The Disc-Endplate-Bone-Marrow Complex classification: Progress in our understanding of Modic vertebral endplate changes and their clinical relevance

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Abstract

Background context: The disc, endplate (EP), and bone marrow region of the spine form a single anatomical and functional interdependent unit; isolated degeneration of any one structure is rare. Modic changes (MC), however, are restricted to the subchondral bone alone and based on only T1 and T2 sequences of MRI. This results in poor reliability in differentiating fat from edema and hence may give a false impression of disease inactivity.

Purpose: To study the changes in disc, endplate, and bone marrow as a whole in degeneration and propose a classification based on the activity status of this complex with the addition of STIR MRI sequences.

Study design: Observational cohort

Patient sample: Patients with isolated brain, cervical, or thoracic spine injury and patients with LBP who underwent MRI formed the control and study groups, respectively.

Outcome measures: Demographic data, the prevalence of MC and disc-endplate-bone marrow classification (DEBC) changes, EPs undergoing reclassification based on DEBC, and comparison of the prevalence of MC, DEBC, H+ modifier and DEBC with H+ concordance between control and LBP group. The study determined the risk of LBP patients undergoing surgery as well as the incidence of postoperative infection based on DEBC changes. Significance was calculated by binomial test and Chi-square test with the effect size of 0.3-0.5. Prevalence and association of outcome were calculated by Altman's odds ratio with the 95% CI and the scoring of z statistics. Logistic expression was plotted for independent variables associated with each class of both Modic and DEBC against dependent variables surgery and non-surgery.

Methods: Lumbar segments in both groups were assessed for MC types. The DEBC classification was developed with the addition of STIR images and studying the interdependent complex as a whole: Type-A: Acute inflammation; Type-B: Chronic Persistence; Type-C: Latent and Type-D: Inactive. Modifier H+ was added if there was disc herniation. The classification was compared to MC and correlated to clinical outcomes.

Results: 3560 EPs of 445 controls and 8680 EPs in 1085 patients with LBP were assessed. 4 non-MC, 560 MC-II, and 22 MC-III EPs were found to have previously undetected edema in STIR (n=542) or hyperintensity in discs (n=44) needing reclassification. The formerly undescribed Type-B of DEBC, representing a chronic persistent activity state was the most common (51.8%) type. The difference between the control and LBP of H+

(12%vs28.8%) and its co-occurrence with DEBC type 1.1%vs23.3%) was significant (p<0.0001). The odds ratio for the need for surgery was highest (OR=5.2) when H+ and DEBC type change co-occurred. Postoperative deep infection (as determined by CDC criteria) was 0.47% in non-DEBC, compared to 2.4% in patients with DEBC (p=0.002), with maximum occurrence in Type-B.

Conclusion: Classification based on the classic MC was found to need a reclassification in 586 EPs showing the shortcomings of results of previous studies. Considering the DEBC allowed better classification and better predictability for the need for surgical intervention and incidence of postoperative infection rate than MC.

Keywords: Endplate changes, Modic changes, STIR, Disc-Endplate-Bone-Marrow Complex, Disc hyperintensity

Introduction

Modic changes (MC) are discussed extensively in literature from various angles of being a separate clinical phenotype, having a controversial etiology with the possibility of subclinical infection and treatment with antibiotics, having poor outcomes and higher complication rates after surgery[1],[2],[3],[4],[5]. The currently utilized classification, originally described by Roos [6], but more commonly called Modic Changes[7],[8], represents three clinical states: edema and active inflammation, fatty conversion and healing, and sclerosis. The classification is based on differentiating fat and edema but is based on traditional T1 and T2 MRI sequences. This is fundamentally flawed as the advent of Short Tau Inversion Recovery (STIR) has shown that more than 70% of lesions diagnosed purely as fat can be edema or a mixture of both[9]. Further, MC evaluates changes in the bone marrow of the subchondral region alone and this may be very restrictive as it ignores the other changes of the end plate region which includes the cartilaginous and bony end plate, and the peripheral nucleus pulposus, all of which are interdependent in their anatomical integrity and physiological function: a change in one can cause a change in the other [10]. With the current prominence and increased attention of the spine community on the end plate changes, it becomes important that we move forward and beyond the Modic change for accurate understanding. Considering the entire 'disc-endplate-bone marrow complex, or DEBC' as a whole and using STIR sequence to differentiate fat, sclerosis, and edema may be important. The cartilaginous endplate supported by a bony end plate is responsible for anatomical integrity and controls diffusion, the only source of nutrition to the disc[11], [12]. A break in the end plate establishes disc-bone marrow contact leading to possible severe auto-immune inflammation and also neo-vascularization and destruction of the disc[13]. Conversely,

degeneration, herniation, or infection of the disc will end in the destruction of both end plates as well as involve the subchondral bone. It is then logical that this region must be considered together as a whole. To consider any one of them in isolation can lead to the error of overlooking changes in the other structures of the DEBC. The present study has evaluated changes in the DEBC as a whole in controls and patients with low back pain and has evolved a classification.

Materials and Methods

A retrospective, Institutional Review Board (IRB) approved study was performed to examine EP changes and correlate them to clinical outcomes. 445 consecutive patients who underwent whole spine MRI for isolated brain, cervical, or thoracic spine injury without a neurological deficit formed the control group. The MRI of these patients was taken immediately following their presentation to the hospital and before any surgery. 1085 consecutive patients presenting with low back pain (LBP) for a minimum duration of 3 weeks to the outpatient clinic undergoing MRI formed the study group. Patient demographics, clinical presentation, and outcomes up to six months were recorded. All the patients belonged to the south-Indian state of Tamil Nadu. The mean height, weight, and BMI were 167 cm, 69 Kg, and 24, respectively. Patients with instability, a history of trauma to the lumbar spine, tumor, infection, spondyloarthropathies, deformity, or previous surgery were excluded. All radiological images were retrieved from the hospital Picture Archiving and Communication System (PACS).

The lumbar segments including L2-L3 discs to the L5-S1 discs and adjacent EPs in the control and LBP groups were assessed for MC types using a 3T (Siemens, Germany), Magnetom Lumina platform using fast spin-echo. The same segments were also assessed with T1, T2, and STIR images. Based on this, the DEBC changes were classified into four types by 2 senior spine surgeons and senior radiologists, shown in Table 1 and Figure 1. Based on the image findings, these 4 types were found to represent four states or evolutionary stages of infection-/inflammation.

Type A shows T1 hypo, T2 hyper, and STIR hyperintensity signal change and represents an ongoing ‘acute active inflammatory process. Disc hyperintensity may or may not be present and the EP is often indistinct with possible erosions (Fig. 1a-c). **Type B** shows edema in STIR interspersed with either fat or sclerosis and indicates a state of 'chronic persistent inflammation. Disc hyperintensity may or may not be present and the EP shows erosions (Fig. 1d–f). **Type C** shows hyperintensity in both T1 and T2 and hypointensity in STIR and indicates a state of ‘latency’ with no element of acute activity. There is no hyperintensity in discs or EP erosions (Fig. 1 g–i). **Type D** shows hypointense signals on all three sequences and indicates a state of inactivity. There is no hyperintensity in discs or EP erosions. (Fig. 1j – l)

Based on the presence of a disc herniation, an H+ modifier was added. These discs were further classified as concordant if the DEBC change and H+ were on the same side, discordant (Fig. 2). Discs showing hyperintensity (Fig. 3) and EP erosions (Fig. 4) were identified. Patients who required reclassification to Type A or B of DEBC based on edema in STIR or disc hyperintensity were documented (Fig. 5). The influence of DEBC change in the presence and absence of H+ modifier was evaluated to determine the need for surgery and surgical outcome including post-operative infection.

Significance was calculated by binomial test and Chi-square test with the effect size of 0.3-0.5. Prevalence and association of outcome were calculated by Altman's Odds ratio with 95 % CI and the scoring of z statistics. Logistic expression was plotted for independent variables associated with each class of both Modic and DEBC type against dependent variables surgery and non-surgery.

Results

3560 EPs in 1780 discs of 445 patients in controls were compared with 8680 EPs of 4340 discs in 1085 patients in the LBP group. The mean age was 45.96 (42.8 years in control versus 47.25 in the LBP). Males formed 63% of the population (77.5% of controls and 57.1% of LBP patients).

Assessment of MC: The incidence of MC in the control group was 8.8% and 41.3% in the LBP group (p=<0.00001) (Table 2). Of the controls with MCs, the distribution was Type 1 - 5%, Type 2 - 95%, and none in Modic Type 3, while in the LBP group, it was Type 1-8.36%, Type 2-87.14%, and Type 3-4.5% (Table 3). The distribution of Modic change was higher in the lower two lumbar levels in the LBP group (82.4%) compared to the controls (52%) (p=0.00001).

Assessment of DEBC changes: The intra-observer and inter-observer variability correlation for classifying DEBC changes measured by Kappa was 0.84 except for the presence of disc hyperintensity, which was 0.71. Of the controls with DEBC changes, the distribution was Type A-5.4%, Type B-36.6%, Type C-58.0%, and none in Type D, while in the LBP group, the distribution was Type A-8.6%; Type B-51.8%; Type C-37%; Type D-2.6% (Table 3). The subtype previously undescribed in Modic, 'chronic persistence' (DEBC Type B), was found to be the most common accounting for 51.8%.

Classic Modic description based on T1 and T2 alone had misclassified 586 EPs (Table 4). 560 type 2 MC and 22 type 3 MC were reclassified to Type B DEBC, while 4 EPs were classified from non-modic to Type A DEBC, because of the presence of edema in STIR images. The distribution of edema was paradiscal in 422 (77%), perilesional in 80(14.65%), or mixed in 44(8.05%) in LBP (Fig 6). Disc hyperintensity was found in 18 (4%) controls and 224 (20.64%) patients in the LBP group(p=0.000096). The presence of disc hyperintensity

was responsible for the re-classification of 4.1% (40) of type 2 MC to type B. End-plate erosions were found in only 111(3%) controls compared to 752 (8.6%) of all LBP (p=0.000087) and to a variable extent in every patient with DEBC.

In the 445 controls, H+ alone was present in 53 (12%), DEBC alone in 34 (7.6%), and both together in 5(1.12%). This is significantly different from the 1085 LBP group, where H+ alone was present in 313(28.8%)(p=0.00001), DEBC alone in 448(41.3%)(p=0.00001), and both together in 253(23.3%)(p=0.00001). The incidence of concordance of H+ and DEBC change was found in 80% of controls and 85.7% of LBP patients (p=0.08) implying that endplate changes and herniation may have a common etiopathogenesis.

The patients were managed based on the institute's protocol. All patients initially underwent conservative management in the form of NSAIDs, neuropathic medications, and physiotherapy for a minimum period of 8 weeks. The decision to operate was taken based on clinical grounds. In the LBP group without DEBC changes, 557 (87.44%) underwent non-surgical treatment, and surgery (both fusion and nonfusion) was required only in 80 (12.5%) this is significantly different in the DEBC group where surgery was required in 122(27.2%)patients(p=0.00001) (Table 5). The odds of the patient requiring surgery was 0.66 in pure herniations without DEBC, compared to 2.6 with only DEBC, and the highest of 5.2 in patients having both DEBC and H+ (Table 6). The predictability threshold of patients going into surgery for the DEBC classification was found to be 1.2-fold higher than the Modic classification.

The patients were followed at 3 weeks, 6 weeks, 3 months, and 6 months following initiation of treatment in the outpatient by one of the members of the spine team, belonging to the cadre of Senior Registrar and above. There was a total of 14 infections of which 9 were deep infections (4.4%). The incidence of deep infection (diagnosed as per CDC criteria) in the non-DEBC group was 2 (2.5%) compared to 7 (5.7%) in the DEBC group ($p<0.05$) [28]. None of the patients in Type A and D had infection compared to 4(57%) in Type B; and 3(43%) in Type C. Patients with DEBC had a higher rate of infection than those without, with an odds ratio of 5.3.

Discussion:

Although Modic has occupied the center stage of discussion in current literature, it has the disadvantage of utilizing only T1 and T2 for evaluation of fat and edema and also being restrictive to the changes of bone marrow in the subchondral bone. This has probably led to considerable misclassification of the EP status and to a faulted concept that active inflammation was restricted to Type 1 only. This unfortunately focused many clinical, microbiological, and therapeutic investigations on mainly Type 1 disc and could also be the cause of indeterminate and controversial results of various studies.[14],[15],[16],[17]

The most important contribution of this work is the recognition of the DEBC region as a separate anatomical-functional entity and evolving a classification based on the changes of the whole region. The disc, cartilaginous and bony endplate, and subchondral bone are not just neighborhood organs but function together for structural integrity and physiological function. An alteration in one will lead to an alteration in the other. Our results showed that focusing only on the subchondral bone can lead to misclassification in up to 51.8% of the patients.

Assessment of DEBC Changes.

Our study documented a variety of concomitant changes in DEBC which can occur in isolation but more often together. It is not in the scope of this paper to discuss the etiology of these changes and the controversies that exist on whether the inflammation is due to traumatic, inflammatory, or subclinical infection. However, the findings stand as proof of the need for STIR and the evaluation of the DEBC as a whole. STIR identified edema in 560 EPs, considered to be pure fat, and in 22 EPs considered as pure sclerosis and helped to avoid misclassification (Figure 5). Surprisingly STIR identified edema in 4 of the non-MC patients also. Overall, STIR was responsible for diagnosing 58% of endplates correctly to chronic persistent type (Type B) which were previously considered as healing or healed. The interobserver variability in our study is higher for most parameters, except for disc hyperintensity, as compared to the study by Sherwood et al [18] where 20 MRIs were independently reviewed by two neuroradiologists and two spine physiatrists. They reported an overall inter-observer reliability kappa value of 0.5899. Literature abounds with research correlating the type of Modic change to epidemiological studies [19], clinical presentation [1], prospective trials of antibiotic therapy [14], presence of bacteria in the disc [20], presence of inflammatory markers [21],[22] and clinical outcomes [3], [2] without consensus. The high degree of re-classification required when STIR and disc hyperintensity were used questions the validity and conclusion of many of the studies published so far utilizing Modic classification. The contradictory results and controversies could have all along been due to this fundamental flaw of misclassification by MC.

Chronic persistence (Type B) and Latency (Type C)

Chronic persistence with frequent exacerbation is a well-known entity in many inflammatory/infective conditions affecting other bones in the body [23] and was found to be the same for EPs in our study. We defined chronic persistence as any EP having edema in association with fat or sclerosis both of which are found only with chronicity. This group which was most common forming 58% was completely ignored in the classical description by Modic and was wrongly considered healing or healed (Fig 7). The randomized trials of antibiotic

therapy and many studies probing subclinical infection and antibiotic therapy were focused on Type 1 alone, as Type 2 and 3 were thought to be healing or healed[4],[15],[14]. Our study has shown that while pure Type A was only 8.6%, Type B showing chronic inflammation was the most common with 58% incidence.

The distribution of edema was also found to be important. Among the four types of distribution identified (Fig 6), the paradiscal type and patchy type were found to be the most significantly associated with postoperative infections.

The description of pure fatty infiltration was considered as healing in MC but we have preferred to term it as latent (Type C). Latency, in biology, is defined as lying dormant or hidden until circumstances are suitable for development or manifestation [24]. Type C suits this aptly as although there is a radiological image of healing, there is a potency for reactivation both clinically and radiologically. Literature shows the possibility of conversion of Type 2 Modic to Type 1 and vice versa showing that fatty infiltration of marrow is not a complete sign of healing[25]. Clinically, type C patients had frequent exacerbations, and the requirement of surgery and the incidence of infection, although lower than type B, was still significant.

The importance of H+

The special feature of DEBC classification is the addition of H+ with the presence of a disc herniation. This is important because, in clinical practice, the presence of a herniation largely determines the nature of treatment and the need for surgery. There have been reports on the outcomes of Modic changes or herniation but very few reports have concentrated on the influence of Modic change on herniation and the co-existence of both on the outcome. Our study showed clearly the clinical importance of their co-existence. H+ modifier was present in 313 discs, DEBC was present in 448, and both together in 253 Cases. Concordant presence with both the herniation and Modic change on the same side was seen in 217(85.8%) in the LBP group and 4 (80%) in the control group. While it is difficult to comment on the influence of each on the etiopathology of the other or which is the primary pathology, it is obvious that they share a common occurrence and influence the clinical outcome. The probability of requiring surgery was highest when both herniation and DEBC were present. The odds ratio for requiring surgery with only herniation was 0.66; was 2.6 with only DEBC and 5.2 when both were present. The negative influence extended to the post-operative outcome with a rate of infection being 5.7% in DEBC compared to only 2.5 % in DEBC negative (p=0.02)

Erosions and Disc Hyperintensity

Erosions of the bony EP and the presence of disc hyperintensity are strong radiological indicators of inflammation/infection and we included them in DEBC. Overlooking these two important radiological entities is a major handicap of the classical Modic change. In our series, disc hyperintensity was present in 18 (4%) controls and 224 (20.64%) patients in the LBP group (p=0.000096). Disc intensity is a classical sign of early infection and also spondyloarthritis and it accounted for re-classification to chronic persistence (Type B) in 44. Of the 448 DEBC-positive patients, a typical erosive pattern of the EP was present in 66% of patients (Figure 4). A recently published study has analyzed the patterns of erosion by multimodal imaging and has elaborated on the similarity of erosions of MC with that of healing infection[26] (Fig 7). Although that study concentrated on CT analysis of MC, we were able to identify significant erosive patterns in plain MRI in 752 EPs. The odds of a patient requiring surgery when the patient had significant erosion was 3.6. A study by Baker et al[27] reported significantly higher levels of post-operative disability following anterior cervical discectomy and fusion in patients who had Modic-endplate complex changes which provided valuable aid to prognostic assessment of degenerative cervical patients. However, the study did not consider the changes in the disc which forms an integral part of the functional unit of the spine, nor was STIR included in the evaluation which has led to the reclassification of several endplates that were previously thought to be in a healing phase.

Limitations

Although this present study involved the assessment of a total number of 12,240 endplates, it is still a single-center study and will require multicenter validation. The radiological signs had good intra-observer correlation amongst both clinicians and radiologists in our center but will have to be validated in future studies from other centers. The lack of biochemical result correlation and a prospective correlation with clinical findings and outcomes will need to be addressed in future studies.

Conclusion

The DEBC classification is a progressive shift in our understanding of the so-called Modic EP changes by considering the DEBC Complex as a whole. By the usage of STIR and the addition of disc intensity and EP erosions, 3.4% of patients in controls and 21.75% in the LBP group were reclassified to a previously undescribed 'chronic persistence' (Type B of DEBC) group which had the highest incidence and influence for the need for surgery and infection. The addition of an H+ modifier was found to be clinically important as the

co-existence of DEBC and H+ influenced treatment outcomes and the requirement for surgery and surgical infections. We believe that DEBC classification is an important advance in our understanding of the vertebral endplate changes over the classical MC and will have a significant role in both clinical practice and research in disc diseases.

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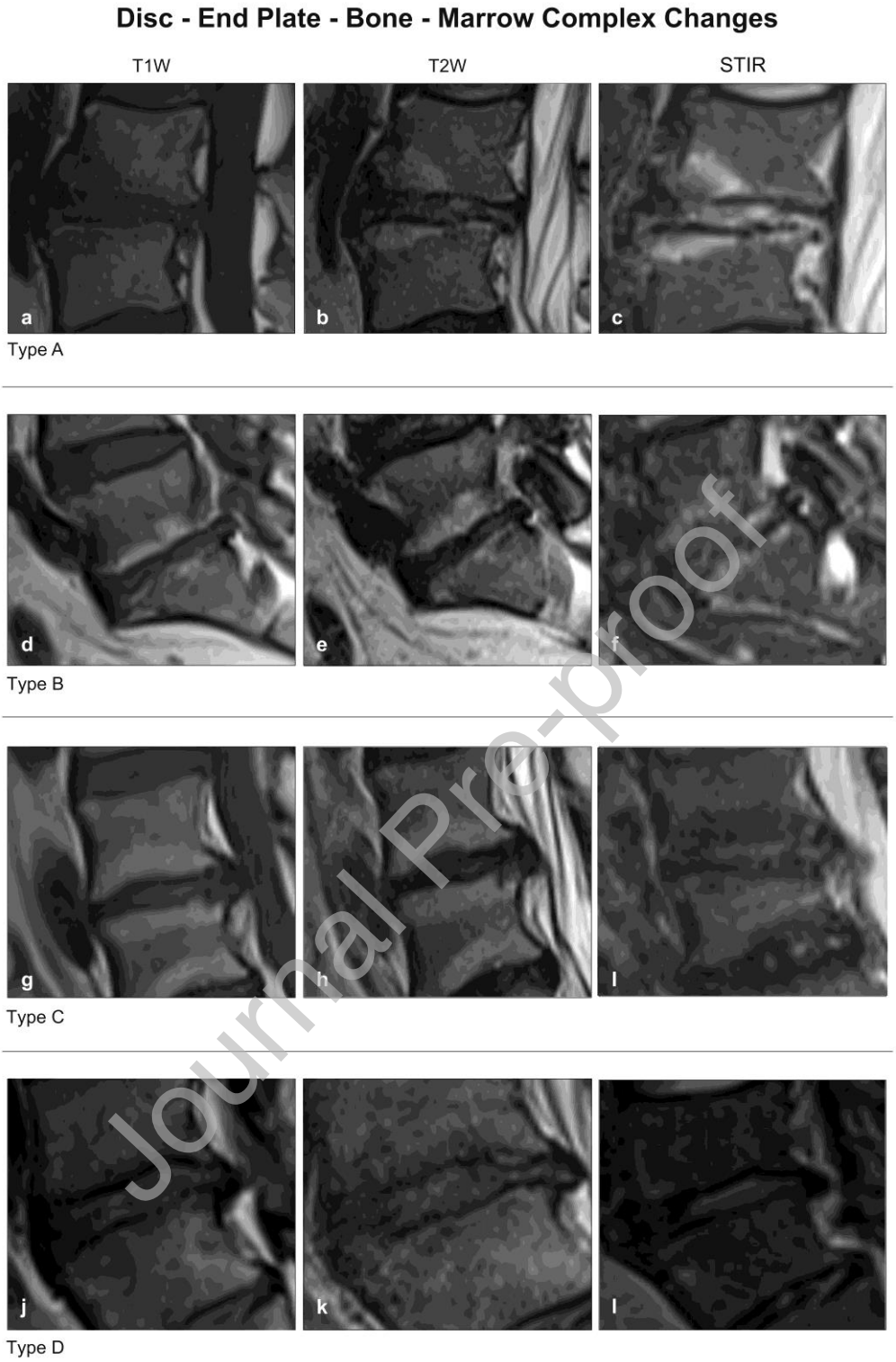


Figure 1. 'Disc- End plate-Bone-Marrow Complex' changes. Figure shows the characteristic features of the four different types – A to D. **Type A**(a to c) represents an ongoing acute active inflammatory process and shows T1W hypo, T2W hyper, and STIR hyperintense signal changes. Disc hyperintensity may or may not be present and the end plate is often indistinct with possible erosions. **Type B** (d – f) represents a state of 'Chronic

persistent inflammation' and shows edema in STIR interspersed with either fat or sclerosis. Disc hyperintensity may or may not be present and the endplate shows erosions. **Type C** (g – i) represents a state of 'Latency' with no element of acute activity and shows hyperintensity in both T1 and T2 and hypointensity in STIR. There is no hyperintensity in discs and end plate erosions are present. **Type D** (j – l) represents inactivity and shows hypointense signals on all three sequences. There is no hyperintensity in discs and end plate erosions are present.

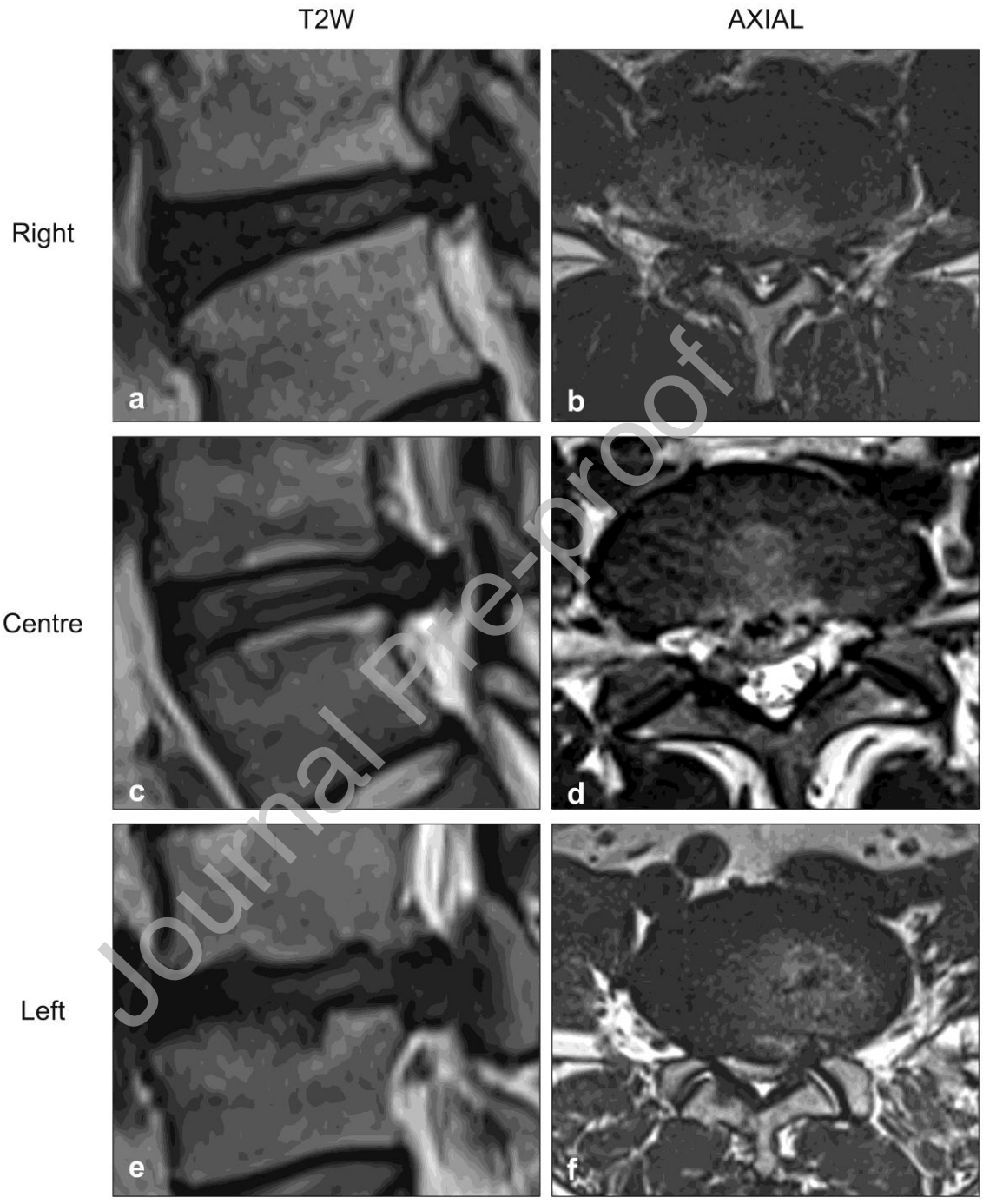


Figure 2. **The figure shows concordant disc herniations in three patients**: a right paracentral disc herniation with an end plate change on the right side (a,b). There is an endplate change in the center with a large central

disc collapse (c,d). An endplate change on the left side is accompanied by disc hyperintensity and left paracentral disc herniation (e,f).

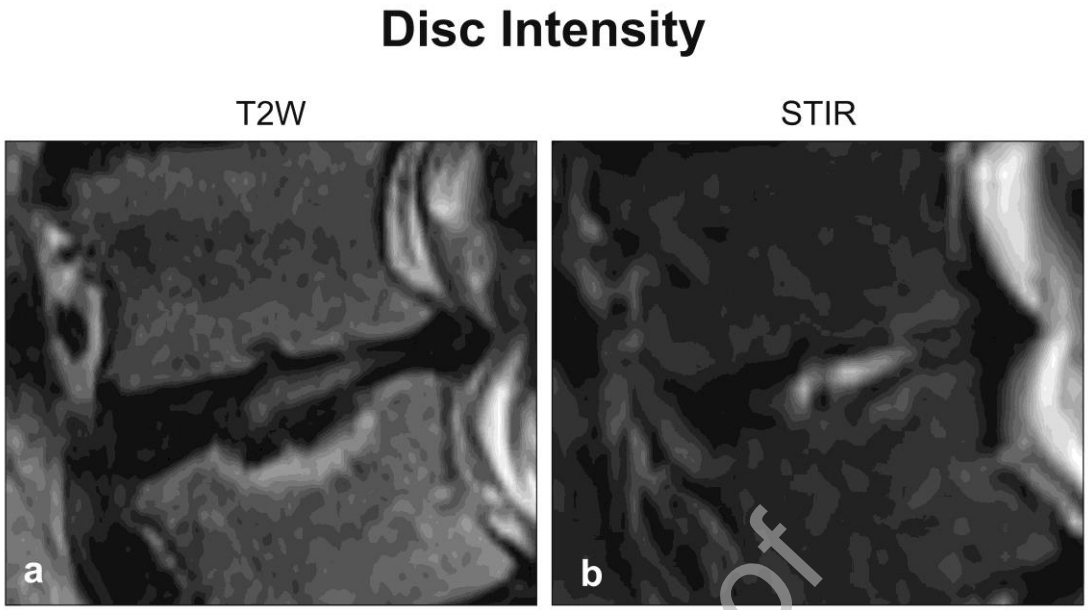


Figure 3. Disc intensity: T2W and STIR images show complete loss of nucleus pulposus and annulus fibrosus distinction with focal amorphous hyperintensity in the nucleus pulposus at the region of endplate erosion.

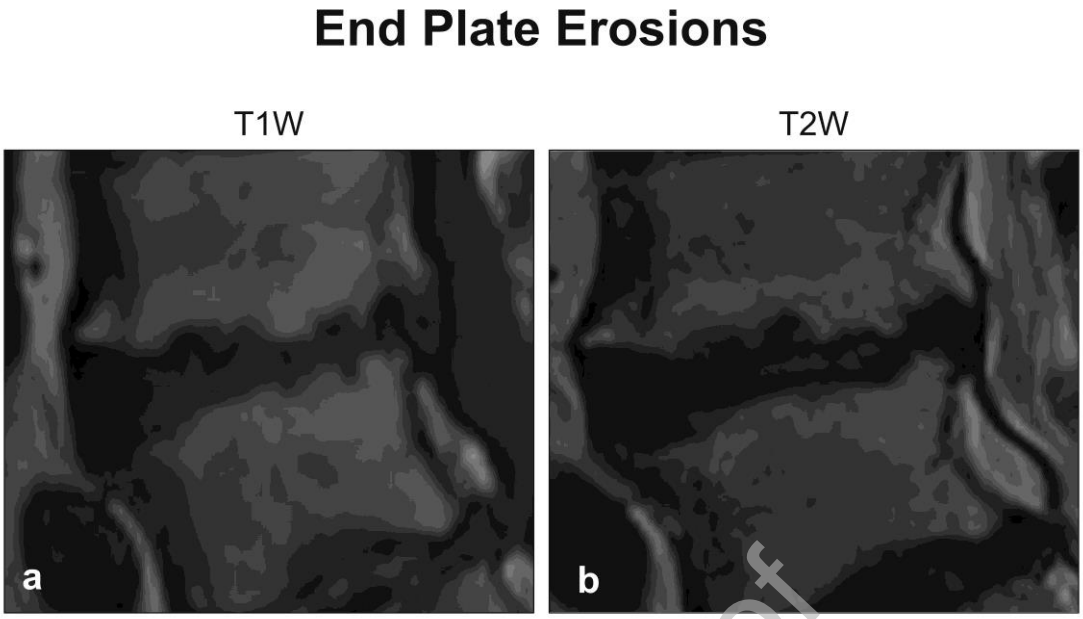
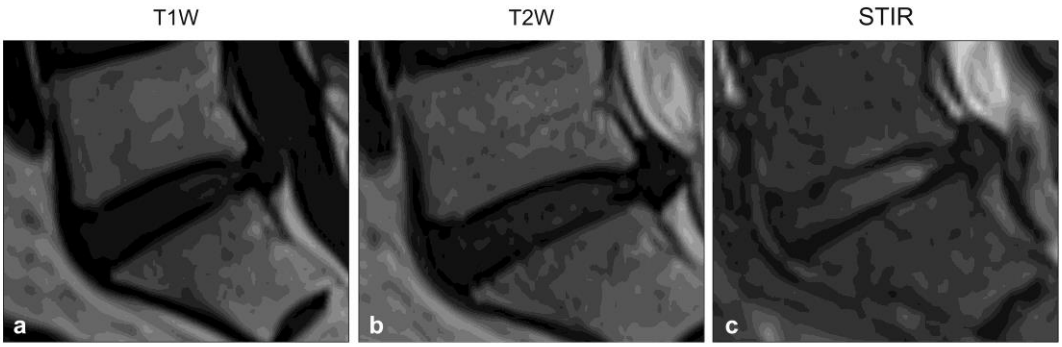
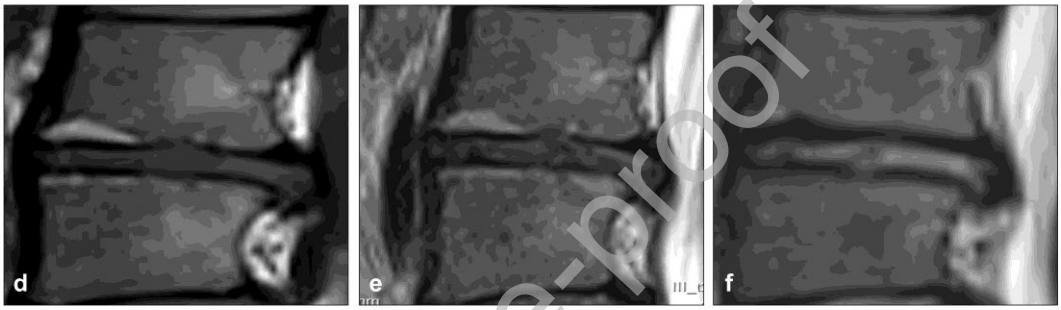


Figure 4. Endplate erosions- Erosions are clearly seen in T1W images as seen in this figure. Here there is diffuse irregularity involving both endplates.

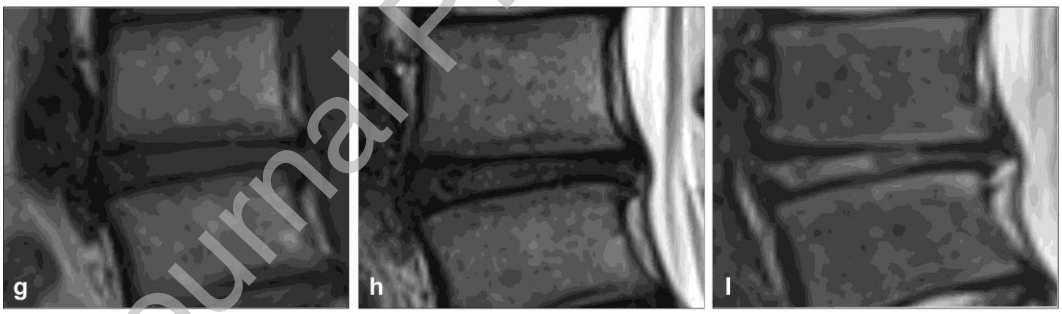
Use of STIR and Disc Hyperintensity in Reclassiying Modic Changes to DEBC



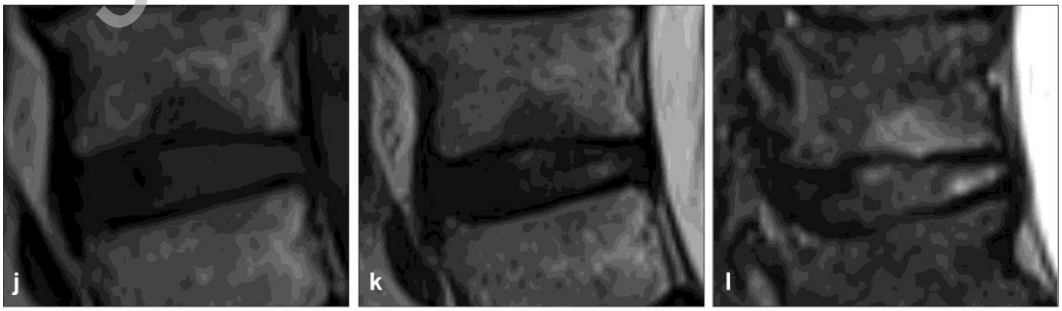
Non Modic to DEBC Type A Due To STIR Hyperintensity



Modic 2 to DEBC Type B due to Disc Hyperintensity



Modic 3 to DEBC Type B due to STIR and Disc Hyperintensity



Modic 3 to DEBC Type B due to STIR Hyperintensity

Figure 5. Use of STIR and disc hyperintensity in reclassifying Modic changes to DEBC – In patient 1 (a-c), T1W and T2W do not show any edema but STIR shows subchondral edema indicating Type A of DEBC instead of classifying it as a non-Modic disc. In patient 2(d-f), the Modic change classifies it as type 2 based on fatty conversion in T1W and T2W indicating a healing status. STIR images show mild marrow edema and prominent disc hyperintensity which indicates chronic activity and hence was classified as DEBC type B. In patient 3(g-i), there is hypointensity in both T1W and T2W classifying it into type 3 which indicates a healed inactive status. STIR however shows minimal marrow and disc hyperintensity indicating chronic activity classifying it as DEBC type B. In patient 4(j-l), Modic again classifies it as type 3 but STIR shows prominent marrow and disc hyperintensity classifying it as DEBC type B

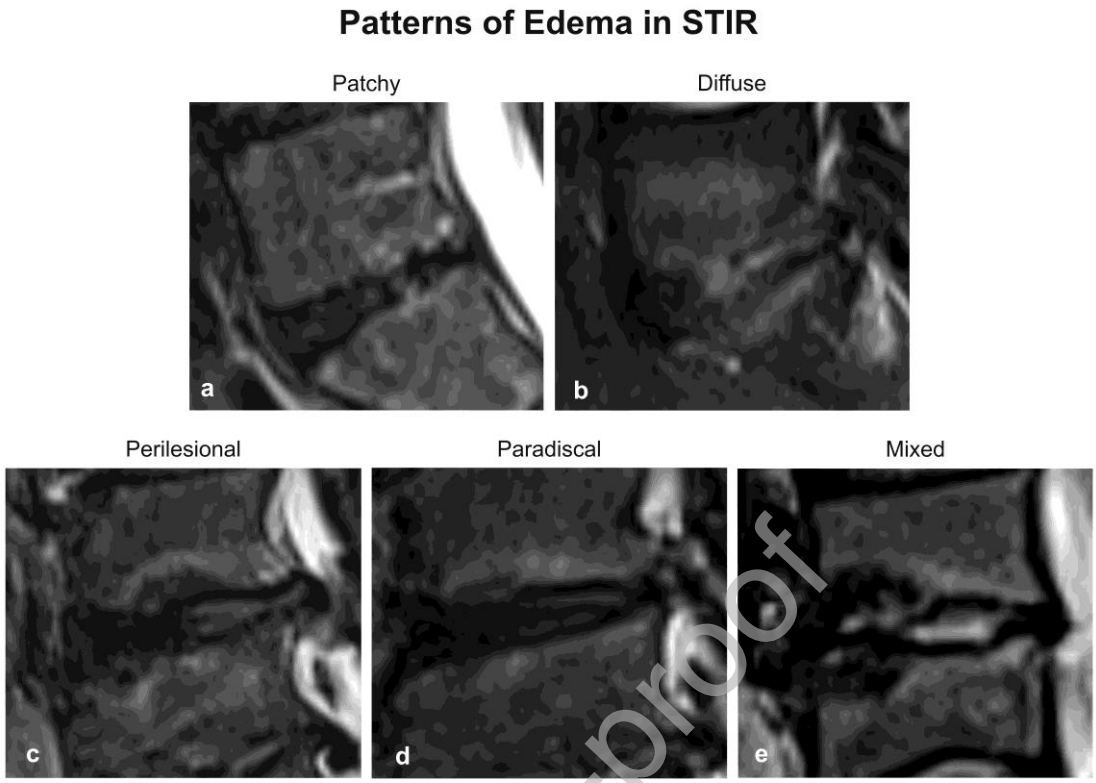


Figure 6. Patterns of edema in STIR- 4 different patterns were seen : (a) Patchy- where the edema is seen in a heterogenous pattern interspersed with fat or sclerosis. (b) Diffuse-edema is spread diffusely in the region of the subchondral change. (c) Perilesional- Fatty or sclerotic changes in the center surrounded by a rim of edema of variable severity. (d) Paradiscal- The edema is concentrated in the paradiscal region lining the bony endplate which can be surrounded by fat or sclerosis. (e) Mixed type- Frequently mixed patterns are also found.

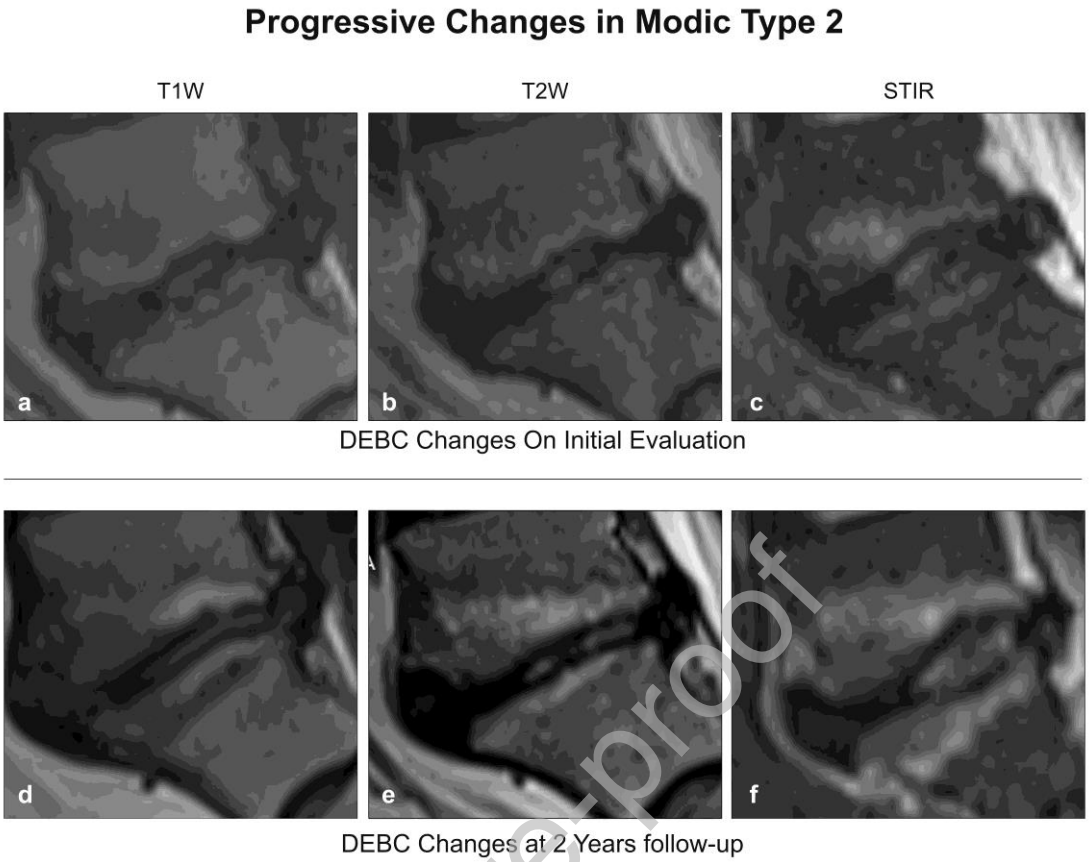


Figure 7. Progressive changes in Modic type 2- An L5-S1 disc in a 40-year-old man with frequent attacks of LBP shows typical Modic type 2(a,b). Clinically this is indicative of a healing status. However, STIR shows chronic activity with prominent subchondral edema and disc hyperintensity(c). The patient continued to have recurrent attacks of LBP, and MRI after 2 years shows progressive edema which is evident in T1W, T2W, and STIR along with fatty changes. In addition, there is a further increase in disc hyperintensity as well. This case clearly demonstrates the fallacy of the 'healing status' of type 2 Modic. DEBC clearly depicts the ongoing inflammation both in the initial and follow-up scans.

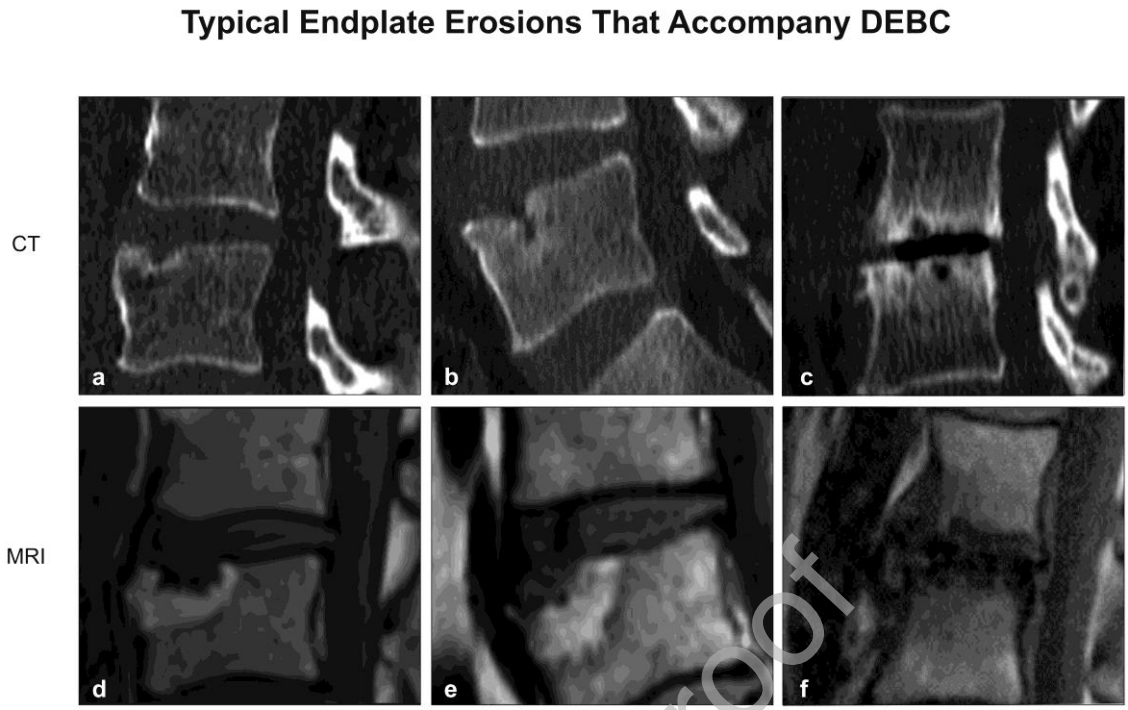


Figure 8. Typical endplate erosions that accompany DEBC- Although they are more prominently seen via CT(a,b,c), these changes can also be recognized on T1W images (d,e,f). We found bony endplate erosions as a significant finding in most cases of DEBC.

Table 1. Radiological basis for classification of the DEBC changes

Components of DEBC[+]	TYPE A (Acute Active)	TYPE B (Chronic/persistent)	TYPE C (Latent)	TYPE D (Healed)
Subchondral Bone	Edema	Fat And Edema	Fat	Sclerosis
End Plate	Indistinct Endplate Erosions ±	Endplate Erosions +	Endplate Erosions +	Endplate Erosions +
Disc Hyperintensity	±	±	-	-

[+] Disc-End plate-Bone marrow Complex

Table 2. Demographic Data

Variable	Control group	Low back pain group	p value
Total Patients Assessed	445	1085	-
Mean Age	42.8	47.25	
Sex Ratio(M:F)	3.45(345/100)	1.3(620/465)	
Number of End Plates Assessed	3560	8680	
Number of patients with DEBC^{+} changes *	39(8.8%)	448(41.3%)	**<0.005**
End Plate Erosions *	47(1.3%)	752(8.6)	**<0.005**
Number of Disc Levels Assessed	1780	4340	
Number of hyperintense Discs *	18(4%)	224(20.64%)	**<0.005**
Number of Herniations (H+) *	53(12%)	313(28.8%)	**<0.005**
Distribution of patients based on Modic changes			-
Type I	2(0.45%)	31(2.8%)	
Type II	37(8.3%)	392(36.1%)	
Type III	0	21(1.9%)	
Distribution of patients based on DEBC changes			
Type A	2 (0.45%)	35 (3.2%)	
Type B *	15 (3.35%)	232 (21.4%)	**<0.005**
Type C *	22 (5.0%)	166 (15.3%)	**<0.005**
Type D	0	15 (1.3%)	
Management distribution			-
Non-Surgical	-	883 (81.4%)	
Surgical	-	202 (18.6%)	

Patients With H+ Modifier Who Had DEBC changes *	5 (9.4%)	253 (80.8%)	**<0.005**
Patients With DEBC change with H+ modifier *	5 (12.8%)	253 (56%)	**<0.005**
Concordance of H+ With DEBC	4 (80.00%)	217 (85.77%)	**0.08**
Post-operative infections		9	
Non-DEBC	-	2 {2.5%}	-
DEBC		7 {5.7%}	
Type B		4/7 (57%)	
Type C		3/7 (43%)	

* Statistically significant

[+] Disc-End plate-Bone marrow Complex

Table 3. Distribution based on Modic and DEBC[+] changes in control and LBP group

Variables		Control		LBP	
		Frequency	Percentage	Frequency	Percentage
Number		93/3560	2.6	1124/8680	12.94
Modic Types	I	5/93	5.4	93/1120	8.36
	II	88/93	94.6	976/1120	87.14
	III	-	-	51/1120	4.5
DEBC Type	A	5/93	5.4	97/1124	8.6
	B	34/93	36.6	582/1124	51.8
	C	54/93	58.0	416/1124	37
	D	-	-	29/1124	2.6

[+] Disc-End plate-Bone marrow Complex

Table 4. Reclassification of Modic to DEBC changes based on TRIM and disc signal changes

Change	Rationale	No of EP
Non-modic to Type A DEBC[+]	**Subchondral bone TRIM hyperintensity**	**4**
Type 3 Modic to Type B DEBC	**Disc hyperintensity**	**4**
	Subchondral bone TRIM hyperintensity	**18**
Type 2 Modic to Type B DEBC	**Disc hyperintensity**	**40**
	Subchondral bone TRIM hyperintensity	**520**
Total		**586**

[+] Disc-End plate-Bone marrow Complex

Table 5. Frequency of Patients with DEBC Progressing to Surgical Management

DEBC[+] change	Frequency	Percentage %
NDEBC	80/637	12.5
Type A	7/35	20
Type B	68/232	29.3
Type C	44/166	26.5
Type D	3/15	20

Modic change	Frequency	Percentage %
Non-modic	81/641	12.63
1	7/31	22.6
2	108/392	27.55
3	6/21	28.57

[+] Disc-End plate-Bone marrow Complex

Table 6. Odds ratio for surgery

Modifiers	Odds ratio	95% CI	p value
End-plate erosions	3.6	2.65 to 5.01	<0.0001
Herniation	2.7	1.99 to 3.72	<0.0001
DEBC[+] changes	2.6	1.90 to 3.56	<0.0001
Herniation and DEBC	5.2	2.29 to 11.93	=0.0001
Herniation without DEBC	0.66	0.37 to 1.15	=0.1490

[+] Disc-End plate-Bone marrow Complex