The importance of mechanics in the pathogenesis of fragility fractures of the femur and vertebrae

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Summary

This review highlights the role played by mechanical imbalances in the pathogenesis of fragility fractures of the femur and vertebrae. Particular attention is paid to vertebral fractures, and the consequences arising from mechanical imbalances are analyzed and evaluated to determine how much they contribute to worsening vertebral deformity and creating a domino effect.

KEY WORDS: Vertebral compression fracture; VCF; Fragility fractures of the vertebrae; Mechanics.

Introduction

Reduction in bone mass, advancing age, fragility fractures after the age of 40, chronic steroid therapy, the possibility of falling and familiarity of fractures are the principal risk factors currently recognized in the pathogenesis of fragility fractures (1).
The risk factors related to bone mass are similar because, albeit through different mechanisms, they all alter the resistance of the 'bone material', which depends on the bone mass peak, loss of bone mass related to age, co-morbidities and their treatments, bone geometry, degree of mineralization and the complex process of remodeling to maintain efficiency (2).
However, bone resistance by itself does not explain the dynamics of a fracture. Other factors need to be taken into consideration, such as mechanics, and the weight and direction of applied force. For example, in a femoral neck fracture, correlation between bone resistance and length of femoral neck, where there is equal bone mass, shows that the longer femoral neck is more exposed to the risk of fracture (3); however, it has not yet been shown that a wide cervico-diaphyseal angle leads to an increase in the risk of fracture although, as we discuss later, we believe it is highly probable.

Women with a vertebral compression fracture (VCF) have a 2–3 times greater risk of a new fracture, and the risk of incurring a new VCF is increased even where there is normal bone mass (4,5). This could be due to the variation in weight generated by mechanical imbalances deriving from vertebral fractures.
Furthermore, VCFs are more frequent in the mid-thoracic area and the thoracolumbar passage (6); however, the mechanisms that explain the frequency of fractures in these sites through the change in mechanical load have not been sufficiently analyzed.
The aim of this paper was to show how mechanical imbalances by itself, and even more when associated with bone fragility, is a significant risk factor for fractures; and, when vertebral fractures do occur, they should taken into account and considered on a par with the other generally accepted risk factors that were already mentioned.
We also want to draw attention to the necessity of evaluating the degree of deformity and the site of VCFs for predicting the consequences on a mechanical level, and the possibility for worsening the degree of deformity with a potential domino effect.

Mechanical pathogenesis of femoral neck fragility fractures

The resistance of the femoral neck is through structural and mechanical components.
The structural components that play a relevant role, together with the degree of cortical thickness and the integrity of the trabecular structure, are the trabecular supporting arciform, cephalic and trochanteric bundles; their development and integrity contribute to normal bone resistance (7).
Wolf's Law states that the individual development and consistency of the single trabecular bundles are related to the width of the cervico-diaphyseal angle in the femoral neck. During walking, different loads are imposed on the femoral neck by forces of compression and tension, according to the width of the cervico-diaphyseal angle; this leads to further development of the trabecular bundles. When the cervico-diaphyseal angle is of normal width, i.e. 130°, the forces of compression and tension are balanced and the development of single bundles is in proportion and harmonized.
In cervico-diaphyseal valgism, the cephalic bundle is more developed than the arciform because the force of compression prevails; in varism, the arciform bundle is more developed through greater tension (8). It follows that the harmonious development of the trabecular bundles in the femoral neck depends on a state of mechanical balance that exists only with a cervico-diaphyseal angle of 130° (Figure 1).
Ward's triangle is a triangular area of minor resistance, situated at the base of the femoral neck and defined by the intersection of the trabecular bundles, the dimensions of which depend on the length and width of the femoral neck and the structural changes through age. Right at the base of the femoral neck, where Ward's triangle is situated, there are particularly intense forces of flexing strain, the degree of which is related to the length of the neck. (9,10).
The flexing force at the base of the femoral neck is taken from the momentum generated by the force of weight, in gravitational terms, multiplied by the arm, which is greater the longer the femoral neck: $M = Fp \times b$ (Figure 2). As already reported (3,11,12), just as with bone mineral density (BMD), a long femoral neck carries a grea-

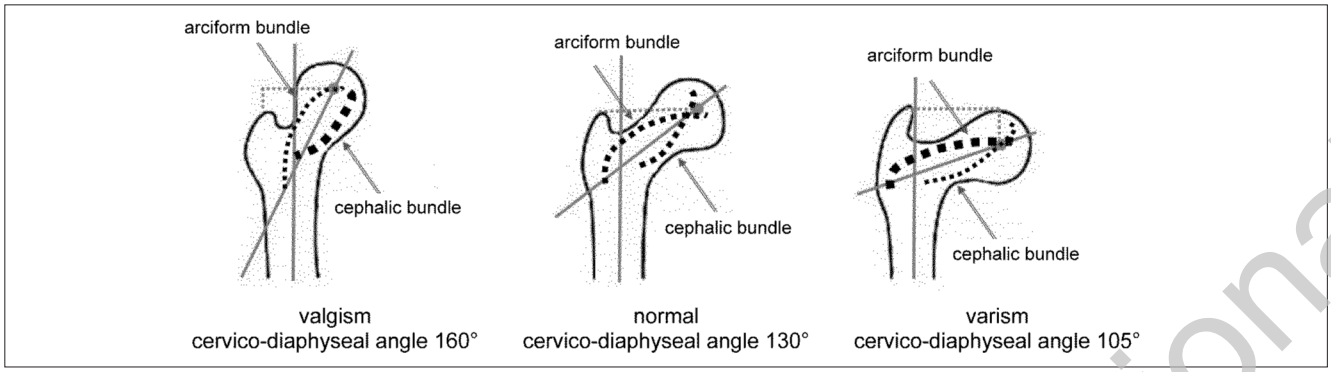

Figure 1 - Development of the strengthening trabecular bundles of the femoral neck compared to the width of the cervico-diaphyseal angle.

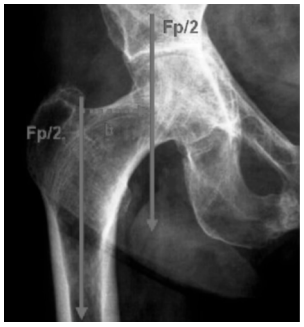

Figure 2 - Flexing momentum in relation to the length of the femoral neck. *Fp/2* = force of weight/2; *Fp/2t* = force of weight/2 transferred on the diaphyseal axis; *M = Fp/2t ×b.*

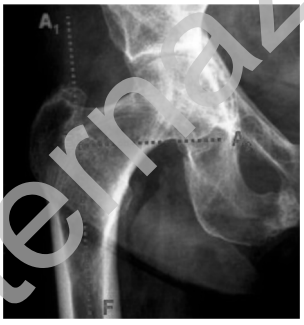

Figure 3 - Flexing momentum in relation to the width of the femoral cervico-diaphyseal angle. $\angle A_1EF = 180°$; $\angle A_2EF = 90°$; M = *Fp/2t* × *b* where $\angle A_1EF$ = *Fp/2t* and $\angle A_2EF$ = *b*.

ter risk of fracture because of a more intense flexing momentum. The width of the cervico-diaphyseal angle is also important in evaluating the risk of fracture of the femoral neck (3,11,12), in fact it is reasonable to expect that the risk increases with varism and decreases with valgism. In the absurd example of a cervico-diaphyseal angle of 180°, the absence of the arm would see momentum coincide with the force of weight, but if the angle was 90°, the presence of the arm would generate a particularly intense momentum. Cervico-diaphyseal varism, where the arm is greater, carries a risk of major fracture compared to valgism (Figure 3). In summary, when the gravitational force remains constant, the arm becomes variable in terms of influencing the risk of fracture.
We can therefore theorize that the risk of fracture of the femoral neck depends not only on its length but also on the degree of varism, and that the simultaneous presence of both these conditions can lead to a further increase in the risk of fracture (Figure 4).

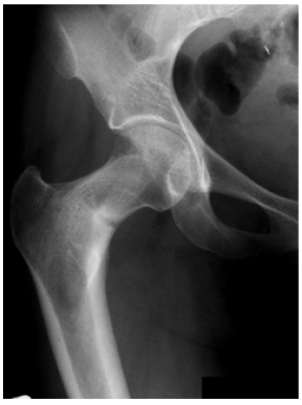

Figure 4 - Risk factors of length and cervico-diaphyseal angle in femoral neck fractures: incomplete basicervical fracture of a femoral neck with excessive length and cervico-diaphyseal varism.

Mechanical pathogenesis of vertebral fragility fractures

The fracture of a vertebral body depends on the resistance (density of bone tissue, architecture and characteristics of the materials , on its volumetric dimensions and the load applied materials (13,14); all these components vary according to gender and age. The vertebral body carries on growing up to the age of 30, the peak point for bone mass, when it reaches its maximum resistance to load (15); all these components vary according to gender and age. After thirty, resistance gradually decreases by thinning and disruption of the trabecular structure and by reduction of the cortical thickness. These processes lead to a reduction in bone mass and mineral density, which can reach 80% of the original value (16).
In defining the overall resistance of a vertebral body, together with cortical thickness and horizontal, vertical and oblique trabecular structures, there is also the so-called *fan bundles*, which, like those in the femoral collar, act as structural reinforcement. A triangular area in the anterior part of the vertebral body exists that is less resistant to load through the absence of a horizontal trabecular structure and bundles (Figure 5). It has been shown that in order to bring about the collapse of the anterior wall in a vertebral body with normal resistance, it is enough to apply a compression force of less than 25% compared to that needed for the collapse of a posterior wall (17).
In the physiological configuration of kyphosis of the dorsal spine, the compression force on the anterior margin of the vertebral bodies is correspondingly greater at T7 and T8 (18) and, because the thoracic column is part of the structure within the thoracic cage, the same force is also present at L1. This hypothesis is suppor-

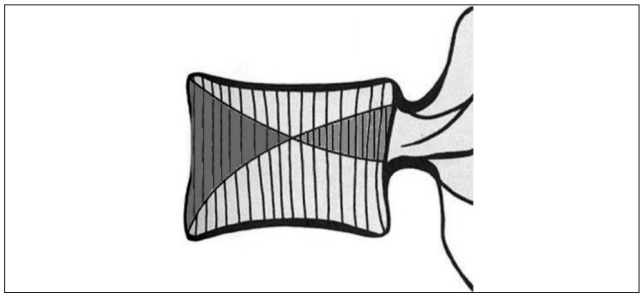

Figure 5 - Vertical section of a vertebral body. The area of least resistance due to the absence of horizontal trabeculature and fan bundles is shown in red; the grey area is that of greatest resistance due to the presence of the intersection of fan bundles and horizontal trabecules.

ted by the results of observational studies (6) which have shown a greater prevalence of stress fractures in these vertebrae.
Now we discuss how the increased occurrence of stress compression fractures of the vertebral body in these areas and the type of deformity (wedge vertebra in the thoracic tract, butterfly vertebra in the thoracolumbar passage, biconcave or herringbone vertebra in the lumbar tract) (19) depend on the degree and direction of mechanical strain in relation to the gravitational axis (20,21,22).
The forces that act on the bones that form the skeleton are compression, traction, torsion, flexing and shear; these can work separately or in different combinations (20,21,22).
In humans, the main force at work on the vertebrae is axial compression, which depends on load. When standing erect and bending all the axial load is supported by the vertebral bodies; when stretched the greater part is supported by the posterior somatic structure. The role of tangential and transverse tensions, relating to the articulated arches and facets, is irrelevant in the pathogenesis of VCF (20,21,22).
The *physiological load* is that which acts on vertebrae during normal daily activity and the *mechanical strain* corresponds to the force applied divided by the superficial area and is expressed in terms of Newtons per square metre (N/m^2).
Bone tissue, in common with all other solid materials, is subject to mechanical stress, and fractures that appear following a reduction of resistance to a repeated load are defined by force/stress or fatigue. Load stresses and resistance to compression are measured in Newtons (N) (16).
As observed when standing and from the side, physiologically the spine displays a curve of posterior convexity (kyphosis) in the thoracic tract and a curve of posterior convexity (lordosis) in the lumbar tract. The extent of curvature varies according to the lumbosacral angle from which the lordotic curve departs perpendicular (23, 24).
The extent of the kyphotic curve determines the size of the arm, expressed by the perpendicular distance from the gravitational axis, and therefore the intensity of momentum, which is higher for the vertebrae of the spinal tract where the convexity of the curve is greatest. The intensity of the bending moment can be calculated using the following formula:

$$m = Fp \times b$$

where *M* is the momentum, *Fp* is force of weight and *b* is the arm.
Because vertebrae T7 and T8 are those that physiologically support the greatest bending moment, they are also more exposed to risk of fracture when if kyphosis is accentuated (Figure 6).
This same mechanism, with a few differences, can explain the greater incidence of load fractures to T12 and L1 where the spine is flexed. Unlike the upright position, with the spine flexed, it is primarily forces perpendicular to the ground that act on the first thoracic vertebrae, and in T12 and L1 particularly intense flexor forces arise.

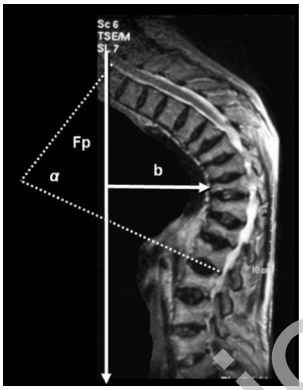

Figure 6 - Dorsal hyperkyphosis acquired through vertebral compression fractures. Particular intensity of the bending moment *(M = Fp × b)* on the dorsal spine originating from hyperkyphosis through VCFs with consequent increase in the arm, pushing the gravitational axis forward and reducing the depth of angle α.

The type of vertebral deformity can also influences mechanical strains and the positioning of the gravitational axis in relation to the physiological curve of the spine. In the dorsal tract, where the curve is kyphotic and the gravitational axis is pushed forward with the vertebrae anchored to the thoracic cage, the type of vertebral deformity through fracture is mainly an anterior trapezoidal wedge type. In the thoracolumbar tract, the meeting point of a rigid segment and an extremely mobile one, in which the gravitational axis tends to be central to T12 and L1, the type of deformity can more easily be butterfly vertebra. Finally, in the lumbar tract, where the curve is lordotic and the gravitational load axis is pushed backwards, the load tends to depress the constraining structure creating a biconcave vertebral deformity.

Mechanical pathogenesis of the domino effect

It has been shown that a postmenopausal woman diagnosed with VCF has a 20% greater risk of encountering a new vertebral fracture within the year (5). The risk of new VCFs increases with the number and degree of severity of previous fractures. This sequence of fracture events is related to the changes deriving from the mechanical imbalances caused by VCFs.
In a vertebra with normal morphology, the straight lines of the limiting structure tend to remain parallel or at most meet at a far distant point. In a wedge vertebra, however, the greater the degree of deformity, the closer to the vertebral body the two lines intersect. The larger the angle, the more severe the deformity. Wedge deformity of the vertebra in the thoracic tract leads to accentuation of the kyphotic curve. The mechanical imbalance derived from the changed morphology of the vertebral body leads to a forward shift of the gravitational axis with a consequent increase of the arm and therefore of the bending moment. This force is capable of aggravating the initial deformity of the vertebra and dragging the other vertebrae into a 'fracture waterfall', thus creating a domino effect. It is likely that the greater the number and the severity of the vertebral deformities, the more intense will be the bending moment and therefore the greater the risk of new and more severe vertebral fractures.
The posture change that occurs after fracture of T7 and T8 and which accentuates the kyphotic curve at T12 and L1 and causes a reduction in the lordotic curve, has a negative effect on the whole spine.
Thus, pathogenesis of the domino effect is almost exclusively mechanical and the movement of the gravitational axis generated by VCFs creates an increase in the bending moment, and the

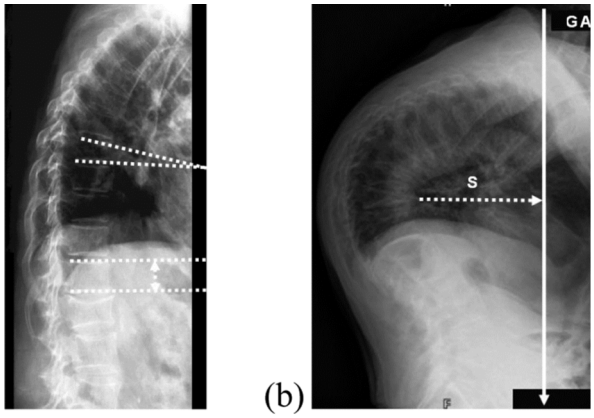

Figure 7 - Mechanical pathogenesis of the domino effect. (a) Tangential lines at the limits of a normal vertebra are parallel; in the case of VCF they intersect; the nearer the intersection to the vertebra the greater the degree of deformity. (b) Accentuation of the dorsal kyphosis in the case of VCFs with forward displacement of the gravitational axis and the domino effect. GA, gravitational axis; S, magnitude of forward displacement of the gravitational axis.

dorsolumbar muscles are unable to counterbalance the effect (Figure 7).

The criticality of VCFs

The degree of vertebral deformity and the spinal area involved, generally when the fractures are localized in the mid-thoracic and thoracolumbar tract, have a decisive role as far as the domino effect is concerned. In patients with VCF, it would be particularly useful to evaluate the level of 'criticality' in order to prevent a further worsening of the deformity and avoid the domino effect.
Criticality pertains to:

(a) The site of the VCF, corresponding to the spinal segment most greatly stressed by the bending moment:
    1. mid-thoracic tract (T7–T8)
    2. thoracolumbar passage (T12–L1)
(b) The degree of vertebral deformity: the mechanical imbalances arising from this could create a domino effect.

The *degree of vertebral deformity* is commonly calculated according to the Genant criteria (19), and vertebral fracture is classified as light, moderate or severe if reduction in height is 20–25%, 25–40% or more than 40% respectively. The degree of vertebral deformity can be calculated as a percentage and expressed as an absolute value by the following formula:

$$d\% = \frac{|h_p - h_a|}{h_p} \times 100$$

where $d\%$ is the percentage degree of deformity, h_p is the posterior height, and h_a is the anterior or medial height.
Detecting a criticality condition in a VCF could lead, together with pharmacological therapy, to a decision to employ mini-invasive surgery, such as vertebroplasty or percutaneous kyphoplasty (25), to reduce or stabilize the vertebral deformity in order to minimize the negative effects of mechanical failure.

Conclusion

When evaluating the risk factors for fractures, mechanical components must also be taken into consideration. Bone resistance on its own does not explain the fracture of a particular skeletal segment, which can be affected by mechanical components such as the degree of load and the direction of force applied. In the femur, the length of the neck and the degree of the cervico-diaphyseal angle influence the intensity of the bending moment. The femoral neck is more exposed to risk of fracture not only when it is long but also when the cervico-diaphyseal angle tends towards varism. We believe that both these mechanical components should be considered as risk factors for fractures, independent of BMD.
The vertebrae of the mid-thoracic tract and the thoracolumbar passage are those at the highest risk of fracture. Thoracic kyphosis contributes to the moment of bending stress, which is particularly intense in the condition of primary or acquired hyperkyphosis. The degree of kyphosis should, however, be considered a risk factor for BMD-independent fracture.
Vertebral fractures in the mid-thoracic tract and thoracolumbar passage cause significant imbalance with regard to the forward displacement of the gravitational axis and consequent increase of the arm and bending moment. The increase in bending force could be the main cause of the domino effect, suggesting largely a mechanical pathogenesis. Vertebral fractures that appear in these "critical" locations are particularly risky with regard to the domino effect, and should be stabilized as soon as possible with mini-invasive surgery such as vertebroplasty or kyphoplasty.
The authors are aware that the biomechanical ideas expressed in this review are based on mathematical calculations, and their foundation needs to be confirmed in clinical studies.

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