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Authors

Berg-Johansen, Britta

Jain, Deeptee

Liebenberg, Ellen C

et al.

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Tidemark Avulsions are a Predominant Form of Endplate Irregularity

Britta Berg-Johansen, PhD, **Deeptee Jain, MD**, **Ellen C. Liebenberg, BS**, **Aaron J. Fields, PhD**, **Thomas M. Link, MD**, **Conor W. O'Neill, MD**, and **Jeffrey C. Lotz, PhD**

University of California, San Francisco

Abstract

Study Design—Descriptive histologic and MRI study of human cadaveric spines.

Objective—To identify and characterize common endplate pathologies to form a histologic foundation for an etiology-based classification system.

Summary of Background Data—Irregularities at the spinal disc-vertebra interface are associated with back pain and intervertebral disc herniation injuries. However, there is currently a lack of consensus regarding terminology for classification. This limits the potential for advancing understanding of back pain mechanisms, and prohibits meaningful comparisons for identifying priorities for prevention and treatment. Prior classification systems largely rely on observations from clinical imaging, which may miss subtle pathologic features.

Methods—Fifteen cadaveric spines with moderate to severe disc degeneration were obtained and scanned with MRI in the sagittal plane using two-dimensional T_1-weighted and T_2-weighted fast spin-echo sequences. Eighty-nine lumbar and lower thoracic bone-disc-bone motion segments were extracted, fixed, sectioned, and stained for histologic evaluation. Focal endplate irregularities were identified and categorized based on features that inferred causation. The presence, type, and anatomic location were recorded. A classification system with three major categories of focal endplate irregularities was created.

Results—Disc-vertebra avulsion and vertebral rim degeneration were more common than subchondral nodes: 50% of irregularities were classified as rim degeneration (75/150), 35% were classified as avulsions (52/150), and 15% were classified as nodes (23/150). Ninety percent of avulsions were sub-classified as "tidemark avulsions," a highly prevalent form of endplate irregularity in which the outer annulus separates from the vertebra at the tidemark. These tidemark avulsions have not been previously described, yet are visible on T2-weighted MRI as high-intensity regions.

Conclusions—This study provides histologic basis for a system to classify focal endplate irregularities. Included is a previously unidentified but prevalent finding of tidemark avulsions, which are visible with both histology and MRI. These observations will help clinicians better organize patients into meaningful groups to facilitate diagnosis, treatment, and clinical research.

Address for reprints and correspondence: Jeffrey C. Lotz, Ph.D., University of California, 513 Parnassus Avenue, S-1157, San Francisco, CA 94143-0514, United States, lotzj@orthosurg.ucsf.edu.

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Keywords

Endplate irregularity; lumbar spine; disc-vertebra interface; tidemark; avulsion; histology; MRI; high-intensity regions; high-intensity zone; disc herniation; Schmorl's nodes; rim degeneration; diagnostics; cartilage endplate; low back pain

Introduction

Endplate irregularities at the disc-vertebra interface are associated with back pain and injury; [1–4] however, studies on pathophysiology, natural history, and clinical significance are limited by a lack of consensus on terminology and classification. A pathologic definition and uniform classification for endplate damage is long overdue. In a recent survey of a working group from the International Society of the Lumbar Spine (ISSLS), approximately 84% of orthopaedic clinicians and 80% of orthopaedic researchers agreed that a standardized endplate nomenclature is clinically needed.[5] Standardized nomenclature will improve the accuracy and consistency of clinical diagnostics, thereby enhancing therapeutic decision-making.

A standard classification scheme for intervertebral disc degeneration is mandatory to advance clinical research in the prevention and treatment of chronic low back pain. Most disc degeneration classification schemes include sub-scores for the major sub-tissues: nucleus, annulus, and cartilage endplate. Of these, the endplate may be most clinically important, since endplate damage is primarily associated with pathologic disc innervation.[3]

Unfortunately, clinical classification of endplate damage is hindered by the use of different terminology to describe a single type of endplate irregularity; for example, separations at the annulus-vertebra interface have been described as “avulsions,” “cartilaginous herniations,” and “endplate junction failures.”[1–3,6–9] Conversely, the same term is often used to describe two or more different types of endplate irregularities; for example, nodes with traumatic expulsion of disc material vs. nodes showing more erosive morphologies are both termed “Schmorl’s nodes,”[10,11] despite evidence suggesting that they have very different etiologies. [12] Another example is the use of the term “rim lesion” to describe both bony lesions and annular lesions near the endplate junction.[13–15] These inconsistencies in terminology may contribute to the high proportion of clinical cases where chronic low back pain remains unexplained.

To address the need for standardized nomenclature, the aim of this study was to identify and classify focal endplate pathologies in a cadaveric population using histology. Rather than relying solely on clinical imaging, histology provides a high-resolution depiction of tissue features and structures involved, thereby providing the mechanistic underpinnings for future clinical classification.

Materials and Methods

Study Population and Imaging

Fifteen cadaveric spines with moderate to severe disc degeneration (11 male / 4 female, age range 49–67 years, mean 58.2 ± 5.8 years) were obtained from donors within 72 hours post-mortem (UCSF Willed Body Program) and scanned *in situ* in the sagittal plane with 3T MRI (GE Signa HDx scanner; GE Healthcare, Waukesha, WI, USA). All 15 spines were scanned using two-dimensional T_1-weighted and T_2-weighted fast spin-echo sequences, and 8 of the 15 spines were also scanned using a three-dimensional ultrashort echo-time (UTE) sequence. Imaging was used to exclude motion segments with prior surgery, fracture, or deformity (scoliosis, spondylolisthesis). Medial history information was not available for the donors.

Histologic Analysis

Following imaging, 89 motion segments (2 T10-T11, 4 T11-T12, 13 T12-L1, 14 L1-L2, 15 L2-L3, 13 L3-L4, 15 L4-L5, and 13 L5-S1) were extracted from the fifteen intact spines using a band saw. Motion segments were cut into 5- to 8-mm-thick parasagittal slabs, and ~1 cm thick parasagittal slabs from areas with known endplate irregularities were fixed in 10% formalin for histology. After fixation, slabs were decalcified with an ion-exchange decalcification solution (unit DCIEDLT, American MasterTech, Lodi, CA), embedded in paraffin, and cut into 7-μm thick sections. Several sections from each motion segment were stained using the following stains: Hematoxylin and Eosin (H&E) for observing overall morphology, Masson trichrome and Mallory-Heidenhain for visualizing connective tissue, including annular lamellae and their junction with the bony endplate, and Safranin O Fast Green for clear visualization of the cartilage endplate (CEP). Sections were imaged on a microscope with and without polarized light with 0.5X, 2X, 4X, 10X, and 20X objectives. Histology images were assessed by two raters. A classification scheme of endplate irregularities was generated, and incidence of each type was recorded. Only focal tissue-scale irregularities were recorded (i.e., overall endplate shape, cell shape, etc. were not considered). Anatomical location (anterior/posterior and inferior/superior) of each endplate irregularity was also recorded.

Based on a novel histology finding showing separations of the annulus from the vertebra at the tidemark – where outer annulus fibers insert into the calcified cartilage layer – we assessed innervation at these “tidemark avulsions.” Specifically, two specimens showing tidemark avulsions were immuno-stained for the general neuronal marker protein gene product 9.5 (PGP 9.5; AbD Serotec, Kidlingdon, United Kingdom) using a polymer detection system (MACH 4 HRP; Biocare Mdical). PGP-9.5 is present in nerves, and immuno-stained slides were assessed for the absence or presence of nerves in the bone adjacent to endplate irregularities.

MRI Analysis

MR images of each spine were examined for the presence or absence of tidemark avulsions, including anatomic location (anterior/posterior and inferior/superior). MR images were

assessed by two clinical orthopaedic specialists who were different from the raters used for histology and blinded from the histologic findings.

Results

Histology Classification

Based on location and patho-anatomical features, endplate irregularities were grouped into the following categories and subcategories:

- **I.** *Avulsion*: tissue separation at the disc-vertebra interface
  - **i.** Tidemark avulsion: separation along tidemark, where outer annulus fibers insert into the calcified cartilage layer (Figure 1A and Figure 2C&D)
  - **ii.** CEP-bone avulsion: separation between the cartilage endplate (CEP) and bone (Figure 1B)
- **II.** *Nodes*:
  - **i.** Traumatic: herniation of nucleus material through endplate (Figure 1C)
  - **ii.** Erosive: bony erosion and abnormal fibrocartilage ingrowth (Figure1D)
- **III.** *Rim degeneration*: degeneration of bone-annulus interface, including bone marrow changes and loss of annular fiber organization (Figure 1E)

Prevalence of Endplate Irregularities

Due to the purposeful selection of moderately to severely degenerated spines, 104 of the 178 total endplates (58%) had at least one type of endplate irregularity. Avulsions and rim degeneration were more common than nodes (Figure 1A): 50% of irregularities were classified as rim degeneration (75 total), 35% were classified as avulsions (52 total), and only 15% were classified as nodes (23 total). Avulsions and rim degeneration occurred most frequently in the lumbar spine, while nodes occurred most frequently in the thoracic spine (Figure 2B). Linear regression showed that the number of cases of each endplate irregularity did not correlate significantly with age ($p > 0.3$).; however, the three oldest spines (ages 63, 65, and 67) had the highest numbers of tidemark avulsions (11, 7, and 8, respectively). Often, similar endplate irregularities were observed in multiple discs of the same spine; for example, 20 of 23 nodes were distributed amongst only 5 of the 15 spines, with 7 nodes occurring in a single spine (Figure 3; A,B,C&D).

Ninety percent of avulsions were tidemark avulsions (Figure 2). Annulus fibers were observed to change direction upon crossing the tidemark (Figure 2A&B). Tidemark avulsions occurred in 35 of 89 discs, and of those discs, 14 had multiple avulsions. Eighty-seven percent of all avulsions occurred anteriorly.

Tidemark Avulsions as High-Intensity Regions on T_2-weighted MRI

Tidemark avulsions were visible on T_2-weighted MRI as linear high-intensity regions in the corners (Figure 4). It was noted that even small tidemark avulsions were visible, and in fact,

appeared larger on MRI than on histology. Tidemark avulsions were not pronounced on T_1-weighted and UTE MRI (Figure 5).

Increased Innervation at Tidemark Avulsions

Vertebral bone adjacent to endplate irregularities showed marrow changes and increased innervation, as observed in previous work.[3] Even small tidemark avulsions showed increased nerve density in adjacent bone (Figure 6; A,B,C&D).

Discussion

The design of any classification system depends, in large part, on its goals. One is to organize the many chronic low back patients into subsets for better diagnosis and treatment planning. Another is to infer etiology as a basis for clarifying pathogenesis, risk factors, and designing new therapies. This current study uses histology as the gold-standard, and describes three primary forms of endplate damage: avulsions, nodes, and rim degeneration. The histologic features infer etiology, and include a mix of biomechanical features (avulsions and traumatic nodes) and biological features (erosive nodes and rim degeneration).

While rim lesions are well-documented,[15,16] the precise failure location of annulus fibers along the tidemark has only been described in biomechanical studies of excised animal tissue[7] and has not been previously described in human spines. Several prior studies have described the tidemark in human spines,[17,18] which is also an important feature of tendon-bone insertions[19] and articular cartilage-bone interfaces.[20] Around the periphery of the disc, the cartilage endplate is calcified throughout its entire thickness, and the tidemark lies between the annulus and this calcified cartilage. In the central portion of the disc, the cartilage endplate is mainly uncalcified hyaline cartilage, and the tidemark joins the hyaline cartilage and a very thin or indiscernible layer of calcified cartilage (Figure 7).[6,18] Our observation that annulus fibers change direction upon crossing the tidemark agrees with previous observations.[17,6]

The fact that tidemark avulsions can be seen with MRI has potential clinical significance. Damage at the disc-vertebra interface has been linked with both pain and injury,[1–4] and our current data shown that even minor separations at the disc-vertebra interface are visible on T_2-weighted MRI as areas of linear high intensity. This raises the potential for disc-vertebra interface injuries to be identified and targeted with specific prospective treatments. The corresponding high-intensity T_2 MRI findings suggest that avulsion defects collect fluid. Alternatively, gas may accumulate within the separation from surrounding extracellular spaces, much like the “vacuum phenomenon” that has been observed in disc spaces and noted as high-intensity zones on T_2-weighted MRI.[21,22] We noted that tidemark avulsions appeared larger on MRI than histology, which may be explained by fluid and/or gas collections being lost during *ex vivo* sectioning. The presence of high intensity regions on MRI coincident with the annular avulsions makes it unlikely they are artifacts of histologic preparation, and demonstrates the potential for clinical utility.

The term “high intensity zone (HIZ)” for spinal MRIs typically refers to areas of high signal intensity within the posterior annular substance on T_2-weighted MRI, and often reflects annular fissures.[23,24] The relationship between these annular HIZs and back pain is controversial. Early studies found that HIZs may be a highly-specific signal for back pain, [25,26] whereas recent work has shown that HIZs are prevalent in both symptomatic and asymptomatic individuals.[117] The high-intensity regions observed in the current study differ from the classically-observed annulus HIZs in that they are located at the peripheral disc-vertebra interface. We have shown that these interfacial high-intensity regions associate with delamination injury as seen with histology. Because of the aforementioned associations between endplate defects and back pain, these high-intensity regions may represent painful lesions. Furthermore, recent work has found that the Oswestry Disability Index (ODI) score, an index used to quantify disability for low back pain, was significantly higher for discs with abnormally elevated anterior disc widening during dynamic flexion/extension imaging.[29] Anterior disc widening may, in theory, be caused by tidemark avulsions allowing the disc to detach and separate from the vertebra. This agrees with our findings that the majority (87%) of tidemark avulsions occurred anteriorly.

The mechanism of failure at the tidemark remains unclear. A biomechanical study of excised ovine tissue found that disc-bone specimens were more likely to fail at the tidemark in axial tension than in torsion or in-plane tension, and hypothesized that this may be due to acute fiber bending where the annulus fibers enter the calcified cartilage.[7] This suggests that tidemark failures may occur during bending, putting the annulus is tension. Despite our finding that the majority of tidemark avulsions occur anteriorly, traumatic avulsions coincident with disc herniation (not included in our study population) often occur posteriorly, with the inner annulus avulsing the cartilage endplate during forward bending. Our previous work has shown that human bone-disc-bone specimens pulled in axial tension to failure also occur at the inner annulus region, with cartilage endplate being stripped from underlying bone.[30] Based on the current study, the avulsion injuries induced in the previous study may occur specifically at the tidemark between the hyaline cartilage endplate and thin calcified cartilage layer, as supported by SEM results showing that cartilage-like tissue was present on both failure surfaces.[30] Various other studies have also observed cartilage endplate-bone avulsions,[1,2,7,31] and further studies are needed to confirm with high-resolution imaging whether these avulsions occur at the tidemark.

The high prevalence of tidemark avulsions at the outer annulus without associated disc herniation identified in the current study suggests that this injury has a different mechanism than the traumatic cartilage endplate-bone avulsions induced in our prior work. One possibility is that outer annulus tidemark avulsions are fatigue injuries from repetitive loading, while the inner annulus cartilage endplate-bone avulsions associated with disc herniations[1,2] may be a traumatic injury (e.g. during excessive forward bending). Further studies are needed to test this hypothesis, such as biomechanical studies of the tidemark under fatigue loading.

This study has several limitations. First, we performed MRI on cadaveric spines using higher resolution than typical clinical MRI. It is possible that tidemark avulsions may be more difficult to discern clinically due to limited image resolution. Thus, further studies are

needed to confirm the visibility of tidemark avulsions with clinical MRI. Additionally, we retrospectively analyzed histology slides and MR images collected over the past several years that were not co-registered, and were therefore unable to match the exact location of histology slices with MRI slices. Despite this limitation, we were able to approximately match locations based on disc and endplate morphologies.

The results of this current study provide important new information regarding anatomic features of endplate damage, and may help overcome the challenges and controversies associated with achieving consensus on classification language. While we believe that these new observations will be valuable for standardization, future studies are required to demonstrate that the histologic features of this system are easy to identify with routine clinical imaging with high sensitivity, specificity, and inter-observer agreement. Additionally, prospective studies in a population-based cohort are required to validate clinical utility.

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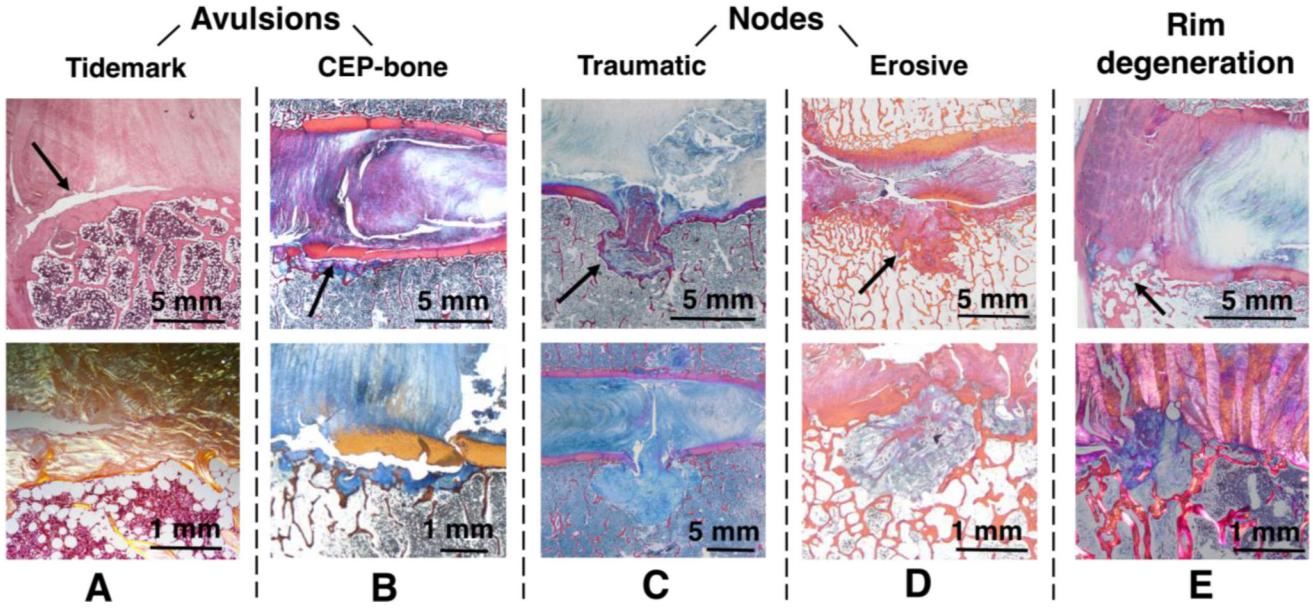

Figure 1.

Categories of endplate irregularities as seen with histology: (A) Tidemark avulsion at the insertion of annulus fibers into the calcified cartilage layer; (B) CEP-bone avulsion; (C) traumatic node; (D) erosive node; (E) rim degeneration. Top images are lower magnification, with arrows pointing to endplate irregularities. Sections in (A) are stained with Hematoxylin and Eosin (top) and Safranin O (bottom), while sections in (B–E) are stained with Mallory-Heidenhain trichrome stain. Bottom images of (A) and (E) are taken under polarized light.

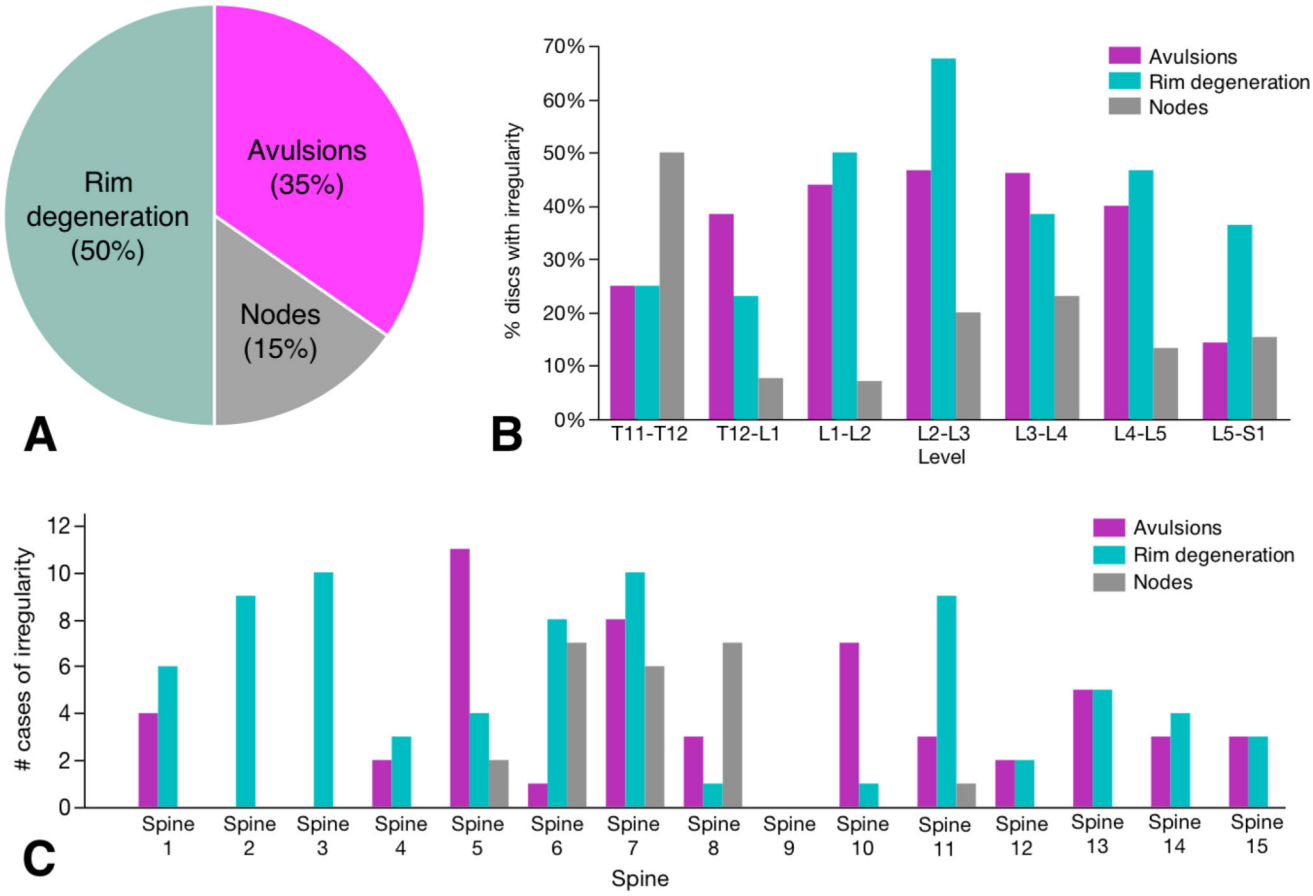

Figure 2.

(A) distribution of endplate irregularities by category; (B) distribution of endplate irregularities by disc level; (C) distribution of endplate irregularities by individual, demonstrating a single category of irregularity often occurring multiple times in a given spine.

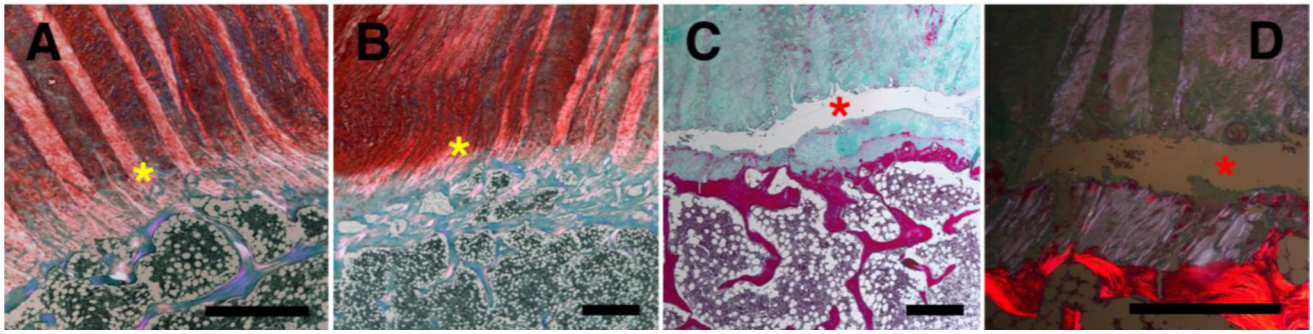

Figure 3.

Histology images of (A&B) intact tidemarks, denoted by yellow asterisks, and (C&D) tidemark avulsions, denoted by red asterisks. Note that collagen fibers change direction at the tidemark (A&B). Sections are stained with (A,B,D) Hematoxylin and Eosin under polarized light, and (C) Masson Trichrome. Scale bars represent 1 mm.

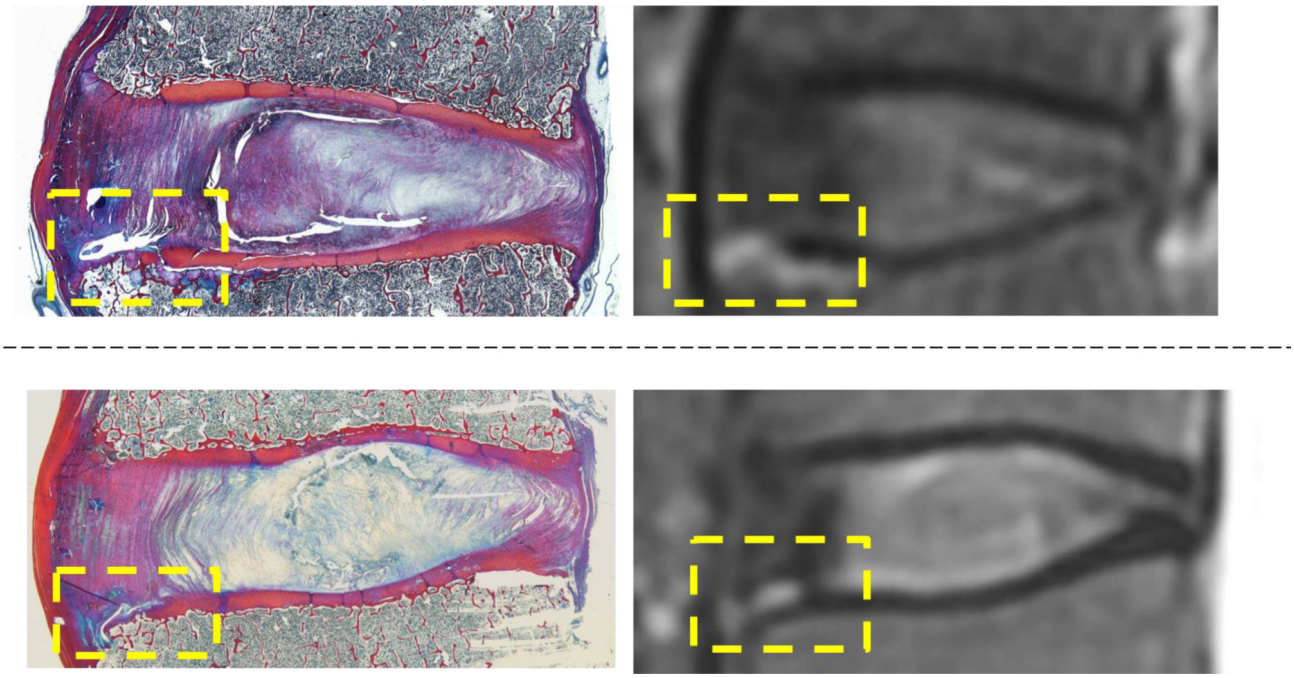

Figure 4.
Tidemark avulsions on histology (left) and T2-weighted MRI (right). Tidemark avulsions appear as high-intensity regions on MRI.

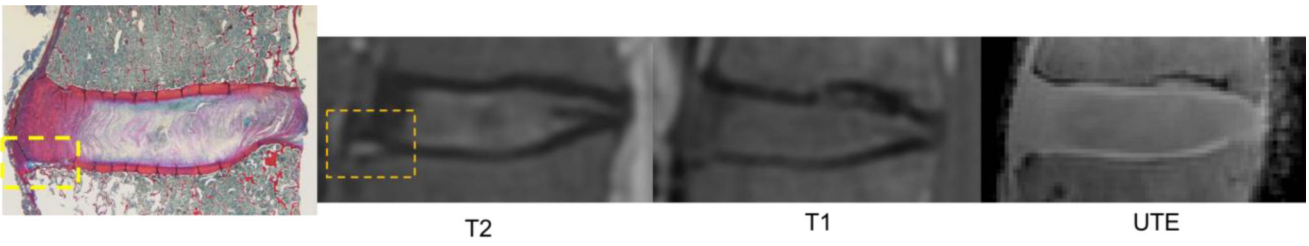

Figure 5.
Tidemark avulsions on histology, T2-weighted MRI, T1-weighted MRI, and UTE. A high-intensity region is visible on T2-weighted MRI. This finding is not pronounced on T1-weighted MRI and UTE.

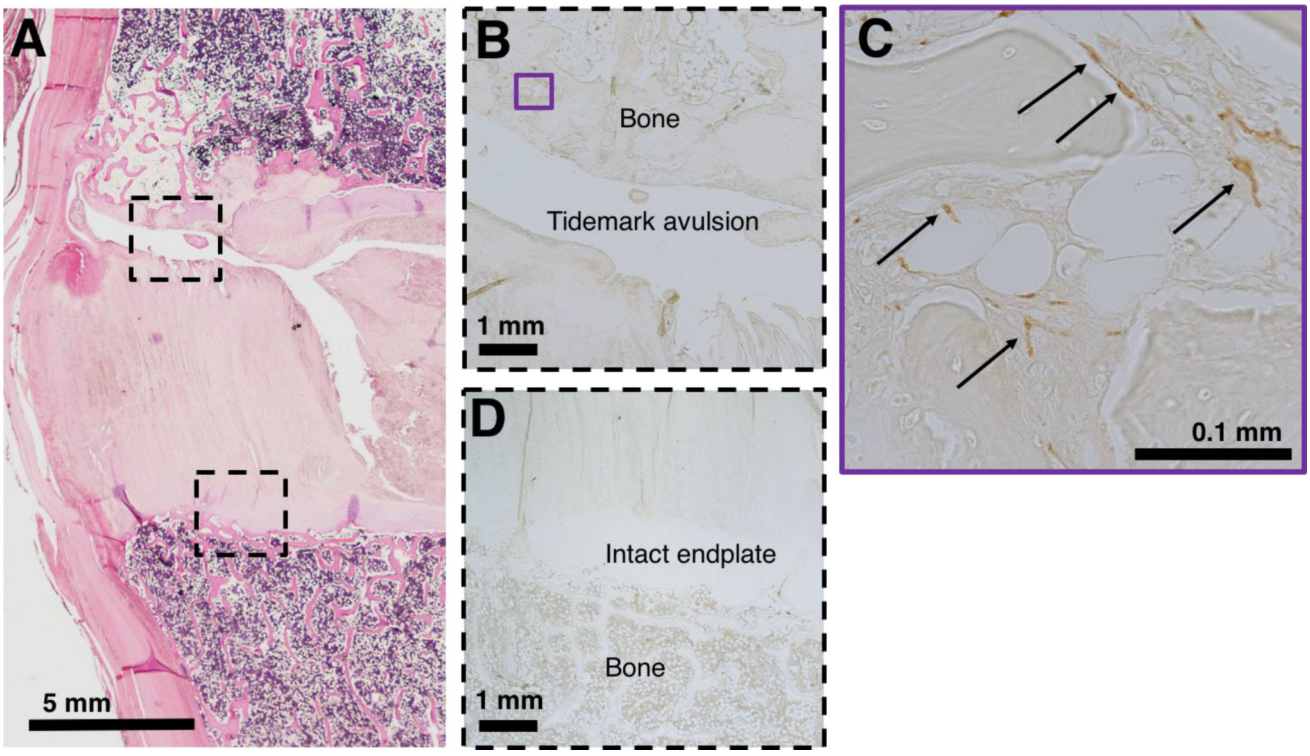

Figure 6.

Tidemark avulsions shows increased innervation in adjacent vertebral bone. (A) Disc with tidemark avulsion at superior endplate, and healthy/intact inferior endplate; (B) 2X image of superior tidemark avulsion and adjacent vertebral bone (top boxed region in A); (C) 40X image of vertebral bone adjacent to tidemark avulsion (boxed region in B) with arrows pointing to protein gene product (PGP) 9.5-positive nerves, visible as dark spots using immuno-staining method; (D) intact inferior endplate (bottom boxed region in A) with no visible nerves.

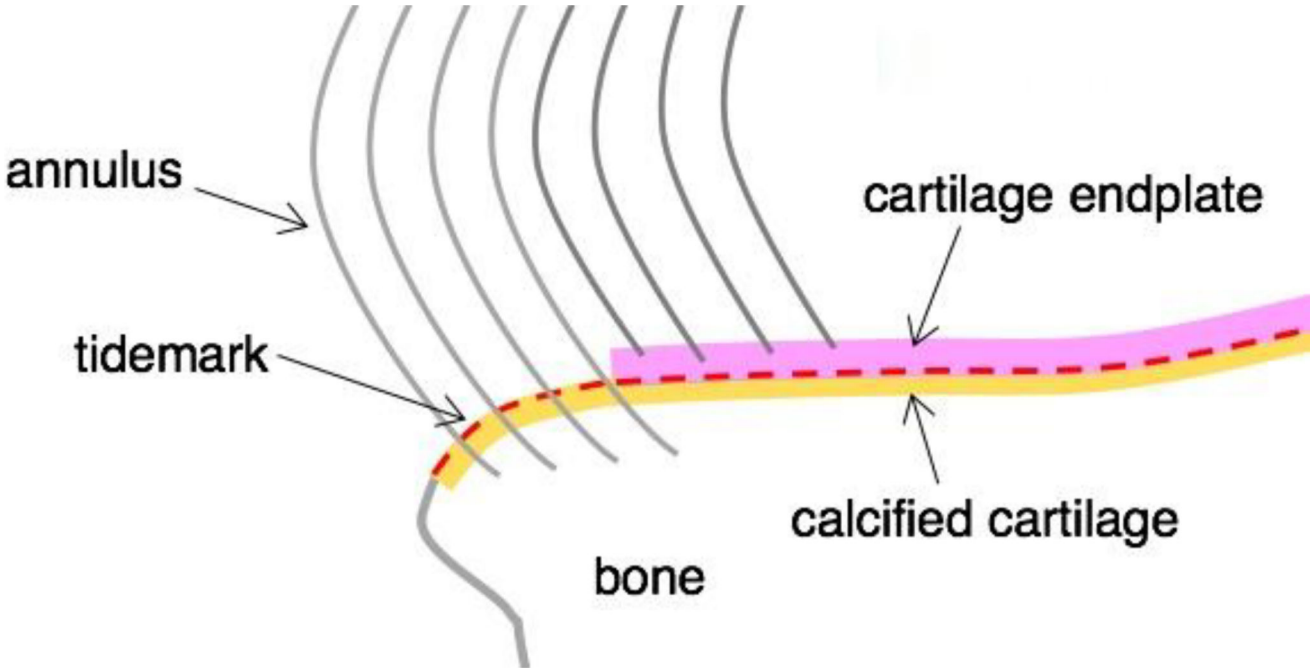

Figure 7.

Tidemark anatomy. In the outer annulus, fibers cross the tidemark (red dotted line) and enter the calcified cartilage layer (yellow line). In the inner annulus, fibers enter the hyaline cartilage endplate (pink line), and the tidemark sits between the cartilage endplate and calcified cartilage layer. The calcified cartilage layer is thicker at the outer annulus and thinner at the inner annulus.