Association Between Myocardial Infarction and Fractures
An Emerging Phenomenon

Yariv Gerber, PhD; L. Joseph Melton III, MD, MPH; Susan A. Weston, MS; Véronique L. Roger, MD, MPH

Background—Data on the association between myocardial infarction (MI) and fractures are scarce. Recent changes in the epidemiology of MI justify exploring this relationship. We evaluated whether MI constitutes a risk factor for osteoporotic fracture and examined secular trends in this association.

Methods and Results—Consecutive Olmsted County, Minnesota, residents with incident MI diagnosed in 1979 to 2006 and community control subjects individually matched (1:1) to cases on age, sex, and year of onset (n=6642) were followed up through 2009. Outcome measures were time to osteoporotic fracture, overall and by anatomic site, and death. Fracture incidence rates were stable in controls but increased markedly over time among MI cases. Accordingly, although an overall excess of fracture risk after MI was observed (adjusted hazard ratio, 1.32; 95% confidence interval, 1.12 to 1.56), substantial temporal variations were noted (1979 to 1989: hazard ratio, 0.81; 95% confidence interval, 0.58 to 1.12; 1990 to 1999: hazard ratio, 1.47; 95% confidence interval, 1.10 to 1.96; 2000 to 2006: hazard ratio, 1.73; 95% confidence interval, 1.32 to 2.27; P for trend <0.001). Trends were similar regardless of age, sex or fracture site. Conversely, the overall hazard ratio for death in MI cases versus controls did not change materially despite a continuous decline in 30-day case fatality rate (12.5% in 1979 to 1989; 6.7% in 2000 to 2006). Observed changes in the baseline prevalence of cardiovascular risk factors, MI characteristics, and comorbidities did not fully account for the trends in fracture risk.

Conclusions—Over the past decades, the association between MI and osteoporotic fractures increased steadily. The trend is consistent with the displacement of post-MI outcomes toward noncardiovascular events, highlighting the need for comprehensive prevention strategies to accommodate the changing epidemiology of MI. (Circulation. 2011;124:297-303.)

Key Words: epidemiology ▪ fractures, bone ▪ myocardial infarction ▪ prevention

A ccumulating evidence links vascular factors to osteoporosis and fractures,1,2 yet much of it is conflicting.3–5 Although several studies included an assessment of fracture risk in patients with cardiovascular disease,6,7 the specific association between myocardial infarction (MI) and fractures has rarely been studied, and temporal trends have not previously been evaluated. Because the burden of MI has shifted in recent decades toward women and the elderly,8,9 in whom osteoporosis is also prevalent,10 the nature and strength of the association need further clarification, the urgency of which is anchored in the major clinical and public health impact of both MI and fractures in aging populations.11,12 Although we and others have recently demonstrated profound changes in the outcomes of MI, characterized by reduced short-term case fatality rates and a displacement of deaths toward noncardiovascular causes,9,13,14 the implications of these important observations on comorbid events among aging MI survivors have not been fully addressed. Because atherosclerotic disease and fractures likely share a common biological pathway,1–7 we used a population-based cohort study design to examine whether incident MI was associated with an excess risk of subsequent osteoporotic fracture, whether this association had changed over time, and whether risk factors for fracture among MI subjects could be identified to help design preventive strategies.

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Methods

Setting, Design, and Participants
This research was conducted in Olmsted County, Minnesota, a location well suited for disease association studies because comprehensive medical records for the residents are available for review and are accessible through a centralized index of diagnoses made by all medical care providers serving the local population (with a few exceptions that are not relevant to this study).15 After approval was granted by the appropriate Institutional Review boards, a matched
cohort study was carried out using this unique record linkage system (the Rochester Epidemiology Project). Because almost all Olmsted County residents are represented in this system, this data source provides a virtually complete enumeration of the source population for many decades. Indeed, the Rochester Epidemiology Project Census enumeration has recently been compared with the US Census enumeration for Olmsted County and demonstrated to have excellent validity for every Census year since 1930.16

Exposed subjects (cases) were consecutive patients with an incident (ie, first ever) MI occurring between 1979 and 2006 (n=3321), identified with an algorithm previously described and validated according to standardized criteria.8,17 Unexposed subjects (controls) were randomly selected from the Olmsted County population and individually matched (1:1) to each case on age (±3 years), sex, and incidence year. By matching on incidence year, we ensured that the controls had sought medical care the same year the case experienced the MI. Among all eligible controls who met the matching criteria, we selected the one with the medical record registration number closest to that of the corresponding case. Because information on exposures before the index date was obtained from community medical records, this matches for the duration of prior medical record documentation, ensures similar opportunities for ascertainment of comorbidities between the compared groups, and avoids biases inherent in many observational studies (eg, differential recall, nonresponse bias, and survivor bias). Potential controls with a prior MI were excluded. All participants were followed up through their complete (inpatient and outpatient) medical records in the community from index time to death or the most recent clinical contact (last follow-up, July 2009). Because virtually all local residents have at least 1 contact with the medical system in any 3-year period and the majority are seen annually, follow-up is quite complete.15

Outcome Measures
Each subject’s medical record was searched electronically for the occurrence of any fracture through comprehensive diagnostic and surgical indexes that record all diagnoses made over time and hence are not limited to chief complaints. Consequently, fractures found incidentally on workups for other problems are included in this system, as are symptomatic fractures presenting for treatment. Because we have access to complete inpatient and outpatient medical records in the community, our data collection processes also identify fractures that occurred elsewhere through follow-up care, patient reports of interim medical history, or subsequent radiographs. This optimizes ascertainment of fractures, which should be nearly complete because the vast majority come to medical attention either directly or indirectly.18 Fractures were classified by anatomic site, with those of the proximal femur (hip), lumbar/thoracic vertebrae (spine), distal forearm (wrist), or humerus (shoulder) regarded as osteoporotic.10 However, all fractures were included in the analysis, and the date of first fracture event both before and after the index time, overall and by anatomic site, was used as the diagnosis date.

Death was ascertained through multiple sources, including death certificates filed in Olmsted County, autopsy reports, obituary notices, and electronic death certificate files obtained from the Section of Vital Statistics, Minnesota Department of Health.17

Additional Clinical Data
For the cases, the medical record was reviewed to ascertain cardiovascular risk factors and MI characteristics through the use of medical indexes that recorded as of the index date or at the closest time before hospital admission. Cigarette use was classified into current or never. Although the compared groups were matched by age and sex, adjustment was carried out for these variables in the survival models to account for possible differences in their distributions as follow-up progressed, owing to early censoring of nonfrailer participants (who might be underrepresented in the MI group). However, similar results were obtained in sensitivity analyses that excluded age and sex from the models. Subsequently, Cox proportional hazards regression models were constructed to evaluate the adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) for osteoporotic fracture (overall and by anatomic site), nonosteoporotic fracture, and death in MI cases compared with controls. Because the proportionality assumption was not met in some of the models, follow-up was uniformly truncated at 5 years, when proportionality was satisfied for all models. This also reduced differences in follow-up time between MI cases and controls (mean difference, 0.7 years). Analyses were carried out for the entire sample and for the above-mentioned 3 time-period categories separately. Temporal trends were formally tested by including a year-by-MI status interaction term in the models. For the latter purpose, year was treated as a continuous variable. Stratified analyses by age group, sex, and prior fracture were performed. Diverging trends by age, sex, and prior fracture status were assessed by including 3-way interaction terms (eg, year by age by MI status) in the models, with the pertinent 2-way interaction terms included.

Secular trends in the age-adjusted prevalence of traditional risk factors and MI characteristics among the cases only and of measured comorbidities in both cases and controls were assessed with logistic regression models, and the adjusted association with fracture risk was estimated with Cox proportional hazards regression models. Analyses were performed with SAS version 8.2 (SAS Institute Inc, Cary, NC).

Table 1. Baseline Characteristics Among Olmsted County, Minnesota, Residents With Incident Myocardial Infarction in 1979 to 2006 Compared With Community Control Subjects

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Myocardial Infarction Status</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases</td>
</tr>
<tr>
<td>n</td>
<td>3321</td>
</tr>
<tr>
<td>Age, mean (SD), y</td>
<td>67.4 (14.2)</td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>1425 (43)</td>
</tr>
<tr>
<td>Prior osteoporotic fracture, n (%)</td>
<td>476 (14)</td>
</tr>
<tr>
<td>Charlson Comorbidity Index, median (quartiles 1; 3)</td>
<td>1 (0, 3)</td>
</tr>
</tbody>
</table>

comorbid conditions weighted according to the degree to which they predict mortality (1, 2, 3, or 6 points for each).

Statistical Analysis
Fracture incidence rates with person-time denominators were calculated for MI cases and controls. Linear increase in rates over time was assessed with the Mantel-Haenszel trend test.

Fracture-free survival curves were then plotted for 3 time periods separately: early years (1979 to 1989), middle years (1990 to 1999), and late years (2000 to 2006), with adjustment for age, sex, and prior osteoporotic fracture. This was done with the direct adjustment method developed by Zhang et al.20 Although the compared groups were matched by age and sex, adjustment was carried out for these variables in the survival models to account for possible differences in their distributions as follow-up progressed, owing to early censoring of nonfrailer participants (who might be underrepresented in the MI group). However, similar results were obtained in sensitivity analyses that excluded age and sex from the models. Subsequently, Cox proportional hazards regression models were constructed to evaluate the adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) for osteoporotic fracture (overall and by anatomic site), nonosteoporotic fracture, and death in MI cases compared with controls. Because the proportionality assumption was not met in some of the models, follow-up was uniformly truncated at 5 years, when proportionality was satisfied for all models. This also reduced differences in follow-up time between MI cases and controls (mean difference, 0.7 years). Analyses were carried out for the entire sample and for the above-mentioned 3 time-period categories separately. Temporal trends were formally tested by including a year-by-MI status interaction term in the models. For the latter purpose, year was treated as a continuous variable. Stratified analyses by age group, sex, and prior fracture were performed. Diverging trends by age, sex, and prior fracture status were assessed by including 3-way interaction terms (eg, year by age by MI status) in the models, with the pertinent 2-way interaction terms included.

Secular trends in the age-adjusted prevalence of traditional risk factors and MI characteristics among the cases only and of measured comorbidities in both cases and controls were assessed with logistic regression models, and the adjusted association with fracture risk was estimated with Cox proportional hazards regression models. Analyses were performed with SAS version 8.2 (SAS Institute Inc, Cary, NC).

Results
The study included 6642 subjects: 3321 incident MI cases (mean age at baseline, 67.4 years; SD, 14.2 years; 43% women) and 3321 age- and sex-matched controls. On average, MI cases had greater comorbidities than controls, whereas the distribution of prior osteoporotic fractures was similar (Table 1).
During a mean follow-up of 4.0 years (SD, 1.7 years), 561 participants developed an incident osteoporotic fracture; 1650 subjects died. The overall rates per 1000 person-years in MI cases versus controls were 24.1 versus 20.9 for fracture and 90.3 versus 39.8 for death, respectively. However, although fracture rates were stable in the control group (22.2 in 1979 to 1989, 19.1 in 1990 to 1999, and 21.7 in 2000 to 2006; \( P \) for trend = 0.88), a steady increase was noted among MI patients (16.3, 22.5, and 34.0, respectively; \( P \) for trend < 0.001). This secular trend persisted after adjustment for age, sex, and prior osteoporotic fracture (Figure 1). On a relative scale, although the overall HR for fracture in MI versus controls was moderate (1.32; 95% CI, 1.12 to 1.56), substantial temporal variations were evident (in 1979 to 1989: HR, 0.81; 95% CI, 0.58 to 1.12; in 1990 to 1999: HR, 1.47; 95% CI, 1.10 to 1.96; and in 2000 to 2006: HR, 1.73; 95% CI, 1.32 to 2.27; \( P \) for trend < 0.001; Table 2). Repeating this analysis with shorter time-period categories supported a steady, monotonic in-

Table 2. Temporal Trends in the Associations Between Incident Myocardial Infarction and 5-Year Risk of Osteoporotic Fracture, Overall and in Specific Subgroups, Nonosteoporotic Fracture, and Death Among Olmsted County, Minnesota, Residents in 1979 to 2006

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Time Period</th>
<th>Cases</th>
<th>HR (95% CI)*</th>
<th>Cases</th>
<th>HR (95% CI)*</th>
<th>Cases</th>
<th>HR (95% CI)*</th>
<th>Cases</th>
<th>HR (95% CI)*</th>
<th>( P_{\text{trend}} )</th>
<th>( P_{\text{interaction}} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteoporotic fracture</td>
<td>Overall</td>
<td>561</td>
<td>1.32 (1.12–1.56)</td>
<td>155</td>
<td>0.81 (0.58–1.12)</td>
<td>191</td>
<td>1.47 (1.10–1.96)</td>
<td>215</td>
<td>1.73 (1.32–2.27)</td>
<td>&lt;0.001</td>
<td>...</td>
</tr>
<tr>
<td>Stratified by baseline age groups</td>
<td>1979–1989</td>
<td>190</td>
<td>1.40 (1.05–1.86)</td>
<td>58</td>
<td>0.99 (0.59–1.66)</td>
<td>62</td>
<td>1.63 (0.99–2.71)</td>
<td>70</td>
<td>1.67 (1.04–2.70)</td>
<td>0.02</td>
<td>0.68</td>
</tr>
<tr>
<td></td>
<td>&gt;75 y</td>
<td>371</td>
<td>1.29 (1.05–1.58)</td>
<td>97</td>
<td>0.72 (0.47–1.09)</td>
<td>129</td>
<td>1.41 (0.99–2.00)</td>
<td>145</td>
<td>1.79 (1.29–2.49)</td>
<td>0.001</td>
<td>...</td>
</tr>
<tr>
<td>Stratified by sex</td>
<td>Overall</td>
<td>393</td>
<td>1.35 (1.11–1.65)</td>
<td>115</td>
<td>0.71 (0.48–1.04)</td>
<td>148</td>
<td>1.72 (1.24–2.39)</td>
<td>130</td>
<td>1.81 (1.27–2.56)</td>
<td>&lt;0.001</td>
<td>0.62</td>
</tr>
<tr>
<td></td>
<td>Women</td>
<td>168</td>
<td>1.25 (0.92–1.69)</td>
<td>40</td>
<td>1.20 (0.64–2.23)</td>
<td>43</td>
<td>0.79 (0.43–1.46)</td>
<td>85</td>
<td>1.62 (1.06–2.49)</td>
<td>0.25</td>
<td>...</td>
</tr>
<tr>
<td>Stratified by prior fracture status</td>
<td>Overall</td>
<td>347</td>
<td>1.45 (1.17–1.79)</td>
<td>90</td>
<td>1.03 (0.68–1.57)</td>
<td>119</td>
<td>1.32 (0.92–1.89)</td>
<td>138</td>
<td>1.98 (1.41–2.79)</td>
<td>0.008</td>
<td>0.98</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>214</td>
<td>1.13 (0.86–1.48)</td>
<td>65</td>
<td>0.58 (0.35–0.99)</td>
<td>72</td>
<td>1.68 (1.04–2.69)</td>
<td>77</td>
<td>1.36 (0.86–2.13)</td>
<td>0.002</td>
<td>...</td>
</tr>
<tr>
<td>Restricted to specific anatomic sites</td>
<td>Overall</td>
<td>183</td>
<td>1.22 (0.91–1.63)</td>
<td>56</td>
<td>0.60 (0.34–1.06)</td>
<td>54</td>
<td>1.94 (1.12–3.34)</td>
<td>73</td>
<td>1.42 (0.89–2.25)</td>
<td>0.004</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>Spine</td>
<td>415</td>
<td>1.45 (1.20–1.76)</td>
<td>109</td>
<td>1.01 (0.69–1.47)</td>
<td>147</td>
<td>1.43 (1.03–1.99)</td>
<td>159</td>
<td>1.94 (1.41–2.66)</td>
<td>0.003</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>Nonspine</td>
<td>269</td>
<td>1.42 (1.12–1.81)</td>
<td>74</td>
<td>0.92 (0.58–1.47)</td>
<td>104</td>
<td>1.75 (1.18–2.58)</td>
<td>91</td>
<td>1.66 (1.09–2.51)</td>
<td>0.01</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>Hip</td>
<td>114</td>
<td>1.47 (1.01–2.12)</td>
<td>27</td>
<td>0.91 (0.42–1.96)</td>
<td>35</td>
<td>1.32 (0.68–2.57)</td>
<td>52</td>
<td>2.00 (1.14–3.50)</td>
<td>0.11</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>Forearm</td>
<td>56</td>
<td>1.69 (1.00–2.87)</td>
<td>16</td>
<td>1.39 (0.52–3.72)</td>
<td>14</td>
<td>0.62 (0.19–2.01)</td>
<td>26</td>
<td>3.57 (1.50–8.51)</td>
<td>0.18</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>Humerus</td>
<td>781</td>
<td>1.20 (1.04–1.38)</td>
<td>187</td>
<td>1.06 (0.80–1.42)</td>
<td>315</td>
<td>1.26 (1.01–1.57)</td>
<td>279</td>
<td>1.21 (0.96–1.53)</td>
<td>0.19</td>
<td>...</td>
</tr>
<tr>
<td>Nonosteoporotic fracture</td>
<td>Overall</td>
<td>1,650</td>
<td>2.47 (2.24–2.74)</td>
<td>574</td>
<td>2.47 (2.08–2.93)</td>
<td>569</td>
<td>2.30 (1.93–2.73)</td>
<td>507</td>
<td>2.77 (2.30–3.34)</td>
<td>0.55</td>
<td>...</td>
</tr>
</tbody>
</table>

HR indicates hazard ratio; CI, confidence interval.
*HRs are adjusted for age, sex, and prior osteoporotic fracture.
increase in fracture risk associated with MI (Figure 2). This increasing trend applied to both vertebral and nonvertebral fractures and did not differ significantly by age, sex, or prior fracture status, whereas little association was found with nonosteooporotic fractures, indicating the specificity of the association between MI and osteoporotic fractures (Table 2). We additionally explored the age-, sex-, and prior osteoporotic fracture-adjusted trend in the association between MI and incident osteoporotic fractures, distinguishing ST-segment elevation (P for trend = 0.02) from non–ST-segment elevation (P for trend = 0.005), and found a robust temporal increase for both MI types. Although the overall adjusted association with death was largely constant over time, a substantial decline in 30-day case fatality rate was observed among MI patients (12.5% in 1979 to 1989, 10.7% in 1990 to 1999, and 6.7% in 2000 to 2006; P for trend < 0.001). This decline was driven mainly by reductions in fatalities among women (17.3% in 1979 to 1989, 9.9% in 2000 to 2006) and patients >75 years of age (22.4% in 1979 to 1989, 13.5% in 2000 to 2006), together responsible for 83% of all early deaths.

We further evaluated temporal trends in the prevalence of traditional cardiovascular risk factors and MI characteristics among the cases and their relationship to subsequent fracture risk. These 2 complementary analyses are summarized in Table 3. Variables associated with an excess fracture risk that increased in prevalence over time were hypertension and non–ST-segment–elevation MI. Obesity, on the other hand, which was inversely associated with fracture, more than doubled in rate during the study period.

We also examined the relationship of comorbidities with osteoporotic fracture and found positive associations with heart failure, peripheral vascular disease, stroke, chronic obstructive pulmonary disease, renal disease, rheumatologic disease, and cancer. However, no diverging trends in the prevalence of these comorbidities between MI patients and controls were detected over time, with the exception of heart failure, which was stable in controls but declined among MI cases. The overall comorbidity burden, as assessed by the Charlson index, increased over time (P < 0.001) in both cases (median, 2 [quartiles 1 and 3, 1 and 5] in 1979 to 1989; 2 [quartiles 1 and 3, 1 and 5] in 2000 to 2006) and controls (0 [quartiles 1 and 3, 0 and 1] in 1979 to 1989; 1 [quartiles 1 and 3, 0 and 3] in 2000 to 2006). In multivariable Cox models adjusted for age, sex, prior fracture, and overall comorbidity, the HRs for fracture in MI patients versus controls were 0.72 (95% CI, 0.51 to 1.02) in 1979 to 1989, 1.38 (95% CI, 1.03 to 1.85) in 1990 to 1999, and 1.50 (95% CI, 1.13 to 1.98) in 2000 to 2006 (P for trend < 0.001).

**Discussion**

The present population-based study provides strong evidence for an emerging association between MI and the risk of osteoporotic fracture in the community. Using data spanning 30 years (1979 to 2009), originating from carefully characterized cohorts with complete ascertainment of outcomes, we detected a steady increase in fracture risk after MI, a novel finding not observed in the general population. This alarming temporal trend was robust across patient groups and fracture sites. Long-term mortality risk, on the other hand, remained largely unchanged in MI cases compared with controls, despite a substantial decline in 30-day case fatality rate. Although several characteristics were associated with an
substantial proportion of these fractures are asymptomatic

tainment bias is theoretically possible for vertebral fractures
eral explanations may be considered for our findings. Ascer-
more intensively with drugs than before.9,13,22 Polypharmacy
suggested,25,26 with a possible relation to MI risk.27 Although

calcium supplementation on falls and fractures have also been
of diagnosed comorbidities9,21,22 and are therefore treated
because more recent MI patients have an increasing number
of cardiovascular risk factors, MI characteristics,
and comorbidities as measured at study entry were examined
as potential mediators of the relationship between fracture
risk after MI and time; however, no such mechanism was
evident. Furthermore, heart failure, which was recently found
to be a strong risk factor for osteoporotic fracture in large-
scale epidemiological studies,6,7 has been shown previously
to decline in occurrence after MI in this cohort.32 Stroke after
MI, on the other hand, had increased somewhat from 1979 to
1988, with no further rise observed afterward.33 Thus, secular
changes in the rates of heart failure and stroke after MI are
probably not responsible for the observed trends in fracture
risk.

Over the past 2 decades, medical breakthroughs related to
technological developments, invasive procedures, and ad-
vanced drugs have led to a steady reduction in cardiovascular
mortality.17 Consequently, the population of MI survivors has
increased, often at the expense of increased disability,34 and
there is increased awareness of the burden of frailty in these
patients.35 Indeed, our data reveal that the dramatic decline in
30-day case fatality rate in Olmsted County was driven
primarily by improved short-term survival of women and
elderly patients. Hence, the trend of increasing fracture risk,
an expected outcome of frailty-related impairments,34,36 in the
aftermath of MI may be a result. Furthermore, although
long-term survival remained largely unchanged among 30-
day survivors in this population13 and others,14 the deaths
shifted from cardiovascular to noncardiovascular causes.13,14
This shift may be partially attributable to the trend of
increased fracture risk after MI identified here because
fracture confers a high mortality risk, especially in men.10,37

Limitations, Strengths, and Clinical Implications

These data emanate from a single Midwestern community
that is predominantly white.15 Yet, the incidence of fractures
in this community is quite comparable to national figures for
US whites.38 Although no population can be representative of
the nation as a whole, these findings should be reexamined in
different racial and ethnic groups. Measurements of bone
mineral density or biochemical markers of bone turnover
were not routinely performed, so the role of bone loss in fracture risk could not be assessed directly. In addition, data on pharmacological treatments possibly related to fractures were not available, nor were data on healthcare encounters (eg, number of visits in a healthcare episode). Finally, temporal changes in definition and ascertainment of MI have occurred over the very long time period studied, as was the case with several covariates evaluated, including hypertension, diabetes mellitus, hyperlipidemia, chronic obstructive pulmonary disease, and renal disease. As in any other secular trend analysis, caution should be taken in the interpretation of these results.

Our study, conducted in a geographically defined population, has several important strengths. Addressing the crucial and challenging issue of chronic disease burden requires the ability to study cohorts relevant to real-world clinical practice such as the present ones. Detecting unfavorable trends that call for intervention requires longitudinal data as can be obtained under the auspices of the Rochester Epidemiology Project.

Our findings, demonstrating a substantial increase in fracture risk among contemporary survivors of an MI, have important clinical and public health implications. Fracture prevention initiatives after MI are likely warranted, particularly among high-risk patient groups such as the elderly, women, and those with prior fracture and/or coexisting illnesses. Furthermore, deficits such as sensory impairments, locomotor disturbances, and cognitive disorders, which are frequently overlooked in the traditional disease-specific model of cardiac care applied to MI patients, should be assessed and treated. Participation in secondary prevention programs, found to be consistently underused in this population as in others, should be aggressively pursued given the beneficial role of physical activity for the prevention of bone loss and falls, crucial elements in fracture prevention.

Conclusions

In this geographically defined population, we found a large excess risk of osteoporotic fracture after MI, which increased markedly over the past 3 decades. The trend applied to all segments of the population and all fracture sites. Given the deleterious impact of fracture on outcomes, fracture prevention is required in the aftermath of MI, with a particular focus on the elderly, women, and patients with coexisting illnesses.

Acknowledgments

We are indebted to Ruoxiang (Rochelle) Jiang for computer programming and data analysis and to Mary G. Roberts and Kristie K. Shorter for administrative assistance.

Sources of Funding

This work was supported by grants from the National Institutes of Health (P01 AG04875 to Dr Melton; R01 HL59205 and R01 HL72435 to Dr Roger) and the National Institute on Aging (R01 AG034676). Dr Roger is an Established Investigator of the American Heart Association. The funding sources played no role in the design, conduct, or reporting of this study.

Disclosures

None.

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CLINICAL PERSPECTIVE

Accumulating evidence links atherosclerotic disease to osteoporosis, but the specific association between myocardial infarction (MI) and fractures has rarely been studied, and temporal trends have not been evaluated. Using data spanning 30 years (1979 to 2009), originating from carefully characterized cohorts (3321 consecutive incident MI patients and 3321 community-based non-MI control subjects individually matched on age, sex, and year of onset) with complete ascertainment of fractures, we detected a steady increase in osteoporotic fracture risk after MI, a novel finding not observed in the general population. This alarming temporal trend was robust across patient groups and fracture sites and is consistent with the displacement of post-MI outcomes toward noncardiovascular events. Thus, fracture prevention initiatives after MI are likely warranted, particularly among high-risk patient groups, such as the elderly, women, and those with prior fracture and/or coexisting illnesses. Furthermore, frailty symptoms, which are frequently overlooked in the traditional disease-specific model of cardiac care applied to MI patients, should be assessed and treated. Participation in secondary prevention programs should be aggressively pursued given the beneficial role of physical activity for the prevention of bone loss and falls, crucial elements in fracture prevention.
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_Circulation_. 2011;124:297-303; originally published online June 27, 2011;
doi: 10.1161/CIRCULATIONAHA.110.007195
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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