

SPONTANEOUS INSUFFICIENCY FRACTURES

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Abstract: Spontaneous insufficiency fractures are caused by normal or physiological stress on weakened bone. The leading cause of insufficiency fractures is osteoporosis which has a propensity to affect older patients. Other causes or associated factors are disorders which affect bone metabolism, collagen formation, bone remodelling and medications such as bisphosphonates and glucocorticoids. Pathological fractures and abuse are important causes of unexplained fractures which warrant careful consideration. Spontaneous fractures of the long bones affect on average 1% of nursing home residents per year and tend to occur in patients who are bed-bound with joint contractures. Preventative measures for spontaneous insufficiency fractures include optimising nutrition to include an adequate intake of protein, calcium and vitamin D, maintaining mobility and preventing long periods of bed-rest and treatment of underlying pre-disposing conditions.

Key words: Spontaneous insufficiency fractures, osteoporosis, older people.

Background

Spontaneous insufficiency fractures are a type of stress fracture which are caused by normal or physiological stress upon weakened bone. They should be differentiated from fatigue fractures which are due to abnormal stresses on normal bones, and pathological fractures which occur as a result of focal pathology such as malignancy (1). Older patients are at an increased risk of spontaneous insufficiency fractures due to associated conditions of osteopenia and osteoporosis, sarcopenia and neuromotor degradation (2).

Fractures can occur spontaneously in this population during normal activity such as ambulating or transferring. Some patients may experience prodromal pain prior to the fracture particularly in atypical femoral fractures (3).

The most common locations for insufficiency fractures are vertebral, sacral and hip, followed by long bone fractures such as the femoral shaft, tibia, fibula and the humerus. Osteoporotic fractures tend to occur primarily in the vertebrae and sacrum while atypical fractures due to bisphosphonates more commonly occur in the femur.

Differential Diagnosis

Spontaneous insufficiency fractures should be regarded as a symptom of an underlying predisposing or precipitating condition. The most common cause of insufficiency fractures is osteoporosis although there are numerous other contributors including disorders affecting bone mineral homeostasis, bone remodelling and collagen formation, medications and local destruction of bone (4).

Pathological fractures should be excluded as a cause of presumed spontaneous fractures especially in a patient with risk factors for or known malignancy. Metastatic disease and multiple myeloma can present with minimal trauma fractures.

Elder abuse should also be considered as a differential of

all patients presenting with an unexplained fracture and should prompt a thorough examination for evidence of other injuries or signs of other forms of neglect or abuse (5). The differential diagnosis for spontaneous fractures is shown in Table 1 below.

Table 1
Differential Diagnosis for Spontaneous Insufficiency Fractures

Mechanism of fracture	Examples
Disruption of bone mineral homeostasis	Osteoporosis
Osteomalacia	
Hyperparathyroidism	
Rheumatoid Arthritis	
Chronic kidney disease	
Diabetes mellitus	
Bone remodelling	Paget's disease
Osteopetrosis	
Collagen Formation	Marfan's Syndrome
Fibrous Dysplasia	
Medication-induced	Bisphosphonates
Glucocorticoids	
Chemotherapy	
Local disruption of bone	Radiation therapy
Peri-prosthetic widening	
Other causes of 'pseudo-spontaneous' fracture	Pathological-bone metastasis, multiple myeloma
	Abuse

Prevalence of spontaneous insufficiency fractures of long bones

Reports on spontaneous long bone fractures in older immobile patients are limited. One case series described six

nursing home residents with a 'spontaneous long bone fracture'. (6) A further survey of 11 nursing homes reported 16 patients with 'minimal trauma fractures' and described the interaction between functional status and low trauma fractures (7).

A prospective study of 30 nursing homes in France reported 'spontaneous long bone insufficiency fractures' occurred in 1% (n=55) over a 30-month period. Femoral fractures were associated with worse outcomes with a two-month mortality of 54% (8). A Japanese observational study of 500 long term care residents followed up over 6 years reported a rate of 3.6% (n=18) for spontaneous insufficiency long bone fractures. The most common site for fractures was the femur followed by the humerus. The authors observed that all fractures occurred near joint contractures during daily care procedures, without any external trauma or abuse. Over half of cases had a history of fractures in long bones that were either traumatic or non-traumatic and a third of fractures recurred in the same bone indicating that older people with previous long bone fractures are prone to recurrent events (9). Stroke patients have a more rapid decrease in bone mineral density on the paretic than on the non-paretic side. This corresponds to an increased fracture risk on the affected side (10).

Association with Bisphosphonate and Glucocorticoid Therapy

Bisphosphonates are an effective and generally well tolerated treatment for osteoporosis. Prolonged bisphosphonate therapy is associated with an increased risk of atypical femoral fractures. The estimated incidence is 1 per 1000 per year or 0.1%. These fractures seldom heal spontaneously and most patients will require surgical intervention (11).

Common features are prodromal pain, minimal or absence of trauma and a simple, transverse pattern and hypertrophy of the diaphyseal cortex. This may result from propagation of a stress fracture whose repair is retarded by diminished osteoclast activity and impaired microdamage repair resulting from prolonged bisphosphonate use (12). Regular assessment of patient's bone health, consideration of drug holiday from bisphosphonates after 5 years therapy, or preferably alternative treatment options (e.g. Denosumab, Teriparatide) for those who require ongoing therapy for osteoporosis is recommended.

Long term glucocorticoid use is also associated with the development of insufficiency fractures. It is estimated that over half of chronic glucocorticoid users will develop reduced bone mineral density and fractures. Regardless of the mechanism of the fracture, the risk of hip, forearm and shoulder fractures increased two-fold while the risk of vertebral fractures is even higher.

A meta-analysis showed the relative risk (RR) of any fracture ranged from 1.66-1.98 for any fracture and the RR of 2.48-4.42 for hip fracture depending on age. This risk is largely independent of bone mineral density and has a dose-dependent relationship (13). Management/prevention includes minimising

steroid dosage and the duration of therapy, utilisation of steroid-sparing alternatives (e.g. Methotrexate in rheumatoid arthritis and biologic agents in inflammatory bowel disease). A regular bone health assessment should be carried out in patients requiring long term steroid therapy. Recent osteoporosis guidelines recommend women taking >7.5mg/prednisolone per day or equivalent should be considered for bone protective therapy. Additionally, consideration of anti-resorptive therapy should be considered for male patients and pre-menopausal women if taking high dose glucocorticoids (14). Denosumab is also licensed for treatment of bone loss associated with systemic glucocorticoid therapy.

Immobility and Spontaneous Fractures

Prolonged bed rest leads to disuse osteoporosis due to an increase in bone resorption and decrease in bone formation. One study of 25 healthy volunteers showed bone mineral density reduced by 5% at 60 days with a corresponding increase in bone resorption markers and osteoclast number (15). Muscle mass is lost quickly following immobility with disuse atrophy occurring at a rate of 0.5-1% reduction in muscle mass per day and muscle strength decreases by 50% over three to five weeks (16).

Mechanical force through gravity or muscle contractions affects skeletal health of both the internal and external architecture as well as bone quality. Therefore, periods of immobility and bed rest negatively impact on the quality and quantity of bone. Muscle atrophy and weakness begin to occur early following immobilisation (1).

The Effect of Nutritional Factors

Spontaneous insufficiency fractures are most common in older, immobile patients. Nutrition has a major role in bone health. Adherence to calcium and vitamin D supplementation is poor in the osteoporotic population. Up to 50% of patients are non-compliant with prescribed treatment at 6 months (17).

Similarly, a compliance rate of 50% is observed with bisphosphonate therapy due to side effects, inconvenience of administration, cost and issues such as forgetfulness in patients with cognitive impairment (18). Untreated osteoporosis will increase the risk of spontaneous insufficiency fractures.

The majority of the literature describes spontaneous insufficiency fractures of long bones occurring in nursing home residents. As well as a higher prevalence of osteoporosis, nursing home residents have a higher level of functional dependence and frailty. 'Sarcopenia with limited mobility' is measured by reduced muscle mass and impaired gait speed and is increasingly recognised in the older population (19). Sarcopenia is common in nursing home residents due to disuse atrophy, co-morbidities associated with ageing and reduced muscle loss as well as dietary factors such as inadequate protein intake (20). Neuromotor degradation due to cognitive

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and motor disorders, previous stroke and polypharmacy also play a role. Institutionalised residents may have inadequate sunlight exposure if the outdoors environment is inaccessible due to impaired mobility or lack of opportunity. Malnutrition is common due to dysphagia and weight loss which often coincide with advanced cognitive impairment (21). Adequate dietary vitamin D, calcium and protein intake are essential for bone health and maintaining muscle mass.

Conclusions

Spontaneous insufficiency fractures will become increasingly prevalent given the ageing population and predicted increase in osteoporosis.

The clinical features are fractures that occur in the setting of minimal or absent trauma which may be associated with prodromal pain and typically affect the vertebrae, hip of long bones. Management involves comprehensively searching for precipitating and associated factors, reviewing the indications for bisphosphonates and glucocorticoids, and maintaining a vigilant watch for evidence of abuse or pathological fractures due to a malignancy process.

Preventative measures include prompt diagnosis and treatment of osteoporosis; maintaining mobility and avoiding periods of non-weight bearing if possible; optimisation of nutrition including adequate intake of protein, calcium and vitamin D; treating concurrent conditions such as metabolic bone diseases, collagen disorders and bone remodelling.

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