For athletes, a speedy recovery from injury is vital. This article will describe how knee overuse injury and arthritis can lead to stress fractures and bone marrow lesions in the subchondral bone. We then discuss prognosis with traditional treatment options and present an emerging option – subchondroplasty – for treating bone marrow lesions. Finally, we present case studies from our practices in which sports-injury-related bone marrow lesions were treated with subchondroplasty.

Overuse, injury and osteoarthritis (OA) place undue stress on the knee joint, leading to microtrabecular fractures, visualised as bone marrow lesions on MRI. The subchondral bone is exposed to high loads, especially during exercise, when muscle-driven accelerations increase the effective load by multiples of body weight (e.g. walking 1 to 2x; running 3 to 5x; explosive jumping/landing 6 to 8x body weight). Several anatomical adaptations allow the subchondral bone to accept these loads. The meniscal cartilage in the knee joint serves to spread the femur’s load over a larger area of the tibial plateau (stress = force/area). Additionally, the articular cartilage and properly functioning muscle units cushion dynamic articular forces, spreading impact loads over a greater number of milliseconds, thereby reducing peak load.

The subchondral bone

The subchondral bone’s ability to bear load can be compromised by a host of injuries to the knee joint, ranging from overuse to exercise-related injury. For example, a meniscus tear resulting in a partial meniscectomy surgery will reduce the contact area of the articulation between the femur and tibia, thus increasing the stress in the subchondral bone in both the tibia and femoral condyle. Sports injuries such as ACL tear and patellar dislocation can delaminate the articular cartilage, resulting in compromised dynamic load sharing. In Figure 1a, such injuries are represented as a leftward shift along the X-axis.
Microtrabecular fractures

Normal trabecular bone maintains structural homeostasis: microtrabecular fractures occur naturally and injured bone is removed and replaced to maintain the health and strength of the bone. This steady state cannot be maintained when higher loads (represented in Figure 1a as upward shifts along the Y-axis) cause the rate of microtrabecular fractures to exceed the bone's ability to repair them. The result is a stress fracture (shaded region above the diagonal in Figure 1a), which can be detected by bone scan and MRI. At first the stress fracture is asymptomatic, but as it progresses it becomes increasingly painful. When a stress fracture occurs in a healthy and uncompromised knee joint (i.e. involvement of abnormal stress on a normal joint), it is termed a ‘classic’ or ‘fatigue’ stress fracture. When it occurs in a knee-joint-compromised patient (i.e. normal stress on an abnormal joint), it is termed an ‘insufficiency’ stress fracture.

Microtrabecular stress fractures and imaging

Microtrabecular stress fractures lead to changes in the marrow between trabeculae. These trabecular fractures are rarely detectable on plain film radiographs. When radiologists first saw these changes on MRI, they noted the signal was similar to that associated with water and thus assigned the misnomer ‘bone marrow oedema’. In the early stages of overloading, it may truly be water content. However, over time these changes, known as bone marrow lesions (BMLs) by orthopaedic surgeons, are actually indicative of microtrabecular fractures and associated bone remodelling. Radiologists are concerned that the term BML could imply sarcoma or other pathology and are slowly changing their terminology to ‘bone marrow oedema-like lesions’. Histological analyses of BMLs from bone retrieval studies demonstrate trabecular abnormalities, necrosis and fibrosis consistent with the appearance of a non-healing chronic stress fracture. These MRI findings are also frequently present with the altered loading during deterioration due to OA. Importantly, stress fractures invariably compromise the ability of the bone to bear load. An athlete

Figure 1: (a) Relationship between load, capacity to bear load and fractures. (b) Illustration of typical clinical courses. Scenario 1: patient exercised too much and developed a classic stress fracture (1x). Diagnosis led to the recommendation of extended rest of the joint, which eventually resulted in healing of the affected tibial trabecular bone (1y). Scenario 2: patient developed a classic stress fracture (2x), but did not reduce exercise, resulting in progression to a macroscopic fracture (2y). Scenario 3: patient tore her ACL, resulting in delamination of articular cartilage (3x). She did not heed exercise restriction recommendations, resulting in an insufficiency fracture (3y). She was treated with subchondroplasty and rest and her fracture healed (3z).
continuing to overload a stress-fracture-compromised joint risks escalation to a macroscopic fracture, as illustrated by Scenario 2 in Figure 1b.

BMLs are associated with pain and loss of function and indicate an increased likelihood of total knee replacement when they occur in OA patients.

Athletes may develop BMLs as a result of overuse or from mechanical overloading due to malalignment or meniscal loss and/or dysfunction. BMLs may also be associated with a focal cartilage lesion. Finally, a BML may be present after an impact injury such as a direct blow to the knee, traumatic ACL tear, patellar subluxation, dislocation or impaction injury.

The presence of a BML on MRI is strongly associated with the presence and severity of knee pain and is even predictive of subchondral bone attrition and loss of cartilage volume. In OA joints, the presence of a BML predicts further progression of the disease and a possible need for joint replacement in the future. Scher et al found that OA patients with a BML on MRI were nearly nine times as likely to progress to total knee arthroplasty compared to those without a BML.

Treatment of bone marrow lesions

Current treatment options for BMLs can be slow, ineffective or invasive.

Conservative treatment options may include activity modification or periods of non-weight-bearing, particularly for stress fractures.

- Bracing may also be utilised to unload the compartment associated with a BML or to stabilise the patella. In a recent study of patellofemoral OA, Callaghan et al found that patients who used a patellofemoral brace for 6 weeks saw a reduction both in BML volume in the patellofemoral compartment and in knee pain.
- Physical therapy can also be useful: incorporating exercises to increase core stability, pelvic tilt and to strengthen and co-ordinate the muscles of the hip and knee, may serve to alleviate overloading due to muscle imbalance. Successful implementation of conservative treatment is illustrated by Scenario 1 in Figure 1b.

- In some cases, surgery may be warranted to correct significant mechanical deficiencies:
  - For patients with compartments overloaded due to malalignment, osteotomy may be helpful. Meniscal deficiencies may be corrected through meniscal transplant.
  - For patients with BML associated with a focal cartilage lesion, cartilage restoration may be indicated.

In many cases, chronic BMLs persist even after conservative care and/or surgery to correct deficiencies. Additionally, many athletes are unable to rest for the long recovery durations required for conservative care.

Consequently, there is a need for a BML-targeted therapeutic option which allows for a shorter recovery time.

Subchondroplasty

Subchondroplasty is a minimally invasive surgical technique that targets and treats subchondral bone pathology associated with BMLs, which in turn allows bone healing. It is usually performed along with arthroscopy for guided insertion and concurrent treatment of other pathology inside the joint. During subchondroplasty, a calcium phosphate bone substitute material (BSM), is injected into the trabeculae of cancellous bone in the subchondral region of the knee joint (Figure 2). The flowable, synthetic calcium phosphate readily fills subchondral defects, crystallising endothermically in approximately 10 minutes with a porosity and strength that mimics healthy cancellous bone. This provides a scaffold for endogenous osteoclasts and osteoblasts to grow into and repair the bone. Over time, the calcium phosphate is resorbed and replaced with new healthy bone.

Subchondroplasty treatment of subchondral defects can result in reduced pain and improved function in OA patients.

OA can result in BMLs, which herald severe joint degeneration and progression towards the need for joint replacement. Joint-preserving treatments that reverse the progression of pain and immobility are limited for patients who have OA.

A recent retrospective study evaluated the effectiveness of subchondroplasty for relieving pain and improving function in 66 patients with documented BMLs and advanced knee OA. Significant improvements in both pain and function following subchondroplasty with arthroscopic debridement were observed, as measured by the visual analog scale and the International Knee Documentation Committee Subjective Knee Evaluation Form, through 2 years post-operative follow-up. Given that arthroscopic debridement alone has been previously shown to yield insignificant pain relief beyond 6 months post-surgery, the results of Cohen et al suggest that subchondroplasty may be a promising approach for the treatment of BMLs that may be associated with OA.
Sports-related injuries and subchondroplasty

Subchondroplasty may be a valid treatment for bone repair after injury. Sports-related injuries that have not responded to conservative therapies are problematic for the pathophysiological reasons outlined above. In addition, chronic BMLs that result from untreated injuries can contribute to the progression of OA. The success of subchondroplasty in treating OA-related BMLs provides a rationale for exploring whether this approach will extend to chronic sports-injury-related BMLs and the associated trabecular homeostasis that is critical to long-term bone health.

Case study 1

A 38-year-old male engineer presented with left medial knee pain that began when he increased his running mileage to train for a mini marathon. The patient described the pain as a constant, dull ache, becoming moderately intense and sharp with weight-bearing activity. The symptoms caused him to limp and were unresponsive to medication.

Upon examination, there were no associated signs such as redness, effusion or swelling. There was no patellar instability or loose body sensation. Imaging revealed a transverse stress fracture at the medial tibial plateau (Figures 3 and 4). An initial trial of minimal weight-bearing with crutches was not tolerated by the patient, who needed unaided ambulation to work. We discussed that the MRI was normal and, therefore, there was no reason to perform arthroscopy. The patient underwent subchondroplasty of the tibial plateau (Figure 5).

A needle was placed intra-articularly at the joint line for reference. Fluoroscopy was used and the fracture was noted to be 2 cm distal to the joint line. This was estimated and a spinal needle was placed at this point. It was noted that the stress fracture reaction is more readily seen on T2 fat-suppressed sequence (a).
Figure 5: Case study 1: intra-operative radiographs. (a) Injection of calcium phosphate BSM into the region of the bone marrow lesion. (b) Radiograph blush of calcium phosphate BSM following injection.

Figure 6: Case study 1: post-operative radiographs. (a and b) 1 month post-operative radiographs demonstrating calcium phosphate persists at site of injection. (c and d) 18 months post-operative radiographs demonstrating evidence of some degree of calcium phosphate resorption.

BSM=bone substitute material.
then reinserted and held in position for 10 minutes while the BSM solidified. The obturator and cannula were removed. There was no extraneous BSM at the site of the hole. The skin was approximated with #3-0 nylon using vertical mattress technique and Steri-Strips. The wound was dressed with Adaptic gauze, 4x4 flats, ABD pads, Kerlix wrap and a thigh-high TED hose.

At 1 week after surgery the patient had decreasing pain and no swelling and showed no signs of infection. He was completely pain-free at 2 weeks and after 5 weeks he was able to return to light jogging (against medical advice) and use of an elliptical machine. By 18 months post surgery the patient had fully recovered and was able to resume all preoperative activities. Radiographs at 18 months showed the presence of residual calcium phosphate in the tibia around the area of the original fracture (Figure 6). He was running 10 miles per week and able to participate in approximately three half-marathons per year. The patient was pleased with the overall comfort and functional status of the knee.

Case study 2

A 31-year-old male professional football player presented with a long history of left knee problems. He had undergone multiple arthroscopic procedures on the knee relating to meniscal tears and chondral defects. Nineteen months prior to his presentation he had undergone an osteochondral autograft transfer to the medial femoral condyle for a full thickness chondral defect. Prior to that procedure he had a microfracture of the same chondral defect. He was performing well with minimal knee issues during the early part of a training camp until his knee began swelling and he developed increasing medial pain. He initially had a corticosteroid injection which failed to improve his symptoms, followed by a viscosupplementation series.

Examination revealed a standing alignment of 5 degrees of varus and a range of motion of full extension to 112 degrees of flexion. He had a positive effusion of approximately 20 cm³ with medial joint line and medial femoral condyle tenderness. His ligamentous examination was stable. Weight-bearing X-rays revealed medial compartment joint space narrowing and spurring (Figure 7). An MRI scan revealed

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evidence of a prior osteochondral autograft transfer procedure to the medial femoral condyle with residual medial compartment chondral loss, synovitis, loose chondral debris in the supra-patella space and significant bone marrow oedema in the medial femoral condyle and medial tibial plateau (Figure 8).

The surgical and nonsurgical options were reviewed with the patient at that point and included continued conservative management with oral nonsteroidal anti-inflammatory drugs, bracing, injections (platelet-rich plasma, viscosupplementation, corticosteroid) and return to play as tolerated. The surgical options included arthroscopic debridement, synovectomy, loose body excision and subchondroplasty. The patient elected for arthroscopy with subchondroplasty, which was performed. At the time of surgery, the patient had significant synovitis with 3 loose chondral bodies measuring 5 mm. He had International Cartilage Repair Society grade 3 chondral changes to the trochlea and medial femoral condyle and grade 2 changes to the central/lateral facet of the patella. There was no meniscal or ligamentous pathology. There were osteophytes and spurring of the medial femoral condyle and tibial plateau. A debridement was performed in the medial and patellofemoral compartments as well as partial synovectomy and loose body excision. Subchondroplasty was performed under fluoroscopic guidance and a total of 6 cm$^3$ of BSM was injected into the medial femoral condyle and 4 cm$^3$ in the medial tibial plateau.

The patient was allowed weight-bearing and range of motion as tolerated. He advanced his rehabilitation with the team’s athletic training staff. He began a jogging/running programme by 2 to 3 weeks after surgery and returned to full practice by 5 weeks. At 7 weeks his postoperative MRI showed injection of calcium phosphate into the femoral condyle and tibia plateau with some residual oedema (Figure 9). The patient was allowed to continue progression of activity as tolerated and returned to full game participation at 7 weeks.

**CONCLUSION**

Subchondroplasty is a minimally invasive surgical technique that targets and treats subchondral bone pathology to allow bone healing. Prior work indicates that subchondroplasty is a promising treatment for bone marrow lesions related to OA. In the case studies presented, subchondroplasty successfully repaired the underlying defect, thereby restoring full knee functionality to two patients who had BMLs resulting from sports-related injury/overuse. The demonstrated success of subchondroplasty in treating BMLs related to OA and the initial success shown in these case studies warrants further exploration of the benefits of using subchondroplasty to treat BMLs resulting from sports-related injuries.

**Acknowledgements**

The authors would like to thank Vanessa Bender, Dillen Wischmeier, Iman Ahmad and Margeaux Rogers for critical review of the manuscript and Brett Mensh for the conceptualisation of Figure 1.

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**Figure 8:** Case study 2: pre-operative MRI. Subchondral oedema in the medial femoral condyle and medial tibial plateau.
Figure 9: Case study 2: 7 weeks post-operative MRI. Calcium phosphate in the femoral condyle and tibial plateau with some residual subchondral oedema.

References

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