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Hypointense signal lesions of the articular cartilage: a review of current concepts☆

B. Keegan Markhardt [a,*], Eric Y. Chang [b,c]

[a] Department of Radiology, Community Division, University of Wisconsin, Madison, WI
[b] Department of Radiology, VA San Diego Healthcare System, San Diego, CA
[c] Department of Radiology, University of California, San Diego Medical Center, San Diego, CA

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ABSTRACT

Discussion of articular cartilage disease detection by MRI usually focuses on the presence of bright signal on T2-weighted sequences, such as in Grade 1 chondromalacia and cartilage fissures containing fluid. Less emphasis has been placed on how cartilage disease may be manifested by dark signal on T2-weighted sequences. The appearance of the recently described "cartilage black line sign" of the femoral trochlea highlights these lesions and further raises the question of their etiology. We illustrate various hypointense signal lesions that are not restricted to the femoral trochlea of the knee joint and discuss the possible etiologies for these lesions.

1. Introduction

Many clinical radiologists and researchers rely solely on fluid-sensitive sequences, whether intermediate-weighted or T2-weighted, to assess articular cartilage [1,2]. Increased signal intensity on fluid-sensitive sequences creates the familiar contrast between diseased and normal cartilage [3]. For example, cartilage swelling and increased signal has been correlated with the arthroscopic diagnosis of Grade 1 chondromalacia, and cartilage fissures and defects are manifested by joint fluid extending into cartilage. Less emphasis has been placed on how cartilage disease may be manifested by decreased signal on T2-weighted sequences.

There has been renewed discussion in the literature exploring the meaning of decreased signal with the recent identification of what has been termed the *cartilage black line sign* of the central femoral trochlea by Stevens et al. [4]. This lesion is defined as a linear, low-signal lesion on T2-weighted sequences, oriented perpendicular to the subchondral bone, which proved to represent a deep cartilage cleft at arthroscopy (two patients) and at computed tomography (CT) arthrography (one patient). Broderick et al. [5] and Konig et al. [6] first described linear regions of perpendicularly oriented, low T2 signal abnormality to represent cartilage degeneration at arthroscopy and biopsy, respectively. In distinction to these other studies, Stevens et al. suggest that the cartilage black line sign is not likely to be related to chondrosis, as these lesions represent actual linear fissures, which the arthroscopists could probe, and not merely degenerative change [4].

Wissman et al. [7] studied the central femoral trochlea lesion further by examining the arthroscopic findings of eight patients and found that all but one cartilage lesion was occult on arthroscopy. The authors suggested that if the superficial layer were to remain intact over a cartilage fissure, this would explain why the MRI abnormality does not fill with fluid and why it is not seen at arthroscopy in their experience. Moreover, they suggest that these hypointense signal lesions might relate to deep, incomplete cartilage fissures that do not frequently communicate with the joint.

Recent research has revealed several possible etiologies for the cartilage black line sign and other hypointense signal lesions of the articular cartilage, some of which do not require there to be a fissure at all. In this article, we further explore the probable etiologies of dark or hypointense signal cartilage lesions and provide a case series to illustrate how such lesions are not limited to the femoral trochlea.

2. Cases

3. Discussion

The etiology of hypointense signal lesions in articular cartilage has not been precisely determined. Although the precise etiology may be

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* Corresponding author. Meriter Hospital Department of Radiology, 202 S. Park Street, Madison, WI 53715. Tel.: +1 608 347 5001; fax: +1 608 417 7959.
E-mail addresses: keegan.markhardt@gmail.com (B.K. Markhardt), ericchangmd@gmail.com (E.Y. Chang).

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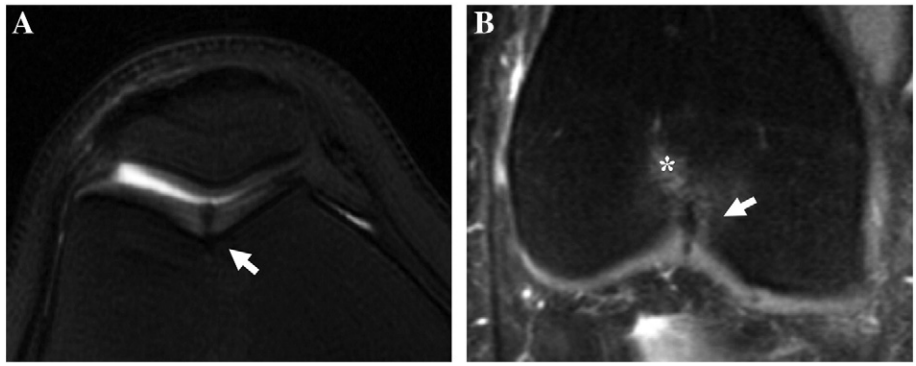

Fig. 1. Two cases of the cartilage black line sign (arrows) in the central aspect of the femoral trochlea in (A) a 25-year-old and (B) a 47-year-old woman with knee pain. Subchondral marrow reactive changes are frequently noted with these lesions (asterisk). Sequences are axial (A) and coronal (B) T2-weighted fat-sat (T2FS).

uncertain, there is consensus in the literature that such lesions indicate cartilage disease. The major factors determining the signal intensity of articular hyaline cartilage are anisotropy, magnetization transfer, water content, truncation artifact, and chemical shift artifact. In addition, after an osteochondral injury, there may be introduction of fibrocartilage, resulting in alterations of signal intensity. Considering the different contexts in which hypointense signal lesions are seen, the etiology of a specific lesion is more likely related to some of these factors and less likely to be related to others.

The directional orientation or anisotropy of articular cartilage architecture is a major factor in its MRI appearance [8]. Articular cartilage contains a relatively small number of chondrocytes (4% by weight), and the bulk of the tissue consists of an extracellular matrix composed of water (65–85%), collagen (10–20%), and proteoglycans (5–10%) [9,10]. Freeze-fracture sectioning and scanning electron microscopy reveals columnar organization of the extracellular matrix with fibrous structure that radiates from the subchondral bone and curves through an arc of 90°, like blades of grass [11–14]. This fibrous organization is oriented to the plane of stresses applied, and the structure of the extracellular matrix changes from a more vertical organization in the central region of the articular surface to a more oblique orientation at the margin of the joint [11,12] (Fig. 8A). It is important to note that the anisotropy of cartilage is physically apparent at all levels, and there is no random or isotropic layer [12,15].

The gradual change in shading of signal intensity seen in MRI of normal articular cartilage is related to changes in this anisotropy relative to the plane of the main magnetic field (B_0). As the orientation of these fibrous structures is tilted away from the direction of B_0, increasing prolongation of T2 signal occurs due to proton dipole–dipole interactions [16–19]. Maximal T2 prolongation is observed at approximately 54.7°, the so-called *magic angle*. As relative fiber orientation progresses beyond 54.7°, the T2 prolongation effect abates. These effects explain why that at the margin of a joint, where the matrix has a more oblique orientation, there are changes in T2 signal [15]. For example, in the knee there is commonly increase in T2 signal at the posterior aspect of the weight-bearing portion of the femoral condyle, and decrease in signal is seen at the superior pole of the patella and inferior aspect of the femoral trochlea (Figs. 3a and 4b). Moreover, the reported T2 values of normal cartilage range from 30 to 70 ms, depending on the depth of cartilage; however, because of magic angle effect the T2 signal in the same region of healthy cartilage can vary by as much as 80% depending on its orientation to B0 [19,20].

Articular cartilage injury by its very nature is sure to disrupt fiber anisotropy. This could result in either disorganization with loss of anisotropy or localized change in anisotropy orientation (Fig. 8B, C). Either change could explain a hypointense signal lesion. Loss of anisotropy could result in loss of the normal increased chondral signal related to magic angle effects. More specifically, disruption of the anisotropy of cartilage could decrease the proton dipole–dipole interactions and prevent the normal T2 prolongation (Fig. 8B). Alternatively, rather than loss of anisotropy, it is possible that direct or adjacent cartilage injury results in the orientation change of relatively normal cartilage (Fig. 8C). Such findings are consistent with our experience, where areas of hypointense signal are not infrequently seen adjacent to fluid-filled fissures and defects (Figs. 2, 3, 4 and 5). This hypothesis is supported by ex vivo and in vivo cartilage compression studies. Ex vivo micro-MRI studies have shown progressive decrease in T2 signal in the transitional zone with higher degrees of cartilage compression that is dependent on the cartilage angle relative to B0 and therefore related to anisotropy [21,22]. Diffusion tensor imaging and

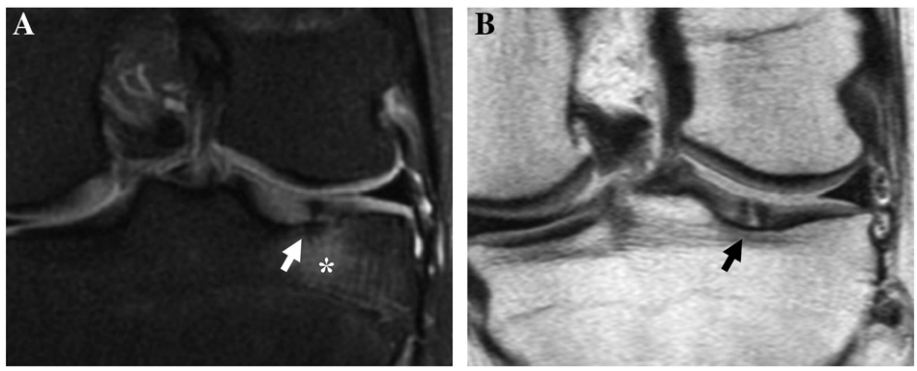

Fig. 2. Two cases of hypointense, perpendicular cartilage lesions involving the lateral tibial plateau (arrows) viewed in the coronal plane: (A) a 19-year-old male soccer player with lateral pain shows subchondral bone marrow reactive changes (asterisk), and (B) a 39-year-old man with 2 months of continued knee pain after running injury shows hypointense signal lining a deep fluid-filled fissure. Sequences are coronal T2FS and Proton Density (PD), respectively.