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Effects of endplate healing morphology on intervertebral disc degeneration after pedicle screw fixation for thoracolumbar fractures

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Abstract

The cartilage endplate plays an important role in the stress distribution and nutrition metabolism of the intervertebral disc. The healing morphology of the endplate after spinal fracture and its effect on the intervertebral disc degeneration are still unclear.

This was a retrospective study. Patients with traumatic single-level thoracolumbar fractures treated in our orthopedic trauma service center from June 2011 to May 2019 were included and the relevant data were collected from the medical records. Based on combined computed tomography and MRI images, the endplate injury status was determined (no endplate injury, unilateral and bilateral endplate injury). According to the location of the injury, endplate injury was further divided into endplate central injury and endplate peripheral injury. The degree of posttraumatic disc lesions and disc degeneration during follow-up were classified based on the Sander classification and the Pfirrmann classification, respectively. According to the T1 image of MRI at the final follow-up, the healing morphology of endplates was determined and classified. Univariate analyses and correlation analyses were performed to evaluate the within- and between-group differences.

There were in total 51 patients included in this study. Cartilage endplate fracture was significantly closely related to the degree of degeneration of the intervertebral disc ($P = .003$). Injuries in different parts of the endplate have no significant effect on the intervertebral disc degeneration ($P = .204$). The healing morphology after endplate fracture significantly affected the degree of intervertebral disc degeneration ($P = .001$). The comparisons of groups showed that the effects of irregular healing and traumatic Schmorl nodes on disc degeneration were not statistically significant, but were significantly significant with increased curvature.

These results suggest that the irregular healing and the traumatic Schmorl nodes are closely related to intervertebral disc degeneration. The presence and severity of the endplate injury can provide valuable information for individualized clinical decision-making processes.

Abbreviations: BMD = bone mineral density, CT = computed tomography, IEC = increased endplate curvature, IH = irregular healing, MRI = magnetic resonance imaging, T1WI = T1-weighted imaging, T2 = T2-weighted imaging, TIRMs = turbo inversion recovery magnitude sequences, TSN = traumatic Schmorl node.

Keywords: endplate injury, intervertebral disc degeneration, related factors, Schmorl nodes, spinal trauma

1. Introduction

With the development of pedicle screw technology, most patients with spinal fractures have obtained favorable clinical and radiographic results.[1] However, some patients have chronic instability and low back pain after surgery, even severe disc degeneration in some cases, which seriously affect quality of life.[2] The intervertebral disc plays an important role in maintaining the stress distribution of the spine.[3] Because of its elastic biomechanical characteristics, intervertebral discs can absorb energy and exert a moderating influence to make stress distribution more uniform during stress transmission. Therefore, before a vertebral fracture occurs, the intervertebral disc has absorbed a large amount of energy, possibly leading to its injury.[4] Due to the three-dimensional complexity of injuries, the imaging manifestations of intervertebral disc injuries are also complicated, making it difficult to describe the degree of injury with a standard and widely recognized evaluation system. Sander et al reported a new evaluation system for traumatic intervertebral disc lesions based on the morphological and signal changes of injured intervertebral discs, which is simple and has been widely used in clinical practice. However, this approach does not describe the clinical outcome of the injured intervertebral disc and

fails to clarify the relationship between each injury type and intervertebral disc degeneration.[5]

As a part of the intervertebral disc, the cartilage endplate plays an important role in facilitating the nutrition supply, metabolic exchange, and biomechanical transmission of the whole intervertebral disc. Otherwise, the structure damages or changes of composition of the cartilage endplate will significantly affect the infiltration and exudation of nutrients and metabolic waste, resulting in undesirable metabolism of intervertebral disc cells and promoting the degeneration of intervertebral disc tissue. Although the bony part of a vertebral fracture can completely heal and restore the normal strength, the pedicle screw system cannot directly reduce the endplate fracture, which makes the healing morphology of the cartilage endplate unpredictable. However, by far as we know, the relationship between the healing morphology and intervertebral disc degeneration has still been unclear. We hypothesized that cartilage endplate injury would accelerate the degeneration of the intervertebral disc and the aim of this study was to explore the relationship between the healing morphology of the cartilage endplate and intervertebral disc degeneration by follow-up magnetic resonance imaging (MRI).

2. Materials and methods

2.1. Patient inclusion and exclusion

Prior to its commencement, this retrospective study was approved by the institutional review committee of the 3rd Hospital of Hebei Medical University. We reviewed the medical records of 51 patients with traumatic single-level thoracolumbar fractures treated in the orthopedic trauma service center from June 2011 to May 2019. An electronic medical records system was sought to review the injury mechanisms. This study only included T11 to L4 thoracolumbar fractures. The exclusion criteria included pathological spinal fracture, spondylitis, osteoporotic fracture (age over 60 years or T value of dual X-ray bone mineral density (BMD) measurement less than 2.5), and degenerative disc disease without injury to the intervertebral disc.

2.2. Imaging

All patients underwent standard X-ray (including anteroposterior view and lateral view), computed tomography (CT) and MRI preoperatively. According to the severity of the fracture and the surgeon's preference and experiences, patients underwent open reduction and short segment fixation, open reduction and intermediate pedicle screw techniques, or open reduction and long segment fixation. All surgical procedures were performed under general anesthesia. For short segmental fixation, we exposed the levels above and below the injured segment via posterior midline approach in the prone position. Pedicle screws were inserted into bilateral pedicles of the adjacent vertebrae. Long posterior fixation with pedicle screws and rods two-levels above and below the fracture level provided more stable fixation. For intermediate screw fixation, additional screws were inserted into the pedicles of the fractured vertebrae. Indirect reduction of collapsed vertebral bodies and correction of the segmental kyphosis were performed via ligamentotaxis after connecting rods distraction toward the cranially and caudally in sequence. For all surgical procedures, no bone graft fusion was performed between the transverse process or spinous process, and no transpedicular bone grafting was performed. The mean time until the internal fixator was removed was 13.5 months after the injury (range: 8–18 months). The average total follow-up time was 28.2 months (range, 11–66 months). MRI scanning was performed using a 3.0T MAGNETOM Verio, 1.5T MAGNETOM Avanto, or 1.5T MAGNETOM Symphony MRI system (Germany, Siemens) with a spine surface matrix coil (Spine Matrix) and turbo spin echo imaging (TSE). T2-weighted images were obtained in the axial plane. In the supine position, images were obtained with T1, T2, and T2 turbo inversion recovery magnitude sequences (TIRMs) in the sagittal plane. Sagittal T1-weighted imaging (T1WI) and T2-weighted imaging (T2WI) was performed with field of view (FOV) of 280 mm/300 mm, a matrix of 288×384, 256×512, or 224×320, and a layer thickness of 4 mm. Sagittal T2WI with TIRM was performed with an FOV of 280 mm/300 mm, matrix of 230×256, 192×256, or 192×320, and a layer thickness of 4 mm. Axial T2W1 was performed with an FOV of 210 to 240 mm, matrix of 240×320, 288×384, or 204×512, and a layer thickness or 4 mm. The following sagittal MRI sequences were performed: T1WI TSE (repetition time/echo time (TR/TE), 540–650/9.6–11), T2WI TSE (TR/TE, 1650–3000/88–101), T2WI TIRM (TR/TE, 2560–4000/70–80), axial T2WI TSE (TR/TE, 2300–3800/99–112).

2.3. Data collection, measurements, and definition

According to the AO classification, the fracture patterns were classified on the initial X-ray and CT images as follows: Type A, vertebral compression; Type A1, simple compression fracture without involving the posterior wall of the vertebral body; Type A2, coronal splitting fracture of the bilateral endplates without involving the posterior wall of the vertebral body; type A3, burst fracture involving the posterior wall of the vertebral body; Type B, tension band injury; and Type C, displacement/translational injury. Based on fracture status evaluated by the initial CT and MRI scans, the endplate injury was classified into without presence of endplate injury, unilateral endplate injury, and bilateral endplate injury. The endplate injury group was further divided into the central endplate injury and peripheral endplate injury groups according to the injury site. On the median sagittal and median coronal images, the endplate was divided into 3 equal parts.[6] If the most obvious area of the endplate fracture showed the angulation was located in the middle third portion, it was defined as a central endplate injury (equivalent to the nucleus pulposus in the disc), and an injury of circumjacent third was defined as a peripheral endplate injury (equivalent to the annulus fibrosus in the disc). According to Sander classification of traumatic intervertebral disc lesions, the intervertebral disc injuries were classified based on the preoperative MRI.[5] A grade 0 injury showed no morphological change in the intervertebral disc of the fractured vertebral body, indicating there being no injury. The manifestation of a grade 1 injury was high signal on T2-weighted or T2 TIRM images, suggesting disc edema. A grade 2 injury was defined as a reduction in the original hyperintensity around the lesion on T2-weighted or T2 TIRM images and the appearance of isointensity to hyperintensity on T1-weighted images, indicating disc rupture with intradiscal hemorrhage. The criteria for grade 3 injury were intervertebral disc invasion, annular tear, or endplate protrusion. According to the Pfirrmann classification system, the degree of intervertebral disc degeneration was evaluated on MRI at the final follow-up.[7] The cranial and caudal discs of fracture were compared with uninjured discs to take into account age-related changes in disc signal intensity.
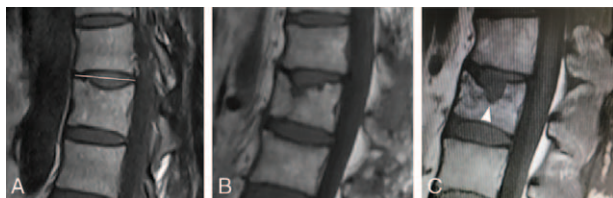
Figure 1. Increased endplate curvature (A), irregular healing (B), and traumatic Schmorl nodes (C) images on T1-weighed magnetic resonance images. Landmarks in midsagittal plane: the white straight line represents the connecting line between the anterior and posterior edges of the vertebral body. The white arrow represents traumatic Schmorl node formation due to unsatisfactory reduction of the endplate.

According to the median sagittal T1-weighted MRI scans, the healing morphology of the endplate was divided into increased endplate curvature (IEC), irregular healing (IH), and traumatic Schmorl node (TSN). Specifically, a straight line was drawn along the median sagittal image of the endplate on the anterior and posterior edges of the vertebral body. If there was intervertebral disc tissue between the straight line and the edge of the endplate, and compared with the adjacent endplate without fracture, the indentation degree was increased, then there was IEC (Fig. 1A). If the signal of the cartilage endplate was continuous, but the signal pattern was not smooth, and there were local bumps or indentations, then there was IH (Fig. 1B). If the signal of the cartilage endplate was continuous, and there was a round or irregular herniation through the cartilage endplate, in which the intervertebral disc tissue can be seen and intruded, then there was TSN (Fig. 1C).

2.4. Statistical analysis

Data collection, measurements, and evaluation were performed by an experienced traumatic orthopedic surgeon and a senior radiologist experienced in musculoskeletal imaging. Three months after the first assessment, the traumatic orthopedic surgeon and radiologist reevaluated all data in the same way at their own independent workstations. IBM SPSS 21.0 (IBM Corporation, Armonk, NY) was used for statistical analysis. The Wilcoxon rank sum test and Kruskal–Wallis H test were used to statistically analyze the grading data. The least significant difference (LSD) test was used for pairwise comparisons. A $P < .05$ was considered statistically significant.

3. Results

3.1. Patient characteristics

A total of 51 patients were enrolled in this study, including 39 males and 12 females. The average age was 42.2 years (range, 22–59 years). Fall injuries accounted for 47.2% (24/51) of the 51 cases, followed by heavy objects 25.4% (13/51), fall from height 17.6% (9/51), and traffic accidents 9.8% (5/51). Among them, 23 patients were treated with open reduction and short segment fixation, 16 patients with open reduction and intermediate pedicle screw techniques, and 12 patients with open reduction and long segment fixation.

3.2. Analysis of disc degeneration by fracture patterns

7.8% of the injuries involved T11 level, 23.5% at T12, 45.4% at L1, 17.6% at L2, 1.9% at L3, and 3.8% at L4 level. 31.1% (16/51) were classified as type A1 fractures, 5.8% (3/51) as type A2 fractures, 37.7% (19/51) as type A3 fractures, 23.5% (12/51) as type B fractures, and 1.9% (1/51) as type C fractures. As the fracture severity increased, the degree of intervertebral disc degeneration significantly increased ($P = .006$) (Table 1). Among the groups, the degree of disc degeneration of A1 fractures was significantly lower than that of other fracture types (A1 vs. A2 $P = .012$, A1 vs. A3 $P = .003$, A1 vs. B $P = .001$).

3.3. Analysis of disc degeneration according to endplate lesion

Among the 51 patients, 33 patients had a unilateral endplate injury with corresponding 33 intervertebral discs, 15 patients had bilateral endplate injuries with corresponding 30 intervertebral discs included for analysis. The remaining 3 patients without presence of endplate injuries with corresponding 6 intervertebral discs were considered as control group. The existence of an endplate injury was significantly related to the degree of

Table 1

Comparisons of degree of disc degeneration according to the fracture classification.

| Fracture classification | Classification of disc degeneration during follow-up | | | | χ^2 | P |
	II	III	IV	V		
A1	12 (37.5%)	13 (40.6%)	7 (21.9%)	0 (0.0%)	14.517	.006
A2	0 (0.0%)	2 (33.3%)	4 (66.7%)	0 (0.0%)		
A3	3 (7.9%)	17 (44.7%)	18 (47.4%)	0 (0.0%)		
B	2 (8.3%)	9 (37.5%)	11 (45.9%)	2 (8.3%)		
C	1 (50.0%)	0 (0.0%)	1 (50.0%)	0 (0.0%)		

(A1 vs. A2 $P = .012$; A1 vs. A3 $P = .003$; A1 vs. B $P = .001$).

Table 2

Comparisons of degree of disc degeneration according to the endplate injury.

| Endplate injury | Classification of disc degeneration during follow-up | | | | χ^2 | P |
	II	III	IV	V		
Unilateral endplate injury	0 (0.0%)	20 (60.6%)	12 (36.4%)	1 (3.0%)	11.886	.003
Bilateral endplate injury	0 (0.0%)	11 (36.7%)	18 (60.0%)	1 (3.3%)		
No endplate injury	4 (66.6%)	1 (16.7%)	1 (16.7%)	0 (0.0%)		

Table 3

Comparisons of degree of disc degeneration according to the injury site of endplate.

Injury site of endplate	Classification of disc degeneration during follow-up			Z	P
	III	IV	V		
Central injury	13 (59.1%)	9 (40.9%)	0 (0.0%)	1.271	.204
Peripheral injury	18 (43.9%)	21 (51.2%)	2 (4.9%)		

Table 6

Intergroup comparison of the effect of endplate healing morphology on intervertebral disc degeneration by LSD.

Pairwise comparison	Mean difference	P	95% Confidence interval	
			Lower bound	Upper bound
IEC vs. IH	−16.839	<.001	−25.290	−8.388
IEC vs. TSN	−12.490	.014	−22.310	−2.671
IH vs. TSN	4.348	.356	−5.005	13.702

IEC = increased endplate curvature, IH = irregular healing, TSN = traumatic Schmorl node.

intervertebral disc degeneration ($P = .003$) (Table 2). There was no significant difference in the degree of intervertebral disc degeneration between unilateral endplate injury and bilateral endplate injury (unilateral vs. bilateral $P = .075$, unilateral vs. no injury $P = .010$, bilateral vs. no injury $P = .001$). Among the 63 injured endplates considered as samples, 65.1% (41/63) involved the central area and 34.9% (41/63) involved the peripheral area, but it was not significant regarding the degeneration of intervertebral discs ($P = .204$) (Table 3).

3.4. Analysis of disc degeneration according to intervertebral disc lesion and endplate healing morphology

The grades of 102 intervertebral discs in 51 patients based on the preoperative original MRI were as follows: grade 0 in 23.5% (24/102), grade 1 in 37.4% (38/102), grade 2 in 26.4% (27/102), and grade 3 in 12.7% (13/102) of the lesions. The grades of traumatic disc lesions based on Sander classification system were significantly related to the degree of degeneration ($P < .001$) (Table 4). At the last follow-up, 63 cartilage endplate fractures healed with the following healing morphology: IEC in 33.3% (21/63), IH in 42.9% (27/63), and TSNs in 23.8% (15/63). The healing morphology after an endplate fracture significantly affected the degree of intervertebral disc degeneration ($P = .001$) (Table 5). However, there was no significant difference between IH and TSN in intervertebral disc degeneration ($P = .356$). Compared with the other 2 groups, the IEC group had significantly different intervertebral disc degeneration (IEC vs. IH $P < .001$, IEC vs. TSN $P = .014$) (Table 6).

4. Discussion

The endplate is a layered composite material composed of semiporous thickened cancellous bone (0.6–1 mm) and transparent cartilage (0.2–0.8 mm).[8] Because of its unique anatomical morphology and material properties, the cartilage endplate injury could have significant effect on biological transport and stress conduction.[9] In this study, we retrospectively investigate the relationship between several variables and occurrence of intervertebral disc degeneration. Our results suggested that irregular healing and the traumatic Schmorl nodes are closely related to intervertebral disc degeneration. The presence and severity of the endplate injury can provide valuable information for individualized clinical decision-making processes.

The destruction of the integrity of the endplate may cause a pathological cascade, eventually leading to degeneration of the adjacent intervertebral disc. First, the protrusion of the nucleus pulposus into the vertebral body leads to a direct loss of water and proteoglycans in the nucleus pulposus, and the accompanying inflammatory/immune reaction can further destroy the homeostasis of the intervertebral disc and damage cell metabolism.[10] Second, endplate damage and the secondary repair may destroy or block the contact channel between the intervertebral disc tissue and the bone marrow in the vertebral body, hindering the nutritional supply (mainly oxygen and glucose) of the intervertebral disc, which was thought to be the primary cause of nucleus pulposus cell death.[11] In addition, changes of the matrix stress distribution near the intervertebral disc from endplate injury may further aggravate this process.[12,13] These studies

Table 4

Comparisons of degree of disc degeneration according to the grade of intervertebral disc injuries.

Grade of intervertebral disc lesions	Classification of disc degeneration during follow-up				χ^2	P
	II	III	IV	V		
0	16 (66.7%)	5 (20.8%)	3 (12.5%)	0 (0.0%)	41.131	<.001
1	2 (5.3%)	25 (65.8%)	11 (28.9%)	0 (0.0%)		
2	0 (0.0%)	8 (29.6%)	17 (63.0%)	2 (7.4%)		
3	0 (0.0%)	3 (23.1%)	10 (76.9%)	0 (0.0%)		

Table 5

Comparisons of degree of disc degeneration according to the endplate healing morphology.

Endplate healing morphology	Classification of disc degeneration during follow-up			χ^2	P
	III	IV	V		
IEC	17 (81.0%)	4 (19.0%)	0 (0.0%)	13.296	.001
IH	8 (29.6%)	17 (63.0%)	2 (7.4%)		
TSN	6 (40.0%)	9 (60.0%)	0 (0.0%)		

IEC = increased endplate curvature, IH = irregular healing, TSN = traumatic Schmorl node.

supported our finding that traumatic endplate injury regardless of unilateral or bilateral injury can lead to disc degeneration.

The synergistic effect of physical and biological mechanisms also contributes to intervertebral disc degeneration. The endplate near the annulus fibrosus is thicker than that in the central nucleus pulposus region, and the lower endplate of the same vertebral body is thicker than the upper endplate.[8] We observed that the central nucleus pulposus area is the main area involved in endplate fracture and the fracture line often extends to the peripheral region, and vice versa. Endplate fracture leads to an abnormal stress distribution in the adjacent intervertebral discs and increases the risk of internal rupture and degeneration. In vitro studies have also found that endplate damage promotes a low expression of anabolic genes (aggrecan) and high expression of catabolic genes (MMP-1, -3, -13) and proinflammatory genes (TNF, IL-6) and speeds up the process of intervertebral disc degeneration.[14] According to studies on AO spinal fracture classifications, the morbidity of type A1 and A3 fractures is the highest and that of type C fractures is the lowest,[15] which is consistent with the distribution of fracture types in our study. As the severity of the fracture increases, the form of intervertebral disc injury can also manifest as intervertebral disc edema, intradisc hemorrhage, the nucleus pulposus protruding into the vertebral body, which was found to be closely related to the injury to the adjacent intervertebral disc, and the finding was consistent with ours.[16]

Improvements have been consistently made to indicate the instability and severity of injury to the intervertebral disc. Sander et al[5] used routine MRI to identify the morphological and signal changes of the injured intervertebral disc, and thereby established a grading system from grade 0 to grade 3 and demonstrated a high degree of credibility (interobserver and intraobserver reliability, Cohen kappa coefficient $\kappa=0.96$). This method plays an especially irreplaceable role in the imaging of intervertebral discs and cartilage endplates. In this study, therefore, we used MRI to explore the relationship between the classification of traumatic intervertebral disc lesions and postoperative intervertebral disc degeneration, and the results were similar to those in a previous study by Sander et al.[17] In their study, the disc injury is progressive, especially for less severe discs with only signal changes but no morphological changes. For severer grade 3 injuries, no repair of the intervertebral disc was observed during follow-up, but contrarily obvious degeneration occurred. This is consistent with our current finding that the more severe the intervertebral disc injury, the more marked the disc degeneration at follow-up. This also proves that the traumatic disc lesion classification system can predict the degree of disc degeneration to a certain extent. The difference is that we paid more attention to the prognosis of intervertebral disc injuries and use of the Pfirrmann degeneration classification system instead of the traumatic disc lesion classification system in the follow-up.

The morphology of the endplate has also been established to play an important role in maintaining the mechanical function of the spine or intervertebral disc. Pappou et al[18] analyzed the relationship between the morphology of the lumbar endplate and the degree of degeneration by MRI, and found the shape of the endplate was related to disc degeneration and Modic changes. In our study, the degree of intervertebral disc degeneration corresponding to IEC was lower than that corresponding to IH and TSN, but that was not significantly different between IH and TSN. In all fractured vertebrae, we did not observe the significant Modic changes in the MRI signal. In a study on the

relationship between vertebral endplate morphology and lumbar disc degeneration, researchers found that the level of vertebral endplate flattening was related to the severity of intervertebral disc degeneration.[19] IEC seemed to be a "protective mechanism" in the process of intervertebral disc degeneration. The potential explanation is that the IEC group usually showed a light simple fracture on the endplate. Based on Wolff's law statement that when the load on the bone changes the bone tissue is remodeled to accommodate the load on the bone, IEC may be the result of endplate remodeling to adapt to the changed stress distribution under axial stress. On the T1 MRI scans at follow-up, we observed that the signal of the endplates with IH and TSN was continuous, so we considered these 2 kinds of healing forms as "malunion," which were often seen in patients with obvious displacement of the endplate fracture. Pedicle screws can reduce the height of the vertebral body but cannot directly reduce the endplate, leading to the often unsatisfactory reduction and secondary malunion of the endplate. TSNs had 2 forms in our observations: the first type was due to a grade 3 injury of the intervertebral disc. The nucleus pulposus broke into the vertebral body through the endplate. The endplate could not be reduced after surgery, so the nucleus pulposus tissue still remained in the vertebral body, and TSN formed after the endplate was healed. These TSNs were often irregular in shape in the endplate. The other type was that the trauma only caused a slight fissure fracture of the endplate. Under repeated stress stimulations, the nucleus pulposus tissue gradually herniated into the vertebral body through the weak area of the endplate (Fig. 2). The formation mechanism of these TSNs is similar to that of the degenerative Schmorl nodes, while Williams et al[20] found Schmorl nodes to be closely related to lumbar disc degeneration

Figure 2. A, The lateral view of a 59-year-old man with a L1 vertebral fracture due to the fall injury. B, The preoperative image on T1-weighed image. The white arrow represents a slight fissure fracture of the upper endplate. C, The postoperative image with open reduction and intermediate pedicle screw in lateral view. D, The follow-up image on T1-weighed image. The black arrow represents traumatic Schmorl node formation with a smooth and round outline. E, The follow-up image on T2-weighed image. The upper intervertebral disc was classified as Grade IIIby Pfirmann degeneration classification.

and low back pain, although these nodes were not independent predictors of low back pain.

Some limitations to this study must be mentioned. First, our study is lack of clinical evaluation of disc degeneration corresponding to endplate injury. Indeed, degenerative changes of the intervertebral disc do not necessarily appear to correspond to clinical symptoms, and the symptoms can occur for a few years or even more than 10 years.[21] The relatively short follow-up period (average, 28.2 months) may be used for explanation. The value of simple imaging changes without confirmed clinical relevance is limited. Second, because this study is a retrospective analysis of patient imaging data, some inherent limitations are inevitable, such as inaccuracy in data collection or recall bias for some clinical variables. Third, this is a single-center study with a small sample size and the findings need multicenter, prospective studies with a large sample size to confirm.

5. Conclusion

The normal anatomy and integrity of the endplate play an important role in providing biomechanical conduction, stress redistribution, and nutrition metabolism in the intervertebral disc. As the common forms of healing after vertebral fracture, IH and TSN are closely related to intervertebral disc degeneration. The existence and severity of the endplate injury can provide valuable information for individualized clinical decision-making processes.

Author contributions

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