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Why have temporal trends in STEMI and NSTEMI incidence and short-term mortality changed in recent years? A nationwide 35-year cohort study in Iceland

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ABSTRACT

Objectives Temporal trends in the incidence of ST-elevation myocardial infarction (STEMI) have been declining in many countries, while the incidence of non-ST elevation myocardial infarction (NSTEMI) has reached a plateau or even increased. The reasons for these changing trends have yet to be explained. We analysed these trends and short-term mortality from acute coronary syndromes in a nationwide cohort study over 35 years in Iceland.

Design Retrospective cohort study using a national MI registry.

Setting Iceland.

Participants All cases of myocardial infarction in individuals aged 25–74 years in Iceland 1981–2015.

Methods Each case was classified as STEMI, NSTEMI or no ECG taken. ECG recordings were classified according to Minnesota criteria.

Outcome measures Trends of STEMI and NSTEMI incidence and 1-day and 28-day mortality were obtained from the National Death Registry.

Results A total of 10 348 cases were identified (mean age 61 years, 76.4% male). These were categorised as STEMI (32.7%), NSTEMI (45.8%) and no ECG taken (21.5%). We detected a significant 3.7% annual decline in the incidence of first MI. The age-adjusted incidence of STEMI showed an 83.2% decline, most pronounced after 1994, while for NSTEMI the decline was 66.5%, reaching a plateau from the year 1989 onwards. In Iceland, the uptake of highly sensitive biomarkers was initiated in 1997 (cardiac troponin T) and 2012 (high-sensitive troponin T), respectively.

Conclusions The different temporal trends in the incidence of STEMI and NSTEMI cannot be explained only by the uptake of highly sensitive biomarkers in 1997 and 2012. The change in population-level risk factor exposure is likely to have influenced atherosclerotic plaque burden and thrombotic mechanisms. Finally, increasing uptake of cardioprotective pharmacological and interventional therapy may have resulted in a primary preventive effect on plaque rupture and thrombosis and thus on the rates of STEMI and NSTEMI disproportionally.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ This paper provides a nationwide perspective of the trends of ST-elevation myocardial infarction and non-ST elevation myocardial infarction incidence and mortality over an extended period of 35 years.
⇒ The use of national registries and national personal identity number systems in Iceland provides a unique environment for the study.
⇒ The study followed the prospective systematic classification of ECGs according to the Minnesota coding criteria.
⇒ The Icelandic population is mostly Caucasian and of European descent which might limit interpretability for other ethnic groups and regions.

INTRODUCTION

Ischaemic heart disease is a leading cause of death representing approximately one-third of deaths worldwide.[1] The clinical presentation is broad and ranges from chronic disease to acute coronary syndromes (ACS) and sudden cardiac death.[2] Interestingly, in recent decades there have been changes in the presentation of ACS in most countries with a declining incidence of ST-elevation myocardial infarction (STEMI) (previously Q-wave or transmural myocardial infarction) and stable or increasing rates of non-ST elevation myocardial infarction (NSTEMI) (previously non-Q-wave or subendocardial infarction).[3–7] These trends are paralleled by a steady decline in mortality rates of MI during the last decades.[8]

The changing incidence of STEMI and NSTEMI has yet to be explained. The two groups have different pathophysiology, management, and prognosis. STEMI is most commonly associated with total coronary occlusion in a single vessel and bears higher short-term mortality, whereas NSTEMI is

associated with more extensive coronary atherosclerotic plaque burden and higher long-term mortality.[3 9 10]

There are several factors that may contribute to these different trends in the incidence and mortality of STEMI and NSTEMI. First, the cardiovascular risk factor profile in the general population has changed in recent decades with steadily decreasing smoking rates, mean population cholesterol and blood pressure levels, while the prevalence of obesity and type 2 diabetes has increased.[3 4 11] As modifiable risk factors have been shown to account for most cases of MI, it seems likely that changing risk factor profiles have a direct effect on the different clinical presentation of ACS.[12] Also, there have been major advances in the diagnosis and treatment with more sensitive biomarkers, primary preventive strategies, pharmacological treatment and reperfusion therapy.[2 3 13]

In recent years, there have been some studies analysing the trends in the incidence and mortality of ACS patients. Many of these are cross-sectional studies or observational studies over a short period of time. There have only been a few nationwide outcome studies with long-term follow-ups of consecutive patients. Most of these studies either account for the mortality of MI patients in general or stem from hospital registries focusing on the MI subclasses of STEMI and NSTEMI. Generally, the attenuated decline in the incidence of NSTEMI as compared with STEMI has been explained by the introduction of highly sensitive biomarkers, such as cardiac troponin T (cTnT) in recent years. There is, however, limited data to support this assumption due to a lack of long-term follow-up over extended periods. Therefore, we conducted a nationwide study of all cases of incident MI in Iceland over a 35-year period in order to relate the temporal trends in the incidence of STEMI and NSTEMI to the introduction of cTnT and previously reported risk factor trends in Iceland.

METHODS
Icelandic MI registry
The Icelandic Heart Association has kept a nationwide register of all cases of incident MI in Iceland since the year 1981 in collaboration with the Directorate of Health (the Icelandic MI registry). The registry was initially established in 1981 as part of the WHO: Multinational Monitoring of Trends and Determinants in Cardiovascular Disease (MONICA) study conducted by the WHO and has been extensively used for research since then.[14 15] The register includes data on every MI detected among people aged 25–74 years in Iceland. These age limits were set according to the WHO MONICA protocol in order to present trends in incident MI and mortality for the whole study period. For the purpose of this study, each case was individually linked to data from the Directorate of Health (the Icelandic Causes of Death Register) to provide cause-specific mortality data.

Identification of myocardial infarctions
Cases were identified by a diagnosis of acute MI according to the International Classification of Diseases, Ninth and Tenth Revisions, Clinical Modification (ICD-9-CM and ICD-10-CM), Disease Related Group codes from hospital and national healthcare records. The cases were classified as definite or possible MI according to definitions in the MONICA study protocol.[16] Biomarkers used in clinical practice in Iceland during the study period included CK and CK-MB until 1997, cTnT from 1997 to 2012 and high-sensitive troponin T (hs-TnT) from 2012 onwards. The criteria for the diagnosis of MI were based on symptoms, ECG changes, biomarkers and/or autopsy findings indicating myocardial infarction according to the MONICA protocol. Most of the patients with fatal out-of-hospital cardiac arrest (OHCA) had either an autopsy or ECG to verify the diagnosis of MI as this was common practice in fatal cases of OHCA at the time of the study. However, in some cases, the clinical circumstances or clinical course of the deceased will have been taken as sufficient evidence of an MI to certify the cause of death. In case of a fatal outcome, the diagnosis was based on a formal certificate of the cause of death, issued either by an attending physician or the medical examiner. In each case, the diagnosis was adjudicated according to the prespecified MONICA criteria by an independent trained observer (physician or nurse) at the Icelandic Heart Association. The applied diagnostic and ECG criteria are listed in online supplemental tables S1 and S2.

All ECGs from the index MI event were classified according to the Minnesota code by the Icelandic Heart Association.[17] Code 9–2 indicates ST-segment elevation and is applied at three sites: anterolateral site (ST-segment elevation ≥ 1.0 mm in any of leads I, aVL, V_6), posterior site (ST-segment elevation ≥ 1.0 mm in any of leads II, III or aVF) and anterior site (ST-segment elevation ≥ 1.0 mm in lead V_5 or ≥ 2.0 mm in any of leads V_1, V_2, V_3 or V_4). ECGs with code 9–2 in anterolateral, posterior or anterior sites were categorised as STEMI and other ECGs were categorised as NSTEMI. The ECGs were coded according to the WHO MONICA protocol (online supplemental tables S1 and S2) by research nurses and cardiologists that had received training from the MONICA study research group in the USA. External quality control was performed by WHO from 1981 to 1992 while the MONICA study was running, but the data collection has continued beyond the MONICA study endpoint. For patients with OHCA, the diagnostic ECG changes were obtained, when available, from Emergency Dispatch Responders on site, during transport to the hospital or in the ER on hospital arrival. However, a large proportion of OHCA patients had the diagnosis confirmed by autopsy.

Cardiac care in Iceland
The Icelandic healthcare system is state-driven and provides hospital services to all Icelandic citizens free of charge. More than two-thirds of the current population of 383 000 is centralised to the greater Reykjavik area.

Icelanders are predominantly Caucasian and of European descent. Although five regional hospitals treat MI cases locally, there is only one percutaneous coronary intervention (PCI) centre, located at the University Hospital of Iceland in Reykjavik. This centre has provided 24-hour PCI service since 2003, currently treating 95% of STEMI cases in the country. In Iceland, there is a long tradition of public health statistic registries held by the Directorate of Health since 1981 and the national MI registry holds complete data on all cases of MI according to clinical notes, autopsy reports and death certificates. Personal identification codes of all Icelanders provide the opportunity to compile data from these registries to study the incidence and mortality of MI on a national level. Although the sample size is moderate, the data set is complete and accurate so as to represent the incident and mortality trends of MI in Iceland.

Statistical analysis

Statistical analysis was done in R, V.4.1.0 and RStudio, V.2023.09.0+463. Incidence and mortality rates were calculated with age standardisation using ageadjust. direct from the Epitools package and the Icelandic population in the year 2015 as a reference. Comparison of incidence rates between sexes was done by Poisson regression. Smooth regression lines for incidence rates were fitted using Poisson regression models. The Joinpoint trend analysis software from the National Cancer Institute, V.5.02, was used to test for significant join points in the fitted regression lines for the incidence and mortality rates. A change in the annual percent change was considered significant at the alpha=0.05 level. Population numbers were obtained from Statistics Iceland.[18] The statistical code is included in online supplemental file 1.

Patient and public involvement

No patients or members of the public were involved in the design, conduct or reporting of this study.

RESULTS

Study population

A total of 10 988 cases of incident MI in people aged 25–74 years were identified during the study period 1981–2015. There were 640 cases (5.8%) where ECGs had been recorded but were missing in the database. These were excluded from the analysis, leaving a study cohort of 10 348 cases. The mean age was 61 years and 76.4% were male. Based on Minnesota codes of available ECGs, ST elevation was found in 3381 cases (32.7%) and NSTEMI in 4740 cases (45.8%) (table 1). In 2227 cases (21.5%), ECGs were not taken, mostly because of OHCA or ongoing resuscitation on hospital arrival. Sex distribution and mean age at first MI were similar in all three groups (76.6%, 74.8% and 79.4% male, respectively and 60, 62 and 63 years, respectively).

Nearly all of the STEMI and NSTEMI cases received treatment in a hospital (99.9% and 99.3%, respectively). However, most of the groups with no ECG taken were patients in OHCA where no attempt at cardiopulmonary resuscitation (CPR) was made, some of which were never transferred to the hospital and thus medically unattended (65.5%). In these patients, the diagnosis of MI was either based on autopsy findings or other prespecified criteria (online supplemental table S1). In addition, 14.3% of patients with no ECG taken were patients without ECG activity on hospital arrival, where some attempt at CPR was performed, although no restoration of cardiac activity was reached. Further, 4.2% of the patients with no ECG recorded had OHCA in the home and 16% in other or unknown locations. Of the deceased with no ECG taken,

Table 1	Patient management and outcome			
	STEMI (n=3381)	NSTEMI (n=4740)	No ECG taken (n=2227)	Total (n=10 348)
Sex (male)	2591 (76.6)	3544 (74.8)	1769 (79.4)	7904 (76.4)
Mean age at first MI	59.8	61.7	62.8	61.3
Management				
Hospital	3379 (99.9)	4709 (99.3)	318 (14.3)	8406 (81.2)
Home/nursing home	1 (0)	1 (0)	94 (4.2)	96 (0.9)
Medically unattended	0	0	1459 (65.5)	1459 (14.1)
Other/unknown	1 (0)	30 (0.6)	356 (16.0)	387 (3.7)
Mortality				
1-day mortality	74 (2.2)	140 (3.0)	1918 (86.1)	2132 (20.6)
28-day mortality	361 (10.7)	562 (11.9)	2105 (94.5)	3028 (29.3)
Autopsy				
Autopsy performed	135 (4.0)	251 (5.3)	1278 (57.4)	1664 (16.1)
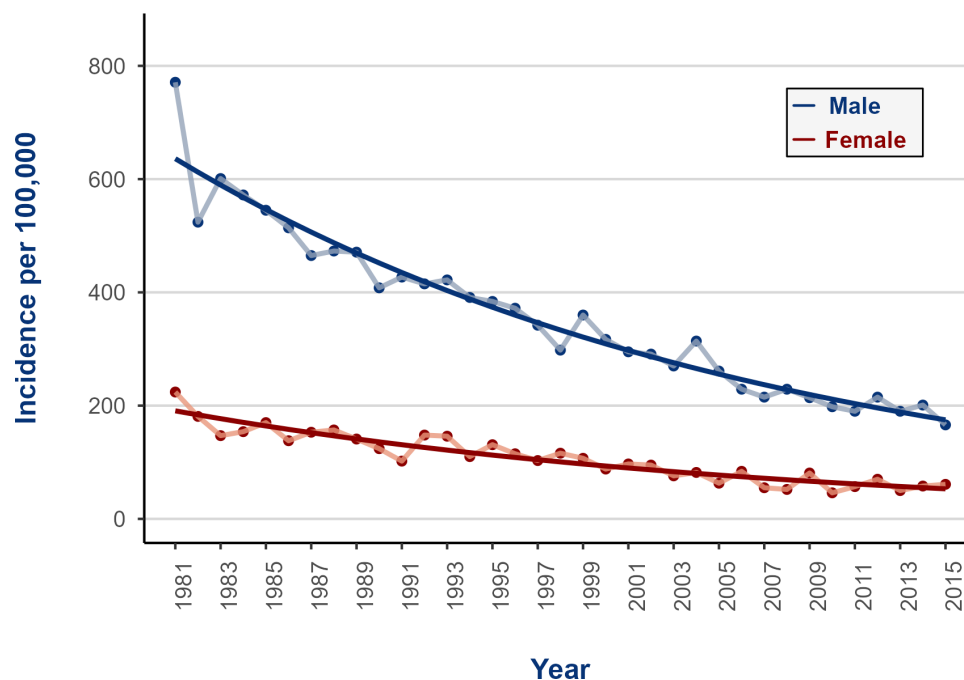
Figure 1 Age-adjusted incidence of myocardial infarction per 100 000 people, categorised by sex, from 1981 to 2015.

57.4% had an autopsy, mostly medico-legal autopsies (84%) but also clinical hospital autopsies. In comparison, an autopsy was performed in 4.0% of the STEMI cases and 5.3% of the NSTEMI cases (table 1).

Incident MI per 100 000 individuals

The age-adjusted incidence of MI was reduced by 76.9% over the study period, declining from 493 to 114 events per 100 000 individuals which is an annual percentage change (APC) of −3.7%. The incidence decreased by 78.5% among males (from 771 to 166 events per 100 000) and 72.8% among females (from 224 to 61 events per 100 000) (figure 1). Notably, a significant disparity in incidence between sexes persisted throughout the entire observation period, with an overall incidence ratio of 3.35 for males compared with females.

Although the event rates of MI were higher among older age groups, a declining trend was observed across all age categories throughout the study period. However, the decline was particularly pronounced among the older age groups. The incidence decreased by 71.5% in the oldest group (individuals aged 65–74 years), while in individuals 45 years or older, the incidence decreased by 68.8%. In the youngest age group (individuals aged 25–34 years), the decrease was only 43.1% (figure 2).

The age-adjusted incidence for STEMI showed a decrease of 83.2%, declining from 137 to 23 per 100 000 individuals during the study period. The APC between 1981 and 1994 did not change significantly, but there was a significant decline of −6.2% between 1994 and 2015. The age-adjusted incidence of NSTEMI declined somewhat less by 66.5%, decreasing from 239 to 80 per 100 000 individuals (figure 3). The APC was −7.9% between 1981 and 1989 and −1.0% between 1989 and 2015. Notably,

cTnT was implemented in Iceland in the year 1997 and hs-TnT in the year 2012.

Mortality per 100 000 individuals

Throughout the study period, the age-adjusted 1-day and 28-day (including day 1) mortality associated with MI displayed a notable reduction. Specifically, the 1-day mortality of MI declined by 91.8%, decreasing from 122 to 10 per 100 000 individuals (figure 4) with a reduction of 93.5% among males and 78.6% among females. The APC was −4.4% between 1981 and 2005, −18.3% between 2005 and 2010, but did not change between 2010 and 2015. The 1-day mortality was primarily driven by mortality in the no-ECG group. Similarly, the 28-day age-adjusted mortality decreased by 92.3%, declining from 195 to 15 per 100 000 individuals (online supplemental figure). This reduction was consistent for both sexes or 92.9% for males and 90.0% for females. The APC was −5.1% between 1981 and 2005, −20.4% between 2005 and 2008, but did not change between 2008 and 2015.

Further analysis was conducted on the 28-day mortality within the three subgroups, STEMI, NSTEMI and cases with no ECG taken. The 28-day age-adjusted mortality for STEMI decreased by 85.0%, from 20 to 3 per 100 000 individuals. Similarly, the 28-day mortality for NSTEMI decreased by 95.4%, from 65 to 3 per 100 000 individuals, reflecting significant improvements in mortality outcomes for both subgroups (online supplemental figure). The group where no ECG was taken had the highest age-adjusted 28-day mortality among the three ECG categories, amounting to 2105 cases of the 2227 (94.5%) as shown in table 1. Furthermore, the patients with no ECG taken had a 90.9% reduction in age-adjusted 28-day mortality declining from 111 to 10 per 100 000 individuals
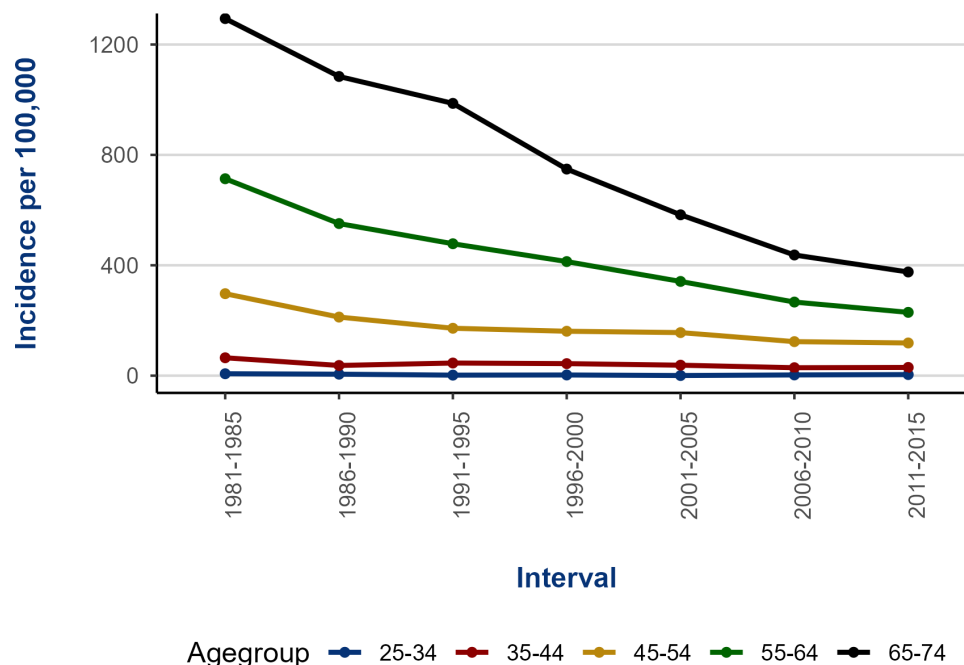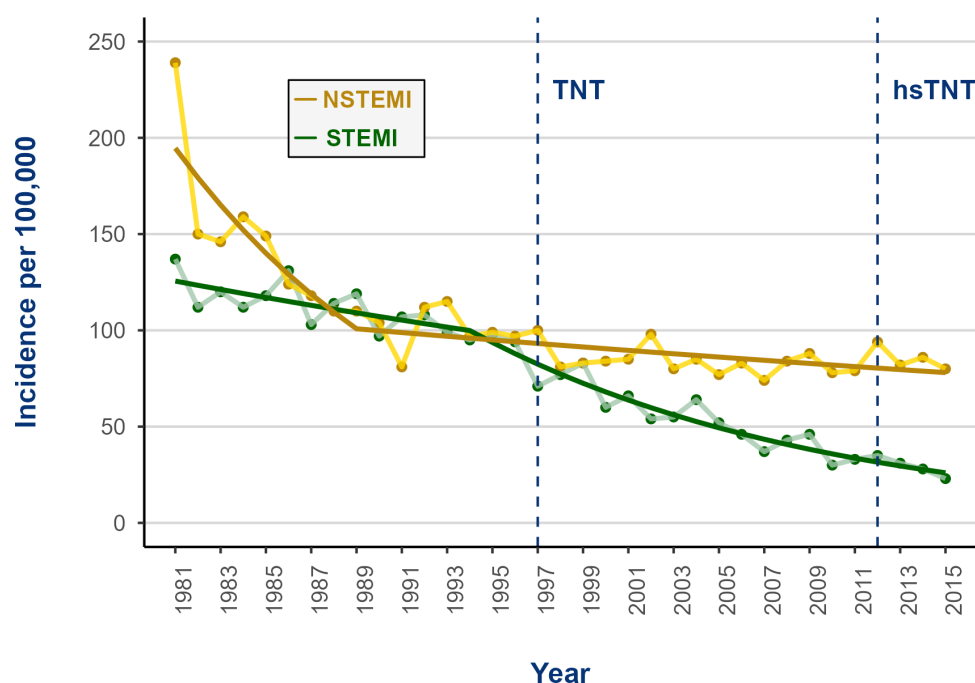
Figure 2 Incidence of myocardial infarction per 100 000 from 1981 to 2015 stratified by age groups.

reflecting the overall reduction in MI mortality in the population.

DISCUSSION

In this nationwide cohort of all incident cases of MI, we found a consistent 3.7% decline per year in age-adjusted incidence of MI during 35 years. This decline was most profound in the older age groups and was apparent both for new cases of STEMI as well as NSTEMI. Including data from discharge records from all hospitals treating

MI in the country and the National Causes of Death Registry, we identified all cases of incident MI according to MONICA criteria, including clinical, biochemical, electrocardiographic and autopsy findings. Importantly, we used pre-specified Minnesota code criteria to categorise each patient as STEMI, NSTEMI or no ECG taken. This allowed us to identify the high-risk group of MI patients with no ECG taken, a group generally medically unattended, comprised to a large extent of victims of OHCA. The linkage of each case to data from the National

Figure 3 Age-adjusted incidence of STEMI and NSTEMI per 100 000 people from 1981 to 2015. hsTNT, high-sensitive troponin T; NSTEMI, non-ST elevation myocardial infarction; STEMI, ST-elevation myocardial infarction.
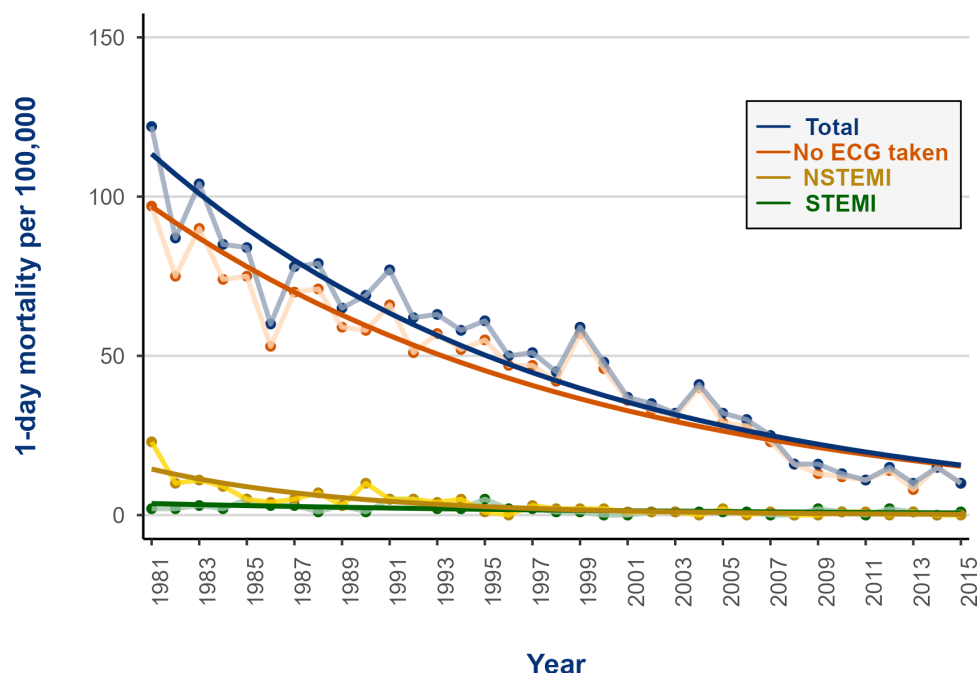
Figure 4 Age-adjusted 1-day mortality per 100 000 people from 1981 to 2015. Data is shown for all cases and for the subgroups no ECG taken, STEMI and NSTEMI. NSTEMI, non-ST elevation myocardial infarction; STEMI, ST-elevation myocardial infarction.

Causes of Death Registry allowed us to analyse trends in mortality of each diagnostic category over time and to directly compare the prognosis of hospitalised versus non-hospitalised MI patients. This method has, to our knowledge, not been used in previous publications on incidence and mortality trends in MI patients.

Declining incidence of MI

In the present study, we found a decline in age-adjusted incidence of 83.2% for STEMI and 66.5% for NSTEMI between 1981 and 2015. However, as shown in figure 3, there was a difference in the temporal trends of age-adjusted incidence between STEMI and NSTEMI patients. While the decline in the incidence of STEMI was accelerated after 1994, the declining incidence of NSTEMI was attenuated after 1989. These shifts in temporal trends of STEMI and NSTEMI incidence occurred 3 and 8 years before the introduction of cTnT in 1997. Therefore, the shifts in temporal trends of STEMI and NSTEMI incidence cannot exclusively be related to the uptake of highly sensitive biomarkers. The most plausible explanation is the changes in population risk factor exposure during the same period. These shifts not only involve rapidly declining smoking rates and declining mean blood pressure and blood cholesterol levels in the population but also a significant increase in the incidence of obesity and type 2 diabetes.[4] Furthermore, cardioprotective treatment with the introduction of aspirin and statins as well as the increased use of coronary interventions and thienopyridine platelet inhibitors are likely to have had a significant impact on primary prevention.[19] The net effect of these changes on the population level risk factor burden and treatment of coronary artery disease in

recent decades is likely to have impacted the incidence of STEMI and NSTEMI differently. Although most previous reports on the relative incidence of NSTEMI compared with STEMI in recent years have shown similar trends in declining incidence of STEMI and NSTEMI[6 8 20 21] they have been of shorter duration and have not been able to uncover these important temporal trends in relation to the uptake of sensitive biomarkers, risk factor exposure and cardioprotective pharmacological treatment.

Several reports on temporal change in incident MI have been published during the last few decades. Generally, these studies show significant reductions in the overall incidence of MI.[6 8 20–22] However, the incidence of STEMI and NSTEMI shows more variable results. While most studies show a substantial decline in the incidence of STEMI after the turn of the millennium, the results for NSTEMI are varied. Many authors report a steady or even increasing rate of NSTEMI during the last decades,[6 8 20 21] while others have found declining rates.[22] The reasons for this disparity are probably multiple. First, the populations studied may vary; some are derived from registries, private insurance patient groups or other selected cohorts, while others are derived from national databases of hospital admissions or pharmacological registries. Second, the period studied may vary between recent decades, thus influencing the comparability of results. The different timing of the introduction of highly sensitive biomarkers will affect the diagnostic sensitivity of minor MIs, mostly NSTEMI, some of which were earlier diagnosed as unstable angina.[23–26] Further, the effect of cardioprotective pharmacotherapy prior to incident MI may also have had an impact.[19] A paper from Denmark

showed that primary preventive treatment with aspirin, statins, beta-blockers, ACEIs/ARBs and particularly thienopyridine platelet inhibitors may differently affect the incidence of ACS groups in favour of STEMI.[19] Many studies exclude the oldest age categories which are an increasing proportion of the total population with long-term exposure to risk factors, a high prevalence of ACS and the most rapid decline in incident MI compared with younger age categories. Finally, the changes in prevalence and duration of risk factor exposure in the general populations of different countries may affect plaque composition and thus lead to different clinical presentations of ACS in different populations.[5]

Declining mortality

The temporal trends in declining MI incidence are reflected in continuing trends of declining coronary heart disease (CHD) mortality in most Western countries. Although most epidemiological studies report on total mortality from MI, including OHCA and hospitalised patients, others focus on case fatality in each of the two main categories: STEMI and NSTEMI. Our data and others show that a considerable proportion of MI mortality stems from the high-risk group of OHCA or medically unattended MI patients.[20 27] As seen in figure 4, the 1-day mortality of MI is almost completely due to deaths in the no-ECG group. Similarly, deaths in the no-ECG group account for more than half of the overall 28-day mortality after MI (online supplemental figure). Therefore, when exploring the driving factors for declining CHD mortality during the last two to three decades it is necessary to account for this high-risk group.

While the short-term mortality of STEMI and NSTEMI patients in the present study is remarkably similar, the prognosis of the no-ECG group is very poor, 94.5% of which had died in the first 28 days after MI (table 1). Interestingly, when analysing the rate of decline in age-adjusted 28-day mortality between the three diagnostic categories, STEMI, NSTEMI and no ECG taken, they remain quite similar at 85.0%, 95.4% and 90.9%, respectively. This illustrates that the prognosis of these three diagnostic groups improved at a similar rate, although the time period from 1981 to 2015 holds much greater improvement in pharmacologic and interventional therapy for hospitalised patients with MI than victims of OHCA treated with CPR or medically unattended.[28] Therefore, as has previously been shown, the attenuated incidence and mortality of MI patients may be explained by improved risk factor profile rather than by all interventions combined.[4 29 30]

The declining trends in age-adjusted mortality are even more pronounced in the older age groups where the absolute risk levels are higher, possibly further reflecting a different risk profile and longer duration of risk factor exposure. Data from the Icelandic Heart Association has in fact shown distinct changes in risk factors in the Icelandic population. When looking into declining mortality rates from CHD between 1981 and 2006 they found that approximately 73% was attributable to population risk factor reduction, most importantly lower cholesterol levels (changing from 6.01 mmol/L to 5.14 mmol/L, explaining 32% of the overall risk reduction), lower smoking prevalence (changing from 46.5% to 22.6%, explaining 22% of the overall risk reduction) and lower mean systolic blood pressure (changing from 126.3 mm Hg to 121.2 mm Hg, which explains 22% of the overall risk reduction), with adverse trends for diabetes (increasing from 1.7% to 3.6%, explaining 5% increased risk) and obesity (increasing from 25.0% to 27.0%, explaining 4% increased risk). The overall treatment effect accounted for 25% of the mortality decrease, mainly through secondary prevention (8%), heart failure treatment (6%), treatment for ACS and revascularisation (6%).[4] A Norwegian study showed similar trends with risk factor reduction accounting for 66% of the decline in CHD events from 1994 to 2008.[20]

Many of the published studies analysing short-term mortality after MI are dated to the era before reperfusion in STEMI and early invasive therapy in NSTEMI became standard of care. Also, the use of cardioprotective pharmacological therapy was less established than in recent years. The treatment goals of LDL cholesterol to less than 1.8 mmol/L in post-MI patients with very high risk were put forward in the 2012 ESC Guidelines on cardiovascular disease prevention in clinical practice,[31] and were reduced from the previous target of 2.5 mmol/L. Also, the targets of blood pressure and blood glucose treatment were less aggressive at the time of this study than currently. Finally, we have witnessed rapidly declining smoking rates during the last two decades in most Western countries. The increasing prevalence of obesity and diabetes is a risk factor counteracting the positive mortality trends due to most other CVD risk factors. Overall, these changes to a varying degree have impacted short- and long-term outcomes of patients after MI in different regions of the world.

Strength and limitations

The main strength of the present study is that it is a population-based cohort, including all consecutive cases of first MI in individuals aged 25–74 years over a 35-year period in Iceland. This offers a nationwide perspective over a long time, which is particularly interesting as there have been profound changes in risk factor profile, diagnosis and treatment of MI during this period. The use of national registries and national personal identity number systems in Iceland provides a unique environment for such studies. Furthermore, the study followed the standardised MONICA study protocol and the prospective systematic classification of ECGs according to the Minnesota coding criteria. Importantly, the study conveys information on OHCA victims, which is a high-risk group that is often under-represented due to a lack of data. A high rate of autopsies was performed in this group which supports the diagnosis, although the cases cannot be categorised into STEMI or NSTEMI.

The study also has limitations. The Icelandic population is small, mostly Caucasian and of European descent which might limit interpretability for other ethnic groups and regions. Second, the risk factor profile may differ from other countries. Moreover, there were 640 cases with missing ECGs accounting for 5.8% of the study group which might introduce some bias, as they cannot be categorised into STEMI or NSTEMI. Also, a proportion of STEMI and NSTEMI patients will not have sought medical services but survived and therefore were not detected in our study. Furthermore, the ECGs were coded according to the Minnesota coding system which is useful for epidemiological purposes but may not always resemble the interpretation of clinicians. Cases of posterior STEMI and new left bundle branch block may thus be misclassified as the ECGs may not show an ST-segment elevation.

CONCLUSIONS

In conclusion, we report a significant 3.7% annual decline in consecutive cases of incident MI during a 35-year period from 1981 to 2015 in Iceland. The decline in incident STEMI and NSTEMI was 83.2% and 66.5%, respectively. The difference in temporal trends in the incidence of STEMI and NSTEMI after 1994 and 1989, respectively cannot be explained only by the uptake of cTnT measurements in 1997. Rather, the shifting risk factor exposure on the population level as well as increasing uptake of cardioprotective therapy is likely to have had a different effect on the incidence of these two main categories of MI.

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