Changes in Disability Before and After Myocardial Infarction in Older Adults

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Background: Disability in older adults is thought to occur primarily as a consequence of clinical disease episodes. However, the temporal relationship between clinical disease and disability has received little systematic attention.

Methods: Data from a prospective population-based study of 2812 older adults were analyzed to examine changes in disability before and after acute myocardial infarction. Disability outcomes included up to 9 yearly assessments of disability in activities in daily living, disability in basic physical functions, and disability in tasks requiring basic mobility and strength.

Results: A total of 279 myocardial infarctions occurred during 9 years of follow-up. After adjustment for age and sex, the average yearly increase in disability in activities of daily living and basic physical functions was not significantly greater in the 1-year period after myocardial infarction than in the 3-year period before myocardial infarction (P values > .20). Disability in basic mobility and strength showed a significantly greater increase in the year after myocardial infarction (P = .02). The results did not change after adjustment for comorbidity and chest pain or when restricted to incident cases of myocardial infarction or survivors. An additional exploratory analysis suggested that the rate of increase in some forms of disability may start to accelerate at about 1 year before the event, rather than after the event.

Conclusions: The increase in disability after myocardial infarction may form a continuation of increases that occur before the event and challenge commonly held notions about the temporal relationship between clinical disease and disability. Changes in disability before acute disease episodes may be related to subclinical disease.

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STUDY POPULATION

Data come from the New Haven site of the Established Populations for the Epidemiologic Studies of the Elderly (EPESE) project, 1 of 4 sites funded by the National Institute on Aging, Bethesda, Md. Details of the study design have been described previously.15 Briefly, the New Haven EPESE cohort was based on a stratified probability sample of the noninstitutionalized New Haven population 65 years and older. At baseline, the cohort was composed of 1169 men and 1643 women, for a total of 2812 subjects (response rate, 82%). Baseline data collection took place during in-home face-to-face interviews in 1982. Follow-up interviews were conducted at yearly intervals through 1990/1991, including face-to-face interviews in 1985 and 1988 and telephone interviews in all other years. There was complete information on vital status for the entire follow-up period. The study was approved by the Human Investigation Committee of Yale University's Medical School, and all participants provided written informed consent.

ASCERTAINMENT OF MYOCARDIAL INFARCTION

Cases of AMI were identified through a surveillance system that conducted weekly reviews of all admissions in the 2 local hospitals. Additional information on hospital admissions was obtained from the Medicare Part A Beneficiary Bill History data of The Centers for Medicare & Medicaid Services. Matching data on hospitalization from these 2 sources indicated that the surveillance system identified 95% of all coronary heart disease–related admissions. All hospitalizations with a discharge diagnosis of AMI (International Classification of Diseases, Ninth Revision, Clinical Modification [ICD-9-CM] codes 410.0-410.9) were identified, and the medical records were reviewed with a standardized abstraction instrument.18 Records of subjects with a discharge diagnosis of unstable angina were reviewed as well to capture potentially misclassified AMIs. A diagnosis of AMI required at least 2 of the following 3 criteria: (1) central anterior chest pain lasting at least 15 minutes or other symptoms consistent with myocardial infarction (ie, acute pulmonary edema, cardiogenic shock, or cardiac arrest); (2) characteristic electrocardiographic modifications (ie, new Q waves >0.04 seconds) or ST-segment elevations or depressions in at least 2 leads; or (3) typical rise and fall of serum creatine kinase (CPK) level with increase of the MB fraction (CPK-MB) to 4% or greater. If there were no Q waves, then serum enzyme elevations were required for the diagnosis of AMI. Subjects who had more than 1 AMI during follow-up were analyzed only in relation to their first event.

STUDY MEASURES

Assessment of disability was based on 3 widely used self-reported measures of basic daily functions, which represent a broad range of functional tasks and abilities that are considered part of overall disability among older adults.2,3 The first measure focuses on activities of daily living (ADL) and assesses the ability to perform 6 basic self-care tasks without help (eg, bathing, dressing, and eating).17 The second measure assesses the ability to perform 4 basic upper and lower extremity functions (pulling or pushing large objects; stooping, crouching, or kneeling; reaching or extending arms above shoulder level; and writing or handling small objects).18 The third measure assesses the ability to perform tasks requiring mobility and strength and included 3 items (walking up and down stairs; walking half a mile; and doing heavy work around the house).19 For ease of presentation, the last 2 measures will be referred to as Nagi and Rosow-Breslau disability, respectively. For each measure, a summary score was formed by adding the number of tasks the subject reported being unable to do. Thus, higher scores indicate greater disability.

Other variables included in the analysis included age (in years), sex, exertional chest pain, and comorbidity. Exertional chest pain was ascertained based on standardized questions from the London School of Hygiene Chest Pain Questionnaire.20 Chest pain was assessed during face-to-face interviews only, and we used information from the last pre-AMI interview for the present analysis. Comorbid conditions were ascertained annually on the basis of self-reported information of a physician-diagnosed history of AMI, stroke, cancer, high blood pressure, diabetes, hip fracture, and arthritis. We summed the number of positive responses across the 7 conditions from the first pre-AMI interview that was used in the analysis as measure of comorbidity.

STATISTICAL ANALYSIS

Yearly collected disability data were linked to the date of AMI, using data from up to the last 3 pre-AMI interviews and the first post-AMI interview. Multivariable regression models were used to estimate change in disability outcomes during the pre-AMI and post-AMI period. Owing to their nonnormal distributions, disability scores were considered as the number of tasks a person was unable to perform of the total number of tasks on each measure. They were then analyzed using generalized estimating equations with a logit link function, a binomial error structure, and an exchangeable working correlation matrix.21 Estimated regression coefficients were expressed as odds ratios, which represent the linear effect of a predictor variable on the odds of the proportion of tasks a person was unable to perform.

We first computed a regression model for each outcome with a single estimate for the average rate of yearly change in disability score during the entire pre-AMI to post-AMI period. We repeated this regression model after replacing the single estimate for change over time with 2 separate estimates for the average rate of yearly change during the pre-AMI and post-AMI period, respectively. Coefficients for the separate periods were estimated using piecewise regression models with an a priori–defined change point set at the time of the AMI.22 A standard Wald χ² test was used to test whether the change estimates for the pre-AMI and post-AMI periods were significantly different from each other, which constituted the formal test of our hypothesis. A negative result of no difference indicated that the estimated rate of change in disability score after AMI did not differ from the rate of change before AMI. Significance testing was based on a level of .05.

In a secondary analysis, we examined the degree to which pre-AMI exertional angina and comorbid conditions modified differences in pre-AMI or post-AMI changes in disability score. This was done by adding terms for each variable to the primary regression models. We also reran the primary regression models restricted to incident AMIs to see if differences between pre-AMI and post-AMI changes in disability score were similar among first AMIs and not due to previous episodes of AMI. Finally, we repeated the models after excluding subjects who died before the first post-AMI interview to see if post-AMI survival affected differences in pre-AMI and post-AMI changes in disability score. All regression models were adjusted for age and sex and computed using the GENMOD Procedure of SAS version 8 (SAS Institute Inc, Cary, NC).

We conducted an additional exploratory analysis to investigate the possibility that disability changes related to AMI may
begin at a different point in time than the actual date of the event itself. We plotted the longitudinal disability data in relation to the time of the AMI and fitted smoothed curves based on local-weighted regression functions without assumptions about the parametric form of the change in disability over time (S-Plus 6 for Windows; Insightful Corporation, Seattle, Wash). The results of this analysis informed an additional piecewise regression model with a different change point than the time of AMI.

RESULTS

A total of 279 subjects had an AMI during follow-up. Data from 1 subject was missing and was excluded from analysis. The mean±SD age at the time of AMI was 78.9±6.7 years; 138 subjects (50%) were female, and 41 (15%) were black; and for 195 subjects (70%), the event was an incident AMI (Table 1). Seventy-five subjects (27%) reported pre-AMI exertional chest pain, and subjects reported a mean±SD of 1.9±1.2 comorbid conditions, the most common of which were hypertension (58%) and arthritis (49%). The 3 yearly pre-AMI interviews occurred on average at 2.7 years (range, 4.2-1.8 years), 1.7 years (range, 3.1-0.5 years), and 0.6 years (range, 1.9-0.0 years) before the event, and the first post-AMI interview occurred on average at 0.5 years (range, 0.0-1.4 years) after the event.

Models with a single estimate for change over time showed that there was a significant increase in disability on all 3 measures during the entire pre-AMI to post-AMI period (Table 2). In piecewise regression models, the average rate of yearly increase in disability after AMI was not significantly different from the rate of increase before AMI for ADL disability (Wald $\chi^2=1.32; P=.34$) or Nagi disability (Wald $\chi^2=0.92; P=.34$). There was a significant difference in the rate of change in Rosow-Breslau disability score before and after AMI (Wald $\chi^2=5.06; P=.02$). The rate of yearly increase was higher after AMI (adjusted odds ratio [OR], 2.60; 95% confidence interval [CI], 1.56-4.35) than before AMI (adjusted OR, 1.37; 95% CI, 1.23-1.53).

To illustrate the nature of change in disability before and after AMI, we plotted the predicted disability scores for a typical 75-year-old woman (Figure 1). The scores were derived from the piecewise regression models (Table 2), converting the predicted proportion of tasks a person was unable to perform back to the original scale scores. As expected in older persons, disability scores increased over time. The average rate of increase was not higher after AMI for either ADL or Nagi disability. If anything, disability levels appeared to level off slightly after AMI on these outcomes. There was a greater increase in Rosow-Breslau disability score in the year after AMI compared with the pre-AMI period.

In secondary analysis, we found essentially the same pattern of findings after adjustment for pre-AMI chest pain and comorbid conditions or when we restricted the analysis to patients with incident AMI or surviving until the first post-AMI interview (Table 3). The pattern did not change even after adjustment for comorbidity scores obtained at the last pre-AMI interview rather than the first pre-AMI interview (data not shown). A stratified analysis by median age (78 years) and sex did not suggest that these associations varied substantially between younger-old and older-old patients or between male and female patients (data not shown).

In the additional exploratory analysis, we found a fairly uniform rate of increase in ADL disability score during the entire pre-AMI to post-AMI period, consistent with the findings presented previously (Figure 2). However, the rate of change in disability appeared to increase starting at about 1 year before AMI for Nagi and

### Table 1. Basic Characteristics of the Study Sample of 278 Patients

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at AMI, y</td>
<td>78.9 ± 6.7</td>
</tr>
<tr>
<td>Female</td>
<td>138 (50)</td>
</tr>
<tr>
<td>Black</td>
<td>41 (15)</td>
</tr>
<tr>
<td>Incident cases of AMI</td>
<td>195 (70)</td>
</tr>
<tr>
<td>Pre-AMI exertional chest pain</td>
<td>75 (27)</td>
</tr>
<tr>
<td>Baseline prevalence of medical conditions AMI</td>
<td>83 (30)</td>
</tr>
<tr>
<td>Stroke</td>
<td>24 (9)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>160 (58)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>66 (24)</td>
</tr>
<tr>
<td>Cancer</td>
<td>35 (13)</td>
</tr>
<tr>
<td>Hip fracture</td>
<td>14 (5)</td>
</tr>
<tr>
<td>Arthritis</td>
<td>137 (49)</td>
</tr>
<tr>
<td>No. of comorbidity conditions</td>
<td>1.9 ± 1.2</td>
</tr>
</tbody>
</table>

*Abbreviation: AMI, acute myocardial infarction.
*Data are given as number (percentage) or mean ± SD. Number of participants of the New Haven Established Populations for the Epidemiologic Studies of the Elderly study with an AMI during follow-up (1982-1991).

### Table 2. Yearly Change in Disability Outcomes From 3 Years Before AMI to 1 Year After AMI in 278 Patients

<table>
<thead>
<tr>
<th>Yearly Change</th>
<th>ADL Disability Score</th>
<th>Nagi Disability Score</th>
<th>Rosow-Breslau Disability Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Entire period</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yearly change</td>
<td>1.65 (1.44-1.88)</td>
<td>1.43 (1.32-1.56)</td>
<td>1.48 (1.35-1.63)</td>
</tr>
<tr>
<td>Separate periods (last 3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yearly change before AMI</td>
<td>1.74 (1.48-2.04)</td>
<td>1.47 (1.33-1.63)</td>
<td>1.37 (1.23-1.53)</td>
</tr>
<tr>
<td>Yearly change after AMI</td>
<td>1.25 (0.75-2.07)</td>
<td>1.20 (0.83-1.74)</td>
<td>2.60 (1.56-4.35)</td>
</tr>
<tr>
<td>Wald $\chi^2$</td>
<td>1.32</td>
<td>0.92</td>
<td>5.06†</td>
</tr>
</tbody>
</table>

*Abbreviations: ADL, activities of daily living; AMI, acute myocardial infarction.
*Data are given as odds ratio (95% confidence interval) unless otherwise specified. Odds ratios represent the average yearly change in the odds of the proportion of tasks a person is unable to perform, adjusted for age and sex.
†$P<.05$. 

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Rosow-Breslau disability scores. This pattern continued after AMI for Rosow-Breslau disability, but it leveled off for Nagi disability. Based on this pattern, we repeated the piecewise regression models for Nagi and Rosow-Breslau disability with a change point set at 1 year before AMI, rather than the time of AMI itself. In this analysis, the rate of change in the period starting at 1 year before AMI was significantly different than the rate of change prior to that point for Rosow-Breslau disability (Wald $\chi^2=8.86; P=.003$), but not for Nagi disability (Wald $\chi^2=3.10; P=.08$). Owing to the exploratory nature of this analysis, the results of these last 2 tests should be interpreted with caution.

**COMMENT**

Age-related disability is conceptualized as the result of clinical manifestations of underlying chronic diseases that affect the ability to perform basic physical and social functions.\(^2\) The purpose of this investigation was to provide a more systematic examination of the temporal relationship between clinical disease episodes and disability in the context of AMI. We did not find clear evidence that AMI is associated with a marked increase in disability in the year following the clinical event. Instead, our data suggest that the increase in disability after an AMI
is a continuation of the changes that occurred before the event.

There may be a number of reasons why we did not find greater differences in the rate of increase in disability after AMI relative to the pre-AMI period. Owing to the design of the study, we may have underestimated the magnitude of disability increases after AMI. We had relatively few data from the early post-AMI period, which limited our ability to estimate disability changes during the most acute phase of the disease, when more pronounced changes may have occurred. However, our primary focus was on the impact of clinical disease on longer-term changes in disability, rather than those occurring during the acute phase of disease. More data from the immediate post-AMI period might also have captured greater increases in disability among those who died early after AMI, but such data would also have had less relevance for a better understanding of the long-term implications of clinical disease for disability. Finally, it is possible that the lack of more substantial increases in post-AMI disability is due to patients changing the evaluation of their own abilities and reporting fewer limitations in response to a down-shifted standard of optimal functional capacity after acute clinical disease.

Another reason for the failure to find more pronounced differences in disability changes before and after AMI may be that increases in disability before AMI were greater than expected. Pre-AMI changes in disability may have occurred as a result of other aging-related processes unrelated to the AMI itself. Such changes could have been due to previous disease episodes, to symptomatic manifestations of the underlying disease process, such as chest pain, or to other comorbid medical conditions. However, none of these factors appeared to account for the lack of observed differences in pre-AMI and post-AMI changes on 2 of the 3 disability outcomes. These changes could also be related to other age-related physiological changes that may increase disability, such as decreased immune function and increased inflammation. Owing to the lack of data, we were unable to examine the influence of these processes on pre-AMI changes in disability.

Overall, our findings suggest that AMI is associated with significant preclinical increases in disability, that is, increases that occur before the event. Although we cannot infer a direct link between the preclinical increases in disability and the occurrence of the AMI from our data, they raise the possibility that they are related to each other. The strongest evidence for a possible link was found for basic physical functions, especially those requiring basic mobility and strength, such as walking and doing strenuous household tasks. In exploratory analysis, we found some evidence that a decline in these functions appeared to accelerate at about 1 year before AMI. One explanation for the preclinical changes in disability is that they are related to progression of subclinical disease in the cardiovascular system. Although there is little evidence that progression of subclinical cardiovascular disease is associated with increasing disability, data from the Cardiovascular Health Study have shown a significant cross-sectional relationship between subclinical cardiovascular disease and frailty, a condition of decreased overall physical health and function related to disability. The extent to which disability may be related to subclinical disease would also explain the prospective association between disability and risk for cardiovascular events such as AMI and stroke observed in previous studies.

To the extent that these findings can be replicated in other patient populations and for other conditions, they offer a new perspective on the relationship between chronic disease and age-related disability. Previous views of the disability process have suggested that chronic diseases characterized by acute, severe, although infrequent disease episodes are typically associated with dramatic increases in disability following such episodes. Although this may be generally true for the immediate aftermath of acute clinical events, our data indicate that disability changes beyond the acute phase may be much less dramatic and possibly reflect a continuation of preclinical changes in disability. The exact nature of these preclinical changes in disability is poorly understood, although we speculate that progression of subclinical disease may be a factor contributing to these changes.

This view of the relationship between disease and disability may have important clinical consequences. In clinical practice, minimizing the disabling consequences of chronic conditions is usually limited to rehabilitation efforts after a clinical episode. Our findings suggest that changes in disability deserve broader clinical attention, inasmuch as increases in disability may mark a period of increased risk for clinical disease episodes. This would mean that disability and its potential subclinical origins could be an important target for intervention, not only for the reduction of disability itself but also for the prevention of acute clinical disease episodes or for reducing the severity of such events. The results from a recent clinical trial show that such interventions are feasible and associated with reduced disability in older adults who live at home. Future trials should test the degree to which such interventions are effective in reducing the risk for clinical disease outcomes.

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