Health Status before and Mortality after Hip Fracture

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ABSTRACT

A cohort study was done to determine the direct impact of hip fracture on mortality in older people. Survival was compared between 211 hip fracture patients from a defined area and 201 non–hip fracture control subjects randomly selected from the same area. The mortality rate 1 year after hip fracture was 21.7%; 1-year mortality in the comparison group was 4.7%. The crude hazard ratio for hip fracture and mortality was 4.0 (95% confidence interval [CI] = 2.2, 7.4); adjusting for multiple health-related variables reduced it to 3.3 (95% CI = 1.7, 6.5). This finding suggests that the observed excess mortality after hip fracture is not explained by poor prefracture health status. (*Am J Public Health.* 1996;86:557–560)

Introduction

Although it is clear that hip fractures are a major public health problem, the impact of these fractures on mortality is uncertain because people who have hip fractures tend to have a number of other health problems that would put them at increased risk of death even if they did not have a hip fracture.[1–3] Numerous studies have compared mortality in hip fracture patients with expected mortality[4–20]; however, none of these studies has assessed the effect of prefracture health status on the observed excess mortality in hip fracture patients. In the study reported on in this paper, we compared the mortality in a group of hip fracture patients with that in a group of people from the same community who had not had a hip fracture, controlling for a number of health-related factors.

Methods

This study involved follow-up of subjects who were originally recruited for a population-based case–control study of hip fracture etiology.[21,22] The study population comprised Australians aged 65 years and over from a defined area in western Sydney. Subject recruitment occurred between March 1990 and August 1991; follow-up took place between November 1991 and May 1992.

Selection of Subjects

Most hip fracture subjects were recruited from Westmead Hospital. They were identified by daily contact with the coordinator of an early-discharge scheme for hip fracture patients and by weekly review of the logbooks of the accident and emergency department. Eleven other hospitals treating hip fracture patients from the study area were also contacted regularly to ascertain eligible hip fracture admissions. Patients were ineligible for the study if their hip fracture was definitely related to neoplastic disease (n = 3).

Four case subjects died before their baseline data were collected, so only basic demographic data and survival times were available for these individuals.

Area probability sampling was used to select subjects without hip fracture living in the community. Ten census collectors' districts in the study area were randomly selected by the Australian Bureau of Statistics, and all dwellings in these districts (n = 2560) were visited. (Collectors' districts are clusters of dwellings defined so that one census collector can cover all dwellings at the 5-year Australian census.) Data were sought for all people aged 75 years and older, and for a 10% random sample of those aged 65 to 74 years.

Six nursing homes and three hostels for the aged were randomly selected from the 28 nursing homes and 12 hostels in the study area. Five people aged 75 years and older were randomly chosen from each of these places.

Baseline and Follow-Up Data Collection

An interviewer-administered questionnaire was used to measure exposures of interest. Health status was assessed by asking subjects to rate their health as excellent, good, fair, or poor. Subjects were also asked if they had seen a doctor within the past year about any of the following conditions: diabetes, heart attack, emphysema, angina, Parkinson's disease, cancer, or osteoporosis. Current medication use was assessed by self-report.

The amount of physical activity was estimated from the average number of times per week in the previous year that subjects went for a walk lasting at least 15 minutes and from the average number of hours per week subjects worked in the house or garden. Body mass index was calculated from self-reported height and weight. The reproducibility of responses to these questions is reported elsewhere.[23]

Pfeiffer's Short Portable Mental Status Questionnaire was used to assess

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This paper was accepted August 25, 1995.

TABLE 1—Baseline Characteristics of Study Subjects: 412 Sydney Residents Aged 65 Years or Older

	Hip Fracture Subjects (n = 211), %	Non–Hip Fracture Subjects (n = 201), %
Females	82.5	65.7
Age group, y		
65–74	19.4	16.4
75–79	18.0	37.3
80–84	27.0	29.4
85–89	21.8	11.9
90–100	13.7	5.0
Residence at baseline		
Community (alone)	22.1	27.4
Community (other)	39.4	52.2
Nursing home	33.2	13.4
Hostel	5.3	7.0
Self-rated health[a]		
Excellent	24.2	25.3
Good	47.5	52.9
Fair	22.5	17.1
Poor	5.8	4.7
Number of medications		
0	11.1	4.5
1–3	36.5	44.8
4–6	38.0	32.3
7–14	14.4	18.4
Medical conditions		
Diabetes	6.8	10.0
Myocardial infarction	5.3	3.5
Parkinson's disease	5.8	2.0
Stroke	16.9	12.9
Arthritis	43.0	48.8
Emphysema	4.4	6.0
Angina	13.5	10.0
Osteoporosis	11.1	9.5
Cancer	4.4	11.4
Total conditions		
0	31.1	28.0
1–3	63.1	69.0
4–10	5.8	3.0
Falls in past year		
0	41.1	64.7
1–2	28.0	28.9
≥3	30.9	6.5
Mental state score[b]		
0–4	34.9	11.3
5–8	40.3	30.8
9–10	24.7	57.9
Walks per week, mean no.	3.7	3.5
Work done per week, mean no. hrs	9.4	12.9
Current smoker	15.0	11.0
Body mass index[c]		
15–22	52.8	34.5
23–24	26.7	35.6
25–35	20.5	29.9

[a]Data on self-rated health were not collected for subjects interviewed by proxy (87 hip fracture subjects, 28 control subjects).
[b]Mental state score was not assessed in 25 hip fracture subjects and 6 control subjects, most of whom were clearly cognitively impaired.
[c]Data for calculation of body mass index were incomplete for 50 hip fracture subjects and 27 control subjects.

cognitive status.[24] Hip fracture patients were interviewed when it was judged, in consultation with relatives or nursing home staff, that cognitive function had returned to its prefracture level. In the early stages of the study, mental state scores were not estimated for subjects in whom it was clear that a proxy respondent would be required (n = 27). For subjects unable to answer the full questionnaire (usually because of cognitive impairment), a shortened questionnaire was administered to proxy respondents. This did not include the question on self-rated health.

Follow-up data were collected at interviews conducted in order of date of baseline interview. Subjects (or proxy respondents) were contacted by telephone or in person.

Statistical Analysis

Survival analysis was used to analyze the data from this study. Cox proportional hazard models were constructed to calculate hazard ratios and 95% confidence intervals for the impact of hip fracture on mortality.

Prognostic variables included in the survival analyses were chosen (1) if they were thought a priori to be prognostically important (age, sex, place of residence, self-rated health, mental state, total number of medications, need for proxy respondent), or (2) if, in a log-rank test in the nonfracture group, the variable was significant at the .2 level in predicting mortality (amount of work and walking done, previous myocardial infarction, diabetes, and Parkinson's disease).

Results

Four hundred twelve subjects participated in this study: 211 hip fracture subjects (mean age = 81.2) and 201 subjects without hip fracture (mean age = 79.0). Baseline participation rates were 98% for those with hip fracture and 83% for those without. Baseline data were collected from proxy respondents for 41% of hip fracture subjects and for 14% of non–hip fracture subjects. Follow-up data were collected for 99% of hip fracture subjects and for 97% of subjects without hip fractures. The mean length of follow-up was 394 days for hip fracture subjects and 441 days for control subjects.

Table 1 shows some baseline characteristics of study subjects. The major differences between subjects with and without hip fractures were that hip fracture subjects tended to be older and were more likely to be female, live in nursing homes, have frequent falls, be cognitively impaired, and have low body weight.

The crude effect of hip fracture on mortality is shown in Table 2. For hip fracture subjects, the 1-month mortality

rate was 4.7% and the 1-year mortality rate was 21.7%. In contrast, in non–hip fracture subjects, there were no deaths at 1 month, and the mortality rate at 1 year was 4.7%.

The possibility that the effect of hip fracture on mortality was due to confounding by poor prefracture health was assessed using proportional hazard models (Table 3). In the 378 subjects with complete data on important prognostic factors, the crude hip fracture–mortality hazard ratio was 4.0 and the adjusted hazard ratio was 3.3. In the 287 subjects who were directly interviewed and in whom it was also possible to adjust for self-rated health, the crude and adjusted hazard ratios were 2.7 and 3.1, respectively.

TABLE 2—Proportion of Hip Fracture and Non–Hip Fracture Subjects Surviving after Various Time Periods

	Hip Fracture Subjects (n = 211)		Non–Hip Fracture Subjects (n = 201)	
Time	Surviving %	Cumulative Deaths, No.	Surviving %	Cumulative Deaths, No.
30 days	95.3	10	100.0	0
90 days	90.5	20	99.0	2
183 days	87.2	27	96.5	7
365 days	78.3	45	95.3	9
548 days	70.1	55	91.7	13

Note. Percentages of subjects surviving were calculated with life table methodology.

TABLE 3—Crude and Adjusted Hazard Ratios and 95% Confidence Intervals (CIs) for Mortality after Hip Fracture

Subjects in Model	Crude Hazard Ratio (95% CI)	Adjusted Hazard Ratio (95% CI)
All (n = 412)	4.48 (2.45, 8.20)	. . .
All with complete covariate data (n = 378)	4.00 (2.16, 7.42)	3.31 (1.68, 6.51)
Directly interviewed subjects with complete covariate data (n = 287)	2.71 (1.34, 5.48)	3.07 (1.40, 6.71)

Note. Hazard ratios and confidence intervals adjusted for age, sex, place of residence, mental state score, number of walks per week, number of hours worked per week, total number of medications, and self-reported diabetes mellitus, myocardial infarction, and Parkinson's disease. The hazard ratio in all subjects is also adjusted for use of a proxy respondent; the hazard ratio in directly interviewed subjects is also adjusted for self-rated health.

Discussion

This study has several limitations. Most importantly, control for prefracture health status is likely to be far from complete. Poor measurement of health status and failure to control for other known (and unknown) prognostic factors might have led us to overestimate the size of the hip fracture–mortality relationship. Self-rated health was probably the least well measured of our variables because subjects made their assessment after the fracture. The extent of the bias introduced by poorly measured self-rated health is unclear: self-rated health may not be as strongly associated with hip fracture as some of the other health-related variables (cognitive state, physical activity, and specific medical conditions) that we assessed.[25,26]

Another problem in our study is that the baseline participation rate for control subjects (83%) was lower than that for case subjects (98%). Thus, if nonparticipating controls were less healthy than participants, we would have overestimated the impact of hip fracture on mortality.

Despite these limitations, we believe our findings support the hypothesis that much of the excess mortality after hip fracture is due to the fracture itself. Controlling for prefracture differences in health status between hip fracture patients and control subjects had little effect on the observed three- to fourfold increase in mortality after hip fracture. Using the formula for attributable risk percent[27(p 38)] and a hazard ratio of 3.3, we estimate that as many as 70% of the deaths that occur in the year or so after hip fracture can be directly attributed to the fracture and its sequelae. This is comparable to the figures from a British study that involved examination of the medical records of 257 hip fracture patients who died within a year of their fracture.[15] In that study, cause of death was classified as being directly due to fracture (25% of deaths), possibly related to fracture (42%), or totally unrelated to fracture (33%).

Other investigators have argued that hip fractures directly cause excess mortality. Weiss et al., who restricted their study to women who had had a fracture in hopes of controlling for factors that lead to falls and fractures (including prefracture health status), found higher 1- and 2-year mortality rates in women with hip fractures than in women with forearm fractures.[4]

A recent US study compared survival in patients with vertebral, forearm, and hip fractures to that of all residents of the same area.[19] The investigators concluded that the observed excess 5-year mortality from hip fractures (mostly in the first 6 months postfracture) was due to the interaction of the injury with comorbid conditions. This pattern was quite different from that in patients with vertebral fractures in whom there was a gradual departure from the expected survival rates, making it unlikely that the fracture itself had any effect on mortality. There was no excess mortality in patients with forearm fractures.

In conclusion, we found evidence that hip fractures may be directly responsible for a threefold increase in mortality in the year or so after the fracture. The mechanism by which hip fractures cause older people to die is obscure and requires further research. □

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Trends and Differentials in Adolescent and Young Adult Mortality in the United States, 1950 through 1993

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ABSTRACT

Using data from the National Vital Statistics System and the National Longitudinal Mortality Study, this study examined mortality trends and differentials from 1950 through 1993 among US adolescents and young adults according to sex, race/ethnicity, education, family income, marital status, and cause of death. No appreciable reduction in youth mortality has occurred, especially among men. Declines in youth mortality from accidents have been nearly offset by increases in death rates from homicide, suicide, and firearm injuries. American Indians, Blacks, males, and those with least education and income were at increased risk of both overall and injury-specific youth mortality. (*Am J Public Health.* 1996; 86:560–564)

Introduction

Adolescents and young adults aged 15 to 24 years[1] are a sizable demographic group and represent about 15% of the total US population.[2] Premature death among them, especially due to preventable causes such as homicide, suicide, motor vehicle crashes, and other injuries, results in an enormous toll each year on the years of potential life lost.[3]

Although mortality for the general population in the United States has declined consistently since 1950, no such decline in mortality has occurred for those aged 15 to 24.[4–6] In fact, mortality for the latter has changed very little since 1982.[4,5] Furthermore, the US youth mortality remains substantially higher than that of many industrialized countries, largely because of excess mortality from homicide, suicide, and unintentional injuries.[7–9]

Studies examining trends and differentials in US adolescent and young adulthood mortality by sex, race/ethnicity, socioeconomic status, and cause of death are either scarce or nonexistent.[4,9,10] To fill these gaps, this paper examines long-term mortality trends from 1950 through 1993 and estimates the effects of sociodemographic covariates on overall and injury-specific youth mortality.

Materials and Methods

To analyze long-term mortality trends, sex-, race/ethnic-, and cause-of-

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This paper was accepted October 24, 1995.

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