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REVIEW ARTICLE

Injuries of the adolescent girl athlete: a review of imaging findings

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Abstract

With the rising participation of girls in sports at both the recreational and elite levels, there has also been increased awareness of injuries common in this athlete population. Anatomic differences between boys and girls cause girl athletes to be predisposed to certain injuries. Certain behavioral patterns, such as eating disorders, also cause problems specific to girl athletes that may result in injury. Imaging plays a large role in diagnosis and ongoing management, but there has been only scant literature dedicated to the specific topic of imaging in girl athletes. The purpose of this article is to review the imaging findings and recommendations for injuries and other conditions affecting the adolescent girl athlete. This article first provides an overview of the key anatomic differences between boys and girls, including both static and dynamic factors, as well as non-anatomic differences, such as hormonal factors, and discusses how these differences contribute to the injury patterns that are seen more typically in girls. The article then reviews the imaging findings in injuries that are commonly seen in girl athletes. There is also a discussion of the "female athlete triad," which consists of osteoporosis, disordered eating, and amenorrhea, and the role of imaging in this condition.

Keywords Female athlete · Sports medicine · MRI · Female athlete triad · Pediatrics

Introduction

Over the past several decades, there has been a dramatic increase in youth sports participation. In 2008, a survey by the National Council of Youth Sports found that 60 million children ages 6 to 18 years old participate in some form of organized athletics [1]. Sports participation among women has also skyrocketed in recent decades, and with it the number of girl athletes: a 2012 longitudinal national survey found that the number of high school aged girls participating in competitive sports has increased tenfold (1000%) over the past 35 years [2]. Although there has also been an increase in high-school-aged boys participating in competitive sports, this has paled in comparison to the rate seen with girls. Over the 30 years spanning 1972 to 2011, there has been a 23% increase in high school male athletes [3]. Significant growth in the population of adolescent female athletes make it increasingly important to recognize injuries that are more common in, and more specific to, the girl athlete. Imaging plays a crucial role in diagnosis, and knowledge of the differences in anatomic, physiologic, and behavioral factors between girls and boys helps the radiologist to understand gender-specific patterns of injury.

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Anatomic differences in girl athletes

Differences in injury patterns between girls and boys start to emerge during and after puberty; we will refer to “girl athletes” and “boy athletes” in this manuscript as being in the post-pubertal stage, unless otherwise noted. Once developmental changes have taken place, anatomic differences between genders can be viewed in two major categories: static (difference is present at rest) and dynamic (difference occurs upon movement).

Static anatomic differences between genders

Girls and women generally have a lower center of gravity than boys and men, as their limbs are shorter and smaller relative to body size, and they have greater laxity of their joints [4], which further increases during pregnancy [5]. Boys and men have proportionally more muscle mass than girls and women in the upper extremities [6] and upper trunk [7]. Girls' and women's pelvises tend to be wider than boys' and men's, and there are resultant increases in hip varus angle [8], knee valgus angle [9], and femoral anteversion [10]. This results in girls and women typically having a greater Q angle, defined as the angle formed between a line bisecting anterior superior iliac spine (ASIS) and the central patella and a line bisecting the central patella and the tibial tubercle [11]. This greater Q angle in women may lead to a relative increased lateral force of the quadriceps muscles on the patellar tendon compared to men, which results in an increased risk of patellar pathology in women, as discussed below. All of these differences are highlighted in Fig. 1.

As boys mature, their femoral condylar size increases along with the width of their femoral intercondylar notch. Girls' intercondylar notch size increases to a lesser degree [12] (Fig. 2).

It should be noted that there are exceptions to these general patterns in anatomic differences. There are geographical differences in body size: women in the Netherlands, for example, might on average be taller than men in some countries in South America and Southeast Asia. This may result in different mechanical forces in one group of women compared to another.

Dynamic anatomic differences between genders

As discussed previously, girls have increased laxity and flexibility compared to boys. Among other things, this can result in delayed hamstring and quadriceps contraction when planting the foot [13]. In general, girls' posture is more upright than boys', which can cause injury (discussed below) [14]. When landing from a jump, girl athletes have different muscular activation patterns than boys, with increased activity of the rectus femoris and decreased activity of the gluteus maximus [15]. Postural differences also cause girl athletes to place more weight on the forefoot than boys [16].

Gender-related non-anatomic differences predisposing to injury patterns

During and after puberty, girls have higher levels of estrogen than boys as well as fluctuating hormone levels, which affects certain musculoskeletal structures such as ligaments [13]. Higher estrogen levels also result in augmented laxity and diminished neuromuscular control of the knee [13].

In addition, there are differences between boys and girls in their training and activity patterns. For example, boy athletes might be more likely to have required strength training and participate in strength training year-round [17].

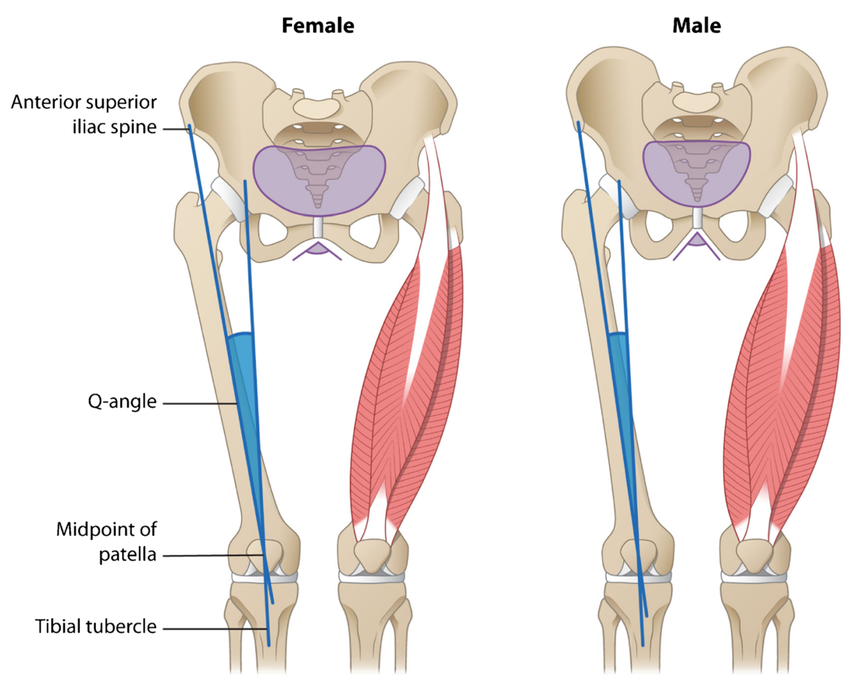

Fig. 1 Illustration demonstrating the increased Q angle (*blue*) and decreased muscle bulk of the female pelvis when compared with the male pelvis. The female pelvic outlet and angle between the ischial tuberosities (*purple*) are wider

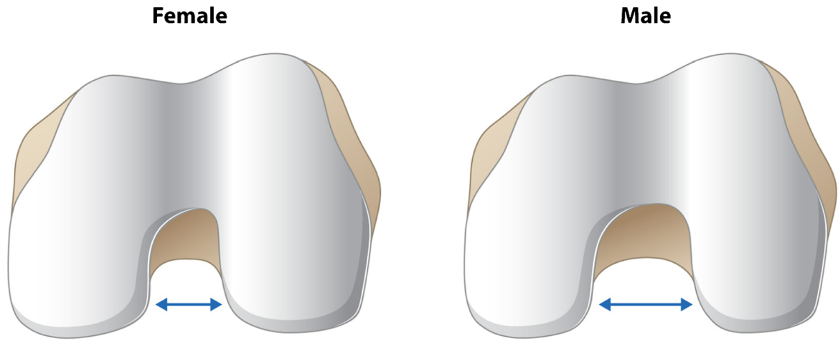

Fig. 2 Illustration demonstrating the narrower intercondylar notch of the female femur (*left*) compared to the male femur (*right*), associated with increased risk of ACL injury in females

Injury patterns typically seen in girl athletes

Hip

A 2016 study by Stracciolini et al. recently demonstrated that girls show a greater increase in hip injuries with advancing age during the teen years than boys [18]. Girls are more likely to have soft tissue-related hip injuries, while boys are more likely to have osseous-related injuries [18]. Overuse injuries, including tendinopathy, are being seen on a larger scale in the adolescent athlete population, with adolescent girl athletes specifically at higher risk than their male counterparts [18].

Labral tears

Incidence of acetabular labral injury is increased in athletes who participate in sports that require pivoting motions on the loaded femur, including ballet, soccer, and golf [19]. Symptomatic labral tears are more common in girls than boys [20]. Acetabular dysplasia, specifically of the pincer type, which is more common in girls and women, is thought to play a role in labral injury [21, 22]. Labral tears are best seen on magnetic resonance (MR) arthrography as contrast filling of a defect in the labrum (Fig. 3), and reports demonstrate the accuracy rate of MR arthrography in the diagnosis of labral tears as greater than 95% [23]. Interpretation of labral tears can be challenging, however, especially in the setting of a contrast-filled cleft between the acetabulum and labrum. An entity known as a sublabral sulcus has been described, which is thought to represent a partially healed tear or normal variant and can be confused for a symptomatic labral tear [24]. However, a retrospective 2015 study that analyzed 41 patients 17 years of age and under who underwent MR arthrography of the hip found that none of these adolescent patients met the criteria for a sublabral sulcus [25]. Thus, a labral base cleft seen in adolescent patients should not be necessarily be considered a normal variant.

Acetabular dysplasia, as discussed above, should be distinguished from femoroacetabular impingement (FAI) syndrome. FAI is a clinical syndrome seen in young athletes who present with hip pain and restricted motion, generally secondary to participation in elite levels of sport or overtraining [26]. The pincer configuration of the acetabulum generally occurs in women at a greater rate than in men, leading to over-coverage of the femoral head with resultant anterior superior labral injury and thinning of the adjacent cartilage. Cam-type configuration is more common in men.

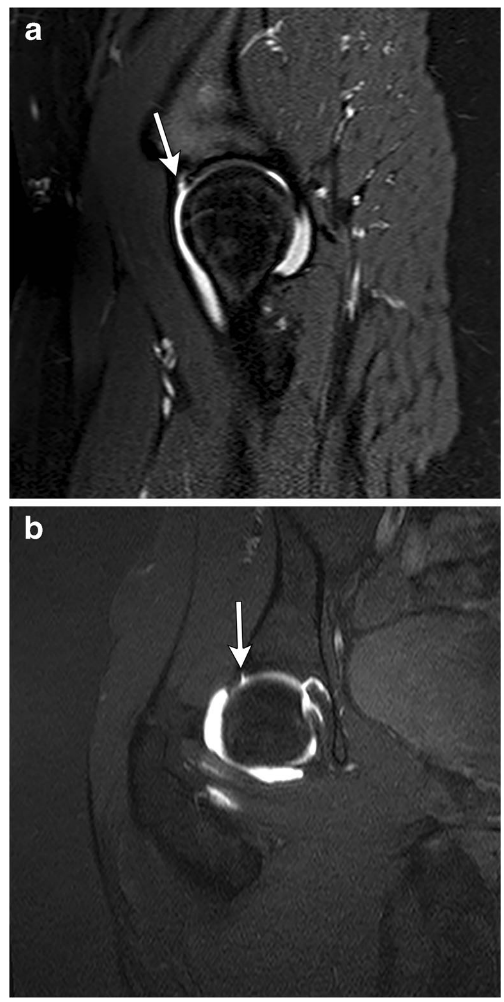

Fig. 3 A 16-year-old girl with labral tear. **a** Oblique sagittal proton density fat-saturated MR arthrogram image of the right hip demonstrates a high signal cleft at the 11 o'clock position (*arrow*) consistent with tear. **b** Coronal proton density fat-saturated image demonstrating the same labral tear (*arrow*)

Women may also be more likely to have a mixed-type FAI configuration, which some describe as a combination of the pincer and cam-type deformities [21, 27].

Snapping hip syndrome

Snapping hip syndrome can occur in adolescent athletes, and because the condition is most frequently seen in ballet dancers, it is especially prevalent in girls [28]. There are two main categories of snapping hip syndrome: extra-articular and intra-articular. Extra-articular etiologies include the actual snapping of tendons over bone, including external snapping hip resulting from the iliotibial band as it moves over the greater trochanter and internal snapping hip resulting from the iliopsoas tendon as it moves over the iliopectineal eminence (Fig. 4). These entities are best assessed with ultrasound, as dynamic imaging can be performed while the patient performs an exacerbating motion. In contrast, intra-articular snapping hip refers to the sensation of snapping caused by etiologies such as intra-articular bodies or acetabular labral tears [29], and these entities are best assessed using magnetic resonance imaging (MRI) or MR arthrography.

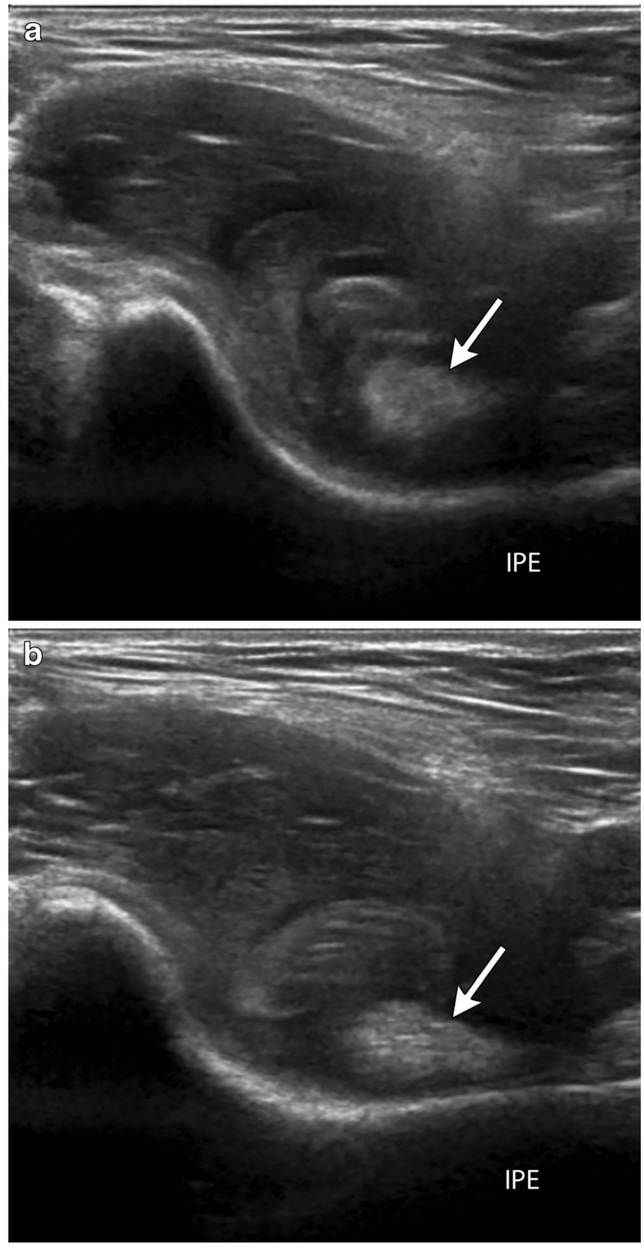

Fig. 4 A 14-year-old dancer with snapping hip syndrome. Transverse grayscale ultrasound images show abrupt motion of the iliopsoas tendon (*arrows*) as the hip is moved between flexion/abduction (**a**) and extension (**b**). IPE = iliopectineal eminence

Apophysitis

A common chronic injury in the adolescent athletic population is traction apophysitis, in which repeated, strong, but submaximal force is applied to an apophysis by a tendon, resulting in microscopic avulsions and inflammation to the apophyseal surface [30]. These injuries tend to be more common in boys, but girls participating in certain sports including gymnastics, dance, and running are also at risk. When apophysitis involves the pelvis, it can present as chronic hip pain and most commonly occurs at the iliac crest (attachment site of the abdominal oblique musculature), the ischial tuberosity (attachment site of the hamstring musculature; Fig. 5), the anterior superior iliac spine (attachment site of the sartorius), and the anterior inferior iliac spine (attachment site of the direct or straight head of the rectus femoris) [29]. On radiographs, the affected apophysis may appear sclerotic, irregular, or fragmented at its margin with the physis. The underlying physis is usually normal or slightly widened without overt displacement [30]. On MRI, increased fluid-sensitive weighted signal is seen in the associated bone and tendon [30].

Knee

ACL injuries

ACL injuries are common in girl and women athletes, occurring at a two to eight times higher incidence than in boys and men [31], and have been extensively discussed in the literature. Certain factors discussed previously have been implicated in this difference in injury rate. A greater Q angle in girls has been speculated to play a role in ACL injury by increasing the lateral force of the quadriceps, resulting in greater valgus stress across the knee [32]. Similarly, the increased activity of the rectus femoris compared to the gluteus maximus also may make girls more susceptible to ACL injury [15]. Girls' upright posture results in decreased knee and hip flexion [33] and increased anterior tibial translation, which can lead to ACL tear [14]. Girl athletes are prone to landing or cutting with the leg in pronation, the knee in valgus, and the

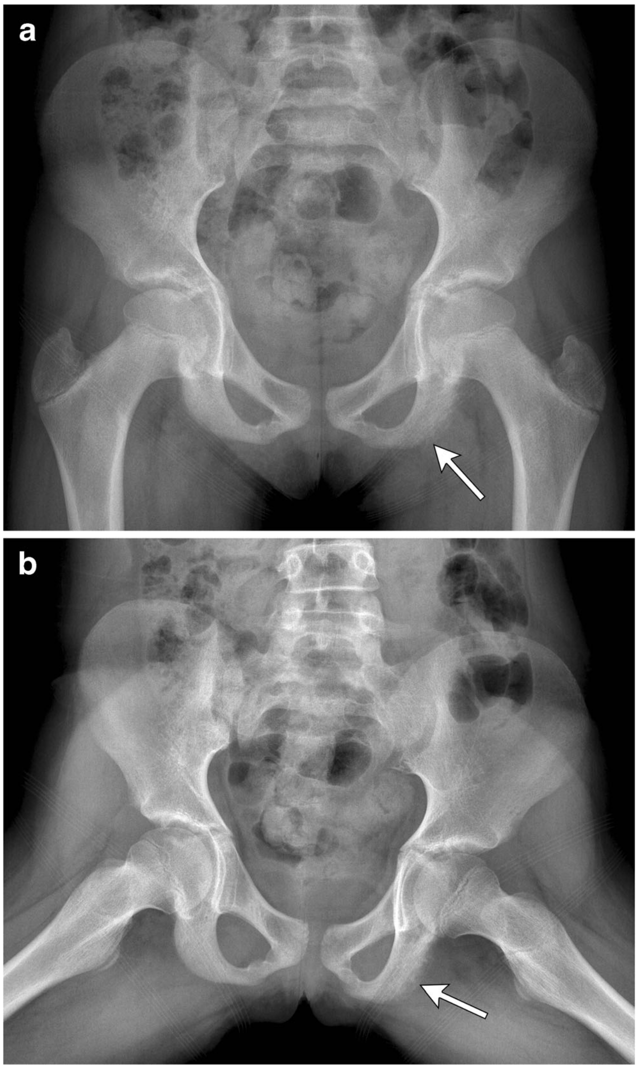

Fig. 5 A 12-year-old girl with ischial tuberosity apophysitis presenting as left groin pain. **a** AP pelvic radiograph demonstrating osseous irregularity and early fragmentation at the left ischial tuberosity (*arrow*). **b** Frogleg pelvic radiograph demonstrating the same findings with left ischial tuberosity osseous irregularity and fragmentation (*arrow*)

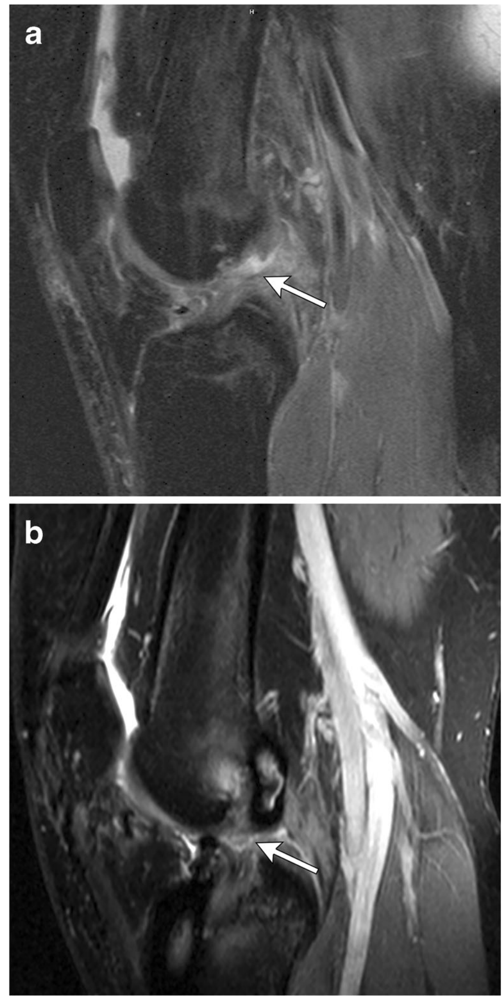

Fig. 6 ACL tear and subsequent re-tear of ACL graft in a 16-year-old girl soccer player. **a** Sagittal proton density fat-saturated MR image through the knee demonstrates complete disruption of ACL fibers (*arrow*) with abnormal orientation. **b** Sagittal proton density fat-saturated MRI image of the same patient following ACL graft repair demonstrates complete tear of the ACL graft (*arrow*)

foot in pronation (the so-called "position of no return"), which places excess strain on the ACL, leaving it prone to tearing [11]. A narrower intercondylar notch in girls is thought to cause increase impact on the ACL [34], and a smaller-sized ACL in girls may also cause increased ligament injury rates [35]. It has been demonstrated that ACL fibroblasts have specific receptors for estrogen [13], and exposure of these receptors to higher estrogen levels in girls reduces collagen synthesis, decreasing ACL bulk and strength [36].

Patients with ACL tears typically present with a discrete knee injury associated with a "popping" sensation. MRI remains the most accurate tool in diagnosing ACL injuries however, with imaging findings of complete tear including frank discontinuity or abnormal position-dependent slope configuration of the ACL during MRI examination (Fig. 6) [37]. Prior studies have demonstrated that neuromuscular training (modification of dynamic aligning factors) can modify ACL injury risk in girl athletes [38].

Patellofemoral pain syndrome (PFPS)

PFPS is defined as anterior knee pain, generally under or around the patella, after other etiologies of pain have been excluded [39]. PFPS, also referred to as "runner's knee" or "jumper's knee" secondary to its predilection to affect athletes who perform these activities, is part of a spectrum that includes lateral femoral condyle friction syndrome, where there is impingement of fat between the lateral femoral condyle and patellar tendon (shown in Fig. 7). PFPS also includes excessive lateral pressure syndrome, defined as anterior knee pain and lateral patellar cartilage degradation due to increased

Fig. 7 A 15-year-old girl cross-country runner with anterior knee pain. Axial (**a**) and sagittal (**b**) proton density fat-saturated MR images of the knee demonstrate edema within Hoffa's fat pad (*arrows*)

lateral patellar tilt. These entities are more common in girl and women athletes, who seek sports medicine clinic visits for patellofemoral pain at a significantly higher rate than boys and men [40]. A 2014 study of girl middle school athletes by Foss et al. demonstrated that the knee was the most common site of injury in this cohort (74%) with 31.3% of knee injuries further classified as patellofemoral pain syndrome [41].

Factors that predispose girls to PFPS include increased Q angle, increased femoral neck anteversion, and a higher incidence of both patella alta and trochlear dysplasia [42, 43]. Trochlear dysplasia is demonstrated on an axial CT or MRI image or axial knee radiograph when the trochlear sulcus measures more than 150 degrees (Fig. 8) [30]. A common measurement of trochlear dysplasia obtained from axial CT or MR images is the tibial tuberosity-trochlear groove (TT-TG) distance, which is defined as the distance between the midpoint of the tibial tuberosity and the midpoint of the trochlear groove [44]. A distance of greater than 15–20 mm is considered abnormal and may be an indication for surgical realignment [45]. Other measurements to assess dysplasia include the lateral trochlear inclination, which describes the shallowness of the angle of the lateral trochlear facet (less than 11 degrees indicates dysplasia), trochlear facet asymmetry, which describes the size of the medial trochlear facet compared to the lateral trochlear facet (a ratio of less than 40% indicates dysplasia), and trochlear depth, which describes the depth of the deepest point of the trochlear groove (3 mm or less indicates dysplasia).

Fig. 8 A 16-year-old girl basketball player with knee pain. Axial proton density fat-saturated MR image through the knee in this patient with knee pain demonstrates the morphology of a shallow trochlear groove (*arrow*)

Dejour et al. have also described four morphologic categories of trochlear dysplasia (Fig. 9). A type A trochlea demonstrates a shallow trochlear groove but is otherwise structurally normal, while a type B trochlea demonstrates a notably flattened or possibly convex trochlea. The type C trochlea has a flattened surface due to a hypoplastic medial femoral condylar facet and high lateral femoral condylar facet. A type D trochlea has the features of a type C trochlea with an additional vertical "cliff" between the lateral and medial facets [46, 47].

Patellar instability

In addition to an increased likelihood of PFPS, multiple studies have demonstrated that girls are more likely than boys to experience patellar instability and dislocation [48]. In addition to the morphologic differences of the patellofemoral

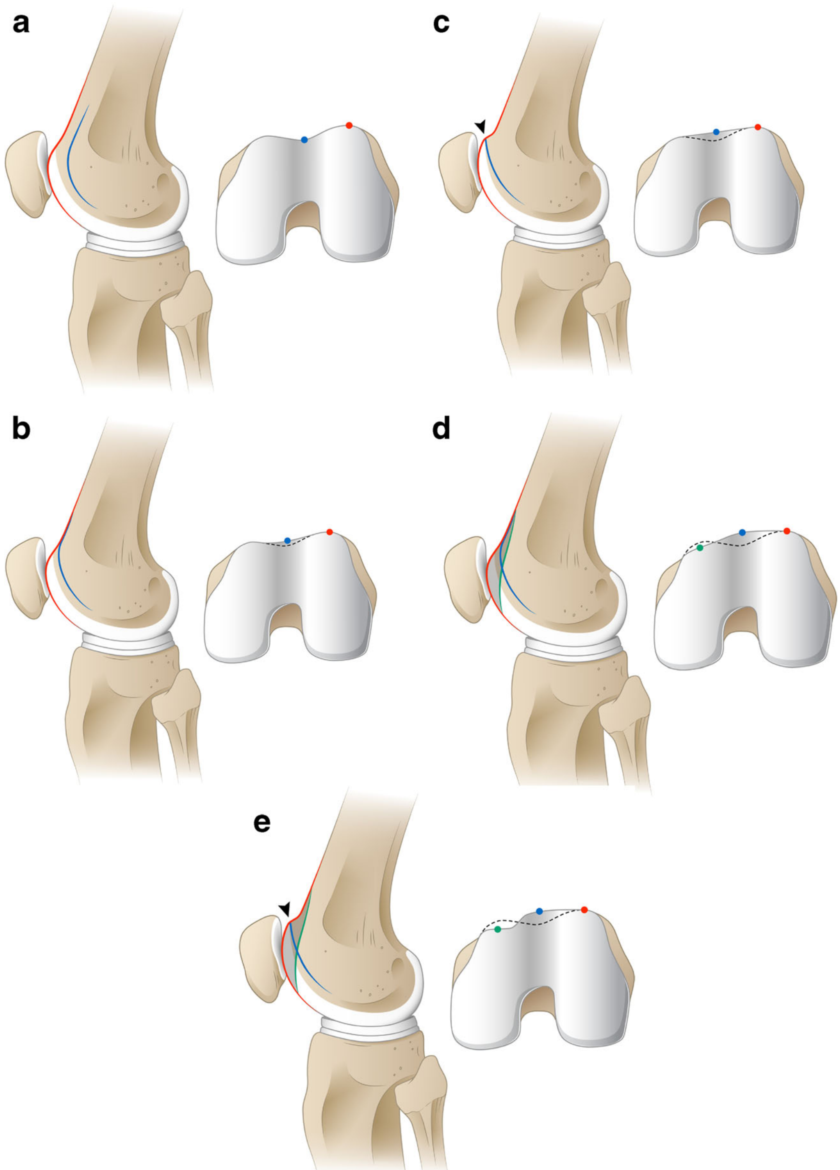

Fig. 9 Illustration of the four categories of trochlear dysplasia as described by Dejour. Sagittal views are on the left and axial views are on the right. **a** Normal appearance of the trochlea. The *red line* indicates the contour of the lateral femoral condyle on the sagittal view, and the *red dot* indicates the lateral condyle on the axial view. The *blue line* indicates the contour of the trochlear groove on the sagittal view, shown as a *blue dot* on the axial view. **b** Type A trochlea with shallow groove; the "crossing sign" is present on the sagittal view, in which the trochlear groove (*blue line*) overlaps with the lateral femoral condyle (*red line*). The *dotted line* on the axial image traces the normal contour of the trochlea. **c** Type B trochlea with a flat, or possibly convex, trochlear groove. The crossing sign is still present along with a trochlear spur (*arrowhead*), which is the osseous protrusion along the anterior femur seen on the sagittal view. **d** Type C trochlea with hypoplastic medial femoral condylar facet and protuberant lateral femoral condylar facet. The *green line and dot* indicate the medial femoral condyle. The crossing sign is present as in type A, and the "double contour sign," in which *red and green lines* are distinguishable, indicates that the contour of the medial femoral condyle is markedly smaller than the contour of the lateral femoral condyle. **e** Type D trochlea with hypoplastic medial femoral condylar facet, protuberant lateral condylar facet, and steep, convex "cliff" between the trochlea and medial facet. The crossing sign, double contour sign, and trochlear spur (*arrowhead*) are all present

compartment discussed above, another contributing factor to this susceptibility includes a relative weakness of hip abductor and external rotator muscles in girls. This weakness results in a predisposition to hip adduction and internal rotation, which consequently increases lateral stress on the patellofemoral joint [49]. Girls also demonstrate preferential use of the lateral quadriceps muscles over the hamstrings and medial quadriceps muscles, possibly because of different neural-controlled preparatory muscle activation compared to boys [50]. This further increases lateral forces on the patella, contributing to patellar instability, subluxation, and dislocation (Fig. 10) [51, 52] .

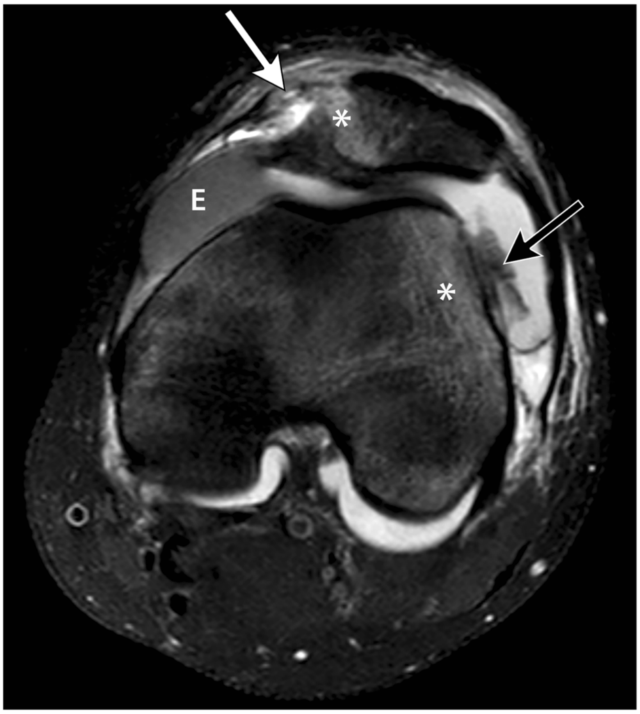

Fig. 10 13-year-old girl soccer player with transient patellar dislocation. Axial proton density fat-saturatedMR image through the knee demonstrates a full-thickness tear of the medial patellar retinacular structures (*white arrow*), bone contusions/edema in the medial patella and lateral femoral condyle (*asterisks*), cartilage fragments/intra-articular bodies (*black arrow*) from a donor site in the patella (not included on this image), and a large, heterogeneous joint effusion (E)

Osgood–Schlatter disease and Sinding–Larsen–Johansson syndrome

Osgood–Schlatter disease (OSD) and Sinding–Larsen–Johansson syndrome (SLJ), both primarily clinical diagnoses, are due to traction apophysitis involving the patellar tendon and are common causes of knee pain in the adolescent athlete. While both OSD and SLJ are more common in boys, girls are also affected, especially those participating in activities that involve squatting, kneeling, or jumping [53]. OSD occurs at the patellar tendon insertion on the tibial tuberosity secondary to excess stress from the quadriceps muscles [53] and occurs in the pre- or early-adolescent age group (8–12 years old). In the previously referenced 2014 Foss study of middle school girl athletes, OSD comprised 10.4% of the reported knee injuries [41]. Radiographic findings of OSD include apophyseal irregularity (Fig. 11), physeal widening, and tendon thickening with intra-tendon osseous fragments present in approximately half of cases [53, 54]. On MRI, increased signal on T2-weighted images can be seen in the distal patellar tendon. Sinding–Larsen–Johansson (SLJ), also a traction apophysitis, instead occurs at the patellar tendon origin at the inferior pole of the patella. SLJ tends to be more prevalent in a slightly younger age group than OSD [55]. Like OSD, radiographic and MRI findings of SLJ include apophyseal irregularity, osseous fragments in the tendon, and thickening and increased signal on T2-weighted images at the patellar tendon origin [56].

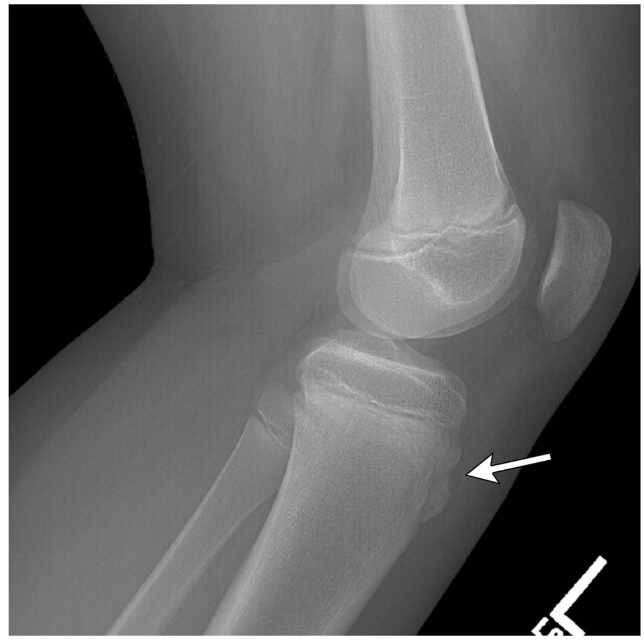

Fig. 11 A 10-year-old girl soccer player with knee pain and Osgood–Schlatter disease. Lateral radiograph of the knee demonstrates apophyseal irregularity at the patellar tendon insertion on the tibial tuberosity (*arrow*)

Foot

Sesamoiditis is a common problem in adolescent athletes who engage in jumping, especially in tennis and ballet, and those who engage in running, which places stress on the forefoot. Girls and boys are equally likely to be affected, but patients with cavus deformity or pronated feet are thought to be particularly predisposed to this condition [57]. Pain and swelling is typically localized to the first metatarsal head [58]. MRI findings include bone marrow edema in one or both sesamoid bones and sometimes edema of the surrounding soft tissues [59]; diagnostic accuracy increases if the edema involves both sesamoid bones.

Shoulder

The shoulder, specifically the proximal humeral epiphysis, is particularly susceptible to chronic overuse injuries in the adolescent athlete. This pattern of injury is typically seen in those who engage in overhead throwing, such as baseball pitchers [60], leading to the moniker “little league shoulder.” While more common in boys, girl athletes who engage in overhead throwing sports are also at risk. Repetitive forces on the proximal humeral epiphysis during throwing leads to physeal microfractures, resulting in a nondisplaced Salter–Harris I pattern of injury

[61]. The pain and arm fatigue associated with this injury can manifest as a decrease in a pitcher's accuracy [62]. Radiographs may be normal, or they may demonstrate physeal widening, subchondral cystic change at the growth plate, sclerosis, or fragmentation [63]. Because the pathologic findings may be subtle, it is often suggested that a radiograph of the contralateral shoulder be obtained [64]. MRI is not routinely performed for this injury but, if obtained, can demonstrate physeal widening and increased signal on T2 weighted images both within the physis and in the underlying subchondral bone, which histologically represents hypertrophied chondrocytes extending into the metaphysis [61].

Wrist

Wrist pain is a common entity among girl athletes, with one study reporting that more than 70% of girl gymnasts complain of pain in their wrist [65]. "Gymnast's wrist" is a loosely used term that may refer to a variety of injuries caused by chronic repetitive compressive stress on the wrist [66]. Most often, it refers to chronic stress-related injury to the distal radial physis [61], essentially representing a nondisplaced Salter–Harris 1 type of stress fracture. Common radiographic findings of this entity include irregularity and asymmetric widening of the physis, sometimes with cystic changes and sclerosis of the metaphyseal aspect [67, 68]. If the physeal injury is severe, there may be premature fusion of the growth plates, resulting in a short radius and subsequent positive ulnar variance. Studies have shown an increased rate of positive ulnar variance in gymnasts [69]. Occasionally, but not classically, other wrist injuries such as scaphoid stress fracture, triangular fibrocartilage complex tears, and ganglion cysts are also lumped into the category of "gymnast's wrist" [70].

Overuse injuries

Stress fractures occur in women collegiate athletes at approximately a 2:1 ratio when compared with men [71], and they are also common in adolescent girl athletes. There are two overall categories of stress fracture: fatigue fracture, in which abnormal stress (overtraining) is placed on normal bone, and insufficiency fracture, in which normal stress is placed on abnormal (osteopenic) bone [72]. On radiographs, stress fractures appear in normal density bone with a range of findings including band-like sclerosis, cortical thickening, periosteal reaction and, when advanced, a linear lucency. Findings are usually more pronounced and can be detected earlier on MRI as periosteal edema which progresses to bone marrow edema, followed by signal changes in the cortex, and eventually a linear, low signal fracture line (Fig. 12).

Greater muscle mass in boy athletes compared to girls is thought to be protective against stress fractures [73]. Other factors that place the girl athlete at increased risk for stress fracture include increased peak hip adduction, increased peak hind foot

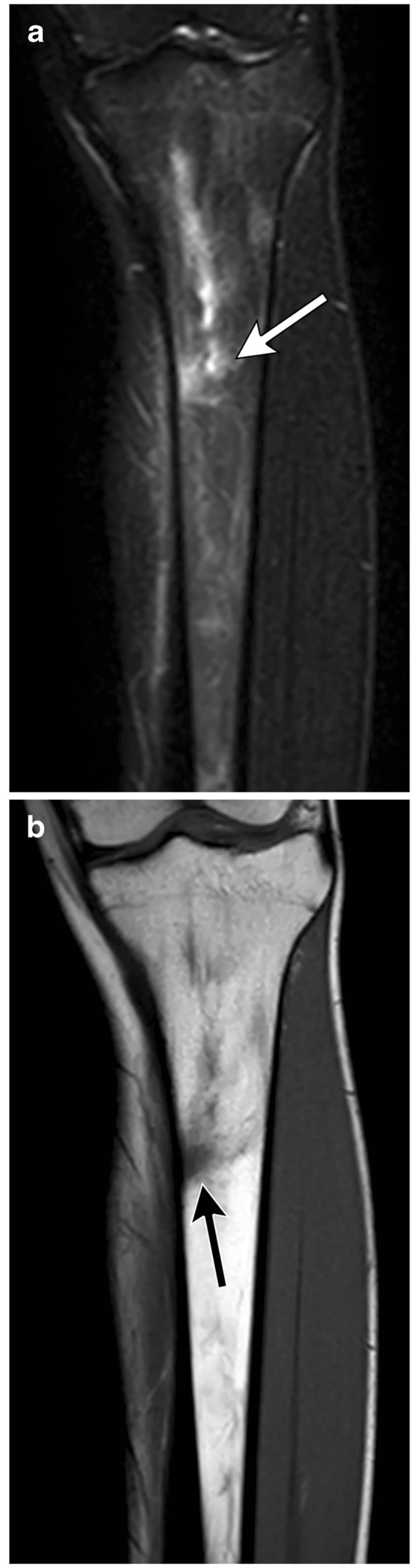

Fig. 12 A 16-year-old girl runner with tibial stress fracture. **a** Coronal STIR MRI image through the tibia demonstrates bone marrow edema in the mid tibial diaphysis (*arrow*). **b** Coronal T1-weighted image from the same study demonstrates corresponding linear low signal (*arrow*) consistent with a stress fracture

eversion, and shorter stature [74, 75]. It has been suggested that stress fractures can be prevented by heavy resistance training with the goal of increasing bone density and muscle mass, but overtraining can also result in abnormal bone remodeling, inadvertently increasing the risk of fracture. A compromise strategy is to ensure adequate rest and incremental training increases [72, 76].

Female athlete triad

The "female athlete triad" is defined as disordered eating, amenorrhea, and osteoporosis [77]. In a study by Byrne et al., it was shown that 31% of girl and women athletes who participate in sports with an aesthetic component (i.e., "thin build" sports such as gymnastics, ballet, and cross-country running) demonstrate disordered eating compared to 5.5% in the control group [78]. More than twice as many girl and women athletes with disordered eating have menstrual irregularities than those who demonstrate non-disordered eating, and athletes with menstrual irregularities including oligomenorrhea or amenorrhea have been shown to have significantly lower bone mineral density than athletes with eumenorrhea [79, 80]. Lower bone mineral density increases the risk of stress fracture, likely explaining why menstrual dysfunction and stress fractures demonstrate a positive correlation [81]. Additionally, girls with anorexia have been demonstrated to have a higher composition of yellow marrow, which tends toward differentiation into adipocytes, in the peripheral skeleton while healthy controls demonstrate a higher composition of red marrow, which tends toward differentiation into osteoblasts [82, 83].

A radiologist may be the first to diagnose the initial finding of decreased bone density on radiographs. Therefore, appropriate radiologic reporting and referral in the setting of a girl athlete with low bone mineral density is crucial to prevent future injury and to direct clinical management. Bone mineral density is typically determined using dual-energy X-ray absorptiometry (DEXA) scans with measurements often made at the hip (femoral neck) and lumbar vertebral bodies. The T score is the measurement expressed in standard deviations compared with "normal" measurements based on that of a 20-year-old woman [84]. For postmenopausal women, a T score less than −1.0 is characterized as osteopenia, and a T score less than −2.5 is characterized as osteoporosis [84]. However, some have suggested that for premenopausal women, the phrase "bone density below expected range for age" should be used instead of "osteoporosis", and the word "osteopenia" should not be used [85]. Additionally, there has been recent controversy as to whether the Z score (comparison to age-matched control) should be used instead of the T score for premenopausal woman [86].

Conclusions

After puberty, there are both anatomical/mechanical and non-anatomical differences between boy and girl athletes that contribute to gender-specific injury patterns. Adolescent girl athletes are more likely to suffer from ACL injuries, patellofemoral pain syndrome, gymnast's wrist, stress fractures, and symptomatic hip labral tears. Other injuries seen in girl athletes include Osgood–Schlatter Disease, Sinding–Larsen–Johansson disease, snapping hip syndrome, and pelvic traction apophysitis. The female athlete triad of disordered eating, amenorrhea, and osteoporosis is common and can increase risk of injury. The radiologist can play a crucial role in initial recognition of low bone mineral density and subsequent referral for DEXA. Knowledge of these gender specific injuries will enable the radiologist to provide better care for young female athletes.

Compliance with ethical standards

Conflicts of interest The authors declare that they have no conflicts of interest.

References

1. Report of Trends and Participation in Organized Youth Sports. In: Sports NCoY, ed. NCYS membership survey 2008.
2. Carpenter LJ, Acosta RV. Women in intercollegiate sport: a longitudinal national study; thirty-five year update, 1977-2012. Human Kinetics. 2012.
3. High School Athletics Participation Survey. In: Associations NFoSHS, ed. 2010–2011.
4. Jansson A, Saartok T, Werner S, Renstrom P. General joint laxity in 1845 Swedish school children of different ages: age- and gender-specific distributions. Acta Paediatr. 2004;93(9):1202–6.
5. Marnach ML, Ramin KD, Ramsey PS, Song SW, Stensland JJ, An KN. Characterization of the relationship between joint laxity and maternal hormones in pregnancy. Obstet Gynecol. 2003;101(2):331–5.
6. Gallagher D, Visser M, De Meersman RE, Sepulveda D, Baumgartner RN, Pierson RN, et al. Appendicular skeletal muscle mass: effects of age, gender, and ethnicity. J Appl Physiol (1985) 1997; 83(1):229–239.
7. Abe T, Kearns CF, Fukunaga T. Sex differences in whole-body skeletal muscle mass measured by magnetic resonance imaging and its distribution in young Japanese adults. Br J Sports Med. 2003;37(5):436–40.
8. Griffin LY, Agel J, Albohm MJ, Arendt EA, Dick RW, Garrett WE, et al. Noncontact anterior cruciate ligament injuries: risk factors and prevention strategies. J Am Acad Orthop Surg. 2000;8(3):141–150.
9. Zeller BL, McCrory JL, Kibler WB, Uhl TL. Differences in kinematics and electromyographic activity between men and women during the single-legged squat. Am J Sports Med. 2003;31(3):449–56.
10. Braten M, Terjesen T, Rossvoll I. Femoral anteversion in normal adults. Ultrasound measurements in 50 men and 50 women. Acta Orthop Scand. 1992;63(1):29–32.
11. Hilibrand MJ, Hammoud S, Bishop M, Woods D, Fredrick RW, Dodson CC. Common injuries and ailments of the female athlete;

pathophysiology, treatment and prevention. Phys Sportsmed. 2015;43(4):403–11.
12. Shelbourne KD, Davis TJ, Klootwyk TE. The relationship between intercondylar notch width of the femur and the incidence of anterior cruciate ligament tears. A prospective study. Am J Sports Med. 1998;26(3):402–8.
13. Hewett TE, Myer GD, Ford KR. Anterior cruciate ligament injuries in female athletes: part 1, mechanisms and risk factors. Am J Sports Med. 2006;34(2):299–311.
14. Ford KR, Myer GD, Smith RL, Vianello RM, Seiwert SL, Hewett TE. A comparison of dynamic coronal plane excursion between matched male and female athletes when performing single leg landings. Clin Biomech (Bristol, Avon). 2006;21(1):33–40.
15. Zazulak BT, Ponce PL, Straub SJ, Medvecky MJ, Avedisian L, Hewett TE. Gender comparison of hip muscle activity during single-leg landing. J Orthop Sports Phys Ther. 2005;35(5):292–9.
16. Willson JD, Ireland ML, Davis I. Core strength and lower extremity alignment during single leg squats. Med Sci Sports Exerc. 2006;38(5):945–52.
17. Reynolds ML, Ransdell LB, Lucas SM, Petlichkoff LM, Gao Y. An examination of current practices and gender differences in strength and conditioning in a sample of varsity high school athletic programs. J Strength Cond Res. 2012;26(1):174–83.
18. Stracciolini A, Yen YM, d'Hemecourt PA, Lewis CL, Sugimoto D. Sex and growth effect on pediatric hip injuries presenting to sports medicine clinic. J Pediatr Orthop B. 2016;25(4):315–21.
19. Mason JB. Acetabular labral tears in the athlete. Clin Sports Med. 2001;20(4):779–90.
20. McCarthy JC, Noble PC, Schuck MR, Wright J, Lee J. The Otto E. Aufranc Award: the role of labral lesions to development of early degenerative hip disease. Clin Orthop Relat Res. 2001;393:25–37.
21. Beck M, Kalhor M, Leunig M, Ganz R. Hip morphology influences the pattern of damage to the acetabular cartilage: femoroacetabular impingement as a cause of early osteoarthritis of the hip. J Bone Joint Surg Br. 2005;87(7):1012–8.
22. Bache CE, Clegg J, Herron M. Risk factors for developmental dysplasia of the hip: ultrasonographic findings in the neonatal period. J Pediatr Orthop B. 2002;11(3):212–8.
23. Ziegert AJ, Blankenbaker DG, De Smet AA, Keene JS, Shinki K, Fine JP. Comparison of standard hip MR arthrographic imaging planes and sequences for detection of arthroscopically proven labral tear. AJR Am J Roentgenol. 2009;192(5):1397–400.
24. Dinauer PA, Murphy KP, Carroll JF. Sublabral sulcus at the posteroinferior acetabulum: a potential pitfall in MR arthrography diagnosis of acetabular labral tears. AJR Am J Roentgenol. 2004;183(6):1745–53.
25. Magerkurth O, Jacobson JA, Morag Y, Fessell D, Bedi A, Sekiya JK. Prevalence of the acetabular sublabral sulcus at MR arthrography in patients under 17 years of age: does it exist? Skelet Radiol. 2015;44(7):953–61.
26. Anderson SE, Siebenrock KA, Tannast M. Femoroacetabular impingement: evidence of an established hip abnormality. Radiology. 2010;257(1):8–13.
27. Tannast M, Siebenrock KA, Anderson SE. Femoroacetabular impingement: radiographic diagnosis—what the radiologist should know. AJR Am J Roentgenol. 2007;188(6):1540–52.
28. Piechota M, Maczuch J, Skupinski J, Kukawska-Sysio K, Wawrzynek W. Internal snapping hip syndrome in dynamic ultrasonography. J Ultrason. 2016;16(66):296–303.
29. Hotchkiss BL, Engels JA, Forness M. Hip disorders in the adolescent. Adolesc Med State Art Rev. 2007;18(1):165–81. x-xi
30. Davis KW. Imaging pediatric sports injuries: lower extremity. Radiol Clin N Am. 2010;48(6):1213–35.
31. Sutton KM, Bullock JM. Anterior cruciate ligament rupture: differences between males and females. J Am Acad Orthop Surg. 2013;21(1):41–50.
32. Nguyen AD, Boling MC, Levine B, Shultz SJ. Relationships between lower extremity alignment and the quadriceps angle. Clin J Sport Med. 2009;19(3):201–6.
33. Loudon JK, Jenkins W, Loudon KL. The relationship between static posture and ACL injury in female athletes. J Orthop Sports Phys Ther. 1996;24(2):91–7.
34. Uhorchak JM, Scoville CR, Williams GN, Arciero RA, St Pierre P, Taylor DC. Risk factors associated with noncontact injury of the anterior cruciate ligament: a prospective four-year evaluation of 859 West Point cadets. Am J Sports Med. 2003;31(6):831–42.
35. Giugliano DN, Solomon JL. ACL tears in female athletes. Phys Med Rehabil Clin N Am. 2007;18(3):417–38. viii
36. Slauterbeck J, Clevenger C, Lundberg W, Burchfield DM. Estrogen level alters the failure load of the rabbit anterior cruciate ligament. J Orthop Res. 1999;17(3):405–8.
37. Renstrom P, Ljungqvist A, Arendt E, Beynnon B, Fukubayashi T, Garrett W, et al. Non-contact ACL injuries in female athletes: an International Olympic Committee current concepts statement. Br J Sports Med 2008; 42(6):394–412.
38. Hewett TE, Lindenfeld TN, Riccobene JV, Noyes FR. The effect of neuromuscular training on the incidence of knee injury in female athletes. A prospective study. Am J Sports Med. 1999;27(6):699–706.
39. Boles CA, Ferguson C. The female athlete. Radiol Clin N Am. 2010;48(6):1249–66.
40. DeHaven KE, Lintner DM. Athletic injuries: comparison by age, sport, and gender. Am J Sports Med. 1986;14(3):218–24.
41. Barber Foss KD, Myer GD, Hewett TE. Epidemiology of basketball, soccer, and volleyball injuries in middle-school female athletes. Phys Sportsmed. 2014;42(2):146–53.
42. Earl JE, Vetter CS. Patellofemoral pain. Phys Med Rehabil Clin N Am. 2007;18(3):439–58. viii
43. Dejour H, Walch G, Nove-Josserand L, Guier C. Factors of patellar instability: an anatomic radiographic study. Knee Surg Sports Traumatol Arthrosc. 1994;2(1):19–26.
44. Schoettle PB, Zanetti M, Seifert B, Pfirrmann CW, Fucentese SF, Romero J. The tibial tuberosity-trochlear groove distance; a comparative study between CT and MRI scanning. Knee. 2006;13(1):26–31.
45. Hinckel BB, Gobbi RG, Filho EN, Pecora JR, Camanho GL, Rodrigues MB, et al. Are the osseous and tendinous-cartilaginous tibial tuberosity-trochlear groove distances the same on CT and MRI? Skelet Radiol. 2015;44(8):1085–93.
46. Dejour H, Walch G, Neyret P, Adeleine P. Dysplasia of the femoral trochlea. Rev Chir Orthop Reparatrice Appar Mot. 1990;76(1):45–54.
47. Diederichs G, Issever AS, Scheffler S. MR imaging of patellar instability: injury patterns and assessment of risk factors. Radiographics. 2010;30(4):961–81.
48. Taunton JE, Ryan MB, Clement DB, McKenzie DC, Lloyd-Smith DR, Zumbo BD. A retrospective case-control analysis of 2002 running injuries. Br J Sports Med. 2002;36(2):95–101.
49. Ireland ML, Willson JD, Ballantyne BT, Davis IM. Hip strength in females with and without patellofemoral pain. J Orthop Sports Phys Ther. 2003;33(11):671–6.
50. Palmieri-Smith RM, Wojtys EM, Ashton-Miller JA. Association between preparatory muscle activation and peak valgus knee angle. J Electromyogr Kinesiol. 2008;18(6):973–9.
51. Palmieri-Smith RM, McLean SG, Ashton-Miller JA, Wojtys EM. Association of quadriceps and hamstrings cocontraction patterns with knee joint loading. J Athl Train. 2009;44(3):256–63.
52. Witvrouw E, Lysens R, Bellemans J, Cambier D, Vanderstraeten G. Intrinsic risk factors for the development of anterior knee pain in an athletic population. A two-year prospective study. Am J Sports Med. 2000;28(4):480–9.

53. Gholve PA, Scher DM, Khakharia S, Widmann RF, Green DW. Osgood–Schlatter syndrome. Curr Opin Pediatr. 2007;19(1):44–50.
54. Rosenberg ZS, Kawelblum M, Cheung YY, Beltran J, Lehman WB, Grant AD. Osgood-Schlatter lesion: fracture or tendinitis? Scintigraphic, CT, and MR imaging features. Radiology. 1992;185(3):853–8.
55. Lyon RM, Street CC. Pediatric sports injuries: when to refer or X-ray. Pediatr Clin N Am. 1998;45(1):221–44.
56. Auringer ST, Anthony EY. Common pediatric sports injuries. Semin Musculoskelet Radiol. 1999;3(3):247–56.
57. McManama GB Jr. Ankle injuries in the young athlete. Clin Sports Med. 1988;7(3):547–62.
58. Hunter-Griffin LY. Injuries to the leg, ankle, and foot. In: Sullivan JA, Grana WA, editors. The pediatric athlete. Park ridge, IL: American Academy of Orthopaedic Surgeons; 1990. p. 187–98.
59. Ashman CJ, Klecker RJ, Yu JS. Forefoot pain involving the metatarsal region: differential diagnosis with MR imaging. Radiographics. 2001;21(6):1425–40.
60. Kinsella S, Carl R. Upper extremity overuse injuries. Clin Pediatr Emerg Med. 2013;14(318)
61. Davis KW. Imaging pediatric sports injuries: upper extremity. Radiol Clin N Am. 2010;48(6):1199–211.
62. Osbahr DC, Kim HJ, Dugas JR. Little league shoulder. Curr Opin Pediatr. 2010;22(1):35–40.
63. Adams JE. Little league shoulder: osteochondrosis of the proximal humeral epiphysis in boy baseball pitchers. Calif Med. 1966;105(1):22–5.
64. Hoang QB, Mortazavi M. Pediatric overuse injuries in sports. Adv Pediatr Infect Dis. 2012;59(1):359–83.
65. Zetaruk MN. The young gymnast. Clin Sports Med. 2000;19(4): 757–80.
66. Little JT, Klionsky NB, Chaturvedi A, Soral A, Chaturvedi A. Pediatric distal forearm and wrist injury: an imaging review. Radiographics. 2014;34(2):472–90.
67. Carter SR, Aldridge MJ, Fitzgerald R, Davies AM. Stress changes of the wrist in adolescent gymnasts. Br J Radiol. 1988;61(722): 109–12.
68. Roy S, Caine D, Singer KM. Stress changes of the distal radial epiphysis in young gymnasts. A report of twenty-one cases and a review of the literature. Am J Sports Med. 1985;13(5):301–8.
69. Dwek JR, Cardoso F, Chung CB. MR imaging of overuse injuries in the skeletally immature gymnast: spectrum of soft-tissue and osseous lesions in the hand and wrist. Pediatr Radiol. 2009;39(12):1310–6.
70. Mandelbaum BR, Bartolozzi AR, Davis CA, Teurlings L, Bragonier B. Wrist pain syndrome in the gymnast. Pathogenetic, diagnostic, and therapeutic considerations. Am J Sports Med. 1989;17(3):305–17.
71. Hame SL, LaFemina JM, McAllister DR, Schaadt GW, Dorey FJ. Fractures in the collegiate athlete. Am J Sports Med. 2004;32(2): 446–51.
72. Arendt EA. Stress fractures and the female athlete. Clin Orthop Relat Res. 2000;372:131–8.
73. Markey KL. Stress fractures. Clin Sports Med. 1987;6(2):405–25.
74. Giladi M, Milgrom C, Simkin A, Stein M, Kashtan H, Margulies J, et al. Stress fractures and tibial bone width. a risk factor. J Bone Joint Surg Br. 1987;69(2):326–329.
75. Behrens SB, Deren ME, Matson A, Fadale PD, Monchik KO. Stress fractures of the pelvis and legs in athletes: a review. Sports Health. 2013;5(2):165–74.
76. Rauh MJ, Macera CA, Trone DW, Shaffer RA, Brodine SK. Epidemiology of stress fracture and lower-extremity overuse injury in female recruits. Med Sci Sports Exerc. 2006;38(9):1571–7.
77. Yeager KK, Agostini R, Nattiv A, Drinkwater B. The female athlete triad: disordered eating, amenorrhea, osteoporosis. Med Sci Sports Exerc. 1993;25(7):775–7.
78. Byrne S, McLean N. Elite athletes: effects of the pressure to be thin. J Sci Med Sport. 2002;5(2):80–94.
79. Nichols JF, Rauh MJ, Barrack MT, Barkai HS, Pernick Y. Disordered eating and menstrual irregularity in high school athletes in lean-build and nonlean-build sports. Int J Sport Nutr Exerc Metab. 2007;17(4):364–77.
80. Nichols JF, Rauh MJ, Lawson MJ, Ji M, Barkai HS. Prevalence of the female athlete triad syndrome among high school athletes. Arch Pediatr Adolesc Med. 2006;160(2):137–42.
81. Barrow GW, Saha S. Menstrual irregularity and stress fractures in collegiate female distance runners. Am J Sports Med. 1988;16(3): 209–16.
82. Ecklund K, Vajapeyam S, Feldman HA, Buzney CD, Mulkern RV, Kleinman PK, et al. Bone marrow changes in adolescent girls with anorexia nervosa. J Bone Miner Res. 2010;25(2):298–304.
83. Ackerman KE, Misra M. Bone health and the female athlete triad in adolescent athletes. Phys Sportsmed. 2011;39(1):131–41.
84. Assessment of fracture risk and its application to screening for postmenopausal osteoporosis. Report of a WHO study group. 1 ed: World Health Organization 1994.
85. Leib ES, Lewiecki EM, Binkley N, Hamdy RC. International Society for Clinical D. Official positions of the International Society for Clinical Densitom. J Clin Densitom. 2004;7(1):1–6.
86. Beals KA, Hill AK. The prevalence of disordered eating, menstrual dysfunction, and low bone mineral density among US collegiate athletes. Int J Sport Nutr Exerc Metab. 2006;16(1):1–23.