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Osteoporosis and vertebral trabecular bone health: an historico-anthropological perspective

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Abstract: This brief review article aims to recapitulate the history of osteoporosis from the most ancient observations to the current clinical definition, by offering a perspective on trabecular bone health and degeneration, which has become of paramount important both in clinical, radiological and biological anthropological studies.

Key words: osteoporosis, trabecular bone, endocrinology, history of anthropology, biological anthropology, palaeopathology.

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The first identification of a link between a structural abnormality in bones and subsequent fractures is to be ascribed to the British surgeon and anatomist Sir Astley Paston Cooper (1768–1841) (Lorentzon and Cummings 2015), while the technical term naming this condition, 'osteoporosis' (Fr.: *ostéoporose*), dates back to 1833, when the German-born French pathologist and surgeon Jean G.C.F.M. Lobstein (1777–1835) was able to describe an expansion of the marrow spaces at the expense of the trabecular bone in osteological specimens (Brand 2011).

Nowadays it is known that trabecular bone has a characteristic network of lamellar bony plates and rods which shows less density, homogeneity and lower degree of parallel orientation. It shows a wide variability in strength and stiffness to be related with this type of bone's apparent density (Osterhoff et al. 2016). Osteoporotic loss occurs as a result of an imbalance of the remodelling process governing bone homeostasis, noting that this remodelling activity is higher in the central skeleton, which explains bone loss-related vertebral fractures (Osterhoff et al. 2016). Although trabecular bone accounts only for approximately 20% of the total skeletal bone mass, it is responsible for most of a skeleton's turnover, which is particularly apparent in individuals younger than 65 years of age (Osterhoff et al. 2016).

Lobstein wrote that osteoporosis implies a reduction of internal cohesion between the molecules of the bones (*elle suppose une diminution de cohésion entre les molecules de l'os*), a diminution which he thought was caused by a certain 'expansive force' (*force expansive*). Concurring with previous similar observation by the Italian anatomist Antonio Scarpa (1751–1831), he defined this force as responsible for the softening, swelling and rarefaction (*le rammollissment, le goflement et la rarefaction*) of the bone the result of the activity of excited nerves (*l'activité exaltée de nerfs*), possibly activated by special pathological principles connected with some of the major diseases such as the venereal, the arthritic, the variolar, morbillar, etc. (Lobstein 1833; Schapira & Schapira 1993), since at the time he was not aware of the currently known aetiology of osteoporosis.

While it is now believed that, in fact, what Lobstein commented on may have been osteogenesis imperfecta type I, yet this appears to be the earliest known mention of trabecular bone pathology (Lorentzon and Cummings 2015). Consequently, the word osteoporosis became rapidly popular in medical circles by the late 19th century (Brand 2011).

While still unable to name a precise cause for this disease, in the 1850 edition of German pathologist August Förster's (1822–1865) anatomical pathology textbook, a description closer to the current one is to be found: 'This condition of macerated bone [*des macerierten Knochens*], called Lobstein's osteoporosis [*diese von Lobstein Osteoporose*], is caused by various processes, some of which are still unknown to us [*ist durch verschiedene, und zum Theil noch unbekannte Vorgänge bedingt*]. Most frequently, inflamed or rachitic bones, when macerated at certain periods, i.e. after healing but before complete ossification, take on the shape described; older and newer authors also assume hypertrophy of the medulla as a condition for expansion of the medullary spaces and inflation of the bone [*eine Hypertrophie des Markes als Bedingung der Ausdehnung*

der Markräume und Aufblähung des Knochens]' (Förster 1850).

By the late 19th century, the general pattern of age (advanced) and sex (female) distribution for this disease had become known, particularly with reference to fractures (at the time the principal factor leading to a diagnosis), while the manifestation of osteoporosis in males and its lower frequency were not yet completely understood (Brand 2011).

Growing knowledge on bone physiology owing to the work of Albert von Kölliker (1817–1905), who named osteoclasts in 1873, and of Carl Gegenbauer (1826–1903), who described osteoblasts, together with more information being gradually available on the impact of parathyroid hormone and estrogen on bone (resorption vs new production) would make a better understanding of osteoporosis possible (Grob 2014).

During WW2, in 1941, Albright and colleagues examined clinical data of 42 cases of what they called 'generalized osteoporosis', *a priori* excluding patients aged over 65 years because senescence could then become a primary factor in the pathophysiological process: 40/42 patients were female individuals who had passed the menopause and they had not developed osteoporosis before it, while 2/40 were males for whom no apparent causal explanation was identified (Albright et al. 1941). This analysis stressed the fact that osteoporosis was caused by deficient osteoblastic activity, hence it was a deficit of formation rather than mineralization (Albright et al. 1941; Forbes 1991).

It was precisely this study, which saw the implementation of X-ray imaging, that determined the predilection for the spine and pelvis by osteoporosis and that showed how long bones became affected merely in instances of more severe involvement. It also highlighted how osteoporosis typically does not affect the skull, unlike hyperparathyroidism (Albright et al. 1941). Albright and colleagues' study also presented the main radiological characteristics of spinal involvement in osteoporosis: fractured/crushed vertebrae, 'fish vertebrae' (i.e. biconcave vertebral bodies due to the expansion of intervertebral disks), Schmorl's nodes (herniation of the nucleus pulposus) (Albright et al. 1941).

This research additionally indicated how, while an osteoporotic vertebral lesion can be revealed by X-ray imaging after a patient's report of back pain, it can also be an incidental finding because the vertebral changes can develop completely asymptomatically (Albright et al. 1941). With reference to vertebral damage, one of the key merits of the Albright et al. study was also to show that the administration of long-term therapy was capable of arresting it, including statural loss, in postmenopausal women affected by osteoporosis or prevented them altogether if started in the early stages of the disease (Forbes 1991).

In 1973 Gallagher and colleagues examined the nature and presentation of osteoporosis-induced vertebral fractures in 58 postmenopausal women, a condition which they named 'The Crush Fracture Syndrome', combining radiological, histological and metabolic data (Gallagher et al. 1973). This study proved that osteoporotic fractures included patients with various clinical pictures, irrespective of their mineralization rates, and that fractures had to be related to a severe reduction in the amount of trabecular bone. This reduction of bone in vertebral bodies was reflected by a similar loss of trabecular bone in biopsies from patients'

iliac crests which were subjected to histological analysis (Gallagher et al. 1973). This study also pointed out that, while loss of trabecular bone is of the primary relevance in the pathogenesis of osteoporotic fractures, also cortical bone is affected by the pathological process, in that it is lost 'at a faster rate than normally occurs in postmenopausal women', and this becomes ever more apparent if the patient manages to survive long enough and the disease chronicizes (Gallagher et al. 1973). The authors concluded that osteoporosis-related crush fracture syndrome could be seen to be a self-limiting disease only at the spinal level, whereas such an attribute could not be given to its metacarpal manifestation (Gallagher et al. 1973).

Following in the footsteps of the Albright et al. study and the Gallagher et al. one, the continued implementation of lateral thoracic radiographs in subsequent research indicated that 'in some cases, vertebral fractures may be the result of a gradual loss of vertebral height rather than sudden vertebral collapse' (Hedlund et al. 1989), which could explain the asymptomatic nature of some of the vertebral changes first described by Albright et al. Hedlund et al.'s study demonstrated how the first stage of non-traumatic spinal fractures involved an initial wedging of 1–2 mid-thoracic or thoracic-lumbar vertebrae, which would then likely involve ≥ 4 vertebrae, hence ultimately making a final posterior vertebral collapse much more frequent (Hedlund et al. 1989). The final stage of the process presents with an even distribution between T6 and L2 manifesting with anterior and posterior collapse (Hedlund et al. 1989). In this study it was also shown that vertebral fractures did not only reduce vertebral height but also explained wider vertebrae (Hedlund et al. 1989).

This research also highlighted that anterior vertebral fractures were found to be more frequent in the mid-thoracic spine, while a more lumbar distribution was to be described for posterior fractures, a difference that could be explained with the direction of compressive forces according to the natural kyphotic (thoracic) and lordotic (lumbar) curvatures of the spine (Hedlund et al. 1989). Hedlund et al.'s paper finally concluded that an individual's body mass seemed to be more related to spinal vertebral fractures than vertebral size but did not exclude the possibility that density of nonfractured vertebrae could be less in osteoporotic patients (Hedlund et al. 1989).

As research on osteoporosis continued to progress a new radiological methodology was implemented, that is dual-energy X-ray absorptiometry which allowed for the assessment of bone mineral density (BMD), the current diagnostic gold standard as it is considered a surrogate marker of bone strength, although it only accounts for 60% of bony fragility variation (McDonnell et al. 2007).

However, BMD does not fully explain changes in trabecular architecture, tissue properties and accumulation of microdamage, as it has been shown in subsequent studies that a patient's risk of fracture is influenced by bone quality as defined by the aforementioned aspects (Osterhoff et al. 2016), especially when one considers that a parameter like loss of strength is more affected by perforation of the trabeculae than by their general thinning (McDonnell et al. 2007).

Further advances were made possible by the application of microCT analysis, which is nonetheless not allowed on living patients' due to the high radiation

exposure, but proved excellent for *in vitro* studies, cadaver or anthropological studies (McDonnell et al. 2007). In particular it permits scientists to produce detailed and accurate 3D reconstructions of trabecular volumes (McDonnell et al. 2007).

With a special focus on vertebral changes the following characteristics are to be considered:

a. microstructure and density are not uniform in the vertebral centrum;
b. the regions of the centrum closest to the endplates and in the posterio-lateral regions show the highest volume fraction and BMD;
c. the middle and anterior regions of the centrum show the highest trabecular separation and degree of anisotropy;
d. the high percentage of anterior wedge vertebral fractures can be explained by the the anterior region's relatively low density and high degree of anisotropy in the anterior region (Osterhoff et al. 2016).

Additional studies have shown that vertebral trabecular architecture increasingly becomes anisotropic with the progress of bone loss, which can be a significant factor in fracture risk due to the fact that trabecular architecture tries to adapt to compensate for the loss of bone (Osterhoff et al. 2016).

A study on population sample of 541 women and 490 men aged 17 to 88 years, trabecular vBMD in both sexes decreased from the T1 to L3 in all age categories, L3 showing the lowest vBMD of all other thoracic and lumbar vertebrae (Chen et al. 2013).

A cadaveric study on 56 L4 vertebrae from Asian donors aged between 57 and 98 years showed, at microCT scan and scanning electron microscopic analysis, that trabecular bone volume fraction (BV/TV) and trabecular number (Tb.N) significantly decreased with advancing age, with a similar pattern in males and females. Additionally, trabecular separation augmented with increasing age, while decrease in trabecular thickness (Tb.Th) was not found to be statistically significant (Chen et al. 2013).

Moreover, some studies suggest that the loss of horizontal trabeculae is the only one occurring while other studies indicate that both horizontal and vertical trabeculae are lost with age (Chen et al. 2013).

Future studies investigating trabecular health will likely show a pattern of deterioration of such structures fundamental for bone strength and their assessment in past populations, thanks to the analysis of large skeletal populations, could provide an evolutionary perspective on this condition.

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Conflict of interests

We have no competing interests.

Authors' contribution

Both authors contributed equally to this paper.

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