Age-specific gender differences in long-term recurrence and mortality following incident myocardial infarction: a population-based study

Short title: Gender differences in myocardial infarction outcomes

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INTRODUCTION

Coronary heart disease (CHD) contributes significantly to the burden of morbidity and mortality in the general population [1]. Despite improvements in short and long-term survival following a myocardial infarction (MI) over recent decades [2,3], the risk of a subsequent MI or death remains elevated [4]. There is evidence of gender differences in survival, with women reported to have higher short-term mortality rates [5], however there are less data available on gender differences in long-term outcomes. It has been suggested that age and a greater prevalence of comorbidities may be associated with this apparent difference [5,6]. More recent reports suggest that the burden of adverse outcomes is also evident in younger women who experience an MI [6,7]. Given reports of an increase in the incidence of MI in younger people [8], and specifically in younger women in WA [9], this question warrants investigation in an Australian context to determine whether age-specific gender disparities exist.

Significant gaps still remain in the delivery of guideline-recommended levels of secondary prevention measures post-MI [10], and age and gender are important variables in determining the risk of future cardiovascular events in CHD patients. It is therefore imperative that age-specific gender outcomes are described to ascertain particularly high-risk target groups for enhanced secondary prevention measures. Thus the study aim was to determine the age-specific impact of gender on long-term MI recurrence and mortality in 30-day survivors of incident MI in a population-based setting.

METHODS

Data source

Data for this study were obtained from two of the core datasets of the WA Data Linkage System (WADLS) - the Hospital Morbidity Data Collection (HMDC) and Death Register. These data are linked centrally by the WADLS using probabilistic matching, with >99% accuracy for this process [11]. The majority of acute coronary care and all invasive revascularisation procedures are undertaken in the tertiary hospitals (public and private) situated in the capital city, Perth. The person-based linked dataset available for this study contained all records for any patient hospitalised with or dying from cardiovascular disease (CVD) in WA from 1985-2010. Variables available included demographic information, principal discharge diagnosis, 20 secondary discharge diagnosis fields, and inpatient procedures. Discharge pharmacy data was available for incident MI cases admitted to the three adult tertiary hospitals, and linked to the matching hospital admission by a unique admission identifier. The data used in this study are de-identified, and the study was granted a waiver of informed consent from each ethics committee. Approval for this study was obtained from the ethics committees of The University of Western Australia and the WA Department of Health.

Incident MI cohort

All MI cases hospitalised in WA from 2003 to 2009 were identified using the principal discharge diagnosis field (ICD-10-AM I21,I22). Cases were classified as incident if there were no hospitalisations for acute coronary syndromes in the 16 years prior to the MI.

admission [12]. Patients aged 35 to 84 years of age who survived greater than 30 days following the incident MI, were included in the cohort.

Patient characteristics

Comorbidities were identified from the linked dataset if recorded in the 16 years prior to or on the incident admission. These included: hypertension (ICD-10-AM I10-I15 and ICD-9-CM equivalent), diabetes (E10-E14), heart failure (I50), atrial fibrillation (I48), stroke (I60-I64), peripheral arterial disease (I70-I79), and chronic kidney disease (CKD)[13]. History of coronary heart disease (CHD) was identified where there was prior hospitalisation for stable angina or other CHD (I20.1-I20.9, I24-I25). Revascularisation procedures (percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG)) occurring during the incident episode (including following transfer to a metropolitan hospital) were identified from any of the 11 procedure fields. Utilisation of evidence-based medications following MI was determined by identifying drugs dispensed at discharge for tertiary hospital patients with a length of stay greater than one day and at least one pharmacy record in the dataset. The analysis was restricted to this sample of patients because discharge pharmacotherapy data are only available for linkage for tertiary hospital patients. The drug groups analysed were antiplatelet therapy (eg, aspirin), angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin II receptor blockers (ARBs), lipid-lowering drugs, beta blockers, and calcium channel blockers. Aspirin use in pain management was excluded by checking the directions on prescriptions for aspirin 300mg. Drugs dispensed as part of a clinical trial were excluded.

Outcomes

The endpoints of recurrent MI, CVD mortality and all-cause mortality were identified.

Recurrent MI was identified where recorded in the principal discharge diagnosis field >30 days following the incident hospitalisation. CVD deaths were identified where the underlying cause of death was coded as any cardiovascular-related cause (ICD-10-AM I00-I99). Follow-up data were available to 30th June 2011 for all patients, providing minimum and maximum follow-up periods of 18 months and 8.5 years respectively.

Statistical Analyses

Patient characteristics and discharge pharmacotherapy are presented separately for men and women. Differences in the proportion of men and women dispensed drugs from each category were compared by chi-squared tests. Unadjusted risks were derived from Kaplan Meier survival curves. Time to event was calculated from the date of the incident MI hospitalisation to the date of the event (MI, CVD death and all-cause death), or censored at the end of followup or at an intervening event (death for recurrent MI endpoint or non-CVD death for the CVD death endpoint), whichever came first. The log-rank p-value test was used to compare men and women within each age group.

Cox regression models were used to examine the effect of gender on the risk of recurrent MI or death, stratified by age group. The proportional hazards assumption was tested using an interaction term between sex and time. No consistent evidence of violation of this assumption was found. In addition to the primary analysis where the full follow-up available for each patient was used, a further analysis was carried out using a fixed follow-up of 2 years. Additionally, we performed landmark analyses at 2 and 4 years, where patients were only included if alive and event-free at these time points. Age-adjusted (continuous variable) and multivariate models were run for each of these analyses. Variables entered into the

multivariate models included age (continuous and with it's square where needed), indigenous status, diabetes, CKD, hypertension, atrial fibrillation, stroke, peripheral arterial disease, CHD, MI type (ST-segment elevation MI (STEMI), non-ST segment elevation MI (NSTEMI) or unspecified type, as recorded in the linked dataset), and PCI or CABG during the incident episode. For the landmark analyses, binary variables indicating revascularisation during the followup period but before the landmark time point were also included. Results are reported as hazard ratios (HR, 95% confidence interval, CI). All analyses were carried out using SAS v9.4 (Carey, USA), and the statistical significance level set at $p < 0.05$.

RESULTS

There were 12,420 30-day survivors of incident MI from 2003 to 2009. Mean follow-up time was 4.05 years (SD 2.2 years). Males comprised 71.2% of the study cohort (Table 1). 28.6% of men and 50.6% of women were in the 70-84 year age group. The mean age for men was 61.7 years (SD 11.9) and 67.4 years (SD 12.1) in women. There was a higher proportion of indigenous patients in women, with the greatest disparity in the 35-54 year age group (19.8% in women versus 9.3% in men). There was a higher prevalence of diabetes, hypertension and heart failure recorded for women compared with men within the three age groups.

Pharmacotherapy at hospital discharge

Of the total cohort, 5763 (46.4%) patients were admitted to a tertiary hospital with a length of stay greater than one day and had a discharge pharmacy record, and were therefore included in the analysis. A greater proportion of older patients had no pharmacy record at discharge, particularly females. Coverage was greatest for lipid-lowering drugs in both men (87.7%) and

women (77.9%), though in younger women coverage was greatest for low-dose aspirin (86.5%; Table 2). Chi-squared comparisons within each age group and overall revealed a statistically significant lower proportion of women than men receiving statins or other lipid-lowering drugs, aspirin, ACEI/ARB, and beta blockers.

Long-term survival

Unadjusted risk of recurrent MI and CVD mortality, stratified by age group, are presented in Figures 1 and 2 respectively. Table 3 shows risk estimates by gender and age group for each outcome. The 8-year risk of recurrent MI was higher in women (14.9% versus 11.6% in men, log-rank $p=0.04$), with no significant difference between men and women in any of the age groups, although rates were nearly 4% higher in women than men in the 55-69 year age group (Table 3). There was a larger disparity for cardiovascular death (women 17.6%, men 9.7%, log-rank $p<0.0001$), with a significantly higher unadjusted risk of cardiovascular mortality in women than men in the 35-54 year age group (log-rank $p=0.003$). This difference persisted in women in the older age groups, although the differences were smaller and not statistically significant. The proportion of all deaths attributed to cardiovascular causes was similar between men and women in each age group (35-54 years, 45.0% versus 44.7%; 55-69 years 36.8% versus 37.2%; 70-84 years 48.2% versus 46.0% respectively).

Multivariate adjustment

After multivariate adjustment, the hazard ratio comparing women to men for recurrent MI was generally less than one, although only statistically significant in the 35-54 year age group (HR 0.66, 95% CI 0.47, 0.94). This difference was also apparent in the fixed 2-year

followup. There was generally little difference between men and women for CVD mortality in the fully adjusted model. However, there was a tendency towards increased rates in 35-54 year old women (age-adjusted HR 2.08, 95% CI 1.26, 3.42) which was diminished after adjustment for comorbidities and demographic factors (HR 1.28, 95% CI 0.77, 2.15). A similar pattern was seen in each of the age groups when all-cause mortality was the endpoint. In 35-54 and 55-69 year olds, the variables mainly responsible for attenuating the rates between the age-adjusted and multivariate models were diabetes, heart failure, chronic kidney disease and indigenous status. There was little attenuation between the age-adjusted and multivariate models for the 70-84 year age group.

The landmark analyses which included 2- and 4-year survivors respectively demonstrated similar hazard ratios for each of the endpoints as for the full cohort, although the reduced rate of recurrent MI in 35-54 year old women versus men was no longer statistically significant (data not shown).

DISCUSSION

This study provides important data on gender differences in long-term recurrence and mortality following incident MI in an Australian population-based setting. It affords a unique perspective because there is limited data on long-term outcomes in this high-risk patient group nationally. Our study shows that in patients who survive the acute phase of a first-ever MI, women have a higher unadjusted risk of recurrent MI and cardiovascular mortality than men up to 8 years following the incident event. The disparity between men and women for recurrent MI is underpinned by the higher risk in 55-69 year old women, whereas a higher risk of CVD death in women is evident across all age groups. These differences were generally attenuated after multivariate adjustment, with the adjusted rate for recurrent MI

being lower although not significantly so in women versus men, indicating the impact of increased CVD risk factors and comorbidities in women on long-term outcomes.

Comparisons of mortality outcomes:

Previous studies have shown worse outcomes in women than men following MI [5,14,15]. Higher all-cause mortality at 1-year in women aged <50 years [6], and at 2-years in women aged <60 years [7] has been demonstrated to persist even after adjustment for demographic characteristics, comorbidities and early treatment. However Griffiths et al in a single-centre study reported increased long-term mortality in women at 7 years following MI which was accounted for by these factors [16]. A systematic review of five to ten year mortality outcomes following MI concluded that differences in age, comorbidities and treatment are responsible for the higher mortality risk in women even with very long-term followup, although there was significant heterogeneity in the magnitude of risk [17]. Our results are consistent with these findings, although we were unable to fully adjust for in-hospital drug treatment during MI admissions.

Apparent gender differences may also be related to differing MI severity within study populations. The cohort of women in some studies may represent a higher-risk female population [18] with higher rates of in-hospital complications [17]. Higher case-fatality in women may reflect these factors [18,19], and therefore exclusion of early deaths from our study cohort would limit the impact of this factor. The similar findings of the landmark analyses in our study indicate that the impact of comorbidities and treatment at baseline persist into the longer term, with limited impact of subsequent revascularisation on rates.

Higher unadjusted CVD mortality risk in women versus men persisted across age groups, with similar disparities for all-cause mortality. Although a lower risk of CVD death may be expected in younger women than men, we found that women in the 35-54 year age group had a 5% absolute higher risk of CVD death than the corresponding men, and a doubling of risk which remained elevated, although not significantly so, after multivariate adjustment. Our results are in concordance with those from a national inpatient registry in Sweden, with evidence of a higher risk of all-cause mortality at 4 years following MI in 25-54 year old women versus men [20]. However, men in this age group were more likely to have a CVD-related death than women (55.4% vs 34.1% respectively). This is in contrast to our study, where the proportion of CVD deaths was similar between men and women across all age groups. This implies that the mortality differences in our study are not driven by a greater proportion of non-CVD deaths in women relative to men, and highlights the importance of addressing vascular risk, even in younger women with CHD.

The multivariate analyses in our study show that the poorer long term survival in women may be associated with the higher comorbidity burden relative to men. Indigenous status, diabetes, heart failure and chronic kidney disease appeared to explain much of the increased risk in women for recurrent MI and mortality in the 35-54 and 55-69 year age groups, although the impact of indigenous status was greater in the younger age group. Although excluding indigenous people from the analysis reduced the disparity in absolute risk to a greater degree in 35-54 versus 55-69 year olds, further analysis showed that diabetes attenuated adjusted rates in the youngest age group independent of indigenous status. This is of importance, as over a quarter of women in the 35-54 year age group in our study had diabetes and an adverse risk factor profile in younger women with diabetes is associated with relatively higher levels of all-cause mortality [21].

The use of evidence-based medications, including beta-blockers, ACEI/ARBs, statins and antiplatelet drugs, is associated with improved survival in patients with CHD [22]. Although there were reasonably high levels of dispensing of evidence-based drugs at discharge in our study, men had higher levels of dispensing across all age groups compared with women. Evidence suggest that this disparity is one of the factors which contributes to increased long-term risk in women [14,22]. Hence, it is likely that this level of drug uptake is not maintained in the long-term. Use of evidence-based drugs is lower in community-based CHD patients compared with patients following an acute event [23]. Yusuf et al reported that women and younger people are less likely to take medication in a community setting, and that use of antiplatelet therapy, statins and ACEI/ARBs decreases significantly with increasing time after index event [24]. Women with CHD are less likely to be prescribed statins in the primary care setting, despite a higher prevalence of hyperlipdaemia [25]. It has also been reported that three-quarters of all acute coronary syndrome patients don't receive optimal evidence-based secondary prevention, including referral to rehabilitation and lifestyle modification, by the time of hospital discharge [10]. These issues may all contribute to the gender differences reported. Whether these patterns are related to patient adherence or system-level issues such as formal pathways for medical follow-up and rehabilitation requires explanation. It is also possible that because increasing age is a known risk factor for cardiovascular events, less aggressive cardiovascular management is provided to younger women, even in the presence of existing CHD.

Strengths and Limitations

The strength of our study lies in the ability to capture all incident MI's occurring in a whole-population, and in the availability of complete long-term follow-up data for this cohort.

Because of the person-based record linkage available in WA and long hospitalisation history available for this study, we were able to accurately identify first-ever MI cases and capture comorbidity history in this population. However, linked administrative data has inherent limitations which need to be considered. These include the accuracy of recording of MI in hospital morbidity data, which has been validated in our population (sensitivity 74%, positive predictive value 94% against American Heart Association epidemiological criteria) [26]. Additionally, data for baseline clinical indicators and in-hospital medications are not available in the administrative datasets. We also did not have data related to continued use of pharmacotherapy and other secondary prevention measures following hospital discharge in these patients. Nor was there any data on preadmission pharmacotherapy, so we did not have any information on drugs used prior to admissions. The use of increasingly sensitive troponin assays in MI diagnosis during the study period could lead to an increasing proportion of lower severity MI cases in our cohort, which is unlikely to lead to a higher risk of CVD mortality in women. Other potential confounders of age-specific gender disparities such as smoking, diet, psychosocial factors and socioeconomic status were not able to be accounted for in our analyses, and therefore residual confounding for the relationship of age and gender with outcomes may be present.

Conclusion

This study contributes to the limited data on gender differences in long-term outcomes following MI in an Australian setting. It highlights the elevated cardiovascular risk in women compared with men over the long-term, and that this difference is not limited to older women. These differences are apparent despite reasonable, although not optimal, levels of dispensing of evidence-based drugs at discharge from hospital. These data demonstrate that

the need for ongoing secondary prevention is imperative, and that measures should not be restricted to the early period following an acute event. In particular, health professionals should be aware that women, even in the younger age group, have a high CVD risk factor and comorbidity burden and remain at high risk of adverse outcomes over an extended period of time.

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Table 1. Patient characteristics stratified by gender and agegroup.

	35-54 years		55-69 years		70-84 years		Total (35-84 years)	
	Men (n=2639)	Women (n=627)	Men (n=3672)	Women (n=1141)	Men (n=2527)	Women (n=1814)	Men (n=8838)	Women (n=3582)
Mean age, years (SD)	47.5 (5.0)	47.4 (5.0)	61.7 (4.3)	62.5 (4.2)	76.4 (4.1)	77.4 (4.2)	61.7 (11.9)	67.4 (12.1)
Indigenous status	9.3	19.8	2.5	6.2	1.0	1.2	4.1	6.0
Acute PCI	60.7	41.0	56.1	42.9	37.4	31.0	52.1	36.5
Acute CABG	3.3	2.4	5.9	4.1	6.3	2.7	5.2	3.1
Comorbidities								
CHD	5.1	8.0	9.8	9.4	21.8	18.7	11.8	13.8
Diabetes	16.4	26.9	21.6	27.3	26.2	28.2	21.4	27.7
Chronic kidney disease	3.8	10.7	6.0	11.0	16.5	13.8	8.3	12.4
Hypertension	36.5	43.7	47.9	59.1	63.6	71.7	49.0	62.8
Heart failure	4.4	7.8	8.4	11.9	24.0	27.9	11.7	19.3
Stroke	0.8	0.8	1.9	4.0	6.4	6.3	2.8	4.6
Atrial fibrillation	3.5	3.0	9.2	9.0	24.8	25.5	12.0	16.3
Peripheral arterial disease	1.9	2.9	5.4	6.5	14.8	10.2	7.0	7.8

Table 2. Proportion (%) of patients who received pharmacotherapy on discharge from hospital in a subset of 30-day survivors of incident myocardial infarction in Western Australia (patients admitted to a tertiary hospital and with a length of stay >1 day), n=5763.

	35-54 years		55-69 years		70-84 years		35-84 years	
	Men (n=1247)	Women (n=259)	Men (n=1756)	Women (n=532)	Men (n=1162)	Women (n=807)	Men (n=4165)	Women (n=1598)
Statin and other lipid-lowering drugs	92.5	85.7 [†]	89.2	84.4 [†]	80.5	71.1*	87.7	77.9*
Aspirin (low dose)	91.4	86.5 [†]	87.7	82.3 [†]	79.0	70.3*	86.4	76.9*
Other antiplatelet drugs	86.8	82.2	85.0	75.4*	70.9	67.5	81.6	72.5*
ACEI/ARB	89.0	79.2*	84.5	78.6 [†]	74.2	69.8 [†]	83.0	74.2*
Beta blocker	88.8	80.3 [†]	86.4	77.1*	77.1	68.2*	84.5	73.1*
Calcium channel blockers	4.2	5.0	5.4	11.1*	10.8	13.8	6.6	11.5*

*p<0.0001, †p<0.05, from chi-squared test comparing men and women.

ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin II receptor blockers.

Table 3. Estimates from Kaplan-Meier curves for the risk of recurrent myocardial infarction, cardiovascular disease mortality and all-cause mortality following incident myocardial infarction.

Age group, years	Follow-up time, years	Event risks, % (95% CI)					
		Myocardial infarction		Cardiovascular disease mortality		All-cause mortality	
		Men	Women	Men	Women	Men	Women
35-54	2	4.5 (3.7, 5.3)	3.4 (2.0, 4.9)	1.0 (0.6, 1.3)	1.8 (0.8, 2.8)	2.1 (1.6, 2.6)	3.5 (2.1, 5.0)
	4	6.9 (5.9, 8.0)	6.0 (4.1, 8.1)	1.5 (0.1, 2.0)	2.9 (1.5, 4.3)	4.0 (3.3, 4.7)	6.9 (4.8, 9.1)
	8	11.0 (9.3, 12.8)	11.2 (6.8, 15.5)	3.3 (2.0, 4.6)	8.3 (2.1, 14.5)*	8.1 (6.3, 9.9)	14.4 (8.0, 20.8) [†]
55-69	2	3.8 (3.2, 4.4)	4.6 (3.4, 5.8)	1.7 (1.3, 2.2)	1.6 (0.8, 2.3)	4.4 (3.8, 5.0)	4.8 (3.6, 6.1)
	4	5.8 (5.0, 6.6)	6.5 (5.0, 8.1)	2.4 (1.9, 3.0)	2.9 (1.8, 4.0)	7.4 (6.7, 8.2)	8.4 (6.6, 10.1)
	8	9.4 (7.9, 10.9)	13.2 (8.9, 17.6)	4.5 (3.5, 5.3)	6.9 (4.3, 9.6)	13.9 (12.1, 15.6)	16.3 (12.6, 20.1)*
70-84	2	7.8 (6.7, 8.9)	7.2 (6.0, 8.5)	8.3 (7.2, 9.4)	8.7 (7.4, 10.1)	16.1 (15.0, 17.2)	15.9 (14.2, 17.6)
	4	11.1 (9.8, 12.5)	10.1 (8.6, 11.7)	14.6 (13.0, 16.1)	13.3 (11.6, 15.0)	27.2 (25.8, 28.7)	27.1 (24.9, 29.3)
	8	16.0 (13.5, 18.5)	17.4 (14.1, 20.7)	25.6 (22.8, 28.5)	28.0 (24.0, 32.2)	49.3 (46.7, 51.8)	50.7 (46.8, 54.5)
35-84	2	5.1 (4.6, 5.6)	5.7 (4.9, 6.5)	3.3 (2.9, 3.7)	5.1 (4.4, 5.8)	6.9 (6.4, 7.5)	10.3 (9.3, 11.3)
	4	7.6 (7.0, 8.2)	8.2 (7.2, 9.2)	5.5 (5.0, 6.0)	8.1 (7.1, 9.0)	11.8 (11.1, 12.6)	17.7 (16.3, 19.0)
	8	11.6 (10.6, 12.7)	14.9 (12.6, 17.2)*	9.7 (8.7, 10.6)	17.6 (15.1, 20.1) [†]	21.2 (19.8, 22.6)	34.1 (31.4, 36.8) [†]

Log-rank p-value comparing men and women, *p<0.05, [†]p<0.0001.

Table 4. Age and multivariate-adjusted hazard ratios (95% CI)* for the rates of recurrent myocardial infarction, cardiovascular disease mortality and all-cause mortality in women compared with men in 30-day survivors of incident myocardial infarction.

Follow-up	Age group, years	Hazard ratios (95% CI) [†]					
		Myocardial infarction		Cardiovascular disease mortality		All-cause mortality	
		Age adjusted	Multivariate	Age adjusted	Multivariate	Age adjusted	Multivariate
All [‡]	35-54	0.90 (0.65, 1.26)	0.67 (0.48, 0.95)	2.10 (1.28, 3.46)	1.30 (0.77, 2.19)	2.13 (1.52, 2.98)	1.29 (0.91, 1.84)
	55-69	1.20 (0.94, 1.54)	1.03 (0.79, 1.32)	1.19 (0.83, 1.70)	0.84 (0.58, 1.21)	1.17 (0.94, 1.45)	0.87 (0.70, 1.09)
	70-84	0.90 (0.74, 1.08)	0.89 (0.74, 1.08)	0.86 (0.73, 1.00)	0.86 (0.74, 1.01)	0.92 (0.83, 1.02)	0.92 (0.82, 1.02)
	All (35-84)	0.98 (0.85, 1.12)	0.88 (0.77, 1.01)	0.95 (0.83, 1.09)	0.91 (0.79, 1.05)	1.02 (0.93, 1.12)	0.96 (0.87, 1.05)
2 years	35-54	0.75 (0.47, 1.19)	0.54 (0.34, 0.88)	1.95 (0.95, 3.98)	1.39 (0.66, 2.94)	2.04 (1.23, 3.39)	1.39 (0.81, 2.38)
	55-69	1.19 (0.87, 1.65)	0.99 (0.71, 1.38)	0.86 (0.51, 1.47)	0.62 (0.36, 1.07)	1.06 (0.78, 1.44)	0.80 (0.59, 1.10)
	70-84	0.87 (0.69, 1.09)	0.84 (0.67, 1.07)	0.92 (0.74, 1.14)	0.95 (0.77, 1.19)	0.89 (0.76, 1.04)	0.90 (0.77, 1.05)
	All (35-84)	0.93 (0.78, 1.11)	0.83 (0.69, 0.98)	0.96 (0.79, 1.16)	0.93 (0.77, 1.13)	0.96 (0.84, 1.10)	0.92 (0.80, 1.05)

*Adjusted for 5-year agegroup, indigenous status, diabetes heart failure, hypertension, atrial fibrillation, stroke, peripheral arterial disease, chronic kidney disease, coronary heart disease, acute percutaneous coronary revascularisation, acute coronary artery bypass grafting and myocardial infarction type.

[†]Reference level for each agegroup comparison is male gender.

[‡]Maximum follow-up for the whole cohort 30 June 2011.

Figure legends:

Figure 1. Kaplan Meier curves for recurrent myocardial infarction following an incident myocardial infarction in men (A) and women (B), stratified by age group.

Figure 2. Kaplan Meier curves for cardiovascular disease mortality following an incident myocardial infarction in men (A) and women (B), stratified by age group.

Fig.1A

Fig. 1B

Fig. 2A

Fig. 2B