The designation here of stable or unstable refers to movement on time and relatively quiescent, whereas Modic Type 1 (MT1) changes are considered unstable and more symptomatic. The authors report two cases in which MT2 changes were symptomatic and evidently unstable, and in which chronic low-back pain severity remained unaltered despite a MT2→MT1 reverse transformation. Two women (41 and 48 years old) both presented with chronic low-back pain. Magnetic resonance (MR) images demonstrated degenerating discs at L5–S1 associated with well-established MT2 changes in adjacent vertebrae. Repeated MR imaging in these two patients after 11 months and 7 years, respectively, revealed reverse transformation of the MT2 changes into more florid MT1 changes, despite unaltered chronic low-back pain severity. Following anterior disectomy and disc arthroplasty, immediate abolition of chronic low-back pain was achieved in both patients and sustained at 3-year follow up. Modic Type 2 changes are therefore neither as stable nor as quiescent as originally believed. Each type can change, with equal symptom-generating capacity. More representative imaging–pathological correlates are required to determine the precise nature of MT changes.

The authors present 2 cases in which they determined that conversion from MT2 to MT1 occurred. They concluded that “Modic Types 1 and 2 are interchangeable and equipotent in symptom-generating capacity, a fact that may more accurately reflect their associated endplate changes rather than their associated adjacent bone marrow changes.”

I completely disagree on all assertions. The conversion from a theoretically unstable MT1 to a more stable MT2 has been described by many authors.

Moreover, the authors did not include in their references the paper by our group from Strasbourg. This paper by Esposito et al. reports the findings of a prospective study of a cohort of 60 consecutive patients with chronic discogenic low-back pain (duration > 6 months) that was refractory to conservative treatment. The individuals (30 men, 30 women) constituted a clinically homogeneous cohort. All were severely disabled (Visual Analog Scale [VAS] scores ≥ 6; Japanese Orthopedic Association [JOA] scores ≤ 10), with advanced disc degeneration (Grades 3–5 according to the classification system of Pfirrmann). When there were ≥2 discs with degenerative appearance on MR images, discography was performed to determine the painful level. Patients underwent either a posterior 1-level instrumented arthrodesis and posterolateral autograft (38 patients) or interbody fusion with carbon-fiber composite (polyetheretherketone) cages filled with bone graft (22 patients). Changes were classified as MT0 in 15 patients, MT1 in 22, MT2 in 14, and MT1/2 (defined by us as a transitory state) in 9. The Wilcoxon paired sample test was used to assess the significance in the results of pre- and postoperative assessments of pain and functional status, with probability values < 0.05 considered significant. Patients harboring MT1 changes improved much more than others (p < 0.01), with good/excellent results in 72.7%. In the MT2 group, the results were poor, with good/excellent results in only 14.3% of the patients and a nonsignificant difference between pre-/post-op status. For the MT1/2 group, clinical outcome was comparable to that in patients who had presented with pure MT1 changes (p < 0.01). In the MT0 group, there was also significant improvement in both VAS and JOA scores, but in a smaller proportion of patients than in the MT1 and MT1/2 groups (p = 0.0395).

We concluded that the combination of low-back pain of discal origin and severe degenerative disc disease with MT1 lesions on MR images may lead to excellent results after fusion in a large proportion of patients. Conversely, arthrodesis for patients harboring MT2 changes is likely to result in smaller benefit of doubtful clinical significance.

Clearly the patients in our series were representative of a surgical series, and we didn’t consider the natural evolution (without surgery) of the Modic changes for each patient. However, in spite of our not having had a control group of patients who were not treated surgically, the results of our study can make a very interesting contribution to the understanding of the dynamics of Modic changes. The clinically significant results obtained after fusion for patients with MT1 may hypothetically correspond to a dramatic acceleration of the healing process of this inflammatory state. The micromovement being stopped, the inflammatory state would heal and the outcome would be better. Given that the inflammatory state may persist for a long time, fusion would allow such patients to quickly obtain a better quality of life.

Mitra and colleagues performed a longitudinal MR imaging study of MT1 changes and over time they found complete transformation to MT2 in 37.5% of cases, partial
transformation in 14.5% of cases, and maintenance of MT1 changes in 48%, but never spontaneous regression.

Kuisma et al.7 affirmed that Modic changes at L5–S1 and MT1 lesions are more likely to be associated with pain symptoms than other Modic changes or changes located at other lumbar levels. Ohtori et al.8 stated that endplate abnormalities are related to inflammation and axon growth induced by tumor necrosis factor.

So, we disagree with Dr. Marshman’s conclusion that “Modic Type 1 and 2 are equipotent in symptom-generating capacity, a fact that may more accurately reflect their associated endplate changes rather than their associated adjacent bone marrow changes.” The endplate changes are intimately related with the associated adjacent bone marrow changes. In fact, the endplate changes seem to precede the associated adjacent bone marrow changes. The endplate changes are probably a consequence of the lack of support from the degenerated disc (to the endplates).3 The intervertebral disc is largely avascular; hence, it depends on nutrition supplied to the endplates and the subchondral bone.9 The association between the endplates, the subchondral bone, and the intervertebral disc is of paramount importance.3 When degeneration occurs, natural disc nutrition would be impaired. The adjacent bone marrow would react and display the known MT1 changes, through inflammation and hypervascularization. Thus, endplate changes and associated adjacent bone marrow changes are not distinct things.

Malinin and Brown1 used nonhuman primates to investigate changes in the vertebral bodies adjacent to acutely narrowed intervertebral discs. By inducing acute disc degeneration, they caused adjacent bone marrow changes.

Their study can be considered an animal model of accelerated disc degeneration. They affirmed that disruption of the endplate vasculature by sudden loss of the disc supporting the endplates may explain the pathogenesis of lesions found in their study. Their study confirms the role of the normal nucleus pulposus as a distributor of weight-bearing forces evenly onto the adjacent vertebral bodies. Thus, when there is modification of the normal weight-bearing properties of the nucleus pulposus, weight-bearing forces on the spine become distributed unevenly on the adjacent vertebrae, resulting in microfractures and bone necrosis. The same authors anticipate that, as the result of healing and stabilization of disc function, the lesions in the vertebral bodies may heal.1 Might this experiment confirm the passage from MT1 to MT2? Unfortunately the authors did not perform sequential MR imaging.

This study clearly demonstrates that the bone lesions adjacent to acutely narrowed discs are due to changes in the biomechanical environment around the vertebral bodies.3 Thus, the most important cause of acceleration of disc degeneration would be the lack of support to the endplates, but not their integrity. The restoration of support to the endplate would theoretically allow the transition to a more stable state. When the endplate is in an inflammatory, hyper-vascular state (MT1), the restoration of support would permit the stabilization of the discovertebral unit and thus the transition to an MT0 state.

RESPONSE: We thank for Dr. Pinheiro-Franco for his interest in our article. However, we are obviously concerned that he “completely disagrees” on “all [our] assertions.” Our conclusions were straightforward, logical statements that accurately summarized our clinical findings. We described 2 cases in which florid MT2 changes were associated with invariable, disabling chronic low-back pain (CLBP); and in whom florid reverse MT2–MT1 transformation occurred despite invariable CLBP severity.

Because CLBP severity remained invariant during the MT2–MT1 radiological transformation in both our cases, MT2 lesions were therefore associated with as much pain as with MT1 lesions: in other words, they were “equipotent” (our first conclusion).

Modic et al.7 stated that MT2 lesions were “stable” lesions that did not change with time. In contrast, MT1 lesions were frequently “unstable,” transforming over time into either MT2 or MT0 lesions.8 Quite clearly, both our cases contradicted this view: MT2 lesions were evidently unstable over time (our second conclusion).

Some of the difficulty may have related to an apparent confusion over the context used for the word “stable.” For example, in the second paragraph of his letter (in which Dr. Pinheiro-Franco appears to contradict himself by appearing to accept our second conclusion), Dr. Pinheiro-Franco conjectures that “the conversion from MT2 to MT1 would be explained by the movement that occurred from an initial more stable MT2.” As we made abundantly clear in our original article, however, the use of the word “stable” related—as in Modic’s original article—to invariance over time: it did not refer to mechanical “microstability.”

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References