A review of anatomical and mechanical factors affecting vertebral body integrity

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Abstract

Background: The aetiology of osteoporotic vertebral fracture is multifactorial and may be conceptualised using a systems framework. Previous studies have established several correlates of vertebral fracture including reduced vertebral cross-sectional area, weakness in back extensor muscles, reduced bone mineral density, increasing age, worsening kyphosis and recent vertebral fracture. Alterations in these physical characteristics may influence biomechanical loads and neuromuscular control of the trunk and contribute to changes in subregional bone mineral density of the vertebral bodies.

Methods: This review discusses factors that have received less attention in the literature, which may contribute to the development of vertebral fracture. A literature review was conducted using electronic databases including Medline, Cinahl and ISI Web of Science to examine the potential contribution of trabecular architecture, subregional bone mineral density, vertebral geometry, muscle force, muscle strength, neuromuscular control and intervertebral disc integrity to the aetiology of osteoporotic vertebral fracture.

Interpretation: A better understanding of factors such as biomechanical loading and neuromuscular control of the trunk may help to explain the high incidence of subsequent vertebral fracture after sustaining an initial vertebral fracture. Consideration of these issues may be important in the development of prevention and management strategies.

Key words osteoporosis, vertebral fracture, bone density, spinal biomechanics, neuromuscular control

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1. Introduction

Osteoporosis is a metabolic bone disorder characterised by low bone mass and micro-architectural deterioration of bone tissue, leading to enhanced bone fragility and a consequent increase in fracture risk [93]. Osteoporosis is increasingly recognised as an important public health problem due to the significant physical, economic and psychosocial ramifications of fracture. Vertebral fractures are a hallmark of osteoporosis and are associated with back pain, functional disability, reduced health-related quality of life and increased mortality. These associations become more significant with increasing numbers of vertebral fractures [24, 69].

Previous reviews have outlined the influence of bone mineral density (BMD), bone loss and important engineering principles such as bone geometry, mechanical properties and load transmission on the capacity of a vertebral body to resist physiologic loads that may contribute to vertebral fracture or deformity [32, 31, 65]. With advances in densitometric technologies and \textit{ex vivo} analyses of trabecular bone properties, it is now evident that heterogeneity exists within the trabecular bone of a vertebral body. This observation may assist in the understanding of the complex aetiology underlying osteoporotic vertebral fracture. However, we postulate that trabecular bone properties cannot explain entirely why a difference in the prevalence rate of vertebral fractures exists among individuals with comparable BMD. It is likely that factors other than BMD operate to influence fracture risk, for example physiologic loading of vertebral bodies and the neuromuscular characteristics of trunk musculature.

Previously, the factors discussed have been viewed as mutually exclusive, but consideration of their potential interactions may assist understanding of the complex aetiology of these fractures. Therefore, the aim of this review is to highlight factors identified in the literature that may act to influence the risk of sustaining an osteoporotic vertebral fracture.

The risk of osteoporotic vertebral fracture can be conceptualised as a function of loading conditions and the ability of the human spine to withstand the load. Bone strength depends on its micro-architectural and macro-architectural features [80]. These features can be affected by the pathophysiology of osteoporosis and the local and global environment in which the bone operates. The local environment includes muscle and connective tissue attachments and the adjacent intervertebral disc, while the global environment can be considered to be the entire body operating in space (see figure 1).

\textbf{Figure 1.} Diagram of the systems approach that may be applied to explain the multifactorial aetiology underlying osteoporotic vertebral fracture. Bone, macroscopic, local and global factors may interact to influence the risk of sustaining a vertebral fracture.
2. Bone Structural Features

2.1. Trabecular micro-architecture

The design of the trabecular network allows for the efficient distribution of compressive and shear forces. This efficiency exists via the orientation and spacing of the three-dimensional arrangement of osseous plates and struts that are continuous with the inner surface of the cortex. Vertically-orientated trabeculae attenuate axial forces, while horizontal plates respond to the tensile (shear) stresses transferred from the intervertebral disc [88]. An excess of bone resorption over formation leads to trabecular deterioration and loss. Deterioration in the trabecular architecture transforms the densely connected, plate-like trabeculae into discontinuous rod-like structures [1, 82]. Such transformations, if severe enough, are a hallmark of osteoporosis.

Trabecular bone strength is in part attributable to its large surface to volume ratio. This ratio is approximately four times greater than that observed in cortical bone [72]. With age and menopause, the trabecular bone mass in the vertebral body (centrum) declines at a faster rate than cortical bone mass. Cortical bone therefore dominates in the elderly spine. Generally, women lose 20% of peak cortical bone mass and 40-50% of trabecular mass by 90 years of age [58]. This results in a decline in the load-bearing capacity of the vertebral body. For example, load bearing capacity in a lumbar vertebra can decrease from 1000kg in a young individual to 60-90kg with age [62]. In osteoporosis, the normal age-related changes in cortical thickness and trabecular density become more pronounced resulting in a significant decline of compressive strength in the vertebral body, increasing the likelihood of fracture. A loss of trabecular bone in particular, will contribute significantly to a decline in mechanical strength of the vertebral body [94]. Ex vivo investigations demonstrate that mechanical characteristics of vertebral bone to strain is dominated by trabecular bone properties [43, 81].

2.2. Subregional Bone Mineral Density (srBMD)

The characteristic reduction in bone mass per unit of volume, i.e. volumetric BMD in osteoporosis interferes with the mechanical support properties of the bone. Various measures of BMD correlate strongly with compressive failure loads of vertebrae and can therefore be used as convenient and generally reliable indicators of compressive strength of vertebrae in vivo when measured with tools such as dual energy x-ray absorptiometry (DXA) and quantitative computed tomography (QCT) [31, 65]. Approximately 75-90% of the variance in bone strength is accounted for by apparent BMD [53, 87]. Low BMD is one of the most important risk factors for osteoporotic fracture [13]. However, examination of BMD alone is insufficient to explain the basis for vertebral fracture. Studies have shown differences in prevalence rates for fracture among individuals with comparable BMD [1, 12, 15, 18, 28, 46, 61, 73, 79, 89, 90]. These observations suggest that a single measure of BMD may not be representative of vertebral capacity and/or that other factors may operate independent of BMD measures. For example, risk variation between individuals may lie in the more subtle subregional density differences within the vertebral body. Subregional differences are hidden when whole vertebral body BMD is measured [46].

During aging, there is a non-uniform loss of bone within the vertebral body, resulting in trabecular bone density becoming non-homogenous throughout the centrum [2, 3, 56]. BMD therefore becomes disproportionate between different regions of the vertebral body. In an examination of cancellous bone morphometry of lumbar vertebrae, Simpson and colleagues [82] discovered that BMD, as measured by the ratio of bone volume to total volume in the vertebra, showed significant subregional differences. Posterior margins of the vertebrae showed greater BMD than anterior regions, while central zones had the lowest BMD, lowest trabecular number and greatest trabecular separation. Generally vertebrae are less dense anteriorly and superiorly and more dense posteriorly and inferiorly with heterogeneity between symmetrical sites within the vertebral body [2, 3, 25, 56, 67, 78]. The lower density and therefore lower compressive strength of the anterior zone of the vertebral body may account for the greater incidence of anterior crush fracture in individuals with osteoporosis. Therefore, a better understanding of the mechanics of vertebral fracture may be ascertained by investigating differences in subregional BMD between individuals with and without vertebral fracture. Overall vertebral BMD may remain the same in the presence of differential responses in trabecular architecture to mechanical loading [82]. The mechanisms underlying variation in subregional BMD remain unknown. However, it is likely that axial force transmission through the vertebral body may play a role. Adjacent musculature and the intervertebral disc modulate axial force.

3. Macroscopic factors

3.1. Vertebral Geometry

The shape of a vertebra is well designed to accept axial loads. The orientation and framework of the vertebral body assures that in normal circumstances, compressive load is transferred through the
4.1. Fracture Risk

Fracture risk is commonly based on measurements of vertebral BMD. However, other factors such as bone size and thickness of the cortical rim and endplates need to also be considered [16]. Consideration of these simple engineering principles may assist in fracture risk prediction [31]. Each of these factors is influenced independently with age, immobilisation, metabolic bone disease, disc degeneration and osteophyte formation. It is therefore difficult to determine the exact role of each factor and their individual contribution to the aetiology of vertebral fracture. The size of a vertebral body, often measured using computed tomography (CT) to calculate cross sectional area (CSA), has been found to be a predictor of risk of fracture [28, 65]. CSA provides an index of the ability to resist fracture given that bone strength is positively correlated with bone size [20].

Mechanical stress in a vertebral body during axial loading is inversely proportional to the CSA of the bone [16, 27]. Mazess et al. [54] found that individuals with osteoporosis had a significantly smaller vertebral CSA than individuals without osteoporosis. In a study comparing osteoporotic women with and without vertebral fracture, it was found that those with fracture had a reduced vertebral CSA in T12-L4 when matched for age, height, weight and BMD [28]. Another study comparing women with and without vertebral fracture found that vertebral volume was significantly lower in the fracture group when matched with controls for age, height, weight and BMD [15]. The smaller vertebral size in the fracture group would increase mechanical stress in the vertebral body, thereby limiting the loading capacity of the spine. Superimposing smaller vertebral size with the pathology of osteoporosis could increase fracture risk substantially.

4. Local Environment

4.1. Muscle Force

Muscles that attach to the vertebral column have a role in producing movement and providing protection to the spine by stabilising its structures. Without muscular support, the spine has a compression threshold of just 2kg before buckling [6, 66]. The paraspinal extensor muscles play an important role in maintaining static equilibrium of the trunk by resisting the flexion moment imposed by gravity and any mass carried anterior to the spine (see figure 2). The role of the erector spinae as a tonic muscle group has been confirmed by a histological study that found a predominance of type I muscle fibres (slow twitch, associated with endurance) at both thoracic and lumbar levels [50].

However, forces produced by muscle contraction may also be injurious to spinal structures. The muscles responsible for balancing spinal gravitational moments attach closely to the axis of rotation of the segment, therefore being at a mechanical disadvantage when considering the lever arm length. Relative to other muscles in the body, the spinal extensors have a very short lever arm. Therefore the force generated by these muscles must be sufficiently large to stabilise the trunk and overcome this mechanical disadvantage. The result is a higher compressive reaction force delivered to the intervertebral discs and vertebral centrum [14, 45, 68, 92]. Lever arm length can be affected by vertebral geometry and vertebral body position, as a function of spinal posture. Women have a smaller vertebral body size than men [6]. This helps to explain the observation that women have a 9% smaller lever arm from the erector spinae muscle to the centre of the vertebral body [27]. Gilsanz et al. [28] discovered that women with osteoporotic fractures had smaller lever arms (by 5.8%) than osteoporotic women with no fractures. The combination of reduced vertebral CSA and shorter lever arm resulted in increased mechanical loading by 8% during erect stance and by 15% during trunk flexion in the fracture cohort.

In a flexed spinal posture, the lever arm lengths of the lumbar erector spinae may decrease by up to 13.3% [9, 92]. Therefore, in order to maintain the same torque, muscle force must increase since torque is the product of force and lever length. Under normal conditions, the musculoskeletal system is able make this adjustment safely. However, given the pathology of osteoporosis, vertebral bone may be incapable of sustaining the resultant increase in compressive load. Vertebral fracture has been associated with an increase in thoracic kyphosis and a decrease in lumbar lordosis [84]. This change in spinal alignment will shorten the lever arm lengths since the spine will be positioned in greater flexion. Additionally, flexion will shift the body’s line of gravity further anteriorly from the vertebral bodies. This will increase flexion moments, requiring large extensor counter-moments and therefore an increase in vertebral compression load. Compression force may also be increased due to the change in fibre orientation of the erector spinae when the spine flexes. Lumbar flexion is associated with a change in the line of action of longissimus and iliocostalis, thus reducing their capacity to resist shear force and
increasing the compression vector [57]. Spinal posture and geometry may therefore significantly influence the risk of osteoporotic vertebral fracture.

**Figure 2.** Calculation of segmental vertebral load using a flexion moment at T5. The length of the lever arm (r) to the force vector (F) is partly related to the curvature of the spine at that level. The T12 lever arm is less than the T5 lever arm.

The flexion moment ($\tau_f$) for a given vertebra is calculated as the product of the vertical compression force (F) and the lever arm distance (R) from the vertebral centroid.

$$\tau_f = |F_1 \times R_1| + |F_2 \times R_2| + |F_3 \times R_3| + |F_4 \times R_4| + \ldots + |F_n \times R_n|$$

The extensor force (E) provides the opposing extensor moment ($\tau_e$). This force is a result of muscle and connective tissue tension, it sum of both compression (Ec) and shear (Es) vectors.

$$\tau_e = Ec_1 \times Re_1 + \ldots + [Ec_n \times Re_n]$$

To maintain equilibrium the net flexor moments must equal the net extensor moments.

**4.2. Muscle Strength**

Spinal posture may be influenced by the strength of paraspinal muscles. Back extensor strength is negatively correlated with thoracic kyphosis in some studies [39, 84, 85], but not others [17]. The strength of muscles around the thoracic spine is believed to protect against thoracic fracture in patients with osteoporosis [85]. Several studies have shown back extensor strength decreases with age [17, 47, ...
83] and has an inverse relationship with BMD [74, 86, 83, 91], both of which are established correlates of osteoporosis. The magnitude of thoracic kyphosis is only moderately correlated with mean vertebral BMD and vertebral height ratios. This suggests that factors other than vertebral density and morphometry may contribute to kyphosis, for example muscle strength [19]. Extensor muscle weakness can result from an age-related decline in motor unit numbers, progressive habitual inactivity or pain inhibition [60]. As weakness in the extensor muscles increases, they are less able to maintain the necessary extensor moment to support an erect posture. Consequently kyphosis increases and the length of the extensor muscles increases. This lengthening may further reduce their torque generating capacity when considering the length-tension relationship of skeletal muscle [55]. As kyphosis increases, either as a result of muscle weakness or prevalent vertebral fracture, the compressive stress that vertebrae sustain increases as the flexion moment increases (see figure 2). This results in potential overload of the anterior portion of the vertebral body and structural failure. A mathematical modelling study by Keller et al. [42] found that an increase in kyphosis of 41.7° due to osteoporotic deformity resulted in a 25.2% reduction in spinal height and produced a 15.1cm anterior translation of C2. These changes resulted in a 19% increase in compressive force and a 40% increase in spinal extensor force required at T7/T8. Mueller et al. [64] found intramuscular pressure in the erector spinae to be significantly greater in individuals who adopted a kyphotic posture compared to an erect spinal posture, suggesting a significant increase in muscle tension with kyphosis. It is likely that as kyphosis increases, a vicious cycle of abnormal tissue loading will ensue, and if prolonged, will manifest as pathology, for example vertebral bone failure [30].

4.3. Neuromuscular control

In addition to the role of muscle activity in maintaining an erect spinal posture, the control of this activity should also be considered. The spine is inherently unstable. Muscles around the spine and trunk are responsible for providing controlled movement and stability. Maintaining controlled movement and stability in the trunk may minimise the risk of sustaining a vertebral fracture. To ensure stability and optimal movement control, the trunk muscles must have sufficient strength, endurance and correct recruitment patterns [71]. The patterns of recruitment and relative onset times between muscles are modulated by the central nervous system (CNS). Neuromuscular control relates to the role of the CNS in ensuring optimal muscle performance.

Prior research has confirmed the role of deep trunk muscles in maintaining stability of the lumbarpelvic region [35, 36, 63]. Poor trunk stability as manifested through maladaptive muscle recruitment patterns and fatigue of deep abdominal (transversus abdominis) and deep spinal muscles (multifidus) may have an influential role in the aetiology of osteoporotic vertebral fracture. Research has confirmed that spinal intersegmental movement of 2° or more is associated with the development of local microtrauma [29]. Instability of the trunk may lead to a biomechanical overload of already weakened vertebral and perpetuate subregional microtrauma due to suboptimal movement control and muscle recruitment.

Hodges and Richardson [36] suggested that dysfunction of the transversus abdominis (TrA) muscle would lead to suboptimal control of the spine against any forces that challenge its integrity. These researchers found that activation of TrA preceded other body movements sufficient to perturb the trunk in a normal population. A similar recruitment characteristic was noted for the deep fibres of multifidus [63]. When the same test was performed in subjects with low back pain, the TrA demonstrated latency in activation, suggesting dysfunction in neuromuscular control.

Other studies have found a decrease in the CSA of multifidus in patients with chronic low back pain [11, 34, 33, 40]. Pain from symptomatic vertebral fracture may contribute to atrophy of the multifidus and further compromise intersegmental stability. Maladaptive motor control of the TrA and multifidus may also be related to other mechanisms such as alerted posture, muscle imbalance, pain and the development of a sedentary lifestyle. These factors may contribute to poor deep muscle control. O’Sullivan et al. [70] observed a decrease in multifidus activation in people who assumed a slumped sitting posture (thoracic and lumbar flexion). It is feasible that the increase in thoracic kyphosis, decrease in lumbar lordosis and change in sacral inclination associated with osteoporotic vertebral fracture may similarly affect paraspinal muscle control thereby compromising intersegmental spinal stability. This hypothesis is yet to be directly evaluated.

4.4. Intervertebral Disc Integrity

The intervertebral disc attaches to the vertebral body via the communication between the collagenous fibres of the annulus fibrosus and the vertebral end plate. The disc facilitates intersegmental movement while maintaining alignment of the vertebral column and has a primary role in attenuating and dispersing axial load. With aging, the water content of the nucleus pulposus decreases, compromising the properties of the disc and placing individuals at greater risk of vertebral injury [52]. As the disc degenerates, the mechanical load on the vertebral body may increase. Disc stiffness has been positively correlated to the degree of discal degeneration and amount of cyclical, repeated loading...
Any increase in disc stiffness will reduce movement at the motion segment. Therefore a potentially harmful increase in muscle force is required to obtain a desired movement. This response would obviously lead to greater mechanical stress through the vertebral body and thus increase the risk of fracture in an osteoporotic vertebral body.

The load bearing ratio between the cortex and trabecular bone depends on the integrity of the intervertebral disc. In healthy discs, the highest effective stresses are located in the centre of the bony end plates. For degenerated discs, the stresses are found in the lateral aspects of the end plates, in the cortical wall, and in the vertebral body rim [10, 37, 44]. These are areas of the vertebral body that have a lower threshold to axial load compared to intact trabecular bone in the centrum. Degenerative changes in the intervertebral disc have also been suggested to cause abnormal load distributions between the anterior and posterior zones of the vertebral body and neural arch [75-78]. These findings suggest that disc integrity can affect vertebral loading and potentially contribute to fracture risk by modulating changes in subregional BMD. Bone within the vertebral body could respond adaptively by altering density in response to changes in axial force transmission.

5. Global Environment

5.1. Body Position and Activity

The global environment relates to the activity and position of the body in space. As discussed previously, axial load transferred to the vertebral bodies is a function of body weight, tension in adjacent structures and intervertebral disc properties. Mechanical loading increases when body mass above the vertebra moves anterior to its axis of rotation. For example, loading will increase in situations where an individual bends forwards or rises from a chair. Duan et al. [16] estimated that activities associated with flexion of the upper body resulted in an approximate ten-fold increase in the compressive stress imposed on the vertebra compared to upright standing. For any loading scenario derived extensor moment and could therefore be considered as a significant contributor to the incidence of vertebral fracture [16]. The centre of mass will also move anterior to its axis of rotation with increasing kyphosis, which is a known correlate of osteoporotic vertebral fracture and back extensor muscle weakness. Furthermore, kyphosis is known to increase with successive vertebral fractures [4, 7, 8, 21].

Vertebral load will increase when compression forces increase, for example, carrying an additional load. The resulting compression force will then be a function of body weight, the mass of the load and the distance of the load to the vertebral body. Mannion et al. [51] found that for every increase in Newton weight held in the hands, there was a corresponding increase of compressive force of 5.23N on the lumbar spine. Vertebral fractures are assumed to occur from activities that maximise loads in vertebral bodies, particularly in vertebral bodies with altered extrinsic and intrinsic structural integrity. Peak loads in individuals will vary due to differences in activity levels and incidences of traumatic events such as falls [16]. Approximately 33% of vertebral fractures arise from falls however, in many cases the aetiology is unknown [5]. Osteoporotic vertebral fracture may simply be the result of weak vertebral bone being loaded during activities of daily living [42, 56, 59].

6. Conclusion and clinical implications

Physiologic load on vertebral bodies plays a significant role in the mechanics of vertebral fracture. However applying load estimates to predict fracture risk in vivo is difficult due to the complex geometry of the human skeleton. Loading estimates are often made with several assumptions, and this may threaten the internal validity of any model used. Furthermore, fracture risk prediction is complicated by the complexity in measuring skeletal loads during activities of daily living and traumatic events [31]. To achieve clinically sensitive measures of fracture risk, other factors should be considered in addition to our knowledge of spinal loading characteristics.

Bone microstructure, density, geometry and perhaps mineralisation will influence load bearing capacity. These factors are known to correlate well with the capacity of a vertebral body to withstand axial load, since both intrinsic (bone quality) and extrinsic (bone geometry) features can modulate load bearing capacity. Therefore, to arrive at more sensitive predictions of fracture risk, these factors should be considered and potentially measured simultaneously. Utilisation of QCT and high resolution magnetic resonance imaging (MRI) may present advantages over the more commonly used tools such as DXA and radiography. Estimations of multidimensional vertebral geometry cannot be made from DXA. Information derived from QCT allows isolation of densitometric and geometric changes in both cortical and trabecular bone [31]. MRI has the potential to make measurements of bone geometry and bone quality [22, 26, 48, 49].

Clinically, the advantages offered by QCT and MRI may be offset by the greater expense, longer scanning time and greater ionising radiation dosage delivered by QCT compared to DXA. With advances in DXA, lateral scanning of vertebral bodies in vivo with sufficient precision is now available.
Clinical applications of DXA could be directed towards measuring areal BMD in vertebral subregions from lateral scans. However, the precision and accuracy of this application is yet to be evaluated. If these parameters were researched and optimised, clinically important information concerning fracture risk prediction and response to therapies may be provided.

The capacity of the osteoporotic spine to withstand axial loads may be influenced by trabecular bone quality, subregional BMD and vertebral body geometry. However, there is limited information in the literature regarding in vivo loading of the thoracic spine, other than a mathematical modelling study by Keller et al. [42]. It remains unclear if vertebral fractures significantly affect the segmental loading profile of the spine. Future research should be aimed at examining changes in loading profiles in individuals with osteoporotic vertebral fractures. Keller and colleagues [42] discuss the implication of increasing kyphosis on vertebral load, however it is not known if a magnitude threshold exists for kyphosis to significantly affect vertebral load and therefore fracture risk. To determine this, accurate and clinically applicable tools to measure thoracic kyphosis need to be appraised and applied to cohort studies. The effect of changes in spinal curvature and pain on the recruitment characteristics of thoracic paraspinal musculature in individuals with vertebral fractures also needs to be investigated. It is likely that changes in thoracic posture will influence the recruitment characteristics of these muscles as changes in strength, force vectors and length-tension relationships ensue.

With more information on subregional vertebral BMD, vertebral loading and neuromuscular control of trunk musculature, strategies to prevent further vertebral fracture and identification of individuals at high risk of fracture may be improved. Information about impairments in neuromuscular control and vertebral loading may assist health practitioners, such as physiotherapists, who treat patients with osteoporosis. Their treatment plans may be refined to include specific motor retraining and strengthening interventions and modalities to reduce vertebral loading. Additionally, specialists in bone health may be provided with valuable information on vertebral bone metabolism and the efficacy of pharmacotherapies by investigating subregional BMD within vertebral bodies.

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Conflict of interest
The authors have declared that no conflict of interest exists.

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