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LETTER TO THE EDITOR

Modic changes: "*Age, si quid agis*"

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I read with interest the article "Modic changes and their associations with clinical findings" from Kjaer et al. [3]. The authors stated that that the aim of their study was not to specifically identify the clinical profile of people with Modic changes (MCs), but mainly to investigate if MC is a separate entity, one that would deserve to be treated as a potential diagnostic subgroup with specific clinical consequences.

They looked at the pattern of associations between the clinical picture and MRI findings of MC and/or disc degeneration (DD) in three subgroups, of descending "severity", described as follows: the first consisted of all people with both DD and MC; the second consisted of people with DD but no MC; the third consisted of people with neither DD nor MC. They studied several variables related to clinical history and clinical examination. In their population-based sample of 412 40-year-old Danes, they found that MC had a specific clinical profile.

I found very interesting in the proposal of Dr. Kjaer et al. concerning their assumption that "the aim of the study was not to specifically identify the clinical profile of people with Modic changes (MC) but mainly to investigate if MC is a separate entity." Their goal was achieved.

Notwithstanding, the authors did not mention at any instance on their article that more and more evidence is pointing out that Type-1 MC and Type-2 MC seem to correspond to distinct clinical histopathological pictures. In the light of the actuarial knowledge, the readers of this article must keep in mind that Type-1 MC may correspond to a inflammatory, hypervascular state, where some kind of "microinstability" would happen [7, 9]. This supposed "unstable" state may be the sign of an active degenerative state [7, 9]. Modic 2 may define a more stable state, with fatty replacement, a continuum in the degeneration process [1, 7, 9, 11].

This possible distinction between Type-1 and Type-2 MC was expressed in articles demonstrating the longitudinal "evolution" from Type-1 to Type-2 MC, also in papers demonstrating the eventual association between Type-1 MC and low back pain (LBP); and finally in surgical series presenting a possible "acceleration of the natural history" of Type-1 MC after arthrodesis.

The conversion from a theoretically unstable Modic 1 to a more stable Modic 2 has been described by some authors [1, 6, 7, 11]. Modic et al. [7], in their first description of MC, performed a longitudinal study and had five out of six Type-1 MC patients presenting at least partial conversion to Type-2 MC in 14 months to 3 years. Mitra et al. [6] studied sequential MRIs from patients with Modic 1 changes and had a complete (37.5%) or partial (14.5%) transformation to Modic 2 with time or a keeping in Modic 1 (48%), but never spontaneous regression.

Some authors affirmed that Type-1 MC may be painful. Toyone et al. [10] found an association between type I change and LBP (73% Type-1 MC and 11.5% Type-2 MC had significant LBP). Kuisma et al. [4] stated that MCs at L5-S1 and Modic type I lesions are more likely to be associated with pain symptoms than other types of MCs or changes located at other lumbar levels. Kjaer et al. [2], in a previous study, analyzed all types of MCs in relation to LBP variables. They found that Type-1 MC increased the associations in relation to LBP during the last month and year (OR 1.9–2.2 and 4.2–5.7, respectively). They finally

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stated that MCs are strongly associated with LBP during the past year.

Malinin and Brown [5] used nonhuman primates to investigate changes in the vertebral bodies adjacent to acutely narrowed intervertebral discs. On inducing acute DD, they caused adjacent bone marrow changes.

Their study can be considered an animal model of accelerated DD. They affirm 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 normal nucleus pulposus as a distributor of weight-bearing forces evenly onto the adjacent vertebral bodies. Thus, when there is modification on 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 authors anticipate that, as a result of healing and stabilization of disc function, the lesions in the vertebral bodies may heal [5]. Pinheiro Franco argued if this experiment was not defining the passage from Modic 1 to Modic 2 [9]. Unfortunately, the experiment was not documented with sequential MRI.

Finally, two surgical series recently published corroborates the data that Type-1 MC and Type-2 MC may be distinct clinicohistopathological entities and therefore may be studied separately [1, 11].

Vital et al. [11] performed a prospective study on the outcome of the Type-1 MC in 17 patients with chronic LBP 6 months after instrumented posterior lumbar arthrodesis. Disc disease had occurred subsequently to discectomy ($n = 7$), rapidly destructive disc disease ($n = 5$), or spondylolisthesis resulting from spondylolysis ($n = 5$). At the 6-month postoperative examination, all of the patients were clinically improved, and Modic Type 1 evolved in 13 patients to Type-2 MC and in four to no Modic sign.

Esposito et al. [1] did a prospective study on a cohort of 60 consecutive patients with chronic discogenic LBP for more than 6 months refractory to conservative treatment. The individuals constituted a clinically homogeneus cohort. All the patients (30 males, 30 females) were severely disabled (VAS ≥ 6; JOA ≤ 10), with advanced DD: grades 3–5 according to Pfirrmann et al.'s classification [8]. When there were two or more discs with degenerative MRI appearance, discography was performed to assess the painful level. Patients underwent a posterior one-level instrumented arthrodesis and posterolateral autograft ($n = 38$) or interbody fusion with carbon–fiber composite (PEEK) cages filled with bone grafting ($n = 22$). There were 15 patients with Modic Type 0, 22 patients with Modic Type I, 14 patients with Modic Type II, and 9 patients with Modic Type I/II (defined by us a transitory state). For the statistical analysis, the Wilcoxon paired test was used to assess significance in pre- and post-operative differences in term of pain and functional status, with the level of significance set at $P < 0.05$. Patients harboring Modic Type I changes improved much better than others ($P < 0.01$), with good/excellent results in 72.7%. For Modic Type II, results were poor, with 14.3% good/excellent results and no significant difference between pre-/post-operative status. For the Modic Type I/II, clinical outcome was comparable to those presenting with pure Modic Type I changes ($P < 0.01$). In the Modic Type 0 group, patients were also significantly improved for both VAS and JOA scores, but in a lower proportion rather than Modic Type I and I/II groups ($P = 0.0395$) [3].

These authors concluded that combination of LBP of discal origin and severe DDD with Modic Type 1 lesions on MRI may lead to excellent results after fusion in a large proportion of patients. Conversely, arthrodesis for patients harboring Modic Type II changes implicates smaller benefit of doubtful clinical significance [1]. Kjaer et al. surmise that DD, per se, is a fairly quiet disorder, but it constitutes a true clinical entity when MC is present.

I thank the authors for their very important and interesting paper, emphasizing the need to consider MC in our clinical thinking. However, we must remind that when talking and writing on MCs, distinct Type-1 and Type-2 study and considerations are imperative, as from the ancient latin "*Age, si quid agis*" (Plautus, Miles Gloriosus, 215), which means: if you do something, do it (Type-1 MC and Type-2 MC must be analyzed separately).

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