Chapter 10
Tennis Injuries of the Hip and Knee

Steven M. Andelman and Alexis C. Colvin

Introduction

Extensive efforts to both quantify and qualify injuries common to the young tennis athlete have come to represent a growing body of literature with contributions from multiple specialists including orthopedic surgeons, physiatrists, athletic trainers, and biomechanical scientists. Early research into the subject focused mainly on epidemiologic studies, attempting to enumerate the most common injuries encountered in competitive youth tennis. Later research concentrated on describing the etiology, diagnosis, and treatment of tennis-specific injuries. The majority of these efforts have focused on conditions of the upper extremity at the elbow and shoulder. It has not been until recently that interest has turned toward the unique biomechanical, anatomic, and pathologic stresses placed on the hip and knee during tennis.

This chapter will serve as a review of the literature of injuries to the hip and knee encountered in the young tennis athlete. Following the trend of the available literature, this review will begin with analysis of the numerous attempts to describe the epidemiology of injuries to the lower extremities within various populations of tennis athletes. The focus will then shift toward the specific injuries of the hip and knee seen in young tennis athletes and end with future directions for research.

S.M. Andelman, MD (✉) • A.C. Colvin, MD
Department of Orthopaedic Surgery,
Mount Sinai Medical Center,
New York, NY, USA
e-mail: steve.andelman@gmail.com

A.C. Colvin, J.N. Gladstone (eds.), *The Young Tennis Player: Injury Prevention and Treatment*, Contemporary Pediatric and Adolescent Sports Medicine,
DOI 10.1007/978-3-319-27559-8_10

Epidemiology of Tennis Injuries

Many early attempts at characterizing tennis injuries were aimed at determining their incidence in different age groups at varying levels of play. A comprehensive 2006 review of literature regarding injuries associated with tennis identified 31 total epidemiologic studies from 1966 to 2006 [1]. These ranged from small prospective studies of elite-level athletes to large, survey-based retrospective studies of recreational-level players. In addition to the variety of studies, each set of authors also provided differing definitions of what constitutes an "injury," thus leading to conflicting levels of reported incidence. Table 10.1 provides a sample of the available literature and demonstrates the variability of study structure as well as the associated inconsistency in the reported results.

Hutchinson et al. followed 1440 participants in the United States Tennis Association (USTA) Boy's Tennis Championships over 6 years and reported an injury rate of 21.5/1000 athletic exposures, with one athletic exposure equivalent to a tennis match [5]. Contrast this to Winge et al. who followed 89 elite-level young male and female athletes over one tennis season and reported 2.3 injuries/1000 h of tennis [3]. Assuming the relative comparability between "athletic exposures" and "hours of tennis," this nearly tenfold difference can most likely be attributed to the strict definition of an injury by Winge in comparison to that used by Hutchinson. Winge defined an injury as that which "handicapped" an athlete or required "special attention" from medical personnel, while Hutchinson defined an injury as anything requiring medical assistance.

Better clarity, especially from the perspective of the treating physician, can come from studies with a more strict definition of injury. Defining an injury as that which prevented tennis play for greater than seven days, Jayanthi et al. reported an incidence of 3.0 injuries/1000 h [9]. Similarly, Lanese et al. prospectively followed 12 male and 11 female college-level athletes monitoring for injuries that prevented participation in competitive play and reported an incidence of 1.6 and 1.0 injuries/1000 h, respectively [4]. Corroborating the results of these early epidemiologic studies, Hjelm et al. reported a rate of 1.7 and 0.6 injuries/1000 h of tennis for male and female athletes, respectively [9]. Using a more strict definition of "injury" as an incident that causes an athlete to be unable to compete, the true incidence of injury for both recreational and competitive tennis players likely lies between 0.6 and 3.0 injuries/1000 h as reported above.

To put the incidence of tennis-related injuries in young athletes into context, they can be compared to reported rates of injury for other common sports. Hootman et al. published 16 years of data on collegiate injuries sustained during practice and competitive events [12]. In this retrospective review, an injury was defined as that which caused an athlete to miss at least one day of competitive play. All data was expressed in terms of "athlete events" (AE), defined as the participation of one athlete in one practice or game. Reported rates of injury ranged from 35.9 injuries/1000 AE in men's football to 4.3 injuries/1000 AE in women's softball. While this data was expressed in terms of "athletic events," and not in "hours of sport"

Table 10.1 Selected epidemiologic publications on tennis injuries

Study	Format	Population	Number	Definition of injury	Incidence of injury
Reece [2]	Retrospective	Elite tennis athletes ages 16–20	45	An injury that required medical attention from a physician or athletic trainer	M: 2.5 injuries/player/year F: 2.9 injuries/player/year
Winge [3]	Prospective	Elite M + F Danish young tennis athletes	89	An injury that handicapped an athlete or required special treatment	Total: 2.3 injuries/1000 h of tennis M: 2.7 injuries/1000 h F: 1.1 injuries/1000 h
Lanese et al. [4]	Prospective	M + F college athletes	23	Any injury resulting in absence of play	M: 1.6 injuries/1000 h F: 1.0 injury/1000 h
Hutchinson [5]	Retrospective	USTA boys championship < 18 years old	1440	All injuries that required medical attention	9.9 new injuries/100 athletes 21.5 injuries/1000 athletic exposures
Hahn [6]	Retrospective	Danish competitive athletes 14–35	339	Any knee pain in preceding 12 months	42.5 % with knee pain 15 % with absence due to pain
Sallis [7]	Retrospective	Mixed sport M + F college athletes 18–22	3767 total athletes, injuries separated by sport, no sport-specific "N" given	An injury that required attention of athletic trainer	M: 0.465 injuries/player/year F: 0.425 injury/player/year
Silva [8]	Prospective	Brazilian junior tennis circuit M + F 12–18	258	Any consultation or treatment given to an athlete during a tournament	6.9 medical treatments/1000 games played
Jayanthi [9]	Retrospective questionnaire	M + F recreational tennis players	428	Any injury preventing play for > 7 days	3.0 injuries/1000 h
Hjelm [10]	Prospective	Swedish competitive tennis players 12–18	55	An injury that makes it impossible to participate in regular tennis on at least 1 occasion	M: 1.7 injuries/1000 h F: 0.6 injury/1000 h

Table partially modified from Pluim et al. [1] and Abrams et al. [11].

Table 10.2 Common sites of tennis-related injuries

Study	% Lower extremity	% Upper extremity	% Trunk	% Knee injuries	% Hip injuries
Reece [2]	59 %	20 %	21 %	–	–
Winge [3]	39 %	46 %	24 %	4.3 %	–
Hutchinson [5]	49 %	26 %	25 %	19.2 %	8.8 %
Sallis et al. [7]	M: 62.2 % F: 70.7 %	M: 23.1 % F: 21.9 %	M: 14.6 % F: 7.2 %	M: 6.11 % F: 11.0 %	M: 3.7 % F: 8.5 %
Jayanthi et al. [9]	41 %	43 %	11 %	12 %	5 % *
Hjelm [10]	50 %	25 %	25 %	14 %	5 % **

*denoted as thigh/groin strain
**denoted as groin strain

Table 10.3 Common types of tennis-related injuries

Study	Mechanism of injury
Winge [3]	Sprain: 17 % Strain: 14 % Fracture: 2 %
Hutchinson [5]	Sprain: 58 % Strain: 21 % Contusion: 7 %
Silva et al. [8]	Muscle contracture: 26 % Strain: 11 % Sprain: 6 %

such as the aforementioned epidemiologic data for tennis injuries, it is appropriate to assume that tennis has a relatively low injury rate when compared to other common sports.

A subset of the epidemiologic studies also reported on the location and type of injury sustained during tennis play. Table 10.2 represents a review of the location of injury with special attention to the hip and knee. Although differences exist, the majority of data suggests that lower extremity injuries predominate with approximately twice as many injuries occurring at the knee compared to the hip.

Of those studies that reported on etiology of injury, chronic injuries are shown to be more common than acute injuries [3, 9, 10]. Looking specifically at acute injuries, sprains, strains, and muscular contusions occur with relative frequency, while fractures and ligamentous tears were rarely reported (Table 10.3). Only Hjelm looked specifically at mechanism of injury, reporting that 30 % of all injuries occurred while hitting a ball, 24 % occurred during training exercises, 9 % were attributed to twisting injuries, and 5 % were attributed to acceleration/deceleration movements [10].

Overall, the epidemiologic studies of injuries in young tennis athletes reveal a relatively low incidence of injury with a predominance of chronic injuries over acute occurring most frequently in the lower extremities.

Development and Anatomy of the Hip and Knee

A basic understanding of the development and anatomy of the hip and knee is essential prior to discussion of common lower extremity injuries seen in young tennis athletes.

The knee is composed of two articulations – tibiofemoral and patellofemoral. The tibiofemoral articulation is a ginglymus or "hinge-type" articulation that allows for rotation as well as flexion and extension in the sagittal plane. The tibiofemoral articulation functions to transmit forces from the femur to the tibia. Stability to the tibiofemoral articulation is derived from the surrounding ligamentous and tendinous structures. The medial and lateral collateral ligaments resist valgus and varus stress, respectively, while the anterior cruciate ligament is the primary restraint to anterior translation of the tibia on the femur, and the posterior cruciate ligament is the primary restraint to posterior translation of the tibia on the femur. Multiple other dynamic stabilizers play a role in tibiofemoral function including the gastrocnemius, popliteus, and hamstring muscles. In the developing skeleton two primary physes are present – one at the distal femur and one at the proximal tibia. These two physes allow for the highest rate of longitudinal growth in the body with the distal femoral physis growing at a rate of 0.9 mm/year and the proximal tibial physis growing at a rate of 0.6 mm/year [13]. Closure of the distal femoral and proximal tibial physes occurs at the onset of skeletal maturity, considered to be between 13 and 14 years of age in females and 15–16 years of age in males [13] (Fig. 10.1).

The patellofemoral joint is a gliding articulation that functions to transmit forces from the quadriceps tendon to the patellar tendon to facilitate extension of the tibiofemoral joint. The patella sits within the trochlea of the distal femur. The quadriceps tendon inserts on the superior pole of the patella, while the patellar tendon originates at the inferior pole of the patella and inserts distally on the tibial tubercle. Further stability of patellar motion is derived from the insertions of the vastus medialis and lateralis as well as the medial patellofemoral ligament. The primary structure of importance in the developing skeleton is the tibial tubercle apophysis into which the patellar tendon inserts. This secondary growth center is cartilaginous up until age 11 after which an apophysis begins to form that is visible on radiographic examination. Apophyseal maturation occurs from age 11 to 14. From age 14 to 18, the apophysis fuses with the tibial epiphysis, and after age 18 the fused epiphysis and apophysis fuse to the rest of the tibia [14].

The hip is a ball-and-socket joint with the proximal femoral head articulating into the acetabulum. The hip allows for multidirectional movement including flexion, extension, adduction, abduction, and internal and external rotation. Hip stability and motion is determined both by the bony articulation between the femoral head and the acetabulum as well as the many dynamic stabilizing muscles that cross the hip joint including the gluteus medius and minimus (abduction); the adductor magnus, longus, and brevis (adduction); the gluteus maximus (extension); the iliopsoas (flexion); and the short external rotator muscles (external rotation). Further

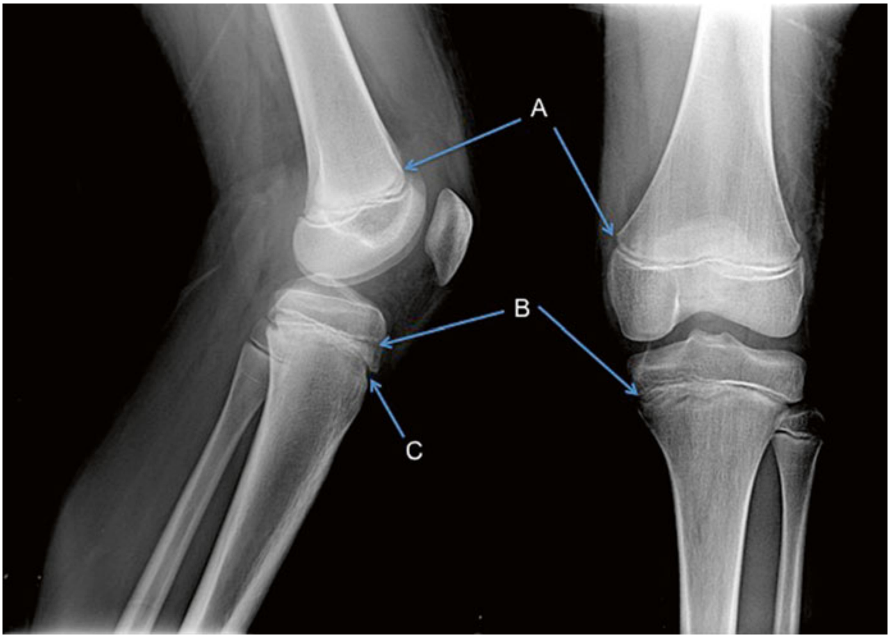

Fig. 10.1 Lateral and AP radiographs of the pediatric knee. (*A*) Distal femoral physis, (*B*) proximal tibial physis, (*C*) tibial tubercle apophysis, and attachment of the patellar tendon

stability comes from the hip labrum, analogous to the labrum of the shoulder, which provides a deepening of the acetabular socket.

The developing pediatric hip is composed of multiple ossification centers that arise at varying ages. While the femoral shaft and femoral capital epiphysis are present at birth, the femoral head appears at 4–6 months of age, the greater trochanter appears at 2–4 years of age, and the lesser trochanter appears later during puberty [15]. All ossification centers fuse after the onset of puberty between 14 and 18 years of age. The acetabulum is composed of the triradiate cartilage that represents the confluence of the ilium, ischium, and pubic innominate bones. Not a true secondary center of ossification, the triradiate cartilage fuses between 14 and 18 years of age [16, 17] (Fig. 10.2).

Knee Injuries

From a biomechanical perspective, tennis places a high level of stress on the knee joint and surrounding ligamentous and tendinous stabilizing structures. Tennis play requires short bursts of high-intensity running interspersed with the stopping, starting, pivoting, and twisting motions necessary to reach the location of the ball, maneuver to set up the impending stroke, and return the ball to the opponent. The

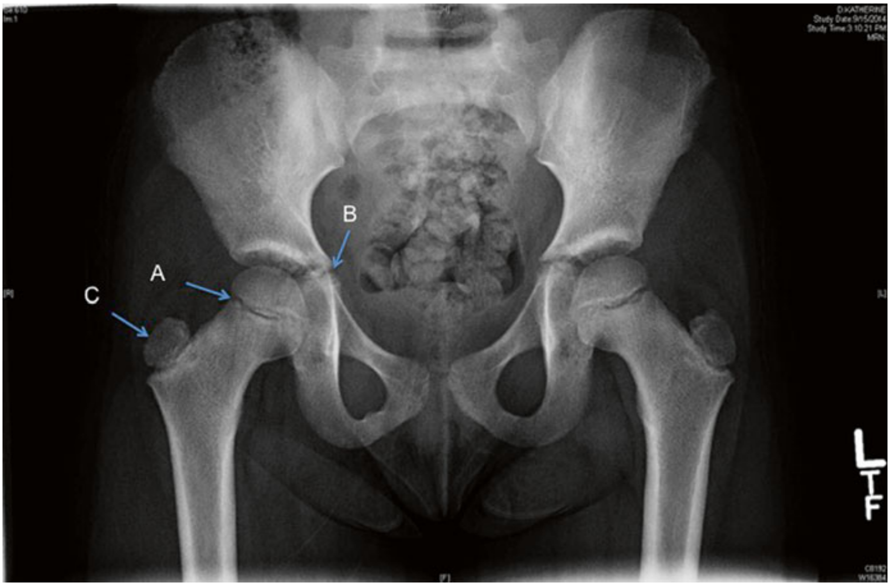

Fig. 10.2 AP radiograph of the pediatric hip. (*A*) Proximal femoral physis, (*B*) apophysis of the greater trochanter, (*C*) triradiate cartilage

average tennis point involves more than eight changes of direction, each placing a load of 1.5–2.7 times the involved body weight through the knee joint and surrounding structures [18]. Available data suggests that 4.3–19.2 % of all tennis-related injuries occur to the knee [3, 5, 8, 9]. While the majority of tennis injuries in young athletes tend to be chronic overuse injuries to the patellofemoral compartment and extensor mechanism, the twisting and pivoting motions necessary for tennis play produce a unique profile of injuries to the tendinous and ligamentous stabilizing structures of the knee.

Chronic Knee Pain

A significant amount of knee injuries encountered in young tennis athletes have been characterized as "overuse" injuries [2–4]. The repetitive knee flexion and extension during tennis play leads to microtrauma to the knee joint. Published rates of chronic knee injuries have varied from 30 to 72 % [5, 10, 19]. The majority of these injuries can be localized to the extensor mechanism and patellofemoral compartment with patellofemoral pain syndrome, quadriceps and patellar tendonitis, Osgood-Schlatter's disease (OSD), iliotibial band (ITB) syndrome, and bursitis being the most commonly encountered diagnoses [10, 23, 37]. There have been few attempts to determine the true incidence of the each individual diagnosis. Hjelm et al. reported on 14 chronic knee injuries out of 100 total injuries. Within this subset, the authors reported on one case of patellofemoral pain syndrome (7 %), three cases of patellar or quadriceps tendinopathy (21 %), two cases of ITB syndrome (14 %), and two cases of OSD (14 %) [10].

Differentiating between causes of chronic knee pain and arriving at a single diagnosis can be difficult due to the subtle differences of presentation between the varying pathologies. The following represents an overview of the most common causes of chronic knee pain in young tennis athletes including etiology, diagnosis, and initial treatment modalities.

Patellofemoral Pain Syndrome

Patellofemoral pain syndrome (idiopathic chondromalacia patellae) is characterized by aching anterior knee pain exacerbated by knee flexion. Pain is caused by maltracking of the patella within the trochlear groove leading to abnormal forces within the patellofemoral compartment. It can arise due to a multitude of intrinsic anatomic factors including a high "q angle" caused by increased femoral anteversion or external tibial torsion, trochlear dysplasia, or lateral patellar instability [20]. The reported incidence of patellofemoral pain syndrome in young tennis athletes is between 7 and 16 % [10, 21]. It is important to note that patellofemoral pain syndrome is due to inherent anatomic variations to the patellofemoral compartment that are exacerbated by the repetitive knee flexion required by tennis play (Fig. 10.3).

Patellofemoral pain syndrome is a difficult diagnosis to make and is many times considered a diagnosis of exclusion. Presence of obvious anatomic causes as

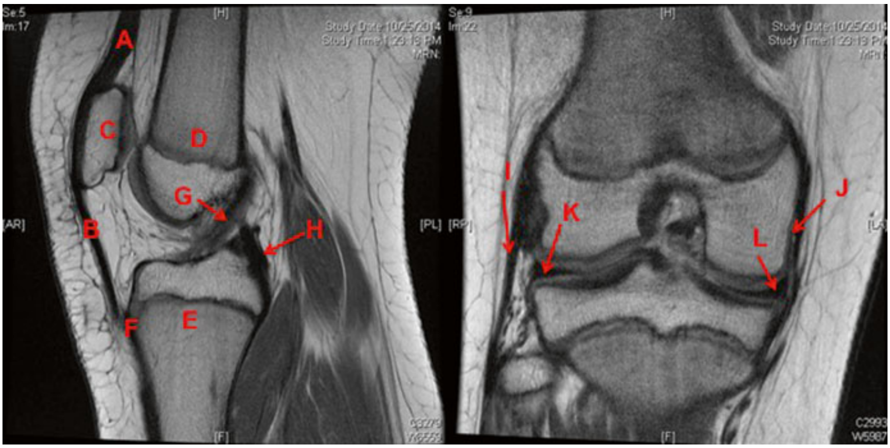

Fig. 10.3 Sagittal and coronal views of the pediatric knee. (*A*) Quadriceps tendon, (*B*) patellar tendon, (*C*) patella, (*D*) distal femoral physis, (*E*) proximal tibial physis, (*F*) insertion of patellar tendon into tibial tubercle apophysis, (*G*) anterior cruciate ligament, (*H*) posterior cruciate ligament, (*I*) lateral collateral ligament, (*J*) medial collateral ligament, (*K*) lateral meniscus, (*L*) medial meniscus

described above as seen on either x-ray or advanced imaging facilitates such a task. In the absence of such findings, many patients will complain of generalized anterior knee pain without point tenderness. Patients may complain of pain with other actions requiring deep knee flexion such as navigation of stairs. If a diagnosis of patellofemoral pain syndrome is suspected, initial treatment always involves rest, nonsteroidal anti-inflammatory medications, and long-term physical therapy directed toward core and quadriceps strengthening [22].

Quadriceps and Patellar Tendonitis

Quadriceps and patellar tendonitis occur at the insertion of the quadriceps tendon at the superior pole of the patella and the origin of the patellar tendon at the inferior pole of the patella, respectively. Both diagnoses are seen often in sports requiring knee flexion and extension due to repetitive eccentric contractions of the extensor mechanism leading to microtears at the bone-tendon interface [21, 23]. Quadriceps and patellar tendonitis are thus understandably seen in young tennis athletes given the repetitive deep knee flexion and extension necessary to complete a tennis stroke.

While both quadriceps and patellar tendonitis cause diffuse anterior knee pain, they can be differentiated by location of point tenderness and exacerbating maneuvers. Whereas quadriceps tendonitis will lead to pain with palpation of the superior pole of the patella and be exacerbated with knee extension, patellar tendonitis will lead to pain with palpation of the inferior pole of the patella and be exacerbated with knee flexion. Clinical examination is usually sufficient to diagnose quadriceps and patellar tendonitis. However, knee ultrasound and magnetic resonance imaging (MRI) can help confirm diagnosis or aide in diagnosing more subtle presentations.

Treatment is based on conservative measures including rest, nonsteroidal anti-inflammatory medications, and physical therapy initiated upon resolution of pain directed first at range of motion exercises before progressing to loading of the quadriceps mechanism [24, 25].

Osgood-Schlatter's Disease (OSD)

Osgood-Schlatter's disease (tibial tubercle traction apophysitis) is another common cause of pain in adolescent athletes who participate in sports that involve jumping or flexion activities. It arises due to repetitive tension placed on the insertion of the patellar tendon at the apophysis of the tibial tubercle in skeletally immature patients prior to fusion between the apophysis and tibial epiphysis. Repetitive traction forces placed on the unfused apophysis lead to point tenderness and pain over the tibial tubercle. In this manner OSD can be seen as analogous to patellar tendonitis in skeletally immature tennis athletes [26].

Diagnosis of OSD is made purely on a clinical basis in skeletally immature patients with tibial tubercle tenderness associated with knee flexion. Imaging rarely plays a role in diagnosis, although x-ray imaging can demonstrate irregularity of the tibial tubercle [27].

Like patellar tendonitis, initial treatment is conservative and aimed at rest, nonsteroidal anti-inflammatory medications, and physical therapy directed at quadriceps stretching to decrease tension on the unfused apophysis. Recalcitrant cases can be treated with an extended period of long leg cast immobilization or, if skeletally mature, excision of any residual fragmented ossicles of the tibial tubercle [28] (Fig. 10.4).

Iliotibial Band Friction Syndrome

Iliotibial band (ITB) syndrome arises from excessive friction between the ITB and lateral femoral condyle. It has been associated with a variety of anatomic and physiologic abnormalities including weak hip abductors, a tight ITB, increased tibial

Fig. 10.4 Lateral radiograph of the knee demonstrating tibial tubercle apophyseal irregularities associated with OSD [29]

internal rotation, genu varum, and mismatch between hamstring and quadriceps strength – all of which can cause maltracking of the ITB and irritation at the area overlying the lateral femoral condyle. ITB friction syndrome is seen commonly in sports that require repetitive knee flexion and extension such as cycling and running as well as in those athletes who undergo a sudden, rapid increase in training intensity [30]. ITB friction syndrome has been identified in young tennis athletes, especially at the onset of training during preseason conditioning [21].

Diagnosis of ITB friction syndrome can be made clinically with point tenderness over the lateral femoral condyle in the region of the ITB with reproduction of pain with deep knee flexion. The Ober test can help to identify ITB pathology. In this maneuver the patient is placed lateral lying on the unaffected side. The affected knee is brought from flexion and abduction to extension and adduction. Decreased ability to adduct is suggestive of ITB tightness, while pain during the maneuver is suggestive of ITB inflammation. Radiographs and MRI do not so much aide in diagnosis as help to identify possible underlying anatomic abnormalities and rule out lateral knee compartment intraarticular pathology [30].

After a diagnosis of ITB friction syndrome, conservative treatment is initiated with a focus on stretching the ITB and strengthening hip abductors. In the rare instance of recalcitrant ITB friction syndrome, corticosteroid injections have proven effective. Surgical ITB lengthening represents an option of last resort after all conservative measures have failed [30].

Bursitis

There are multiple bursas about the knee that can become irritated and inflamed from the flexion, twisting, and pivoting motions that occur throughout a tennis match. The most commonly encountered diagnoses are prepatellar bursitis, pes anserinus bursitis, and semimembranosus bursitis [21]. There is no published literature concerning the incidence of these injuries in young tennis athletes. Diagnosis of bursitis is clinical and based on tenderness to the bursa in question and pain with associated provocative movements. Treatment is aimed at conservative management with rest and nonsteroidal anti-inflammatory medications with corticosteroid injections relegated for those patients unresponsive to more conservative measures.

Acute Knee Injuries

Acute ligamentous injuries of the knee are less common than chronic overuse injuries. Reported incidence of acute knee injuries varies widely depending on the definition of injury, with published rates ranging from 28 to 70 % [10, 19]. The most common types of acute knee injuries are ligamentous "sprains," representing

6–58 % of all knee injuries, although there is no specification as to which knee structure is injured in these studies [3, 5, 8, 10]. Historically, the medial collateral ligament (MCL) was the most frequently injured knee structure in tennis [18, 21]; however there is little published data on tennis injuries to the MCL. Based on the available data, the medial meniscus is the most frequently injured structure followed by the anterior cruciate ligament (ACL) and lateral meniscus [31, 32].

Intraarticular Knee Injuries

A review of 129 tennis-related knee injuries as a subset of over 3000 sport-related intraarticular injuries over a 10-year period diagnosed via MRI or arthroscopy revealed 66 injuries to the medial meniscus, 33 ACL injuries, and 19 lateral meniscal injuries, while only two injuries to the MCL were noted [31]. The 51.2 % incidence of medial meniscal injuries was nearly five times the 10.8 % average incidence of medial meniscal injury across all sports reviewed. In contrast to the high rate of meniscal injuries, tennis was associated with a relatively low rate of injuries to the ACL. ACL injuries were the most common pathology encountered across all sports, diagnosed in 45 % of all subjects. This is nearly double the 26 % incidence of ACL injuries encountered in tennis athletes. This data confirms an earlier review of 128 arthroscopies performed for injuries sustained during racquet sports, including tennis, which reported a 36 % incidence of meniscal injury and a 10.8 % incidence of ACL injury [32].

As tennis is a noncontact sport dependent on repetitive pivoting and twisting motions throughout a given match, a predominance of injuries to the menisci and a relatively low rate of injuries to the ACL are consistent with regular tennis play. However, it is important to note that both aforementioned studies involve athletes of all ages and do not focus solely on young tennis players. It is generally felt that young tennis athletes have a relatively low rate of intraarticular ligamentous injuries [18, 19] (Fig. 10.5).

Diagnosis of intraarticular knee injuries should be guided by clinical exam with MRI used only for confirmatory purposes. Differentiation between meniscal and ACL injuries can be challenging. Injuries to the ACL are usually associated with an immediate large knee effusion, while the relatively avascular nature of the meniscus causes more subtle effusions. Patients with an ACL injury will complain of knee instability and will have increased anterior translation of the proximal tibia on the distal femur during anterior drawer and Lachman testing. In contrast, patients with meniscal injuries will likely complain more often of locking or mechanical knee symptoms with associated tenderness to palpation over the affected tibial plateau joint line [33].

A full discussion of the treatment of intraarticular knee injuries in tennis is beyond the scope of this chapter. Broadly, decisions regarding treatment should take into account the age, competitive level, associated injuries, injury type, patient preference, and surgeon expertise.

Fig. 10.5 Sagittal view of pediatric knee demonstrating a tear of anterior cruciate ligament

Tennis Leg

Tennis leg is an eponym for an acute muscular strain or tear at the myotendinous origin of the medial head of the gastrocnemius [34, 35]. This injury occurs with sudden transition from ankle plantar flexion to dorsiflexion with the knee in extension, as seen on the back leg during the tennis serve [36, 37]. Originally described in adult and recreational athletes, tennis leg is felt to be relatively uncommon in young tennis players [19].

Diagnosis is based on a compelling history of injury with tenderness over the course of the medial head of the gastrocnemius muscle, more commonly occurring in the proximal half of the muscle belly. MRI can be used as an adjunct to confirm suspected diagnosis demonstrating inflammation along the muscle belly. As with the chronic knee injuries described above, treatment is conservative with an initial period of rest and nonsteroidal anti-inflammatory medications followed by physical therapy for range of motion and strengthening exercises.

Hip Injuries

The hip and pelvis play an essential role in tennis in generating the rotational torque necessary to allow for transmission of power from the lower extremities to the upper extremities and eventually through the swinging racquet. Biomechanical studies

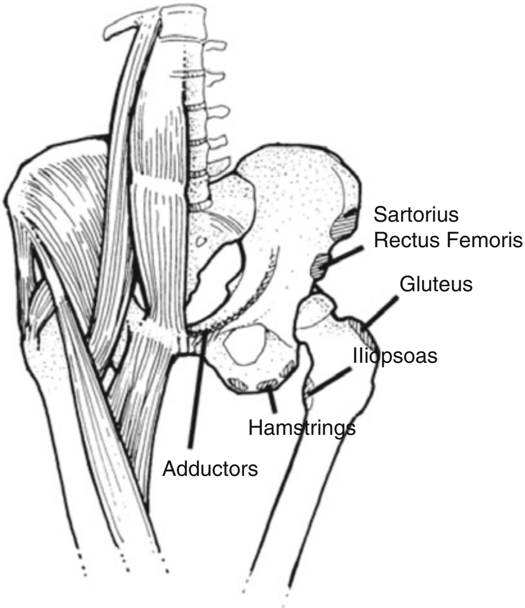

Fig. 10.6 Schematic representation of the major muscular stabilizers of the hip joint. The adductors are a common site of injuries in young tennis athletes [39]

have demonstrated that the trunk and pelvis rotate at 350°/s [18] with an associated pelvic angular displacement of 54–60° throughout the backhand and forehand strokes [38]. Generation of such movements is reliant on hip adduction at the front or leading leg and hip extension at the back leg, with subtle differences dependent on stroke type [38]. An understanding of the forces transmitted through the hip provides a starting point for studying the injury patterns to the hip in young tennis athletes (Fig. 10.6).

Adductor Muscle Strains

Available data suggests that the incidence of hip injuries, including injuries to the "groin," represents 3.7–8.8 % of all tennis-related injuries [5, 7, 10]. The majority of all documented injuries to the hip are adductor muscle strains. Empirically, this is felt to be due to the extreme leg abduction that can occur while reaching for a ball in the "split" position. The high incidence of adductor strains is further explained by the lead hip adduction observed during the tennis stroke as discussed above.

Diagnosis and treatment are similar as for strains of the medial head of the gastrocnemius. Diagnosis is clinical with pain over the medial hip at the insertion of the adductors. Treatment is conservative and based on limiting abduction, nonsteroidal anti-inflammatory medications, and gradual return to play.

Intraarticular Injuries

In the aforementioned epidemiologic studies, there have been no attempts to describe hip injuries in young tennis athletes beyond “sprains” and “strains” to the groin. Recently, there have been endeavors to characterize the effects of tennis play on both hip rotation and the dynamic stabilizing muscles of the hip joint given the repetitive asymmetric loading of the hip during tennis.

Using MRI in to characterize the major stabilizers of the hip joint, Sanchis-Moysi et al. found that young, elite tennis athletes had asymmetric hypertrophy of the nondominant (back leg) iliopsoas and symmetric hypertrophy of the gluteal muscles when compared to control subjects [40]. However, asymmetric differences in the dynamic stabilizers of the hip do not appear to affect hip rotation. Ellenbecker et al. described the normative hip internal and external rotation values for 147 young, elite male and female tennis athletes and found no statistically significant difference in internal or external rotation between the dominant and nondominant hips [41]. While there may not be differences in dominant and nondominant hip rotation in asymptomatic patients, Vad et al. found a statistically significant decrease in dominant hip internal rotation in tennis athletes with self-reported low back pain [42].

While differences in muscle development can exist between the dominant and nondominant hips, these differences do not necessarily impact hip range of motion. This is especially relevant given the recent interest in femoroacetabular impingement as a source of hip and low back pain in young athletes. Femoroacetabular impingement, described as abnormal contact between the femoral head/neck junction and the anterior lip of the acetabulum, is associated with pain and decreased range of motion on hip internal rotation [43–45]. Therefore, it should not be presumed to be “normal” if a young athlete presents with differences in dominant and nondominant hip rotational range of motion as this may be an indicator of hip pathology requiring further attention [41].

Thus careful attention to history and physical exam must be taken in examining the young tennis athlete complaining of hip pain. Hip pain associated with decreased hip internal rotation, pain with internal and external hip rotation, or a history of a “snapping” or “clunking” hip should raise suspicion of intraarticular hip pathology and warrant consideration of advanced imaging with MRI.

Effect of Playing Surface on Hip and Knee Injuries

Tennis is unique in that athletes commonly play on vastly different surfaces with frictional properties that place varying levels of stress on the hip and knee. Research has shown that there is a decreased rate of lower extremity injury on surfaces that allow for sliding (clay) compared to hard court surfaces (asphalt) [46, 47], although the overall rate of early match retirement due to injury in professional tennis was found to be equal between hard court and clay court surfaces [48]. Biomechanical studies have demonstrated surface-based differences in knee flexion during approach to the ball. Hard court surfaces with a higher coefficient of friction were associated with greater knee flexion than clay surfaces where the athlete is able to slide [49]. Thus play on hard court surfaces places greater eccentric stress on the extensor mechanism during knee flexion. This could be one reason for the observed high rates of chronic patellofemoral pain and injuries to the extensor mechanism in tennis athletes.

There have been no known analogous studies to the effects of playing surface on the biomechanics of the hip. However, it would be expected that surfaces with decreased friction would lead to an increased risk for adductor strains while sliding with the legs in abduction.

Risk Factors for Lower Extremity Injuries

In order to adequately address injury prevention in tennis athletes, it is essential to identify risk factors for injury. In a 2-year prospective study of 55 young tennis athletes, Hjelm et al. determined that the only statistically significant risk factor for injury in tennis was existence of a previous injury [50]. Playing greater than 6 h of tennis a week was associated with increased back pain but did not lead to increased rates of injuries to the lower extremities. These findings confirm previous investigations that showed no difference in injury rate based on sex or level of play [7, 9]. The association between sustaining a previous injury and developing a new injury has led some to suggest that injury risk increases with inadequate rehabilitation and treatment of old injuries [50]. By allowing young athletes to return to sport prior to full rehabilitation, they may be at increased risk to reinjury or may make adjustments to compensate for the previous injury that leads to new injury from altered biomechanics.

Conclusion

In conclusion, the young tennis athlete is subject to a unique profile of lower extremity injuries. In the knee, chronic overuse injuries to the patellofemoral compartment and extensor mechanism predominate. The most frequent acute knee injuries are sprains to ligamentous structures, with injuries to the medial meniscus and ACL being most prevalent. In the hip and groin, the most common injuries are adductor strains. Less is known regarding intraarticular injuries to the hip and its surrounding stabilizing structures, although recent research into hip rotation has begun to shed light on intraarticular hip pathology. The biomechanics of hip morphology and range of motion and their relationship to injury in tennis is a growing field that requires further study. There have been recent attempts to identify risk factors for injuries that have demonstrated a link between having sustained an old injury and developing a new injury, although more work is required on this matter. Currently, researchers have a relative grasp of the incidence and types of injuries to the hip and knee that occur in tennis. Future research should be directed toward further elucidation of the risk factors for developing such injuries in order to help tennis athletes avoid future injury.

References

1. Pluim BM, Staal JB, Windler GE, Jayanthi N. Tennis injuries: occurrence, aetiology, and prevention. Br J Sports Med. 2006;40(5):415–23.
2. Reece L, Fricker P, Maguire K. Injuries to elite young tennis players at the Australian Institute of Sports. Aust J Sci Med Sport. 1986;18:11–5.
3. Winge S, Jorgensen U, Lassen Nielsen A. Epidemiology of injuries in Danish championship tennis. Int J Sports Med. 1989;10:368–71.
4. Lanese RR, Strauss RH, Leizman DJ, et al. Injury and disability in matched men's and women's intercollegiate sports. Am J Public Health. 1990;80:1459–62.
5. Hutchinson MR, Laprade RF, Burnett QM, Moss R, Terpstra J. Injury surveillance at the USTA Boys' Tennis Championships: a 6-yr study. Med Sci Sports Exerc. 1995;27(6):826–30.
6. Hahn T, Foldspang A. Prevalent knee pain and sport. Scand J Soc Med. 1998;26(1):44–52.
7. Sallis RE, Jones K, Sunshine S, et al. Comparing sports injuries in men and women. Int J Sports Med. 2001;22:420–3.
8. Silva RT, Takahashi R, Berra B, et al. Medical assistance at the Brazilian juniors tennis circuit–a one-year prospective study. J Sci Med Sport. 2003;6:14–8.
9. Jayanthi N, Sallay P, Hunker P, et al. Skill-level related injuries in recreational competition tennis players. Med Sci Tennis. 2005;10:12–5.
10. Hjelm N, Werner S, Renstrom P. Injury profile in junior tennis players: a prospective two year study. Knee Surg Sports Traumatol Arthrosc. 2010;18(6):845–50.
11. Abrams GD, Renstrom PA, Safran MR. Epidemiology of musculoskeletal injury in the tennis player. Br J Sports Med. 2012;46(7):492–8.
12. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. J Athl Train. 2007;42(2):311–9.
13. Weinstein SL, Flynn JM. Lovell and Winter's pediatric orthopedics. Vol 2, Ch. 28: limb length discrepancy. Lipincott Williams & Wilkins; Philadelphia, PA 2006.
14. Ogden JA, Southwick WO. Osgood-Schlatter's disease and tibial tuberosity development. Clin Orthop Relat Res. 1976;116:180–9.
15. Panattoni GL, D'amelio P, Di stefano M, Isaia GC. Ossification centers of human femur. Calcif Tissue Int. 2000;66(4):255–8.
16. Fabricant PD, Hirsch BP, Holmes I, et al. A radiographic study of the ossification of the posterior wall of the acetabulum: implications for the diagnosis of pediatric and adolescent hip disorders. J Bone Joint Surg Am. 2013;95(3):230–6.
17. Weinstein SL, Flynn JM. Lovell and Winter's pediatric orthopedics. Vol 2, Ch. 23: developmental hip Dysplasia and dislocation. Lipincott Williams & Wilkins; Philadelphia, PA 2006.
18. Kibler WB, Safran MR. Musculoskeletal injuries in the young tennis player. Clin Sports Med. 2000;19(4):781–92.
19. Kibler WB, Safran M. Tennis injuries. Med Sport Sci. 2005;48:120–37.
20. Collado H, Fredericson M. Patellofemoral pain syndrome. Clin Sports Med. 2010;29(3):379–98.

21. Renström AF. Knee pain in tennis players. Clin Sports Med. 1995;14(1):163–75.
22. Fulkerson JP. Patellofemoral pain disorders: evaluation and management. J Am Acad Orthop Surg. 1994;2(2):124–32.
23. Gecha SR, Torg E. Knee injuries in tennis. Clin Sports Med. 1988;7(2):435–52.
24. Rutland M, O'connell D, Brismée JM, Sizer P, Apte G, O'connell J. Evidence-supported rehabilitation of patellar tendinopathy. N Am J Sports Phys Ther. 2010;5(3):166–78.
25. Hak DJ, Sanchez A, Trobisch P. Quadriceps tendon injuries. Orthopedics. 2010;33(1):40–6.
26. Ducher G, Cook J, Spurrier D, et al. Ultrasound imaging of the patellar tendon attachment to the tibia during puberty: a 12-month follow-up in tennis players. Scand J Med Sci Sports. 2010;20(1):e35–40.
27. Kujala UM, Kvist M, Heinonen O. Osgood-Schlatter's disease in adolescent athletes. Retrospective study of incidence and duration. Am J Sports Med. 1985;13(4):236–41.
28. Pihlajamäki HK, Mattila VM, Parviainen M, Kiuru MJ, Visuri TI. Long-term outcome after surgical treatment of unresolved Osgood-Schlatter disease in young men. J Bone Joint Surg Am. 2009;91(10):2350–8.
29. Micheli LJ, Kocher MS. The pediatric and adolescent knee: Ch. 17 tendonopathy of the extensor apparatus of the knee. W B Saunders Company; Philadelphia, PA 2006.
30. Strauss EJ, Kim S, Calcei JG, Park D. Iliotibial band syndrome: evaluation and management. J Am Acad Orthop Surg. 2011;19(12):728–36.
31. Majewski M, Susanne H, Klaus S. Epidemiology of athletic knee injuries: a 10-year study. Knee. 2006;13(3):184–8.
32. Powell JM, Kavanagh TG, Kennedy DK, et al. Intra-articular knee injuries in racquet sports. A review of 128 arthroscopies. Surg Endosc. 1988;2:39–43.
33. Frank JB, Jarit GJ, Bravman JT, Rosen JE. Lower extremity injuries in the skeletally immature athlete. J Am Acad Orthop Surg. 2007;15(6):356–66.
34. Millar AP. Strains of the posterior calf musculature ('tennis leg'). Am J Sports Med. 1979;7:172–4.
35. Delgado GJ, Chung CB, Lektrakul N, et al. Tennis leg: clinical US study of 141 patients and anatomic investigation of four cadavers with MR imaging and US. Radiology. 2002;224:112–9.
36. Kulund DN, Mccue FC, Rockwell DA, Gieck JH. Tennis injuries: prevention and treatment. A review. Am J Sports Med. 1979;7(4):249–53.
37. Perkins RH, Davis D. Musculoskeletal injuries in tennis. Phys Med Rehabil Clin N Am. 2006;17(3):609–31.
38. Akutagawa S, Kojima T. Trunk rotation torques through the hip joints during the one- and two-handed backhand tennis strokes. J Sports Sci. 2005;23(8):781–93.
39. Anderson K, Strickland SM, Warren R. Hip and groin injuries in athletes. Am J Sports Med. 2001;29(4):521–33.
40. Sanchis-Moysi J, Idoate F, Izquierdo M, et al. Iliopsoas and gluteal muscles are asymmetric in tennis players but not in soccer players. PLoS One. 2011;6, e22858.
41. Ellenbecker TS, Ellenbecker GA, Roetert EP, Silva RT, Keuter G, Sperling F. Descriptive profile of hip rotation range of motion in elite tennis players and professional baseball pitchers. Am J Sports Med. 2007;35(8):1371–6.
42. Vad VB, Gebeh A, Dines D, Altchek D, Norris B. Hip and shoulder internal rotation range of motion deficits in professional tennis players. J Sci Med Sport. 2003;6(1):71–5.
43. Safran MR. Evaluation of the hip: history, physical examination, and imaging. Oper Tech Sports Med. 2005;13:2–12.
44. Kelly BT, Weiland DE, Schenker ML, Phillippon MJ. Arthroscopic labral repair in the hip: surgical technique and review of the literature. Arthroscopy. 2005;21:1496–504.
45. Schenker ML, Martin R, Weiland DE, Philippon MJ. Current trends in hip arthroscopy: a review of injury diagnosis, techniques and out- come scoring. Curr Opin Orthop. 2005;16:89–94.

46. Bastholt P. Professional tennis (ATP tour) and number of medical treatments in relation to type of surface. Med Sci Tennis. 2000;11(40):981–990.
47. Nigg BM, Segesser B. The influence of playing surfaces on the load on the locomotor system and on football and tennis injuries. Sports Med. 1988;5(6):375–85.
48. Breznik K, Batagelj V. Retired matches among male professional tennis players. J Sports Sci Med. 2012;11(2):270–8.
49. Damm L, Low D, Richardson A, Clarke J, Carré M, Dixon S. The effects of surface traction characteristics on frictional demand and kinematics in tennis. Sports Biomech. 2013;12(4):389–402.
50. Hjelm N, Werner S, Renstrom P. Injury risk factors in junior tennis players: a prospective 2-year study. Scand J Med Sci Sports. 2012;22(1):40–8.