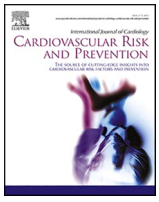
International Journal of Cardiology Cardiovascular Risk and Prevention 19 (2023) 200214

Contents lists available at ScienceDirect

International Journal of Cardiology Cardiovascular Risk and Prevention

journal homepage: www.journals.elsevier.com/international-journal-of-cardiology-cardiovascular-risk-and-prevention



Pre-hospital and in-hospital ST-elevation myocardial infarction from 2008 to 2020 in Australia

Seshika Ratwatte [a,b], Austin Chin Chwan Ng [a,b], Karice Hyun [a,b], Robin Philip [b], Farzaneh Boroumand [c], Courtney Weber [d], Leonard Kritharides [a,b], David Brieger [a,b,*]

[a] Department of Cardiology, Concord Repatriation General Hospital, NSW, Australia
[b] University of Sydney, Faculty of Medicine and Health, NSW, Australia
[c] School of Mathematical and Physical Sciences, Macquarie University, NSW, Australia
[d] School of Population and Global Health, The University of Western Australia, Crawley, Western Australia, Australia

ARTICLE INFO

Handling Editor: D Levy

1. Introduction

Contemporary treatment of ST-elevation myocardial infarction (STEMI) is optimally achieved by timely reperfusion. Urgent percutaneous coronary invention (PCI) is the gold standard for reperfusion in STEMI patients [1]. Rates of STEMI have decreased, whilst rates of PCI have increased in the modern era, both within Australia and internationally [2,3]. In the most populous state of Australia, New South Wales (NSW), statewide initiatives have focused on early recognition and diagnosis of STEMI patients in the community. These include public health campaigns educating the community on early recognition of symptoms [4,5] and the Early Triage of Acute Myocardial Infarction (ETAMI) program which aims to provide pre-hospital triage to expedite reperfusion [6]. There has also been an expansion of PCI capable cardiac catheterisation facilities in NSW in the same period. Similar improvements in treatment strategies have occurred internationally [7,8]. Whilst a corresponding fall in mortality has been noted, the extent of this decline differs between countries, with limited data on temporal mortality trends available after 2010 [8–10].

These improvements in outcomes have largely been seen in patients presenting to hospital with STEMI, defined as pre-hospital STEMI (PH-STEMI). There is much less data on patients who are diagnosed with STEMI as inpatients, or in-hospital STEMI (IH-STEMI). Whilst it is recommended that catheterisation laboratory activation for IH-STEMI should mirror PH-STEMI protocols as closely as possible [11] these patients are less likely to undergo invasive testing and have poorer outcomes [12–14]. The incidence and outcomes of patients with IH-STEMI in Australia are not known. In addition, prior studies were limited by short follow-up, thus, temporal trends on PCI usage and association with outcomes are lacking.

We sought to explore STEMI outcomes in the modern PCI-era. Given the recognised under treatment of IH-STEMI patients, we compared the frequency of PCI and all-cause mortality trends between patients with PH-STEMI and IH-STEMI in a state-wide cohort within Australia.

2. Methods

2.1. Study population

Consecutive patients who were diagnosed with STEMI from 1-July-2008 to 30-June 2020 were identified from the NSW Admission Patient Data Collection (APDC) database (which includes >97% of NSW healthcare facilities) held by the Centre for Health Record Linkage (CHeReL). This is the largest health-related database in Australia and provides health linkage data on NSW residents.

2.2. Patient variables

The primary and secondary diagnoses associated with each STEMI patient were extracted from the APDC database. The APDC database include coding each diagnosis according to the International Statistical Classification of Diseases, Tenth-Revision Australian Modification (ICD-

* Corresponding author. for corresponding author. Cardiology Department, Concord Hospital, The University of Sydney, 1 Hospital Road, Concord NSW, 2139 Australia.
E-mail address: David.brieger@health.nsw.gov.au (D. Brieger).

https://doi.org/10.1016/j.ijcrp.2023.200214
Received 28 May 2023; Received in revised form 12 September 2023; Accepted 17 September 2023
Available online 21 September 2023

10AM) with STEMI defined as I21.0-I21.3. Patients with a primary diagnosis of STEMI were defined as PH-STEMI. To define our patient cohort who had a STEMI during an admission for a separate disorder (i.e. admitted with a primary diagnosis other than STEMI ICD-10AM codes I21.0-I21.3), we used the condition onset flag indicator coded in the APDC database for each admission to identify an in-hospital STEMI event (IH-STEMI) [12,15]. This helps distinguish a secondary diagnosis of STEMI that was coded as a background diagnosis from a prior event from our IH-STEMI event of interest. Additional variables extracted from the APDC database include age, sex, and comorbidities (Supplementary Table 1). We adopted a 2-year look-back period into the APDC database to ensure robust identification of comorbidities. PCI was defined as per the Australian Classification of Health Interventions (ACHI) procedural codes (Supplementary Table 1). STEMI patients who presented to a non-PCI-capable centre that were subsequently transferred to a PCI-capable centre for rescue PCI were included in our analysis. The cohort was split into 3-yearly time-based admission year-groups for temporal trend analysis (Year-group-1: 1-July-2008 – 30-June-2011; Year-group-2: 1-July-2011 – 30-June-2014; Year-group-3: 1-July-2014 – 30-June-2017; Year-group-4: 1-July-2017 – 30-June-2020).

2.3. Study outcome

The main outcome was all-cause mortality tracked from the NSW statewide death registry also held by CHeReL and linked to the APDC database. We separately examined cardiovascular mortality outcome. Cases were limited to NSW residents to minimise loss to follow-up through cross-border emigration. The end-of-study follow-up date was 30-December-2020. Approval of the study protocol was granted by the NSW Population and Health Services Research Ethics Committee, reference number: 2013/09/479.

2.4. Statistical analysis

All continuous variables are expressed as mean ± standard deviation (SD), unless otherwise stated, and categorical data as frequency and percentages. For binary variables, the Cochran-Armitage trend test was used to determine if there was a trend in the proportion across the periods. For continuous variables, univariable linear regression analysis was used to test whether the trend of the mean was linear. When comparing binary variables by PH-STEMI vs IH-STEMI, chi-squared test was used; and for continuous variables, univariable linear regression analysis was used.

Kaplan-Meier curves were used to present survival rates. Cox regression analyses were performed to assess the changes in all-cause and cardiovascular mortality during different year-groups while adjusting for confounders. Variables considered in the multivariable Cox model include age, sex, year-groups, PCI, prior history of heart failure (HF), myocardial infarction (MI), chronic kidney disease (CKD) and diabetes. To assess the relative contribution of PCI during the index STEMI admission on outcomes between different year-groups, the interaction between PCI and year-groups was tested, and if this interaction was significant the interaction term was included in the model.

All analyses were performed using SAS version 9.4 (SAS Institute, Cary NC, USA). A two-tailed probability value < 0.05 was considered statistically significant. All authors had full access to all the data in the study, and the corresponding author had final responsibility for the decision to submit for publication.

3. Results

3.1. Baseline characteristics

A total of 42 533 STEMI patients were identified; 96.7% were PH-STEMI. Compared to PH-STEMI, patients with IH-STEMI were older (76 ± 13 vs 66 ± 14yrs, $P < 0.0001$), more likely to be female (45.7% vs 29.0%, $P < 0.0001$) with higher rates of comorbidities including diabetes, CKD and atrial fibrillation (Table 1).

Common primary diagnoses for IH-STEMI patients include other circulatory diagnoses (including HF, cerebrovascular and vascular presentations) (13.1%), respiratory disease (12.1%), malignancy (11.3%) and sepsis (5.0%) (Supplementary Table 2). Acute coronary syndromes (ACS) other than STEMI comprised 4.7% and non-ACS ischemic heart disease presentations comprised 4.6%.

3.2. STEMI volumes and rates of PCI

The total number of STEMI presentations decreased overall from year-group 1 to year-group 4 in the PH-STEMI cohort (11 144–9638, $P < 0.0001$ for trend, Fig. 1). Conversely the number of STEMI presentation in the IH-STEMI cohort increased progressively over time from 176 in year-group 1 to 446 by year-group 4 ($P < 0.0001$ for trend, Fig. 1).

Patients with IH-STEMI were less likely to undergo PCI compared to patients with PH-STEMI during the study period (23.6% vs 64.0% respectively, $P < 0.0001$). Whilst the rate of PCI increased over time in both the PH-STEMI and IH-STEMI cohorts, rates remained lower for the former (14.8%–32.7% vs 56.1%–74.8% respectively from year-group 1 to year-group 4, Fig. 1).

In PH-STEMI patients who did not undergo PCI there was a progressive increase in comorbidities over time. These include prior bleeding (8.4% in year-group-1 vs 12.3% in year-group-4) and diabetes (16.9% in year-group-1 vs. 28.7% in year-group-4, Supplementary Table 3). Progressive increases in co-morbidities in IH-STEMI patients who did not undergo PCI were noted for diabetes (22.7% in year-group-1 vs 33.3% in year-group-4), Supplementary Table 4).

3.3. Study outcome

The in-hospital mortality rate was markedly higher in the IH-STEMI compared to the PH-STEMI cohorts (33.5% vs 8.4% respectively, $P < 0.0001$). There was no significant change over time for in-hospital death rates in either population (Fig. 1).

During a median follow-up of 7.9 years (interquartile range: 5.0–11.0 years), there were 11 291 deaths (27.4%) in PH-STEMI cohort compared to 899 deaths (64.8%) in the IH-STEMI cohort (Supplementary Fig. 1). In the PH-STEMI cohort, unadjusted mortality was significantly lower in later year-groups compared to the reference year-group 1 (range of hazard ratios [HR] 0.63–0.85 for year-group's 2–4 vs year-

Table 1
Baseline characteristics.

	Pre-Hospital STEMI N = 41 145	In-Hospital STEMI N = 1388	P value
Demographics			
Age, years	66 ± 14	76 ± 13	<0.0001
Female	11 929 (29)	645 (46.5)	<0.0001
Comorbidities			
Diabetes mellitus	8179 (19.9)	419 (30.2)	<0.0001
Current smoker	12 171 (29.6)	264 (19.0)	<0.0001
Ex-smoker	11 193 (27.2)	574 (41.5)	<0.0001
Atrial fibrillation	4641 (11.3)	431 (31.1)	<0.0001
Peripheral vascular disease	1622 (3.9)	214 (15.4)	<0.0001
Chronic kidney disease	2427 (5.9)	311 (22.4)	<0.0001
Prior stroke	666 (1.6)	90 (6.5)	<0.0001
Prior congestive cardiac failure	1479 (3.6)	154 (11.1)	<0.0001
Prior myocardial infarction	3412 (8.3)	102 (7.3)	0.21
Prior coronary revascularization	1206 (2.9)	47 (3.4)	0.32
Liver disease	492 (1.2)	88 (6.3)	<0.0001
Malignancy	2257 (5.5)	284 (20.5)	<0.0001
Dementia	825 (2.0)	93 (6.7)	<0.0001
Chronic Obstruction Pulmonary Disease	567 (1.4)	72 (5.2)	<0.0001
Prior bleeding	3193 (7.8)	364 (26.2)	<0.0001

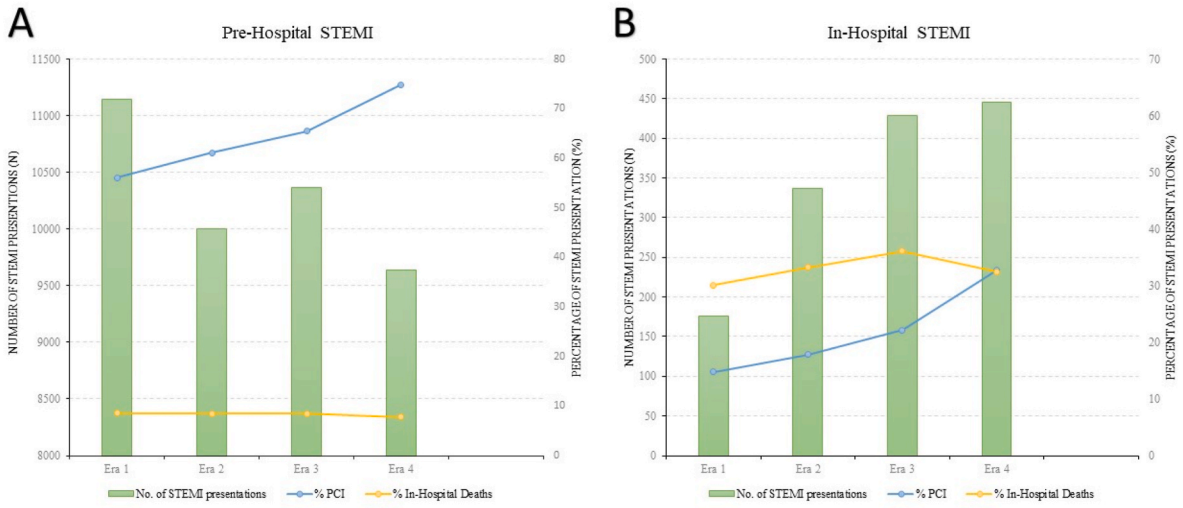


Fig. 1. Number of ST-elevation myocardial infarction (STEMI) presentations, rates of percutaneous coronary intervention (PCI) and In-Hospital mortality across year-groups.
Figures show an overall decrease in the number of STEMI presentations in a) PH-STEMI population, and a progressive increase in STEMI presentations in b) IH-STEMI population; and, increasing rates of PCI and stable in-hospital mortality from earliest year-group (2008–10) to most recent (2017–2019).

group 1) (Supplementary Fig. 2A). These trends were mirrored in cardiovascular mortality (Supplementary Fig. 2B). In the IH-STEMI population, there was no progressive improvement in unadjusted mortality, with year-group 3 having similar mortality risk to the reference year-group (Supplementary Fig. 2C). There were however trends towards improvement in cardiovascular mortality over time, though not significant ($p = 0.11$) (Supplementary Fig. 2D).

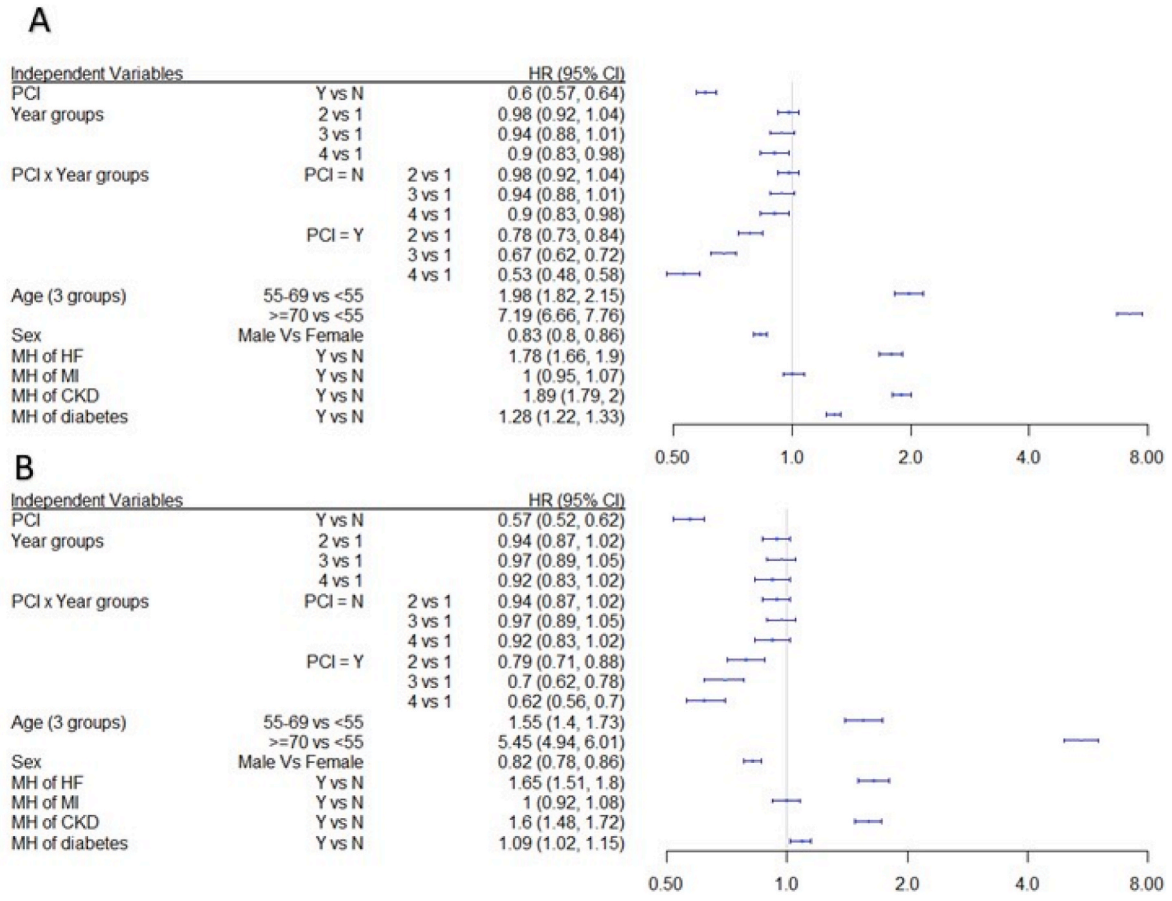


Fig. 2. Adjusted Cox regression analysis for all-cause and cardiovascular mortality in PH-STEMI cohort.
Figure shows the adjusted hazard ratio (HR) for all-cause and cardiovascular mortality and inclusion of interaction term "PCI x Year Group". Stratification across year groups showed a progressive fall in (A) all-cause and (B) cardiovascular mortality over time, most pronounced in those who underwent PCI, $P < 0.0001$.

3.4. Impact of PCI on all-cause mortality following STEMI

PCI was independently associated with a reduction in mortality in the PH-STEMI subgroup (adjusted hazard ratio [aHR] = 0.60, p < 0.001). Stratification by PCI showed a significant progressive fall in mortality risk for PH-STEMI patients who underwent PCI (aHR = 0.78, 95% CI = 0.73–0.84 for year-group 2 vs year-group 1, compared to aHR = 0.53, 95%CI = 0.48–0.58 for year-group 4 vs year-group 1, P < 0.0001). There was also an improvement in mortality, albeit less pronounced, in patients who did not undergo PCI, (aHR = 0.98, 95% CI = 0.92–1.04 for year-group 2 vs year-group 1, compared to aHR = 0.90, 95% CI = 0.83–0.98, for year-group 4 vs year-group 1, P < 0.0001, Fig. 2A). Similar findings were noted for cardiovascular mortality (Fig. 2B).

For the IH-STEMI subgroup, PCI showed a trend toward improvements in mortality but this was not significant (aHR = 0.85, p = 0.51). When stratified by PCI, there was a reduction in mortality risk in those who underwent PCI in later year-groups (aHR = 0.74, 95% CI = 0.43–1.3 for year-group 2 vs year-group 1, compared to aHR = 0.45, 95% CI = 0.27–0.77 for year-group 4 vs year-group 1), but not among those who did not have PCI (p value for interaction 0.048) (Fig. 3A). There was no significant interaction between PCI and year-groups with regards to cardiovascular mortality in the IH-STEMI cohort (p = 0.12). Nevertheless, PCI was associated with lower risk for cardiovascular death (aHR = 0.75, 95% CI = 0.57–0.98, p = 0.04) (Fig. 3B).

4. Discussion

This statewide, observational study including over 42 000 STEMI patients describes the incidence and characteristics of PH-STEMI and IH-STEMI patients within Australia in the modern PCI-era. We confirm the rate of PCI use increased over time in both the PH-STEMI and IH-STEMI cohorts, but rates remained consistently lower for the former. There was a progressive decrease in long-term all-cause and cardiovascular mortality over time in the PH-STEMI population, which is at least in part due to a change in PCI over time. While there were no significant improvements in either all-cause or cardiovascular mortality over time in the IH-STEMI cohort, there was a progressive reduction in all-cause mortality among patients undergoing PCI.

4.1. Incidence and characteristics of IH-STEMI vs. PH-STEMI

We confirm a low incidence of IH-STEMI patients, representing 3.4% of the total STEMI cohort. The incidence of IH-STEMI varied from 1 to 20% of total STEMI populations in the literature [16–19] with the largest previous study reporting an incidence of 4.9% [12]. A lack of standardised definitions for IH-STEMI likely contributes to the variability in published rates. Some studies, including ours, included inpatients who had a primary ACS diagnosis other than STEMI [18,20], whilst others only included patients admitted with a non-ACS diagnosis [12]. Consistent with prior studies, our IH-STEMI cohort was older, more likely female and was significantly more co-morbid compared to the PH-STEMI cohort [12,14,18,20,21].

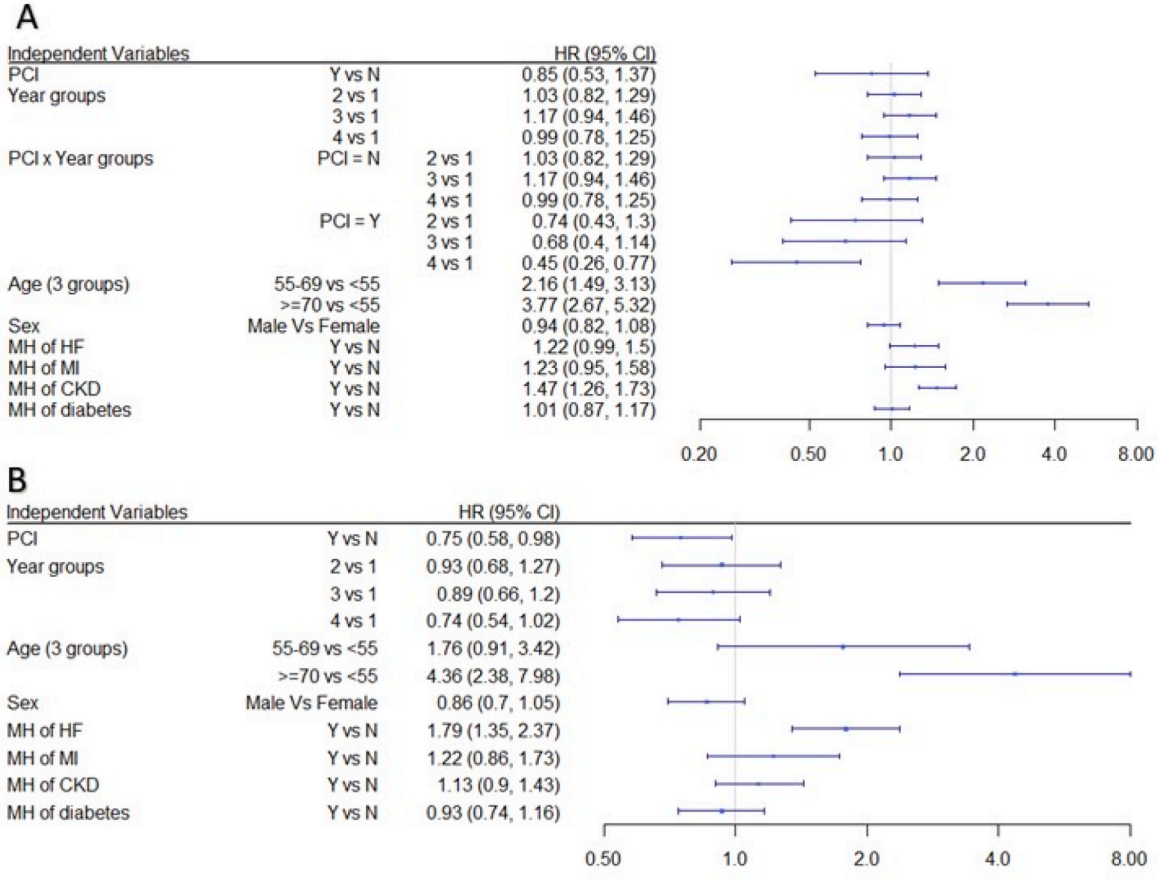


Fig. 3. Adjusted Cox regression analysis for all-cause and cardiovascular mortality in IH-STEMI cohort.
Figure shows the adjusted hazard ratio (HR) for all-cause (A) and cardiovascular mortality (B) respectively in the IH-STEMI cohort. There was a significant interaction between 'PCI and Year-Group' for all-cause mortality (p = 0.048) with significant improvements noted overtime in patients who underwent PCI. There was no significant change in cardiovascular mortality over time (p = 0.11).

4.2. Rates of PCI and uptake within each cohort

Consistent with local and international literature, we observed progressive increase in the rate of PCI use for STEMI over time [2,3,9]. Reasons contributing to this are firstly, an increase in cardiac catheterisation facilities with PCI capabilities throughout metropolitan and regional NSW during the study period. Secondly, the utilisation of pre-hospital assessment for primary PCI whereby patients with STEMI diagnosed by ECG in the ambulance are taken directly to a primary PCI hospital, has increased from 2 to 11 sites across metropolitan NSW [6]. Thirdly, national community education programs have been shown to increase ambulance calls for chest pain [5].

The increased uptake of PCI over time was observed in both cohorts. Rates of PCI within the PH-STEMI cohort in the most recent year-group was 74.8%; similar populations internationally have reported rates of 80% [2]. This mild treatment gap may in part be driven by the geographically vast area of NSW (>800 000 km^2), the majority of which are regional and rural areas not serviced by primary PCI-capable facilities. Consistent with prior literature, PCI rates remained significantly lower in the IH-STEMI cohort [12,14,20]. Increased age and comorbidities, noted in our IH-STEMI cohort, other intercurrent illness leading to hospital admission and marked delays in diagnosis, noted in prior studies, all contribute to increased procedural risk [13,16] and act as barriers to timely reperfusion [14]. Furthermore, the strategies outlined above focus on increasing the utilisation of PCI amongst PH-STEMI patients rather than IH-STEMI.

4.3. Outcomes

4.3.1. In-hospital mortality

In-hospital mortality in our PH-STEMI population (8.4%) is comparable to a recently published US registry study (8.1%) [2] with factors such as left ventricular dysfunction and residual myocardial ischemia contributing to short-term prognosis. Similar to other studies, our IH-STEMI cohort had significantly higher in-hospital mortality compared with PH-STEMI patients, with more than 3-fold greater risk (33.5%) [12,18]. Contributing factors to this disparity likely include a sicker cohort based on their baseline characteristics, intercurrent illness [12], delays in recognition and management and higher rates of bleeding complications and cardiogenic shock [16,17,22]. International populations have reported initial improvements followed by a plateau [2], or a similar lack of improvement in in-hospital mortality [23,24] as seen in both our cohorts.

4.3.2. Long-term mortality

A progressive improvement in unadjusted long-term, all-cause and cardiovascular mortality was noted in the PH-STEMI cohort. These improvements have been noted previously, and in contrast to factors driving short-term prognosis, long-term prognosis is more likely reflecting improvements in access to reperfusion strategies and secondary prevention in altering the progression of atherosclerotic disease [24–27]. In the IH-STEMI cohort neither unadjusted all-cause nor cardiovascular mortality showed progressive improvements. Rather, a slight rise in mortality was noted in year-group 3 with improvements seen in the year-groups before and after. The smaller numbers within this cohort do suggest that these trends should be interpreted with caution.

4.3.3. The impact of PCI on mortality

In adjusted analyses among PH-STEMI patients, there was a progressive reduction in all-cause and cardiovascular mortality over time from the earliest to the most recent year-group. This trend was most pronounced in the patients who underwent PCI. The significant interaction between 'PCI and year-group' suggests that at least in part the improvements in outcomes over time are driven by PCI. The reasons for this may include an improvement in the quality and availability of PCI resulting in shorter door-to-balloon times and use of newer generation stents. Improved pharmacotherapy, and access to cardiac follow-up reviews with a focus on tight cardiac risk factors control are also likely drivers in improving mid to long-term outcomes both in patients who did and did not undergo PCI. Whilst lipid-lowering therapy is not reported in our study, it has been well documented to improve long-term outcomes in STEMI patients with recent studies suggesting that further risk-stratification and more aggressive escalation in lipid-lowering therapy is important in achieving target risk factor control [28,29].

There was a significant interaction between 'PCI and year-group' for all-cause mortality in the IH-STEMI population, showing that those who underwent PCI in this sub-group had similar favourable impacts as those seen in the PH-STEMI patients who underwent PCI. There was no similar interaction noted for cardiovascular mortality, however it is likely that the smaller numbers of patients in this subgroup precluded us from identifying such an association should it exist.

4.4. Clinical implications

Long-term mortality in the IH-STEMI population remained approximately double that of the PH-STEMI cohort throughout the study period with consistently lower utilisation of PCI. Other studies have suggested that improvements in early diagnosis via early ECG acquisition, early STEMI protocol activation as well as education programs are key to improving outcomes in this cohort [13,16]. Clearly this is a unique and complex population and further research is needed to identify strategies to improve outcomes.

4.5. Limitations

Being an observational study derived from administrative dataset, there are several limitations of this study. Diagnosis, comorbidities and outcomes may be susceptible to coding inaccuracies. However, public validation studies using the same database indicated >95% agreement between audited comorbidities and hospital discharge coding [30,31]. Similarly, non-captured deaths are estimated to be <0.02% based on known migration rates in the study period. Given the fact that our cohort had large numbers, small changes in demographics and comorbidities over time can lead to statistically significant but not clinically important temporal changes. Furthermore, factors such as time to PCI, use of other therapies such as dual anti-platelet therapy, thrombolysis, risk factors control, and device therapy were not included in the APDC dataset and may all contribute to changes in outcome over time.

5. Conclusion

Rates of PCI increased steadily in NSW from 2008 to 2020 and were associated with improvements in all-cause and cardiovascular mortality in the PH-STEMI population. IH-STEMI patients had lower rates of PCI and significantly higher rates of in-hospital and long-term mortality. Although a small proportion of the total STEMI population, further efforts should be directed towards addressing the poorer outcomes in IH-STEMI patients.

Credit author statement

S Ratwatte: Conceptualization, Methodology, Writing – original draft, Writing – review & editing. A Ng: Methodology, Data curation, Supervision, Writing – review & editing. K Hyun: Data curation, Formal analysis, Methodology, Writing – review & editing. R Philip: Formal analysis, Data curation. F Boroumand: Formal analysis, Data curation. C Weber: Data curation, Formal analysis, Methodology. L Kritharides: Writing – review & editing, Supervision. D Brieger: Conceptualization, Methodology, Formal analysis, Supervision, Writing – review & editing

Funding

SR has received an Emerging Cardiovascular Researcher Education Scholarship from the Heart Research Institute and a PhD Scholarship from the National Heart Foundation (106796).

KH is funded by the National Health and Medical Research Council Investigator Grant Emerging Leader 1 (GNT1196724).

CW has received a Postgraduate Scholarship from the National Health and Medical Research Council of Australia Centre of Research Excellence in Cardiovascular Outcomes Improvement (#111170).

Declaration of competing interest

All authors declare that they have no conflict of interest and have nothing to disclose.

Acknowledgements

We gratefully acknowledge the Centre for Health Record Linkage for data linkage services and the NSW Ministry of Health for supplying data for these analyses. No sponsors had a role in study design, data collection, data analysis, data interpretation, or writing of the report. All authors had full access to all the data in the study, and the corresponding author had final responsibility for the decision to submit for publication.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcrp.2023.200214.

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