Degenerative disc disease and osteoporosis


In this epidemiological cross-sectional study with a very large sample (7144 patients), the authors found a significant relationship between high bone mineral density (BMD) and low-back pain. The authors hypothesized that degenerative changes in the spine may be a possible explanation for the observed relationship between higher BMD and low-back pain. Of special interest is the increasing evidence in the literature for a significant correlation between high BMD and degenerative changes of the intervertebral discs.

Old pathophysiological theories of spinal degeneration predicted that reduced bone quality would lead to progressive spondylosis, endplate degeneration, and arthritic changes in the facet joints, ultimately culminating in increased disc degeneration. However, several reports have demonstrated that patients with low BMD, despite presenting higher risks of vertebral body fractures (and, in some cases, advanced multilevel vertebral body collapse), seem to paradoxically present reduced rates of intervertebral disc degeneration (Figs. 1 and 2).³,⁴

In a recent study, we have demonstrated that the relationship between intervertebral disc degeneration and BMD can be basically explained by both vascular and mechanical pathways (Fig. 3).⁷ According to such a paradigm, osteoporosis would possibly delay intervertebral disc degeneration because of an increase in intradiscal nutrient diffusion by increased endplate vascularization.⁷ Additionally, because there would be decreased endplate resistance and decreased intradiscal strain due to the low quality of the bone, the overall stress upon the intervertebral discs adjacent to osteoporotic vertebral bodies would be reduced.⁴ Although the long-term effects of low bone quality on the overall health of the spine in terms of its bony, discal, and ligamentous structures may be debatable (especially taking into account the progressive sagittal imbalance related to multilevel vertebral body wedging and fractures), it seems clear that, at least at the local level of individual functional spinal units, the presence of adjacent vertebral bodies with reduced BMD tends to lead to delayed intervertebral disc degeneration.

The findings of the study by Lee et al. are in accordance with such a paradigm, as they clearly demonstrate that, although patients with lower BMD may have acute

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**Fig. 1.** Sagittal MR images of the lumbar spine (T2-weighted sequence [left] and STIR sequence [right]) obtained in a patient with severe osteoporosis. Note the delayed disc degeneration at the levels (T12–L1, L1–2, and L2–3) where the vertebral bodies exhibit evident signs of decreased bone density (such as decreased height, osteoporotic fractures, and more hypointense bone signal) in comparison with those levels (L4–5 and L5–S1) where there is evidence of higher bone density (such as preserved vertebral body height, isointense signal of the vertebral body, and sclerotic/modic changes of the vertebral endplates). At some of the levels at which the osteoporotic changes are more prominent, it is even possible to observe significantly enlarged feeding vessels toward the vertebral endplates (arrows), suggesting increased endplate vascularization, a factor that has been hypothesized to be involved in the pathophysiology of the delayed disc degeneration in patients with osteoporosis.

**Fig. 2.** Left: Reconstructed sagittal CT scan of the lumbar spine of a patient with advanced osteoporosis, demonstrating preserved height of the disc spaces (especially at the L4–5 and L5–S1 levels) adjacent to the lumbar vertebrae with low BMD, as measured in Hounsfield units (hypointensity [arrows]). Right: Reconstructed sagittal CT scan of the thoracic spine demonstrating clear signs of disc degeneration (such as decreased height of the disc spaces) adjacent to vertebral bodies with high BMD (arrowhead). Reproduced with permission from Mattei TA: Osteoporosis delays intervertebral disc degeneration by increasing intradiscal diffusive transport of nutrients through both mechanical and vascular pathophysiological pathways. Med Hypotheses 80:582–586, 2013.
and recurrent episodes of low-back pain related to vertebral body fractures, the overall levels of low-back pain in such a population tend to be lower than in patients with normal BMD. Although delayed intervertebral disc degeneration may at least partially explain such findings, future studies are required in order to investigate other factors, especially the status of the facet joints, that may contribute to the observed correlation between lower BMD and reduced levels of low-back pain.

Finally, as there seems to be increasing evidence that BMD plays a significant role in degenerative disc disease, it seems of paramount importance for future clinical studies to evaluate the long-term effects of currently widespread medical therapies for osteoporosis (such as the use of bisphosphonates, calcium and vitamin D, and parathyroid hormone analogs) on the degeneration status of intervertebral discs.

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Disclosure
The author reports no conflict of interest.

References

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