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SCIENTIFIC ARTICLE

Relationship of Modic type 1 change with disc degeneration: a prospective MRI study

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Abstract
Objective The objective was to study the natural course of Modic type 1 change (M1) in relation to lumbar disc degeneration.
Materials and methods Twenty-four chronic low back pain (LBP) patients with M1 on lumbar spine were selected from 1,015 patients with magnetic resonance imaging from a follow-up study lasting for 18–74 months. Exclusion criteria were any other specific back disorder, age ≥60 years, or a recent spine operation. The association between the development of M1 and degenerative disc changes was studied using multivariate modeling (complex samples logistic regression).
Results At baseline, 20 of 28 (71%) disc spaces with M1 had a decreased disc height (DH) and 16 of 28 (57%) a dark nucleus pulposus, but ten of 28 (36%) a very dark annulus fibrosus and a paradoxically bright nucleus pulposus albeit decreased DH. During follow-up, DH decreased in 13 of 28 (46%) and signal intensity of nucleus pulposus (DSI) in eight of 28 (29%) disc spaces with M1, but it increased in four (14%) discs. In those without M1, only few changes occurred. The larger the M1, the more likely was the DH low or decreased further. Both the presence and changes in M1 were associated with a decrease in DH and changes in DSI and bulges.
Conclusion The degenerative process in discs with adjacent M1 seems to be accelerated and leads to advanced and deforming changes with special morphologic features. M1 may be a sign of a pathologic degenerative process in the discovertebral unit.

Keywords Lumbar spine · Low back pain · Intervertebral disk · Follow-up studies · Magnetic resonance imaging

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Introduction

Modic-type signal intensity (SI) changes in the subchondral bone marrow adjacent to end plates of degenerated intervertebral discs may play an important role in chronic non-specific low back pain (LBP) syndrome [1]. Modic type 1 change (M1) associated with subchondral edema has been found to change to Modic type 2 (M2) associated with fatty degeneration [2]. Modic type 3 (M3) is associated with sclerotic changes.

Modic changes are related to degenerative disc changes [2, 3], but the detailed knowledge of the development is sparse. An association between M1 and bony end plate changes has been found in a follow-up study [4]. Patients with disc herniation or sciatica have been included in many follow-up studies on Modic changes. For a better understanding of the morphologic alterations and the mecha-

nisms of pain production, a more accurate stratification of patient cohorts has been suggested [5, 6]. Therefore, we chose to select patients without disc herniation or other specific back disease for this study. The aim was to evaluate the progress of the degenerative disc changes in relation to the Modic type 1 changes in the lumbar spine.

Materials and methods

The subjects were selected from 1,015 consecutive LBP patients referred for standard lumbar spine magnetic resonance imaging (MRI) from the departments of orthopedics or rehabilitation at a university hospital. Patients with chronic nonspecific LBP for at least 3-month duration and a clearly detectable M1 or a mixed lesion (M1/M2 or M1/M3) covering at least 5% (most >15%) of the area of the vertebra in a sagittal MR image were included. Patients with age ≥60 years, a specific back disease, e.g., fracture, neoplasia, spondylolisthesis (5 mm or more), spinal stenosis, disc extrusion or any other finding with neural compression, an infectious or rheumatic spine disease, or a recent (less than 6 months ago) or a major spine operation at any time were excluded. The mean age of the remaining 24 patients (16 females, eight males) was 43 years (range 28–60 years). Three had had an earlier operation 3 to 7 years prior to the MRI, one laminectomy, and two extirpations of a herniated disc. All patients gave a written informed consent to use their clinical data for the study purposes. The study protocol was approved at the research ethics review committee of the university hospital.

The baseline MRI studies were performed with four high field MRI units using T1- and T2-weighted, conventionally used turbo spin-echo (TSE) and fast spin-echo sequences. Two Gyroscan units (1.0 T), one Sonata (1.5 T), and one Signa (1.5 T) unit were used. The follow-up MRI (sagittal images) was performed with the 1.0-T Gyroscan unit and TSE sequences with an average interval of 42 (18–74) months. The MRI signs used in the analysis were modified from the classification criteria used in previous studies on disc degeneration [2, 7, 8]. The variables were tested in a pilot study, and the feasible ones were chosen based on their prevalences and repeatability. Comparative variables were created to assess the change in M1, bony end plate lesions, disc height, signal intensity, and bulges between baseline and follow-up images.

An experienced radiologist (KL) evaluated all images (hard copies) without knowing the symptoms, age, or other background data of the patients or the timing (baseline/follow-up) of the MR examinations. Twenty images were evaluated twice by one radiologist (KL) and once by another (LK) to estimate the intra- and inter-observer agreements. The maximal area (as a percentage of the sagittal area of the vertebral body) and location of each subchondral hypo- and hyperintense SI change on T1- and T2-weighted images (T1WI and T2WI) were estimated. Bony end plate lesions (EPL), meaning focal defects and hypointensities or irregularities at the border between the intervertebral disc and vertebral body, were assessed in each end plate (n=240).

Disc height (DH) was visually assessed as normal (higher than or as high as the upper not degenerated disc space), slightly decreased (≤33% lower than the upper disc space), decreased (34–66% lower), or strongly decreased (>66% lower) at the anterior and posterior edge and at the center of the disc space. Signal intensity of the nucleus pulposus (DSI) was estimated as follows: bright (normal), gray (slightly decreased), dark (clearly decreased), and black (annulus fibrosus and/or nucleus pulposus region similar to that of cortical bone). The presence of an annular tear and anterior and posterior bulging or protrusion of the intervertebral disc was estimated.

New comparative variables were created for the final analysis using the results of the blinded reading to assess the constancy of the EPL and anterior and posterior bulging or protrusion as: absent, constant, disappeared, and increased or appeared by comparing the findings of the baseline and follow-up studies.

Then, a consensus reading was performed by two experienced radiologists (KL, TV). M1s and decrease of the DH and signal intensity (DSI) were first evaluated at each disc space (n=120) in the baseline images. The same classification was used as in the blinded reading except for DSI for which the grade “black disc” was substituted with grades “black disc” (very dark annulus fibrosus and nucleus pulposus) and “black/white disc” (very dark annulus fibrosus but bright nucleus pulposus).

The development of the findings was assessed by visually comparing the baseline and follow-up images and classified with new, “comparative” variables. The presence and change in the size of M1 was classified as follows: absent, constant, decreased/disappeared and enlarged or a (new) M1 appeared. Additional M2s and M3s and their development were evaluated accordingly.

The constancy/change of DH during the follow-up was classified as follows: constantly normal, constantly decreased (but unchanged), and further decreased. An estimated height decrease of at least 20% was accepted. The constancy/change of DSI during follow-up was classified as follows: constantly normal, constantly decreased (but unchanged), further decreased, and increased.

Statistical analysis

Kappa or intraclass correlation coefficient was calculated for the degenerative changes at each disc level to estimate

the intra- and inter-observer agreements (Table 1). The associations between subchondral signal abnormalities and degenerative disc changes and end plate lesions at the baseline were first studied by cross-tabling and calculating chi-square tests, Spearman tests, or Fishers exact tests between variables of the blinded reading for all disc spaces and end plates and separately for single disc spaces and end plates. The development of the degenerative changes was studied by cross-tabling the baseline and follow-up findings. The associations between the baseline subcondral signal abnormalities and degenerative variables of the consensus reading were studied accordingly, and so were those between the comparative variables. The results were compared. Variables with acceptable repeatability were chosen for the final analysis.

To study if the presence of or change of the subcondral signal abnormality is associated with changes of degenerative disc findings, logistic regression was performed by taking other possible predictors into account: patient age, gender, imaging interval, and end plate location (upper/lower end plate). The constancy of DH and DSI and the dichotomized outcome variable for constancy of M1 (absent or constant/decreased, disappeared, enlarged or appeared), based on consensus reading, were used as independent variables in the analysis. Constancy of the anterior or posterior bulge and bony EPL, comparative variables created from the findings of the blinded reading, were added in the final model.

A single end plate was regarded as an observational unit (n=240). The patients (n=24) were defined as clusters in the analysis, and so were the disc levels (n=5). SPSS 14.0.1. (SPSS, Chicago, IL, USA) with its complex samples module was used. Values of $P<0.05$ were considered significant.

Results

Baseline

Nineteen of the 24 patients had an M1 at one disc space, five at two disc spaces, adjacent to both end plates in 25 of 29 disc spaces. Twenty-eight of 54 M1s were uniformly hypointense on T1WI, 26 of 54 of mixed type (M1/M2). Uniformly hyperintense subchondral marrow changes (M2) were additionally found adjacent to 15 end plates. The inter- and intra-observer agreement concerning presence and size of M1 were 0.82 and 0.88, respectively. The detailed results concerning the presence and development of Modic changes and bony EPLs are presented elsewhere [4].

The prevalences and repeatability of degenerative changes in the 120 disc spaces are presented in Table 1. DH was clearly or severely decreased in most disc spaces with M1 either adjacent to the upper or lower end plate (Table 2). The larger the adjacent M1, the lower was the disc. Sixteen of 24 (67%) of such disc spaces with decreased DH had an adjacent M2 too. An M3 was found in decreased disc spaces only.

DSI was decreased or severely decreased on T2WI in 16 of 28 (57%) disc spaces with M1 adjacent to the upper end plate (Table 2). In ten of 28 (36%) disc spaces, DSI was increased in nucleus pulposus albeit dark in annulus fibrosus, even though DH was decreased (Figs. 1 and 2a). Such a black/white disc was found in only one disc space without M1. Eight of 11 (72%) black/white discs had an M2; in addition, most of them were small (5–10% of the sagittal area of the vertebra) at baseline. Six of the seven M3s were located adjacent to discs with decreased or black/white DSI. Decreased DH and disc

Table 1 Number and prevalences (%) of advanced degenerative changes in discs and end plates of 120 disc spaces L1/L2-L5/S1 on sagittal MRI-images of 24 LBP patients at baseline and after follow-up

Degenerative sign	Advanced degenerative changes				Agreement	
	Number (N=120)		Prevalence (%)			
	Baseline	Follow-up	Baseline	Follow-*up*	Inter-observer	Intra-observer
Decreased DSI	46	53	38	44	0.72[a]	0.76[a]
Decreased disc height	25	33	21	27	0.91[a]	0.91[a]
AF-tear or HIZ	24	22	9	6	0.56[b]	0.73[b]
Posterior bulge	30	25	25	21	0.48[b]	0.58[b]
Anterior bulge	29	29	24	24	0.45[b]	0.61[b]
Upper end plate lesion	47	59	39	49	0.79[b]	0.89[b]
Lower end plate lesion	48	52	40	43	0.79[b]	0.89[b]

Agreement is calculated for 120 discs and for a total of 240 (upper and lower) end plates
[a] Intraclass correlation (ICC)
[b] Kappa value

Table 2 Grade of disc degeneration (decrease of signal intensity of the nucleus pulposus and height of the disc) at baseline in disc spaces L1/L2-L5/S1 with or without an adjacent M1 lesion in the upper end plate (consensus reading)

Degenerative sign								
Grade of disc degeneration (decrease)	Signal intensity of nucleus pulposus				Disc height			
	No M1		M1		No M1		M1	
	Number	Percentage	Number	Percentage	Number	Percentage	Number	Percentage
No decrease	51	56	1	4	76	83	2	7
Slight decrease	24	26	1	4	12	13	6	22
Decrease	13	14	15	53	2	2	11	39
severe decrease	3	3	1	4	2	2	9	32
Black/white disc	1	1	10	35	–	–	–	–
	92	100	28	100	92	100	28	100

bulges were not common without adjacent M1, but decreased DSI was equally common with or without M1 (Table 2). Twenty-six of 78 (33%) discs with normal DH had decreased DSI, but only four of 54 (7%) discs with normal DSI had decreased DH. The results concerning associations between subchondral signal changes and degenerative disc changes were essentially the same when using variables of the blinded reading and those of consensus reading.

Follow-up

During the 18–74 months follow-up, 14 M1s decreased (Fig. 2) or disappeared, 12 enlarged (Figs. 3 and 4), and five new M1s appeared (Figs. 4 and 5) at the same or other disc spaces. Twenty-two of 54 M1s changed into M2s (Fig. 5). Only six remained stable. The prevalence of decreased DH and DSI increased, but that of posterior bulge and AF-tear or HIZ slightly decreased (Table 1). Twelve posterior and seven anterior bulges disappeared and seven combined bulges appeared. Posterior bulges or protrusions did not increase in disc spaces with a changing, unstable M1.

Degenerative changes progressed differently in disc spaces with or without M1 and in those with stable or unstable M1s. The decreasing M1 seemed to have a similar effect like the increasing M1 on the progress of disc degeneration (Figs. 6 and 7).

DH decreased in 13 of 28 (46%) disc spaces with M1. All those 13 disc spaces had an unstable M1 (Fig. 3), nine (69%) a decreased DH, and eight (62%) a black/white DSI at baseline. In three of the discs with decreasing height, the

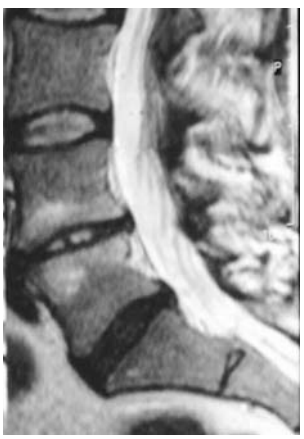

Fig. 1 On T2WI, M1 adjacent to both end plates of disc space L4/L5 with a strong height decrease and an increased signal intensity in the nucleus pulposus of the intervertebral disc. The signal intensity of the L5/S1 disc without M1 is clearly decreased, although its height is only slightly decreased

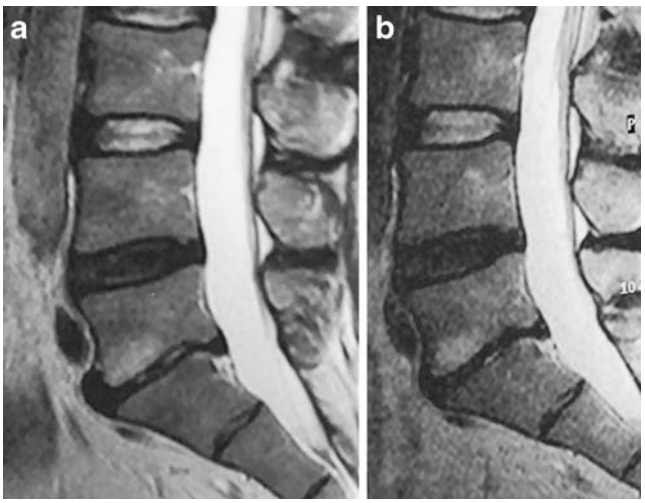

Fig. 2 **a** On T2WI, in a degenerated disc space L5/S1 with anterior extrusion and M1 adjacent to the upper end plate, disc height (*DH*) is decreased but the signal intensity (*SI*) bright in the nucleus pulposus albeit dark in annulus fibrosus (**b**). While M1 decreases and turns to M2, the anterior extrusion decreases, DH and SI of the nucleus pulposus decrease, and small irregularities develop in the bony end plates. In the upper disc space with no M1, the degenerative changes do not show any progress: SI remains decreased but DH remains normal

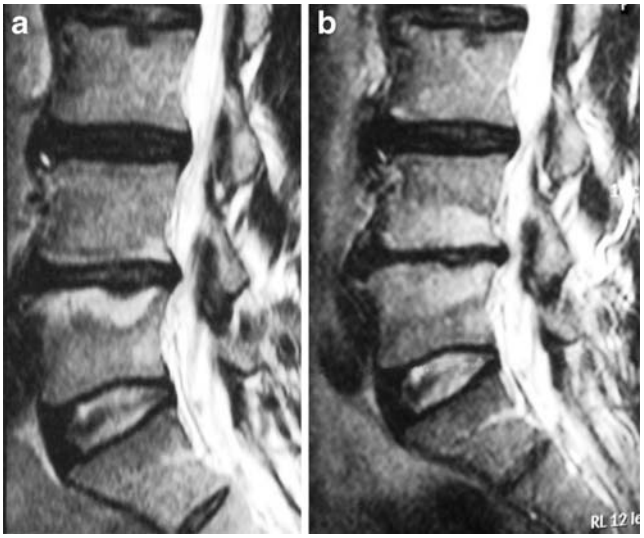

Fig. 3 **a** On T2WI at baseline, M1 adjacent to both end plates of the L4/L5 disc space with a clearly decreased disc height and a higher signal intensity (*SI*) of nucleus pulposus than in the upper, degenerated disc space with decreased DSI and only slightly decreased DH. **b** After follow-up, M1 at L4/L5 disc space enlarges, disc height strongly decreases, and irregularities in the bony end plates increase. Degenerative changes do not appear on L5/S1 disc or progress in the upper discs

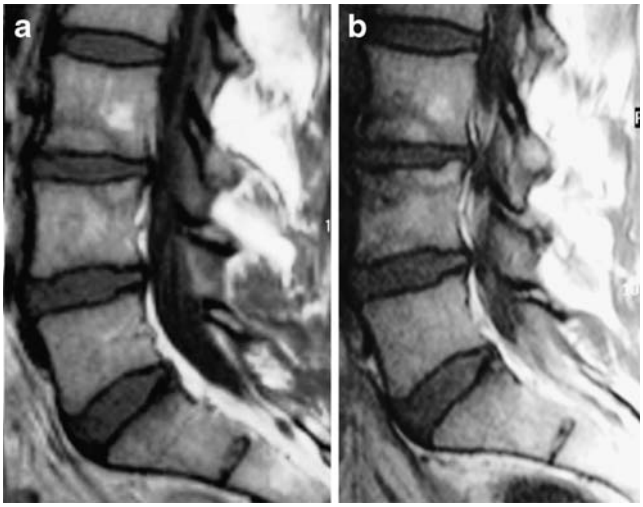

Fig. 5 **a** On T1WI, a bony lesion adjacent to the upper and lower end plate of L3/L4 disc space is surrounded by a hypointensity with a semilunar shape. There is also a slight hypointensity on the upper end plate of L4/L5 disc space, not easily detectable on T1WI. **b** The hypointensities turn to hyperintensities, while disc height slightly decreases. A new bony end plate lesion and a surrounding subchondral hypointensity appear anteriorly adjacent to the lower end plate of L3/L4 disc space. In the other disc spaces, the degenerative changes do not show progress

DSI turned to black/white type. The larger the M1 was, the more likely the DH decreased.

DSI decreased (further; Fig. 2) in eight (29%) or turned to black/white in four (14%) of the 28 discs with an adjacent M1, most often an unstable one. All new black/white discs developed in disc spaces with a decreased DSI and an enlarging or stable M2 (Fig. 4). In eight (73%) of the 11 disc spaces with a black/white disc at baseline, M1 changed into M2 or the preexisting M2 enlarged during the follow-up. In addition, an M3 developed in three of those disc spaces. The increase of DSI was significantly correlated with changes of M1, but the decrease of DSI was not.

In multivariate modeling (complex samples logistic regression), a significant association was found between the instability of M1 and that of the degenerative changes, in particular decrease of DH, appearance of "black/white disc", and appearance or disappearance of anterior bulge (Table 3). The odds ratios varied only slightly according to the applied covariant pattern.

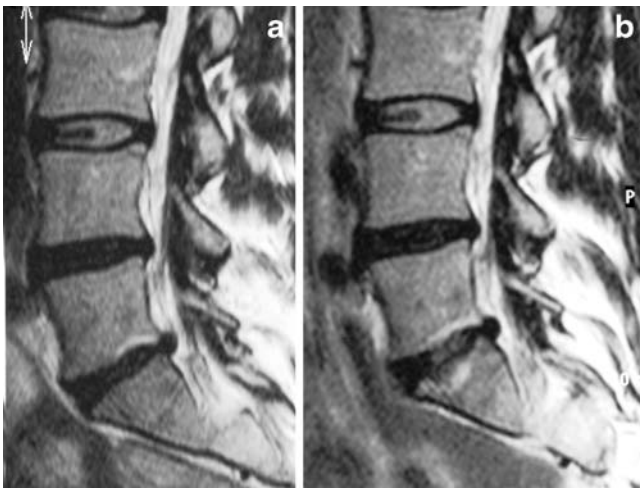

Fig. 4 **a** In a degenerated disc space L5/S1 with decreased disc height and posterior bulge, signal intensity (*SI*) of nucleus pulposus is dark (decreased) on T2WI. **b** During the follow-up, SI of nucleus pulposus becomes bright (increases) and the small M1 lesion adjacent to the upper end plate increases and turns to M2. A new EPL with a surrounding M1/M2 lesion appears adjacent to the lower end plate. In the upper two disc spaces, the degenerative changes do not progress or appear

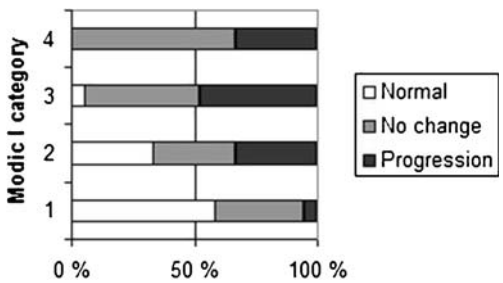

Fig. 6 Constancy of the signal intensity of the nucleus pulposus of the intervertebral disc (constantly normal; constantly decreased; further decreased) in relation to Modic category (1 = no M1 present at any time; 2 = M1 present but remains unchanged; 3 = M1 decreases or disappears; 4 = M1 increases or appears) during 18–74 months follow-up

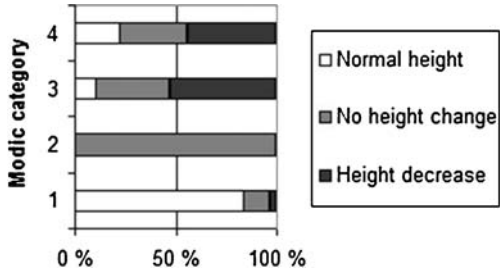

Fig. 7 Constancy of the disc height (constantly normal; constantly decreased, unchanged; further decreased) in relation to Modic category (1 = no M1 present at any time; 2 = M1 present but remains unchanged; 3 = M1 decreases or disappears; 4 = M1 increases or appears) during the 18–74 months follow-up

Discussion

In this study, a relatively rapid progression of degenerative disc changes was found in association with the presence and, in particular, changes in the size and signal intensity of M1s. The degenerative changes were advanced already at baseline in most discs with an adjacent M1, but uncommon in those without. The progressive degenerative changes along with changes in M1 were deforming to the discovertebral unit, as irregularities and even defects developed in the subchondral bone at the end plate border, while DSI changed and DH strongly decreased as a sign of a collapse of the disc.

The weakness of the current study is the small number of patients and the lack of a control group. However, the subjects were selected from a large base population of chronic LBP patients. Only few of them had large M1s. Therefore, the collection of the subjects with the strict inclusion criteria lasted years. Control subjects with the same LBP symptoms and inclusion criteria but without M1 would not have been easy to find in reasonable time. There are follow-up studies in which M1 lesions are not common, and the progress of degenerative disc changes in general population, LBP patients, or asymptomatic subjects can be estimated for comparison.

Although the number of selected patients was small, they had 120 discs and 240 end plates to be analyzed. We decided to use the disc spaces without M1 as internal controls for those with M1 to compensate for the lack of the control subjects. The possible confounding factors and the location of the findings at different disc levels and end plates were taken into account, also the interval between baseline and follow-up study. The possible effect of the varying interval on the results was thus controlled. The effect of the interval on the development of the subchondral signal changes has been presented earlier [4].

Table 3 Effect of changes in disc degeneration and bony end plate lesions on stability of Modic 1 lesion (increased or decreased M1/No M1 or constant size of M1)

Degenerative change	Grade	OR	Confidence limit		*p* value
			95% Lower	95% Upper	
Decrease of SI					0.040
	Normal SI	1.0			
	Constantly decreased SI	9.58	1.87	49.11	
	Decrease of SI	3.79	0.18	78.24	
	Increase of SI	17.69	1.97	158.88	
Bony EPL					0.015
	No EPL	1.0			
	Constant EPL	24.96	3.63	171.77	
	Change of EPL	15.21	1.56	147.93	
	Increase of EPL	12.67	1.84	87.26	
Anterior bulge					0.006
	No bulge	1.0			
	Constant bulge	0.77	0.17	3.52	
	Bulge disappeared	16.45	2.59	104.49	
	New bulge	4.81	1.15	20.10	
Disc height					0.004
	No decrease	1.0			
	Constantly decreased height	3.84	0.70	21.04	
	Decrease of height	58.60	5.40	636.00	

Complex samples logistic regression: model including the anterior disc protrusion. Adjusted for end plate location (superior/inferior), gender and age of the patient and interval between studies
SI signal intensity of the intervertebral disc, *EPL* end plate lesion

The MRI analysis was more detailed than in many earlier studies. Since new variables were used for assessment of change in subchondral signal abnormalities and in DH and DSI, joint reading was performed to increase reliability and to decrease the possibility of false positive findings. We found comparative variables more feasible than scoring the degenerative disc signs and signal intensity and size of M1s or M2s. They were chosen to increase sensitivity to detect small changes and to assure that changes with the same location are compared between baseline and follow-up images, e.g., some M1s were found to be partly or totally isointense at the time of the follow-up MRI and difficult to detect without comparing with baseline images.

The use of different MRI equipments for baseline images may have affected the estimation of signal intensity since T1 is known to be field-dependent. However, all were high field units and the difference in DSI or subchondral signal abnormality between baseline (1.5 or 1.0 T) and follow-up (1.0 T) study was expected to be of minor importance. In the visual assessment, the signal intensity was compared with that of other discs, vertebrae, and surrounding structures in the same image instead of direct comparison between studies. Since standard imaging protocol was used for lumbar spine MRI in all hospitals of the university hospital, imaging parameters did not vary much between patients. The follow-up images were obtained with the same 1.0-T unit and protocol and thus comparable. Since only large findings were observed and morphological changes assessed in addition to signal intensity changes, we do not believe that differences in imaging equipments have significantly affected our results.

The inclusion of a STIR or T2fs sequence may have improved the assessment of end plate edema and change in DSI. However, our subjects were chosen from general LBP patients with the standard lumbar MRI protocol which does not include such sequences. We did not use them in the follow-up MRI either for uniformity and because we wanted to use the original criteria set by Modic for assessing subchondral signal abnormalities.

All images were read on hard copies for uniformity because PACS was not yet used in the whole university hospital at the time our first patients were collected. A more accurate assessment of subchondral signal changes and degenerative disc changes would have been possible on PACS workstations than in visual assessment of the hard copies, as well as quantitative assessment of DH. However, measurement of DH is not reliably repeatable even on workstations [9]. The visual estimation of signal intensity changes was considered more feasible than measurements because different MR units were used. In visual estimation, morphologic changes could be taken into account too and signal intensity of M1 lesion or disc compared with that of adjacent discs and other surrounding structures.

An association of M1 with disc herniation has been found earlier [10, 11]. We found an association with anterior bulges. Desiccation and collapse of the disc as result of the relatively rapidly progressing degeneration along with changes in M1 may explain why the bulges were found to decrease rather than increase.

The association of M1 with an increase of DSI has not been presented earlier. Degenerative subchondral changes have been found adjacent to collapsed discs [8]. The very bright signal in the nucleus pulposus of discs with advanced degeneration may be a sign of destruction in a collapsed disc in which fluid-filled fissures and dense collageneous fibers have replaced the normal fibrocartilaginous structure [8, 12]. The fissures may become prominent during MR imaging in the recumbent position because the intradiscal pressure is diminished and the disc height slightly increased [7, 12]. A transient phase with changed water binding capacities of the nucleus pulposus tissue could also explain the high DSI in a collapsing disc.

The appearance of a new M1 adjacent to the lower end plate after the baseline lesion adjacent to the upper end plate (Fig. 4) may suggest a uniform process spreading to the whole discovertebral unit. The radiological appearance and development of M1 resembles that of spondylodiscitis and spondylarthropathies. Fibrovascular tissue found in vertebrae with M1s suggests an inflammatory process [2]. Inflammatory mediators have been found in end plates with M1s [13]. It has been proposed that they could lead to a progressing inflammatory process in the end plates as well as in the subchondral bone [6, 14, 15]. Whether the finding of an increased SI in some discs with an associated M1 could be a sign of an inflammatory process in disc as well as in the subchondral bone or end plate remains to be studied.

In recent studies, EPLs have been shown to affect diffusion through the end plate and thus to be critical for the nutrition of the disc [16, 17]. Endplate damage correlated with disc degeneration and was considered a crucial event leading to structural failure precipitating to degenerative disc disease [17]. Bony end plate lesions have been found to change along with subchondral signal abnormalities [4]. In this follow-up study, degenerative disc changes were found to change relatively rapidly in disc spaces with M1, like EPLs, during the follow-up of 18–74 months. On the other hand, the incidence of new degenerative findings (decreased DH and DSI) or even progress of those findings in disc spaces without M1 was low. Low incidence of degenerative findings was also reported in a 5-year follow-up study [18] and in asymptomatic subjects followed during 3 years [19]. This may indicate that in most discs, the degenerative process progresses slowly. However, our

results suggest that in certain discs with distinctive features, the progress is relatively fast. Degenerative disc changes and EPLs seem to develop in pace with adjacent M1s. M1 may indicate a progressing, relatively fast process harmful to the integrity of the intervertebral disc.

It has been suggested that subjects with LBP and MODIC changes have a specific type of LBP [1]. We found distinctive morphologic features in degenerated discs associated with M1. They may represent pathological disc degeneration, possibly with inflammatory changes. Whether those findings could explain the variation in LBP symptoms will require further analysis in a prospective setting.

Decrease of DSI has been found to precede that of DH [20]. In our study, the different degenerative disc findings did not develop at the same pace. Our results do not support the view that degenerative disc findings, at least those associated with M1, linearly increase. Variables with a combination of different degenerative signs for classifying disc degeneration may need to be used with caution.

Conclusions

Special morphologic features are found in intervertebral discs and end plates in the disc spaces with Modic type 1 change. Relatively rapid changes occur in subchondral signal abnormalities in line with progressive deforming degenerative changes in discs and bony end plates. Modic type 1 change may be a sign of a distinctive degenerative process in the discovertebral unit.

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