The Roles of a Fracture Liaison Service

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ABSTRACT

The roles of a fracture liaison service (FLS) are extensive and include, but are not limited to: 1) providing a standardized framework for the evaluation and management of low-energy fractures, also known as fragility fractures; 2) improving patient outcomes through the recognition of fragility fractures as signal events requiring further diagnostic explanation; and 3) lowering direct and indirect healthcare expenditures. One of the central tenets of the FLS is its recognition of fragility fractures as warning signs of underlying pathology, often osteoporosis or other metabolic bone diseases. This understanding, combined with the application of a multidisciplinary management team specialized in diagnosing and treating such pathologies, allows for better short- and long-term management of patients and concordant improvement in outcomes. This article should be viewed as a thematic introduction to the FLS, with others in this volume each illustrating specific examples of how FLS paradigms facilitate the roles described herein.

KEYWORDS: Fracture Liaison Service (FLS); osteoporosis; fractures; falls

INTRODUCTION

Fracture Liaison Services (FLSs) have emerged from two clinical observations. First, low-energy fractures, particularly in individuals over age 60, often result in significant functional morbidity, pain, loss of independence, and death. Second, low-energy fractures can be prevented by medical, physical, and environmental interventions, all dependent upon a sensitive understanding of the physiology of the fractures and their consequences. Low-energy fractures, also known as fragility fractures, are signal events, often the presenting symptoms of an underlying pathology requiring further diagnostic exploration. Therefore, in a very real sense, patients with low-energy fractures are not "asymptomatic." On the contrary, they are frequently found to have metabolic bone diseases such as osteomalacia, hyperparathyroidism, and especially osteoporosis that result in decreased bone density and disruption of bone micro-architecture leading to diminished ability to withstand applied stresses. Presentations in this volume will concentrate on metabolic contributions to fractures and on common fracture patterns that should be recognized for their association with functional impairment and secondary fractures. Thematically, the volume makes several points:

- The signal value of fragility fractures should not be ignored or minimized.
- Certain fractures, especially vertebral compression fractures, often signal an increased incidence of subsequent fractures.
- Screening bone density is only an initial step in the treatment of fractures. It does not yield a diagnosis, and, in itself, is not a basis for therapy. All patients with fragility fractures deserve a metabolic bone evaluation.
- The evaluation of bone density in the context of fractures frequently reveals medical conditions that contribute to decreased bone density and falls.
- Appropriate therapy depends upon accurate diagnosis of causal and contributory factors to the fracture history and bone density.
- Several medications are available for the treatment of decreased bone density depending upon the extent of bone loss, fracture history, and co-existing medical conditions.

CLINICAL FINDINGS IN THE RHODE ISLAND FRACTURE LIAISON SERVICE

By carrying out a metabolic bone investigatory program as described here, we have made some surprising findings that have enabled more targeted approaches to therapy. Reports of disease prevalence are particular to each FLS referral base. In our case, fractures are the triggering symptom. Renal and transplant patients are treated on those services, respectively, and are discussed in a later article in this volume. Nonetheless, some observations can reinforce the consensus on coordinated evaluation of fracture patients. Within the last 2 years, of 265 consecutive patients with osteoporosis and fractures on our service, we detected 28 patients with primary hyperparathyroidism and 27 patients with monoclonal gammopathy of uncertain significance (MGUS), both discussed in subsequent articles, as well as 10 patients with hematologic malignancies including multiple myeloma, Waldenström's macroglobulinemia, chronic lymphocytic

leukemia, mast cell leukemia, and lymphomas. In addition, the FLS has evaluated and treated osteoporosis associated with several genetic diseases including osteogenesis imperfecta, Marfan's syndrome, Ehlers-Danlos syndrome, and familial exudative vitreoretinopathy (FEVR), as well as neuromuscular conditions including muscular dystrophy and cerebral palsy. As discussed in an ensuing article, malabsorption-related osteomalacia has posed challenges to raising vitamin D levels depending upon the severity and cause of the malabsorption. Combined, 75/302 (25%) of patients referred for osteoporosis or osteoporotic fractures were diagnosed with another condition, usually serious, and therapy was targeted at those diseases. This reflects a major thematic approach of our FLS. Without a diagnostic approach such as that described below, these conditions could be missed and, therefore, mistreated.

CLINICAL APPROACH

A fragility fracture is often defined as one resulting from a fall from a standing height. However, there are other considerations such as the surface upon which one falls, the circumstances of the fall, etc. In general, the fractures of concern are low-energy fractures and ones with unusual injury mechanics. The clinical approach to the patient with a fragility fracture consists of a history focused on skeletal biology, a focused physical examination, occasional X-rays, determination of bone density, laboratory evaluation of skeletal biology, and a treatment plan that takes into account the patient's medical and fracture history, severity of bone loss, chemistry, and clinical diagnoses.

1. History – A general medical history with an emphasis on the skeleton inquires about the patient's fracture history, history of falls, acuity of vision and hearing, balance, loss of height, the home environment, diet (especially dairy intake or lactose intolerance), medications that may have an adverse effect on the skeleton, medical exposures such as corticosteroids, and general bone hygiene including caffeine intake, alcohol consumption, and smoking habits (**Table 1**). The physical and environmental contributions to fractures should be part of every screening conversation. Causes of secondary osteoporosis are described in the *Osteoporosis and Fragility Fractures* article in this volume.

2. Physical Examination – A focused skeletal examination includes gait and balance stability, core muscle strength, and spinal deformity (especially kyphosis) that might indicate previous fractures. Rapid, simple tests of lower extremity strength are available, such as "timed up and go" and rising from a chair, that suggest a need for physical therapy for muscle strengthening and gait stability to prevent falls.[1,2] In the kyphotic patient, lateral thoracic spine X-rays may be indicated to detect previous vertebral compression fractures since these fractures can be almost asymptomatic yet result in deformity and indicate risk for subsequent fractures.

Table 1. Static and Potentially Alterable Risk Factors for Osteoporotic Fractures

Static Factors	Potentially Alterable Factors
Demographic factors: old age, female sex, Asian or White race	Low bone density
Personal and/or family history of adult fracture	Lifestyle factors: low BMI, sedentary lifestyle, smoking, excessive alcohol consumption, low dietary calcium intake
Menopause onset prior to age 45	Iatrogenic contributions: glucocorticoids, sedatives, anti-hypertensives, polypharmacy
Physical frailty	Environmental factors: obstructed walkways, poor lighting, unequal surfaces, etc.
Deterioration in mental status	Estrogen deficiency

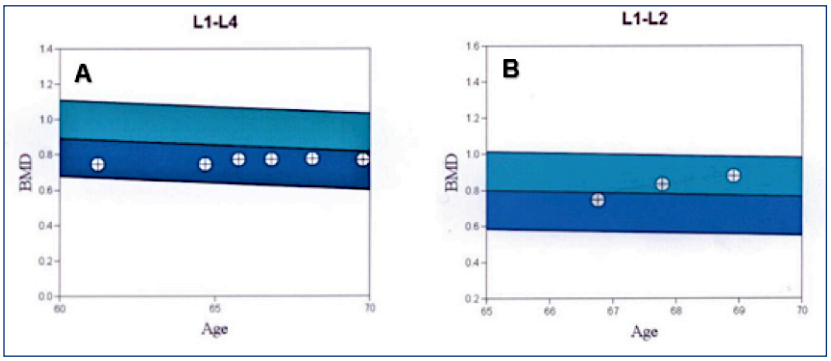
Sensitivity to loss of thoracic height can be important since vertebral compression fractures can result in the lower ribs abutting the pelvis, resulting in impaired sitting and loss of thoracic volume. Several studies have found that pulmonary function can be compromised in patients with osteoporotic vertebral compression fractures compared to patients with chronic low back pain without osteoporosis.[3] Reductions in both vital capacity and forced expiratory volume (FEV1) have been correlated with spinal osteoporotic fractures and resulting spinal deformity and decreased thoracic volume, indicating that reductions in pulmonary function can be correlated with osteoporotic spinal deformities that can be detected and quantified by clinical and radiographic assessment.

3. Determination of Bone Density – Dual-energy absorptiometry (DEXA) scanning has several major advantages in the evaluation of the skeleton. It is rapid, noninvasive, and non-enclosed. The radiation exposure is trivial (**Figure 1**). It provides 2-dimensional measurements of bone density which are reported in g/cm^2 and compares them to age-related norms (Z-score) and values at age 20 (T-score). The

Figure 1. Patient undergoing DEXA scan and technician
Bone mineral density or bone strength is measured with a type of low-energy X-ray called a DEXA scan. The scan is entirely noninvasive, non-enclosed, and takes approximately 15 minutes to complete. The measurement of an individual's bone mineral density is compared to values of the normal population to determine the extent of bone loss. Patients under active treatment should have follow-up DEXA scans at 1-2 year intervals.

Figure 2. DEXA scans of the lumbar spine

A: A patient with breast cancer, treated with tamoxifen, showing a steady increase in bone density. **B**: Documentation of treatment efficacy in another patient.



well-established, age-related normative values are based upon very large numbers of individuals. Graphic display of data is often helpful, particularly in serial studies following progression and treatment (**Figure 2**). It does have some limitations in that normative values are available for the spine, hips, and wrist/forearm only. While the output of DEXA appears simple, one has to interpret the data with sensitivity to the effects of deformity, arthritis, and previous fractures that can give spurious results, especially in the spine. Some vertebrae may need to be excluded from the analysis. Particular attention must be paid to the femoral neck density since this is a frequent site of hip fractures. Extrapolation of density from one anatomic site to another should be done in the total context of the patient's history. Additionally, not all DEXA machines are equally calibrated, and care has to be taken when comparing results between machines. Ideally, the patient would be serially scanned on the same well-calibrated machine. While DEXA is a safe and widely available clinical tool for monitoring bone density and can accurately determine areal (two-dimensional) bone mineral density, it cannot be extrapolated to a three-dimensional volumetric density. Additionally, it does not have the ability to accurately assess structural micro-architecture nor to differentiate between cortical and trabecular bone compartments. Collectively, these constraints limit the ability of DEXA to estimate bone strength and do not allow it to provide microstructural information which can be used to assess bone quality.[4]

It must be appreciated also that the DEXA scan measures only bone density and does not give a clinical diagnosis of osteoporosis. Other conditions can produce low bone density and are treated quite differently, so that a biochemical analysis of the patient with low bone density is important to make an accurate diagnosis and target therapy, with or without fractures.

4. Laboratory Analysis – In 1994, Henry Mankin, then chair of orthopedics at Harvard and Massachusetts General Hospital, wrote a seminal paper on metabolic bone diseases in the orthopedic setting and described the relevant comprehensive analysis of the skeleton.[5] While clinicians often have their individual preferences as to what constitutes core and supplementary studies, one well-targeted initial approach was described by Mankin and reviewed several times (**Table 2**). The laboratory approach to the skeleton consists of 1) exploring conditions other than osteoporosis that can be associated with decreased bone density such as hyperparathyroidism or hematologic malignancies, most commonly multiple myeloma; 2) identifying modifiable conditions, commonly low serum 25-hydroxy vitamin D, chronic renal failure, and secondary hyperparathyroidism; and 3) informing the appropriate choice of pharmacotherapy once primary osteoporosis is identified as the cause of low bone density. In this last category, bisphosphonates require adequate renal function, denosumab requires adequate serum calcium and vitamin D, and osteoanabolics require a normal, or at least explainable, bone-specific alkaline phosphatase.

Table 2. Routine Post-DEXA Diagnostic Procedures in Osteoporosis Evaluation

Goals	Procedures
Exclude non-osteoporotic causes of low bone mass and skeletal fragility	Laboratory tests: Serum calcium, creatinine, alkaline phosphatase, protein electrophoresis, 24-hour urine chemistry, serum 25-hydroxy vitamin D, serum parathyroid hormone, bone turnover markers
Identify modifiable conditions such as low serum 25-hydroxy vitamin D, chronic renal failure, secondary hyperparathyroidism	
Assess the presence of vertebral fractures when clinically indicated	X-ray of thoracic and/or lumbar spine for patients with loss of height and/or deformity

Bone turnover markers (BTMs) are a series of protein biomarkers released by osteoblasts and osteoclasts, or from bone matrix during bone remodeling. They can reflect the degree of bone formation or resorption and can provide prognostic information on the risk of fracture, progression of disease, and response to therapy. Many BTMs are under study with several currently in clinical use. Two clinically used BTMs are the C- and N-terminal telopeptides of type I collagen, the most common protein in bone matrix. The two terminal telopeptides, notated as CTX and NTX, respectively, are released by osteoclastic resorption of bone and are useful in identifying the degree of bone resorption. CTX is generally assayed in serum; NTX is assayed in urine. They have broadly similar clinical utility.[6] Two BTMs reflect bone formation in remodeling: bone-specific alkaline phosphatase and osteocalcin. There are several genetic isoforms of alkaline phosphatase produced by the liver, intestine, and

placenta, but it is the bone-specific isoform that reflects bone formation. Osteocalcin is the most abundant non-collagenous protein in bone matrix and is secreted by osteoblasts. While of investigatory interest, pre-analytic variables have kept it from routine clinical use. BTMs are clinically useful primarily to assess the resorptive component of bone turnover, predict the responses to anti-resorptive therapy, follow the response to, and compliance with, therapy, and assess increases in bone density and reduction of fracture risk.[6]

ROLE OF AN FLS IN OSTEOPOROTIC FRACTURE CARE

Care of patients with fragility fractures is often compromised by a lack of coordination and shared expertise. Active interventions in an FLS have been shown to result in a decreased incidence of second fractures.[7] Anti-osteoporotic therapy after a fragility fracture results in a 40% decrease in the 3-year risk of a subsequent fracture.[8] The advantages of a coordinated post-fracture program were demonstrated by an FLS that achieved accurate diagnosis and secondary fracture prevention in 414/430 (96%) of patients with an osteoporotic fracture.[9] Many other studies have demonstrated that coordinated care of osteoporotic fractures in organized FLS programs improves evaluation, treatment, and outcomes, and reduces fracture-related morbidity and mortality.[10-13] Coordinated care in an FLS program has also been shown to result in substantial reductions in secondary fractures.[14] As a consequence of these demonstrations, a consensus has developed around the clinical benefits of an organized FLS program. Evidence-based clinical guidelines for the evaluation and treatment of low-energy, geriatric, and fragility fractures have been developed by the American Society of Bone and Mineral Research, 39 stakeholder societies, and the NIH.[15] While these are subject to updating, the overarching recommendation is that the FLS care delivery model is the most effective means for treating patients with low-energy fractures (i.e., fragility fractures), and that FLS programs consistently initiate and comply with best practice diagnosis and treatment guidelines more often than do traditional models.[16]

EPIDEMIOLOGY AND ECONOMICS OF LOW-ENERGY FRACTURES

The FLS takes the position that a fracture is a signal event indicating a propensity for functional decline and reflecting underlying reduced bone density, increased fall risk, and the presence of environmental hazards. Importantly, a previous low-energy fracture is among the strongest risk factors for subsequent fractures.[5,17-20] More specifically, patients with a low-energy fracture of the wrist, hip, proximal humerus, or ankle have a 2- to 4-fold greater risk for subsequent fractures than do individuals who have never experienced a fracture.[17] Up to 30%-40% of patients with a vertebral compression fracture will experience additional vertebral fractures within 3 years.[21] Compared to individuals with no history of fracture, a patient with a vertebral fracture has nearly a 5-fold increased risk of a subsequent vertebral fracture and a 6-fold increased risk of non-vertebral fractures.[22] Notably, fractures of the spine and wrist are associated with an increased incidence of secondary hip fractures.[23] Hip fractures account for over 350,000 hospitalizations/year, or 30% of all fracture-related hospitalizations.[24] Outcomes after hip fractures in the elderly are often complicated by morbidity, disability, and death. Overall, 24% of these patients die within a year after fracture.[12,25] Half of hip fracture patients do not regain their pre-fracture function, and many lose their functional independence and require long-term care. Taken together, these data indicate that patients who have experienced a low-energy fracture of any type have a markedly greater risk of subsequent fractures with associated morbidity than do individuals who have not fractured.

The economics of low-energy fracture care are similarly compelling. It has been estimated that the annual medical cost of treating hip fracture patients in the US exceeds $10 billion.[25] Hip fractures are related to age and the geriatric population, and therefore as the population ages, the incidence of hip fractures can be expected to increase concurrently. As a consequence of demographic changes, medical costs related to hip fractures have been predicted to rise to $25.3 billion by 2025, and costs associated with all osteoporotic fractures are predicted to rise to over $95 billion by 2040.[26,27] Multiple cost-effectiveness analyses have demonstrated the potential for substantial savings resulting from increased use of FLSs within the US healthcare system, with one 2021 study concluding that per every 1 million patients with Medicare coverage receiving secondary fracture prevention after an osteoporotic fracture, $418 million dollars could be saved and 30,000 quality-adjusted life-years could be gained.[11,28,29] These data are especially compelling when observed in concert with the estimation that the annual incidence of osteoporotic fractures in the US is expected to rise to 3.2 million by 2040, indicating a potential cost savings of $1 billion or more per year by that time through the widespread implementation of FLSs.[27] Other studies have demonstrated that the potential for savings is not specific to the US.[30-33] Lastly, it is important to note that the economic benefits of the FLS model extend not only to direct health care expenses, but to patients and their families as well due to lifestyle changes, institutionalization, and family care often associated with the longitudinal effects of recurrent fractures, with one study estimating the indirect cost of fractures in 2018 to be $8.2 billion.[27] The extent to which social and economic factors burden patients and families can be minimized by the timely diagnosis and management of the underlying disease processes by an FLS.

CONCLUSIONS

An FLS provides a systematic approach to osteoporotic fracture care that 1) recognizes low-energy fractures as signal events requiring further inquiry; 2) provides a framework for the comprehensive evaluation of underlying and often unexpected pathology; 3) facilitates appropriate treatment; 4) reduces the risks of secondary fractures with their attendant morbidity and mortality; 5) lowers costs for patients and healthcare systems; and 6) improves patient outcomes. This article provides an entryway into the subsequent presentations in this volume that each highlight distinct yet interconnected roles an FLS plays in providing multidisciplinary, comprehensive, high-quality, and cost-effective patient care.

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