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Editorial

Endplate changes

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Lumbar disc herniation is a common condition that is effectively treated by lumbar discectomy in patients whose symptoms fail to respond to nonoperative measures. Peul et al.[12] showed that early surgery improved recovery rates of leg pain as well as perceived recovery, but the 1-year outcome rates were similar for both early surgery and late surgery. In the Spine Patient Outcomes Research Trial (SPORT), Weinstein et al.[16,17] showed that surgery and conservative approaches both demonstrated substantial patient improvement over a 2-year period, with surgery showing consistently better but not statistically significant improvement compared with conservative treatment. At the 4-year mark, surgery was shown to be superior to conservative treatment in terms of clinical outcome.[16] However, a number of patients with successfully treated lumbar disc herniations eventually develop progressive disc space degeneration that may be associated with characteristic endplate changes initially described by Modic et al. in 1988.[9]

The Modic classification of vertebral endplate changes includes 3 progressive types of degeneration. Type 1 changes are hypointense on T1-weighted imaging and hyperintense on T2-weighted imaging and represent bone marrow edema and inflammation. Type 2 changes are hyperintense on T1-weighted imaging and isointense or slightly hyperintense on T2-weighted imaging and are associated with conversion of normal red hemopoietic bone marrow into yellow fatty marrow as a result of marrow ischemia. Type 3 changes are described as hypointense on both T1- and T2-weighted imaging and are believed to represent subchondral bone sclerosis.

Importantly, the clinical significance of these so-called Modic endplate changes in the postoperative setting remains unclear. Essentially 2 theories have been proposed regarding the etiopathology of Modic vertebral endplate changes. The biomechanical theory proposed by Modic et al.[8,9] suggests that abnormal stresses affect vertebral endplates and the microenvironment of adjacent vertebral bone marrow, resulting in histological changes. These histological changes are reflected on MR imaging as signal intensity changes.

The biochemical theory proposed by Crock[3,4] implicates the upregulation of inflammatory mediators in the nucleus pulposus in a local inflammation associated with low-back pain. The inflammatory reaction by the toxic substances from the degenerative disc may go on to produce Modic changes (MCs).[2] Albert et al.[1] suggested a theory of disc herniation in which a portal of entry for bacteria is created, with a resultant inflammatory reaction that predates MCs. This theory, however, has been less widely accepted due to its lack of proof.

The reliability of the Modic classification system is less controversial. Good to excellent kappa values were obtained for inter- and intraobserver variability by Jones et al.[5] and Peterson et al.[11] It is therefore a reproducible method of describing endplate changes. A review of the published literature suggests that the association between MCs and low-back pain is variable.[7,9,18] Interestingly, Kjaer et al.[6] have suggested that MCs associated with low-back pain represent a separate disease entity. Even less clear is the significance of these changes in the postoperative setting and their association with clinical outcome.

In this issue of the *Journal of Neurosurgery: Spine*, Rahme at el.[14] prospectively studied the radiological progression of Modic vertebral body endplate changes over an average period of 41 months. A total of 41 patients who underwent standard single-level microdiscectomy performed by a single surgeon were recruited for their final study population. The initial number of patients who underwent this procedure during the study period was 97, but only 54 (56%) consented to the study, which may have introduced some degree of selection bias. Of these 54 patients, 13 did not have any preoperative MR imaging, which further excluded them from the final study population of 41 patients.

The authors performed a standard microdiscectomy, which involved sparing of the facet and resection of only disrupted disc material with an effort to spare the anulus and endplates.

Rahme et al.[14] then studied clinical outcome using standardized scoring systems including the Oswestry Disability Index and Patient Satisfaction Index, and correlated these scores with an assessment of the vertebral endplate changes (assessed using the Modic classification) and the degree of disc space collapse on the pre- and postoperative MR imaging.

The patients were evaluated radiologically for preoperative MCs and the progression of these changes for a median period of 41 months (range 32–59 months). The prevalence of MCs preoperatively was 46.3%, which in-

creased to 78% at the final follow-up, with 63.4% cases having Type 2 MCs. Interestingly, there was also a trend for Modic Type 1 changes to progress to Type 2 changes, and for Type 2 changes to be maintained, although with a greater extent of involvement of the endplate.

Rahme et al.[14] then attempted to correlate the Modic vertebral endplate changes with low-back pain. In their analysis, the conversion to Modic Type 2 change was associated with a lower rate of back pain, although this correlation did not achieve statistical significance. They also concluded that there was no correlation between the presence of sciatica or segmental instability, disability score, patient satisfaction, or work status with the progression of MCs.

Although the natural history of MCs typically involves the conversion of Type 1 (unstable) to Type 2 (more stable) throughout a period of 18–24 months,[9,15] surgical intervention does appear to alter the course of conversion, with rigid stabilization procedures potentially resulting in stabilization of the endplate changes. Interestingly, Putzier et al.[13] suggested that dynamic stabilization may prevent progression of the degenerative endplate changes following lumbar discectomy. At 3-months' follow-up, accelerated segmental degeneration existed in the solely nucleotomized group but no progression of disc degeneration was noted in the dynamically stabilized group.

Rahme et al. are to be congratulated for their efforts to correlate the progression of postdiscectomy vertebral endplate changes with the development of low-back pain. The authors suggest that degenerative vertebral body endplate changes progress after lumbar discectomy, although these radiological changes were not strongly associated with the development of clinically significant back pain.

Further prospective clinical studies specifically examining the evolution of vertebral body endplate changes and their relationship to objective outcomes would help us better understand the temporal evolution of degenerative discogenic changes in the lumbar spine following surgical intervention. In addition, newer MR imaging modalities, including sodium double-quantum-filtered nuclear MR spectroscopy, will likely add further insights into the evolution of degenerative disc disease with or without surgical intervention.[10]

References

1. Albert HB, Kjaer P, Jensen TS, Sorensen JS, Bendix T, Manniche C: Modic changes, possible causes and relation to low back pain. **Med Hypotheses 70:**361–368, 2008
2. Braithwaite I, White J, Saifuddin A, Renton P, Taylor BA: Vertebral end-plate (Modic) changes on lumbar spine MRI: correlation with pain reproduction at lumbar discography. **Eur Spine J 7:**363–368, 1998
3. Crock HV: Internal disc disruption: a challenge to disc prolapse fifty years on. **Spine 11:**650–653, 1986
4. Crock HV: A reappraisal of intervertebral disc lesions. **Med J Aust 1:**983–989, 1970
5. Jones A, Clarke A, Freeman BJ, Lam KS, Grevitt MP: The Modic classification: inter- and intraobserver error in clinical practice. **Spine 30:**1867–1869, 2005
6. Kjaer P, Korsholm L, Bendix T, Sorensen JS, Leboeuf-Yde C: Modic changes and their associations with clinical findings. **Eur Spine J 15:**1312–1319, 2006
7. Mitra D, Cassar-Pullicino VN, McCall IW: Longitudinal study of vertebral type-1 end-plate changes on MR of the lumbar spine. **Eur Radiol 14:**1574–1581, 2004
8. Modic MT: Editorial. Modic Type 1 and Type 2 changes. **J Neurosurg Spine 6:**150–151, 2007
9. Modic MT, Steinberg PM, Ross JS, Masaryk TJ, Carter JR: Degenerative disk disease: assessment of changes in vertebral body marrow with MR imaging. **Radiology 166 (1 Pt 1):** 193–199, 1988
10. Ooms KJ, Cannella M, Vega AJ, Marcolongo M, Polenova T: The application of 23Na double-quantum-filter (DQF) NMR spectroscopy for the study of spinal disc degeneration. **Magn Reson Med 60:**246–252, 2008
11. Peterson CK, Gatterman B, Carter JC, Humphreys BK, Weibel A: Inter- and intraexaminer reliability in identifying and classifying degenerative marrow (Modic) changes on lumbar spine magnetic resonance scans. **J Manipulative Physiol Ther 30:**85–90, 2007
12. Peul WC, van Houwelingen HC, van den Hout WB, Brand R, Eekhof JA, Tans JT, et al: Surgery versus prolonged conservative treatment for sciatica. **N Engl J Med 356:**2245–2256, 2007
13. Putzier M, Schneider SV, Funk JF, Tohtz SW, Perka C: The surgical treatment of the lumbar disc prolapse: nucleotomy with additional transpedicular dynamic stabilization versus nucleotomy alone. **Spine 30:**E109–E114, 2005
14. Rahme R, Moussa R, Bou-Nassif R, Maarrawi J, Rizk T, Nohra G, et al: What happens to Modic changes following lumbar discectomy? Analysis of a cohort of 41 patients with a 3- to 5-year follow-up period. Clinical article. **J Neurosurg Spine 13:**562–567, 2010
15. Vital JM, Gille O, Pointillart V, Pedram M, Bacon P, Razanabola F, et al: Course of Modic 1 six months after lumbar posterior osteosynthesis. **Spine 28:**715–721, 2003
16. Weinstein JN, Lurie JD, Tosteson TD, Skinner JS, Hanscom B, Tosteson AN, et al: Surgical vs nonoperative treatment for lumbar disk herniation: the Spine Patient Outcomes Research Trial (SPORT) observational cohort. **JAMA 296:**2451–2459, 2006
17. Weinstein JN, Lurie JD, Tosteson TD, Tosteson AN, Blood EA, Abdu WA, et al: Surgical versus nonoperative treatment for lumbar disc herniation: four-year results for the Spine Patient Outcomes Research Trial (SPORT). **Spine 33:**2789–2800, 2008
18. Weishaupt D, Zanetti M, Hodler J, Min K, Fuchs B, Pfirrmann CW, et al: Painful lumbar disk derangement: relevance of endplate abnormalities at MR imaging. **Radiology 218:**420–427, 2001

Response

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We would like to thank Drs. Fehlings and Chua for their positive review. In our opinion, the main finding from this study is that in contrast to the natural history of MCs in patients with nonoperated sciatica, lumbar discectomy promotes the development of Type 2 changes and the conversion from Type 1 to Type 2 in the 1st 3–5 years. From a biomechanical perspective, this may reflect increased stability in the disc space following lumbar

discectomy, possibly as a result of postsurgical fibrosis and scarring, which may not necessarily occur when a lumbar disc herniation is managed conservatively. This is further supported by a trend toward less low-back pain in patients who developed Type 2 changes following lumbar discectomy.

As the reviewers note, a significant proportion of eligible patients (43 patients, 44.3%) were not enrolled in this study. Of these 43 patients, 1 had died from an unrelated cause, 19 were living abroad and could not be contacted, and 3 refused to undergo repeat MR imaging because of claustrophobia. Only 20 patients (20.6%) declined participation in the study. Although this may have introduced a selection bias in the analysis, the direction of this bias, in our opinion, is completely unpredictable. In fact, symptomatic patients may well have been overrepresented because these patients would have been particularly motivated to undergo clinical evaluation and repeat MR imaging. This is also supported by the relatively high rate of persistent sciatica (29.3%) in this patient series.

All patients had undergone MR imaging of the lumbar spine preoperatively according to our standard protocol. Unfortunately, for 13 of the 54 consenting patients, these images could not be obtained at the time of reassessment, which necessitated their exclusion from the analysis.

Finally, we agree with the reviewers that additional prospective studies focusing on the progression of MCs and their relationship to clinical findings and patient outcomes following both conservatively managed and surgically treated lumbar degenerative disc disease should help improve our understanding of this complex disorder. Such studies are therefore to be encouraged. *(DOI: 10.3171/2010.2.SPINE1041)*