidence rates increased over the period 1992–2004 in both the non-Indigenous and Indigenous NT populations, by 89% (IRR, 1.05; 95% CI, 0.98–1.13) and 116% (IRR, 1.07; 95% CI, 1.01–1.12), respectively.

After adjustment for risk factors (Box 4), AMI incidence was 56% lower in women than in men. For non-Indigenous people, incidence increased with age by 10% per year, was 102% higher in remote than urban areas, and decreased by 20% between 1992 and 2004 (IRR, 0.98; 95% CI, 0.97–1.00). For Indigenous people, incidence increased with age by 6% per year (1.06; 95% CI, 1.05–1.06), was similar in urban and remote areas (IRR, 1.05; 95% CI, 0.93–1.21), and increased by 60% between 1992 and 2004 (IRR, 1.04 per year; 95% CI, 1.02–1.06).

The IRR for Indigenous compared with non-Indigenous people varied by age at diagnosis, year of diagnosis and residence (Box 4). Compared with the non-Indigenous urban population, at the median age of AMI diagnosis (58 years) Indigenous AMI incidence was 95% higher in 1992 (IRR, 1.95; 95% CI, 1.60–2.37) and 290% higher in 2004 (IRR, 3.90; 95% CI, 3.27–4.65).

Mortality from AMI

The proportion of deaths from AMI that occurred without hospital admission (prehospital deaths) was lower in women than in men (OR, 0.75) and increased with age (OR, 1.03 per year of age) (Box 4). The proportion was also higher in Indigenous than in non-Indigenous people (OR, 1.88, at median age of diagnosis [58 years] and middle year of study period [1998]).

For non-Indigenous people, the proportion of AMI deaths without hospital admission was greater for remote than urban residents (OR, 1.70) and did not change over time (OR, 1.10). For Indigenous people, the proportion was similar in remote and urban areas (OR, 1.09, 95% CI, 0.85–1.40) and decreased by 56% between 1992 and 2004 (OR, 0.93 per year; 95% CI, 0.91–0.96).

For patients admitted to hospital, the death rate after an AMI was higher for Indigenous than for non-Indigenous patients (HR, 1.75 at median age of diagnosis and middle year of study period) (Box 4), but decreased for both groups between 1992 and 2004, by 75% for non-Indigenous people and 50% for Indigenous people (HR, 0.94 per year; 95% CI, 0.90–0.99). The death rate for hospitalised patients did not differ by sex or by remoteness of residence, but did increase with age at diagnosis, by 7% per year for non-Indigenous patients and 3% per year for Indigenous patients (HR, 1.03; 95% CI, 1.02–1.05).

For all people with AMI (ie, with and without hospital admission), the death rate was higher for Indigenous than for non-
Indigenous people (HR, 1.44; 95% CI, 1.21–1.70) at median age at diagnosis for urban cases (Box 4). The death rate was 11% lower for women than men, and decreased by 29% between 1992 and 2004. For non-Indigenous people, the death rate increased with age at diagnosis by 3% per year, and was 27% higher for remote than urban residents. For Indigenous people, the death rate increased with age at diagnosis by 2% per year (HR, 1.02; 95% CI, 1.01–1.02) and was similar for remote and urban residents (HR, 1.06; 95% CI, 0.90–1.25).

DISCUSSION

We found that annual incidence of AMI in the non-Indigenous population of the NT was similar to that in the general Australian population between 1993 and 1999, and decreased at a similar rate during this time. During our study period, 1992–2004, there was also a considerable increase in survival rates for patients hospitalised with AMI. Increased survival is consistent with the growth of specialised coronary care services and the growing emphasis on post-hospital management of patients with AMI. However, offsetting this improvement was our finding that the risk of pre-hospital death changed little, so that the improvement in non-Indigenous all-cases survival was smaller than that in hospitalised patients (death rates decreased by 29% and 75%, respectively).

For the Indigenous population of the NT, the pattern was different. AMI incidence was similar to national rates in the early 1990s, but increased in subsequent years (while the national rate was falling) to be around twice the national rate by 1998. The increase in incidence was offset by an improvement in all-cases survival in the NT Indigenous population, a result of a decrease in both pre-hospital mortality and mortality among those who reached hospital (death rates reduced by 56% and 50%, respectively). The change in pre-hospital mortality indicates a substantial improvement in early management of AMI — a combination of patients’ recognition and response to their condition, initial primary health care management and access to hospital care. However, there is still much room for improvement, as Indigenous people with AMI in the NT have a 44% higher risk of death than non-Indigenous people.

From these results, we conclude that the previously reported rising rate of deaths associated with IHD in the NT Indigenous population is due to increased incidence of IHD in this population, moderated by the effect of improved survival rates.

Our results also highlight the varying contributions of sex and remote residence to outcome. Being male or a non-Indigenous resident of a remote area was associated with increased risk of pre-hospital death, but had no impact on mortality for those who reached hospital. Urban residence was not “protective” for the Indigenous population, with those living in urban locations having a similar rate of pre-hospital death as those living in remote areas.

Many factors contribute to the poorer outcomes observed for the Indigenous NT population after first hospitalisation for AMI. These include higher rates of AMI-related risk factors, and poorer access to coronary procedures that improve outcomes. Access to procedures is itself influenced by the individual’s preference and consent to treatment, geographic remoteness and clinical decision making. The rate of coronary procedures has been reported as being lower for Indigenous than non-Indigenous people, not only during the index admission but also during subsequent admissions. Other factors that influence outcome include delays to hospital presentation, affordability of medications, compliance with clinical management plans and access to primary care services.

In addition, the difference in AMI incidence between remote and urban non-Indigenous populations reinforces the need for tailored responses to improve outcomes in different populations.

Our study had several limitations. Retrospective linking of hospital and deaths data provided a reliable, but not perfect, data source. For example, 40 of 667 patients (6%) known from hospital data to have died were not identified as deceased when matched to the NDI. Data may also have been duplicated for patients with more than one hospital admission and a variation in their identification details.

A further limitation was that some people with AMI may have been missed because they were misdiagnosed or not admitted to hospital. Missed cases would result in underestimation of true AMI incidence. Survival might also be underestimated because patients missed from hospital data survived the AMI. This effect would be balanced by those early deaths that were incorrectly attributed as AMI deaths. However, these deficits would have little effect on time trends.

A 2003 study in Western Australia showed the importance of a clearance period in data collection, reporting a 13% overestimation of AMI incidence in the early years of studies that did not use such a period. We incorporated a 1.5-year clearance period into our study, which maximised the availability of hospital cases within the available dataset, but a small proportion of previous AMI admissions may have been overlooked.

Our study demonstrates the usefulness of routinely collected data for investigating population health trends and health system performance that previously could not be measured, particularly on a systemwide basis. The national study of AMI incidence and case fatality was not able to measure survival rates, as data for individuals could not be linked.

In this study, the availability of unique patient identifiers across all NT public hospitals allowed the separate calculation of the proportion of people who died without hospital admission, and survival rates for hospitalised patients. These data provide important measures of health system performance, including rates of access to acute care (proportion who died without hospital admission), health service performance (survival for hospitalised patients) and an integrated measure of “whole system” performance (survival for all people after AMI). The different patterns of change over time for Indigenous and non-Indigenous people with AMI highlight the different issues that need to be addressed in each population group.

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COMPETING INTERESTS

None identified.

AUTHOR DETAILS

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