Bone Marrow Edema in the Knee: A pictorial Review

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Learning objectives

To learn the causes and imaging presentations of bone marrow edema (BME) in the knee, revisiting several MRI cases.

Several cases from the authors' practices will be presented, grouped according to the edema mechanisms as adapted from the original classification by Eustace.[1]
Background

Bone marrow edema represents an increase in interstitial fluid, as such it has the MR characteristics of increased water content, namely a low/intermediate signal on T1-weighted images and a high signal on T2 and PD-weighted images.[2] Water sensitive sequences such as short-tau inversion recovery (STIR) imaging are very sensitive in detecting bone marrow edema as it appears hyperintense compared with normal bone marrow.[3]

Following intravenous gadolinium administration minimal delayed enhancement can be seen with marrow edema, particularly in the vasogenic forms (as explained ahead).[2]

In contrast to stroke, posttraumatic marrow edema is associated with increased diffusion on DWI (diffusion weighted-imaging) sequences.[2]

As stated above, marrow edema results from excessive fluid accumulation in the extracellular space, and its MR presentation is rather characteristic, but one should be aware that the same MRI findings can also represent areas of necrosis and/or hemorrhage.[2]

Several mechanisms can be involved in the pathophysiology of bone edema. We follow the division proposed by Eustace[1]: hyperemic/vasogenic, congestive, tumorigenic, traumatic, and an atypical group. We've reordered them according to incidence, arranged some of the rarer and less consensual entities into the Atypical/Others group (even though some of their mechanisms could fit elsewhere) and named the Traumatic/Degenerative group as such to reflect the microtraumatic nature of the traditionally degenerative classification of osteoarthritis:

1. Traumatic/Degenerative

   • Trauma is the most common cause of bone marrow edema.[2] Direct trauma causes immediate disruption of the trabeculae with seepage of interstitial fluid and hemorrhage into the extracellular space. In traumatic bone edema (commonly referred to as a bone bruise, contusion or trabecular microfractures[4]) the cortex remains intact, while in a true fracture the overlying cortex is disrupted.[2]

2. Congestive
• Impaired removal of fluid can lead to increases in hydrostatic pressure at the capillary bed resulting in leakage to extravascular spaces. Congestion typically results from vascular occlusion or thrombosis either idiopathic (e.g. avascular necrosis) or secondary to other causes of elevated marrow space pressures as in hemoglobinopathies, marrow packing disorders, fat cell hypertrophy or steroids use.[2]

3. Hyperemic/vasogenic

• Increased delivery of blood and serum to the marrow capillary bed (with or without changes in permeability) will lead to an increased accumulation of interstitial fluid. This is promptly identified at or adjacent to sites of inflammation due to infection, along the margins of joints affected by acute synovitis, and in patients with posttraumatic reflex sympathetic dystrophy.[2]

4. Tumorigenic

• Similar mechanism to the congestive capillary leakage. Most tumorigenic edema is a consequence of direct capillary trauma from trabecular destruction with the release of intravascular fluid and associated hemorrhage. Its presence is an indirect marker of malignancy. In a minority of cases, edema is instead secondary to an induced inflammatory response (most commonly in osteoid osteomas via prostaglandin E2).[2]

Bone marrow contusions (which can result either from direct blow to the bone, from compressive forces of adjacent bones impacting one another, or from traction forces that occur during an avulsion injury) typically have a distribution that reflects the mechanism of trauma: five contusion patterns are well described:[3]

1. Pivot Shift Injury
2. Dashboard Injury
3. Hyperextension Injury
4. Clip Injury
5. Lateral Patellar Dislocation
Fig. 1: 1) Pivot Shift Pattern (e.g. skiing accident): involves the posterolateral tibial plateau and the midportion of the lateral femoral condyle. ACL tear is frequently associated. 2) Dashboard Pattern (e.g. car accident): edema on the anterior aspect of the proximal tibia. PCL tear is frequently associated. 3) Hyperextension Pattern (e.g. soccer lesion): "kissing" contusion pattern involving the anterior aspect of the proximal tibia and distal femur. PCL tear is also frequently associated. 4) Clip Injury Pattern (e.g. rugby tackle): large area of edema involving the lateral femoral condyle and a smaller area on the medial femoral condyle. Medial Collateral Ligament (MCL) injuries can be associated. 5) Lateral Patellar Dislocation: edema involving the inferomedial patella and anterior aspect of the lateral femoral condyle. Medial Patellofemoral Ligament (MPFL) is frequently disrupted (see case ahead).

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Examples and further considerations on mechanisms 1, 4 and 5 (pivot shift injury, clip injury and lateral patellar dislocation) can be appreciated on the "Findings and procedure
These localized patterns are among the most characteristic presentations of bone marrow edema around the knee. When the edema is diffuse it's more challenging to find the correct cause, and sometimes the patient and attending physician are not able to provide clues prior to imaging. Transient osteoporosis, neuromuscular dysfunctions, neoplastic bone lesions or even minor trauma can be unknown at the time of the MRI scan. In the absence of trauma history and no other discernible cause for the finding of diffuse edema, transient bone marrow edema syndromes can be diagnosed.
Fig. 1: 1) Pivot Shift Pattern (e.g. skiing accident): involves the posterolateral tibial plateau and the midportion of the lateral femoral condyle. ACL tear is frequently associated. 2) Dashboard Pattern (e.g. car accident): edema on the anterior aspect of the proximal tibia. PCL tear is frequently associated. 3) Hyperextension Pattern (e.g. soccer lesion): "kissing" contusion pattern involving the anterior aspect of the proximal tibia and distal femur. PCL tear is also frequently associated. 4) Clip Injury Pattern (e.g. rugby tackle): large area of edema involving the lateral femoral condyle and a smaller area on the medial femoral condyle. Medial Collateral Ligament (MCL) injuries can be associated. 5) Lateral Patellar Dislocation: edema involving the inferomedial patella and anterior aspect of the lateral femoral condyle. Medial Patellofemoral Ligament (MPFL) is frequently disrupted (see case ahead).

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Findings and procedure details

Below we present a listing of several entities associated with bone marrow edema of the knee, arranged according to the pathophysiology of the edema for each case (slightly adapted from the first classification described by Eustace[1]). Several examples collected from our practice are used to illustrate and further discuss these entities:

1. Traumatic/Degenerative
   a. Osteoarthritis (Fig. 2 on page 34)
   b. Acute Trabecular Microfractures (Fig. 7 on page 34, Fig. 8 on page 35, Fig. 9 on page 36, Fig. 10 on page 37, Fig. 11 on page 37)
   c. Stress Fatigue Fractures (Fig. 3 on page 34)
   d. Stress Insufficiency Fractures
      i. SIF (Subchondral Insufficiency Fracture) formerly known as SONK (Spontaneous osteonecrosis of the knee, also known as Ahlback's Disease) (Fig. 4 on page 38, Fig. 5 on page 39, Fig. 6 on page 40)
   e. Macroscopic Fractures (Fig. 12 on page 40, Fig. 13 on page 41, Fig. 14 on page 42)
   f. Symptomatic Ossicles (Fig. 15 on page 43)
   g. Osgood-Schlatter Disease (Fig. 16 on page 44)
   h. Sinding-Larsen-Johansson Syndrome (Fig. 17 on page 45)
   i. Osteochondritis Dissecans (OCD) (Fig. 18 on page 46, Fig. 19 on page 47)

2. Congestive
   a. Osteonecrosis (Avascular Necrosis, AVN)
      i. Hemoglobinopathies (Fig. 20 on page 48)
      ii. Marrow Packing Disorders
      iii. Fat Cell Hypertrophy
      iv. Steroids Use
v. Pregnancy

3. Hyperemic/Vasogenic
   a. Inflammatory/Infectious Arthritis (Fig. 21 on page 50)
   b. Posttraumatic Reflex Sympathetic Dystrophy
   c. Chronic Recurrent Multifocal Osteomyelitis (CRMO)

4. Tumorigenic
   (e.g. Osteosarcoma (Fig. 22 on page 50), Giant Cell Tumor (Fig. 23 on page 51), Osteoid Osteoma (Fig. 24 on page 52), ...)

5. Atypical/Others
   a. Metabolic Changes
      i. Chronic Kidney Disease
   b. Neurological Changes
      i. Charcot's Joint
   c. Iatrogenic Changes
      i. Radiotherapy
   d. Idiopathic/Multifactorial Edema
      i. Transient Bone Marrow Edema Syndrome
         (also known as Transient Osteoporosis (TO)) (Fig. 25 on page 53)
   a. Regional Migratory Osteoporosis (RMO)
      i. Extra-articular
      ii. Intra-articular (Fig. 26 on page 54)

2. Complex Regional Pain Syndrome (CRPS, comprehends the terms Reflex Sympathetic Dystrophy, Algoneurodystrophy, and Sudeck's Atrophy)
3. Calcineurin Inhibitor Pain Syndrome (CIPS)
ii. Disuse Osteoporosis (DO) (Fig. 27 on page 55)

1. Traumatic/Degenerative

a. Osteoarthritis

![Fig. 2: Osteoarthritis (OA): 58-year-old man with extensive chondral defect at the weight-bearing area of the medial femoral condyle, associated with small marginal osteophytes and secondary subchondral bone marrow edema. Osteoarthritis isn't usually classified alongside traumatic etiologies but rather as a degenerative condition, truth is that the development of BME on OA is secondary to repetitive microtrauma on weight-bearing structures[2] (a: coronal T1-WI; b: coronal PD-FS-WI; c: sagittal PD-FS-WI).](image)

**References:** - Portimao/PT

1. Traumatic/Degenerative

b. Acute Trabecular Microfractures

c. Stress Fatigue Fractures

d. Stress Insufficiency Fractures

i. SIF (Subchondral Insufficiency Fracture) formerly known as SONK (Spontaneous osteonecrosis of the knee, also known as Ahlback’s Disease)

MRI findings of traumatic bone edema (commonly referred to as a bone bruise, contusion or trabecular microfracture) becomes evident within hours of the injury.[4]
It is important to note that acute trabecular microfractures and stress fractures are not synonyms. It’s even common to witness "stress fractures" or "insufficiency fractures" reported as the same entity, although insufficiency fractures are actually a subtype of stress fractures. Stress fractures can be classified as fatigue or insufficiency fractures, the first ones are caused by repeated overloading of normal bony structures while the latter ones occur spontaneously on an already impaired bone (e.g. because of osteoporosis or osteomalacia), without trauma or overloading.[5]

Trabecular microfractures (traumatic) and stress-related fractures (chronic overloading on normal bone or normal loading on impaired bone) can't be distinguished on MRI, only the patient's age and history can help to differentiate them.[5]
Fig. 4: SIF- In the past, every time a subchondral lesion as this one was observed in the medial femoral condyle it was labeled as spontaneous osteonecrosis of the knee (SONK). Now it's considered to be an insufficiency fracture.[5] These insufficiency fractures typically develop at the medial femoral condyle and in the femoral head and are present in older individuals (they are related to osteoporosis and altered biomechanics, as in unstable meniscal lesions).[5] Similar to other stress fractures, a hypointense fracture line is surrounded by hyperintense bone marrow edema on watersensitive sequences (a, b: coronal and sagittal PD-FS-WI).

References: - Portimão/PT...
Fig. 5: Long-standing SIF with subchondral collapse and associated large area of edema on the lateral femoral condyle (less common than the medial location shown on the previous image).

References: - Portimao/PT

Fig. 6: Less common form of SIF on the medial tibial plateau (a, b: sagittal and coronal PD-FS-WI).

References: - Portimao/PT

Fig. 7: Trabecular microfracture: 15-year-old male adolescent with acute pain following traumatic event while playing soccer. Hypointense microfracture on femoral medial condyle both on T1-WI (a, b) and on T2 FS-WI (c). On the water-sensitive sequence (T2-FS-WI) the hyperintense bone marrow edema surrounding the fracture is evident, while on the T1-WI's it translates as an ill-defined hypointense area on the otherwise hyperintense marrow of the distal femoral epiphysis and proximal tibia.

References: - Portimao/PT
Traumatic bone edema, patellar detachment, typical pattern: PD-weighted T2 Fat SAT (a, b, c), coronal (d, e) and sagittal planes (f) of a young adult with transient patellar dislocation and resulting typical contusion pattern: interarticular aspect of the lateral femoral condyle and inferomedial aspect of the patella. It's more common on teenagers or young adults who perform sports that require a twisting motion of the knee while it's flexed.

Associated lesions can be present, as in this patient with osteochondral injury of the patella (*) and tear of the medial patellofemoral ligament (MPFL) (arrow).

References: Portimao/PT
This pattern results from a valgus load applied to a flexed or semi-flexed knee, combined with external rotation of the tibia or internal rotation of the femur. This pattern involves the posterior aspect of the lateral tibial plateau and the midportion of the lateral femoral condyle near the condylopatellar sulcus. The degree of flexion of the knee determines the exact location of the lateral femoral condyle injury (more flexion results in a more posterior bruise, whereas less flexion results in a more anteriorly located edema). Sometimes, as shown in "a", a lateral femoral condyle impaction fracture occurs. Another frequently associated site of bone contusion is the anterior aspect of the medial tibial plateau (as shown in "b"), thought to result from contrecoup forces in the medial compartment at the resolution of the forced valgus forces. The ACL is frequently disrupted in this kind of trauma; therefore when this pattern is recognized a thorough evaluation of the ligament should be performed.

References:
- Portimao/PT
Fig. 10: Traumatic bone edema, typical pivot shift pattern: T2-FS-WI on sagittal planes, different patient from figure 6, same mechanism and similar findings (albeit without fracture on this case).
Fig. 11: Traumatic bone edema, typical clip injury pattern: Trauma sustained by a 23-year-old man while playing rugby. Coronal intermediate-weighted fat suppressed MR image reveals bone marrow edema in the lateral femoral condyle and lateral tibial plateau secondary to direct contusion. The valgus force associated with the mechanism of trauma stresses the MCL, which may tear, as documented on this patient.

References: - Portimao/PT

1. Traumatic/Degenerative

e. Macroscopic Fractures
Fig. 12: Traumatic bone edema, macroscopic fracture (with cortical disruption): This patient had a very faint fracture line on conventional radiography, barely noticeable (not shown here). On MR imaging the contusional bone edema surrounding the fracture makes it impossible to miss (PD-FS-WI hyperintensity and T1-WI hypointensity on the lateral tibial plateau). The fracture line is hypointense on T1-WI (a, c) and isointense on PD-FS-WI (b, d).

References: - Portimao/PT
Fig. 13: Traumatic bone edema, macroscopic fracture (PCL avulsion): When the posterior cruciate ligament is not retracted it can be hard to identify an avulsion fracture of its insertion. The surrounding bone edema makes it an easier diagnosis.

References: - Portimao/PT
Fig. 14: Traumatic bone edema, macroscopic subacute fracture: Even with a little less than obvious fracture line on the CT scan (a, b), MRI bone edema points out where to look (c, d, e, f).

References: - Portimao/PT

1. Traumatic/Degenerative

f. Symptomatic Ossicles
Fig. 15: Up to 2% of all bipartite patellae become painful because of stress across the synchondrosis between the ossicle and the patella, resulting from repetitive pull by the vastus lateralis muscle.[5] On this patient the repetitive overload on the synchondrosis has resulted in local bruising (hyperintense edema) in both the unfused accessory ossification center and the remaining patella (a, b: coronal and axial PD-FS-WI).

References: - Portimao/PT

1. Traumatic/Degenerative

   g. Osgood-Schlatter Disease
**Fig. 16**: Osgood-Schlatter’s disease is a chronic avulsion injury thought to result either from repetitive microtrauma and traction on the tibial tubercle or direct trauma to the patellar tendon at its insertion at the tibial tubercle. It's typically seen in active adolescents, particularly those who participate in sports with strenuous involvement of the knee.[6] The resulting edema on the avulsion site can be an important clue to make this diagnosis, as shown above (a, b: sagittal and axial PD-FS-WI).

**References**: - Portimao/PT

1. Traumatic/Degenerative

h. Sinding-Larsen-Johansson Syndrome
Fig. 17: This 13-year-old boy has Sinding-Larsen-Johansson Syndrome, a chronic traction injury of the immature osteotendinous junction of the proximal end of the patellar tendon as it inserts into the inferior pole of the patella (signaled by the lower pole edema on this sagittal PD-FS-WI).

References: - Portimao/PT

1. Traumatic/Degenerative

i. Osteochondritis Dissecans (OCD)
Fig. 18: Osteochondritis dissecans (OCD). OCD etiology is unknown and likely multifactorial, with injury, vascular factors, pressure changes, developmental differences, and genetics all contributing.[5] The most common site for OCD of the knee is the lateral aspect of the medial femoral condyle (75%).[5] Here is shown a characteristic MRI appearance, typically without additional lesions: a small area of bone marrow edema is evident on b, surrounding the osteochondral fragment. (a, b: coronal T1-WI and coronal PD-FS-WI).

References: - Portimao/PT
Fig. 19: Osteochondritis dissecans (OCD). Same patient as in fig. 18. An osteochondral fragment on the lateral aspect of the right femoral medial condyle is seen on conventional radiography.

References: - Portimao/PT

2. Congestive

a. Osteonecrosis (Avascular Necrosis, AVN)

i. Hemoglobinopathies
Fig. 20: Patients with hemoglobinopathies (for instance sickle-cell anemia, as in this case) are prone to develop osteonecrosis secondary to capillary obstruction. This obstruction leads to raised capillary hydrostatic pressure, which in turn is responsible for the hyperintense edema surrounding the hypointense bone infarcts on water-sensitive sequences. This is an example of BME of a congestive nature (due to impaired removal of fluid from the marrow space capillary bed). This patient also presents extensive red marrow reconversion as depicted on the marrow signal in T1-WI’s (much lower than surrounding fat tissue and slightly higher than muscle tissue) and DP-FS-WI's (doesn't fully saturate, remaining slightly hyperintense) (a: coronal DP-FS-WI; b: coronal T1-WI; c: axial T1-WI; d: sagittal DP-FS-WI).

References: - Portimao/PT
3. Hyperemic/Vasogenic

a. Inflammatory/Infectious Arthritis

**Fig. 21**: This patient's inflammatory arthritis is responsible for the bone marrow edema surrounding the erosive changes on the medial femoro-tibial compartment. It's an example of BME of a vasogenic nature (secondary to an increased delivery of blood and serum to the marrow space capillary bed) (a, b: coronal T1-WI and PD-FS-WI).

*References*: - Portimao/PT

4. Tumorigenic

*(e.g. Osteosarcoma, Giant Cell Tumor, Osteoid Osteoma, ...)*
Fig. 22: Osteosarcoma (a, b: coronal and axial PD-FS-WI): tumorigenic edema is secondary to direct capillary trauma from trabecular destruction with the release of intravascular fluid and associated hemorrhage.[2] It may be difficult to differentiate surrounding edema from tumor invasion.

References: - Portimao/PT
Fig. 23: Giant Cell Tumor (PD-FS-WI): tumorigenic edema is also present. 

References: - Portimao/PT

Fig. 24: Osteoid Osteoma: in these benign tumors edema can be secondary to an induced inflammatory response by prostaglandin E2 [2] (a, b: axial and coronal CT scan; c, d: axial T1-WI and coronal PD-FS-WI).

References: - Portimao/PT

5. Atypical/Others

d. Idiopathic/Multifactorial Edema
i. Transient Bone Marrow Edema Syndrome
(also known as Transient Osteoporosis (TO))

Fig. 25: Transient Bone Marrow Edema Syndrome (Transient Osteoporosis): sagittal intermediate-weighted fat-suppressed MR image of a 30-year-old woman with acute knee pain shows bone marrow edema involving the non-weight-bearing area of the lateral femoral condyle, which underwent almost complete spontaneous resolution over the course of 1 year. Transient Bone Marrow Edema Syndromes are self-limiting entities which manifest as arthralgia associated with bone marrow edema evidence on MRI studies.[8] Its etiology remains uncertain.[8]

References: - Portimao/PT

5. Atypical/Others

d. Idiopathic/Multifactorial Edema

i. Transient Bone Marrow Edema Syndrome
(also known as Transient Osteoporosis (TO))

a. Regional Migratory Osteoporosis (RMO)

ii. Intra-articular
Fig. 26: Regional Migratory Osteoporosis (a Transient Bone Marrow Edema Syndrome with location shifting) (serial coronal PD-FS-WI): In some cases a location shift of the bone marrow edema is seen on serial MRIs, this is known as Regional Migratory Osteoporosis (RMO).[8] Although it's more common to witness a spontaneous resolution of the transient bone marrow edema syndrome, a shift to a different joint can occur in a small fraction of cases, or (even rarer) a shift to a different site within the same articulation (i.e. intra-articular shifting), as seen on this patient.[8] On this intra-articular RMO case there was no trauma history, the edema started on the medial femoral condyle and 3 months later the findings had shifted to the lateral condyle.

References: - Portimao/PT

5. Atypical/Others

d. Idiopathic/Multifactorial Edema

ii. Disuse Osteoporosis (DO)
Fig. 27: Disuse Osteoporosis (DO): the confluent and patchy moderately hyperintense pattern here shown in all sequences on the epiphysis is typical of transient osteoporosis following prolonged immobilization. Resuming activity produces bigger stress in the disused bone than in the normal bone because the trabeculae, which have to support the load, are less and weaker, hence bone edema ensues (a stress reaction is also seen on the medial condyle and marked with an *).[7] An healed lateral tibial plateau fracture, responsible for the immobilization, is also apparent (arrow). (a-e: PD-FS-WI; f: T1-WI). It's also worth noting that Complex Regional Pain Syndrome (CRPS, comprehending the terms reflex sympathetic dystrophy, algoneurodystrophy, and Sudeck's atrophy) has the same MRI bone edema pattern as DO but it's pathophysiology is not related to disuse demineralization and it's always painful (DO is an incidental finding in an asymptomatic patient with known immobilization history).[8]

References: - Portimao/PT

Bone Marrow Edema Pitfalls
Fig. 28: Normal red marrow: this is a possible pitfall when looking for bone marrow edema. Hematopoietic red marrow (either due to normal distribution in younger patients or marrow reconversion in adults) has intermediate signal intensity both on T1-WI (b) and on water-sensitive sequences (a: PD-FS-WI).[4] The characteristic pattern of distribution (usually on the metaphysis) and the higher signal intensity of normal red marrow relative to muscle on T1-WI (as opposed to pathologic marrow that is typically iso- or hypointense to muscle tissue on T1-WI) should enable prompt recognition of this normal finding.[4]

References: - Portimao/PT
Fig. 2: Osteoarthritis (OA): 58-year-old man with extensive chondral defect at the weight-bearing area of the medial femoral condyle, associated with small marginal osteophytes and secondary subchondral bone marrow edema. Osteoarthritis isn’t usually classified alongside traumatic etiologies but rather as a degenerative condition, truth is that the development of BME on OA is secondary to repetitive microtrauma on weight-bearing structures[2] (a: coronal T1-WI; b: coronal PD-FS-WI; c: sagittal PD-FS-WI).

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Fig. 3: Stress (fatigue) fractures: T1-weighted and STIR coronal images of the knees of a 25-year-old woman, who had recently engaged in strenuous physical activity at the gym without adequate conditioning. She complained of medial tibial pain bilaterally. Medial proximal stress fractures are evident in both tibias.

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Fig. 7: Trabecular microfracture: 15-year-old male adolescent with acute pain following traumatic event while playing soccer: hypointense microfracture on femoral medial condyle both on T1-WI (a, b) and on T2-FS-WI (c). On the water-sensitive sequence (T2-FS-WI) the hyperintense bone marrow edema surrounding the fracture is evident, while on the T1-WI's it translates as an ill-defined hypointense area on the otherwise hyperintense marrow of the distal femoral epiphysis and proximal tibia.

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**Fig. 8:** Traumatic bone edema, patellar dislocation typical pattern: PD-FS-WI on axial (a, b, c), coronal (d, e) and sagittal planes (f) of a young adult with transient patellar dislocation and resulting typical contusion pattern: anterolateral aspect of the lateral femoral condyle and inferomedial aspect of the patella. It's more common on teenagers or young adults who perform sports that require a twisting motion of the knee while it's flexed. Associated lesions can be present, as in this patient with osteochondral injury of the patella (*) and tear of the medial patellofemoral ligament (MPFL) (arrow).

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**Fig. 9:** Traumatic bone edema, typical pivot shift pattern: PD-FS-WI on sagittal planes. This type of non-contact injury is commonly seen in skiers or American football players. [3] This pattern results from a valgus load applied to the flexed or semi-flexed knee, combined with external rotation of the tibia or internal rotation of the femur.[3] The edema pattern involves the posterior aspect of the lateral tibial plateau and the midportion of the lateral femoral condyle near the condylopatellar sulcus (a).[3] The degree of flexion of the knee determines the exact location of the lateral femoral condyle injury (more flexion results in a more posterior bruise, whereas less flexion results in a more anteriorly located edema).[3] Sometimes, as shown in a, a lateral femoral condyle impaction fracture occurs. Another frequently associated site of bone contusion is the posterior aspect of
the medial tibial plateau (as shown in b), thought to result from contrecoup forces in the medial compartment at the resolution of the forced valgus forces (b). The ACL is frequently disrupted in this kind of trauma[3], therefore when this pattern is recognized a thorough evaluation of the ligament should be performed.

Fig. 10: Traumatic bone edema, typical pivot shift pattern: T2-FS-WI on sagittal planes, different patient from figure 6, same mechanism and similar findings (albeit without fracture on this case).

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Fig. 11: Traumatic bone edema, typical clip injury pattern: Trauma sustained by a 23-year-old man while playing rugby. Coronal intermediate-weighted fat suppressed MR image reveals bone marrow edema in the lateral femoral condyle and lateral tibial plateau secondary to direct contusion. The valgus force associated with the mechanism of trauma stresses the MCL, which may tear, as documented on this patient.

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Fig. 4: SIF: in the past, every time a subchondral lesion as this one was observed in the medial femoral condyle it was labeled as spontaneous osteonecrosis of the knee (SONK). Now it's considered to be an insufficiency fracture.[5] These insufficiency fractures typically develop at the medial femoral condyle and in the femoral head and are present in older individuals (they are related to osteoporosis and altered biomechanics, as in unstable meniscal lesions).[5] Similar to other stress fractures, a hypointense fracture line is surrounded by hyperintense bone marrow edema on water-sensitive sequences (a, b: coronal and sagittal PD-FS-WI).

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**Fig. 5:** Long-standing SIF with subchondral collapse and associated large area of edema on the lateral femoral condyle (less common than the medial location shown on the previous image).

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**Fig. 6:** Less common form of SIF on the medial tibial plateau (a, b: sagittal and coronal PD-FS-WI).

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**Fig. 12:** Traumatic bone edema, macroscopic fracture (with cortical disruption): This patient had a very faint fracture line on conventional radiography, barely noticeable (not shown here). On MR imaging the contusional bone edema surrounding the fracture makes it impossible to miss (PD-FS-WI hyperintensity and T1-WI hypointensity on the lateral tibial plateau). The fracture line is hypointense on T1-WI (a, c) and isointense on PD-FS-WI (b, d).

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**Fig. 13:** Traumatic bone edema, macroscopic fracture (PCL avulsion): When the posterior cruciate ligament is not retracted it can be hard to identify an avulsion fracture of its insertion. The surrounding bone edema makes it an easier diagnosis.

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**Fig. 14:** Traumatic bone edema, macroscopic subacute fracture: Even with a little less than obvious fracture line on the CT scan (a, b), MRI bone edema points out where to look (c, d, e, f).

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Fig. 15: Up to 2% of all bipartite patellae become painful because of stress across the synchondrosis between the ossicle and the patella, resulting from repetitive pull by the vastus lateralis muscle.[5] On this patient the repetitive overload on the synchondrosis has resulted in local bruising (hyperintense edema) in both the unfused accessory ossification center and the remaining patella (a, b: coronal and axial PD-FS-WI).

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Fig. 16: Osgood-Schlatter’s disease is a chronic avulsion injury thought to result either from repetitive microtrauma and traction on the tibial tubercle or direct trauma to the patellar tendon at its insertion at the tibial tubercle. It’s typically seen in active adolescents, particularly those who participate in sports with strenuous involvement of the knee.[6] The resulting edema on the avulsion site can be an important clue to make this diagnosis, as shown above (a, b: sagittal and axial PD-FS-WI).

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Fig. 17: This 13-year-old boy has Sinding-Larsen-Johansson Syndrome, a chronic traction injury of the immature osteotendinous junction of the proximal end of the patellar tendon as it inserts into the inferior pole of the patella (signaled by the lower pole edema on this sagittal PD-FS-WI).

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**Fig. 18:** Osteochondritis dissecans (OCD). OCD etiology is unknown and likely multifactorial, with injury, vascular factors, pressure changes, developmental differences, and genetics all contributing.[5] The most common site for OCD of the knee is the lateral aspect of the medial femoral condyle (75%).[5] Here is shown a characteristic MRI appearance, typically without additional lesions: a small area of bone marrow edema is evident on b, surrounding the osteochondral fragment. (a, b: coronal T1-WI and coronal PD-FS-WI).

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**Fig. 19:** Osteochondritis dissecans (OCD). Same patient as in fig. 18. An osteochondral fragment on the lateral aspect of the right femoral medial condyle is seen on conventional radiography.

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Fig. 20: Patients with hemoglobinopathies (for instance sickle-cell anemia, as in this case) are prone to develop osteonecrosis secondary to capillary obstruction. This obstruction leads to raised capillary hydrostatic pressure, which in turn is responsible for the hyperintense edema surrounding the hypointense bone infarcts on water-sensitive sequences. This is an example of BME of a congestive nature (due to impaired removal of fluid from the marrow space capillary bed). This patient also presents extensive red marrow reconversion as depicted on the marrow signal in T1-WI’s (much lower than surrounding fat tissue and slightly higher than muscle tissue) and DP-FS-WI’s (doesn't fully saturate, remaining slightly hyperintense) (a: coronal DP-FS-WI; b: coronal T1-WI; c: axial T1-WI; d: sagittal DP-FS-WI).

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**Fig. 21:** This patient's inflammatory arthritis is responsible for the bone marrow edema surrounding the erosive changes on the medial femoro-tibial compartment. It's an example of BME of a vasogenic nature (secondary to an increased delivery of blood and serum to the marrow space capillary bed) (a, b: coronal T1-WI and PD-FS-WI).

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**Fig. 22:** Osteosarcoma (a, b: coronal and axial PD-FS-WI): tumorigenic edema is secondary to direct capillary trauma from trabecular destruction with the release of intravascular fluid and associated hemorrhage.[2] It may be difficult to differentiate surrounding edema from tumor invasion.

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Fig. 23: Giant Cell Tumor (PD-FS-WI): tumorigenic edema is also present.

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Fig. 24: Osteoid Osteoma: in these benign tumors edema can be secondary to an induced inflammatory response by prostaglandin E2 [2] (a, b: axial and coronal CT scan; c, d: axial T1-WI and coronal PD-FS-WI).

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Fig. 25: Transient Bone Marrow Edema Syndrome (Transient Osteoporosis): sagittal intermediate-weighted fat-suppressed MR image of a 30-year-old woman with acute knee pain shows bone marrow edema involving the non-weight-bearing area of the lateral femoral condyle, which underwent almost complete spontaneous resolution over the course of 1 year. Transient Bone Marrow Edema Syndromes are self-limiting entities which manifest as arthralgia associated with bone marrow edema evidence on MRI studies.[8] Its etiology remains uncertain.[8]

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Fig. 26: Regional Migratory Osteoporosis (a Transient Bone Marrow Edema Syndrome with location shifting) (serial coronal PD-FS-WI): In some cases a location shift of the bone marrow edema is seen on serial MRIs, this is known as Regional Migratory Osteoporosis (RMO).[8] Although it's more common to witness a spontaneous resolution of the transient bone marrow edema syndrome, a shift to a different joint can occur in a small fraction of cases, or (even rarer) a shift to a different site within the same articulation
(i.e. intra-articular shifting), as seen on this patient.[8] On this intra-articular RMO case there was no trauma history, the edema started on the medial femoral condyle and 3 months later the findings had shifted to the lateral condyle.

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Fig. 27: Disuse Osteoporosis (DO): the confluent and patchy moderately hyperintense pattern here shown in all sequences on the epiphysis is typical of transient osteoporosis following prolonged immobilization. Resuming activity produces bigger stress in the disused bone than in the normal bone because the trabeculae, which have to support the load, are less and weaker, hence bone edema ensues (a stress reaction is also seen on the medial condyle and marked with an *).[7] An healed lateral tibial plateau fracture, responsible for the immobilization, is also apparent (arrow). (a-e: PD-FS-WI; f: T1-WI). It's also worth noting that Complex Regional Pain Syndrome (CRPS, comprehending the terms reflex sympathetic dystrophy, algoneurodystrophy, and Sudeck’s atrophy) has the same MRI bone edema pattern as DO but it's pathophysiology is not related to disuse demineralization and it's always painful (DO is an incidental finding in an asymptomatic patient with known immobilization history).[8]

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Conclusion

Knee bone marrow edema is a very common finding on MRI scans. However, its pathophysiology is not as simple as one could imagine as there are several different mechanisms through which it can develop. For each of these mechanisms there is a specific set of possible underlying diseases, some with a very characteristic distribution pattern. Having this knowledge is highly helpful in narrowing down differential diagnosis and not missing common associated injuries.
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