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### Bone Bruising and Bone Marrow Edema Syndromes: Incidental Radiological Findings or Harbingers of Future Joint Degeneration?

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#### INTRODUCTION

Increasing use of magnetic resonance imaging for musculoskeletal injuries over the last decade has alerted clinicians to "bone bruising", a phenomenon previously undetected on conventional radiographic techniques (1-12). This entity is recognised as focal signal abnormalities in subchondral bone marrow and the appearances are thought to represent microtrabecular fractures, haemorrhage and oedema without disruption of adjacent cortices or articular cartilage. Since the late 1980's, bone bruising has been increasingly identified in association with soft tissue knee injury, in particular anterior cruciate ligament rupture and, to a lesser extent, injuries of the hip joint and foot. Although some authors suspect these lesions may account for symptoms of pain and have prognostic implications, there are few substantial reports to date clarifying the short-term implications, exact time to resolution and long-term sequelae and their clinical significance is still uncertain.

Bone marrow edema syndromes with no history of trauma, are also increasingly recognised, particularly in the hip joint, but increasingly in the knee joint. Whilst initially assumed to be a precursor of osteonecrosis, the scientific evidence is conflicting and at present they are best considered closely related diseases with overlapping clinical and radiological presentations. Most recently, the presence and persistence of marrow edema patterns in early osteoarthritis are showing promise as a potential marker of actively progressive disease.

#### DIAGNOSIS OF BONE BRUISES AND MARROW OEDEMA

The subcortical marrow cavity consists of cancellous bone that usually demonstrates fatty marrow at all ages. The normal marrow signal on MRI parallels that of subcutaneous fat-high on conventional T1-weighted and intermediate on T2-weighted spin echo sequences. A typical bone bruise appears as an area of signal loss on T1 images and high signal intensity on T2 images, as a result of increased water content in the injured marrow. Further information can be gained with Short T1 Inversion Recovery ("STIR") imaging when signal from normal medullary fat is markedly suppressed and bone bruises show increased intensity ( Figs 1-2). Distinction between bone bruising and marrow oedema syndromes is primarily based on a clinical history of trauma, as the radiological features are largely indistinguishable.

#### CLASSIFICATION OF BONE BRUISES

Mink (6) was the first to identify bone bruising as a distinct entity in 1987 and several authors have since attempted to classify the lesions (3,5,12). Some confusion exists however, in distinguishing bone bruises involving only the marrow, and "occult" fractures, undetected on conventional x-ray, which breach the adjacent cortex or osteochondral surface on MRI. Vellet (12) divided true subcortical lesions into three types dependent on their characteristic bruising pattern - reticular, geographic and linear. He described reticular lesions as regions of "reticular serpiginous stranding" with variable degrees of coalescence within the marrow compartment but distant from the adjacent cortices and articular cartilage. "Geographic" lesions are characteristically large, amorphous, coalescent and continuous with the adjacent cortical bone and are the commonest type seen. Scant attention has been paid to these classifications, as they contribute little to understanding the underlying pathology.

#### INCIDENCE OF BONE BRUISING

Much of the literature to date has focused on bone contusions around the knee joint. Lynch reviewed 434 consecutive patients with acute knee injury and found an incidence of 20%, the majority (77%) associated with anterior cruciate rupture (3). Subsequent authors have almost exclusively focused their attention on the ACL injured population with strikingly consistent findings (1,2,7-11,13). Scans in the acute phase following injury consistently found more than 80% of acute ACL tears had contusions on MRI (8-10,13). Studies done which included patients scanned over a longer period had a smaller incidence ranging from 40-56% (1,2,11).

The pattern of bone contusions associated with ACL tears is very distinctive. In Spindler's series, 86% and 67% of contusions involved the lateral femoral condyle (LFC) and lateral tibial plateau (LTP) respectively and bruising of both occurred in 56%. Lesions in the medial femoral condyle (7%) and tibial plateau (21%) were less common (10). The notion that "matching" lesions in the lateral compartment reflected the valgus force on the knee at the time of injury was supported by Kaplan in her review of 100 MRIs of acute ACL tears (2). All 56 knees with contusions had posterolateral tibial lesions and this was the only finding in 43% of patients, whilst 48% had lesions in both the posterior LTP and LFC. In 200 MRIs without radiological ACL injury, posterolateral tibial bruising was found in only 3 patients, all of whom were later found to have an ACL tear at arthroscopy, suggesting that posterolateral tibial bruising is a pathognomonic sign of ACL injury. McCauley also found a high specificity of 97% for posterolateral plateau contusions alone and

100% when combined with lateral femoral condyle contusions as markers of ACL injury (4).

The preponderance of contusions in the lateral compartment with ACL rupture correlates with the mechanism of injury. The tibia subluxes anteriorly relative to the femur, the lateral plateau subluxing more than the medial side. If this traumatic "pivot shift" occurs with enough axial and valgus force, it is conceivable that a unique pattern of "kissing-contusions" may occur in the middle (weight-bearing) portion of the LFC and the posterior aspect of the LTP as the bones are compressed against one another. The posterior aspect of the LTP may be structurally weaker than the LFC and therefore injured most often. Kaplan's finding of the invariable presence of posterolateral tibial bruising would support this theory. It has also been shown that some injury of the popliteus-arcuate capsuloligamentous complex is commonly associated with bone contusions of the posterior LTP (13), lending support to contemporary opinion by clinicians that subtle injury to the posterolateral corner is frequently overlooked and may account for less optimal outcome after ACL reconstruction.

Murphy made some interesting observations by distinguishing between complete and partial tears of the ACL (7). Whilst bruising of the posterolateral tibia (94%) and lateral femoral condyle (91%) was common with complete tears, only 17% of patients with partial tears had contusions. He suggested that the presence of these MRI lesions indicates ACL "insufficiency" and may influence decisions about reconstruction. These findings were supported by Zeiss, who found that 80% of "partial tears" with contusions were high grade injuries that eventually led to complete rupture within 6 months (14).

Whilst the pattern of lateral joint contusions is explained by axial and valgus load, the mechanism of medial tibial plateau injury is not clearly understood. In 25 patients with medial tibial plateau contusions, all associated with ACL rupture and lateral lesions, Kaplan found consistent injury to the posterior horn of the medial meniscus or meniscocapsular junction (15). She suggested contusions of the posteromedial lip of the tibia result from a "contrecoup" impaction injury as the knee reduces and they imply associated medial meniscal injury. Speer observed a relatively high incidence (29%) of MTP lesions in alpine skiers (9). It has been proposed that, at the moment of ACL rupture, the skier's knee is non-weight bearing and the ultimate tearing force is rotational. The expected number of contusions from axial loading should be less, but this is not supported in Speer's group. However, the higher incidence of posteromedial tibial plateau contusions and meniscal injury may reflect a different mechanism of injury in skiers and requires further investigation. The mechanism behind infrequent anteromedial femoral condyle contusion with ACL rupture is also open to question. Recent work has implicated associated clinical disruption of the posterolateral corner but this theory needs substantiation in large studies (16).

Recent literature has also highlighted contusions with other "non-bony" knee injury. Miller reported an incidence of 45% associated with medial collateral ligament injury, almost all lesions involving the lateral femoral condyle (17). Contusions of the lateral femoral condyle (81-100%) and the medial patella (30%) after patellar dislocation (18-21) and "isolated" bone bruising with uneventful resolution (22) are both recognised. Traumatic hip dislocation has also been implicated in lesions of the femoral head (23) and the ipsilateral knee from dashboard impaction (24). Lesions of the talus and medial malleolus occur in up to 40% of lateral ligament ankle sprains (25-29) and bilateral calcaneal contusions after axial loading has also been reported (30).

#### **CLINICAL, OPERATIVE AND HISTOLOGICAL FINDINGS**

Difficulty arises in identifying clinical signs and symptoms directly attributable to the bone bruising, because of the spectra of associated injuries. However, patients with contusions appear to have a more protracted clinical recovery, with greater effusions and pain scores at matched time intervals and a slower return of motion (31).

Arthroscopic evidence of damage to the joint surface overlying contusions is not universally supported in the literature. Several authors found no arthroscopic evidence of osteochondral lesions corresponding anatomically with contusions in the acute phase (1,12). Coen described normal joint appearance but "dimpling" of the cartilage over geographic femoral bruises when probed (32). Several authors describe articular lesions later at the time of ACL reconstruction (9,10). Speer found a small incidence of fissuring (6%) overlying lateral femoral condyle and posterolateral tibial plateau contusions (9). Although Spindler found 46% of patients had articular lesions, many did not correlate with contusion. The only significant relationship was in the lateral femoral condyle where 40% of contusions had an overlying lesion (10). In contrast, Johnson consistently found evidence of articular cartilage injury over femoral condyle contusions, varying from subtle indentation when probed, to severe fibrillation, fissuring or overt chondral fracture (33).

Some interesting histological information has arisen from biopsy at varying time periods (33-35). In acute lesions, Rangger found microfractures of the trabecular bone, oedema and bleeding in the fatty marrow (34). In Johnson's series, all patients had articular cartilage and subchondral bone changes at ACL reconstruction. Chondrocytes in the superficial zone of the articular cartilage showed different stages of degeneration, and loss of matrix proteoglycan and variable osteocyte necrosis in the underlying subchondral bone was noted (33). Fang supported the evidence for proteoglycan loss and also found a 10-fold increase in matrix protein degradation products in the synovial fluid from injured compared with uninjured knees (35). This clinical data supports previous animal studies suggesting blunt trauma to articular cartilage produces profound changes in its histologic, biochemical and ultrastructural characteristics in the absence of surface disruption (36,37) and lends scientific evidence to the notion that bone bruising may be a precursor of posttraumatic arthritis.

#### **RESOLUTION OF BONE BRUISING**

Few studies to date address resolution of bone contusions or long-term sequelae. Vellet demonstrated complete resolution of MRI contusions at 6-12 months but osteochondral sequelae in 67% of lateral femoral condyle lesions (12). The commonest finding was an overt cartilage defect (48%), but features of osteosclerosis, cartilage thinning and osteochondral defects were also seen. No articular defects occurred over associated reticular bruises in the posterolateral tibial plateau. Bretlau recently reported persistent bruising in 69% and 12% of patients rescanned at 4 and 12 months respectively (38). Miller, in contrast, suggested the majority of lesions resolved in 2-4 months but his study involved patients with isolated medial collateral ligament injury, the benign nature of which may have influenced the rate of recovery (17).

Much anecdotal evidence that contusions resolve within the first few months is inferred from earlier studies on incidence. In Graf's series with a 48% incidence of contusions, no lesions were seen in scans later than 6 weeks (1). Tung reported a significantly shorter interval from injury to MR imaging when bone bruising was present (mean 4.3 weeks) than in those with normal medullary signal (mean 24 weeks) (11). Dimond found scans were consistently negative for contusions by 6 months, but showed a greater incidence of meniscal tears and chondromalacia (39). Whether these are

secondary injuries from instability, or indeed sequelae of a resolved bone bruise is open to question. Two year follow-up studies suggest that 10-15% of patients have persistent marrow oedema at 2 years and up to one third have some evidence of subchondral osteonecrosis or articular cartilage degeneration (40,41).

### **BONE MARROW EDEMA SYNDROMES**

The first use of the term "bone marrow edema" was by Wilson and collaborators in 1988 (75). They found ill-defined bone marrow hyperintensities on T2-weighted MR images in patients with debilitating knee and hip pain. Corresponding standard radiographs were normal or demonstrated non-specific osteopenia. The authors termed this condition bone marrow edema because of "the lack of a better term and to emphasize the generic character of the condition". According to findings of a Medline search starting at 1966, this term was not mentioned before 1988 in the radiology or pathology literature, which probably relates to the fact that MR imaging was not used widely for musculoskeletal disease before the mid-1980s. However, Roemer et al. (41) conclude that widely used term BME should be replaced by "ill-defined signal intensity" as there are many similar but unrelated and non-specific MRI abnormalities. They claim that post-traumatic osteonecrosis, as reported in the literature, must be a rare event after acute knee trauma.

Whilst marrow edema is a recognised non-specific finding in pathologies such as infection, neoplasms and avascular necrosis, the phenomenon of transient bone marrow edema syndrome (BMES) has received much attention in recent years. Most commonly seen in the hip joint, it was initially thought to be synonymous with transient osteoporosis and a possible precursor of avascular necrosis. However, the onset of radiological osteopenia within weeks of clinical symptoms distinguishes transient osteoporosis from BMES, although both are characterised by complete recovery, without intervention.

It has been suggested that transient osteoporosis or the bone marrow edema syndrome may be the initial phase of femoral head osteonecrosis but there is little radiological or histological evidence to date to support this hypothesis. In a series of 200 hips, Kim could not identify a bone marrow oedema pattern on MRI in the early stages of femoral head necrosis (42). Structural damage of the head seemed to result in the later appearance of marrow edema and the development of pain, suggesting that the edema pattern is a secondary reaction associated with the inflammatory response to subchondral fracture.

Various authors have treated BMES of the hip with core decompression, showing marked acceleration in recovery compared with conservative measures (43-45). Interestingly, no osteonecrosis followed with either treatment. Histological examination of the core specimens from the earliest series suggested evidence of early necrosis (46). However, recent studies report edema without osteoporosis or osteonecrosis (43-45). Increased osteoblast activity and transient decrease in mineral density are described but osteoclast resorption is rarely seen. Live trabeculae and active bone formation, however, infer increased repair capacity and may explain the spontaneous reversibility of this syndrome.

The pathophysiological event that triggers BMES is still a complete enigma. Ischaemia has been suggested as the initiating factor. Koo reported angiographic findings of increased femoral head perfusion suggesting a vasomotor response in the pathogenesis (47). The present consensus of opinion is that bone marrow edema syndrome, transient osteoporosis and avascular necrosis may have a common pathophysiology but are distinguished by the early potential for reversibility.

The recent increasingly frequent findings of non-traumatic bone marrow edema in the knees and feet of asymptomatic athletes (48,49) and others with pain (50,51), suggests that BMES is more common than previously recognised.

MR imaging is sensitive to changes in subchondral bone marrow (especially if fat suppression is used), which are difficult, if not impossible, to assess arthroscopically. Those changes reflect changes in overlaying cartilage, but the nature of bone lesions remains unclear. In recent years advances in MRI imaging of articulating surfaces have shown previously unknown subchondral bone changes following cartilage repair: edema-like signal, cysts, irregularity of subchondral bone plate, mismatch with adjacent plate, intralesional osteophytes, etc. Persistent BME and pain following autologous chondrocyte implantation (ACI) repair seem to suggest complications or failed repair. Abnormal subchondral bone marrow signal after cartilage repair is associated with failure of repair tissue integration at different levels. Failed integration of the repair tissue to bone appears as diffuse edema-like signal, while failed integration of the repair tissue to adjacent cartilage appears as focal edema-like signal, fissures, cysts, etc. In summary, early edema-like MRI changes may reflect normal healing, while persistently abnormal marrow signal usually indicates a problem with cartilage repair (76).

### **BONE MARROW EDEMA AND OSTEOARTHRITIS**

Recent studies provide some evidence that marrow edema in osteoarthritis joint is strongly associated with both pain and disease progression. In a large series of arthritic knees, Felson found bone marrow lesions in 77.5% of patients with pain, compared with 30% in those without pain (52). In a further study, he suggested that BME lesions increase the risk more than six-fold for disease progression at a year (53). Pessis also examined the predictive value of subchondral edema and found no patient without oedema on initial MR assessment but 40% of those with lesions developed worsening chondropathy at one year (54).

Cartilage degeneration, although fundamental to the pathogenesis of osteoarthritis, is not the site of origin of pain, which is the predominant symptom of osteoarthritis. Patients with osteoarthritis of the knee often report no or minimal pain while walking but considerable pain after activity, especially at night. These delayed responses can be explained by findings such as those reported by Felson and colleagues - they reflect the time it takes for the marrow spaces to react. Edema of the bone marrow has also been observed in patients with painful, transient regional osteoporosis, which is usually symptomatic for 6 to 12 months. Periosteal edema along with marrow edema has been seen on MRI in patients with otherwise unexplained medial tibial pain after trauma. MR imaging has demonstrated other bone marrow lesions in patients with bone pain, such as those in osteoid osteoma and with sickle-cell crises. Magnetic resonance imaging can also detect early subarticular erosions in rheumatoid arthritis.

Impaired venous drainage from the bone marrow has been suggested as a cause of pain in patients with osteoarthritis, since the resulting venous hypertension would increase intraosseous pressure in the closed spaces of the bone marrow compartments. Such venous hypertension would contribute to the development of marrow edema and may be an aspect of the phenomenon that Felson and colleagues observed. The development of venous hypertension and bone marrow edema may also be related to the development of cysts in the subchondral bone in osteoarthritis. These observations may explain pain that occurs before OA changes are visible on standard radiographs. Ordinary radiographs show the effect of degeneration of joint

cartilage as narrowing of the space between articulating surfaces. However, patients can have considerable pain despite a normal-looking cartilage space, or pain can be mild despite marked narrowing. (77).

It is possible in the future that MR may be a useful screening tool for identifying patients with marrow oedema and high risk of arthritic progression.

## DISCUSSION

As a recently recognised entity, the natural history of bone bruising is unknown. If it represents trabecular microfracture, as the histological evidence to date suggests, one can reasonably expect bony healing of the subchondral lesion. Certainly, there is a consensus that most bone contusions heal in the short term. It has been proposed however, that increased stiffness of the healed bone may decrease the potential for the joint to dissipate load by deformation and this may also increase shear-stress at the bone cartilage interface, precipitating cartilage degeneration (55). However, it is also likely that the initial trauma insults the cartilage microstructure in its own right and the relative influence of the underlying bone bruise on chondral degeneration is still open to question. Further understanding of articular cartilage microstructure in the acute phase is needed, as evidence for chondral injury to date is based on macroscopic appearances and histology at the time of ACL reconstruction.

The high prevalence of bone bruises with ACL rupture has raised questions about its prognostic implications for this injured population. Posttraumatic arthritis is an established complication of nonoperative treatment of ACL rupture (56-66). Some radiological degeneration is seen in up to 80% of patients as early as 3 years post injury (60,62). McDaniel and Dameron found 37% of patients had profound degeneration at 14 years (63). Whilst associated meniscal injury was often considered the determining factor, several authors have shown similar rates of long-term degeneration, regardless of initial meniscal damage (64,66). Two possible explanations exist. Chronic instability may provoke secondary injury to menisci seen to be intact at the time of ACL rupture (67,68). Alternatively, initial damage to the articular cartilage may be the predominant prognostic factor and, if so, bone bruising may be the missing link. Sherman's observation that knees with concomitant medial collateral ligament injury degenerated significantly earlier than those with ACL/meniscal tear patterns could be explained by a more traumatic impact to the lateral compartment with extensive bone contusions.

The commonly held belief that a knee with a chronic ACL injury develops cartilage wear and degeneration because of instability requires further review. The literature to date shows lack of documentation that ACL reconstruction prevents degenerative arthritis (57,59,69-71). Friederich and O'Brien have shown a similar incidence of radiographic arthritis at 5-10 years in surgically and conservatively treated knees, whilst Daniel found an increased incidence of degenerative change after reconstruction (57,59). Both these studies suggest that initial injury to the articular cartilage is the predominant precursor to joint degeneration and bone bruising may be the etiological factor. Long-term prospective trials comparing matched reconstructed groups, with and without bone bruising, will clarify this issue in the future.

Most of the literature to date has focused on ACL associated bone bruises. However, the long-term manifestations of bone bruising will be difficult to clarify in these patients because of the injury complexity and we need to identify better natural history models for follow-up studies. "Isolated" contusions and those associated with medial collateral ligament injury would seem ideal. The speculation that lesions may also occur with meniscal tears must also be explored. The incidence of posttraumatic arthritis after meniscectomy has historically been attributed to increased load-bearing in the affected compartment (72-74). However, contusions in the adjacent bone may be contributory in part.

Many answers on the enigmas of bone contusions will not be answered in the short-term. Future research needs to focus on longitudinal studies to establish natural history and further investigation into the pathophysiology of the lesions and the adjacent cartilage. Until such time as long-term studies are available, as clinicians we must assume that bone bruising as a specific entity is a harbinger of posttraumatic arthritis and practice a cautious approach to management of associated knee injuries.

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