FRACTURE-ASSOCIATED AND IDIOPATHIC SUBCHONDRAL VERTEBRAL LESIONS

MRI and Histopathology of subchondral vertebral lesions that are idiopathic or that develop with vertebral fractures.

Lesions due to growth disturbance were excluded. Radiographs and MR images were analyzed in consensus by two radiologists, and sampled specimens were analyzed by a pathologist.

Results Eleven idiopathic and ten fracture-associated vertebral lesions were available.
On T1-weighted images, all lesion signal intensity was low and homogeneous.
On T2weighted images, all idiopathic lesions showed a heterogeneous signal with a central low or intermediate signal component and a peripheral high or intermediate component.

All but one fracture-related lesions showed a homogeneous intermediate to high signal intensity.
Histological analysis of idiopathic lesions showed a central acellular fibrous connective tissue in all cases surrounded

Introduction
Schmorl's Node : could occur not only during growth— probably due to weakness of the cartilaginous vertebral end plates—but also with spontaneous or posttraumatic vertebral end plate fractures and with degenerative changes of the discovertebral junction.
Nowadays, there is a general agreement on the fact that symptomatic Schmorl's nodes develop in vertebral bodies from adults or elderly patients without degenerative disk disease or without obvious vertebral end plate deformity suggestive of previous growth disturbance or vertebral end plate fracture.

1. Fresh human cadavers to describe the MR imaging and histological features of two patterns of subchondral vertebral lesions: (a) subchondral vertebral lesions associated with obvious vertebral end plate fracture (later called fracture-associated subchondral vertebral lesions) and (b) subchondral vertebral lesions without vertebral end plate deformity and/or without degenerative disk disease (later called idiopathic subchondral vertebral lesions).

Inclusion criterion was the presence of a subchondral lucent defect with at least 3mm diameter that involved a vertebral end plate with normal adjacent disk on section radiographs. Lesions smaller than 3 mm and lesions adjacent to disk with degenerative disease defined by loss of disk height of at least 20% with respect to adjacent disks were excluded.

Fracture-associated subchondral vertebral lesion was defined as a subchondral lucent defect that developed in vertebral bodies at the apex of fractured and displaced vertebral end plates.

Idiopathic subchondral vertebral lesion was defined as a subchondral lucent defect that developed in vertebral bodies in which end plates lacked signs of growth disturbance, fracture, or degenerative disk disease. Fracture-associated and idiopathic subchondral vertebral lesions were selected for this study. Subchondral lesions that were associated with signs of growth disturbance, degenerative disk disease, or bone metastases were not included in the current study.

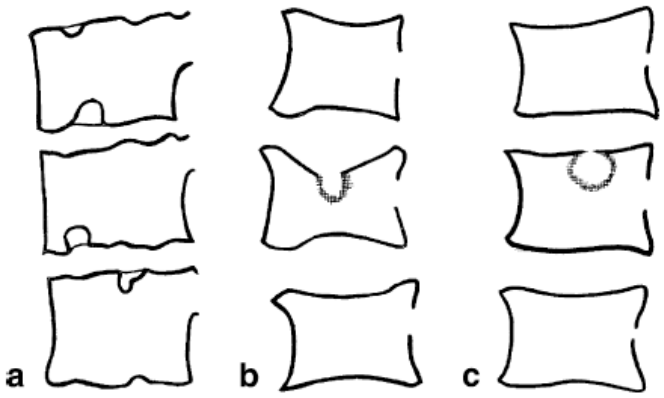


Fig. 1 Schematic drawings illustrating three patterns of subchondral vertebral lesions. a Subchondral vertebral lesion associated with growth disturbance; b subchondral vertebral lesion associated with vertebral end plate fracture; and c idiopathic subchondral vertebral lesion

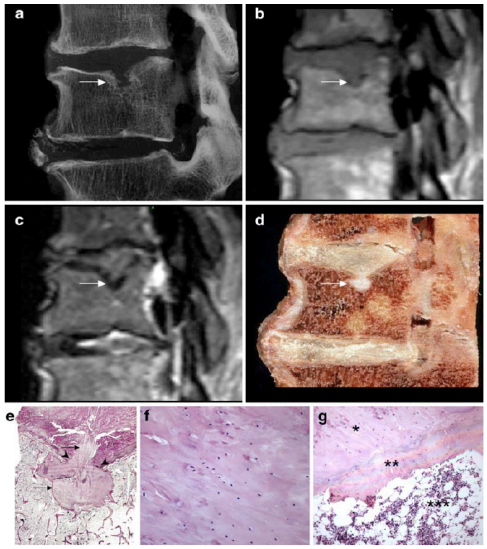
On T1-weighted images, signal intensity of the subchondral lesion was considered to be low or intermediate when it was lower than or equivalent to that of the normal marrow.

On T2-weighted images, signal intensity of the subchondral lesion was considered to be either homogeneous or heterogeneous depending on the presence of only one or at least two signal intensity patterns within the lesion.

Signal intensity of the lesion was considered to be low, intermediate, or high when it was lower than, equivalent to, or higher than that of the normal adjacent disk. The subchondral bone plate was considered to be continuous or interrupted. Presence or absence of the low signal intensity intradiscal cleft was assessed and, when present, its orientation was determined (horizontal or flipped toward the vertebral

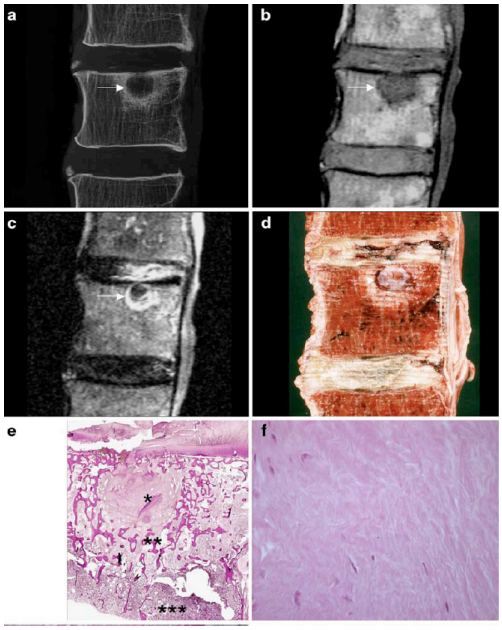
lesion). Adjacent marrow signal pattern was not assessed on T2-weighted images.

End plate fracture



1. X ray: Small defect at the apex of the deformity
2. T2: Homogenous and intermediate
3. Histology: Fibrocartilage tissue with reactive tissue

Idiopathic endplate lesion



X ray: No depression at the subchondral plate

T2: Nonhomogeneous

Hisotology: Central dense collagen tissue surrounded by the peripheral zone of loose firbous tissue with interstial oedema. Rim of reactive tissue. C

Discussion

Subchondral vertebral lesions have been observed in vertebral bodies without degenerative adjacent disk disease in three distinct clinical conditions.

I **Subchondral vertebral lesions that develop during Growth**

Represent disk material herniation favored by a relative or absolute weakness of the cartilaginous vertebral end plate during that period

These lesions occur in patients with radiological signs of growth disturbance in the spine including altered vertebral shape and abnormal vertebral end plate contours at distance of the lesions.

II **Fracture-associated subchondral vertebral lesions**

Correspond to defects that develop at the apex of acute spontaneous or posttraumatic fractures of the vertebral end plate, probably due to migration of disk material through an acute defect of the vertebral end plate.

III **Idiopathic subchondral vertebral lesions**

Observed in patients without radiological and MR signs of growth disturbance, vertebral end plate deformity, or degenerative disk disease.

The origin of these idiopathic subchondral vertebral lesions remains unclear and could result from intra-osseous disk material migration, bone and marrow necrosis, or degenerative cartilaginous end plate lesions.

A better knowledge of the detailed imaging findings observed in these lesions that are presumably secondary to disc or subchondral bone plate disorders could also contribute to the differential diagnosis from lesions secondary to bone marrow disorders, e.g., metastases or multiple myeloma.

First, fracture-associated and idiopathic subchondral vertebral lesions demonstrated different signal intensity patterns on T2-weighted SE images. Fracture-associated subchondral lesions demonstrated a homogeneous signal intensity pattern with intermediate to high signal intensity on T2-weighted SE images. Idiopathic subchondral vertebral lesions demonstrated a heterogeneous signal pattern on T2-weighted SE images that consisted in a central area of low to intermediate signal intensity and a peripheral zone of high signal intensity.

Second, fracture-associated and idiopathic subchondral vertebral lesions demonstrated different histological features. Fracture-associated lesions contained fibrocartilaginous material similar to that of

the adjacent disk material. This finding was consistent with the generally accepted hypothesis to explain their origin. Most likely, subchondral vertebral lesions observed near vertebral end plate fractures are related to disk material migration through the fracture-related defect of the subchondral bone plate. Idiopathic subchondral vertebral lesions consisted in central compact and peripheral loose connective tissue. Our observation is consistent with previous observations from biopsy specimens

Finally, adjacent bone marrow appeared normal on T1weighted spin-echo images in 40% of fracture-associated lesions and in 45% of the idiopathic lesions. Adjacent bone marrow showed decreased signal intensity on T1-weighted images in 20% of fracture-associated lesions and in none of the idiopathic lesions. Adjacent bone marrow showed

In conclusion, the current study showed that fracture-associated and idiopathic subchondral vertebral lesions had different MR and histological appearance. A target organization is observed in idiopathic subchondral vertebral lesions, both on T2-weighted SE MR images and at histology.

Current thinking

Endplate lesions are associated with back pain as well as being closely associated with adjacent DD, with a clear dosage effect. Different types of endplate lesions seem to have different magnitudes of associations with DD. Lumbar endplate lesions may be an important key to better understand both DD and back pain.

Disc degeneration (DD) has long been suspected of playing an essential role in the pathogenesis of back pain and correspondingly the disc is often targeted in medical intervention and scientific research.

Yet, evidence yielded from decades of research shows that the association between DD findings and back pain is generally weak, challenging the traditional view that the disc is the primary back pain generator.

Unlike the poorly innervated intervertebral disc, the adjacent bony vertebral endplate and vertebral body are well supplied by intraosseous nerves and may be another source of back pain.

However, endplate lesions and their associations with DD and back pain have received relatively little attention.

Subsequently, we identified 4 types of endplate lesions, including Schmorl's nodes, fracture, erosion, and calcification lesions.

Endplate lesions are closely associated with adjacent DD, with greater number and greater size of lesions associated with more severe adjacent DD. In addition, evidence suggests that different endplate lesions may have different pathogenic origins, distinct pathological characteristics, and thus, varied magnitudes of pathological influences on the adjacent disc.

Schmorl's nodes, the most common endplate lesions, were thought to be painful. The association between Schmorl's nodes and back pain was not confirmed in a large population-based epidemiological study.

Using clearer definitions and more accurate measurements acquired from cadaveric endplates, compared with radiological studies, this study clarified the important role of endplate integrity in maintaining disc health: endplate lesions of any type were associated with DD. In addition, a greater number of endplate lesions and larger size were associated with more severe adjacent DD. Such a dosage effect, which has been observed previously.

The disruption of endplate integrity may trigger a series of pathological cascades which eventually result in adjacent DD. First of all, the protrusion of the nucleus pulposus into the vertebral body causes a direct loss of nucleus matrix contents, such as water and proteoglycan, leading to DD.
Associated inflammatory and autoimmune reactions could further destroy the homeostasis within the disc and impair the metabolism of the cells.
Second, endplate lesions and the accompanying reparatory reactions may damage or block the marrow contact channels and impede nutrient supply to the disc.

In addition, endplate lesions alter the distribution of matrix compressive stress in the adjacent disc, which may further inhibit disc cell metabolism and lead to progressive structural failure of the disc.

On the contrary, a common association with back injury history suggest that fracture and erosion lesions may share a common origin, such as trauma. In the case of endplate trauma, in addition to consequent inflammation the trauma itself could directly induce disc cell apoptosis and promote DD, resulting in more severe degenerative changes in the adjacent disc than seen with Schmorl's nodes.
It seems necessary to differentiate Schmorl's nodes from other endplate defects. Schmorl's nodes usually

are viewed as synonymous with endplate defects, especially in radiological studies.

Traditional Schmorl's nodes are typically small, involving the endplate center and having modest associations with disc pathology. More commonly affecting the anterior and lateral portions of the endplate, fractures and erosions, which could have been labeled as "traumatic Schmorl's nodes," are relatively large and extensive and tend to have a stronger association with adjacent DD.

To summarize, bony endplate lesions are associated with frequent low back pain. Endplate lesions are closely associated with adjacent DD, with a dosage effect, suggesting that the integrity of the vertebral endplate is important to maintain disc integrity. In addition, different types of endplate lesions may vary in their pathological influence on adjacent discs.

Key Points

- Endplate lesions are associated with back pain history, suggesting that the lesions may be a source of common back pain. This association remained after controlling for DD.
- Endplate lesions are closely associated with adjacent DD, with a dosage effect.
- Different types of lumbar endplate lesions have different pathological features and varying degrees of association with DD, suggesting that they may have different pathogenic origins and effects.
- It is important to differentiate Schmorl's nodes from other morphological endplate defects to further knowledge of either.

References

1.Skeletal Radiology, 2009, Volume 38, Number 3, Pages 245-253

2. SPINE Volume 37, Number 17, pp 1490–1496

3. Schmorl G, Junghans H, Doin G. Clinique et radiologie de la colonne vertébrale normale et pathologique. In: Doin G, editor. Lésion du rachis osseux. Paris: Flammarion; 1956. p. 41–207.

4.Resnick D, Niwayama G. Intravertebral disk herniations: cartilaginous (Schmorl's) nodes. Radiology 1978; 126(1): 57–65.

5. Wang Y , Videman T , Battie MC . Lumbar vertebral endplate lesions: prevalence, classifi cation and association with age . Spine (Phila Pa 1976) 2012 ; 37 : 1432 – 9 .