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).7,8

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). According to such a paradigm, osteoporosis would possibly delay intervertebral disc degeneration because of an increase in intradiscal nutrient diffusion by increased endplate vascularization. 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. 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

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.

**Disclosure**

The author reports no conflict of interest.

**References**


**Response:** No response was received from the authors of the original article.

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Complex regional pain syndrome


The transpsoas approach, which represents an alternative to posterior lumbar interbody fusion (LIF), has many advantages including shorter duration of hospital stay and less morbidity in comparison with anterior LIF or transforaminal LIF. Morr and Kanter reported on the case of a patient presenting with left L-5 radiculopathy after left-side lateral LIF and implantation of a polyetheretherketone (PEEK) interbody graft, complicated by new-onset discomfort, complex regional pain syndrome (CRPS), on postoperative Day 4; no device failure was demonstrated on follow up CT and radiography.

In fact, there are many possible mechanisms predisposing to CRPS, including an inflammatory process, reperfusion injury, and nerve damage–induced CRPS. In their case report, Morr and Kanter did not provide detail about the surgical procedures, such as the docking point of the tube to the disc, or the duration of tube dilatation. These two steps were important for avoiding possible damage of nerves during placement of the tube in the proper anatomical location between the nerve roots of the lumbar plexus and sympathetic chain during lateral LIF.

Additionally, using contrast-enhancing MRI or myelography to exclude the possibility of nerve compression is crucial in managing a case that is complicated by unexpected CRPS after lateral LIF.

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Response: We thank Dr. Cheng et al. for their interest and inquiry in our case and the discussion of CRPS following lateral LIF. In regard to the retractor’s docking point, we typically position the dilators at approximately the midpoint of the disc space, cheating posteriorly as we ascend from L4–5 rostrally since the lumbar plexus is located more posteriorly. Based upon review of our intraop-

Fig. 1. Intraoperative fluoroscopic images revealing retractor system appropriately docked at midpoint of the disc space and slightly ventral cage placement to minimize injury to anterior lumbar plexus at L-4–5.
To the Editor: We read with interest the recent paper by Qureshi and associates on cost-effectiveness of cervical disc replacement surgery (Qureshi SA, McAnany S, Goz V, et al: Cost-effectiveness analysis: comparing single-level cervical disc replacement and single-level anterior cervical discectomy and fusion. Clinical article. J Neurosurg Spine 19:546–554, November 2013). This is certainly a topic in need of a dispositive analysis. However, several of the authors’ assumptions lead us to question their conclusions.

Their utility values are only loosely based on the literature and are assigned somewhat arbitrarily. They make no allowance for impact of perioperative complications on utility, except for surgical failure. Other complications, such as neural injury, infection, and hematomas, vary between operation types and may be very important. Assumptions about rate of device failure and adjacent-level disease have momentous effects on outcome. The authors admit that there are no data on cervical disc replacement (CDR) survival beyond 6 years. Nevertheless, they base lifetime outcomes on an undocumented device survival of 20 years and fail to report adjacent-level disease after CDR surgery. In contrast, most long-term follow-up studies of adjacent-level disease after anterior cervical discectomy and fusion (ACDF) show that the rate does not remain 3% per year over a lifetime. One of the major rationales for CDR is the promise of reduced adjacent-level disease; there is accumulating evidence that this promise has not been fulfilled.

Their cost calculations rely entirely on procedure-related costs. Whitmore et al.2 have demonstrated that a large proportion of the direct medical costs of cervical surgery are related to follow-up costs, such as the costs of medications, rehabilitation, and emergency room and office visits. Medicare reimbursement rates involve bundling for certain combined procedures. For example, this would result in reimbursement for ACDF at far below the $3775.82 quoted by the authors. CDR devices are quite expensive, costing hospitals approximately $4500 to $6000 per device. This amount is not reimbursed by Medicare and appears nowhere in the authors’ calculations. Granted, using Medicare reimbursement as a proxy for medical costs is common. However, in this instance it is not justified.

In summary, this paper cannot address comparative costs and effectiveness over a lifetime, as claimed by the authors. Their conclusions must be evaluated with caution. Rather than guessing at lifetime results with CDR, we should perhaps await solid follow-up evidence.

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