

Medication in Relation to ST-Segment Elevation Myocardial Infarction in Patients With a First Myocardial Infarction

Swedish Register of Information and Knowledge About Swedish Heart Intensive Care Admissions (RIKS-HIA)

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Background: The extent and the severity of acute myocardial infarction (MI) is decreasing. Out-of-hospital medical management before the hospital admission could alter clinical presentation in acute MI. We used a large national patient register to investigate the relation between previous medication use (aspirin, β -blockers, angiotensin-converting enzyme [ACE] inhibitors, and statins) and the risk of presenting with ST-segment elevation MI (STEMI) or non-STEMI.

Methods: We included 103 459 consecutive patients from the Swedish Register of Information and Knowledge About Swedish Heart Intensive Care Admissions (RIKS-HIA) admitted between January 1, 1996, and December 31, 2006, with a first acute MI.

Results: The patients with STEMI (43.5% of the total) were younger, had less prior cardiovascular disease, and used fewer medications before hospitalization. Of the STEMI patients, 61.4% had used no medication vs 45.9%

of the patients with non-STEMI. After multiple adjustments, use of aspirin, β -blockers, ACE inhibitors, and statins before hospitalization were all associated with substantially lower odds of presenting with STEMI. Furthermore, the risk decreased with the number of previous medications, and the use of 3 or more medications was associated with a multiply adjusted odds ratio of presenting with STEMI of 0.48 (99% confidence interval, 0.44-0.52) compared with no medications at admission.

Conclusions: Use of aspirin, β -blockers, ACE inhibitors, or statins before hospital admission in patients with a first acute MI is associated with substantially less risk of presenting with STEMI. The risk decreases with the increasing number of these medications used before acute MI, underlining the benefit of preventive medication in high-risk patients.

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CORONARY HEART DISEASE (CHD) mortality rates and incidence have decreased during the past few decades in most western countries, including Sweden.¹⁻³ In addition to decreasing CHD rates, there has been a shift over time in clinical presentation, with less severe and smaller acute myocardial infarctions (AMIs), more unstable angina pectoris, and lower case fatality rates.⁴

See Invited Commentary at end of article

Risk factors for CHD are well known,⁵ and decreasing cholesterol levels and lower rates of smoking and other risk factors have contributed to the marked decrease in CHD mortality in many European and North American countries.^{6,7} In addition, medi-

cations used in the treatment of several cardiovascular conditions such as hypertension, angina, and heart failure have also contributed to the decrease in CHD mortality. Aspirin, β -blockers, angiotensin-converting enzyme (ACE) inhibitors, and statins have been found to reduce mortality due to cardiovascular disease in randomized controlled trials conducted in various subsets of patients. The extent to which medications influence clinical presentation in CHD has not been widely investigated. In 1 study, the use of statins and β -blockers was associated with a lower risk of presenting with MI compared with stable exertional angina.⁸ In patients with acute coronary syndromes (ACSs), the use of aspirin and statins has been associated with lower risk of presenting with ST-segment elevation as a marker of larger infarctions.^{9,10} Whether other pharmacological agents or medications in combination are

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Table 1. Baseline Characteristics of 103 459 Patients Presenting With a First STEMI or Non-STEMI^a

Characteristic	STEMI Patients (n=45 027 [43.5%])	Non-STEMI Patients (n=58 432 [56.5%])
Men	66.9	64.4
Age, y		
Mean	66.6	68.5
<65	40.6	33.7
Smoking		
Current	31.1	23.7
Past	24.7	29.4
Never	44.2	46.9
Hypertension	32.0	38.6
Diabetes mellitus	18.1	22.1
Heart failure	2.8	7.0
Prior PCI or CABG	3.2	7.2
Prior angina	5.9	14.0
Medications used		
Aspirin	20.4	33.5
β-Blockers	22.5	33.2
ACE inhibitors	10.3	15.8
Statins	8.1	15.0

Abbreviations: ACE, angiotensin-converting enzyme; CABG, coronary artery bypass graft; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction.

^aUnless otherwise indicated, data are expressed as percentage of patients. All differences between STEMI and non-STEMI patients were statistically significant ($P < .001$).

also associated with a lower risk of presenting with ST-segment elevation MI (STEMI) is not known. We used data from a large national register of patients admitted to Swedish coronary care units to investigate the association between previous medication use (aspirin, β-blockers, ACE inhibitors, and statins) and clinical presentation (STEMI or non-STEMI) in patients with a first AMI.

METHODS

The Register of Information and Knowledge About Swedish Heart Intensive Care Admissions (RIKS-HIA) includes all patients admitted to hospitals with coronary care units. The full protocol has been published previously¹¹ (detailed information and the complete protocol are also available at <http://www.ucr.uu.se/rikshia/>). RIKS-HIA started in 1995 with 19 participating hospitals, increasing gradually to 73 (of 77) Swedish hospitals in 2006. The present study is based on all consecutive patients aged 25 to 84 years without a history of AMI who were admitted to a hospital between January 1, 1996, and December 31, 2006, and discharged with a diagnosis of STEMI or non-STEMI.

The ST segment was recorded as the first choice of which the following alternatives accurately described the ST segment on the electrocardiogram at entry: 1 indicates normal; 2, left bundle branch block or pacemaker; 3, ST-segment elevation; 4, ST-segment depression; 5, T-wave inversion; and 6, other changes. The criteria for a diagnosis of AMI were standardized and identical for all participating hospitals using the World Health Organization, Joint European Society of Cardiology, and American College of Cardiology Committee criteria.¹² We defined STEMI as presentation with ST-segment elevation. Information on medication was missing in less than 1% of patients. In cases with missing information, it was presumed that the patient did not use that particular drug. Because prior angina was only registered until 2003, we substi-

tuted use of medication with long-acting nitrates before admission as a proxy for previous angina. The use of aspirin includes aspirin sold over the counter. Use of angiotensin II receptor blockers was only recorded in 2004 to 2006 and was not included. Data on mortality were obtained by merging the RIKS-HIA with the Swedish National Cause of Death Register.

ETHICS APPROVAL

The RIKS-HIA was approved by an ethics committee and the National Board of Health and Welfare of Sweden. The process of merging mortality data with the Swedish National Cause of Death Register was approved by the ethics committee in Uppsala.

STATISTICAL METHODS

Baseline characteristics were summarized as means or percentages as appropriate. The independent associations between STEMI and previous use of aspirin, β-blockers, ACE inhibitors, or statins were assessed by means of logistic regression, in which STEMI was entered as the dependent variable and the following variables were used as covariates (possible confounders): age, sex, year of admission, medications before study entry (aspirin, β-blockers, ACE inhibitors, and statins), and history of smoking (never, current, and former), coronary artery bypass graft, percutaneous coronary intervention, diabetes mellitus, hypertension, heart failure, or angina. To investigate potential interactions between sex and previous medication use and between age and previous medication use, interaction terms (sex × previous medication use and age × previous medication use) were defined and introduced into the models. All statistical analyses were performed using commercially available software (SPSS, version 15.0; SPSS Inc, Chicago, Illinois). Odds ratios (ORs) were calculated from the logistic regression models. Because of the large population, 99% confidence intervals (CIs) were used.

RESULTS

BASELINE CHARACTERISTICS

Of 117 359 patients, we excluded 13 497 with left or right bundle branch block or pacemaker on the electrocardiogram. In addition, we excluded 403 patients with no recorded information of ST segment. However, including patients with left or right bundle branch block did not significantly change the results. After these exclusions, the final sample included 103 459 patients admitted to the hospital for a first AMI. Of those, 43.5% presented with STEMI and 56.5% with non-STEMI. During the study period, 30-day mortality in STEMI patients decreased from 13.0% in 1996 to 5.6% in 2006 and in non-STEMI patients from 10.9% in 1996 to 4.1% in 2006. One-year mortality decreased from 18.0% in both STEMI and non-STEMI patients to 8.8% in STEMI patients and 10.0% in non-STEMI patients.

Table 1 shows baseline characteristics in the STEMI and non-STEMI patients. Patients with STEMI were more often men and slightly younger than patients with non-STEMI. Moreover, smoking was more common: 31.1% of patients with STEMI were current smokers vs 23.7% of non-STEMI patients ($P < .001$). Patients with non-STEMI had significantly more hypertension, diabetes mellitus, heart failure, prior angina pectoris, and known cardiovascular disease with prior percutaneous coro-

Table 2. Type of Medication Used and Risk of Presenting With STEMI vs Non-STEMI

Medication	No. (%) of Patients		OR (99% CI)	
	STEMI (n=45 027)	Non-STEMI (n=58 432)	Age-Adjusted	Multiply Adjusted ^a
Aspirin	9100 (20.2)	19483 (33.3)	0.66 (0.63-0.68)	0.72 (0.69-0.76)
β-Blockers	10018 (22.2)	19279 (33.0)	0.76 (0.73-0.80)	0.82 (0.78-0.86)
ACE inhibitors	4583 (10.2)	9172 (15.7)	0.76 (0.72-0.80)	0.84 (0.79-0.89)
Statins	3597 (8.0)	8670 (14.8)	0.67 (0.63-0.71)	0.79 (0.74-0.84)

Abbreviations: ACE, angiotensin-converting enzyme; CI, confidence interval; OR, odds ratio; STEMI, ST-segment elevation myocardial infarction.

^aAdjusted for sex, age, smoking, hypertension, diabetes mellitus, heart failure, prior angina, prior revascularization, all 4 medications, and year of admission.

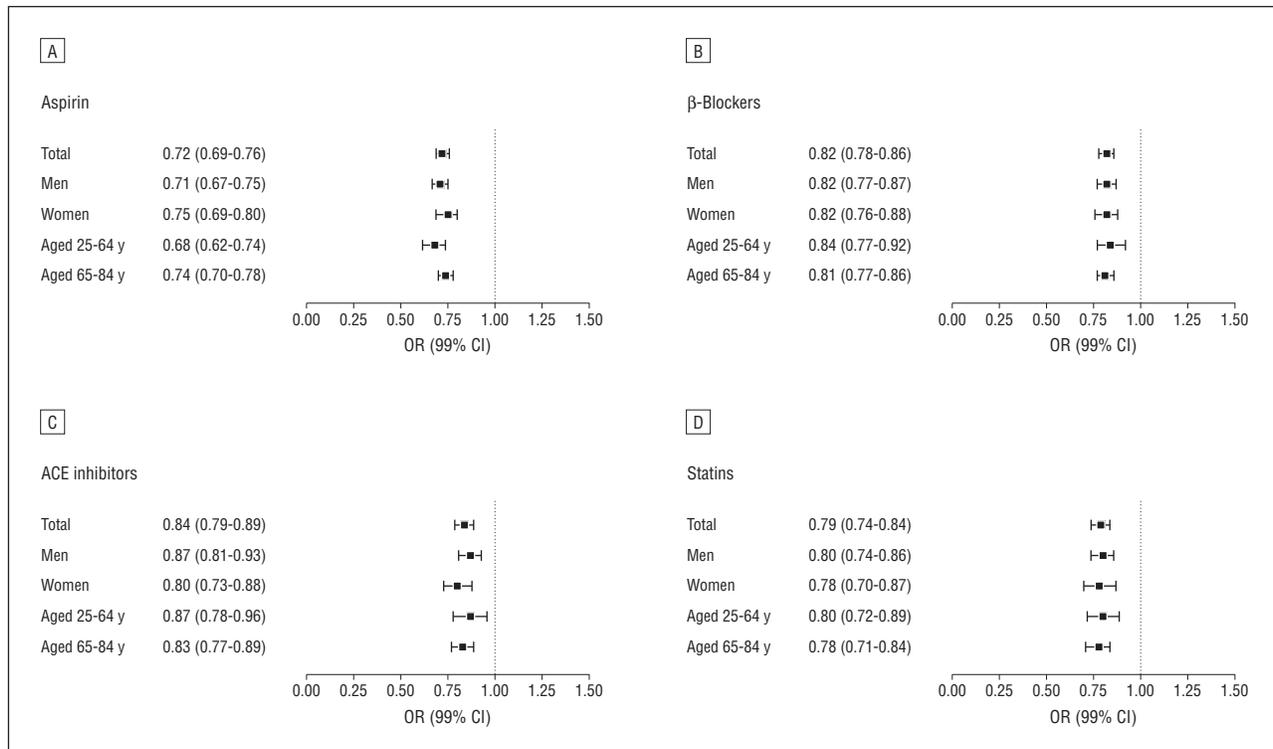


Figure 1. Multiply adjusted odds ratios (ORs) (99% confidence intervals [CIs]) for presenting with ST-segment elevation myocardial infarction (STEMI) vs non-STEMI. Results are shown for all men and women (total), for men and women separately, and by age for previous use of aspirin (A), β-blockers (B), angiotensin-converting enzyme (ACE) inhibitors (C), and statins (D). To provide a more accurate presentation of the 99% CIs, ORs are represented on a linear scale after logarithmic transformation. The vertical dotted lines indicate the reference value.

nary intervention or coronary artery bypass graft. Hence, use of aspirin, β-blockers, ACE inhibitors, and statins was more common in non-STEMI patients. All differences were statistically significant ($P < .001$).

MEDICATION USE AND CLINICAL PRESENTATION

In subsequent analyses, we compared the use of aspirin, β-blockers, ACE inhibitors, and statins before AMI in STEMI and non-STEMI patients. Patients with STEMI used aspirin (20.4% vs 33.5%), β-blockers (22.5% vs 33.2%), ACE inhibitors (10.3% vs 15.8%), and statins (8.1% vs 15.0%) less often than did non-STEMI patients before AMI ($P < .001$ for all; Table 1).

We used logistic regression in a multivariable model to investigate separately how the use of aspirin,

β-blockers, ACE inhibitors, and statins before AMI affected the risk of presenting with STEMI. All models were adjusted for age, sex, hypertension, diabetes, heart failure, angina, prior revascularization, smoking, and year of admission and mutually for previous use of aspirin, β-blockers, ACE inhibitors, and statins. Overall, previous use of aspirin (multiply adjusted OR, 0.72; 99% CI, 0.69-0.76), β-blockers (0.82; 0.78-0.86), ACE inhibitors (0.84; 0.79-0.89), and statins (0.79; 0.74-0.84) were all independently associated with substantially lower risk of presenting with STEMI (Table 2). Next, we investigated the association of each medication separately in men and women and in the groups aged 25 to 64 years and 65 to 84 years for men and women (Figure 1). Effects were consistent for all 4 types of medication, with no suggestion of any interaction effects of age or sex. Of the 103 459 patients, 12 250 had prior coronary disease, in-

Table 3. Characteristics of Patients Presenting With a First MI in Relation to Number of Medications^a

Characteristic	No. of Medications ^b			
	None (n=54 501 [52.7%])	1 (n=24 247 [23.4%])	2 (n=15 982 [15.4%])	≥3 (n=8729 [8.4%])
Women	31.3	39.4	38.0	34.2
Mean age, y	65.4	70.0	71.0	68.8
Current smokers	32.9	21.9	18.2	18.7
Hypertension	14.8	55.9	60.2	65.2
Diabetes mellitus	13.0	24.0	30.3	38.0
Heart failure	1.8	6.7	10.0	13.1
Prior PCI or CABG	1.0	4.1	11.2	26.5

Abbreviations: CABG, coronary artery bypass graft; MI, myocardial infarction; PCI, percutaneous coronary intervention.

^aAll differences between medication number categories were statistically significant ($P < .001$).

^bUnless otherwise indicated, data are expressed as percentage of patients. Because of rounding, percentages may not total 100.

Table 4. Treatment With Medication Before AMI in Relation to Risk of Presenting With STEMI vs Non-STEMI

No. of Medications Before AMI ^a	No. (%) of Patients ^b		OR (99% CI)	
	STEMI	Non-STEMI	Age-Adjusted	Multiply Adjusted ^c
None	27 665 (61.4)	26 836 (45.9)	1 [Reference]	1 [Reference]
1	9967 (22.1)	14 280 (24.4)	0.71 (0.68-0.74)	0.76 (0.72-0.79)
2	5175 (11.5)	10 807 (18.5)	0.49 (0.47-0.52)	0.59 (0.55-0.62)
≥3	2220 (4.9)	6509 (11.1)	0.34 (0.32-0.37)	0.48 (0.44-0.52)
Total	45 027 (100)	58 432 (100)		

Abbreviations: AMI, acute myocardial infarction; CI, confidence interval; OR, odds ratio; STEMI, ST-segment elevation MI.

^aIncludes aspirin, β -blockers, angiotensin-converting enzyme inhibitors, and statins.

^bBecause of rounding, percentages may not total 100.

^cAdjusted for sex, age, smoking, hypertension, diabetes mellitus, heart failure, prior angina, prior revascularization, and year of admission.

cluding angina and/or revascularization procedures. Odds ratios associated with the 4 types of medication were broadly similar irrespective of the presence or absence of prior coronary disease (data not shown).

Table 3 compares characteristics in patients with no, 1, 2, or 3 or more medications, irrespective of type of medication. As expected, patients with more medications had more comorbidities and notably higher rates of hypertension and diabetes but also of heart failure and prior revascularization. A higher proportion of patients with STEMI were receiving no medication before AMI (61.4% for STEMI patients vs 45.9% for non-STEMI patients) (**Table 4**). Conversely, a lower proportion of STEMI patients had 3 or more medications (4.9% for STEMI patients vs 11.1% for non-STEMI patients). After adjustment for comorbidities and other factors, use of 1 medication, regardless of type, was associated with lower risk of presenting with STEMI (multiply adjusted OR, 0.76; 99% CI, 0.72-0.79). When compared with no medication, adding one more decreased the risk by approximately half (OR, 0.59; 99% CI, 0.55-0.62). Treatment with 3 or more medications further decreased the risk of presenting with STEMI (multiply adjusted OR, 0.48; 99% CI, 0.44-0.52) (**Figure 2**), although this group included only a small proportion of the patients. Effects were consistent regardless of age and sex.

Despite a higher risk of presenting with ST-segment elevation, mortality risk did not decrease with the number of medications; 30-day mortality in non-STEMI pa-

tients receiving no medications was 5.5% and in STEMI patients was 7.9%, increasing to 8.3% in non-STEMI patients and 13.5% in STEMI patients receiving 2 medications. In patients receiving 3 or more medications, 30-day mortality was 5.9% and 10.3% in non-STEMI and STEMI patients, respectively.

COMMENT

In this study with data from a large national register that included more than 100 000 patients with a first AMI, previous medication use (ie, aspirin, β -blockers, ACE inhibitors, or statins) was associated with a decreased risk of presenting with STEMI. In addition, the risk decreased with the number of these medications used before AMI. These findings could be useful in explaining some of the beneficial effects of these medications on coronary mortality demonstrated in several randomized controlled trials.

We are aware of few studies that systematically compared medications in patients with different manifestations of coronary disease. In 1 recent study, it was found that the use of statins and β -blockers in adults whose first clinical presentation of coronary disease was AMI or stable exertional angina was associated with lower odds of presenting with an AMI than with stable angina, suggesting that these agents may stabilize the underlying coronary plaque.⁸ The same mechanisms potentially apply in

ST-segment elevation vs no ST-segment elevation in AMI. In the Global Registry of Acute Coronary Events (GRACE) Study, patients with ACS who received long-term aspirin therapy were significantly less likely to have STEMI (OR, 0.35; 95% CI, 0.30-0.40) compared with patients not taking aspirin.¹⁰ The GRACE Study further demonstrated that patients receiving statins before admission were less likely to present with STEMI (OR, 0.79; 95% CI, 0.71-0.88).⁹

These data are consistent with our findings. However, we were able to expand on these previous results by finding a similar effect for ACE inhibitors and β -blockers and, furthermore, to demonstrate that these effects seem to be additive, with less risk of STEMI correlated with the use of more medications.

In general, ST-segment elevation results from transmural acute myocardial ischemia in response to fissuring or rupture of an atheromatous plaque, with total and prolonged occlusion of a major coronary artery. In contrast, non-ST-segment elevation in the setting of ACS is usually associated with incomplete coronary occlusion or occlusion of a small branch artery. Preexisting disease is important in that the existence of prior collateral flow in the affected artery may limit the spread of necrosis, preserving the subpericardial zone.¹³ Research in patients with ACS or a first episode of AMI has shown that the risk of presenting with STEMI was strongly associated with smoking,^{14,15} whereas prior disease (eg, angina, prior MI, percutaneous coronary intervention, and coronary artery bypass graft)¹⁴ and hypertension¹⁵ are associated with less ST-segment elevation.¹⁴ Similarly, in the GRACE Study, a history of angina, MI, stroke, or myocardial revascularization was more frequent among patients with unstable angina than among those with MI and was more common among patients with non-STEMI than among those with STEMI. These findings suggest that not only can prior disease and other factors influence prognosis, they may also modify the disease process and clinical presentation.

To what extent our findings reflect the pharmacological effects of the investigated medications or simply are associated with more preexisting and advanced disease in non-STEMI patients and consequently use of more medications cannot be determined by this cross-sectional study. However, we did control for age and several other medical conditions known to be associated with a lower risk of presenting with ST-segment elevation. The strong and independent effect (lowering the risk of presenting with ST-segment elevation by 15% to 25% for each medication) makes it unlikely that the use of these agents reflects only the presence of more advanced disease. The effect on risk of ST-segment elevation did not translate into lower short-term mortality, which is the net result of a multitude of different influences (not least of these being age and comorbidities, which are associated with lower risk of STEMI but higher risk of death after AMI).

The medicines in the present study have well-documented effects, persuasively proved in a large number of trials and surveys, and have been shown to lower the risk of cardiovascular disease. However, the mechanisms by which they protect from adverse cardiovascular outcomes are quite different for the various agents we

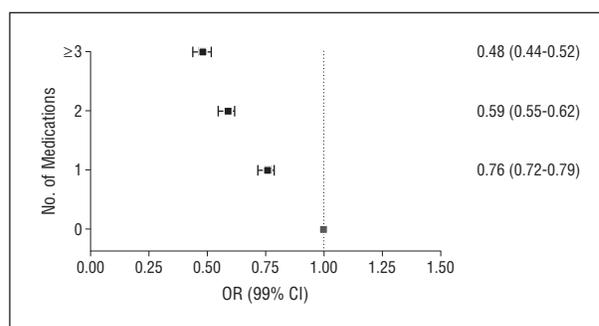


Figure 2. Multiply adjusted odds ratios (ORs) (99% confidence intervals [CIs]) for presenting with ST-segment elevation myocardial infarction (STEMI) vs non-STEMI. Results are shown in relation to the number of medications being received before presentation (regardless of medication type). To provide a more accurate presentation of the 99% CI intervals, ORs are represented on a linear scale after logarithmic transformation. The vertical dotted line indicates the reference value.

investigated. Aspirin has a well-documented antiplatelet or anticlotting effect.¹⁶ Accordingly, the protection from transmural ischemia through this antithrombotic effect is a probable mechanism. β -Blockers reduce the cardiac rate and myocardial metabolic demand, resulting in lower oxygen demand, less widespread infarction, and documented effects on survival.¹⁷ Angiotensin-converting enzyme inhibitors have been shown to exert cardioprotective effects by reducing myocardial and vascular hypertrophy.¹⁸⁻²⁰ Moreover, early initiation of ACE inhibitor therapy reduces mortality in STEMI patients.¹⁹ Finally, ACE inhibitors probably have a vascular protective effect by influencing atherosclerosis progression and plaque rupture, thereby reducing the cardiovascular mortality in high-risk patients. However, the mechanisms involved are not clear.²⁰

A large number of studies have demonstrated the benefit of treatment to lower lipid levels in primary and secondary prevention of cardiovascular disease.^{21,22} Early statin treatment is also associated with reduced mortality in patients with ACS,²³ although there is limited evidence of the benefits of use of statin therapy in AMI. Previously, data from the Swedish Register of Cardiac Intensive Care showed that early initiation of statin treatment in patients with AMI is associated with reduced 1-year mortality.¹¹ Data from the Myocardial Ischemia Reduction with Aggressive Cholesterol Lowering (MIRACL) Study demonstrated that immediate atorvastatin calcium therapy in patients with ACS was associated with reduced recurrent ischemia.²⁴ In addition to the effects of lowering total serum cholesterol levels and slowing the progression and promoting the regression of coronary atherosclerosis,²⁵ statins have demonstrated a number of other positive effects. Such pleiotropic effects, which include anti-inflammatory, antithrombotic, and plaque stabilization effects, may help to explain our findings.^{26,27}

From these and other data, it appears that previous use of medications may exert several effects on the coronary arteries, leading to less risk of transmural ischemia in AMI. Furthermore, with an increasing number of medications, the risk of STEMI decreased. With more restrictive thresholds for what is considered normal or desirable levels of blood pressure and serum lipid levels—

notably, serum cholesterol levels—a larger proportion in the population is likely to be treated with ACE inhibitors, β -blockers, or statins. According to our findings, this may have contributed to the declining severity in incident MI as reported in the Atherosclerosis Risk in Communities Study, which, with carefully documented methods, demonstrated a 2% yearly decline in the proportion of patients with AMI presenting with initial ST-segment elevation.²⁸ The authors noted that increasing knowledge about the benefits of primary prevention may have influenced the use of aspirin, β -blockers, and statins, but because they had no information on preadmission medication use and preexisting medical conditions, this hypothesis could not be tested in their data set. In our study population, the proportion of patients presenting with ST-segment elevation decreased during the study period. Concomitantly, there was an increase in the proportion of patients using medications. However, all multivariable analyses included year of admission.

Our study had several strong points. We studied a large, nationally representative population from a quality-of-care register (ie, the RIKS-HIA) with high coverage levels. More than 95% of all patients admitted to a coronary care unit in Sweden are currently included in the RIKS-HIA. However, only patients surviving long enough to be admitted could be included.

In 2001, the diagnostic criteria for MI were changed, with a lower limit for creatine kinase-MB fraction.¹² Patients who previously would have been diagnosed as having unstable angina pectoris in later years received a non-STEMI diagnosis. This fact would have contributed to a larger proportion of non-STEMI patients during the later part of the study. Even so, the effect of any of the 4 types of medication was strong and independent of the year of hospitalization.

A third limitation is that medications may simply reflect more severe underlying disease, which in itself would lead to less risk of developing STEMI. However, we did control for several premorbid conditions, with a persisting strong effect of all types of medication. Therefore, we believe it is unlikely that residual confounding could account for our findings in any major way.

In conclusion, among patients in the RIKS-HIA register presenting with a first AMI, previous treatment with aspirin, β -blockers, ACE inhibitors, and statins was substantially associated with less risk of presenting with STEMI, a more damaging and severe AMI. This finding held in men and women regardless of age. The risk decreased with the number of medications used before the AMI. Increasing use of preventive medications in the population could contribute to decreasing severity in AMI, further emphasizing the importance of medical treatment in patients perceived to be at high risk.

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INVITED COMMENTARY

Primary Prevention of Myocardial Infarction

Putting the Evidence to Use

Seldom, if ever, have we had as much knowledge to prevent a future epidemic. What is lacking is the wisdom to act upon that knowledge.
Jonathan S. Allan, DVM¹

Since the discovery of coronary artery obstruction by Edward Jenner in the 1780s² and the introduction of the concept of MI by Sir Thomas Lewis in 1934,^{2,3} our understanding of the causes of and methods of preventing MI has grown tremendously.^{4,5}

For asymptomatic high-risk patients (primary prevention)⁶ and patients with established coronary artery disease (secondary prevention),⁷ prevention has relied on pharmacologic treatment coupled with lifestyle changes to normalize the major cardiovascular risk factors of hypertension, dyslipidemia, smoking, sedentary lifestyle, obesity, and hyperglycemia.⁸ These preventive measures along with improved inpatient⁴ and outpatient cardiac care have resulted in a decline in mortality associated with MI.⁹

Björck et al provide compelling information regarding the effect of prior pharmacologic treatment to reduce risk factors of coronary artery disease on initial presentation of MI. Using data from a registry of patients admitted with AMI to Swedish coronary care units, they compared the treatment histories of patients with non-STEMI with those of patients with transmural STEMI. The former is considered less serious, so evidence that prior treatment with aspirin, statins, ACE inhibitors, and β -blockers resulted in a greater likelihood of experiencing non-STEMI rather than STEMI would be evidence for the efficacy of these treatments. Of the 103 459 patients in their study, 43.5% experienced STEMI and 56.5% had non-STEMI. Patients with STEMI were found to have less often used any of the 4 preventive medications prior

to their first-ever episode of MI: aspirin use was 20.2% in the STEMI group vs 33.3% in the non-STEMI group (adjusted OR, 0.72), β -blocker use was 22.2% in the STEMI group vs 33.0% in the non-STEMI group (adjusted OR, 0.82), ACE inhibitor use was 10.2% in the STEMI group vs 15.7% in the non-STEMI group (adjusted OR, 0.84), and statin use was 8.0% in the STEMI group vs 14.8% in the non-STEMI group (adjusted OR, 0.79). Strikingly, Björck et al found that a higher proportion of STEMI patients used no medication at all prior to the first episode of MI compared with non-STEMI patients (61.4% vs 45.9%).

An important limitation of the study by Björck et al is that treatment was not randomized. However, their findings are consistent with those of other researchers. GRACE registry data documented the effectiveness of long-term aspirin use in reducing the incidence of STEMI and lowering mortality in patients with coronary artery disease.¹⁰ Go et al¹¹ demonstrated that statin and β -blocker use was associated with lower odds of presenting with an AMI than with stable angina. Given the large sample size in the study by Björck et al compared with earlier published data, the risk of random error is extremely small.

A perplexing finding of the study by Björck et al is that the prevalence of non-STEMI was higher in younger patients, although no overall difference was observed in mortality at 30 days or 1 year between the STEMI and non-STEMI groups, which corroborates the results of earlier published OPERA registry data.¹² Despite a higher risk of presenting with ST-segment elevation, mortality risk did