Evaluation of Endplate Failure in Lumbar Disc Prolapse and Their Clinical Outcome Correspondence

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ABSTRACT:

BACKGROUND:

Endplate lesions connect in lumbar disc herniation (LDH) evolution, yet correlated clinical course scarcely comprehended.

AIM OF STUDY:

To examine the varieties of endplate failure (EPF) in LDH and their clinical outcome correspondence.

PATIENTS AND METHODS:

A study of sixty-seven cases that have single-level LDH conducted. Magnetic Resonance Imaging (MRI) and computed tomography (CT) scans distinguish EPF. Twenty-five patients have a laminectomy, and forty-two managed conservatively. Pursue of pain scores and clinical outcomes.

RESULTS:

Endplate pathology remarked in 65(97.01%), osseous endplate breakdown (Osseous failure) in 48(73.8%), and confined cartilaginous endplate lesions in 17 patients (26.2%). The group with bone failure possesses comparable pain and outcome scores yet worse neurological shortfall at the primary assessment. The clinical picture ameliorates in all groups, though the resurrection was minor in non-operative osseous failure cases.

CONCLUSION:

Endplate lesions are usually associated with symptomatic LDH. Osseous failure attendance progresses the neurological deficit and lessens the odds of rehabilitation with non-operatively planned cases. The fat-suppressed 3-dimensional fast spoiled gradient (3D FSPGR) mode of MRI efficiently uses for endplate ailment apprehension in the herniated disc.

KEYWORDS: lumbar disc herniation, Endplate failure.

INTRODUCTION:

LDH is a frequent cause of low back pain that hits 1 - 5/1000 population annually. Though its etiology and treatment differ extensively. The roots of disc-related back agony are multifacetedly emerging from mechanical compression, neurophysiological shifts, vascular, inflammatory, and immunologic processes. The nucleus pulposus cited for vast of the symptoms, momentarily other structures are increasingly approved for being a cause.[1]

The endplate affords durability and nourishment to the disc. It is the most vulnerable part of the vertebral-disc unit, praising it to mechanical breakdown. It is likewise the place where tectonic shifts of degeneration are most indisputable. Though preceding postmortem investigations focus on the role of EPF in LDH, none expressed its importance in the clinical setting. Those past investigations employed CT scan to appraise EPF, which increases the radiation and expense. [1]

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Table 1. Rajasekaran Classification of Endplate Junction Failure (Figure 1-6) [2]

1A	Irregularity of the endplate margins without an obvious free fragment
1B	Avulsion evidenced by a thin rim of bone
1C	Frank avulsion of a osseous fragment
1D	Osseous avulsion at the corners of both endplates
2	Herniation without any evidence of endplate junctional failure

Originally, K. et al[3] applied the 3D FSPGR MRI in cadavers to outline the typical anatomy of cartilage endplate and morphological differences in disco-vertebral interchange. Sch. et al[4] exerted marrow signal intensity and vertebral corner defect in MRI to prognosticate the attendance of cartilaginous segments in the extruded disc. Afterward, some scholars employed ultrashort echo time (UTE) or 3D fast low angle shot (3D FLASH) MRI modes to examine the endplate cartilage.[(5)]

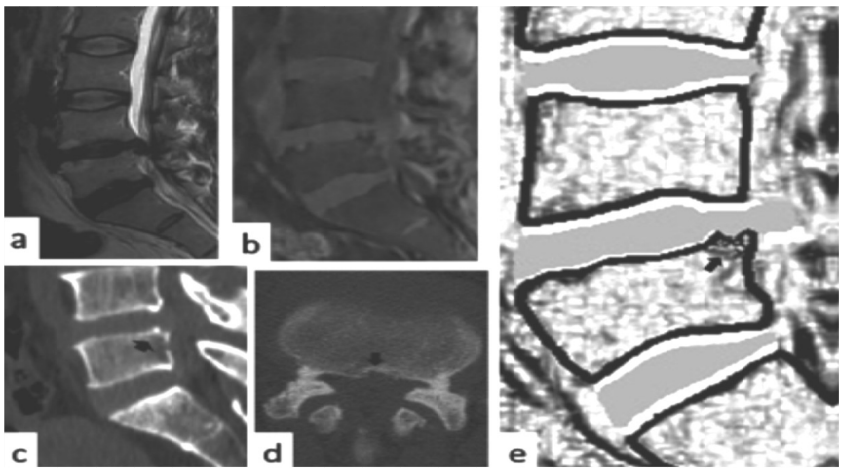

Figure 1:. Type 1A herniation: (a) T2 image with L4-5 LDH, (b) 3D FSPGR image, (c, d) CT scan images, (e) sketch of the 3D FSPGR. Black arrow—Osseous irregularity at posterior superior corner of L5.

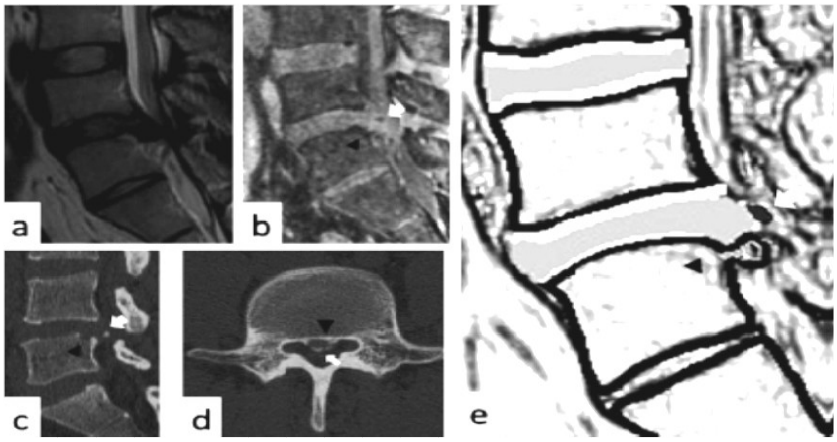

Figure 2: Type 1B herniation: (a) T2 image with L4-5 LDH, (b) 3D FSPGR image, (c, d) CT scan images, (e) sketch of the 3D FSPGR. White arrow—avulsed osseous fragment, black arrowhead—site of end plate failure.

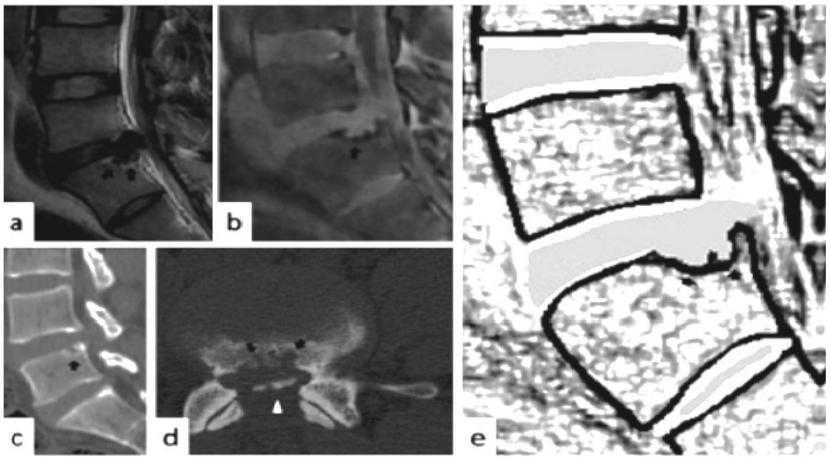

Figure 3:Type 1C herniation: (a) T2 image with L4-5 LDH, (b) 3D FSPGR image, (c, d) CT scan images, (e) sketch of the 3D FSPGR. Black arrows—defect in the inferior endplate along with subchondral sclerosis, white arrowhead—Avulsion of underlying osseous endplate.

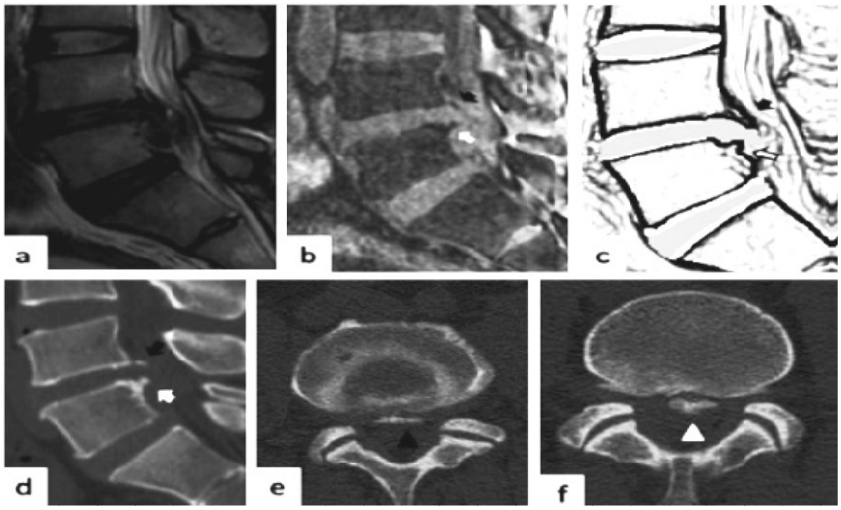

Figure 4: Type 1D herniation: (a) T2 image with L4-5 LDH, (b) 3D FSPGR image, (c) sketch of the 3D FSPGR, (d) midsagittal CT scan, (e) axial CT scan at upper endplate level, (f)axial CT scan at lower endplate level. Black arrows—disruption of superior endplate, white arrows—disruption of inferior endplate, arrowheads—avulsed osseous fragments at superior (black) and inferior (white) levels.

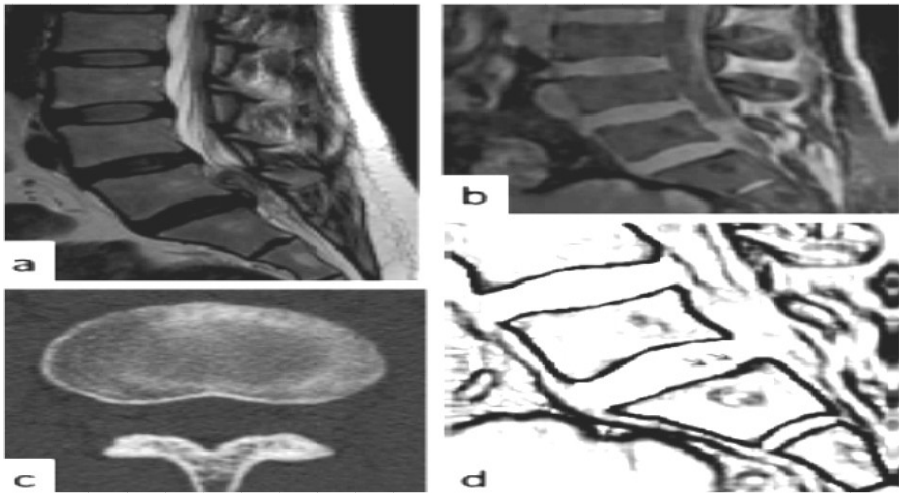

Figure 5:Type 2 herniation with cartilaginous endplate failure: (a) T2 image with L5-S1 LDH, (b) 3D FSPGR image, (c) axial CT scan image,(d) sketch of the 3D FSPGR. Arrows—Absence of hyperintense line of inferior cartilaginous endplate.

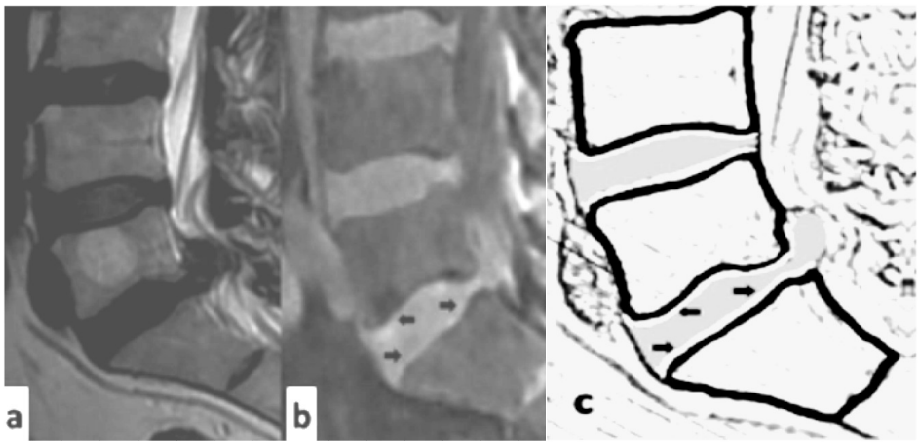

Figure 6:Type 2 herniation without endplate failure: (a) T2 image, (b) 3D FSPGR image, (c) sketch of 3D FSPGR image. Arrows—Hyperintense line between disc and vertebra depicting the intact cartilaginous endplate.

PATIENTS AND METHODS:

This prospective cohort analyses of isolated-level LDH present to the clinic amid February 2018 through May 2020 at the department of neurosurgery, Medical City, Baghdad, Iraq. An approval grants from the ethics committee of the institute. Patients incorporated were between 27 and 51 years of age, with nerve root strain signs +/or neurological abnormalities. Elimination criteria include kyphoscoliosis, preceding lumbar spinal operation, cauda equina syndrome, spinal canal stenosis, multilevel disc prolapse, spondylolisthesis, or spondylodiscitis.

Through MRI, an additional 3D FSPGR mode simultaneously with T1 and T2 phases were used to analyze the situation of the endplate. Sixty-seven patients composed the study group, and their MRI appraisal embodied level, plane (central, paramedian, or lateral), and stage of herniation (protrusion, extrusion, or sequestration). Particular situations of nucleus pulposus, annulus fibrosus,

and endplate remarked, and the LDH classify based on work by Rajasekaran et al[2] (Table 1). Yet, classification in the current study is primarily based on MRI features. Moreover, a CT scan was also performed in 54 cases to examine the osseous element of the endplate failure. All the radiological assessments are by independent radiologists unaware of the study.

A conservative strategy had been carried with analgesics, muscle relaxants, and a physiotherapy plan. The implications for surgery include the neurological deficit, no recovery after two months of non-operative management, or increment of symptoms and neurological deterioration. Discectomy by fenestration was the mode of surgery excluding cases with extensive disc prolapse and severe canal encasement where laminectomy has to consider. The protruded discs had observed for any breach of annulus fibrosus, next the nucleus pulposus was removed with care not to violate the endplates. Remove the loose fragments of discs had performed in extruded and sequestrated discs. The extract elements had been then inspected, tested with fingers, and conveyed for histopathological analysis to recognize any cartilage or osseous portions.

Patients followed up at intervals over 36 weeks, moreover amelioration of the symptoms evaluated by variations in neuro deficit, Straight Leg Raising (SLR), Visual Analog Score (VAS), and Oswestry Disability Index (ODI). Data registered and interpreted by SPSS 20. Discrimination through the assemblies by the Wilcoxon Rank Sum Test and Fisher Irwin Test.

RESULTS

A sum of sixty-seven subjects (forty-nine males and seventeen females) with particularly one-level LDH drafted for the research. As per the 3D FSPGR sequence, forty-six patients had an osseous endplate failure (osseous disruption) and assorted as assemblage one. CT scan distinguished an osseous breakdown in two added cases bringing the cumulative aggregate in gathering one to forty-eight (73.8%). All cases with osseous collapse had accompanied by a cartilaginous component, but then the classification had based on bony lesions. Out of these, eighteen patients had type 1A failure (Figure 1), fifteen had type 1B disruption (Figure 2), seven had disruption type 1C (Figure 3), and eight had failure type 1D (Figure 4).

The 3D FSPGR mode had determined to have superior specificity (98%) and sensitivity (92%) for the discovery of the osseous failure. In contradiction, the T1- and T2-weighted series could identify only 30% of osseous disruptions failing to discover most of the type 1A and 1B endplate failure. Nineteen subjects (29%) had type 2 endplate failure and had assorted as group 2, of which 17 patients had cartilaginous endplate failure (cartilaginous disruption) (Figure 5), and two subjects had just an annulus fibrosus breach (Figure 6).

Eleven subjects in group 1 and six patients in group 2 had a cartilaginous failure at levels added to the LDH level. Four patients in gathering 1 had an osseous disruption at levels distinct from the LDH. In all these four patients, the L3-4 was the herniated level, yet L4-5 had the osseous failure but no herniation. The disruption of the endplate had a statistically strong connection with the herniated discs than the lumbar discs without herniation (Odds ratio 12.40, $P < .01$).

The mean duration of back agony is 5.9 ± 4 months in group one, which is significantly longer than that of group two (2.9 + 2 months). In all patients, the low back pain was accompanied by lower extremities agony with a one-sided spread in 51 patients and two-sided transmission in 15 patients. Ten subjects had neurological abnormality (all in group 1) with the loss of ankle jerk in three of them. Total loss of fine touch was perceived in three patients (solely in group one) and reduced sensation in 29 patients (21 of group one and 8 of group two). Both the groups were comparable in terms of gender allocation (Fisher Irwin Test, $P = .05$), level of herniation (Table 2), degree of disc herniation (Table 3), and canal diameter (Table 4). Fifteen patients had migration of the herniated material of whom 11 had inferior (10 of group 1, 1 of group 2) and 4 had superior (1 of group 1, 3 of group 2) migration.

A total of 25 subjects were operated (twenty-three in group 1 and two in group 2) while 42 patients (twenty-five in group 1 and seventeen in group 2) were managed non-operatively. Eight patients from group 1 were originally designed for a conservative plan but later operated as the manifestations did not recover. Three patients (two in group 1 and one in group 2) were planned for surgery but approached conservatively due to their surgical comorbidities.

One patient from group 1 developed discitis in the postoperative phase, which was managed with intravenous antibiotics. There were no other significant complexities correlated to the surgery. The neurological state optimizes in all except one patient from group 1. In this patient, the discectomy was challenging due to the hardness of the disc.

The mean initial SLR was 27.1º ± 14.2º in group 1 and 33.1º ± 19.1º in group 2. The initial SLR was comparable among the groups, and all groups showed improvement during the follow-up period.

While there was no difference between the surgically treated patients in both groups, the recovery of SLR was significantly lower in group 1 patients treated conservatively. This difference was evident at six weeks and remained as such (Figure 7a). The primary mean VAS was 7.2 ± 0.87 in group 1 and 5.6 ± 0.96 in group 2, on another side the starting mean ODI was 46.9 ± 11.9 in group 1 and 28.7 ± 7.5 in group 2. Though at the start of the study VAS and ODI were significantly worse in group 1 patients than group 2, later on, all patients showed considerable clinical recovery (Figure 7b and c).

Table 2:Distribution of Level of Lumbar Disc Herniation (LDH).

LDH Level	Type 1A	Type 1B	Type 1C	Type 1D	Group 1 Total	Group 2	Fisher Irwin Test Between Group 1 and Group 2
L3-L4	1	4	2	0	7	2	P = .05
L4-L5	12	7	3	5	27	8	
L5-S1	5	4	2	3	14	9	

Table 3: Distribution of Stages of Lumbar Disc Herniation (LDH).

LDH Stage	Type 1A	Type 1B	Type 1C	Type 1D	Group 1 Total	Group 2	Fisher Irwin Test Between Group 1 and Group 2
Protrusion	11	10	5	2	28	11	*P* = .05
Extrusion	3	2	1	3	9	4	
Sequestration	4	3	1	3	11	4	

Table 4: Comparison of the Groups in Terms of Age and Canal Diameter.

	Age, y, Mean + SD	Canal Diameter, mm, Mean + SD
Group 1	37.3 + 7.9	4.93 + 1.20
Group 2	43.5 + 9.2	4.43 + 1.48
P value [a]	.021	.011
[a]P value for comparison of group 1 and group 2 (Wilcoxon Rank Sum Test).		

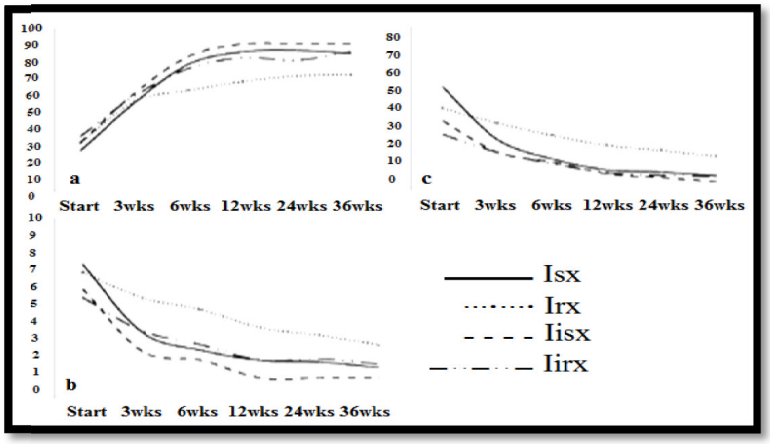

Figure 7:Evolution of clinical pathway throughout management: (a) variation of SLR, (b) development of ODI, (c) differences of VAS. Wks—weeks, Isx (solid line)—mean value of cases with osseous EPF undergoes surgery, Irx (dotted line)—mean value of cases with osseous EPF and conservatively treated, Iisx (dashed line)—mean value of cases without osseous EPF and were operated, Iirx (dash-dot line)—mean value of cases lacking an osseous EPF and conservatively managed. SLR, straight leg raising; ODI, Oswestry Disability Index; VAS, Visual Analog Score; EPF, Endplate Failure.

Table 5: Relation between endplate failure, LDH level, LDH severity, clinical picture & surgical outcome.

EPF STAGE	LDH LEVEL			LDH SEVERITY			CLINICAL PARAMETERS (Preoperative) Mean + SD				SURGICAL OUTCOME (Postoperative) Mean + SD		
	L3-L4	L4-L5	L5-S1	Protrusion	Extrusion	Sequestration	SLR		VAS	ODI	SLR	VAS	ODI
1A	1	12	5	11	3	4	50.2º ± 9.2º		3.2 ± 0.57	25.9 ± 7.9	59.3º ± 10.2º	1.2 ± 0.34	12.9 ± 5.1
1B	4	7	4	10	2	3	35.6º ± 5.2º		4.1 ± 0.21	46.9 ± 9.3	49.5º ± 5.4º	2.2 ± 0.22	21.3 ± 2.9
1C	2	3	2	5	1	1	31.4º ± 1.3º		6.4 ± 0.82	66.2 ± 7.1	40.2º ± 3.1º	4.2 ± 0.81	40.9 ± 5.1
1D	0	5	3	2	3	3	23.1º ± 12.2º		7.2 ± 0.11	80.4 ± 15.3	27.1º ± 14.2º	5.2 ± 0.87	60.9 ± 3.8
	Wilcoxon Rank Sum Test P =.05						Fisher Irwin Test between Groups P = .01						
SLR, Straight Leg Raising; ODI, Oswestry Disability Index; VAS, Visual Analog Score; EPF, Endplate Failure.													

Our study showed a strong correlation (P = .01) of surgical outcome with regard to EPF stage as a lower stage (1A or 1B) transformed into better outcomes (VAS, ODI and SLR) in the postoperative period.

Similarly a direct connection (P = .05) between EPF stage and LDH severity as less severe LDH found in the lower EPF stages (1A or 1B).

DISCUSSION:

The current research adopted MRI to value and analyze the endplate failure and associate it with the clinical setting. Few scholars have admitted breakage of the vertebral rim in the etiology of LDH,[6] Rajasekaran et al[2] are premier to analyze the endplate junctional breakdown correlated with LDH. In the current research, the 3D FSPGR sequenced to appraise the endplate cartilage and ascertained it to possess superior sensitivity and specificity for the apprehension of endplate collapse. Younger patients for this study with osseous failure (Table 4), presumably for, at a younger age, annulus fibrosus is more robust than endplate junction. Consequently, when placed in an utter squeezing or torsion simultaneously amidst axial compression and flexion, the endplate collapses ere annulus fibrosus.[7]

In the existing research, 47 cases (71%) held an osseous failure, and 17 (26%) possessed a cartilaginous breakdown. As all osseous failure cases also have cartilage endplate lesions, 97% of patients had remarkable cartilaginous elements in the herniated materials, which is more than many preceding studies. Nevertheless, most of the past papers had used histopathology to judge the osseous or cartilaginous parts where there were probabilities of missing smaller bits through surgical extraction and subsequent histopathological arrangements.[2,6-8] Anatomical distinctions in the annulus additionally can subscribe to the pattern of EPF. As the cartilage endplate does not reach past annulus halfway, the outside of the annulus is undeviatingly attached to the vertebral bone by Sharpey's fibers and may pluck an osseous piece from the dorsal rim of the vertebral body.[8]

For the annular central region, the collagen filaments are most abundant and append to the cartilage endplate at a distance aside from the bone. Due to the influential bonding among the osseous and cartilaginous endplates here, the dislodged cartilage may seldom take pieces of subchondral bone simultaneously with it, ending in an erosion of the osseous endplate.[8]

That describes the basis of the most basic models of EPF. Close to the inner annulus, agglutinant strength amidst the central cartilaginous endplate and neighboring bone is inadequate. Therefore, even with an average intensity, the nucleus-cartilaginous endplate can shovel the ossein ending in a confined cartilaginous breakdown.[4]

Disc herniations that are common in the younger age group associated with erosion and avulsion of endplates, especially with bigger herniations and migrations[6]. As the subjects in the present research are comparatively youthful aged, so can provide a reason for the likelihood of type 1A and 1B EPF. Four cases possessed an osseous failure at L4-L5 with the LDH was at L3-L4. Because osseous endplate breach may necessitate several months to advance to LDH, these additional level osseous pathology may be the launching etiology for forthcoming disc herniation.[9,12] Aforementioned may afford a shutter of possibility for interference to halt the progression and final herniation.[9]

In the current study, in the osseous failure assemblage, non-operative management resulted in remarkably more subordinate improvement than surgery. That attributed to the stiffness of discs in osseous failure that can yield considerable compression. Such compression is hard to be released but through operative intervention.[7-9] Also, in the current study had difficulty in the removal of the disc in one patient. When connected with an osseous piece (especially if large-sized), it may be challenging to extract the herniated disc by fenestration solely. These sufferers profit from a broader laminectomy. One patient had an enduring motor deficit following the operative intervention, presumably due to long-term contact tension amidst the herniated substance and nerve root ending in attenuation of microvasculature and myelin loss.[7] Another subject with osseous failure acquired discitis after the operation. Unrestricted migration of bacteria following the defect generated due to osseous failure and the anaerobic media encompassing the herniation could have implied this contagion.[8]

Former researches had declared that the attendance of cartilage endplate in the extruded material impedes the neovascularization and resorption, resulting in a more indisposed outcome of LDH.[7,8] Remarkably, in the study we take, cases with solely cartilaginous breakdown manifested noteworthy clinical improvement with conservative management.

No much research that describes this aspect nevertheless introduces a few probabilities. The MRI modes employed in this research could only distinguish the bearing of cartilage endplate pathology, merely quantify the mass of cartilage in the herniated elements.

Concerning the broad fluctuations in cartilage amount, the prognosis consequently is altered.[7] As the severity of neuro deficit was enormously more inferior in solitary cartilaginous failure, possible that these cases had an insignificant volume of cartilage in the herniated substance.[7] W. et al[7] perceive that higher hyaline cartilage mass causes the herniation more cumbersome that points to inferior migration. That had been seen solely in one out of seventeen sufferers among the cartilaginous failure gathering of the present research and as our study showed a strong correlation (P = .01) of surgical outcome with regard to EPF stage as a lower stage (1A or 1B) transformed into better outcomes (VAS, ODI and SLR) in the postoperative period. Similarly, a direct connection (P = .05) between EPF stage and LDH severity as less severe LDH found in the lower EPF stages (1A or 1B).

CONCLUSION:

Endplate abnormalities, particularly the osseous component, are customary in symptomatic LDH. It further reinforces the claim that the endplate is the principal position of pathology.

The clinical outcome predicts through endplate assessment, which can be successfully conducted through a 3D FSPGR MRI sequence, as the involvement of the osseous element raises the necessity for surgery.

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