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Natural history of bone bruises after acute knee injury: clinical outcome and histopathological findings

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Abstract The purpose of this paper is to review the scientific literature on the natural history of bone bruises and the experimental studies regarding the histopathological effects of impaction load on articular cartilage and subchondral bone. Bone bruises with subchondral or osteochondral injuries, or geographic bone bruises seemed to be persistent for years after trauma on MRI. Biopsy samples of the articular cartilage overlying the bone bruise lesions showed degeneration or necrosis of chondrocytes and loss of proteoglycan. Experimental studies using a single impact load revealed chondrocytes death, alteration of the mechanical properties of cartilage explants and/or an increase in the thickness of subchondral bone. These data are indicative of a significant injury to normal articular cartilage homeostasis, and support the suggestion that severe bone bruise is a precursor of early degenerative changes. We recommend delaying return to full weightbearing status when a severe bone bruise is detected to prevent further collapse of subchondral bone and further aggravation of articular cartilage injury.

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Introduction

Occult injuries to the bone detectable only on magnetic resonance imaging (MRI), often referred to as bone bruises or bone contusions, occur in about 80% of patients who have sustained an acute anterior cruciate ligament (ACL) ruptures of the knee [11, 18, 49, 51, 56]. These lesions have also been noted after acute medial collateral ligament (MCL) injury [36], posterior cruciate ligament (PCL) injury [34] and in patients without ligamentous injury [49, 57]. It is also claimed that bone bruises are not uncommon after severe ankle sprains [44] or wrist trauma [28].

Bone bruises detected by MRI have been a subject of interest since the first description [58]. These lesions demonstrate decreased signal intensity on proton-density or T1-weighted images and increased signal intensity on T2-weighted images. This appearance is thought to represent areas of hemorrhage, edema, or infarction secondary to trabecular microfractures. These osseous injuries may be the results of a direct blow to the bone, compressive forces of adjacent bones impacting one another, or traction forces that occur during an avulsion injury.

The most common location of these lesions in patients with ACL injury is within the lateral compartment of the knee, on the lateral femoral condyle at the sulcus terminalis and the posterolateral tibial plateau (Fig. 1). The location of bone bruises seems to be specific to the injured ligament or ligament complex. Investigating bone bruise locations is one possible

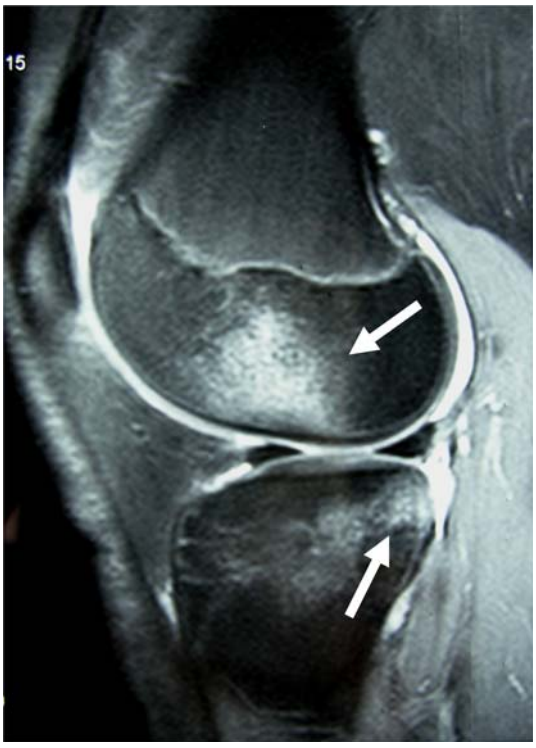

Fig. 1 Sagittal image of the knee in a patient with ACL injury shows bone bruises in the lateral femoral condyle at the sulcus terminalis and the posterolateral tibial plateau

approach to understand the mechanisms of knee injury [18, 29, 51, 52, 56].

Knee ligament injury, especially ACL injury, has long been associated with early occurrence of degenerative arthritis. Cartilage degeneration was originally believed to be due to abnormal knee kinematics and ligament insufficiency. However, even following ACL reconstruction, recent studies have shown that as many of half of the patients will develop radiological degenerative changes within less than 10 years after an ACL injury [9, 16, 39, 40]. These findings suggest that the initial injury to the articular cartilage and subchondral bone may play a role in predisposing the knee to degenerative changes. The present review summarizes and discusses the available literature on the natural history of bone bruises and experimental studies regarding the histopathological effects of impaction load on articular cartilage and subchondral bone to answer the following questions:

1. Is a large bone bruise in a knee joint a precursor of late degenerative arthritis?
2. Should we change our rehabilitation protocol for a patient who has a large bone bruise in a knee joint?

Natural history of bone bruises

It is likely that the etiology of post-traumatic knee arthritis is multifactorial. These degenerative changes may be the result of the initial trauma, meniscal injuries, surgery, joint instability, or other factors. It has been speculated that bone bruises present on MRI may lead to sclerosis of the bone resulting in degeneration of the overlying articular cartilage [8]. However, the natural history of bone bruises remains unknown and has been a subject of great interest since it was first described [58].

Some researchers [18, 36, 51] have suggested that the bruised area is resolved within a few months of the acute injury in all cases. Graf et al. [18] investigated knee MRIs from 98 consecutive patients with clinically diagnosed ACL injuries. They reported that in 71% of the MRIs taken within 6 weeks of injury a bone bruise could be demonstrated, whereas there were no findings of bone bruises on MRIs taken longer than 6 weeks after injury. In a prospective MRI study of 65 patients with grade II or III MCL injuries, Miller and coworkers [36] identified bone bruises in 45% of their patient population. Complete resolution of these lesions was observed in all cases on a repeat MR examination 6–12 weeks after the initial injury. Consequently, they concluded that bone bruises associated with MCL injury are less severe than those associated with ACL injuries.

In contrast to these findings, several studies [3, 56] suggest that some of these lesions might provide evidence of osteochondral sequelae on MRI 6–12 months after injury. Vellet et al. [56], in a prospective study of 120 patients who had acute post-traumatic hemarthrosis of the knee, found 86 patients who had bone bruises on MRI. On follow-up 6–12 months post-injury, resolution with no apparent sequelae at the site of the associated *reticular* bone bruises was demonstrated in all cases. Reticular bone bruises indicate hemorrhage and edema in medullary bone not continuous with the cortical bone of the subadjacent articular surface, and represented 70% of the bone bruises in their study group. However, two-thirds of their patients had evidence of osteochondral sequelae at the site of initial geographic bone bruises. Geographic bone bruises were characterized by increased density and immediate continuity to adjacent cortical bone, and occurred in 25% of their patients. These sequelae included osteosclerosis, apparent cartilage thinning, overt cartilaginous loss or defect, osteochondral defects and cortical impaction with or without abnormal cortical bone.

More recent studies have shown longer-term results of bone bruises. Six years after acute ACL injury and reconstruction Faber et al. [14] studied 23 patients with who initially had normal plain radiographs. However, all their patients had sustained a geographic bone bruise of the lateral femoral condyle. They found that 15 patients (65%) demonstrated persistent MRI evidence of osteochondral sequelae in spite of ACL reconstruction. Costa-Paz et al. [8] investigated 21 patients with ACL ruptures that were reconstructed and followed-up with MRI for a minimum of 2 years. They analyzed the MRI scans using a three-level grading system based on the appearance and location of bone bruises. Type I was defined as diffuse signal with change of the medullary component, often reticular and distant from the articular surface. Type II was defined as a localized signal with continuity to the subjacent articular surface. These are usually crescentic lesions with variable thickness. Type III was defined as disruption or depression of the normal contour of the cortical surface often associated with a type II lesion. In this study, they showed resolution of all type I lesions and 91% of type II lesions. In contrast, all five patients with type III lesions had evidence of persistent abnormality on MRI scans, consisting of cartilage thinning or cortical depression. Based on these results, these authors concluded that a severe occult osteochondral lesion sustained at the time of ACL rupture seemed to persist on MRI, even after a successful cruciate reconstruction.

In a follow-up study regarding the volume of bone bruises, Roemer et al. [48] demonstrated a decrease in volume in all their patients after a minimum of 2 years (mean 44 months). In seven of 49 patients (14%), eight signal changes were seen on the follow-up MRI. Persistent signal alterations were observed more commonly in patients who had a more severe osteochondral injury. Moreover, Davies et al. [10] investigated 30 patients with bone bruises identified on MRI after an acute knee injury and rescanned 12–14 weeks post-injury to study the detailed patterns of bone bruise resolution. They described that bone bruises were still present in all patients at follow-up imaging. Seventeen of the 30 patients had bone bruises that extended to the joint margin, ten of these had associated osteochondral injuries on MRI. Two distinct patterns of bone bruise resolution were demonstrated. In 21 patients, the bone bruises resolved from the periphery towards the center of the bruise, while eight patients showed bone bruises that resolved towards the joint margin, all of whom had associated osteochondral injuries. In addition, the authors showed that the bone bruises with osteochondral injuries resolved more slowly than the group without osteochondral injuries.

In conclusion, bone bruises with subchondral or osteochondral injuries, or geographic bone bruises on MRI seems to be persisting for years after trauma. These persistent signal alterations may result from a change in loadbearing caused by subchondral or osteochondral microfracture. Persistent signal alterations can reflect early changes of post-traumatic degenerative disease. However, histopathological investigations are necessary to understand the effects of a bone bruise in more detail.

Histopathological studies of bone bruises

Excessive levels of mechanical load can generate matrix damage and chondrocyte death in articular cartilage. Arthroscopically, acute articular cartilage injuries that result from a single impact load seems to vary from normal or simple softening and indentation to severe fibrillation, fissuring, or overt chondral fracture [11, 33, 38]. Engebretsen et al. [11] stated that none of 28 bone bruises found on MRI after an acute ACL injury were detected by arthroscopy. In contrast, Johnson et al. [25] reported that all patients had significant arthroscopic articular cartilage irregularity in the area overlying the bone bruise. An understanding of the histopathological changes associated with a bone bruise is important in regards to future changes of articular cartilage.

Hyaline cartilage has a complex avascular, aneural structure in which chondrocytes constitute only 5% of the volume without cell-to-cell contact [5]. Chondrocytes synthesize and maintain an abundant extracellular matrix rich in proteoglycans, mostly aggrecan, and collagens [17]. In normal articular cartilage, a balance exists between synthesis and degradation of matrix components. The presence of isolated injuries to the articular cartilage of the knee is considered a risk factor for more extensive joint damage because cartilage has very limited intrinsic healing capacity. Partial-thickness defects do not heal, and full-thickness defects repair with mechanically inferior fibrocartilage [23]. A variety of methods have been developed to enhance the repair of articular cartilage defects, including abrasion arthroplasty [2, 27], microfracture [37], transplantation of chondrocytes [4, 42], perichondrium [1] and periosteum [20], and osteochondral graft [35]. There is considerable debate over the best treatment for articular cartilage defects.

Numerous researchers have studied the influence of the impaction load that can cause bone bruises on cartilage properties. Below we discuss the histopathological effects of impaction load on articular cartilage.

Biopsy of bone bruises

To analyze the effects of impaction load on articular cartilage, the best model may be to study human biopsy samples of the bone bruise lesions. Some investigators have reported the results of histopathological analysis of human biopsy samples of bone bruises and the surrounding areas [12, 15, 26, 46]. Rangger et al. [46] took biopsies and evaluated the histopathological appearance of bone bruises of the knee detected on MRI. They found microfractures of cancellous bone, edema and bleeding in the fatty marrow corresponding to the MRI findings. Johnson et al. [26] studied biopsy samples of the lesions with continuity to the subchondral bone (geographic lesions) and noted degeneration or necrosis of chondrocytes, loss of proteoglycan, and osteocyte necrosis (empty lacunae) in the subchondral bone. Their findings suggest that a geographic lesion found on MRI indicates substantial damage to normal articular cartilage homeostasis. The authors proposed that large geographic lesion should be an indication to avoid axial loading of the cartilage and underlying subchondral bone.

Fang et al. [15] performed histological and immunostaining analyses of articular cartilage/subchondral bone biopsy specimens overlying MRI-detected bone bruises in 12 patients with ACL tears. Staining with toluidine blue for proteoglycan revealed loss of staining from the superficial portion of the articular cartilage. Immunostaining for cartilage oligomeric matrix protein (COMP) showed an increased staining in the superficial matrix of the articular cartilage. COMP is an abundant noncollagenous extracellular matrix protein in cartilage [19]. Several recent reports [7, 32] suggest that COMP levels in synovial fluid may be used as a marker for cartilage turnover in human diseases. They concluded that their results are indicative of a significant injury to the articular cartilage, and may represent preclinical post-traumatic osteoarthritic lesions.

Experimental study of bone bruises

An experimental single impact load may be interpreted as simulations of acute joint trauma. Several investigators have studied the fate of cartilage with osteochondral damage using either in vivo animal model of joint trauma [41, 53, 54], or by following the changes after impaction of cartilage explants [13, 30, 31, 45, 47, 55]. After application of a transarticular load to the patellofemoral joint in a canine model, Thompson et al. [54] showed osteoarthritic-like degenerative changes at 6 months, but these had stabilized at 12 months. It is not clear if the damage seen in either of these models is sufficient for the joint to progress to full-blown OA. On the day of loading, using MRI and scanning electron microscopy, they also showed multiple, extensive fractures through the zone of calcified cartilage and the subchondral bone with little or no change in the gross appearance of the articular cartilage. Moreover, Newberry et al. [41] impacted the mature rabbit patello-femoral joint and found softening of retropatellar cartilage and an increase in the thickness of subchondral bone of the patella after 12 months. It is possible that some of those subchondral bone injuries heal into a stiffer construction than the previous normal bone, and increased subchondral bone thickness and stiffness may require the articular cartilage to absorb greater loads and subsequently lead to degeneration of articular cartilage [8, 41].

Then, what are the effects of impaction load on chondrocytes in articular cartilage? According to the clinical study with biopsies of geographic bone bruises by Johnson et al. [26], there is degeneration or necrosis of chondrocytes and osteocyte necrosis in the subchondral bone. They also reported that the presence or absence, number, and depth of the injured chondrocytes were directly proportional to the pathological changes of the articular surface.

Numerous researchers have studied cell death that resulted from a single impaction of cartilage explants [13, 30, 31, 45, 47, 55]. Torzilli et al. [55] impacted young mature bovine cranial cartilage with no bone attached at different stress levels and graded the cells as dead or live. They proposed a critical threshold for cell death in the surface layer and collagen damage from a single impact load with an applied stress of 15–20 MPa. They also reported extensive cell death in the deep layer at higher levels of nominal stress. Kurz et al. [30] developed a model that allows strain- and strain rate-controlled loading of cartilage explants to study the effects of injurious compression on the degradation and repair of calf cartilage in vitro. Their results demonstrated that a single compression altered the mechanical properties of cartilage explants, chondrocyte biosynthesis, and chondrocyte response to dynamic compression in a manner that was dependent on the strain rate of the compression. In particular, their study demonstrated that injury affects not only the basal biosynthetic activity of chondrocytes, but also the ability of subsequent low-amplitude dynamic compression to upregulate biosynthetic activity. Normal chondrocytes respond to moderate- or low-amplitude dynamic compression by upregulating biosynthetic activity [43, 50].

Post-traumatic cracking of articular cartilage over bone bruises is a regular finding in human joints. Some animal studies have demonstrated a loss of chondrocytes from the cartilage bordering the wound edge of partial-thickness defects [31, 47]. Moreover, it has been well established that surgical maneuvers involve cell death at the wound edge [22, 24]. Can cracking of articular cartilage cause chondrocytes death? Studies using cartilage explants attached to underlying bone have documented cell death around impact-induced cracks following blunt insult at high rates of loading [31, 47]. On the other hand, some studies suggest that there is no clear spatial association between cell death and matrix damage [45, 55]. Torzilli et al. [55] impacted young mature bovine cranial cartilage, with a single impact of 20 MPa in 571 ms (35 MPa/s). They documented surface fibrillation into the middle zone of cartilage directly under the indentor. Cell death was also localized under the indentor, but extended into the deep layer of the explant. Quinn et al. [45] saw no clear spatial association between cracks in the matrix and cell viability using a very low rate of loading. In response to these reports, Ewers et al. [13] postulated that ''the high rate of loading experiments resulted in cell death adjacent to fissures, whereas the low rate of loading experiments produced a more diffuse distribution of cell death away from the fissures''. In addition, they suggested that greater matrix damage was documented in explants subjected to a high rate of loading, compared to explants exposed to a low rate of loading.

These data are indicative of a significant injury to normal articular cartilage homeostasis, and support the suggestion that severe bone bruise is a precursor of early degenerative changes.

Discussion

The purpose of ACL reconstruction is not only to restore knee stability and function, but also to prevent later degenerative changes. Even following the ACL reconstruction, however, a significant percentage of patients have developed degenerative changes in 5–10 years [9, 16, 39, 40]. It is likely that the etiology of post-traumatic knee arthritis is multifactorial. Taking into account the published literature about natural history and histopathological studies of bone bruises, severe blunt injury to the articular cartilage and subchondral bone can induce early changes of post-traumatic degenerative disease. Therefore, the answer to the first question ''Is a large bone bruise in a knee joint a precursor of late degenerative arthritis?'' is that large bone bruises with subchondral or osteochondral injuries, or geographic bone bruises have a great potential for developing into late degenerative changes. These degenerative changes might be caused by a variety of factors, including necrosis or apoptosis of chondrocytes, loss of proteoglycan, damage to the collagen network and increase in thickness and stiffness of subchondral bone. Concerning chondrocyte death, Chen et al. [6] concluded that in their study about cyclic impact loading to normal cartilage, necrosis occurred first, followed by apoptosis. However, there are no long-term follow-up studies focused on the relationship between bone bruises and appearance of knee osteoarthritis. It is desirable to clarify the long-term progress of the bone bruise in the lateral compartment of the knee after the ACL injury. Moreover, there are some limitations to the experimental studies about bone bruise. One of the most serious limitations is that the amount of clinical impaction load to the articular cartilage at the time of ACL rupture is unknown. The impaction load in the study of Thompson et al. [54] could be a too small as a model for cartilage degeneration. There seems to be a certain amount of load necessary to crack the cartilage and leave a chronic injury such as the one that Torzilli et al. [55] showed.

The second question is if our rehabilitation plan should be changed when there is a large bone bruise in a knee joint. Today, early weightbearing and aggressive rehabilitation is advocated in most cases. The data presented suggest that it may be reasonable to delay return to full weightbearing when we find a large and severe bone bruise to prevent further collapse of subchondral bone and further aggravation of articular cartilage injury. This interval may allow healing to occur in the articular cartilage and subchondral bone. Unfortunately there is no available data to support or refute this hypothesis. However, Johnson et al. [25] reported, in their study of 40 cases of ACL injury including 20 individuals with geographical bone bruise and 20 without associated bone bruise, that patients with a bone bruise had more persistence of effusions, increased pain, and increased number of days required to nonantalgic gait without external aids. In addition, Hooiveld et al. [21] reported in their study using beagle dogs that the experimental joint bleeding when combined with loading of the affected joint resulted in features of progressive degenerative joint damage, whereas similar joint hemorrhages without joint loading did not. These results also suggests that avoiding or minimizing joint loading might be helpful in protecting against late degenerative changes when ACL rupture occurs. Longer-term clinical prospective studies are needed to establish a rehabilitation plan in the case of large and

severe bone bruises. Only a randomized clinical trial can provide evidence on whether delayed weightbearing can prevent or delay cartilage degeneration.

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