Sports participation is a contributing factor in the physical and emotional development of children while promoting habitual exercise and combating childhood obesity. Adolescent participation in sports is increasing with earlier adoption of intense training regimens and repetitive specialized motions. Concurrently, the incidence of sports-related overuse injuries is on the rise. The dynamic nature of the adolescent skeleton results in unique growth-related patterns of injury. The mainstay of treatment in all cases is suspension of activity in conjunction with physical therapy. If left undiagnosed, these injuries can lead to developmental abnormalities that increase susceptibility to future injury. Radiologists play a key role in identifying patterns of overuse injury in adolescent athletes, which facilitates the initiation of a treatment plan. It is imperative that the reviewing radiologist be familiar with associated imaging to avoid misdiagnosis and any subsequent, unnecessary intervention. MRI offers superior sensitivity and specificity compared with other imaging modalities and does not use ionizing radiation, which is essential in the adolescent athlete patient population.

Apophysitis

Apophyses serve as attachment sites for tendons and ligaments in the developing skeleton. Arising from separate ossification centers, apophyses eventually fuse with adjacent bone at variable ages, depending on location. Traction injury to this anchoring bony outgrowth is described as apophysitis. Management is usually nonsurgical, with activity limitation and subsequent graduated rehabilitation of utmost importance. Nonsteroidal anti-inflammatory drugs are effective for pain relief. Barring potential displacement of an avulsion fracture, prognosis is typically excellent. Enlargement of an inflamed apophysis should not be mistaken for more aggressive entities, and awareness of the MRI appearance of developing musculoskeletal anatomy allows for accurate diagnosis.

Apophysitis of the Knee: Osgood-Schlatter and Sinding-Larsen-Johansson Diseases

Apophysitis of the tibial tubercle (Osgood-Schlatter) and inferior patellar pole (Sinding-Larsen-Johansson) are exceedingly common overuse injuries in adolescent athletes who routinely perform running, jumping or cutting motions. In either case, presentation may be bilateral or unilateral and males are more commonly affected. Tibial tubercle ossification occurs from proximal and distal ossification centers, which are separated by residual epiphyseal cartilage. Unfused ossification centers should not be mistaken for avulsion fracture or tibial tubercle apophysitis.

The tibial tubercle apophysis is radiographically evident around age 10, with fusion typically occurring at ages 14 to 18. The tibial tubercle tethers the quadriceps muscles via the patellar tendon and undergoes traction during knee extension and hip flexion.

Osgood-Schlatter disease manifests with gradual onset cyclic pain, swelling and deformity at the tibial tubercle. An association with patella alta has been proposed. MRI will reveal marrow edema of the tibial tubercle, edema within Hoffa’s fat pad, deep infrapatellar bursal fluid, and thickening of the distal patellar tendon (Figure 1). Displaced/nondisplaced cartilaginous and/or osseous fragmentation at the tubercle has also been described.

Traction injury occurring at the patellar attachment of the patellar tendon is known as Sinding-Larsen-Johansson disease. Pain and swelling is localized to the inferior patellar pole. This is easily discernible from Osgood-Schlatter disease by the presence of proximal patellar tendinosis. MRI will reveal a thickened proximal patellar tendon and edema-like signal of the inferior patella and adjacent Hoffa fat pad. Patellar sleeve fracture and symptomatic bipartite patella can present similarly and should be excluded. Persistent pain after growth plate closure in either Osgood-Schlatter or Sinding-Larsen-Johansson disease may be due to a residual ossicle, which can be surgically excised.
Calcaneal Apophysitis: Severs Disease

The calcaneal apophysis is radiographically evident around age 7 with fusion occurring around age 16. The posterior calcaneal apophysis serves as the attachment site for the gastrocnemius and soleus muscles via the Achilles tendon and undergoes traction during plantar flexion and knee flexion. Inferiorly, the plantar fascia also originates from this apophysis. Clinical presentation is typically bilateral or unilateral posterior heel pain aggravated by compression of the Achilles tendon (squeeze test). MRI will reveal apophyseal and subjacent bone marrow edema.

Fragmentation and/or irregularity along the apophyseal margin may be present (Figure 2). Calcaneal fracture and osteomyelitis should be excluded as these can clinically mimic apophysitis.

Ischial Tuberosity Apophysitis

The ischial tuberosity apophysitis is radiographically evident around age 14 with fusion typically occurring between ages 18 and 20. The ischial tuberosity serves as the attachment site for the common hamstring tendon, which includes the long head of the biceps femoris, semitendinosus, and semimembranosus tendons. The biceps femoris and semitendinosus muscles combine as the conjoint tendon, which inserts on the transverse facet of the tuberosity posteromedially. Superolaterally, the semimembranosus muscle originates from the oblique facet of the tuberosity.

Traction at the apophysis occurs with repeated flexion at the knee and extension at the hip. Ischial tuberosity apophysitis is seen commonly in dancers and runners. Clinical presentation is dull hip pain that is aggravated by activity, manual compression, or isometric hamstring contraction. MRI will reveal apophyseal and subjacent bone marrow edema (Figure 3). Linear, hypointense signal on T1-weighted imaging (T1WI) corresponding to a nondisplaced fracture line may be evident. Of note, the ischial tuberosity is the most common site of osseous avulsion in the pelvis. If displaced by at least 2 cm, an avulsed osseous fragment should be surgically fixed.

Medial Epicondyle Apophysitis: Little Leaguer’s Elbow

The medial epicondyle apophysis is radiographically evident at age 6 and fuses around age 15. The common forearm flexors and ulnar collateral ligament attach to the medial epicondyle, which undergoes tensile stress during overhead throwing. During the late-cocking and acceleration phases of throwing, a valgus stress is generated at the elbow joint causing lateral compression and medial tension.
the adult, repeated valgus stress results in degeneration and tearing of the ulnar collateral ligament. In the adolescent, the medial epicondyle apophysis is at increased risk of injury because it is intrinsically weaker than the ulnar collateral ligament. Apophysitis can be attributed to high pitch counts, and throwing off-speed and breaking pitches can accentuate the valgus stress. Clinical presentation includes pain and tenderness at the medial elbow, weakness and limited range of motion.

MRI will reveal apophyseal and subjacent bone marrow edema (Figure 4). The ulnar collateral ligament should also be evaluated for sprain, which will manifest in thickening and increased intrasubstance signal on T2-weighted sequences. High-grade partial and complete ligament tears are less likely in this population but should be excluded. Greater than 3-5 mm of physeal widening is an indication for surgical fixation due to risk of nonunion.

In addition to medial tensile injury, concomitant lateral compressive injuries that may be identified include osteochondral lesion of the capitellum (see section below) and Panner disease. Panner disease is a self-limiting osteochondrosis involving the entire capitellum and is thought to result from vascular interruption secondary to compression. It has been described on MRI as paraphyseal marrow edema in the absence of an osseous or chondral defect. A late MRI manifestation of Panner disease is the replacement of normal fatty marrow signal with patchy sclerosis on T1WI.

**Physeal Stress Injuries**

The physis, or “growth-plate,” is characterized by a radiolucent line on x-ray between the epiphysis and metaphysis in the skeletally immature patient. The physis eventually disappears as endochondral ossification is completed, leaving a physeal scar. Like the apophysis, the physis is subject to stress during repetitive motion. Stress at the physis is due to compressive force, in contrast to the traction force exerted upon the apophysis. The peak incidence of physeal stress injury occurs between ages 11 and 14 years.

Common locations for physeal stress injuries include the wrist, knee and shoulder. Clinical presentation includes pain, swelling and weakness. If left untreated, physeal stress injuries...
can result in growth cessation, length discrepancy, angular deformity and even lifelong disability. The mainstay of treatment is limitation and modification of activity. Diagnosis can be made radiographically with signs including soft-tissue swelling, osseous irregularity and widening of the physis. MRI will reveal physeal irregularity, widening, paraphyseal marrow edema and osseous or fibrous physeal bridging.6

**Radial Physeal Stress Syndrome: Gymnast Wrist**

A common physeal stress injury seen in adolescent gymnasts is gymnast wrist, with a significantly higher prevalence among females. Gymnastics often involve inverted maneuvers resulting in supraphysiologic compressive force at the wrist. Repeated axial loading of the wrist causes injury to the physis leading to premature closure. MRI will reveal physeal widening, which is more pronounced at the volar aspect owing to repetitive hyperextension at the wrist (Figure 5).6 A physeal bar or osseous bridge can develop in some instances further altering development. Long-standing stress injury may manifest in positive ulnar variance and Madelung deformity. Positive ulnar variance can lead to ulnar impaction syndrome causing stress on the triangular fibrocartilage complex (TFCC) and lunate.4 Accordingly, MRI may reveal TFCC and/or lunotriquetral ligament tears and degenerative cystic change along the ulnar aspect of the proximal lunate.

**Stress Fracture**

Stress fractures are characterized by abnormal stress on normal bone and often occur at the metaphyseal-diaphyseal interface. The most common locations for stress fractures occur in the weight-bearing lower extremities, specifically the tibia, tarsal navicular, metatarsal and fibula.7 Clinical presentation includes pain, tenderness, and swelling aggravated by weight-bearing activities. Stress fractures can be subtle to imperceptible on radiographs depending on acuity. MRI is the most sensitive modality and will reveal periosteal and/or marrow edema. Linear, hypointense signal on T1WI corresponding to the fracture line is seen in higher-grade stress fractures.7

**Tibial Stress Injury**

Medial tibial stress syndrome, or “shin splints,” results in diffuse medial tibial pain and is caused by local inflammation due to activity.5 The Fredricson Classification System serves to grade medial tibial stress injuries based on MRI characteristics and thereby assist in management.7 A high-grade medial tibial stress injury displays a
hypointense linear intracortical fracture line with surrounding hyperintense marrow signal on fluid-sensitive sequences (Figure 6). This injury results from repetitive high-impact activity and these fractures may be complete or incomplete. Fractures occurring at the anterior aspect of the tibia typically result from jumping-type stress whereas posterior fractures typically result from running-type stress. There is a higher incidence of tibial stress fracture in female adolescent athletes and those with pes cavus deformity. Patients tend to report a deep, throbbing pain localized to the fracture site. In all cases of tibial stress injury, activity cessation is the first-line treatment.

Osteochondral Lesions

Osteochondral lesion/defect, or osteochondritis dessicans (OCD), is characterized by separation of the osteochondral body from the subchondral bone. Disruption of the articular cartilage can result in local tenderness and a locking sensation. The most common location for development of an osteochondral lesion is the medial femoral condyle. Other commonly implicated locations include the humeral head, talar dome, and capitellum of the humerus. When left undiagnosed, osteochondral lesions may progress to secondary osteoarthritis. Treatment depends on severity with unstable lesions requiring surgical intervention.

Circumferential hyperintense T2-signal along the nonarticular portion of the lesion indicates fragment instability. Other MRI findings suggesting instability include numerous or large underlying cysts, fluid-filled osteochondral defect, and a T2-hyperintense osteochondral fracture line surrounding the defect. In severe cases, the lesion will be identified as a displaced, intra-articular loose body.

Osteochondritis Dessicans: Capitellum of the Humerus

Osteochondral lesions occurring in the capitellum of the humerus are a manifestation of lateral elbow compression. These lesions are frequently encountered in overhead throwing athletes and gymnasts. Osteochondral lesions are located at the anterolateral aspect of the ossifying capitellar epiphysis and occur when ossification is nearing completion. Patients will present with pain and swelling at the lateral elbow and a locking sensation. In contrast to Panner disease, imaging will reveal flattening of the articular surface.

Because of their anterior location, osteochondral lesions are best seen on sagittal and coronal MR images. Early MR findings include crescentic low signal at the subchondral capitellar surface on T1WI. Longstanding injury will demonstrate marrow edema and articular surface cartilage irregularity (Figure 7). The joint space should be evaluated for intra-articular loose bodies. Pseudodefect of the capitellum presents as focal flattening of the posterior...
capitellum with normal overlying articular surface cartilage. A pseudodefect of the capitellum should not be mistaken for an osteochondral lesion.

**Friction Syndromes**

Repetitive compression of soft tissues by bone or musculature can lead to inflammation and damage. Friction syndromes are more common in the adult, but have been described in the adolescent athlete population. Common areas for friction syndromes include the iliotibial band and patellar tendon-lateral femoral condyle. MRI is helpful in diagnosing friction syndromes and identifying potential causes.

**Iliotibial Band Friction Syndrome**

Anatomically, the distal iliotibial band closely approximates the lateral femoral condyle as it inserts on Gerdy’s tubercle at the lateral proximal tibia. With knee flexion and extension, the iliotibial band slides across the lateral femoral condyle surface. Friction is generated with activities such as running or cycling, which causes inflammation and pain at the lateral knee. MRI will reveal soft-tissue, edema-like signal interposed between the distal iliotibial band and lateral femoral condyle. Of note, the lateral synovial recess of the anterior knee should not be confused with soft-tissue edema. In contrast to edema from iliotibial friction, this fluid-filled recess will not extend posterior to the lateral condyle (Figure 8).

**Conclusion**

Increasing incidence of sports-related overuse injuries is associated with early adolescent athletic specialization and high-intensity training. More than ever, MRI is being utilized to effectively and safely identify overuse patterns of injury to expedite care and avoid potentially devastating complications. Treatment is usually conservative with an emphasis on activity cessation and/or modification, pain relief and rehabilitation. Radiologists should be familiar with the clinical presentation and common MRI findings of overuse injuries in adolescent athletes.

**REFERENCES**