Hip Fracture: A Nutritional Perspective (43408)

ROBERT P. HEANEY[1]
John A. Creighton University Professor, Creighton University, Omaha, Nebraska 68178

Abstract. **Hip fracture is the most important skeletal problem confronting the developed nations. In Finland, for example, it accounts for nearly 10% of all acute surgical beds and it annually costs every Western nation in the range of 8 to 20 million U.S. dollars per million population. These already high figures are certain to rise as the number of the old elderly increase. Nutrition plays a role in this problem not simply through the effect of calcium intake on bone mass, but in the falls that precede most fractures, in the amount of soft tissue hip padding to cushion the impact of a fall, and in the recovery both from the injury and from the even greater assault of its repair.**

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Hip fracture is the most important skeletal problem confronting the developed nations. It currently accounts for nearly 10% of all acute surgical beds in Finland (1) and annually costs every Western nation in the range of 8 to 20 million U.S. dollars per million population. These already high figures are certain to rise as the number of the old elderly increase.

In many parts of the world the hip fracture rate has risen dramatically in the years since World War II (2–4), even after adjusting for the shift in age distribution. Judging from Minnesota data, that same rise is occurring currently in the United States for men only. The rate for U.S. women rose during the 1930s and '40s and has been approximately stable since 1950, at something on the order of 130 cases/100,000 population/year (5).

Hip Fracture and Osteoporosis

Hips break at two main sites, through the intertrochanteric region or across the femoral neck. These two types of fracture are about equally prevalent in most developed nations. Each is usually thought of as simply one of the fractures of osteoporosis, although several authors have raised questions concerning the relevance of osteoporosis in this context (6, 7). Both sides of that issue have typically meant low bone mass when they said "osteoporosis," and it is bone mass about which the discussion centers in regard to hip fracture. The new Copenhagen Consensus Conference definition of osteoporosis (8) finesses that problem, in a sense, since it defines osteoporosis as a condition of fragility, and shifts low bone mass to the status of a cause of that fragility. Thus, whether or not reduced mass is the major reason for common hip fractures, there can be no argument about the fragility of the bone in patients who suffer hip fractures. In that sense, they are unquestionably osteoporotic.

The causes of osteoporotic fragility, according to the Copenhagen definition, include, in addition to decreased bone mass, microarchitectural deterioration of bone tissue. This means, among other things, loss of trabecular connectivity and an increased burden of unrepaired fatigue damage in both cortical and trabecular bone—but especially in cortical bone.

Low bone mass is essentially universal in the elderly—those most prone to hip fracture—and, thus, its detection does not usefully predict risk. However, that does not necessarily mean that its presence does not contribute to risk. The extent to which it does so can be assessed principally by examining whether there is an increasing gradient of risk for successive reductions in bone mass. That seems clearly true for the intertrochanteric form of hip fracture. Some, though not all, studies suggest that low mass may be less of a factor for fracture of the femoral neck (5, 7, 9–12).

By contrast, this fracture exhibits a number of features in the elderly strongly suggestive of the second fragility component from the Copenhagen definition, i.e., microarchitectural deterioration of bone tissue. In 102 consecutive cases in a recent report from Israel (13), 90 patients exhibited no evidence of cell-based remodeling at the site of the fracture, while iliac crest

[1] To whom requests for reprints should be addressed at the Office of John A. Creighton University Professor, Creighton University, California at 24th Street, Omaha, NE 68178.

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biopsies from the same patients taken at the time of surgery showed what would be considered normal remodeling activity. Fatigue damage is currently undetectable *in vivo* and requires special techniques for its recognition *in vitro*. These were not employed in this study, but, since remodeling is necessary for removal and replacement of fatigue-damaged bone, it seems likely that such damage would have accumulated at the site where the fracture ultimately occurred.

Other studies have emphasized the importance of local bone tissue deterioration in this fracture. Dodds *et al.* (14) found decreased activity of pentose shunt enzymes in osteoblasts from the cortical bone of the femoral neck in fracture patients, but not in the trabecular osteoblasts of the neck region in the same patients, nor in cortical osteoblasts of age-matched controls coming to hip surgery for osteoarthrosis. The strength of the femoral neck region is mainly determined by cortical bone (15), and thus the localization of the defect to cortical osteoblasts is especially suggestive.

Dunstan *et al.* (16) found that osteocyte viability in the femoral neck declines with age, and that many patients with fracture of the femoral neck show extensive osteocyte death. While the mean proportion of viable cells was the same in hip fracture patients and age-matched controls, the variance of the hip fracture population was nearly twice as large as the control, suggesting substantial heterogeneity among the hip fracture cases.

These reports raise questions about the relevance, for certain types of fracture, of studies either of cell-based remodeling or of bone mass when performed at sites different from those of the fracture involved. They also emphasize the essentially local nature, not only of the fracture itself, but of the fragility that is an essential part of the low-trauma fracture. Such fractures are determined not only by the location of the impact, but by the point at which the structure is weakest. While low bone mass tends to be a panskeletal phenomenon, and thus appendicular bone density or radiogrammetry predicts both spine and intertrochanteric fractures, fractures more related to local factors would not be well predicted from remote site measurement, and to some extent it appears that that may be the case for fractures of the femoral neck.

Osteomalacia is also a factor that can weaken bone at the site of hip fracture. In northern latitudes it has been found in as many as 11–12% of hip fracture patients (17), but is substantially less common in the United States. Apart from frank osteomalacia, serum 25(OH)D levels are almost invariably low in the old elderly. In some studies, serum 25(OH)D has been found to be even lower in hip fracture patients than in age-matched controls (e.g., 18.5 nmol/liter vs 32.9) (18, 19). Others (20) found equally low values in both groups—roughly only 25% of the level suggested by Francis *et al.* (21) and Krall *et al.* (22) as reflecting minimal vitamin D sufficiency. It remains uncertain whether this kind of vitamin D "insufficiency" exerts a harmful effect. Certainly, levels such as found in hip fracture patients are associated with large increases in endogenous parathyroid hormone secretion and poor calcium absorption (22, 23), both of which would plausibly weaken bone.

In addition to vitamin D insufficiency, nutrition is recognized to contribute to osseous fragility mainly through the effect of low calcium intakes on bone mass. Four population studies, two cross-sectional and two longitudinal, have found increased risk of hip fracture for persons ingesting low calcium intakes (24–27), though one found the effect only for men (27). Pointing in the same direction are two recent epidemiological studies of thiazide use, both showing protection against hip fracture (28, 29). One found the effect to be related to exposure in a dose-dependent manner (29), and noted, as well, that there was no protection associated with other antihypertensive treatment, suggesting that it was the thiazides, particularly, rather than the underlying condition or associated medications, that were responsible for the effect. Presumably, thiazides act in this context through their well-recognized reduction in urinary calcium excretion. Urinary losses constitute an important and inadequately appreciated aspect of calcium nutrition. A diet may be inadequate in calcium not simply because it is absolutely low, but also because it is insufficient to offset prevailing excretory losses.

To the extent that low bone mass is an important factor in fracture risk, nutrition plays a correspondingly important role in this multifactorial disorder. By contrast, it is not known whether nutritional factors are involved in the failure adequately to repair fatigue damage. To the extent that the phenomenon is purely local, nutritional causes become less plausible.

The Population at Risk

The hip fracture rate rises exponentially with advancing age, with the highest rates occurring in the oldest old (85 and above). Thus, those at highest risk would seem to be the elderly. While true, this generalization overlooks an important distinction. Simonen (30), for example, found admission rates to be 9-fold greater for patients who, prior to fracture, had been in institutions for the elderly than for free-living elderly of the same age. In that sense, hip fractures can be said to occur predominantly not in the elderly generally, but specifically in the most feeble members of that cohort, those who already have the greatest burden of comorbidity and who, for a variety of reasons, may be the least well nourished as well.

This latter point is reflected, for example, in the patients with hip fracture described by Delmi *et al.* (31), who, on admission following fracture, had significantly

reduced mean levels of serum albumin, carotene, and retinol-binding protein, among other evidences of nutritional deficiency. The poor nutritional status of many patients admitted with hip fracture is further evident in their general emaciation and the scarcity of muscle and fat over the hip. Nutrition plays an undramatic, but probably important, role in such fracture-related features as propensity to fall, postural reaction during a fall, and the sustaining of injury on impact. Pruzansky *et al.* (32) have clearly shown that low body weight is a significant risk factor for hip fracture in both blacks and whites. The importance of soft tissue padding over the hip is often overlooked, but is illustrated dramatically by the simple device of dropping an ordinary drinking glass first on a carpeted floor and then on a concrete or tile floor. The force of the fall is the same under both circumstances, but even thin carpeting is usually enough to prevent the glass from breaking. It is not that the carpet (or soft tissue) absorbs the energy of the impact. Rather, it distributes it over a broader area, so that no one point receives the full brunt of the impact. Better nutritional status in the fragile elderly might well reduce the harvest of fractures that otherwise results from the inevitable falls that the aged suffer. (Failing that, suitable carpeting in institutions for the elderly might accomplish for hips what it demonstrably does for glassware.)

Recovery from Hip Fracture

It is commonly said that excess mortality in the elderly in the first few months following hip fracture is on the order of 10–20%, and that perhaps as many as half of the survivors lose a substantial portion of their prefracture independence. The study of Delmi *et al.* (31) strongly suggests that these outcomes may not be inevitable. Fifty-nine patients (mean age, 82) admitted with hip fracture were randomly assigned to receive either a protein-based multinutrient supplement at bedtime in addition to their regular hospital diets or, alternatively, simply the regular hospital diet (which was otherwise designed to be nutritionally adequate). Outcomes, both acutely and at 6 months, were dramatically better in the supplemented group. Subsequent work from the same group of investigators (P. Bonjour, personal communication) showed that it was the protein in the supplement that was responsible for the improvement. The regular hospital diet contained sufficient protein, of course, but it was of little practical value because the patients rarely consumed it. Both groups experienced a further decline in serum albumin during hospitalization and surgical repair, but it was greater in the unsupplemented patients. As in most institutions, short staffed and cost conscious, no one made the effort to feed the patients. By contrast, care was taken to ensure that the investigational liquid supplement was consumed at bedtime. Hence, it seems that, in the face of surgical assault on these patients, their entering malnutrition got worse under medical "care."

The conclusion suggested by this study is that better salvage (and lower cost) would result from the preeminently low-tech solution of simply helping our patients to eat. Possibly a standard protocol for the fragile elderly admitted with hip fracture would include some regimen of concentrated nutritional replacement, designed both to repair deficiencies commonly present on admission and to aid tissue rebuilding after surgical repair.

Conclusion

Hip fractures are concentrated in the relatively more frail, often-institutionalized elderly, and they have many contributory causes. A number of nutritional inadequacies are relatively common in the elderly and appear to play a role both in predisposing to development of hip fracture and in producing poor outcomes after repair of such fractures. These include protein-calorie undernutrition, low bone mass due partly to low calcium intakes and/or excessive excretory loss, severely depressed circulating vitamin D levels, and, in a few cases, frank osteomalacia superimposed on preexisting osteopenia. While it may not be possible to maintain optimal nutrition in the entire cohort at risk, it does appear that aggressive nutritional supplementation, particularly with protein, offers promise of substantial improvement in outcome after fracture.

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