Nomenclature of Subchondral Nonneoplastic Bone Lesions

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OBJECTIVE. The purpose of this article is to summarize the nomenclature of nonneoplastic conditions affecting subchondral bone through a review of the medical literature and expert opinion of the Society of Skeletal Radiology Subchondral Bone Nomenclature Committee.

CONCLUSION. This consensus statement summarizes current understanding of the pathophysiologic characteristics and imaging findings of subchondral nonneoplastic bone lesions and proposes nomenclature to improve effective communication across clinical specialties and help avoid diagnostic errors that could affect patient care.

Many conditions can manifest as subchondral nonneoplastic bone lesions. Increased use of MRI and awareness of the underlying pathophysiology necessitate a uniform nomenclature of these lesions. However, there is controversy about not only the nomenclature but also the pathophysiologic characteristics of these lesions. The purpose of this white paper is a comprehensive review of the nomenclature of nonneoplastic conditions affecting the subchondral bone through a review of the medical literature and the expert opinion of the Society of Skeletal Radiology (SSR) Subchondral Bone Nomenclature Committee. The white paper is divided into two parts. The first part focuses on the nomenclature of descriptive terms and the second part on clinicopathologic entities affecting subchondral bone that includes a brief description of each lesion and clinicopathologic entity, a summary of current terms and pathophysiologic characteristics, current controversies, and a recommendation for the appropriate nomenclature.

The Practice Guidelines and Technical Standards Committee of the SSR identified nomenclature for subchondral nonneoplastic bone lesions as a topic for the white paper and selected members of the SSR to compose the Subchondral Bone Nomenclature Committee. This committee of 12 musculoskeletal radiologists with expertise in musculoskeletal imaging and evidence-based medicine was tasked with developing a consensus on nomenclature of subchondral nonneoplastic bone lesions. Several conference calls and face-to-face meetings were held to identify specific topics and controversies regarding nonneoplastic abnormalities affecting the articular cartilage, subchondral bone, and marrow. The committee was then divided into subgroups assigned to specific sections and questions. Members of each subgroup performed a literature review using a screening process based on article titles and abstracts

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and the following predefined inclusion criteria: original scientific papers that pertained to the key questions of each subgroup; study population of more than 10 patients; and English language abstract available. The subgroups reviewed the relevant literature and provided recommendations for the nomenclature. For each term and clinicopathologic entity, a precise definition was proposed; debate followed, and the participants voted for or against use of a term. On this foundation, a second consensus was reached by the entire group, with each term classified as to use or not use. The final recommendations were presented for comment to the entire SSR membership during the annual SSR meeting.

Definitions of Descriptive Terms

Descriptive terms are summarized in Table 1 and Figure 1.

Edemalike Marrow Signal Intensity

Many terms have been used interchangeably to describe ill-defined subchondral areas of increased signal intensity on MR images obtained with fluid-sensitive sequences that do not completely replace marrow fat on T1-weighted images [1]. These terms include "bone edema" [2], "bone bruise/contusion," "bone marrow lesions" [3, 4], "bone marrow edema" [5], "bone marrow edemalike signal" [6], "bone marrow edemalike lesions" [7], "bone marrow edema pattern" [5], and "osteitis" [8], to name a few. In general, the language used has been vague and varied with the clinical situation (e.g., "bone marrow lesion" in osteoarthritis). Only rarely have histologic findings been considered for terminology (e.g., "osteitis" for subchondral changes in signal intensity in inflammatory arthritis) [8] (Fig. 2).

Controversy—The term "bone marrow edema" refers to the pathologic finding of eosinophilic extracellular fluid and swollen fat cells within bone marrow [5]. On the basis of histopathologic correlations, however, the MRI signal-intensity abnormalities originally described as edema are nearly always produced by a variety of components and are associated with many processes, including injury, arthritis, overlying cartilage abnormality, and osteonecrosis. For example, when abnormal signal intensity is found in patients with osteoarthritis, pathologic bone marrow edema is not a major constituent [9] but may be present at times [5, 10]. Instead, other findings, such as trabecular changes like thickening and microfractures [10–14], marrow infiltration by fibrovascular tissue or granulation tissue [5, 10, 12–15], marrow necrosis [5, 10, 13], and blood products [5], may account for the signal intensity. In rheumatoid arthritis and spondyloarthritis, inflammatory cellular infiltrates (osteitis) [16–19] and myxoid and fibrovascular tissue [17] are common.

Recommendation—To acknowledge the lack of specificity of this MRI finding and to avoid confusion with a specific pathologic finding, the term "bone marrow edema" should be avoided. Instead, the term "edemalike marrow signal intensity" or "bone marrow edemalike signal intensity" should be used because these terms accurately describe the abnormal signal intensity without naming a specific histopathologic finding. Over a decade ago "bone marrow edema pattern" was coined to refer to these signal-intensity abnormalities [5] and is an acceptable term, but it may be advantageous to reserve the word "pattern" to refer to distributions of edemalike marrow lesions. In specific clinical situations, more precise terms may help radiologists indicate the underlying pathologic condition. After injury, the term "bone contusion" should be used to describe the presence of edemalike marrow signal intensity without a visible fracture line. When edemalike marrow signal intensity is related to inflammatory arthritis, such as rheumatoid arthritis, osteitis may be invoked as the probable cause.

Cystlike Lesion

The presence of edemalike marrow signal intensity on MR images can be associated with the subsequent development of subchondral cystlike lesions in the same location [6, 20]. The pathologic definition of a cyst is a cavitary, fluid-filled lesion with an epithelial lining [21]. Thus, "cyst" is not an accurate term for the lesions encountered at imaging of subchondral bone. Rather, these subchondral lesions are typically lined by a connective tissue membrane, such as collagen [22–24] and filled with a variable combination of inflammatory cells [24–26], mucoid or myxoid material [22, 23, 26], bone or fat necrosis [23, 24], and even solid, vascularized tissue [26] (Fig. 2). The term "geode" was suggested to describe these lesions on radiographs [27].

Controversy—In the osteoarthritis and inflammatory arthritis literature, the terms used for cystlike lesions have been inconsistent. Several of the semiquantitative grading systems used for clinical trials and other research studies have included terms that do not accurately reflect the pathologic definition of a cyst. These include "bone edema" [28], "bone marrow lesions" [3, 29], and "cysts" [1, 3, 29]. The term "bone marrow lesion" has been used to refer to subarticular edemalike marrow signal intensity or cystlike lesions related to cartilage abnormalities found in osteoarthritis and is commonly used in the arthritis literature [30]. However, this term may unnecessarily evoke concern about neoplasia and other nonarthritic entities. For example, metastases are often referred to as bone marrow lesions.

Recommendation—To be more consistent with the histopathologic findings, the term "cystlike changes" or "cystlike lesion" should be used. We acknowledge that the term "bone marrow lesion" is commonly used in the arthritis literature but recommend that it be avoided, because it might invoke concerns about neoplasia.

Reporting the location of cystlike lesions within the bone may also help to infer a particular cause. For example, "subarticular" and "subchondral" may relate to processes occurring subjacent to cartilage abnormalities, whereas "subcortical" abnormalities, such as those at the tibial spines and the humeral greater tuberosity, are more often related to the insertion of a ligament or tendon.

Osteochondral Lesion

"Osteochondral lesion" is a broad and nonspecific term that has been used to refer to any lesion that involves the articular surface and subchondral region of a joint, affecting cartilage, bone, or both.

Recommendation—The term "osteochondral lesion" should be used only as a nonspecific term for osteochondral lesions in which a causative process, such as trauma or osteoarthritis, is not identified.

Osteochondral Defect

An osteochondral defect is a type of osteochondral lesion characterized by a localized focal defect of the articular cartilage and subchondral bone (Fig. 3). Osteochondral defects may be the result of acute traumatic injury, subchondral collapse, or displacement of an unstable fragment in patients with osteochondritis dissecans [31]. Although it is widely accepted and its definition agreed on, the term "osteochondral defect" provides no specific information about the underlying cause.

Recommendation—The term "osteochondral defect" should be used to describe a fo-

TABLE 1: Summary of Controversies Regarding Nomenclature on Subchondral Nonneoplastic Bone Lesions With Panel Consensus Statements and Recommendations

Current Terms	Controversy	Recommended Terms	Terms to Be Avoided
Bone edema, bone bruise/contusion, bone marrow lesion, bone marrow edema, bone marrow edemalike signal, bone marrow edemalike lesion, bone marrow edema pattern, osteitis	Term may not correspond to underlying histopathologic findings	Edemalike marrow signal intensity, bone marrow edemalike signal intensity	Bone marrow lesion, bone marrow edema
		"Pattern" should be reserved for distributions of edemalike marrow signal intensity	
		Precise terms indicating specific pathologic entity in appropriate clinical situations (i.e., bone contusion, osteitis)	
Subchondral cysts, cystlike lesions, cystlike changes, geode	Term may not correspond to histopathologic findings	Cystlike lesions, cystlike changes	Cysts, subchondral cysts, geodes
Osteochondral lesion, osteochondral abnormality, osteochondral defect, OCD	Distinction between osteochondral lesion and osteochondral defect	"Osteochondral lesion" should be used as an umbrella term to describe a focal osteochondral abnormality of any cause and should be reserved for cases in which the exact diagnosis is unknown	Abbreviation OCD should not be used for this abnormality to avoid confusion with osteochondritis dissecans
	Confusion between osteochondral defect and osteochondritis dissecans	"Osteochondral defect" should be used to describe a focal defect in the articular cartilage and subchondral bone	
		When possible, the cause and chronicity of an osteochondral defect should be stated	
Epiphyseal collapse, articular collapse, subarticular collapse, subchondral cleft fracture, collapse	Distinction between collapse and fracture	Epiphyseal collapse, articular collapse	With acute trauma, "osteochondral fracture" should be used rather than "collapse"
		"Collapse" should be used to describe a fracture of the subchondral bone plate that occurs in the absence of acute trauma	
		Epiphyseal or articular collapse should be reported in the context of another diagnosis, such as osteonecrosis, subchondral insufficiency fracture, or rapidly progressive idiopathic arthritis	

(Table 1 continues on next page)

TABLE 1: Summary of Controversies Regarding Nomenclature on Subchondral Nonneoplastic Bone Lesions With Panel Consensus Statements and Recommendations (continued)

Current Terms	Controversy	Recommended Terms	Terms to Be Avoided
Acute traumatic osteochondral lesions (ATOLs), acute osteochondral injury, bone bruise, bone contusion, trabecular microfracture, trabecular impaction subchondral fracture, osteochondral fracture, articular impaction, impaction injury	Term to convey clinical significance of edemalike marrow signal intensity of cartilage and subchondral bone in acute trauma	"Bone contusion" should be used for edemalike marrow signal intensity without fracture	Trabecular microfracture, bone bruise, articular impaction, trabecular impaction
		"Osteochondral fracture" should be used when a contour deformity or fracture line involves cartilage and subchondral bone	
		"Subchondral fracture" should be used for subchondral linear signal intensity not involving articular cartilage or subchondral bone plate	
		"Acute traumatic osteochondral lesion" or "acute osteochondral injury" should be used only as an umbrella term for various acute posttraumatic osteochondral abnormalities	
Subarticular stress response (SSR), stress reaction	Term for atraumatic edemalike marrow signal intensity without fracture	Subarticular stress response, stress response for edemalike marrow signal intensity without fracture or history of acute trauma	
Osteochondritis dissecans (OCD)	Inconsistent use of OCD for different osteochondral entities	OCD should only be used for chronic osteochondral lesion occurring in children and young adults in classic locations	OCD should not be used for osteochondral fractures or lesions outside of the typical locations and age demographic
Subchondral insufficiency fracture (SIF), subchondral insufficiency fracture of the knee (SIFK)	Differentiating SIF from acute traumatic subchondral fractures	SIF should be used for subchondral fractures in absence of acute trauma	
	SIF as a distinct entity versus a component of another diagnosis	SIF can be reported in isolation or in association with other diagnoses, including transient osteoporosis of the hip, osteonecrosis, osteoarthritis, osteoporosis, or meniscal tear	
		In overuse injuries without associated risk factors, "subchondral stress fracture" or "subchondral fatigue fracture" should be used instead of SIF	
Spontaneous osteonecrosis of the knee (SONK)	Distinction between SONK and SIF	SIF	SONK should be replaced with SIF

(Table 1 continues on next page)

TABLE 1: Summary of Controversies Regarding Nomenclature on Subchondral Nonneoplastic Bone Lesions With Panel Consensus Statements and Recommendations (continued)

Current Terms	Controversy	Recommended Terms	Terms to Be Avoided
Transient osteoporosis of the hip (TOH), transient bone marrow edema syndrome (TBMES), transient painful bone marrow edema, bone marrow edema syndrome of the hip	Term may not reflect histopathologic findings or temporal relation between radiographic and MRI findings		
	SIF and transient bone marrow edema syndrome–TOH may not be separate entities	"Transient osteoporosis of the hip" should be used in presence of radiographic findings of osteopenia and preferred over "transient bone marrow edema syndrome" because it implies the risk of development of SIF	
		"Transient bone marrow edema syndrome" can be used when radiographic findings are normal or not available	
		SIF is a separate entity that may coexist or result from transient bone marrow edema syndrome–TOH	
Osteonecrosis (ON), avascular necrosis, bone infarct	Preferred term for bone necrosis	Osteonecrosis	
Rapidly destructive osteoarthritis (RDOA), rapidly progressive idiopathic arthritis (RPIA), rapidly destructive hip disease, rapidly destructive arthropathy/arthrosis, rapidly progressive osteoarthritis (RPOA),	Preferred term for this condition	"Rapidly progressive idiopathic arthritis" for rapid and extensive subchondral bone resorption in the absence of another cause, such as septic arthritis, osteonecrosis, neuropathy of joint, or inflammatory arthritis	
	Differentiating between rapidly destructive osteoarthritis and SIF	SIF should be reported as an associated finding in rapidly progressive idiopathic arthritis rather than a distinct diagnosis	
Osteoarthritis (OA), osteoarthrosis	Definition of osteoarthritis from the imaging, clinical, and research perspectives and the role of inflammation	Osteoarthritis	

cal defect in the articular cartilage and underlying subchondral bone. When possible, osteochondral defects should be described as acute, subacute, or chronic, depending on the degree of bone marrow edemalike signal intensity and bone remodeling seen at the margins of the defect. A specific diagnosis, such as osteochondral fracture, osteochondritis dissecans, or epiphyseal collapse should be rendered when possible. Abbreviating osteochondral defect as OCD should be avoided because of potential confusion with osteochondritis dissecans, a distinct diagnostic entity.

Osteochondral Fracture

An osteochondral fracture occurs in acute traumatic events but can become subacute or chronic. Osteochondral fractures violate the joint surface and involve the articular cartilage and subchondral bone plate. The fracture line can extend through the articular surface completely or incompletely and can encircle a portion of subchondral bone. It can also manifest as subchondral bone depression or fragmentation, creating an osteochondral fragment [31, 32] (Fig. 4).

Recommendation—The term "osteochondral fracture" should be used in the setting of acute trauma if the fracture lines involve both the articular cartilage and subchondral bone (see Acute Traumatic Osteochondral Lesions).

Subchondral Fracture:

A subchondral fracture is characterized by a curvilinear fracture line that is hypointense on T1-weighted and hyperintense or hypointense on fluid-sensitive MR images and surrounded by bone marrow edemalike signal intensity. The fracture line is subjacent to the subchondral bone plate but at some distance away from the bone plate. The fracture line may be irregular, serpentine, discontinuous or open-ended, parallel or convex, or, rarely, concave in relation to the articular surface [33–35] (Fig. 5). Although subchondral fractures can be caused by a single traumatic event, they are more commonly caused by chronic repetitive trauma, as in other stress fractures, for which the term "subchondral insufficiency fracture" (SIF) is used [33, 36] (see Subchondral Insufficiency Fracture). Histologic findings that account for the linear abnormal signal intensity on MR images depend on the morphology and acuity of the fracture. These findings consist of fractured bone trabeculae with a variable amount of associated fracture callus and granulation tissue [37–39].

Recommendation—The term "subchondral fracture" should be used if subchondral linear signal-intensity abnormality is found at a distance from the bone plate. If there is associated contour deformity or involvement of the articular surface, the term "osteochondral fracture" should be used. Accompanying epiphyseal collapse (see Epiphyseal Collapse) should be reported when present.

Epiphyseal Collapse

The terms "collapse," "epiphyseal collapse," and "articular collapse" refer to fracture of the subchondral bone plate in an atraumatic setting. Epiphyseal collapse often occurs in patients with an SIF or collapse due to osteonecrosis or rapidly destructive osteoarthritis [38, 40, 41]. Failure of the bone plate can be visualized in two patterns. The first is deformity of the subchondral bone plate. In the hips, this classically involves loss of normal sphericity of the femoral head, focal depression, a discrete cortical stepoff, or a more generalized area of collapse of the subchondral cortical bone [37, 38, 42]. The second pattern is a fluid-filled cleft underlying the subchondral bone plate, referred to as subchondral cleft, which is indicative of fracture, separation, and ultimately collapse. On fluid-sensitive images this manifests as an abnormal area of fluidlike linear hyperintensity undermining and separating the subchondral bone plate from the rest of the epiphysis [43–45] (Fig. 6).

Recommendation—The term "collapse" should be used to describe a fracture of the subchondral bone plate that occurs in an atraumatic setting. In acute trauma, the preferred term is "osteochondral fracture" (see Acute Traumatic Osteochondral Lesions). The terms "epiphyseal collapse" and "articular collapse" are both acceptable and should be used as descriptive terms with the understanding that they are not specific for any singular entity, because collapse can occur in osteonecrosis, SIF, and rapidly destructive osteoarthritis [46, 47]. It is important that articular collapse be accurately identified and characterized because it represents progression of disease that is nonreversible and often requires joint replacement [44].

Subchondral Hypointense Area

The term "subchondral hypointense area" refers to the finding on T1-weighted and fluid-sensitive MR images of a subchondral area of low signal intensity immediately subjacent to the subchondral bone plate that produces apparent thickening of the plate (Fig. 7). This observation differs from the aforedescribed subchondral fracture that runs a short distance from the bone plate. A subchondral hypointense area may be seen in several conditions. First, in patients with an insufficiency fracture, this hypointense crescentic area often represents a combination of a subchondral fracture line and granulation tissue and frequently secondary osteonecrosis interposed between the fracture and the bone plate [37, 39, 48]. In early stages the subchondral bone plate and overlying articular cartilage are intact. Second, in patients with a subchondral bone plate fracture, seen in either acute trauma or atraumatic collapse, an apparent thickening of the bone plate represents impacted trabeculae and, depending on chronicity and cause, fracture callus, reactive cartilage, granulation tissue, and osteonecrosis [43, 48, 49]. A deformity of the bone plate is typically present. Third, in osteoarthritis, subchondral sclerosis or eburnation may also produce subarticular hypointensity related to deposition of new bone on preexisting trabeculae, trabecular microfractures with callus formation, or secondary (shallow) osteonecrosis [39, 46, 50]. Overlying cartilage denudation is invariably present because it represents an origin of these changes.

Recommendation—Attention should be given to the finding of a subchondral hypointense area because it frequently represents a fracture and secondary osteonecrosis. Dedicated MRI with a small FOV is recommended to help differentiate associated conditions, although overlap may be present, particularly in advanced lesions. Additional findings, such as fracture line, collapse, and cartilage loss should be sought. The extent of a subchondral hypointense area has prognostic significance in insufficiency fractures (see Subchondral Insufficiency Fracture).

Clinicopathologic Entities

Acute Traumatic Osteochondral Lesions

Acute traumatic osteochondral lesions encompass a wide range of traumatic injuries to the cartilage and subchondral bone. Histologic analysis of these injuries reveals a spectrum of findings, ranging from bone marrow edemalike signal intensity in mild cases and microfracture of the subcortical trabeculae [51, 52] to hemorrhage of the fatty marrow and osteocyte necrosis in more severe injuries [53–58]. Acute traumatic osteochondral lesions can be differentiated from other types of subchondral marrow lesions on the basis of a history of acute trauma and recognition of typical locations of acute traumatic osteochondral lesions in different trauma patterns.

Controversy—Many terms have been used to describe the subchondral changes seen in acute traumatic osteochondral lesions, including "bone bruise," "bone contusion," "subchondral fracture," "trabecular microfracture," "trabecular or articular impaction," and "impaction injury" (Fig. 8). When involvement of the overlying cartilage is identified, the term "osteochondral fracture" is the most commonly used.

Recommendation—The term "acute traumatic osteochondral lesion" or "acute osteochondral injury" can be used. Injuries to the cartilage without involvement of the underlying bone should be referred to as chondral injuries, and because they do not result in subchondral changes in signal intensity, they are out of the scope of this paper. Injuries that involve subchondral bone should be referred to (in increasing severity) as bone contusions, which cause reticular or confluent changes in marrow signal intensity without a discrete fracture line or associated cortical disruption of the subchondral bone plate; subchondral fractures, which are characterized by linear areas of low T1 and T2 signal intensity (frequently seen in regions of bone contusion) but do not result in cortical disruption or deformity; and osteochondral fractures, which result in cortical disruption or cortical depression [32, 59]. Impaction fractures, such as deep lateral femoral sulcus occurring after anterior cruciate ligament tear, represent a specific type of osteochondral fracture. The degree of depression and displacement of osteochondral fractures should be described.

Subarticular Stress Response

Subarticular stress response, stress response, and stress reaction are terms that refer to pathologic findings that can be the result of either repetitive stress on normal bone, commonly referred to as fatigue stress response, or the result of normal stress on abnormally weakened bone, commonly referred to as insufficiency stress response [60]. On MR images, stress response initially presents as bone marrow edemalike changes in signal intensity, sometimes accompanied by cortical thickening without a discrete hypointense line [61] (Fig. 9). These subarticular stress responses are most often found in the knee and hip. In the knee, focal subchondral bone marrow edemalike signal intensity

in the peripheral rim of the medial tibial plateau is a specific sign for a medial meniscal tear or root ligament tear, presumably a reaction to the resultant abnormal femorotibial contact forces, and can be considered a form of stress response [62]. With continued stress, cortical disruption and a stress fracture may occur and appear on radiographs and CT images as linear sclerosis perpendicular to the trabeculae and parallel to the articular surface and on MR images as a fracture line surrounded by edemalike marrow signal intensity [63].

Recommendation—Stress response should be considered in patients with subacute or chronic pain who engage in repetitive loading activities or have osteopenia. In this clinical situation, the term "stress reaction" or "stress response" is appropriate for describing marrow changes in which there is no visible fracture line.

Osteochondritis Dissecans

The Research in Osteochondritis Dissecans of the Knee group has defined the term "osteochondritis dissecans" (OCD) as "focal, idiopathic alteration of subchondral bone with risk of instability and disruption of adjacent articular cartilage that may result in premature osteoarthritis" [64]. The most commonly accepted cause is repetitive microtrauma resulting in disruption of endochondral ossification [65–67]. This cause is supported by the common occurrence of OCD in typical areas in young patients engaged in athletics that expose them to repetitive trauma [68–70] and in pediatric patients with obesity [71]. The classic locations of OCD include the femoral condyles and the capitellum. The imaging appearance of OCD depends on the size of the lesion, its mineralization, the presence of findings suggesting instability, and the skeletal maturity of the patient. On radiographs OCD appears as an oval lesion in the subchondral bone with variable amounts of central lucency and surrounding sclerosis. On MR images OCD appears as an irregularity of the subchondral bone with underlying marrow edemalike signal intensity (Fig. 10).

Controversy—Differentiating OCD from variants of normal ossification can be challenging, but the presence of intact overlying cartilage and lack of underlying changes in marrow signal intensity favor a normal ossification variant over OCD [72]. Although there is agreement that the term is not accurate because no inflammation is involved [73], the term "osteochondritis dissecans" is still in use and unlikely to be replaced.

Recommendation—OCD is a well-described entity in the medical literature and a widely recognized term. The term is appropriate for describing a subacute or chronic osteochondral lesion occurring in childhood to young adulthood in the classic anatomic locations. The term should not be used to describe osteochondral fractures or other lesions outside of these locations and this age demographic.

Subchondral Insufficiency Fracture

SIF results from one or a combination of the following factors: diminished bone strength, decreased protective function of cartilage or menisci, or increased physical activity [59, 74]. SIF may present with acute pain and swelling and effusion, and focal articular collapse may be apparent on radiographs. The clinical outcome of SIF varies. The lesion may either heal with conservative management or progress accompanied by articular collapse. Several investigators [75–78] have evaluated the prognostic value of the location, size, and morphologic features of SIF on MR images and radiographs. The length of the subchondral fracture lines and hypointense areas, sometimes collectively referred to as band lesion, and the ratio of the band length to the weight-bearing portion of the femoral head and acetabular head index are important predictive factors for femoral head collapse (Fig. 11).

Controversy—There is controversy about differentiating SIF from acute traumatic subchondral fracture. Such a distinction may be clinically relevant because of the association between SIF and subchondral bone collapse and secondary osteonecrosis [38]. In the case of SIF, there is usually more extensive edemalike signal intensity surrounding the fracture line. Acute fractures typically have fracture lines that are hypointense on T1-weighted images and hyperintense on images obtained with fluid-sensitive sequences, whereas SIFs are more likely to have hypointense fracture lines in all pulse sequences [79]. The presence of ligamentous, meniscal, or other soft-tissue injury in conjunction with the clinical history of acute trauma favors acute fracture [32, 79]. In overuse injuries without associated risk factors of diminished bone strength, the terms "subchondral stress fracture" and "subchondral fatigue fracture" are preferred over "subchondral insufficiency fracture."

There is controversy regarding which imaging findings in SIF are predictive of irreversible lesions. Such assessment often depends on use of dedicated MRI with a small FOV and may not always be possible. Prognostic features defined for early lesions may not be applicable in the presence of accompanying substantial cartilage loss. Extensive subchondral areas of low signal intensity on fluid-sensitive images are associated with poor prognosis for early lesions [37, 77, 78]. When SIF is associated with epiphyseal collapse, the lesions are irreversible and have a poor prognosis [77, 78].

Recommendation—Although several MRI features help to differentiate SIF from acute traumatic subchondral fracture, clinical history remains essential. In equivocal cases, follow-up imaging may be prudent to monitor healing. Dedicated MRI with a small FOV should be used to evaluate for the presence and extent of individual morphologic findings in SIF, such as a fracture line, a subchondral hypointense area, subtle contour changes, cartilage loss, and the presence of collapse. Lesions with collapse should be reported as irreversible. It is important to assess for associated cartilage loss and, in the knee, meniscal abnormalities that can contribute to an unfavorable prognosis for SIF healing.

Spontaneous Osteonecrosis of the Knee

The term "spontaneous osteonecrosis of the knee" (SONK) has been used to describe a clinical entity typically affecting elderly women who present with severe pain in the knee that has a sudden onset with no associated trauma [80]. This lesion was thought to be due to osteonecrosis even though very little osteonecrosis was found at histologic examination [80]. At knee MRI, SONK has been used to denote a spectrum of findings with SIF at one end and collapse and secondary osteoarthritis at the other [31, 81–83] (Fig. 12). Technologic advancements in MRI have improved visualization of the subchondral plate, resulting in better understanding of this condition [82–84]. The landmark review of pathologic specimens by Yamamoto and Bullough [39] revealed that in SONK a subchondral insufficiency fracture is the primary event that may lead to localized osteonecrosis confined to the fracture area. Association with meniscal tears, particularly medial meniscus root tears, further supports the theory of mechanical stress and chronic microtrauma as the cause of SONK [84–87].

Controversy—There is a controversy about whether SONK represents a discrete entity. This notion is supported by a distinct clinical course of SONK and findings of osteonecrosis in collapsed specimens. SONK is now con-

sidered a misnomer. The consensus is that this entity represents an SIF of the knee that has progressed to subchondral collapse and secondary osteonecrosis [39, 82, 84, 88].

Recommendation—The term "spontaneous osteonecrosis of the knee" should not be used and should be replaced by "subchondral insufficiency fracture." MRI should be used to identify irreversible findings, such as subchondral bone plate collapse, and findings associated with poor prognosis, such as extensive subchondral hypointensity, which may denote secondary osteonecrosis (see Subchondral Insufficiency Fracture, Recommendation).

Transient Osteoporosis of the Hip

Transient osteoporosis of the hip (TOH) is an idiopathic disease more common in men (5:1) with sudden onset of hip pain, antalgic gait, and decreased range of motion [45, 89–91]. It is treated conservatively with limited weight-bearing and antiresorptive medications. It typically resolves within 12 months. The disease most often involves two common subtypes of patients: men in middle age and women in the third trimester of pregnancy. However, it certainly occurs in both sexes in all stages of adulthood. When recurrent, TOH is called regional migratory osteoporosis, which can occur in as many as 20% of patients with TOH [45]. Imaging findings of TOH have been described for radiography (osteopenia of the affected hip from the 5th to the 16th week after onset of symptoms), nuclear medicine (uniform increased uptake in the involved hip), and MRI (edemalike signal intensity of the involved bone with or without joint effusion) [40, 78, 89, 92] (Fig. 13). Histopathologically, TOH is characterized by osteoporosis with vascular congestion and absence of osteonecrosis [90]. Given the increased perfusion of the hip in TOH compared with osteonecrosis, research [91, 92] has shown that dynamic contrast-enhanced MRI with high temporal resolution may help in differentiating osteonecrosis from TOH.

Controversy—It is debated whether diffuse ill-defined bone marrow edemalike signal intensity of the hip with no other findings can represent either early osteonecrosis or TOH. However, studies conducted with improved MRI techniques have shown that other findings of osteonecrosis, such as serpentine band, epiphyseal line, and subchondral low signal intensity are present before the development of edema [93]. Investigators in one study [78] also recommended obtaining dedicated small-FOV MR images of the involved hip so that subchondral abnormalities would not be missed. The smaller FOV was essential to ruling out any subchondral abnormalities in addition to the ill-defined edemalike signal intensity of the hip. The condition of patients who originally presented with diffuse ill-defined bone marrow edemalike signal intensity and no other findings did not progress to osteonecrosis [78]. At MRI, absence of other subchondral hip findings outside of the diffuse ill-defined bone marrow edemalike signal intensity has excellent specificity and positive predictive value (approaching 100%) for diagnosing TOH as long as the patient does not have other clinical evidence of infection or underlying rheumatologic disease [78].

Another controversy is that SIF is a separate entity from TOH and that there is no overlap. It is true that SIF is a separate entity from TOH; however, the conditions can coexist. The development of SIF is one of the complications found in patients with TOH who do not limit weight-bearing. The rate of SIF in patients with TOH has been reported to range from 5% to 50%. This discrepancy is thought to be related to the rate of patient adherence to limited weight-bearing.

A third controversy concerns the name "transient osteoporosis of the hip." Other terms used to describe the MRI findings of diffuse ill-defined bone marrow edemalike signal intensity of the hip in a patient with clinical findings that fit TOH are "transient bone marrow edema syndrome" and "bone marrow edema syndrome of the hip." These terms all describe the same clinical process as TOH with identical clinical course and imaging findings [78, 92]. There is a temporal relation between disease manifestation with different imaging modalities. The preferred term is TOH, which fits both the clinicopathologic findings of osteoporosis [90] and implies the clinical risk of SIF given the underlying osteopenia.

Recommendation—Dedicated small-FOV images of the involved hip are recommended for detection of any subchondral abnormalities that may suggest SIF or osteonecrosis. The diagnosis of TOH should be made when images obtained with fluid-sensitive sequences show only diffuse ill-defined bone marrow edemalike signal intensity in the hip. If any other associated abnormal subchondral linear signal intensity is present, the differential diagnosis is broader and includes SIF and osteonecrosis.

SIF can occur in association with other entities, such as osteonecrosis, TOH, and underlying osteoporosis. TOH is the preferred term for this entity in the presence of radiographic findings. Both "transient osteoporosis of the hip" and "transient bone marrow edema syndrome" accurately describe this entity as visualized with different imaging modalities and at various stages of the process. However, the word "osteoporosis" in TOH implies that the bone is weakened and that patients are at increased risk of SIF if they do not restrict their weight-bearing. The term "transient bone marrow edema syndrome" should be used when radiographs are normal or not available.

Osteonecrosis

Osteonecrosis or avascular necrosis results from a reduction or complete loss of blood supply to bone. The condition has numerous causes, the most frequent being trauma and corticosteroid use, or it can be idiopathic [41, 49]. The term "osteonecrosis" is preferred for describing these regions of devitalized bone because it is more inclusive than "avascular necrosis" and because all necrosis is by definition avascular.

The histologic characteristics of chronic or healing phase osteonecrosis occur in three zones: necrotic bone trabeculae, creeping bone substitution, and living bone [47, 49, 94]. At MRI, regions of osteonecrosis typically consist of central mummified fat or yellow marrow outlined by a distinct uninterrupted rim of primarily low signal intensity. This rim at the epiphysis represents reparative tissue formed around a crescentic, wedge-shaped, or ringlike region of osteonecrosis (Fig. 14). The double line sign on T2-weighted images—in which an outer low-signal-intensity rim corresponds to sclerosis from creeping substitution and a high-signal-intensity inner zone represents reparative granulation tissue at the reactive interfaces—has been identified in 65–85% of patients and is considered pathognomonic of osteonecrosis [39, 49, 95]. Epiphyseal collapse in osteonecrosis is represented by a deformity of the subchondral bone or a fluid-filled cleft or both. It may be better visualized on radiographs or CT images, likely because of the higher spatial resolution. Although osteonecrosis may result in osteoarthritis, this is usually a late occurrence after articular collapse. In such cases, foci of osteonecrosis may be found in portions of bone unaffected by osteoarthritis, and radiographic findings may be more severe on the femoral side of the joint [43, 50].

Controversy—It can be difficult to differentiate osteonecrosis from SIF of the fem-

oral head, particularly if collapse has occurred. Before femoral head collapse, this is an important distinction because osteonecrosis is considered irreversible, whereas SIF may resolve with conservative management [34, 42]. SIF is rarely bilateral and is more common in elderly women with obesity and osteoporosis, whereas osteonecrosis is often bilateral (50–70%) [41, 49]. On MR images, SIF typically appears as a low-signal-intensity discontinuous and irregular fracture line parallel to the articular surface of the femoral head. This is in contradistinction to the generally wider, geographic lesions of osteonecrosis with a serpentine contour [43, 49, 96, 97]. The depth of the low-signal-intensity band from the articular surface has also been reported as helpful in differentiating SIF from osteonecrosis; the mean depth is 1.56 mm for SIF and 15.36 mm for osteonecrosis [75].

MRI with IV contrast enhancement may be useful for discerning osteonecrosis from SIF. The subchondral portion of the femoral head proximal to the fracture plane in patients with SIF is enhancing (indicating perfused, living bone) unlike the nonenhancing regions of dead bone in osteonecrosis [98]. Dynamic contrast-enhanced MRI has been reported as a complementary technique for differentiating SIF from osteonecrosis. Osteonecrosis has markedly lower maximum enhancement values, longer time to peak, and lower slopes [99].

"Osteonecrosis" is an umbrella term that includes bone infarcts (metaphyseal) and avascular necrosis (epiphyseal). "Avascular necrosis" has historically been applied to areas of epiphyseal involvement. However, "osteonecrosis" and "avascular necrosis" are often used interchangeably for the same entity, which can lead to confusion.

Recommendation—The morphologic features of subchondral changes should be used to differentiate osteonecrosis from SIF. In cases in which it is difficult to differentiate SIF without underlying osteonecrosis from SIF with underlying osteonecrosis, contrast-enhanced studies may be helpful. The term "osteonecrosis" is preferred for describing regions of devitalized bone because it is all-inclusive.

Rapidly Destructive Osteoarthritis

The condition frequently described as "rapidly destructive osteoarthritis" [100], "rapidly destructive hip disease" [101], "rapidly destructive arthropathy/arthrosis of the hip" [46, 102], and "rapidly progressive osteoarthritis" [103, 104] is rare. It is increasingly seen owing to the increasing size of the elderly population. Rapidly destructive osteoarthritis occurs most commonly in elderly women but may also occur in younger patients after trauma [103]. The condition was first described in the hip and has subsequently been identified in the shoulder as destructive arthropathy that radiographically mimics septic arthritis, osteonecrosis, neuropathic joint, and rheumatologic disease [101, 105, 106]. The cause of this entity remains poorly understood. Histologically, the degenerative changes in rapidly destructive osteoarthritis are very similar to those of primary osteoarthritis with the synovitis characterized as reactive rather than inflammatory [103]. Calcium hydroxyapatite crystals, foci of necrosis, capsular fibrosis, and subchondral fracture are variably present [100].

The imaging findings of rapidly destructive osteoarthritis have two stages: marked chondrolysis followed by rapid and extensive subchondral bone resorption and destruction. In the hip, rapid progression of joint space loss has been defined as greater than 2 mm per year or greater than 50% joint space loss in a year [100, 106, 107] (Fig. 15). In the early chondrolytic stage, hip joint space narrowing is superolateral, similar to that in primary osteoarthritis, but there is minimal to no osteophytosis or subchondral sclerosis with subchondral cystlike changes. Some authors [104] have considered exuberant subchondral cystlike change one of the hallmarks of rapidly destructive osteoarthritis. Early MRI findings include diffuse cartilage denudation with joint effusion and synovitis and pericapsular edema, which can mimic infection. However, the most striking feature is the marked bone marrow edemalike signal intensity at both the femoral head and the acetabulum, which may be accompanied by SIF of the femoral head [46]. In the late subchondral resorptive-destructive stage, radiographs show marked femoral head flattening and remodeling that may be so extensive as to mimic the surgical-like resection margins of atrophic neuropathic arthropathy [108] (Fig. 15).

Controversy—There is controversy about which of the many terms referring to rapidly destructive osteoarthritis is the most accurate. The cause of the condition remains unknown and likely has multiple factors, often unrelated to osteoarthritis. The preferred term should reflect the typical clinical and imaging features and emphasize that this idiopathic condition differs from similar-appearing processes with a known cause, such as septic arthritis and rheumatoid arthritis. For these reasons, the committee is proposing the new term "rapidly progressive idiopathic arthritis" for this condition and considers this term more accurate.

There is controversy about the relation between rapidly progressive idiopathic arthritis and SIF. Evidence suggests that SIF likely plays a role in the development of rapidly progressive idiopathic arthritis in both the shoulder and hip [100]. Although SIF has been clearly identified in many patients with rapidly progressive idiopathic arthritis, it remains uncertain whether SIF is the inciting event for chondrolysis and bone destruction or simply one possible outcome of a multifactorial process [103]. It is also poorly understood why some patients with SIF have rapidly progressive idiopathic arthritis but others have SIF that resolves with conservative management [105, 106].

Recommendation—The term "rapidly progressive idiopathic arthritis" should be used to describe rapid and extensive subchondral bone resorption and destruction in the absence of septic arthritis, osteonecrosis, neuropathic joint, or rheumatologic disease. SIF should be reported as an associated finding in rapidly progressive idiopathic arthritis rather than as a distinct diagnosis.

Osteoarthritis

Osteoarthritis has been recognized for centuries, yet a firm, uniform definition remains elusive as understanding of the disease evolves. Clinical findings of inflammation, that is, redness, swelling, heat, and pain, are not prominent features of osteoarthritis; therefore, some have preferred to refer to the entity as "osteoarthrosis," and both terms have been accepted in the literature. However, with the recognition of the important role of inflammatory mediator production in this disease, "osteoarthritis" is a more accurate term [109]. The pathophysiologic mechanism of osteoarthritis involves degradation and loss of cartilage followed by osseous changes [110]. There have been calls for standardization [111, 112], and imaging-based definitions have been used in clinical and research settings.

Controversy—Should an imaging diagnosis of osteoarthritis be made? If so, how early in the disease process can radiologists make this diagnosis? Although these questions may appear important only for theoretic discussion, real potential clinical implications ex-

ist. Preexisting conditions are important for those who change jobs or insurance companies. In addition, if a joint is labeled osteoarthritic at an early stage, regulatory agencies or insurance companies may approve the use of certain therapies for cartilage defects but deny other procedures. In the future, therapies for early osteoarthritis may be based on serologic markers or compositional cartilage matrix changes that are apparent at imaging before any loss of tissue is visible.

Recommendation—The term "osteoarthritis" rather than "osteoarthrosis" is recommended, reflecting the important role of inflammatory mediators in the pathogenesis of the disease.

Conclusion

Controversy exists about the nomenclature and pathophysiologic characteristics of subchondral nonneoplastic bone lesions. This consensus statement summarizes current understanding of the pathophysiologic and imaging findings of subchondral nonneoplastic bone lesions and proposes nomenclature to improve effective communication across clinical specialties and help avoid diagnostic errors that could affect patient care.

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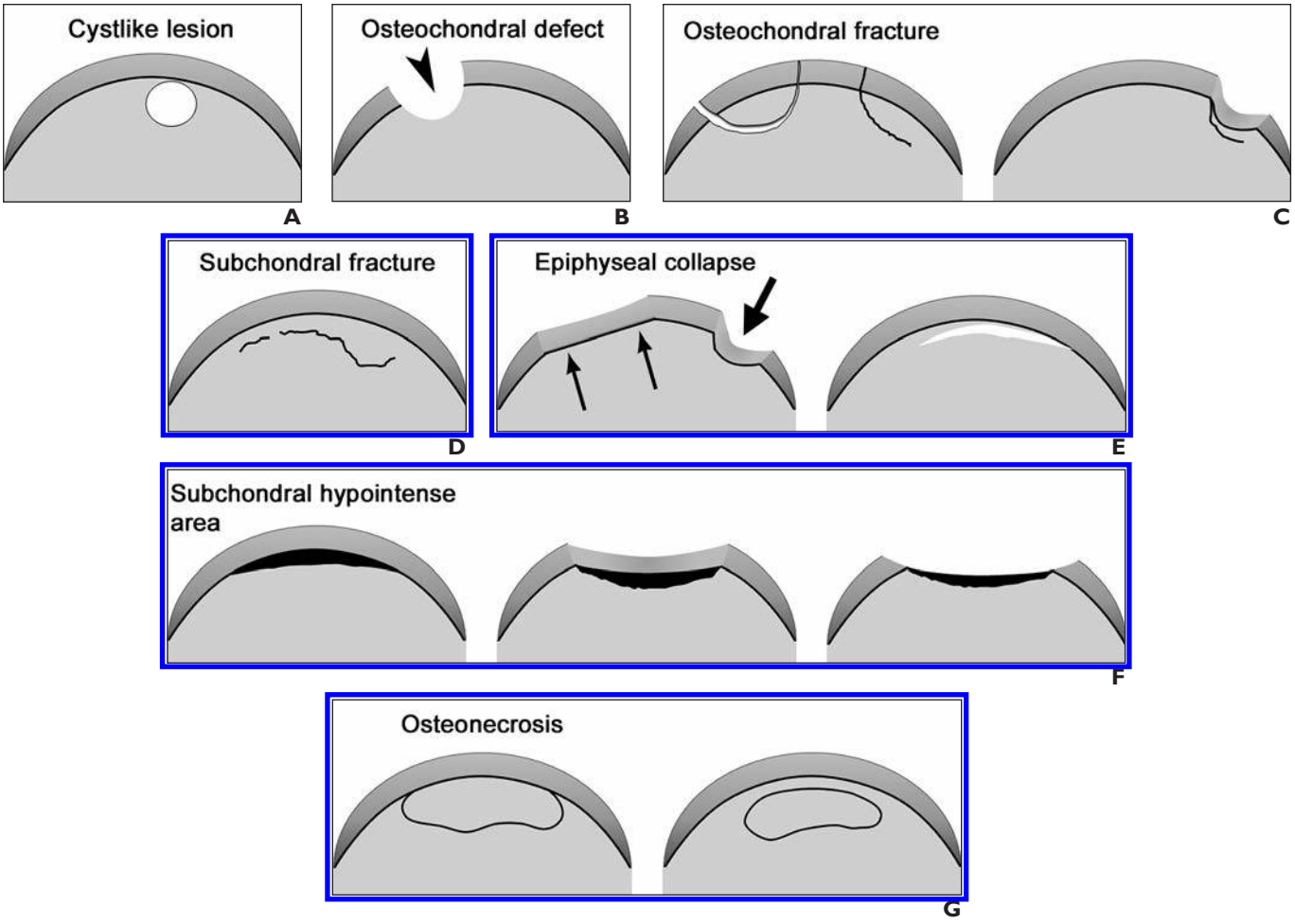


Fig. 1—Descriptive terms applied to nonneoplastic morphologic abnormalities affecting subchondral bone and marrow. Diagrams depict generic epiphyseal surface of bone on T2- or intermediate-weighted MR images. Dark gray denotes articular cartilage; black line, subchondral bone plate; light gray, underlying trabecular bone and marrow.
A, Cystlike lesion. Rounded well-marginated area of high signal intensity on T2-weighted images and loss of normal marrow signal intensity on T1-weighted images. Histologically such lesions are typically lined by connective tissue membrane and contain inflammatory cells, myxoid material, and necrotic bone.
B, Osteochondral defect. Localized defect (*arrowhead*) encompasses articular cartilage, subchondral bone plate, and underlying trabecular bone and marrow.
C, Osteochondral fracture. Acute traumatic fracture involves articular cartilage and subchondral bone plate represented by fracture line that extends through articular surface (*left*) completely or incompletely encircling portion of subchondral bone or deformity with subchondral bone plate depression (*right*).
D, Subchondral fracture. Line or band of low signal intensity running at short distance from subchondral bone plate. Line represents fractured trabeculae with variable amount of associated fracture callus and granulation tissue. Line is usually irregular, serpiginous, often discontinuous or open ended, parallel, convex, or, in rare instances, concave in relation to articular surface. It typically does not delineate large marrow area.
E, Epiphyseal collapse. Fracture of subchondral bone plate in atraumatic setting occurs in two patterns. First is deformity of subchondral bone plate resulting in loss of normal epiphyseal sphericity (*left*), which manifests as flattening (*thin arrows*) or abrupt focal depression (*thick arrow*) of bone plate. Second pattern is fluid-filled cleft underlying subchondral bone plate, representing fracture cleft (*right*). These patterns may be present individually but often coexist.
F, Subchondral hypointense area. Subchondral area of low signal intensity immediately subjacent to subchondral bone plate produces apparent crescentric thickening of plate (*left*). This finding represents combination of fracture line directly beneath subchondral plate, granulation tissue, and often secondary osteonecrosis between fracture and articular surface. Apparent thickening of bone plate often accompanies subchondral bone plate collapse, representing impacted trabeculae, granulation tissue, and secondary osteonecrosis (*middle*). Subchondral sclerosis or eburnation in osteoarthritis may produce similar subarticular hypointensity related to deposition of new bone on preexisting trabeculae and to trabecular microfractures with callus formation (*right*).
G, Osteonecrosis. Subchondral lesion consisting of area of preserved marrow signal intensity outlined by peripheral rim. Area encompasses devitalized marrow, and rim represents reactive interface between necrotic and viable bone. Lesion may involve marrow immediately subjacent to bone plate (*left*) or, less commonly, away from it (*right*). Rim is typically smooth, concave to articular surface, and complete and encircles infarcted area without interruption.

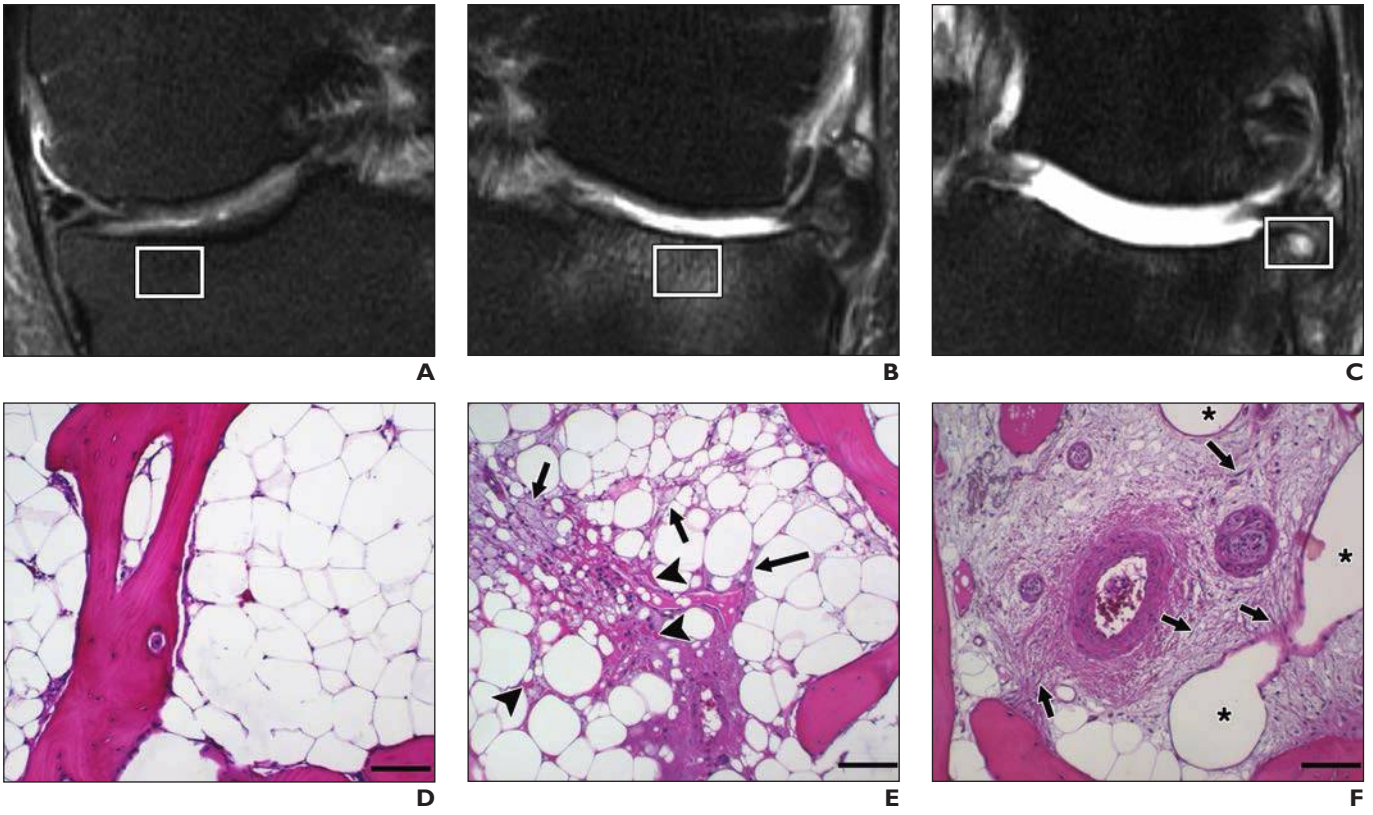


Fig. 2—65-year-old man with left knee pain. Example of bone marrow edemalike signal intensity and cystlike lesions.
A, Preoperative coronal T2-weighted fat-suppressed MR image (TR/TE, 3500/65) shows marrow signal intensity at medial tibial plateau. Box indicates area shown in **D.**
B, Preoperative coronal T2-weighted fat-suppressed MR image (TR/TE, 3500/65) shows subchondral edemalike marrow signal intensity at lateral tibial plateau. Box indicates area shown in **E.**
C, Preoperative coronal T2-weighted fat-suppressed MR image (TR/TE, 3500/65) shows cystlike lesion at peripheral aspect of lateral tibial plateau. Box indicates area shown in **F.**
D, Postoperative photomicrograph (H and E, ×10; bar, 100 μm) corresponding to box in **A** shows normal marrow with few hematopoietic cells and small vessels among many adipocytes.
E, Postoperative photomicrograph (H and E, ×10; bar, 100 μm) corresponding to box in **B** shows edematous region of marrow with eosinophilic globules (*arrowheads*) and enlarged, hypocellular space between adipocytes (*arrows*).
F, Postoperative photomicrograph (H and E, ×10; bar, 100 μm) corresponding to box in **C** shows cystlike lesion consisting of well-vascularized hypocellular regions containing abundant extracellular matrix with numerous large capillarylike venous spaces (*asterisks*) and frequent flattened fibroblastlike cells (*arrows*).

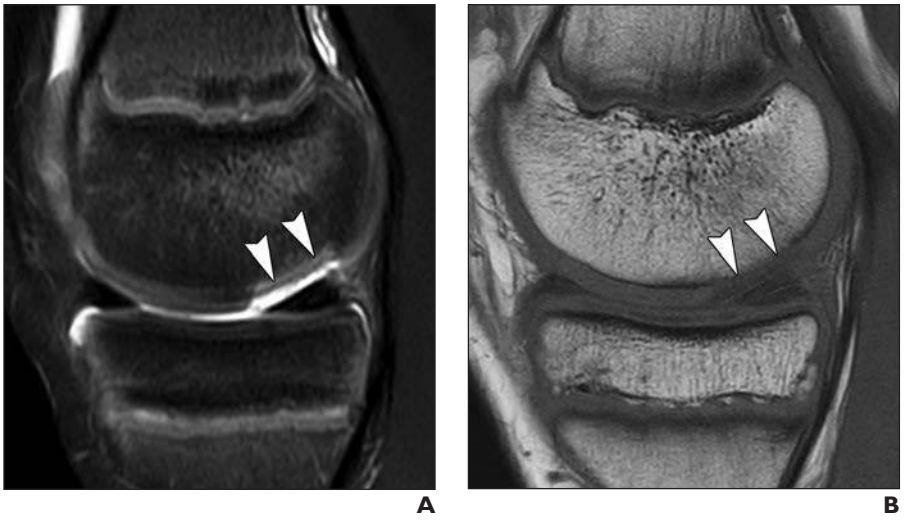


Fig. 3—13-year-old male basketball player with osteochondral defect and 2-week history of left knee pain, swelling, and occasional locking; symptoms may not necessarily be due to osteochondral defect. Case serves as imaging example of such defect.
A, Sagittal proton density–weighted fat-suppressed MR image shows full-thickness cartilage defect with mild subchondral edemalike signal intensity (*arrowheads*).
B, T1-weighted MR image shows defect of subchondral bone plate (*arrowheads*) with absence of normal low-signal-intensity cortical line in this region.

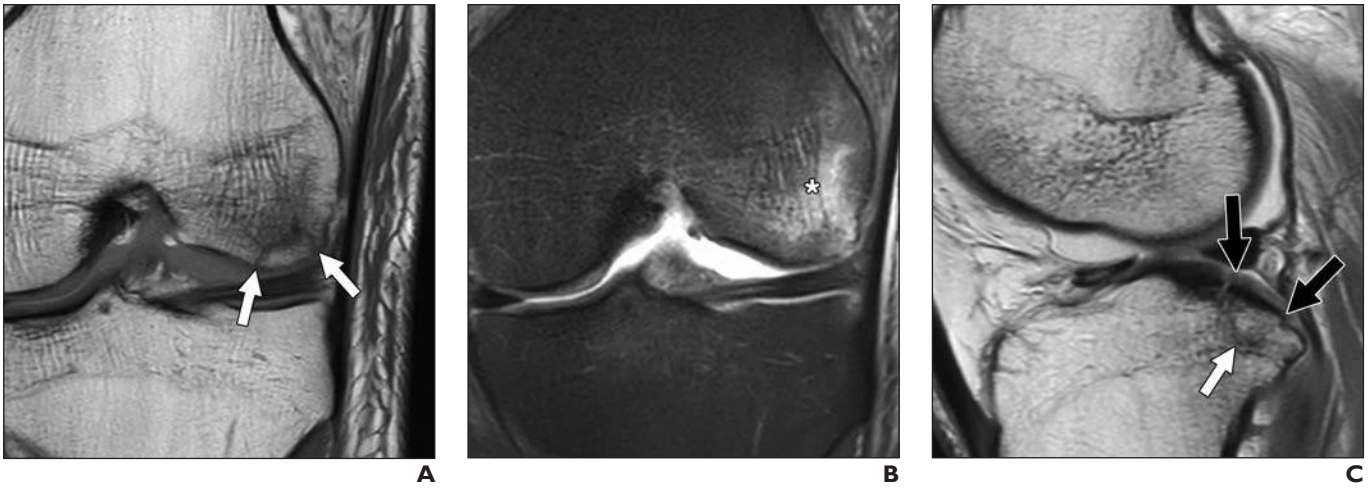
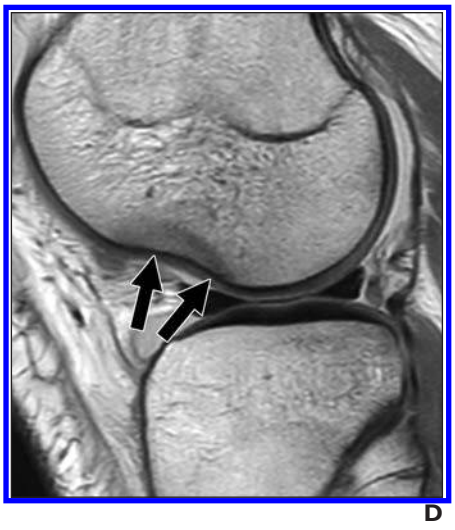


Fig. 4—Osteochondral fractures in acute knee injury with anterior cruciate ligament rupture.
A–C, 45-year-old man. Coronal T1-weighted (**A**), fat-suppressed proton density–weighted (**B**), and sagittal proton density–weighted (**C**) MR images show fracture line extending to subchondral bone plate (*white arrows*, **A** and **C**) with visible disruption of articular cartilage and subchondral bone plate (*black arrows*, **C**). Bone marrow edemalike signal intensity (*asterisk*, **B**) associated with fracture is evident.
D, 18-year-old man. Sagittal proton density–weighted MR image shows contour deformity with subchondral bone plate depression (*arrows*, **D**).



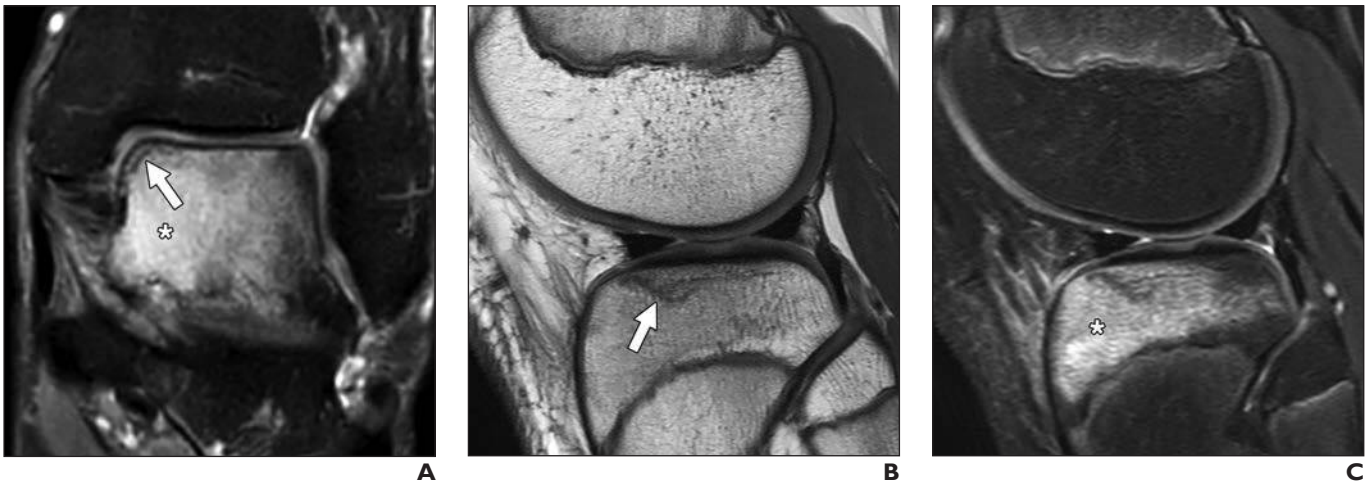


Fig. 5—Subchondral fractures of talar dome.
A, 64-year-old man without history of trauma. Coronal proton density–weighted fat-suppressed MR image shows line of low signal intensity coursing at short distance from subchondral bone plate (*arrow*) with bone marrow edemalike signal intensity (*asterisk*) on both sides of line. Irregular and discontinuous appearance of line is concave in relation to articular surface.
B and **C,** 15-year-old boy after acute injury to lateral tibial plateau. Sagittal proton density–weighted (**B**) and proton density–weighted fat-suppressed (**C**) MR images show line of low signal intensity short distance from subchondral bone plate (*arrow,* **B**) with bone marrow edemalike signal intensity (*asterisk*, **C**) on both sides of line. Irregular and discontinuous appearance of line is convex in relation to articular surface.

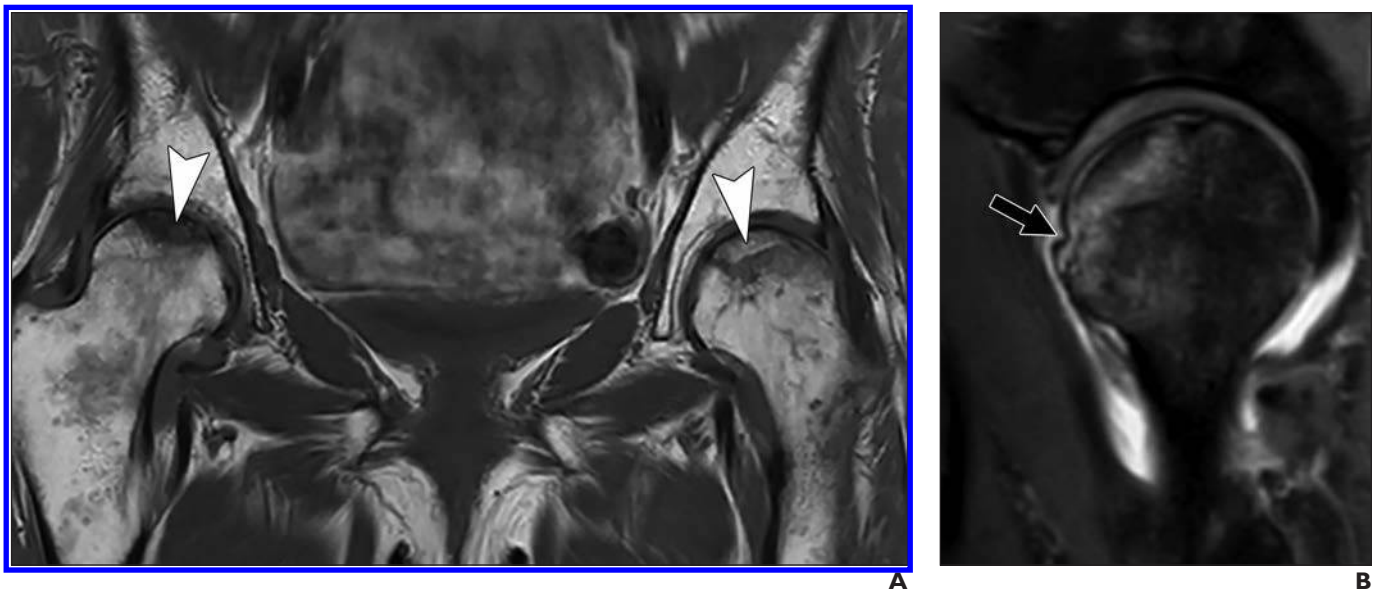


Fig. 6—Epiphyseal collapse.
A–C, 53-year-old man with bilateral femoral head osteonecrosis. Coronal T1-weighted MR image of pelvis (**A**) and sagittal proton density–weighted fat-suppressed image of right (**B**) and left (**C**) hips show subchondral areas of infarct (*arrowheads*, **A**) with collapse manifesting as abrupt focal depression of bone plate (*arrow*, **B**) and fluid-filled cleft underlying subchondral bone plate (*arrow*, **C**).
D, 35-year-old man with history of acute myeloid leukemia, bone marrow transplant, and osteonecrosis. Sagittal T2-weighted fat-suppressed MR image shows extensive collapse with subchondral bone plate flattening resulting in loss of normal sphericity (*black arrows*) of posterior aspect of medial femoral condyle and large fluid-filled clefts (*white arrows*) in infarcted areas of femoral condyle and medial tibial plateau.

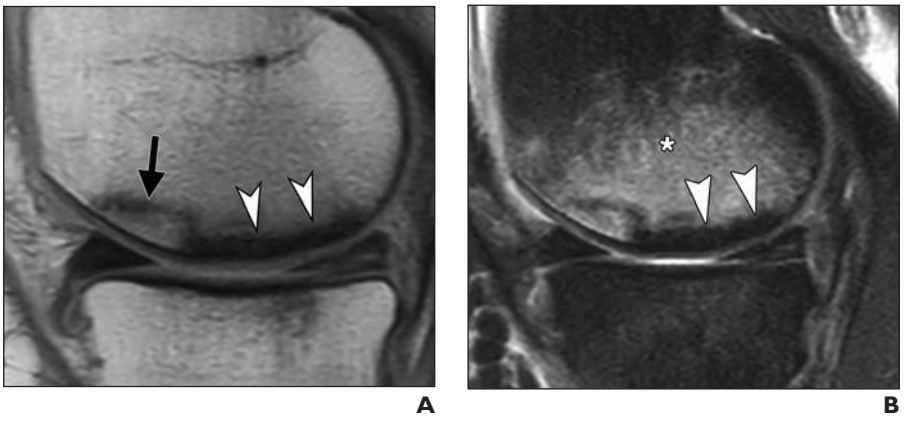


Fig. 7—Subchondral hypointense area.
A and **B,** 64-year-old woman with insufficiency fracture of medial femoral condyle. Sagittal proton density–weighted (**A**) and T2-weighted fat-suppressed (**B**) MR images show low-signal-intensity area (*arrowheads*) immediately subjacent to subchondral bone plate without contour deformity. Distinct subchondral fracture line (*arrow*, **A**) is also present in anterior aspect. Extensive bone marrow edemalike signal intensity associated with subchondral fractures (*asterisk*, **B**) is less localized than edemalike signal intensity in osteoarthritis (*asterisk*, **E**).

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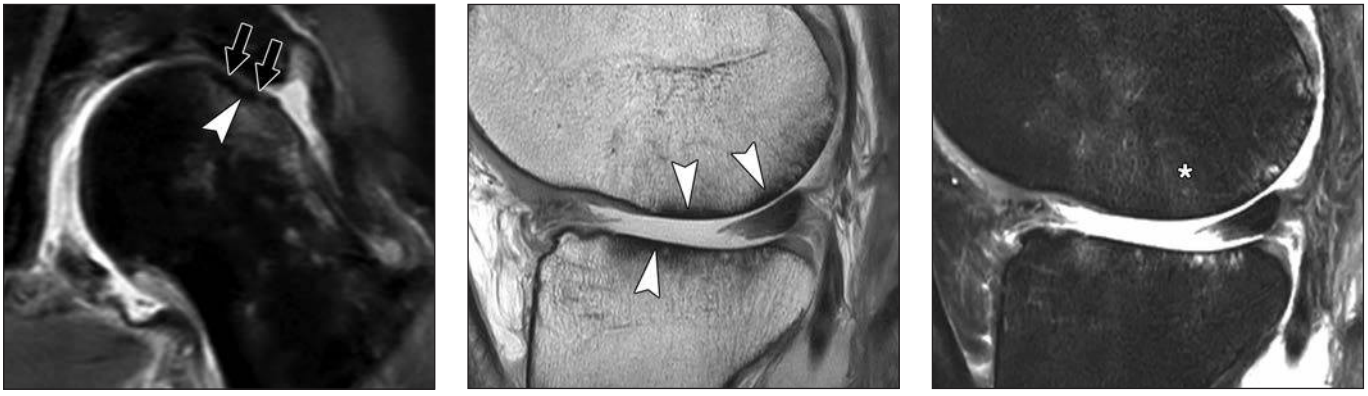

Fig. 7 (continued)—Subchondral hypointense area.
C, 68-year-old woman with femoral head insufficiency fracture and collapse. Coronal proton density–weighted fat-suppressed MR image shows hypointense area (*arrowhead*) representing impacted trabeculae beneath flattened articular surface (*arrows*).
D and **E,** 68-year-old man with osteoarthritis of knee. Sagittal proton density–weighted (**D**) and T2-weighted fat-suppressed (**E**) MR images show subchondral sclerosis in areas of cartilage denudation reflected by subarticular hypointensity (*arrowheads*, **D**). Edemalike signal intensity associated with osteoarthritis (*asterisk*, **E**) is more localized than bone marrow edemalike signal intensity in subchondral fracture (*asterisk*, **B**).

Fig. 8—17-year-old male football player with 2-week history of knee pain after direct blow to lateral knee causing bone contusion.
A and **B,** Sagittal fat-suppressed (**A**) and T1-weighted (**B**) proton density–weighted MR images show bone marrow edemalike signal intensity (*asterisk*) in anterior tibial plateau. No fracture line or contour deformity is present.

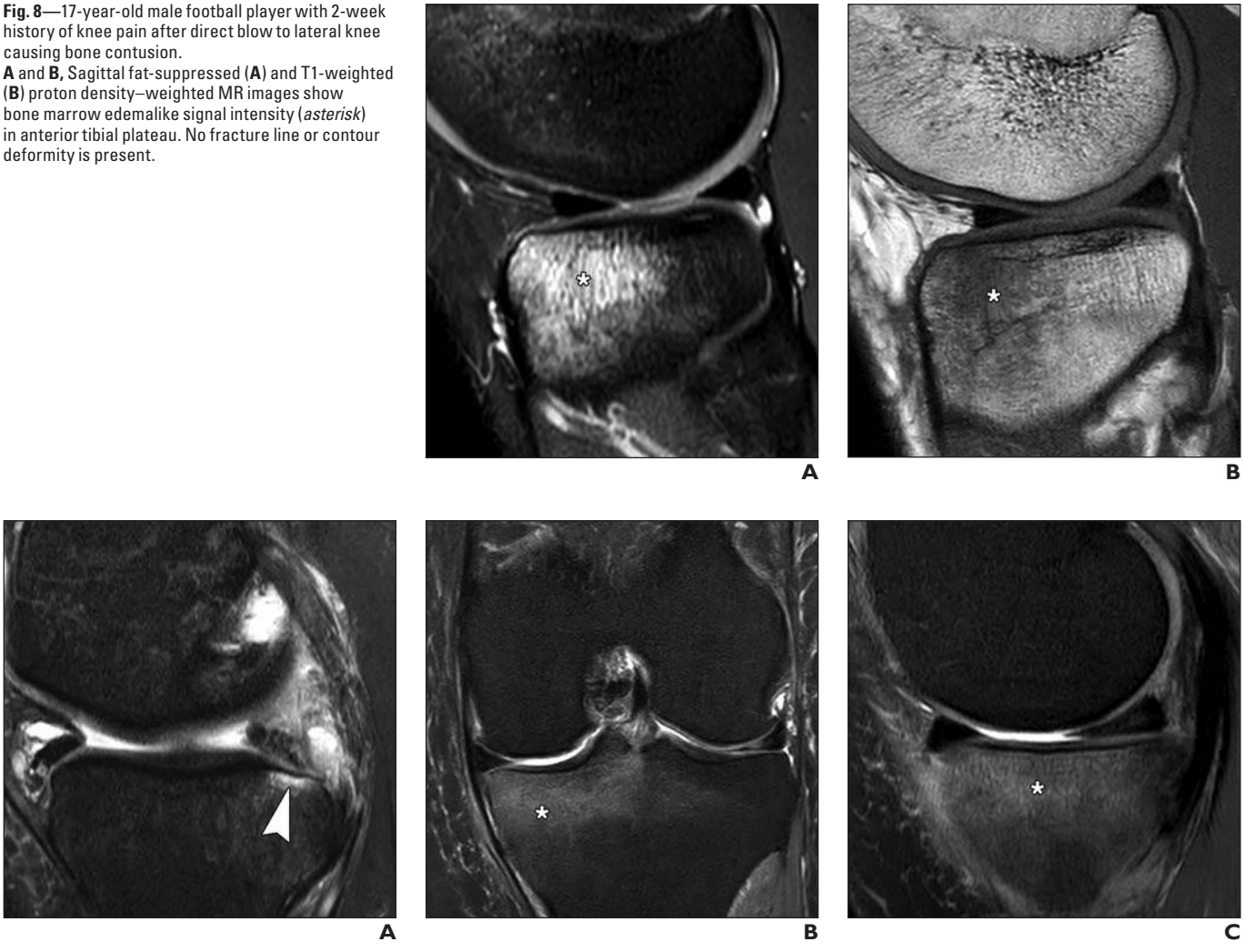

Fig. 9—Stress response.
A, 48-year-old man with tear of posterior root of medial meniscus causing subarticular stress response. Sagittal proton density–weighted fat-suppressed MR image shows linear subchondral area of bone marrow edemalike signal intensity (*arrowhead*) localized to area deep to posterior meniscal root.
B and **C,** 74-year-old woman with stress response and 8-week history of progressively worsening medial knee pain and no history of trauma. Coronal (**B**) and sagittal (**C**) proton density–weighted fat-suppressed MR images show diffuse edemalike signal intensity (*asterisk*) within subchondral marrow of medial tibial plateau. No associated thickening of subchondral bone plate or linear signal-intensity change is present to suggest fracture.

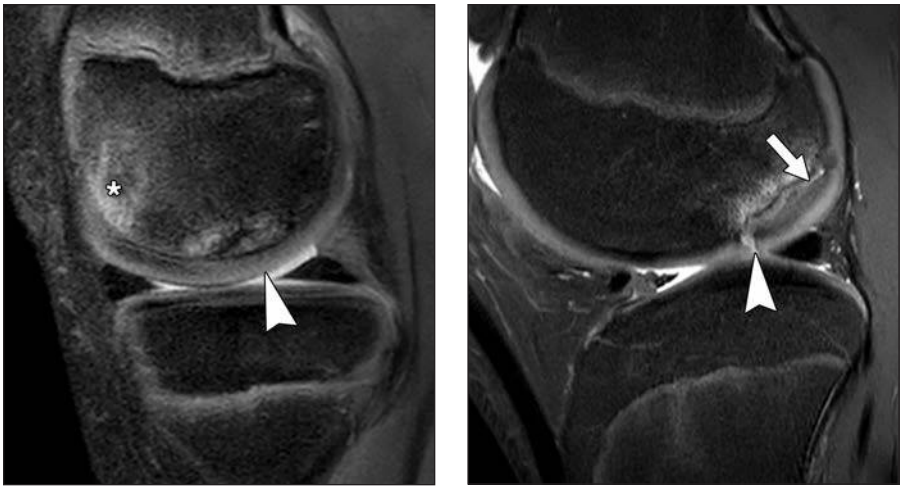

Fig. 10—Osteochondritis dissecans (OCD).
A, 13-year-old boy with stable OCD presenting with 3 weeks of knee pain after direct blow to knee. Sagittal proton density–weighted fat-suppressed MR image shows OCD lesion of posterior weight-bearing surface of medial femoral condyle with intact overlying cartilage (*arrowhead*). Bone contusion (*asterisk*) from recent trauma is evident more anteriorly.
B, 14-year-old boy with unstable OCD with 2 months of lateral knee pain and popping. Sagittal proton density–weighted fat-suppressed MR image shows OCD lesion of posterior aspect of lateral femoral condyle with anterior disruption of cartilage and bone plate (*arrowhead*) and fluidlike signal intensity (*arrow*) at interface between lesion and parent bone.

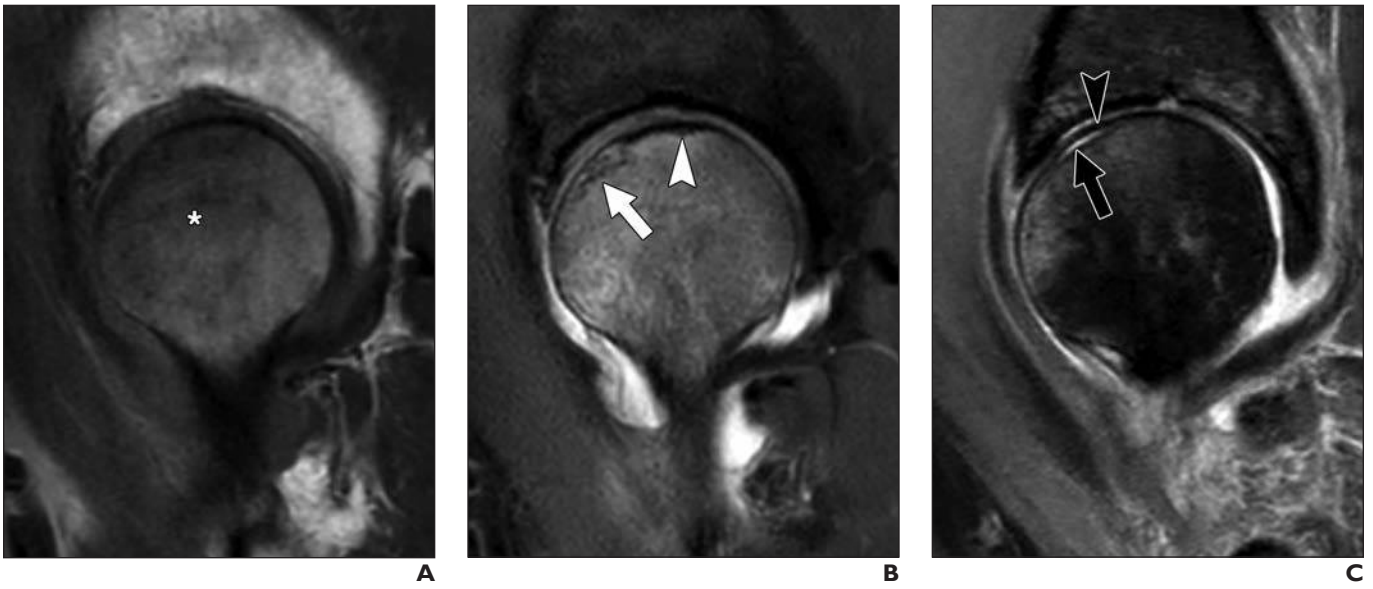

Fig. 11—Subchondral insufficiency fracture (SIF).
A and **B,** 48-year-old man with SIF of femoral head. Sagittal T1-weighted (**A**) and proton density–weighted fat-suppressed (**B**) MR images show open-ended line of low signal intensity (*arrow*, **B**) running at short distance from subchondral bone plate with bone marrow edemalike signal intensity (*asterisk*, **A**) extending from articular surface. In posterior aspect, line runs in proximity to bone plate (*arrowhead*, **B**), creating subchondral hypointense area.
C, 68-year-old woman with collapse in femoral head SIF. Sagittal proton density–weighted fat-suppressed MR image shows fluid cleft (*arrow*) under subchondral bone plate (*arrowhead*).

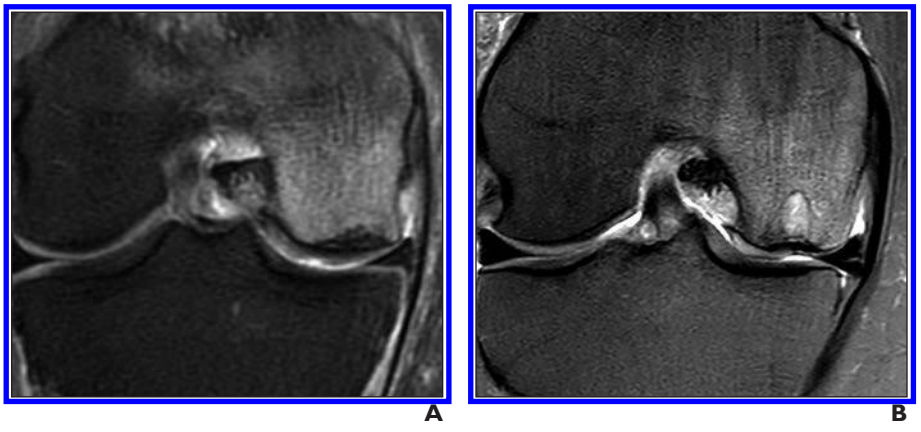

Fig. 12—Subchondral insufficiency fracture (SIF) of medial femoral condyle in different patients with spectrum of imaging findings of SIF of knee. Characteristic extensive bone marrow edemalike signal intensity involves nearly entire femoral condyle in all patients.
A, 53-year-old woman. Coronal proton density–weighted fat-suppressed MR image shows subchondral hypointense area.
B, 68-year-old woman. Coronal proton density–weighted fat-suppressed MR image shows articular collapse and subchondral cystlike changes.
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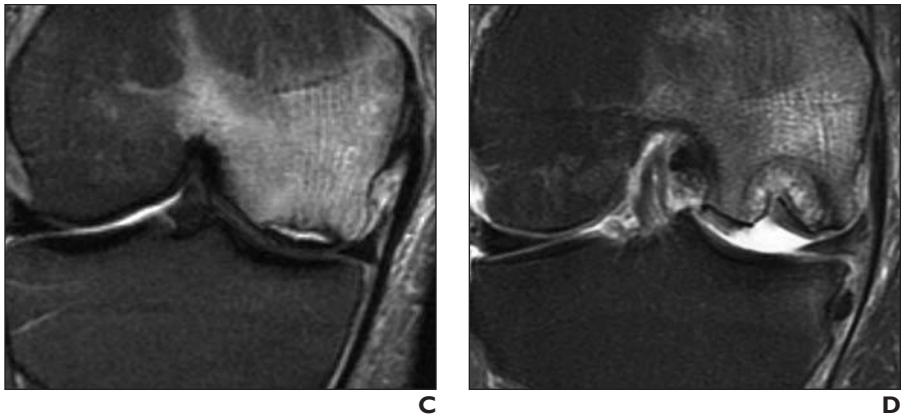

Fig. 12 (continued)—Subchondral insufficiency fracture (SIF) of medial femoral condyle in different patients with spectrum of imaging findings of SIF of knee. Characteristic extensive bone marrow edemalike signal intensity involves nearly entire femoral condyle in all patients.
C, 56-year-old man. Coronal proton density–weighted fat-suppressed MR image shows collapse with subarticular fluid cleft.
D, 88-year-old man. Coronal proton density–weighted fat-suppressed MR image shows collapse with subchondral fractures involving bone plate.

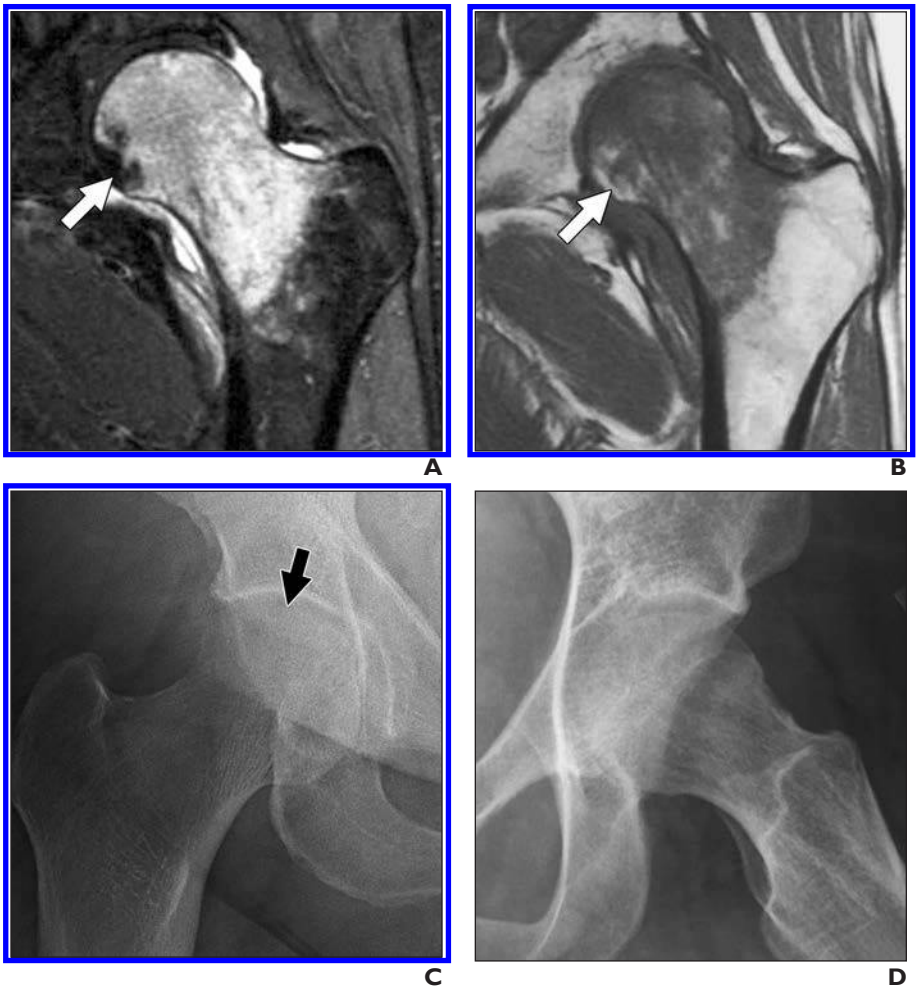

Fig. 13—Transient osteoporosis of hip (TOH).
A and **B,** 44-year-old man. Coronal T2-weighted fat-suppressed (**A**) and T1-weighted (**B**) MR images show classic bone marrow edemalike signal-intensity pattern of transient osteoporosis of hip. No subchondral abnormalities are present in **A**. Images also show classic sparing of medial aspect of femoral head (*arrow*).
C and **D,** 48-year-old man (**C**) and 26-year-old woman (**D**) with TOH. Radiographs of hip show osteoporosis of femoral head with prominent rarefaction of subchondral bone (*arrow*, **C**) resulting in near disappearance of femoral head contour. Preserved joint space and normal acetabular contour are evident.

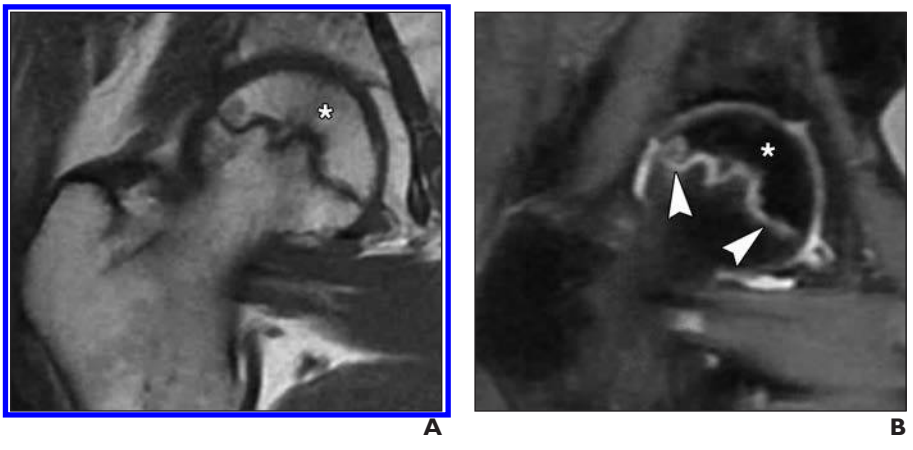

Fig. 14—49-year-old woman (**A** and **B**) and 50-year-old man (**C** and **D**) with osteonecrosis of hip.
A–D, Coronal T1-weighted (**A** and **C**) and proton density–weighted fat-suppressed (**B** and **D**) MR images show subchondral lesions consisting of area of preserved marrow signal intensity (*asterisk*, **A–C**) outlined by peripheral smooth high-signal-intensity rim (*arrowheads*, **B** and **D**). Necrotic area in **A** and **B** involves marrow immediately subjacent to bone plate, whereas in **C** and **D** necrotic area is circular and located at short distance form bone plate. Rim is smooth and completely encircles infarcted area without interruption. No diffuse edemalike marrow signal intensity is present, in contradistinction to osteonecrosis accompanied by collapse (Figs. 6A–6C).

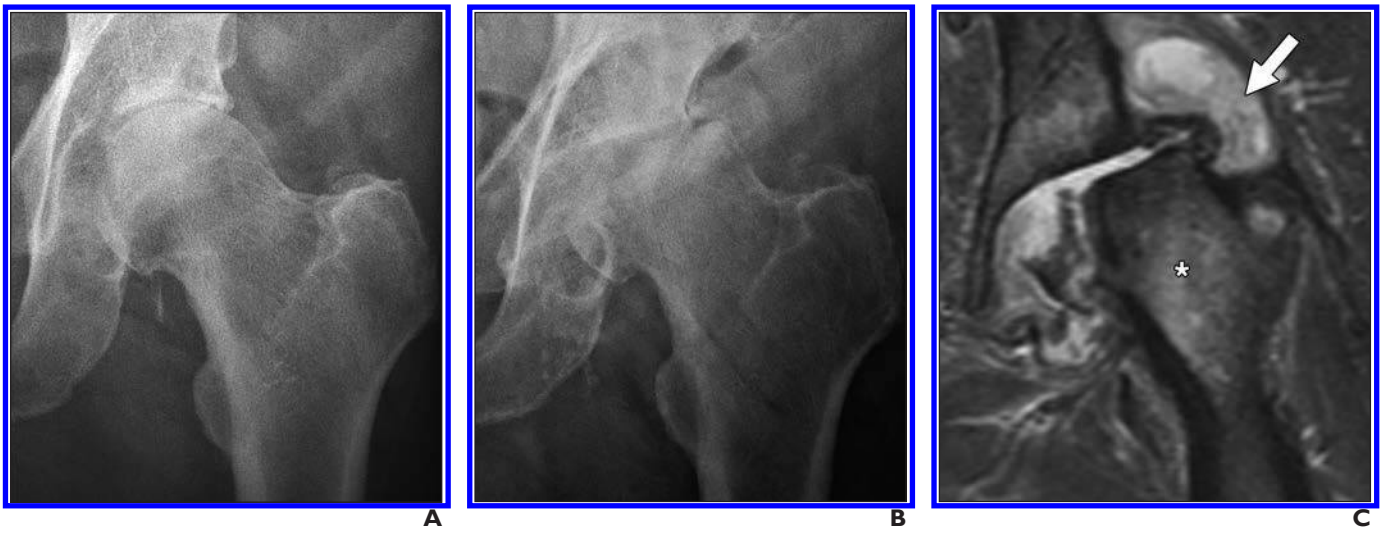

Fig. 15—64-year-old woman with rapidly progressive idiopathic arthritis of hip. (Courtesy of Song A, Loyola University Medical Center, Maywood, IL)
A, Initial frontal radiograph of hip shows moderate narrowing of hip joint space and small osteophytes.
B, Follow-up frontal radiograph of hip obtained 5 months after **A** shows marked interval progression of joint space loss with atrophic-appearing femoral head destruction.
C, Coronal STIR MR image of hip obtained at same time as **B** shows markedly abnormal bone marrow edemalike signal intensity (*asterisk*) extending to intertrochanteric line with complex joint effusion and synovitis (*arrow*).

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