# Traumatic Bone Bruises in the Athlete’s Knee

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In 1988, Wilson et al introduced the term bone marrow edema in describing a group of patients with atraumatic debilitating knee and hip pain. On T2-weighted magnetic resonance (MR) images, they recognized an ill-defined hyperintensity in the bone marrow where standard radiographs showed nonspecific osteopenia or normal findings. This discrepancy in the imaging studies was labeled bone marrow edema owing to the "lack of a better term and to emphasize the generic character of the condition." The proposed pathogenesis of these marrow changes asserts that increased blood pooling, edema, reactive hyperemia, and possible microfracture of the trabecular subchondral bone alter the marrow signal intensity. In time, bone marrow edema earned the moniker bone bruise to reflect its traumatic nature.

The ability to identify a bone bruise is unique to MR studies. On T1-weighted images, the alterations in the subchondral bone marrow signal are characterized by ill-defined low signal intensity, compared with the unaffected bone marrow. In contrast, on T2-weighted, proton density–weighted, fat-suppressed fast spin echo or short tau inversion recovery images, these lesions are characterized by areas of high signal intensity. The hypervascularity in the involved areas is clearly illustrated as the administration of intravenous contrast agents results in further enhancement of these bone marrow signal alterations.

However, the term bone bruise should be applied exclusively to subchondral lesions that exhibit the typical MR findings. These lesions are acute traumatic noncystic areas of bone marrow edema, and they can be distinguished from cystic lesions and atraumatic presentations. The pathophysiology of bone marrow edema is currently nonspecific, and...
differentiation of the different causes can be difficult. In particular, distinguishing among osteochondritis dissecans, spontaneous osteonecrosis of the knee, and idiopathic transient bone marrow lesion syndrome is difficult because ischemic and microtraumatic factors both contribute to their presentation.32 Although many causes may result in bone marrow lesions (Table 1), this review addresses acute traumatic noncystic bone marrow edema (bone bruises).

### TRAUMATIC BONE BRUISES: ANTERIOR CRUCIATE LIGAMENT–RELATED INJURIES TO ARTICULAR CARTILAGE, SUBCHONDRAL BONE, AND MARROW

A bone bruise is clinically concerning: Not only has the geography of the lesion been used to estimate lifetime risk of osteoarthritis, but bone bruise was believed to be associated with knee pain after anterior cruciate ligament (ACL) tear.25 A bone bruise is associated with more than 80% of complete ACL ruptures, and a geographic lesion is present in one-third of these patients.2,25,26 Geographic lesions have a discrete focus of low signal intensity on a T1-weighted image that is confluent and in contiguity to the subchondral plate. (Other types are reviewed in the discussion of occult fractures.) At the time of ACL rupture, bone bruising results from the impact and translation of the femur on the tibia.2,25,26 The resulting phenomenon constitutes a “footprint” left at the time of the rupture, a telltale signature of the injury.23 The characteristic bone bruise involves the posterior aspect of the lateral tibial plateau and the lateral femoral condyle at the sulcus terminalis (Figure 1).

Although the lateral compartment of the knee is commonly involved, marrow changes can be seen in the medial compartment.20,16,22,15,26 As such, a bone bruise is a static representation of the injury and may be used to gain insight into the mechanism of rupture.23 The extent of the anterior translation of the tibia on the femur can be appreciated by the relative location of the bruising in the sagittal plane.

### CONTACT VERSUS NONCONTACT ACL RUPTURES

Contact ACL injuries occur when a valgus load is applied to a planted or fixed leg, resulting in the impaction of the lateral femoral condyle into the lateral tibial plateau.3 In contrast, noncontact ACL injuries can result from quadriceps loading of a slightly flexed knee, which culminates in a valgus moment about an internally rotated tibia.3,6,14,19 Noncontact injuries were traditionally hypothesized to result from a pure valgus force, leading to bruising mainly in the lateral compartment. However, prospective data have revealed that the injury pattern in noncontact ACL injuries is the product of anterior translation affecting the medial and lateral compartments.29

In an evidence-based medicine level II cohort, Viskontas et al investigated how the degree of bone bruising was related to the energy of the injury (contact versus noncontact) and the prognosis of knee function.29 They found that bone bruising was “more frequent, deeper, and more intense” on the lateral tibial plateau in the noncontact injuries. Additionally, medial compartment bone bruising was seen more frequently than previously reported, particularly in the noncontact group.

In the short term, the clinical significance of bone bruising is unclear. In 2007, an evidence-based medicine level II cohort study found no relationship between the presence of bone bruise and pain severity.11 A more recent cross-sectional cohort study found that the presence of a bone bruise on MR imaging concurrent with an ACL tear is not associated with patient-reported pain at time of index ACL reconstruction.8 The typical bone bruise pattern on a T2-weighted MR image resolves

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**Table 1. Differential diagnosis of bone marrow lesions.**21

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<tr>
<th>Category</th>
<th>Lesion Description</th>
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<tr>
<td>Traumatic</td>
<td>Contusions (subchondral impaction)</td>
</tr>
<tr>
<td></td>
<td>Fractures (chondral, subchondral, osteochondral, insufficiency, stress)</td>
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<tr>
<td></td>
<td>Spontaneous osteonecrosis of the knee</td>
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<td></td>
<td>Overuse (repetitive microtrauma)</td>
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<tr>
<td>Nontraumatic</td>
<td>Vascular (periarticular bone infarction)</td>
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<tr>
<td></td>
<td>Osteoarthritis associated</td>
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<td></td>
<td>Transient bone marrow edema syndrome</td>
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<td></td>
<td>Osteochondritis dissecans</td>
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<td></td>
<td>Reactive inflammatory (polyarthritis, reactive arthritis, bacterial arthritis, osteomyelitis)</td>
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<tr>
<td>Tumor / malignant marrow infiltration</td>
<td>Lymphoma, multiple myeloma, etc</td>
</tr>
<tr>
<td>Other</td>
<td>Postoperative, subchondral cysts, red marrow</td>
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However, the long-term significance of bone bruise with ACL tear has not been studied in a multivariable analysis. In a longitudinal 12-year follow-up of a prospective ACL reconstruction cohort, the presence of bone bruise did not correlate with lower patient-reported International Knee Documentation Committee scores. Owing to limited sample size, a multivariable analysis was not possible. This difficulty is compounded by the inability to predict future osteoarthritis using MR imaging.

In a qualitative investigation of 10 consecutive ACL-deficient patients with a concomitant geographic bone bruise, biopsy of articular cartilage overlying a bone bruise before ACL reconstruction revealed histologic evidence of chondrocyte damage in 35% of patients. Direct inspection during arthroscopy by the authors revealed softened cartilage with dimpling, fissuring, or overt chondral fracture. On histologic examination, chondrocyte degeneration was identified: loss of toluidine blue staining consistent with the loss of proteoglycan. Additional evidence of articular cartilage surface thinning has been seen on follow-up MR imaging in the area of the bone contusion from 1 to 6 years after the initial injury. Repeat MR imaging has demonstrated persistence of the bony lesion in some patients as late as 12 months after injury. When the most severe lesions have been followed longitudinally, thinning of the articular cartilage with depression has been observed more than 2 years following injury, whereas 29% of patients demonstrated residual sequelae of the original osteochondral lesion.

Future clinical studies are required to determine if the presence of a bone bruise is a risk factor for poorer patient-reported outcomes secondary to initiation of posttraumatic osteoarthritis.

**Bone Contusions Related to Patella Dislocation**

Like ACL injuries, bone bruise presentation following patella dislocation occurs in a unique and predictable pattern on MR imaging. After an acute lateral dislocation, the impact results in focal marrow edema on the lateral aspect of the lateral femoral condyle and on the inferomedial patella (Figure 2). Increased signal is not present on the distal end of the lateral femoral condyle at the sulcus terminalis or on the lateral tibial plateau, distinguishing a patella dislocation from an acute ACL tear. On occasion, an osteochondral fracture of the lateral femoral condyle may accompany the dislocation. Careful inspection of the patella may reveal an osteochondral defect or a bone contusion related to the dislocation/relocation event on patella, often involving the medial or odd facets. The axial MR images may illustrate the depth of the lesion and the involvement of the subchondral plate, advancing understanding of the injury and its treatment options.

**Occult Fractures**

Whereas ACL ruptures and patella dislocations produce bone bruises, bony contusions are associated with subclinical, or occult, fractures. This type of traumatic injury is not visible on plain radiographs and has been classified on the basis of its MR appearance (see Table 2). Vellet’s system separates occult
fractures into 3 groups with subtypes: occult subcortical, stress, and osteochondral fractures. As a subtype, a reticular lesion has serpiginous regions of diminished T1-weighted signal intensity distant from the subchondral bone plate, whereas a geographic lesion has a discrete focus of low signal intensity on a T1-weighted image that is confluent and in contiguity to the subchondral plate. Linear subcortical fractures have a discrete linear zone of diminished signal intensity on the T1-weighted images that is usually less than or equal to 2 mm wide, with a sharp zone of transition to the adjacent marrow fat. When depression of the articular surface occurs in conjunction with a geographic-type lesion, it is defined as an impaction fracture. However, if a geographic lesion has a discrete low-intensity interface that separates the lesion from the surrounding trabecular bone and communicates with the joint space, it is best described as an osteochondral fracture. Using this classification system, researchers of an evidence-based medicine level IV study found that isolated subcortical trabecular lesions had a favorable short-term recovery after restricted weightbearing and initial activity modification. Most athletes returned to sport within 6 months. Although several studies have indicated that blunt articular cartilage injury alters future cartilage metabolism, the long-term functional significance of MR-identified trabecular injury is not known. In middle-aged athletes, the presence of a subchondral fracture has been associated with the development of spontaneous osteonecrosis.

CONCLUSIONS

Bone marrow edema is identified with MR imaging and may result from traumatic or atraumatic causes. When acute trauma produces a subchondral lesion with low T1-weighted and high T2-weighted signal intensity, the resulting bony contusion is best described as a bone bruise. Table 3 shows these traumatic bone bruise patterns, mechanism of injury, and associated injuries. Predictable patterns of bone edema accompany contact and noncontact ACL ruptures, as well as patella dislocations. However, the long-term significance of the lesions is not known. Increased marrow edema can also be associated with an occult fracture.
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