A Case-Based Approach to Knee Pain

A Pocket Guide to Pathology, Diagnosis and Management

Michelle Leong · Grant Cooper
Joseph E. Herrera
Peter Murphy *Editors*

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Editors
Michelle Leong
Department of Rehabilitation
Mount Sinai Hospital
New York, NY, USA

Joseph E. Herrera
Department of Rehabilitation Medicine
Mount Sinai Hospital
New York, NY, USA

Grant Cooper
Princeton Spine and Joint Center
Princeton, NJ, USA

Peter Murphy
Princeton Spine and Joint Center
Princeton, NJ, USA

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For Mark, Sandy, Tommy, and Riley

Michelle Leong

For Ana, Mila, Lara, Luka, Twinkle, and Lili

Grant Cooper

Tara, Cassandra, and Dr. Maheep Vikram

Peter Murphy

Foreword

Scabbed knees were a badge of honor of sorts during our elementary years; however, as a reader of this manuscript you most likely are wondering what the mechanism of injury was, and whether it warrants an x-ray given the evidence of trauma. We start our lives weight-bearing directly onto our knees, progress to scrapes and bruises, and then load them daily while allowing gravity to play its role too—all proof that knees are quite resilient.

As clinicians who are faced with the increasing incidence of knee pain (which plagued 25% of adults 20 years ago, with a significant increase in incidence over the last two decades), it is important to become familiar with the anatomy of this joint and the pathologies that can combat its many structures. With the most subtle of misaligned movements, one is at risk of disrupting the many structures that both protect and make up the joint. It is not just activity that causes this magnificent joint to become the center of a chief complaint, but also the lack of activity proving to be just as harmful. I am confident that as you read through the pages of this manuscript, you will widen your knowledge and ability to differentiate between affected structures in your patients and the cause of their pathology. Through this case-based approach, you will have a greater understanding of the appropriate workup and treatment for the most common diagnoses associated with knee

pain. With the shared expertise of these authors, I am confident that you will be better equipped to treat any patient to walk through your door seeking your help in treating their resilient, indispensable knees.

Icahn School of Medicine at Mount Sinai Mariam Zakhary
New York, NY, USA

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Michelle Leong

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Grant Cooper

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Joseph E. Herrera, DO, FAAPMR
Chair and Lucy G. Moses Professor
Department of Rehabilitation and Human Performance
Mount Sinai Health System

Director of Sports Medicine
Icahn School of Medicine at Mount Sinai
Interventional Spine and Sports Medicine Division
Department of Rehabilitation and Human Performance

I want to thank everyone who helped come together to make this book possible. I am honored to be a part of this. A loving thank you to my wife Tara and daughter Cassie. I'd also like to thank Dr. Maheep Vikram whose mentorship through the years has been invaluable.

Peter Murphy

Contents

Knee Anatomy

1

Laurenie Louissaint
and Aditya Raghunandan

Learning Objectives

- To recognize normal anatomy of the knee adapting a layered approach.
- Understand the function of anatomical structures.

Introduction

Conceptually, the knee can be seen as being composed of two joints: the tibiofemoral joint and the patellofemoral joint. The overall stability of the knee is dependent on static (cruciate and collateral ligaments) and dynamic (hamstring muscles, extensor mechanisms, and the popliteus muscle) stabilizers, muscle attachment sites, menisci, the joint capsule, and the knee's bony topography. As we dive into its anatomy, readers will gain an understanding of how all these structures reinforce the knee stability and function.

L. Louissaint (✉)
Department of Human Performance and Rehabilitation, Icahn School of Medicine, Mount Sinai Hospital, New York, NY, USA

A. Raghunandan
Department of Rehabilitation Medicine, Long School of Medicine at the University of Texas Health Science Center at San Antonio, San Antonio, TX, USA
e-mail: raghunandan@uthscsa.edu

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The Anterior Knee

Anterior Knee Joints

The knee is a hinged capsular joint with three separate articulations: the patellofemoral, medial, and lateral tibiofemoral joints (Fig. 1.1).

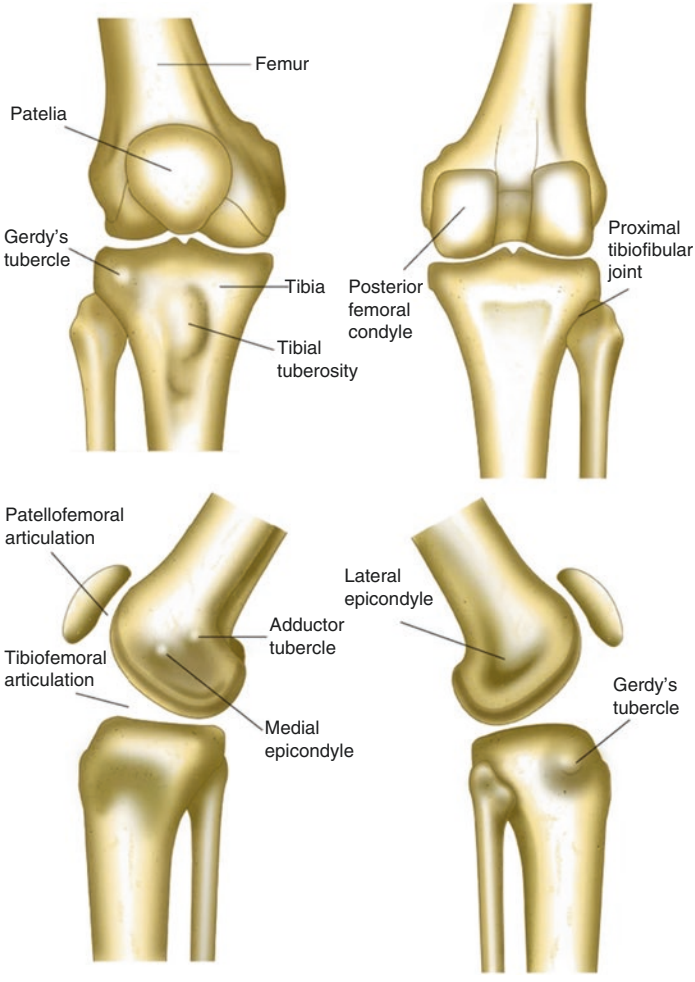

Fig. 1.1 Bones of the knee: anterior and posterior view, side view medial and lateral [3]

The tibiofemoral joint is a hinge joint that aids in some of the knee's primary functions, such as transmission of body weight from the femur to the tibia by allowing sagittal plane rotation and a small degree of tibial axial rotation [1]. The medial and lateral femoral condyles are the femur's distal convex surfaces that articulate with the tibia's medial and lateral tibial plateau, respectively. The medial tibial plateau has a concave articular surface while the lateral plateau has an anteroposterior convexity. This topography accounts for the "screw-home mechanism" or internal rotation of the femur, on the fixed tibia as the knee approaches extension [2].

The patellofemoral joint, a gliding joint, is the articulation between the patella and the femoral sulcus. The patella is the body's largest sesamoid bone. It has a concave superficial surface and an articular surface with a vertical central ridge that separates the medial and lateral facets which articulate with the femur. It has a distal attachment to the deep layer of the patellar tendon and a proximal attachment to the vastus intermedius [1].

Extensor Mechanism

The patellofemoral articulation is also referred to as the extensor mechanism, which is composed of the quadriceps muscle group, the patella, and the patellar tendon (Fig. 1.2). Disruption of any of these components impedes a person's ability to actively extend the knee or resist passive flexion.

The quadriceps muscle group forms the primary motor unit of the extensor mechanism. The rectus femoris merges with vastus medialis obliquus, vastus medialis, vastus lateralis, vastus lateralis oblique which terminate in an aponeurosis; these in turn merge into the anterior-third joint capsule's retinacular layer that extends to form the quadriceps tendon attachment to the patella [2]. The patellar tendon originates at the inferior pole of the patella and inserts at the tibial tubercle, the distal component of the extensor mechanism. Rectus femoris' attachment at the anterior inferior iliac spine allows it to also function as a hip flexor. The quadriceps muscle group is innervated by the femoral nerve (L2–L4) and receives vascular supply from femoral artery [5].

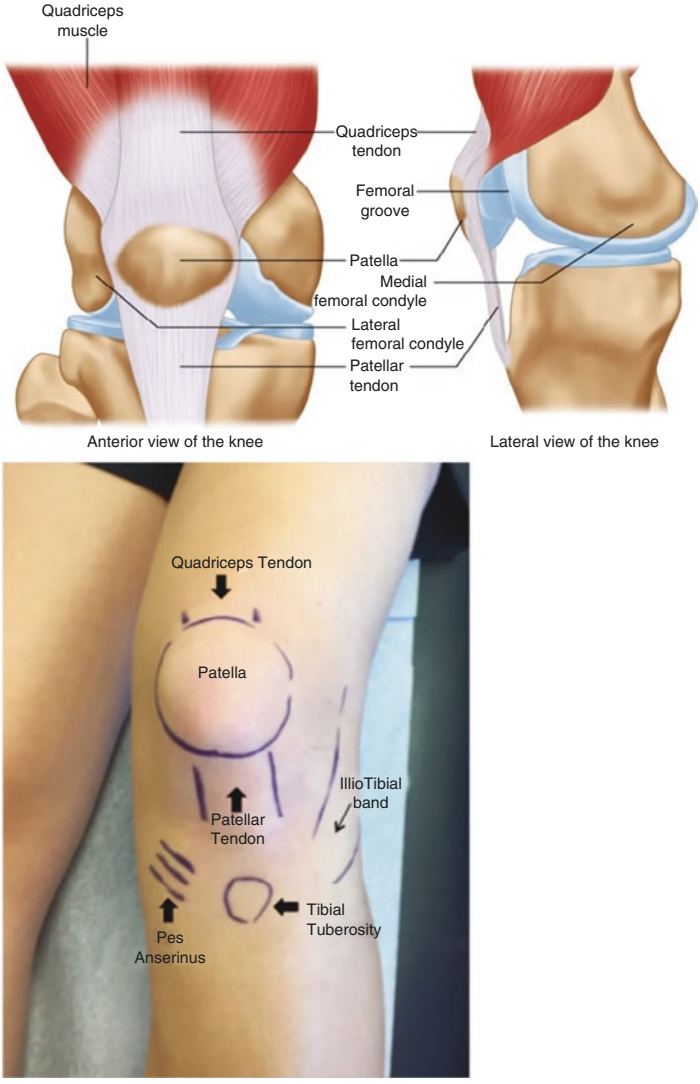

Fig. 1.2 The extensor mechanism [4]

Bursae are small fluid-filled sacs that reduce friction between moving parts in your body's joints. There are two anterior bursae; the prepatellar bursa lies superficial to the patella and the infrapatellar bursa lies between the patellar tendon and underlying tibia.

Articular Cartilage and Capsular Ligaments

The knee's articular surfaces are covered with hyaline cartilage which aids in reducing friction during joint movements. The meniscus is another form of articular cartilage known as fibrocartilage. The medial and lateral menisci are interposed in the tibiofemoral joint (Fig. 1.3).

Menisci play a crucial role in load-bearing, load transmission, shock absorption, as well as lubrication and nutrition of articular cartilage [6]. The medial meniscus is C-shaped and less mobile while the lateral meniscus is saucer-shaped in cross-section and more mobile [2, 5].

The meniscus receives its vascular supply from the superior and inferior medial and lateral geniculate arteries with most of vascular supply distributed to its peripheral outer third. The

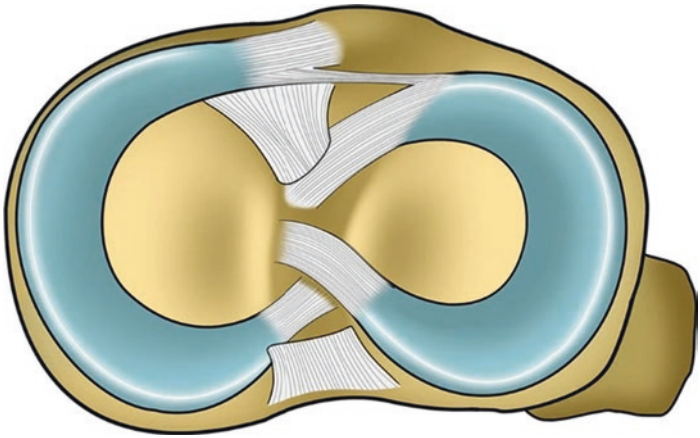

Fig. 1.3 Top view of the medial and lateral meniscus [3]

meniscus may thus be described into three zones according to its vascularity: firstly the outer (red) zone, which is red and rich in blood vessels, secondly the inner (white) zone is avascular, and lastly its intermediate (red-white) zone. The distribution of this vascular supply has great significance in meniscal injuries as tears in the outer zone (red zone) heal while most tears to the inner (white) zone mostly do not heal [5]. The knee joint is innervated by the posterior articular branch of the posterior tibial nerve and the terminal branches of the obturator and femoral nerves [7]. Similar to its vascular supply, the outer third of the body of the meniscus is more densely innervated than the middle third [8].

A capsule, which is part of the retinacular layer, surrounds the entire joint and extends into the suprapatellar pouch. It contains a synovial lining that also covers the cruciate ligaments, making them intra-articular but extrasynovial [5]. The four ligaments branch off the retinacular layer. The medial and lateral patello-femoral ligaments course along the distal and deep structures of the vastus medialis obliques and the vastus lateralis obliquus, then attach at superior poles of the patella. Attached to the inferior poles are the medial and lateral patellotibial ligaments [1]. The Arciform layer, the most superficial layer, is a thin peritendinous membrane which blends anteriorly into the patellar tendon [1]. The plica is a remnant of embryonic development. Its size varies from one person to another. It can be tissue paper thin but can thicken, scar down, and become contracted (plica syndrome) [1].

Ligaments of the Anterior Knee

The anterior cruciate ligament (ACL) is one of the most important ligaments in the anterior knee (Table 1.1). Some of its primary functions are preventing anterior translation of the tibia on the femur and preserving normal biomechanics of the knee motion to prevent meniscal damage [2, 5]. It originates between the intercondylar eminences of the tibia, courses posteriorly, and attaches to the posteromedial portion of the lateral femoral condyle

Table 1.1 Anterior structures of the knee

Ligament	Attachment	Function/comment
Anterior cruciate ligament (ACL) Anteromedial bundle (AMB) Posterolateral bundle (PLB)	Origin: between the intercondylar eminences of the tibia Insertion: posteromedial portion of the lateral femoral condyle	– Prevents anterior translation of the tibia on the femur – AMB is tight in knee flexion, lax in extension – PLB is tight in knee extension, lax in knee flexion
Anterior intermeniscal ligaments	Connects both anterior horns of the menisci to tibia	Stabilizes the menisci
Anterior plica	Distal femoral articulation	– Remnant of embryonic development – It can be tissue paper thin but can thicken, scar down, and become contracted (plica syndrome)
Infrapatellar fat pad	Posterior to the patellar tendon	Separates the patellar tendon from the underlying tibia and acts as a cushion. Can be a source of knee pain once fibrotic or impinged (Hoffa syndrome)

(Fig. 1.4). The ACL is innervated by the tibial nerve which provides mechanoreceptors that contribute to its proprioceptive function. It receives vascular supply primarily from the middle genicular arteries [2].

The anterior intermeniscal ligament is another structure within the anterior knee that attaches transversely across the anterior aspects of the convex margins of the medial and lateral menisci [5]. The posterior collateral ligament (PCL) inserts at the posterior intercondylar fossa of the tibia and its primary function is in the posterior joint. The PCL will be discussed in more detail in a later section.

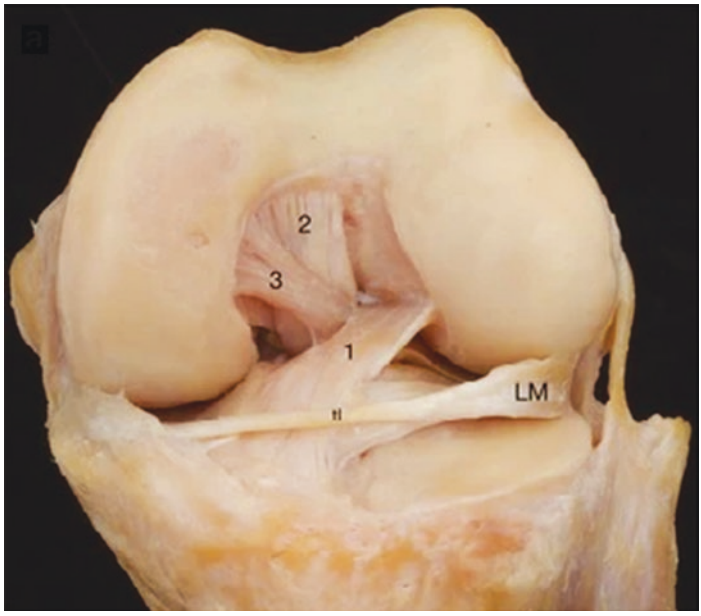

Fig. 1.4 Cadaveric specimen of human left knee joint. (*1*) Anterior cruciate ligament (ACL). (*2*) Posterior cruciate ligament (PCL). (*3*) Anterior meniscofemoral ligament. *LM* lateral meniscus, *tl* transverse ligament [9]

Posterior Knee

Popliteal Fossa and Posterior Knee Structures

It is important to note the anatomy of the popliteal fossa when evaluating posterior knee pain (Fig. 1.5). We will start by discussing its anatomic boundaries (Table 1.2) which include the following: Superomedially, the semimembranosus and semitendinosus muscle, superolaterally, the bicep femoris short and long heads, and inferomedially and inferolaterally, the medial and lateral heads of the gastrocnemius muscle, respectively [5].

The popliteal fossa is composed of nerves (posterior femoral cutaneous, common peroneal, and tibial), vascular structures (small saphenous vein, popliteal artery, and popliteal vein), bursae, lymph nodes, and fat [11]. The posterior cruciate ligament

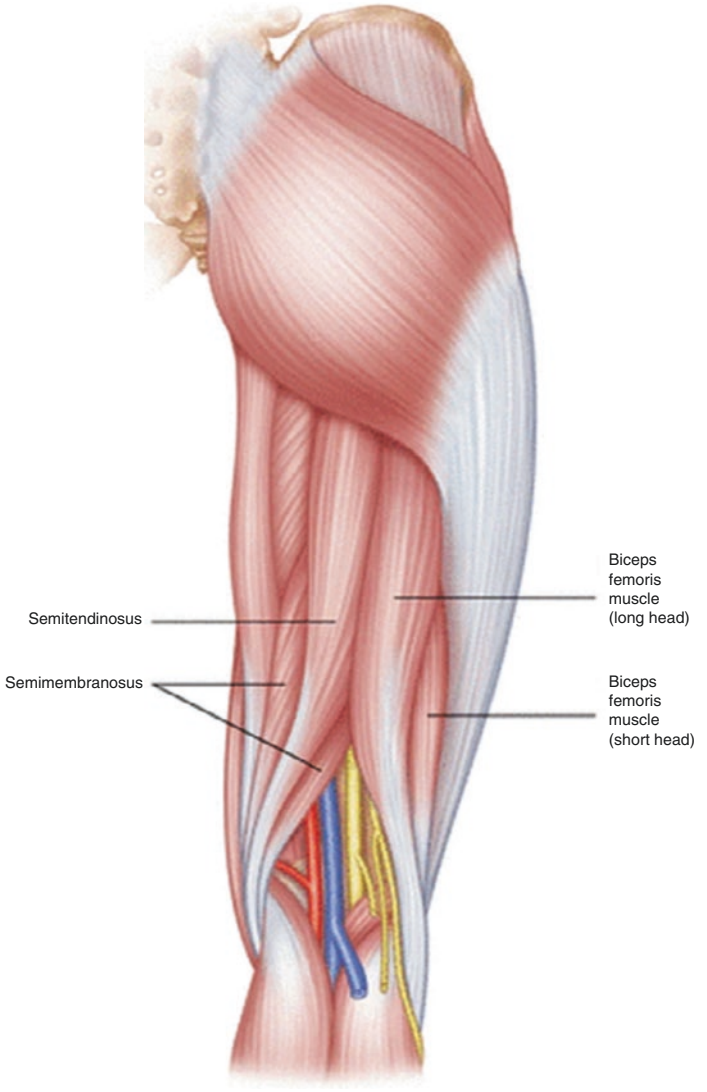

Fig. 1.5 Anatomy of popliteal fossa [10]

Table 1.2 Muscles within the popliteal fossa

Borders	Muscle(s)	Attachment Origin (O) Insertion (I)	Innervation	Vascular supply
Supero-medial	Semi-membranosus Semi-tendinosus	O: Ischial tuberosity I: Posterior medial tibial condyle I: Proximal medial tibia (pes anserinus)	Sciatic N. (tibial branch)	Branches of the profunda femoris, inferior gluteal artery, and popliteal artery
Supero-lateral	Bicep femoris Long head O: Short head	O: Fibular head I: Ischial tuberosity O: Linea aspera I: Tibial (Gerdy's) tubercle	Sciatic N: – Tibial N. branch – Peroneal N. branch	Deep femoral artery
Infero-medial	Medial gastrocnemius	O: Medial femoral condyle I: Calcaneus	Tibial N.	Medial sural artery
Infero-lateral	Lateral gastrocnemius	O: Lateral femoral condyle I: Calcaneus	Tibial N.	Lateral sural artery

(PCL) is the primary ligament of the posterior knee. It originates on the lateral aspect of the medial femoral condyle and inserts at the tibia's posterior intercondylar fossa between its plateaus (Fig. 1.6). The PCL's primary function is to prevent posterior translation of the tibia relative to the femur. Its primary vascular supply is the middle genicular artery. The PCL is innervated by both the tibial and obturator nerves which primarily provide a proprioceptive function. Another important set of ligaments are the ligaments of Humphrey and Wrisberg, which serve as secondary stabilizers to posterior translation. They originate from the

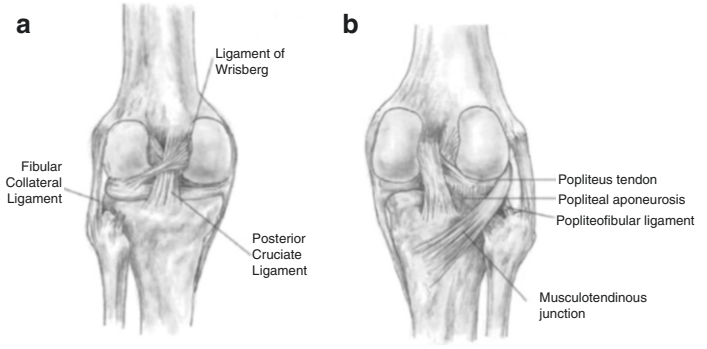

Fig. 1.6 (**a**) Left knee posterior view of PCL with overlying ligament of Wrisberg. (**b**) Right knee posterior view of PCL with other surrounding popliteal structures identified [12]

Table 1.3 Posterior knee

Ligaments	Attachment	Comments
Posterior cruciate ligament (PCL) Anterior bundle Posterior bundle	Origin: lateral aspect of the medial femoral condyle Insertion: tibia's posterior intercondylar fossa between its plateaus	– Prevent posterior translation of the tibia relative to the femur – Secondary stabilizer to prevent external rotation to the tibia and excessive varus and valgus angulation of the knee – Tight in knee flexion, lax in extension – Tight in knee extension, lax in flexion
Meniscofemoral ligament Ligament of Humphrey Ligament of Wrisberg	Origin: posterior horn of the lateral meniscus Insertion: medial femoral condyle Humphrey: Anterior to PCL Wrisberg: Posterior to PCL	– Secondary stabilizers to posterior translation – Contributes to PCL function and stabilizes menisci

posterior horn of the lateral meniscus and insert at the medial femoral condyle while surrounding the PCL both anteriorly and posteriorly (Table 1.3).

An additional layer composed of the capsular ligaments provide additional tibiofemoral stability. The medial and lateral midthird and posterior capsular ligaments cup and buttress the entire posterior medial and posterolateral corner of the knee. They essentially prevent any subluxation of the femoral condyle on the tibial plateaus. The posterolateral corner will be discussed in more detail in a later section [5, 11, 13].

Lateral Knee

The lateral knee is composed of three bony structures: the distal femur, proximal tibia, and fibular head. The lateral tibiofemoral joint's convex-on-convex structures contribute to the joint's inherent instability [14]. There are multiple structures that contribute to the static and dynamic stability of the lateral knee. The three primary static stabilizers include the fibular collateral ligament (FCL) also known as the lateral collateral ligament (LCL), popliteofibular ligament (PFL), and the popliteus tendon (PLT) [14] (Fig. 1.7). These structures compose the posterolateral corner (Table 1.4), which prevent varus stress, external rotation, internal rotation, and posterior tibial translation [5]. Injury to any or all of these structures may subsequently result in residual instability of the knee. Posterolateral corner injuries are commonly associated with ACL or PCL tears, with only 28% of all PLC injuries occurring in isolation [15].

The iliotibial band is a broad band of fascial structure that connects the pelvis to the tibia and covers the lateral thigh [14]. It primarily inserts at the lateral (Gerdy) tubercle, which is the lateral condyle of the proximal tibia. It is important to mention that portions of the anterior arm of the short head of the bicep femoris also insert at Gerdy's tubercle. The anterior arm has anatomic relevance as it is responsible for lateral avulsion fractures (Segond fracture or lateral capsular sign commonly associated with ACL injury). Also coursing through the posterolateral corner are its neurovascular structures, the common peroneal nerve, and the lateral inferior genicular artery [14].

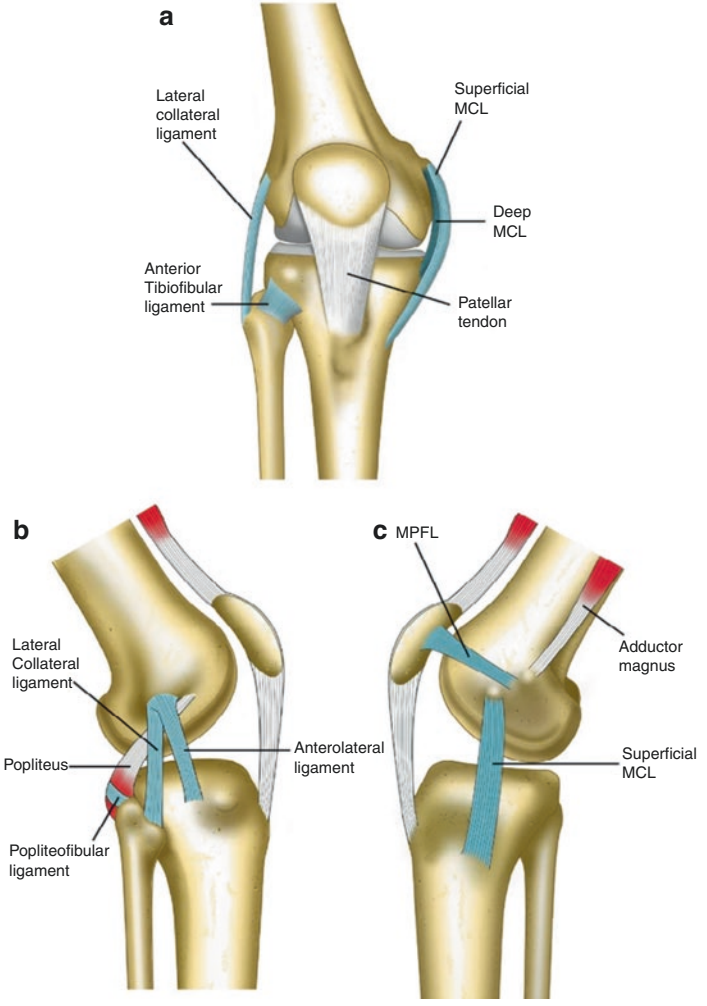

Fig. 1.7 Extra-articular ligaments of the knee. (**a**) Anterior, (**b**) lateral, (**c**) medial view [3]

Table 1.4 The posterolateral corner

Structure	Attachment	Neurovascular supply	Function/ comment
Fibular/lateral collateral ligament	Origin: lateral epicondyle proximal Insertion: medial fibular head	Innervation: tibial nerve Vascular: branches of the superior and inferior lateral genicular arteries [17]	Primary restraint to varus stress, also resists external rotation
Popliteus muscle and tendon	Inserts anterior and distal to the LCL origin	Innervation: tibial nerve Vascular: inferior medial and lateral genicular arteries	Initiating the flexion of the fully extended ("locked") knee Resist tibia external rotation, varus and posterior translation
Popliteofibular ligament	Inserts at popliteus musculotendinous junction to fibula head	Innervation: tibial nerve Vascular: inferior medial and lateral genicular arteries	Primary static restraint to external rotation

The deepest layer of the lateral knee is composed of the mid-third capsular ligament, LCL, popliteus tendon, among other structures (Fig. 1.8). The lateral patellofemoral ligaments and lateral patellar retinaculum make up the middle layer.

The iliotibial band and the bicep femoris make up the most superficial layer of the lateral compartment of the knee. The LCL originates at the lateral epicondyle proximally and posterior to the PLT's attachment [5]. The popliteus muscle courses obliquely in the posterior and inferior direction and wraps around the posterior capsule in the medial direction. The LCL has anterior and posterior divisions that attach to the tip of the fibular styloid process.

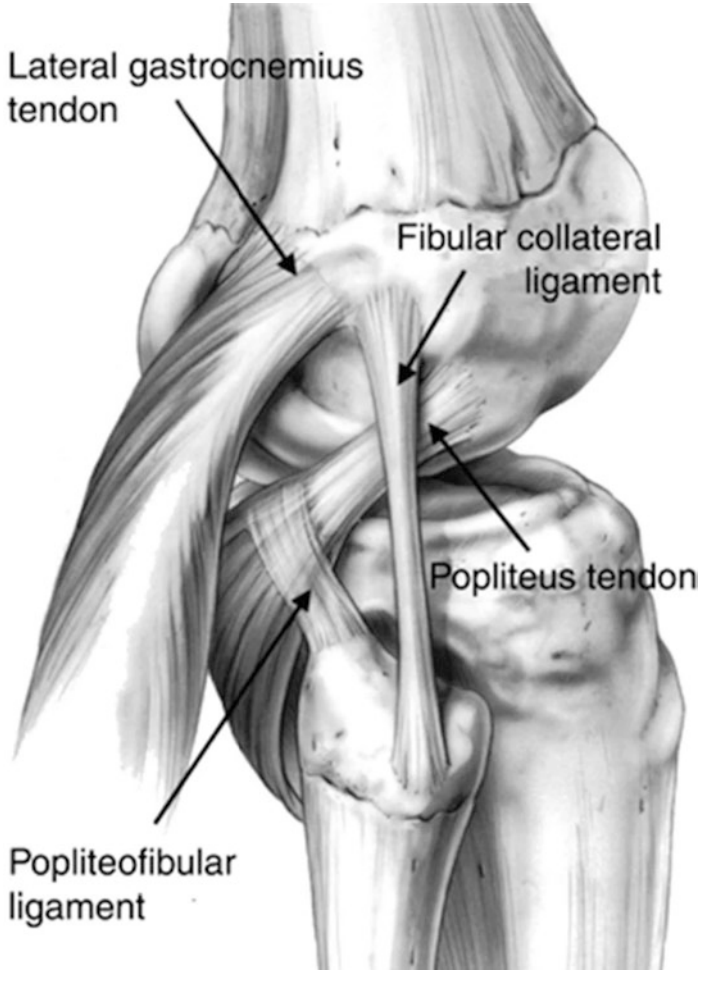

Fig. 1.8 Deepest layer of lateral knee [16]

Medial Knee

Bony Anatomy

In the medial view of the knee, we find the medial tibiofemoral joint. The medial epicondyle's concave articular surface is the most anterior and distal osseous prominence over the medial aspect of the convex medial femoral condyle [18]. There are two important bony prominences of anatomical significance: the medial supracondylar line and the adductor tubercle. The medial supracondylar line is a distal continuation of the linea aspera. It ends below the summit of the medial condyle, in a small tubercle, the adductor tubercle. The adductor tubercle is the common attachment site for the abductor magnus tendon (AMT), medial patellofemoral ligament (MPFL), medial gastrocnemius tendon (MGT), and posterior oblique ligament (POL) [5].

Connective Tissue

The ligaments of the medial knee are the superficial medial collateral ligament (sMCL), deep medial collateral ligament (dMCL), and POL. The MCL has both a deep and superficial layer to help maintain medial knee stability. The sMCL or tibial collateral ligament has two attachment sites: proximally at the medial femoral condyle and distally at the tibial joint line. The dMCL has both a meniscotibial and meniscofemoral component which run parallel and deep to the sMCL [5, 18].

The MCL is innervated by the medial articular nerve, a branch of the saphenous nerve and the branches of the superior and inferior genicular arteries provide its vascular supply [19]. The POL attaches from the adductor tubercle to multiple sites: the posterior tibia, posterior horn of the medial meniscus, and the posterior capsule [11] (Table 1.5).

Other Important Structures

The semimembranosus, a more superficial structure medially, is the dynamic motor structure of the medial knee. In a counterclockwise approach, the capsular, anterior/tibial, direct, inferior,

Table 1.5 Medial knee structures

Ligament	Attachment Origin (O) Insertion (I)	Function/comment
Medial collateral ligament (MCL) Superficial MCL (sMCL) Deep MCL (dMCL): Meniscotibial and meniscofemoral component that run parallel and deep to sMCL	Origin: medial femoral condyle (deep to pes anserinus) Insertion: tibial joint line O: both tibial and femoral I: medial meniscus and tibia plateau	– Primary restraint to valgus forces, especially at 30° – Secondary stabilizer to anterior translation and internal rotation – Stabilizes the meniscus—also known as the medial capsular ligaments or middle 1/3 capsular ligament
Posterior oblique ligament	O: adductor tubercle to the posterior tibia I: posterior horn of the medial meniscus, and blend into the posterior capsule	– Static stabilizer against valgus forces – Lax in flexion but tightens dynamically due to semimembranosus
Medial patellofemoral ligament (MPFL)	O: medial patella I: medial femoral epicondyle	Functions as an important patellar stabilizer as it assists in sustaining the patella within the trochlear groove

and posterior arms cover the medial and posterior portions of the joint and merge together to form the semimembranosus tendon [11] (Fig. 1.9).

Another important structure is the pes anserine. It is located near the anteromedial aspect of the proximal tibia. The pes anserine is formed by the tendons of the sartorius, gracilis, and semitendinosus. These tendons essentially form the roof of the pes anserine bursa. The pes anserine is a common site of anterior knee pain due to secondary bursopathy in the setting of friction [18].

The MPFL functions as an important patellar stabilizer as it assists in sustaining the patella within the trochlear groove. The vastus medialis obliquus (VMO) muscle is the main origin of the abductor magnus tendon (AMT).

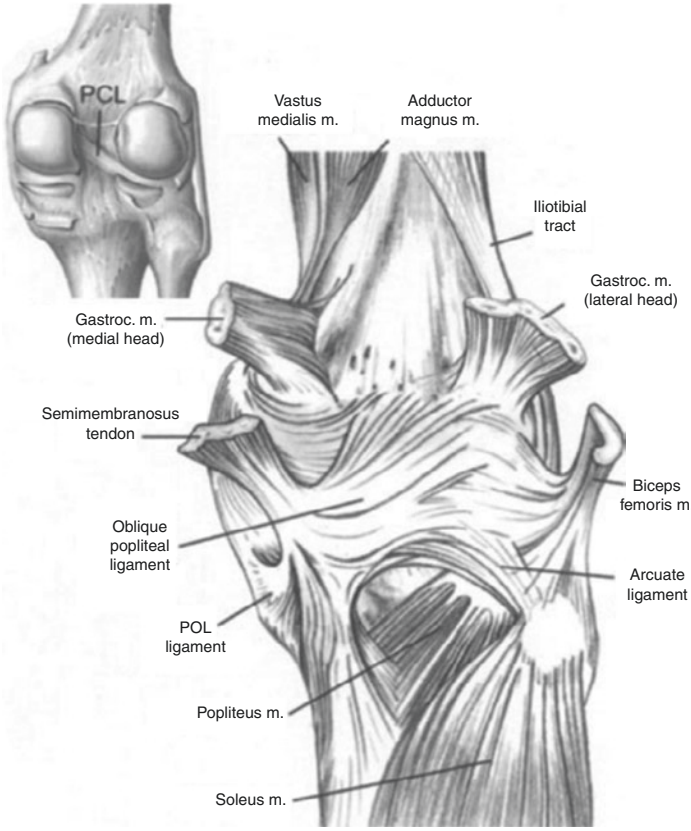

Fig. 1.9 Semimembranosus tendon [12]

The VMO attaches along the proximal edge of the MPFL and the AMT. The VMO applies a medially directed force that has particular importance in relation to the patella, since any injury to this structure disrupts the medial stabilization of the patella [18].

Neurovascular Structures

The saphenous nerve division of the femoral nerve branches off between the gracilis and semitendinosus tendons. It pierces through the sartorius muscle then curves into a close horizontal

path medial to the patellar tendon. The medial inferior genicular arteries branch off the main femoral artery. They supply the upper end of the tibia and the knee joint, anastomosing with the lateral inferior and medial superior genicular arteries which supply structures of the lateral and medial knee, respectively [18].

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Ligament Injuries

2

Monica Gibilisco, Prathusha Maduri, and Richard G. Chang

Anterior Cruciate Ligament

Pathology

The anterior cruciate ligament (ACL) is the most common knee ligament injured in sports [1].

Injuries to the ACL can range from mild, such as a tear/sprain, to severe, when the ligament is completely torn. The ACL courses from its origin at the medial wall of the lateral femoral condyle to its insertion into the middle of the intercondylar area of the tibia. Its function is to resist anterior tibial translation and rotational loads. The ACL has two bundles, an anteromedial bundle, which is tight in flexion, and a posterolateral bundle, which is more tighter in extension. The anteromedial bundle is more responsible for restraining anterior tibial translation, while the posterolateral bundle is more responsible for rotational stability [1].

M. Gibilisco · P. Maduri (✉)
Physical Medicine and Rehabilitation, Icahn School of Medicine at Mount Sinai, New York, NY, USA

R. G. Chang
Department of Rehabilitation and Human Performance, Icahn School of Medicine at Mount Sinai, New York, NY, USA
e-mail: richard.chang@mountsinai.org

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The incidence of ACL injuries in the USA is 1 in 3500 people and is the most injured knee ligament. Some studies suggest that ACL injuries may occur more frequently in women due to weaker hamstrings and quadriceps overuse when decelerating, which stresses the ACL, or due to estrogenic effects causing increased flexibility of tissues. However, the literature for the female demographic is controversial.

ACL injuries can occur through noncontact or contact mechanisms. A noncontact pivoting injury occurs when the tibia translates anteriorly while the knee is in slight flexion and in a valgus position, which may occur in skiers, soccer players, and basketball players. In contrast, contact injuries occur with a direct trauma to the lateral aspect of the knee with the highest risk sport being football [2]. A direct trauma may also cause medial meniscus and medial collateral ligament injury, in addition to ACL injury leading to the commonly known "unhappy triad" [2].

Clinical Presentation

Typically on presentation, a loud pop occurs that can be heard by the individual. Individuals may also report the knee "giving out," signifying knee instability, and will have difficulty bearing weight. Knee effusion or hemarthrosis may occur within the first 2 h. More than 50% of ACL injuries occur with a meniscal tear [1].

Physical Exam

On examination of an acute injury, one will likely see a large effusion and patients will not be able to actively extend their knee. Provocative tests that can be used are the Lachman test, pivot shift test, and the anterior drawer test. The most sensitive exam is Lachman test, with a sensitivity of 95–99% [1].

Lachman test requires the patient to be positioned supine with their injured knee flexed to 20°–30°, and some sources encourage slightly externally rotating the injured leg to relax the iliotibial band (Fig. 2.1). The examiner then uses one hand to stabilize the

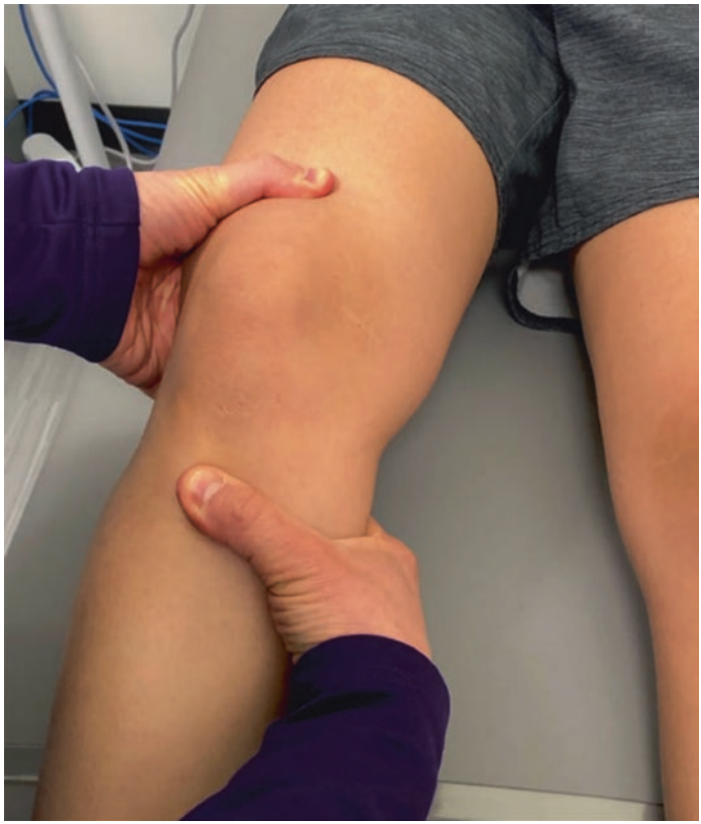

Fig. 2.1 Lachman test [3]

distal femur, and uses their other hand to grasp the proximal tibia. Next, an anterior force is applied to the proximal tibia in an attempt to sublux the tibia forward while keeping the femur stabilized [2]. The test is considered positive if there is excessive anterior translation of the proximal tibia greater than the uninjured side and lack of a firm endpoint. ACL injuries are graded based on the amount of anterior tibial translation compared to uninjured side (Table 2.1).

The pivot shift is not as sensitive and requires the patient to relax as much as possible. However, it is very specific (98–100%) and pathognomonic when performed under anesthesia (Fig. 2.2)

Table 2.1 Grading ACL injuries: degree of laxity determined by amount of tibial translation compared to uninjured side [1]

Grade 1 (mild)	1–5 mm displacement
Grade 2 (moderate)	6–10 mm displacement
Grade 3 (severe)	>10 mm displacement

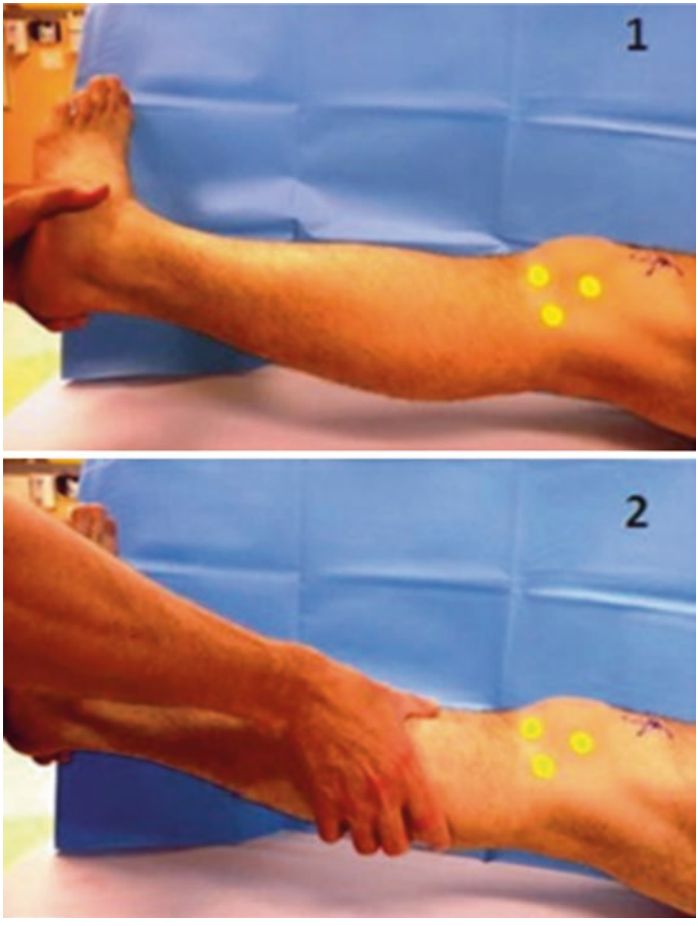

Fig. 2.2 Pivot shift test [4]

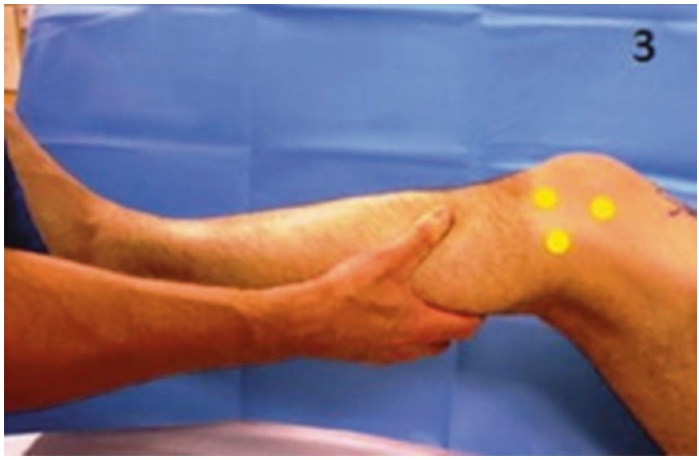

Fig. 2.2 (continued)

[1]. The examiner grasps the heel of the injured leg with the examiners opposite hand placed laterally on the proximal tibia just distal to the knee. The examiner then applies a valgus stress and an axial load while internally rotating the tibia as the knee is moved into flexion from a fully extended position. A positive test is indicated by subluxation of the tibia while the femur rotates externally followed by a reduction of the tibia at 20–30 degrees of flexion.

The anterior drawer test can be used, but lacks the sensitivity and specificity of the Lachman test (Fig. 2.3). The patient lies supine with their hips flexed to 45°, and the injured knee flexed to 90°. Similar to the Lachman test, the examiner then uses one hand to stabilize the distal femur, and uses their other hand to grasp the proximal tibia. Next, an anterior force is applied to the proximal tibia in an attempt to sublux the tibia forward while keeping the femur stabilized. The test is considered positive if there is excessive anterior translation relative to the contralateral side.

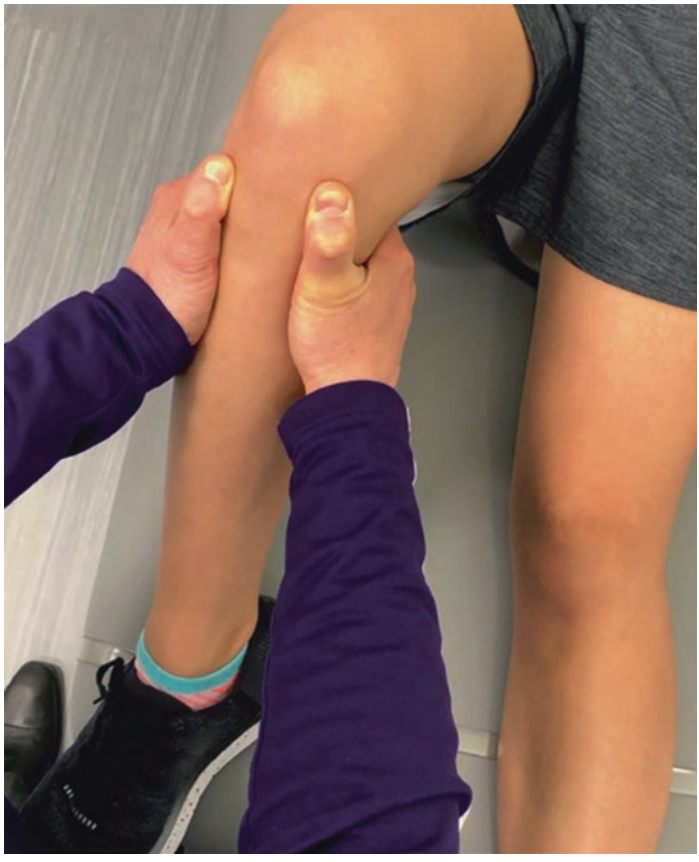

Fig. 2.3 Anterior Drawer test [3]

Diagnostic Studies

Radiographs are typically normal in ACL injuries. Radiographs can be used to evaluate for effusions and bony injuries, especially a Segond fracture. A Segond fracture is a capsular avulsion fracture of the lateral tibial plateau and it is associated with ACL tears 75–100% of the time (Fig. 2.4) [1].

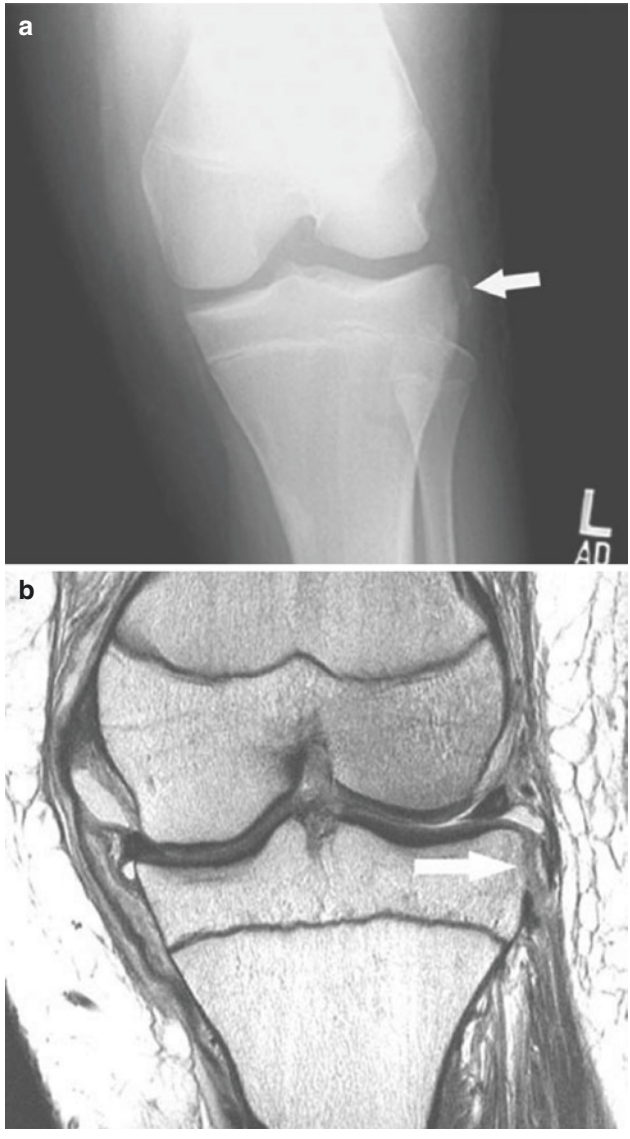

Fig. 2.4 (**a**) Knee X-ray anterior–posterior (AP) view showing a Segond fracture in a 15-year-old girl. (**b**) MRI fat sat AP coronal view showing Segond fracture associated with ACL tear [5]

MRIs will typically confirm clinical diagnosis of ACL rupture and evaluate for concomitant pathology with 86% sensitivity and 95% specificity. Preferred views include T2 sagittal MRIs or STIR sequences, which will show increased signal and edema of the ACL, fiber discontinuity, and change in the ACL course seen as alteration of Blumensaat's line. Blumensaat's line is the line drawn along the roof of the intercondylar notch of the femur. This line is helpful in evaluating for ACL injury as a normal ACL-Blumensaat line angle is less than 15°.

If the angle is greater than 15°, this typically indicates an ACL tear. Research has found that the sensitivity and specificity of the ACL-Blumensaat line angle to detect ACL injury is about 95% [6]. Arthroscopic evaluation is the gold standard in diagnosing ACL injury with 92–100% sensitivity and 95–100% specificity [2].

In one study, dynamic ultrasound was used to diagnose ACL tears with a sensitivity of 52% for partial tears and 79% for complete tears. Specificity was 85% for partial tears and 89% for complete tears. Ultrasound can detect complete ACL rupture but it is not currently standard practice. Overall, diagnostic musculoskeletal ultrasound can be completed at point of injury and further care [7].

Treatment

The acute treatment of an ACL injury is rest, ice, compression, and use of a knee immobilizer or hinged knee brace to aid with pain and stability. Next steps include non-operative rehabilitation or ACL reconstruction/repair.

The ACL has a poor capacity to heal so reconstruction is preferred to repair. The ACL cannot form a fibrin-platelet clot to initiate tissue healing because clot formation is most likely inhibited by factors in the surrounding synovial fluid. Hence, the location of the ACL puts it at a disadvantage to heal when injured compared to extra-articular ligaments [8]. People who are typically recommended to undergo ACL reconstruction are active athletes/patients, have other ligamentous/repairable meniscal injuries,

and/or are experience knee instability. ACL reconstruction can be performed with patellar tendon autografts (native), quadricep tendon autografts or hamstring (semitendinosus or gracilis) autografts or allografts (cadaveric). Advantages with autografts include faster healing, lower risk of re-injury and infection. However, disadvantages include complications at the harvest site, longer surgical procedure times, and constraints around tissue selection such as size and harvest location. Cadaveric allografts are often taken from tendinous structures such as the Achilles, patellar, hamstring, and posterior tibialis tendons. However, these are preferentially used in middle aged athletes engaging in lower impact sports. While allografts perform similarly to autografts, they carry higher rates of re-injury, risk of disease transmission, immunologic reaction, and slower remodeling. Still, allografts may be the preferred choice due to decreased surgical time and less limitations on size and harvest site morbidity. Ultimately, the decision is made after weighing risks and benefits for each individual patient [9].

ACL repair was previously abandoned due to high failure rates in people of all ages, but there has been some increase in repair recently, in certain populations, especially in cases involving proximal ACL avulsions which results in separation of the ACL from the bone. There have been studies where ACL repair in this population can lead to good results, [10] but overall reconstruction is still the preferred surgical method. Current studies suggest that even modern ACL repair techniques have a failure rate of 5–10 times higher than that of ACL reconstruction [11].

If a patient decides for operative management, it is important to begin immediate weight bearing postoperatively and to be involved in early rehabilitation. There is no true standardized difference in rehabilitation protocol for different types of grafts. However, one important precaution is the avoidance of rotational stresses to the knee before initial graft incorporation to bone, which occurs approximately at 4 weeks for bone-tendon-bone grafts and 8–12 weeks for hamstring allografts [12].

There are specific modalities, muscles, and exercise types that should be targeted after a graft is placed. In the acute phase

(1–2 weeks), modalities include cryotherapy and electrical stimulation, while exercises include active assisted flexion, passive extension, isometric quadriceps contractions (between 90° and 45°), dynamic hamstring exercises, and straight leg raises.

In the recovery phase (2+ weeks), patients can start using modalities such as superficial heat, pulsed ultrasound, and electrical stimulation. Exercises are advanced to active flexion and extension training, dynamic quadriceps exercises (between 90° and 30°), and hamstring strengthening [12, 13]. These patients should also focus on closed chain exercises in early rehabilitation (2+ weeks after injury), such as leg-presses or squats. Closed chain exercises are when the hand or foot is fixed in place, whereas in open chain exercises the hand or foot is free to move. Post-ACL reconstruction patients should specifically avoid open chain quadriceps strengthening exercises and should also avoid isokinetic quadriceps strengthening from 15° to 30° during their early rehabilitation [14]. These exercises can put excess stress on the graft. Aquatic exercises, bicycling, swimming, and the elliptical trainer can also be used in the recovery phase. In the recovery phase, if the patient has full flexion and extension, symmetric quadriceps and hamstring strength, and symptom-free progression in a sports-specific program, they are considered to be advanced to the functional phase. The functional phase is the final phase and focuses on general flexibility training, strengthening, power and endurance, neuromuscular control, and proprioceptive training with a return to sport-specific participation [12].

Non-operative management consists of a physical therapy rehabilitation program. People who are considered for non-operative treatment include those who have a more sedentary lifestyle, are recreational athletes, and those without significant knee instability. Physical therapy will focus on range of motion and strengthening of the quadriceps, hamstrings, hip abductors, and core muscles. The rehabilitation protocol is slightly different from postoperative rehabilitation because there is no recommended numerical degree of movement that should be avoided, as there is no graft placement. There is more focus on range of motion in the recovery phase, otherwise the same protocol is used and the same modalities mentioned earlier can be used in non-operative management [12].

Steroid injections as a treatment for ACL injury is unclear, however, most recent literature advises against it. Dexamethasone, a steroid, was found to increase the calcification of ACL cells and caused ACL degeneration through endoplasmic reticulum stress. This suggests that long-term treatment with dexamethasone may cause adverse effects on ACL tissue and increases the risk of long-term rupture [15]. Another animal study in sheep looked at only partial ACL injuries and found that multiple repeated injections of glucocorticoids led to significant proteoglycan loss in the methylprednisolone treated knees. Proteoglycan is a component of a molecule that typically provides hydration and enables tissue to withstand compressional forces. Hence, steroid use in ACL injury is not standard of care [16].

There have been several clinical trials and case reports that demonstrate the effectiveness of prolotherapy injections for ACL injuries. While it is not standard of care, prolotherapy is defined as injection that causes growth of normal cells or tissues. Prolotherapy with intermittent dextrose injections in patients with symptomatic ACL laxity have resulted in clinically and statistically significant improvement in ACL laxity, pain, swelling, and knee range of motion. One particular study used an intraarticular injection consisting of 6–9 cc of 10% dextrose injected at intervals of 0, 2, 4, 6, and 10 months, and then injected with 6 cc of 25% dextrose at 12 months. Afterward, depending on patient preference, injection of either 10% or 25% dextrose was completed every 2–4 months through 36 months [17].

Future treatment directions may include orthobiologic techniques using platelet-rich-plasma, bio-scaffolds with tissue-engineered collagen, and mesenchymal stem cells. Animal studies show encouraging results when these regenerative medicine techniques are used for ligament healing and some preliminary literature suggests some role intra-operatively, but further research must be done to establish clinical impact, operative techniques, and validity [18].

Return to Activities

The postoperative rehabilitation program lasts 6–9 months prior to full return to play [1]. There is no widely accepted specific time

to return to playing sports. It will depend on the patient's restored mobility, flexibility, strength, function, and ability to pass a series of tests that replicate specific sport activities, such as hopping and jumping on one and both legs. Psychological readiness plays a large role in returning to play. The patient must be mentally prepared and the timing of return should not be ignored [9].

Effective ACL injury prevention is a topic of research because once an athlete sustains an ACL injury and undergoes reconstructive surgery, that athlete has an increased risk of injury in both the affected extremity and the contralateral extremity [13]. Mandelbaum and colleagues started the "Prevent Injury Enhance Performance injury prevention program" (PEP). The PEP program focuses on warm-up exercises, agility exercises, plyometric exercises, and stretching and strengthening to prevent ACL injury. For example, warm-up includes jogging and shuttle runs while strengthening includes lunges, hamstring exercises, and single-leg toe raises. Plyometric exercises focus on landing techniques and knee positioning. The program is designed to be completed in 15–20 min. This study noted an 88% reduction in ACL tears in year one. Year two of the study also found significant decreases in ACL injury through training in the prevention program. This example emphasizes the importance of physical therapy and proper rehabilitation exercises months out of an ACL injury [19].

Posterior Cruciate Ligament

Pathology

The posterior cruciate ligament (PCL) is less commonly injured than the ACL. The role of the PCL is to primarily resist extreme posterior translation of the tibia relative to the femur and to prevent hyperflexion. Its secondary role is to prevent excessive rotation between 90 and 120 degrees of knee flexion [20]. The PCL has two bundles, anterolateral bundle, which is tight in flexion, and posteromedial bundle, which is tight in extension. The PCL originates from the anterolateral medial femoral condyle and inserts along the posterior tibial plateau. When the PCL is injured,

other structures in the posterolateral corner including the lateral collateral ligament, popliteus tendon, and the popliteofibular ligament may be injured as well. The mechanism of a PCL injury is most commonly from an impact to the anterior tibia while the knee is in flexion; for example, in a car accident when an individual's knee hits the dashboard [1]. Another mechanism is from a noncontact hyperflexion of the knee with a plantar-flexed foot. This specific mechanism is the most common cause of isolated PCL injuries, without combined ligamentous damage [21]. A PCL injury occurs less often from knee hyperextension, but is also possible [1].

Clinical Presentation

The patient will most likely present with minimal swelling, nonspecific posterior knee pain, and inability to bear weight [1]. A common complaint is apprehension while going down stairs because of a sense of unsteadiness. However, the common complaint of buckling, such as in an ACL injury, is rarely seen in an isolated PCL injury. Thus, if a patient complains of instability, typically other ligaments are also involved representing a combined injury [7].

Physical Exam

Provocative tests for a PCL injury include the posterior drawer test, posterior sag sign, and quadriceps activation test.

The most accurate test for determining a PCL tear is the posterior drawer test at 90°. With the knee at 90 degrees of flexion, a force directed posteriorly is applied to the proximal tibia and posterior tibial translation is then measured. Tears are divided into grade I, grade II and grade III based on the amount of posterior tibial translation during this test (Fig. 2.5, Table 2.2).

The posterior sag sign is tested with the patient lying supine with the hips and knees flexed at 90°, while the physician supports the ankles, and observes for any posterior shift of the tibia. One

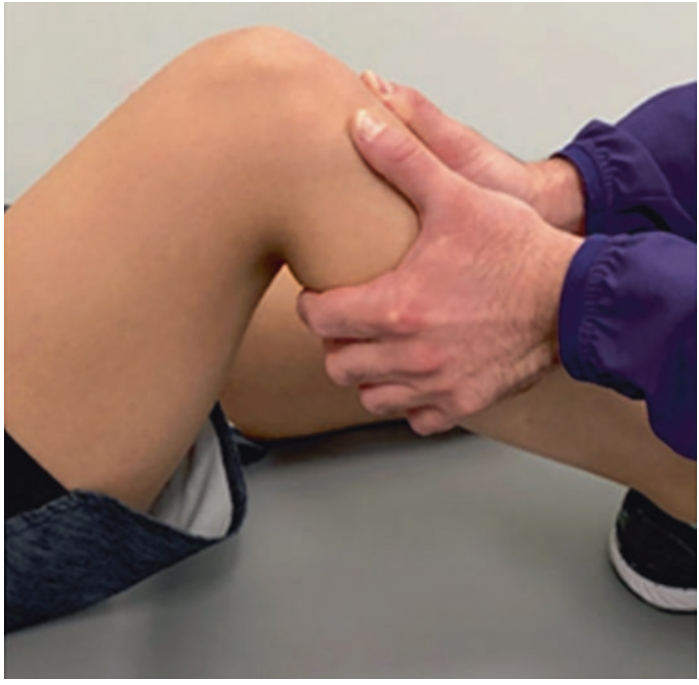

Fig. 2.5 Posterior Drawer test [3]

Table 2.2 Grading PCL tears based on amount of posterior tibial translation compared to uninjured side [21]

Grade I (mild)	1–5 mm partial PCL tear
Grade II (moderate)	6–10 mm complete PCL tear
Grade III (severe)	>10 mm complete PCL tear + capsuloligamentous injury [may include ACL and/or PLC (posterolateral corner) injury]

important point to note is that a quadriceps spasm may cause a false-negative in this test (Fig. 2.6).

The quadriceps activation test has a specificity of 97–100%. It is performed with the patient lying supine, knee flexed to 90° and the foot stabilized by a physician. The patient is asked to slowly

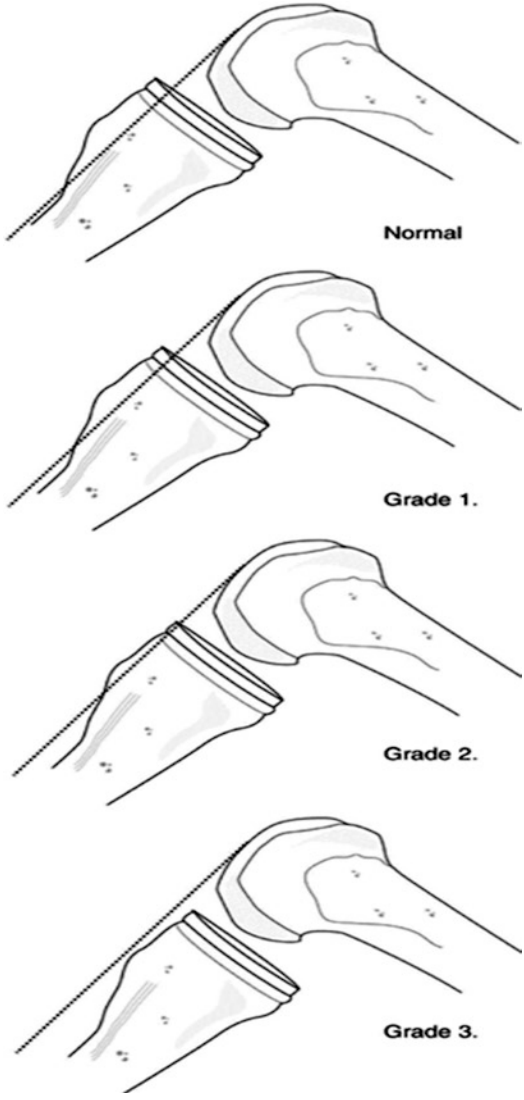

Fig. 2.6 Posterior Sag Sign. Grade 1: Displacement of front of tibia but still in front of anterior aspect of femur. Grade 2: Displacement of front of tibia in line with anterior aspect of femur. Grade 3: Anterior aspect of tibia behind anterior aspect of femur [22]

Fig. 2.7 Quadriceps activation test [23]

slide his or her foot down the table. In a PCL injury, the quadriceps contraction results in an anterior shift of the tibia more than 2 mm relative to the femur (Fig. 2.7) [22, 23].

The posterolateral corner (PLC) should also always be tested when suspecting a PCL injury. The posterolateral corner itself consists of three ligaments, the anterolateral ligament, the popliteofibular ligament, and the fabellofibular ligament. The dial test is a great way to differentiate PLC and PCL injury together or just isolated PLC injury.

The dial test is done with the patient prone, both knees flexed first to 30°, which best isolates the PLC, and then flexed to 90°, with external rotation applied to the tibias at each position. A 10° increase in external rotation, when compared to the contralateral side, at 30 degrees of knee flexion indicates an isolated PLC injury. If the test is positive at also 90 degrees of knee flexion, then there is a concomitant injury to the PCL (Fig. 2.8) [2].

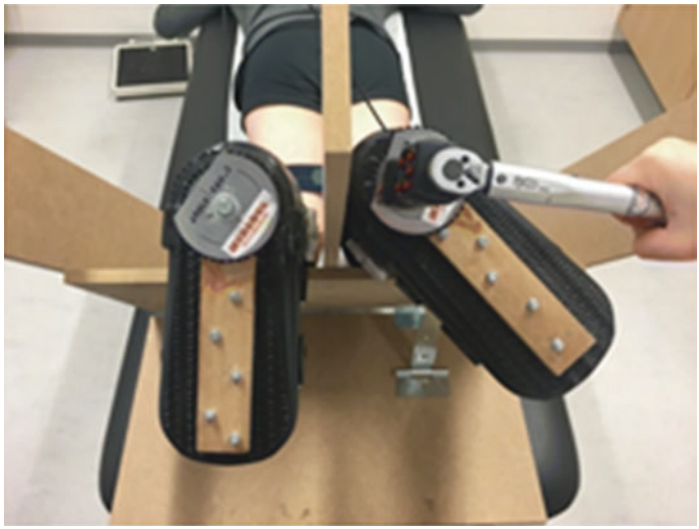

Fig. 2.8 Dial test to differentiate PLC and PCL injury [24]

Diagnostic Studies

Radiographs are being used more often to evaluate PCL injuries, but are still not the gold standard for diagnosis. In an AP and supine lateral view, one can evaluate for associated avulsion fractures of the tibial insertion. The lateral stress view of an X-ray has also become increasingly used (Fig. 2.9). A lateral stress X-ray is when one applies stress to the anterior tibia with the knee flexed to 70°. If there is asymmetric posterior displacement of the tibia, this indicates a PCL injury. Some clinicians consider stress-radiography to be the best test to quantify posterior tibial displacement in PCL insufficiency [25]. MRIs will typically confirm clinical diagnosis of PCL rupture and evaluate for other ligamentous injuries. Arthroscopic evaluation is the gold standard for diagnosis [1].

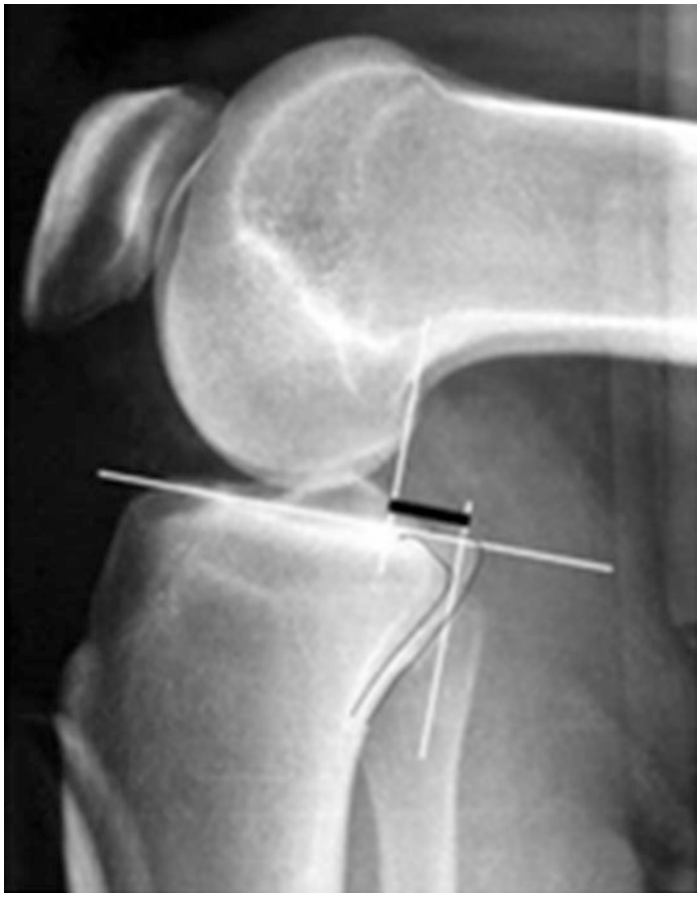

Fig. 2.9 Lateral stress view X-ray demonstrating PCL injury showing posterior displacement of tibia [25]

Treatment

The acute treatment of a PCL injury would include rest, ice, and compression. One can consider a knee brace in full extension or use of crutches if there is significant functional limitation and instability [1]. Next steps of treatment include non-operative care

with rehabilitation, or less commonly, operative management with PCL reconstruction. In general, surgical treatment is only recommended if persistent instability is present and/or other concurrent meniscal/ligamentous injuries are present.

The research on steroid or prolotherapy injection use on PCL injury is minimal. One case report in a 24-year-old male soccer player who presented with a 7-year history of left posterior knee instability, grade 1 posterior drawer and grade 1 posterior sag signs, did undergo one experimental injection of prolotherapy with dextrose hyperosmolar solution. He was injected with a mixture of 1 mL of 50% dextrose, 2 mL of sterile water, and 2 mL of 1% lidocaine was injected. The patient's subjective feeling of looseness and instability resolved by 7 weeks [26].

Studies on the application of orthobiologics and regenerative medicine for PCL injury are lacking. There have been more studies with the use of regenerative techniques for the ACL, as mentioned previously, and in theory these methods could be developed and extended to other ligaments in the knee. One case series of 13 soccer players with isolated partial PCL injuries in Spain used a series of three once-weekly ultrasound-guided white blood cell-poor PRP injections to the PCL, ligament sheath, and popliteal fossa with the goal to enhance the healing of a grade I or II injured PCL. Patients also used a specific PCL brace and participated in an early rehabilitation program. In this study, the treatment regimen was effective to achieve adequate MRI-based healing in 100% of patients and a return to play in 90% of soccer players. Further well-designed studies are needed to appropriately assess this potential treatment technique [27].

The PCL suffers from partial injury more commonly than the ACL, so the grading is particularly important in terms of treatment. Another important factor to consider in PCL injury treatment is other concurrent ligamentous injury. An isolated acute PCL injury, either Grade I or Grade II, will only need 4–6 weeks of limited activity and rehabilitation for treatment. Rehabilitation exercises focus on knee extensor and quadriceps strengthening.

An isolated acute Grade III PCL injury will need 4 weeks in extension bracing to keep the knee in full extension to prevent posterior subluxation of the tibia. If the patient is a young athlete,

surgery can also be considered in this population. If the PCL is combined with injuries of the LCL, MCL or PLC, there should be PCL reconstruction within 2 weeks. PCL reconstruction options include tibial inlay or transtibial methods. Grafts include autograft or allograft. Allografts are typically used as there are multiple graft choices available, such as the Achilles, patellar, hamstring, or anterior tibialis tendons. The transtibial technique is when the graft passes proximally and posteriorly through the tibia and makes a 90° turn around the tibial tunnel before entering the knee joint. This 90° bend in the graft has been shown to create increased internal tendon pressures and possibly lead to graft elongation or even failure. The tibial inlay technique differs because there is arthroscopic placement of the femoral tunnel and the open creation of a bone trough in the posterior tibia. The benefit of this procedure is that the graft is secured to the anatomic tibial attachment site of the PCL, thus avoiding the 90° curve associated with the transtibial tunnel. Controversy continues to exist in PCL reconstruction regarding the optimal location of tibial fixation, ideal placement of the femoral tunnel, number of graft bundles, and appropriate graft tension [28].

Postoperatively the patient should be partially weight bearing with a hinged knee brace locked in extension for 2–4 weeks. Exercises are allowed on the first day after surgery, but should be limited to isometric quadriceps and ankle pump exercises. At 4 weeks, passive range of motion with a physical therapist can be fully performed and active range of motion is not until 8 weeks. Patients should avoid resisted hamstring strengthening exercises in early rehabilitation, such as hamstring curls. This is because the hamstrings can pull on the tibia posteriorly and cause stress on the PCL graft [20].

Return to Activities

Grade I and Grade II tears typically heal quickly and most athletes can return to sports in 4–6 weeks. Return to activity following PCL reconstruction ranges, but is typically between 9–12 months following surgery, and requires sport-specific functional training [20].

Medial Collateral Ligament

Pathology

The incidence of medial collateral ligament (MCL) injuries may be higher than reported since low grade injuries can be missed. The MCL has two components, superficial and deep. The superficial component originates at the medial femoral condyle, inserts at the proximal tibia, and attaches 5–7 cm below the joint line. The deep component is actually contiguous with the medial meniscus and consists of the tibiomeniscal and meniscofemoral ligaments. The primary function of the MCL is to prevent joint gapping during valgus stress [1].

The most common mechanism of injury is when valgus stress is placed on the knee, especially if the knee is in slight external rotation and flexion. Injury to the MCL can be from contact or noncontact injury. A direct contact blow to the lateral knee with a medially directed force will typically result in a high grade ligamentous injury. An example in sports is a football tackle from the side. This contact injury usually causes a rupture of the MCL at its femoral insertion. Noncontact injury is less common than contact injury for the MCL. A noncontact injury can occur when performing a pivoting or cutting maneuver with valgus and external forces, such as during skiing maneuvers. These injuries are more often incomplete.

Clinical Presentation

The clinical presentation of an MCL injury is diffuse pain over the medial knee and joint line. Medial edema will start over the next few hours after injury. The edema may increase and spread to the rest of the knee joint over the next day or two after the original injury. The most common multi-ligamentous knee injury is a combined ACL and MCL injury. After injury, the presence of hemarthrosis on presentation is highly suggestive of an associated ACL–MCL injury. This association is most often with high MCL injuries, injuries

located more superiorly on the MCL. A patient will sometimes also present with a history of a reported "pop" at the time of injury. They may complain of medial joint line pain and report instability during ambulation. Meniscal tears, medial over lateral, are also associated with MCL injury as seen in the "unhappy triad" and this can alter clinical presentation and management as well [29].

Physical Exam

Tenderness along the medial aspect of the knee is common during palpation. Valgus stress testing is the standard physical exam test used to assess the integrity of the MCL (Fig. 2.10). It is performed with the patient supine, and by placing one hand on the outside of the knee palpating the medial joint line, while applying an abducting force at the foot and a valgus force through the knee. If medial gapping is felt compared to the opposite knee, there is MCL injury. Valgus stress testing is typically performed at both 30° and 0° degrees of knee flexion to isolate certain structures. At 30 degrees of knee flexion, the superficial MCL is isolated. Tears are divided into grade I, grade II, and grade III (Table 2.3).

If the test is performed at 0 degrees of knee extension, other structures are included in the valgus stress. When medial laxity is felt with valgus stress at 0 degrees of knee flexion, this indicates combined crucial ligament injury or posteromedial joint capsule injury [9]. Special provocative tests for the ACL, PCL, and medial meniscus should also be performed during the physical exam to evaluate for additional injuries.

Diagnostic Studies

Radiographs are typically normal in MCL injuries but should always be taken after a knee injury to rule out other serious pathologies. Calcifications at the medial femoral insertion site can be seen in X-rays, and are from chronic MCL injury or deficiency. The combination of this calcification and medial knee pain is diagnosed as Pellegrini–Stieda syndrome (Fig. 2.11). When eval-

Fig. 2.10 Valgus stress testing with full knee extension can elicit MCL laxity and injury [3]

Table 2.3 Grading MCL tears based on medial joint line gap compared to uninjured side [29]

Grade I (mild)	1–4 mm gap
Grade II (moderate)	5–9 mm gap
Grade III (severe)	>10 mm gap

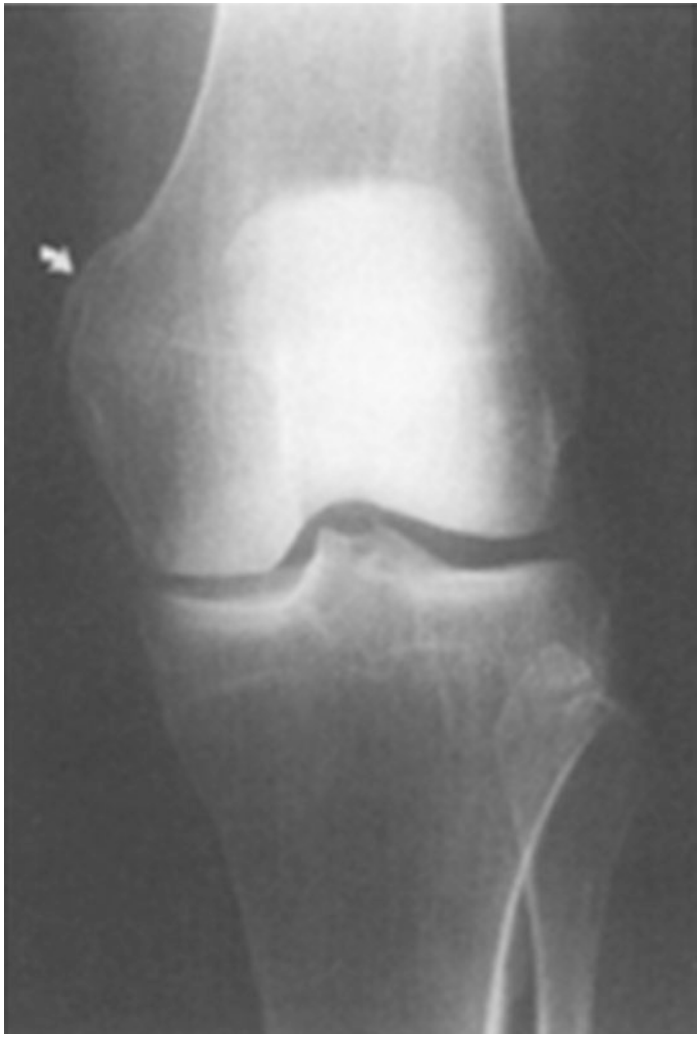

Fig. 2.11 AP radiograph demonstrating a Pellegrini–Stieda lesion: calcification at the femoral attachment site of the medial collateral ligament [30]

uating MCL injury in adolescents, X-rays are important since valgus stress views may reveal gapping through a physeal injury [29]. An MRI is the diagnostic imaging of choice for MCL injuries, especially since an MCL injury frequently involves other ligament injuries as well. Coronal and Axial T1 and T2 MRI images are useful in distinguishing edema within the MCL fibers (Fig. 2.12).

Ultrasound is a quick, low-cost tool to assess MCL tears. Ultrasound can identify MCL pathology by abnormal sonographic appearance of the ligament (examples include heterogeneous fiber quality, hypoechoic fluid, hyperemia, cortical irregularity at

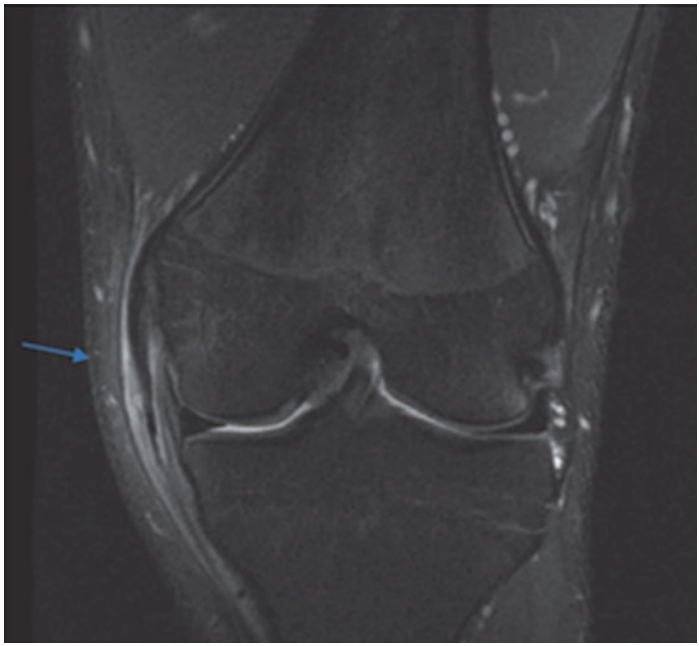

Fig. 2.12 MRI coronal (anterior–posterior view) T2 weighted shows edema (bright signal) within the MCL indicating a Grade III tear [31]

attachment sites) and quantitative assessment of joint gapping with dynamic testing. The role of ultrasound in the assessment of suspected MCL tears is not routinely adapted and MRIs are still needed to confirm findings [32].

Treatment

Treatment of MCL injury is divided into non-operative and operative. Non-operative treatment includes rest, physical therapy, and bracing. Grade I injuries do not need bracing, but rehabilitation is still important. Physical therapy can start with isometric quadriceps contractions and progress to isotonic exercises. Gradually, the range of motion and resistance can be increased. Grade II and Grade III injuries can benefit from bracing. Grade III will only be braced if the knee is stable with valgus stress in full extension and there is no associated cruciate injury. Bracing typically includes a knee immobilizer for comfort and a hinged knee brace for ambulation.

There have been few studies on the use of steroids in MCL injury. A total of 34 patients with chronic pain following grade I or grade II MCL injury were treated with ultrasound-guided injection of local anesthetic and steroid into the deep MCL and were allowed to return to sports immediately. While four were excluded from follow-up and four were lost all patients reported an immediate and sustained resolution of their medial knee pain. At mean follow-up of about 20 months, all were back to their preinjury level of work. Hence, steroid injections in patients with persistent medial joint pain following grade I/II MCL sprain could be useful but not first line [33].

Similar to PCL injury, there have been case reports of prolotherapy aiding in MCL injury recovery. One case study included a rugby player, with a grade 2 MCL injury, who underwent three prolotherapy injections of 15% dextrose, in 1-week intervals. In this case, after prolotherapy and 3 weeks of physical therapy, the patient was pain free, with full knee range of motion [34].

Platelet-rich-plasma (PRP) injections have been used in MCL injury, but the evidence is limited and conflicting. One case study

published was of a 30-year-old professional wrestler with a Grade III MCL injury. Leukocyte-rich (LR) PRP injections combined with rehabilitation was used for his treatment regimen. The patient received a series of three LR-PRP injections evenly spaced 1 week apart with ultrasound guidance. In his case, the use of LR-PRP and early rehabilitation for an isolated MCL tear was beneficial and cut down his total anticipated treatment time. Further research is necessary on PRP but the evidence for rehabilitation is still strong [35].

Operative treatment consists of ligament repair or reconstruction. Repair in grade III injuries is appropriate in the setting of multi-ligamentous knee injury or if there is continued instability despite rehabilitation. Reconstruction is typically performed in chronic MCL injury or if there is loss of adequate tissue for repair.

The use of prophylactic knee bracing to prevent injury or re-injury to the MCL is controversial and studies have mixed results with benefits including stability and kinesthetic reminders to avoid pivoting motions. In some instances, however, disadvantages include weakened surrounding muscles from underuse, which may really impact an athlete depending on a player's specific position in a sport [36].

Return to Activities

Return to activity following an isolated MCL injury can be rapid. A Grade I injury may return to play as early as 7 days, and a Grade II injury may return to play as early as 3–4 weeks.

A Grade III injury may return to play as early as 5–7 weeks, but this varies depending on if reconstruction was done or if other ligaments were also injured [37]. As previously stated, the appropriate time to resume activity after ligamentous injury varies. It is dictated by a combination of physical factors such as activity level, age, type of sport played by athletes and psychological factors such as ability to rehabilitate and cognitive ability.

Lateral Collateral Ligament

Pathology

Isolated lateral or fibular collateral ligament (LCL/FCL) injury is extremely rare, about less than 2% of knee injuries. Many LCL injuries are associated with injury of the posterolateral corner as well. LCL injuries can also be associated with ACL, PCL, or lateral meniscal injuries. The LCL originates at the lateral femoral condyle and inserts on the fibular head (Fig. 2.13). The primary function of the LCL is to prevent joint gapping during varus stress [1].

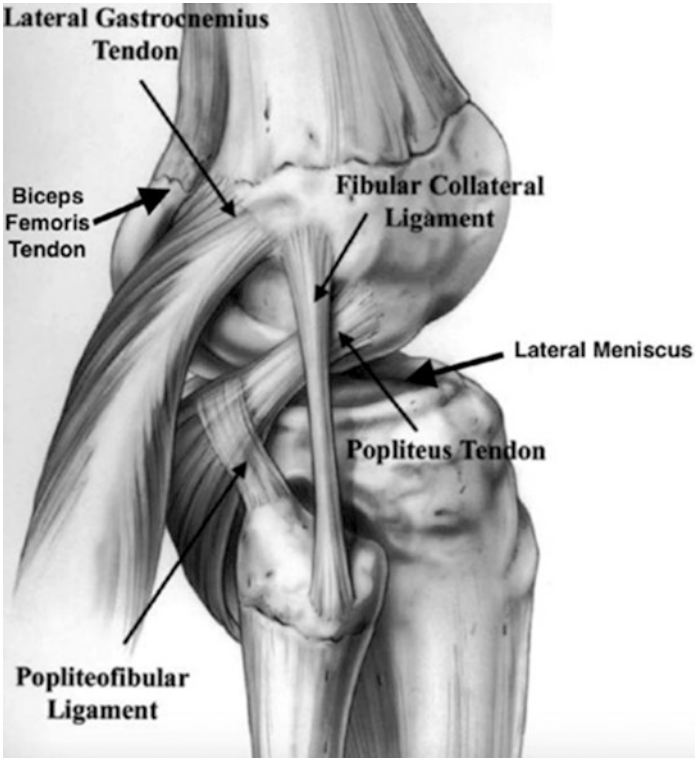

Fig. 2.13 LCL/FCL and the posterolateral corner. The primary static stabilizers of the posterolateral corner include the lateral (fibular) collateral ligament, popliteofibular ligament, and popliteus tendon [38]

LCL injury can be from a traumatic direct blow to the medial side of the knee or excessive varus stress, excessive tibial rotation, or hyperextension. Noncontact injury to the LCL is also possible from a sudden varus moment while the knee is hyperextending. For example, weightlifters with poor lateral knee stability can endure this type of injury [39].

Clinical Presentation

The clinical presentation of a LCL injury is pain diffusely over the lateral knee. Patients will typically complain of a sudden onset of lateral knee pain, swelling, and bruising after the injury. Swelling can happen immediately following the injury or develop up to a few hours after the injury has occurred, spreading along the rest of the knee joint. An example of a contact injury presentation is a football player suffering a blow to the medial aspect of the knee while the foot is planted and knee extended. On presentation, they will report lateral or posterolateral knee pain. Another presentation can be due to a noncontact injury when there is a sudden varus lateral movement while the knee is hyperextending, for example, during a weightlifter's heavy lift. Symptoms that patients typically complain of are instability near full knee extension, difficulty going up and down stairs, difficulty with pivoting, and lateral joint line pain. If additional ligaments are involved in the injury, one may experience additional symptoms such as instability and other areas of pain.

Physical Exam

On initial inspection ecchymosis and lateral joint soft tissue swelling can be observed. Those with concurrent posterolateral corner injury may demonstrate a varus thrust gait (Fig. 2.14). This gait can be observed during foot strike when a gap develops in the lateral aspect of the knee. Because of this, the patient ends up shifting his/her weight during gait to reduce the knee back to normal alignment. On palpation, patients will report tenderness along the lateral knee.

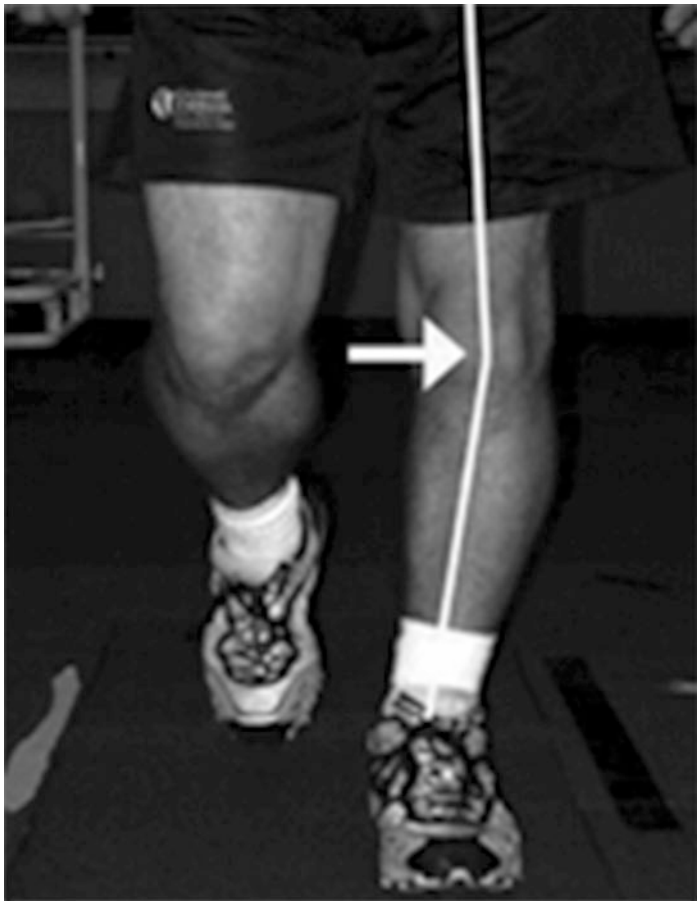

Fig. 2.14 Varus thrust gait [40]

Varus stress testing is the standard physical exam test used for the LCL (Fig. 2.15). It is performed with the patient supine, and the examiner places one hand on the lateral joint line while applying an adducting force at the foot and a varus force through the knee. If lateral gapping is felt compared to the opposite knee, there is likely an LCL injury. LCL injures can be graded based on

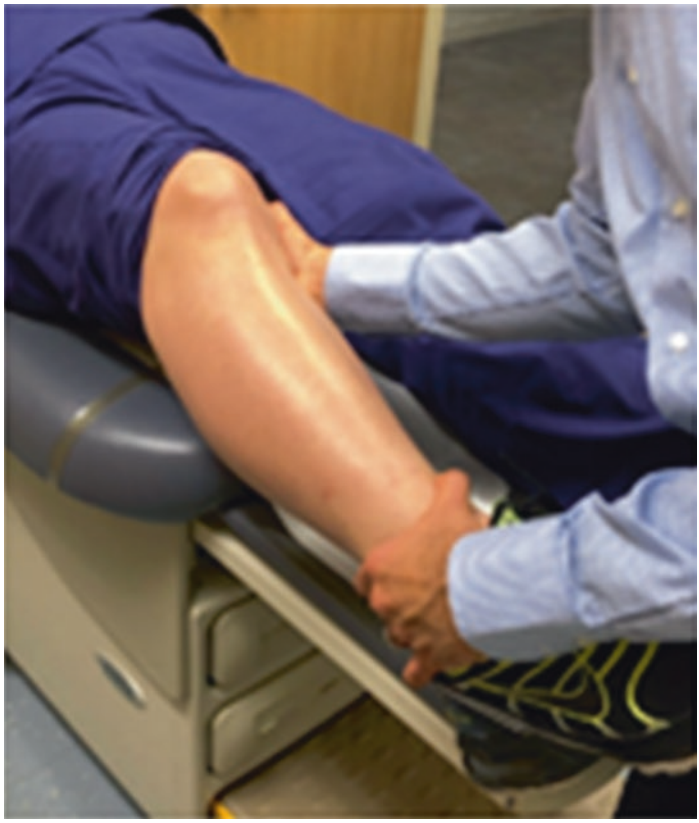

Fig. 2.15 Varus testing with knee in 30 degrees of flexion and palpation of lateral knee [38]

lateral joint line gap during varus stress compared to uninjured side (Table 2.4). Varus stress testing is typically performed at both 30° and 0° degrees of knee flexion to isolate certain structures. Varus instability at 30 degrees of flexion only would indicate an isolated LCL injury, and varus instability at both 0 and 30 degrees of flexion would indicate a combined LCL +/− ACL/PCL injury.

Another provocative test used during physical exam is the dial test (Fig. 2.16) (please see PCL section for further details).

Table 2.4 Grading LCL tears based on lateral joint line gap seen with varus stress compared to uninjured side [38]

Grade I (mild)	0–5 mm gap
Grade II (moderate)	Partial tear 6–10 mm gap
Grade III (severe)	Complete tear >10 mm gap associated with posterolateral corner injury

Fig. 2.16 Physical exam maneuver dial test performed to diagnose LCL injury [38]

Diagnostic Studies

Radiographs cannot show direct damage to the LCL, but they can show small fractures that can increase suspicion for LCL injury. Radiographs can show a Segond fracture, an avulsion fracture of

the lateral tibial plateau typically associated with ACL injury, but can also indicate LCL injury. Varus stress radiographs can also be helpful in the diagnosis of LCL injury. The evidence is not strong, but one may see asymmetric lateral joint line widening which is increased in the lateral joint line when placed under a varus stress. MRI is still the most useful imaging when assessing for LCL injury because it is the modality of choice to also grade severity and particular location of LCL injury.

Musculoskeletal ultrasound can also be used but is technician dependent. The LCL with a grade I or II injury can show a thickened and hypoechoic LCL. A grade III injury can show associated edema, laxity at the lateral joint line and hypoechoic thickening of the LCL with a lack of fiber continuity [39].

Treatment

Similar to all other knee ligament injuries, acute LCL injury is treated with standard interventions, including ice, compression, rest, and analgesics. However, ice should not be applied for longer than 15 min if suspecting LCL injury because of its proximity to the common peroneal nerve. Paresthesia in the distribution of the peroneal nerve, or prolonged foot drop may occur with excessive cryotherapy.

Treatment of LCL injury is divided into non-operative and operative. Non-operative treatment is ideal for isolated grade I or grade II LCL injuries with no instability at 0 degrees of knee flexion, and consists of functional rehabilitation. Physical therapy emphasis is on quadriceps and hamstring strengthening.

Since LCL injuries are typically associated with other knee ligament injuries (such as ACL, PCL) research on isolated steroid or prolotherapy injections to the LCL is slim. However, as demonstrated earlier in this chapter, there has been some efficacy for prolotherapy injections with other knee ligaments, so a comprehensive prolotherapy treatment to the knee as whole could be used for LCL injury.

While it appears that the use of orthobiologics may be similarly applied for LCL injuries, there appears to be sparse published data with isolated LCL injuries.

Operative treatment includes LCL repair or LCL reconstruction for grade III injuries. LCL repair has higher failure rates [39]. The semitendinosus tendon autograft is the preferred graft for LCL reconstruction due to the length of the LCL. In addition, the semitendinosus tendon is closer in anatomical size as compared to other sources for grafts. If there is also a PLC injury, reconstruction may be necessary using other grafts, such as the hamstring or the Achilles tendon. PLC reconstruction involves the LCL, popliteus tendon, and the popliteofibular ligament [41].

For a Grade I injury, hinged bracing is necessary for 4–5 weeks during all weight bearing and physical therapy exercises. Quadriceps and hamstrings strengthening exercises start light and will then progress [39].

For Grade II, physical therapy is typically started on week two, with a hinged brace also worn at all times. Physical therapy range of motion exercises will start with non-weight bearing at week two and then progress to full weight bearing at week three, and continue for up to 16 weeks or as long as needed [39].

Return to Activities

As mentioned throughout this chapter, return to activity is based on many factors. Criteria for return to sport after an LCL injury include full painless knee motion, absence of significant tenderness or ligamentous laxity on exam, quadriceps and hamstring strength that is at least 90% of the unaffected lower extremity, and ability to complete sports-specific warm-up exercises without pain or difficulty.

In general, grade I return to activity is about 4 weeks, grade II is about 10 weeks, and grade III is about 10–14 weeks plus surgical recovery time [39].

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Meniscus Injuries

3

Amanda A. Kelly and Richard G. Chang

Pathology

The menisci are pads of fibrocartilage located between the femoral condyles and tibial plateaus. One of the roles of the menisci is to absorb shock and distribute stress across the knee to protect the cartilage of the knee joint. The menisci also play important roles in knee stability and proprioception. The medial meniscus is described as "C-shaped" and covers about 50–60% of the medial tibial plateau. Medial meniscus tears are more common than lateral meniscus tears. The lateral meniscus is described as "O-shaped," covers about 70–80% of the lateral tibial plateau, and has more excursion than the medial meniscus. The discoid meniscus variant is a thickened, ovoid variant of the lateral meniscus for which the posterior horn attachment is absent, resulting in increased motion and risk of injury [1].

The menisci are composed primarily of water (72%) along with collagen (22%), glycosaminoglycans (17%), DNA (2%), adhesion glycoproteins (<15%), and elastin (<1%). This composition gives the menisci their properties of absorption and joint lubrication.

A. A. Kelly (✉) · R. G. Chang
Department of Rehabilitation and Human Performance, Icahn School of Medicine at Mount Sinai, New York, NY, USA
e-mail: richard.chang@mountsinai.org

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Though primarily avascular structures, the menisci receive their blood supply from the peripheral branches of the popliteal arteries including the medial, lateral inferior, and middle geniculate arteries. Only about 10–25% of the periphery of the lateral meniscus and 10–30% of the medial meniscus are vascularized [2]. Meniscal tears may be classified as red zone or white zone tears depending on their location in relation to vascular supply. The outer 1/3 of the meniscus contains good vascular supply, thus tears located in this area are said to be in the red zone. On the other hand, the inner 2/3 of the meniscus contains poor vascular supply, thus tears located in this area are said to be in the red-white and white zone [1].

Meniscal tears are responsible for about 11% of acute knee disorders and 31% of chronic knee disorders [3]. They are caused by a combination of rotational forces with axial loading leading to shearing of the meniscus [2]. Meniscal tears are typically classified as acute or chronic depending on time course and mechanism of injury. They are also classified by pattern of tear (Table 3.1, Fig. 3.1). Horizontal tears, also known as "cleavage" or "fish mouth" tears, run parallel to the tibial plateau and divide the meniscus into superior and inferior halves. They are typically chronic and occur in the setting of degeneration. Vertical or longitudinal tears run perpendicular to the coronal plane and divide the meniscus into central and peripheral halves. They typically occur following

Table 3.1 Meniscal tear patterns

Tear pattern	Description
Horizontal	Runs parallel to tibial plateau and divides the meniscus into superior and inferior halves, typically chronic
Vertical/ longitudinal	Runs perpendicular to coronal plane and divides meniscus into central and peripheral halves, typically occurs following trauma
Bucket handle	Full-thickness vertical/longitudinal tears involving a displaced meniscal fragment entering intercondylar notch, limits full extension of knee
Radial/ transverse	Runs perpendicular to tibial plateau and long axis of meniscus
Oblique	Occurs at the junction of the posterior and middle body of the meniscus and are unstable
Complex	Involves combination of horizontal, vertical, radial elements

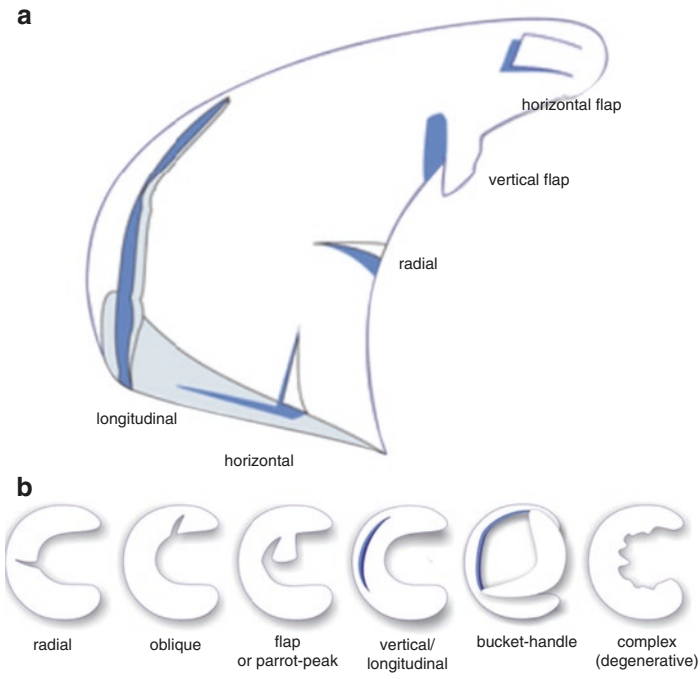

Fig. 3.1 (**a**) ISAKOS (International Society of Arthroscopy, Knee Surgery and Orthopaedic Sports Medicine) classification of meniscal tears. (Image reprinted [6]) (**b**) Meniscus tear patterns. (Image reprinted [6])

trauma in young patients. Bucket handle tears are caused by full-thickness vertical/longitudinal tears and involve a displaced meniscal fragment entering the intercondylar notch, thus limiting full extension of the knee joint. These account for 10% of meniscal tears and are more commonly associated with the medial meniscus. Radial or transverse tears run perpendicular to the tibial plateau and long axis of the meniscus [4]. Oblique tears, including "parrot beak" or "flap" tears, typically occur at the junction of the posterior and middle body of the meniscus and are unstable [5]. Complex tears involve a combination of horizontal, vertical, and radial elements [4]. Lastly, meniscal root tears occur in the anterior or posterior root of the medial or lateral meniscus. They often occur concomitantly with ligamentous injuries, particularly ACL tears [5].

Clinical Presentation

Acute presentations of meniscal injuries are typically traumatic and usually occur in young adults. Patients commonly report immediate onset of pain at the joint line with associated effusion. Chronic presentations of meniscal injuries are usually degenerative in nature and occur in adults greater than 45 years of age. Patients are often unable to recall a specific inciting event, may report pain with activities of daily living, and may report intermittent effusions. It can be difficult to differentiate meniscal injuries from degenerative joint disease in this population, however, those with meniscal injuries may report more localized pain, faster progression of symptoms, and are more likely to report mechanical symptoms.

Mechanical symptoms are common in meniscal injuries and include painful clicking, popping, and catching. If patients report locking with associated decreased range of motion of the knee, consider bucket handle tears. However, locking may also occur with other pathologies such as loose bodies, patella maltracking, and articular cartilage defects [4].

Patients suffering from medial meniscus injuries typically have a history of tibial rotation with knee flexion while weight-bearing. This motion is typically seen in sports such as soccer and football [1]. Pain is localized to the medial joint line, and patients may report pain when crossing their legs. Medial meniscus injuries are also associated with chronic acute cruciate ligament (ACL) deficiencies [7].

The lateral meniscus is commonly injured by squatting in full flexion with rotation. This is seen in sports such as wrestling. In traumatic presentations associated with large effusions, consider a concomitant ACL rupture. Acute ACL ruptures are present in up to 73% meniscus injuries and typically involve the lateral meniscus [8].

Physical Exam

The basic components of a knee exam including inspection, range of motion, palpation, strength testing, and provocative maneuvers should be utilized when evaluating for meniscal injuries. It is important to evaluate the entire extremity including the hip, as well as the back and compare the affected side to the unaffected side.

Inspection

Evaluate for any bony abnormalities, bruising, erythema, and effusions. It is also important to check extremity alignment as varus malalignment increases stress and the chance of injury to the medial meniscus. Valgus malalignment increases stress and the likelihood of injury to the lateral meniscus. Atrophy or asymmetry of muscles can help determine chronicity [7].

Range of Motion

This can often be limited in acute meniscal tears, but may be normal in chronic, degenerative tears [4]. The normal range of motion with knee extension is 0°–10°, and the normal range of motion with knee flexion is 130°–150° [7].

Palpation

Palpate along the joint lines and posterior knee for tenderness. Feel for warmth, effusion, and crepitus.

Strength Testing

Evaluate strength of both lower extremities.

Provocative Maneuvers

Ligamentous Injuries

Evaluate for possible ligamentous injuries by testing the integrity of the cruciate and collateral ligaments with Lachman test, anterior/posterior drawer tests, and varus and valgus instability at 0° and 30° knee flexion [4]. Please see the Ligament Injuries chapter for further information.

Thessaly Test

The maneuver is described in Fig. 3.2. The test is positive if the patient experiences pain at the joint lines or a catching/locking sensation. This test has the highest sensitivity and specificity for meniscal tears with a sensitivity of 98% for medial meniscus and 92% for lateral meniscus, and a specificity of 97% for medial meniscus and 96% for lateral meniscus [9].

McMurray Test

The exam is described in Fig. 3.3. The test is positive if the maneuver reproduces the pain or the examiner feels or hears a click. This test has a low sensitivity but high specificity for meniscal injury [3].

Fig. 3.2 Thessaly test: patient stands on affected lower extremity while lifting the unaffected lower extremity off the ground while the examiner holds patient's outstretched hands (**a**). Patient then rotates the knee and body internally (**b**) and externally (**c**) at 5° and 20° of knee flexion

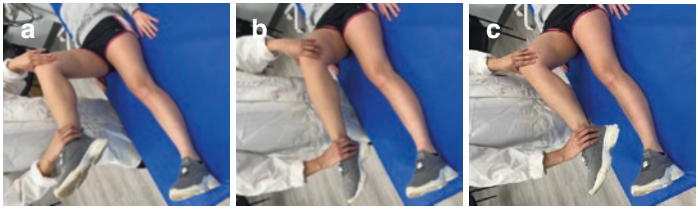

Fig. 3.3 McMurray test: patient lies supine while flexing the affected knee at 20°–30° and hip to 90° (**a**). The examiner places a hand over the knee joint and uses the other hand to hold the lower extremity to extend while internally (assessing lateral meniscus) (**b**) and externally (assessing medical meniscus) (**c**) rotating the tibia

Apley Grind Test

Patient lies prone with affected knee flexed at 90° and hip extended. The examiner then provides axial pressure on the foot and rotates the tibia internally and externally (Fig. 3.4). The test is positive if pain is reproduced at the joint lines [4].

Bounce Home Test

Patient lies supine with knee and hip in full flexion. The patient's heel is placed in the examiner's hand, and the patient is then asked to passively extend. The knee should extend completely (Fig. 3.5). The test is positive if the knee is unable to extend completely or has a rubbery end feel [2].

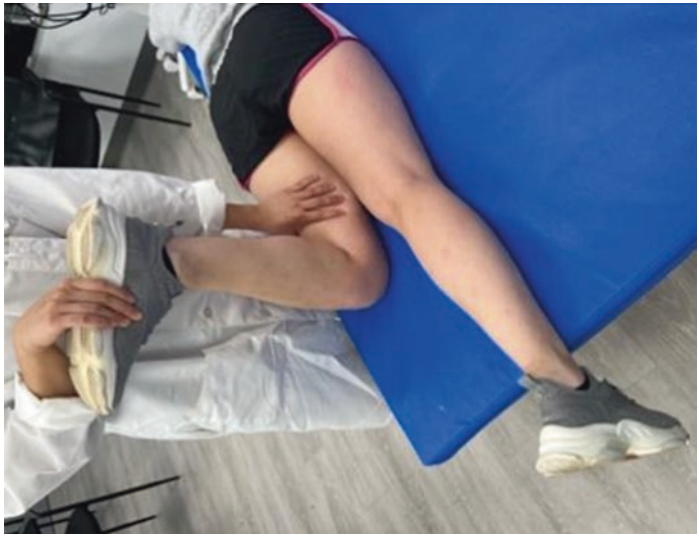

Fig. 3.4 Apley test: patient lies prone with hip extended and knee flexed at 90°. The examiner provides axial force to the foot while rotating the tibia internally and externally

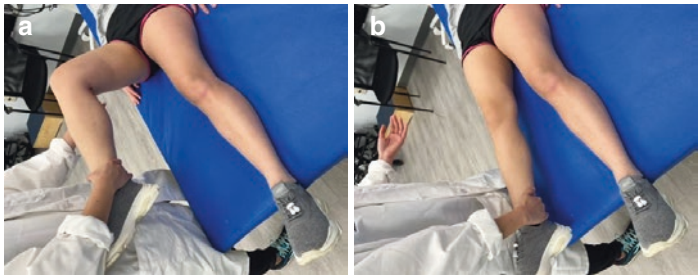

Fig. 3.5 Bounce home test: patient lies supine with lower extremity fully flexed (**a**). Examiner's hand is placed under the patient's heel and the patient passively extends the lower extremity (**b**)

Fig. 3.6 Steinmann part 1 test: patient lies supine with affected knee flexed at 90°. The examiner holds the affected knee with one hand and tibia (**a**) with the other and internally (**b**) and externally (**c**) rotates the lower leg

Steinmann Part 1 Test

The exam is described in Fig. 3.6. The test is positive if pain is reproduced at the joint lines [4]. Of note, Steinmann part 2 test is used to distinguish meniscal pathology from injury secondary to ligaments or osteophytes rather than diagnosis of meniscal pathology.

Diagnostic Studies

Plain Radiographs

Though plain radiographs are not diagnostic of meniscal injury, they are important in ruling out other causes of knee pain such as degenerative changes, loose bodies, chondrocalcinosis, etc. Weight-bearing anterior–posterior, lateral, and patellofemoral views should be obtained. Full-length lower extremity standing views may also be useful to assess alignment and osteoarthritis changes [4].

Magnetic Resonance Imaging

This has high sensitivity and specificity in diagnosing meniscal injuries. MRI sequences used for meniscal pathology evaluation include fat-suppressed and diffusion-weighted fast spin-echo (cartilage) sensitive in axial, coronal, and sagittal views. Normal meniscal structure is signified by uniform low signal intensity on fast spin-echo and fat-suppressed images [5]. Areas of hyperintensity within the meniscal area may demonstrate degeneration.

The following two criteria must be met to diagnose a meniscus tear: abnormal signal in the meniscus suggesting a tear seen on at least two consecutive images and visualization of a meniscal tear in two planes (sagittal and coronal). When both of these criteria are met, the diagnostic accuracy is greater than 90% [4, 10]. MRI classification for meniscal tears is based on meniscus signal intensity [1] and is outlined in Table 3.2 and demonstrated in Fig. 3.7.

Table 3.2 MRI classification for meniscal tears

	Findings
Grade 1	Small focal area of hyperintensity without extension to articular surface
Grade 2	Linear area of hyperintensity without extension to articular surface
Grade 3	Hyperintensity extends to at least one articular surface, referred to as a definite meniscal tear

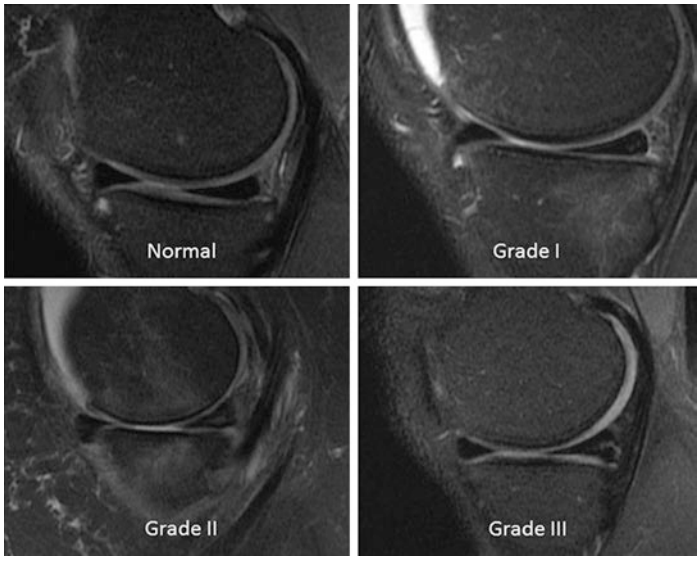

Fig. 3.7 MRI classification for meniscal tears. (Image reprinted [6])

It is important to note that incidental findings of meniscal pathology on MRI are common and increase with age. In patients with radiographic evidence of knee osteoarthritis (Kellgren–Lawrence grade ≥2), the prevalence of a meniscal tear is 63% in symptomatic patients (knee pain, aching, stiffness on most days) and 60% in asymptomatic patients [11].

A discoid meniscus is a congenital variant that describes an enlarged meniscus with further central extension onto the tibial plateau and more commonly occurs in the lateral compared to medial meniscus. The three types of discoid menisci are incomplete, complete, and Wrisberg (lacks normal posterior ligament and capsular attachments) [4]. The Wrisberg type can result in instability and is often referred to as "snapping-knee syndrome" [5]. A diagnosis of discoid meniscus is made on MRI when the

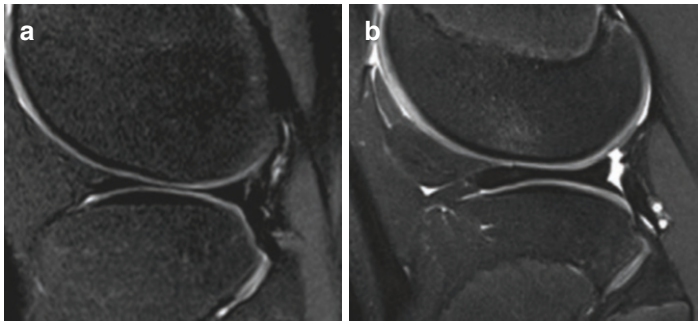

Fig. 3.8 MRI discoid meniscus. (Image adapted [4]). T2-weighted MR images of lateral meniscus (**a**) normal (**b**) discoid

meniscus body measures 15 mm or more on a midline coronal image or when three or more bowtie shapes are seen on continuous sagittal images. The sensitivity and specificity of MRI detection of a discoid tear are highly variable [4] (Fig. 3.8).

CT Arthrography

This may be used in patients with contraindications to MRI. Disadvantages to this modality include the use of intravenous and intra-articular contrast and radiation exposure [4].

Ultrasound

This is not typically used for the diagnosis of meniscal pathology as its accuracy is user dependent. However, studies have shown some promise in the use of diagnostic ultrasound in identifying meniscal injuries. A meta-analysis evaluating seven prospective studies demonstrated summary estimates of sensitivity, specificity, positive likelihood ratio, negative likelihood ratio, and diagnostic odds ratio of ultrasonography in the diagnosis of meniscal

injury to be 0.88 (95% CI 0.84–0.91), 0.90 (95% CI 0.86–0.93), 7.07 (95% CI 4.34–11.52), 0.17 (95% CI 0.10–0.26), and 58.13 (95% CI 24.38–138.62), respectively [12].

Arthroscopy

The gold standard for the diagnosis of meniscal pathology is a complete diagnostic arthroscopy with examination of all intra-articular structures. It is used for both the confirmation of diagnosis and surgical treatment of meniscal injury when nonoperative management is not indicated or has failed [5].

Treatment

Nonoperative management is recommended for patients with minor tears and/or those without significant limitations in function. The types of tears that may be treated nonsurgically include partial-thickness longitudinal (vertical) tears, small (<5 mm) full-thickness peripheral tears, and minor inner rim or degenerative tears [13]. The nonoperative approach aims to manage symptoms rather than heal the meniscal tear as most unrepaired meniscal tears will not heal. Conservative management includes rest, ice, nonsteroidal anti-inflammatory drugs, and activity modification for 6–12 weeks [5]. Other recommendations include cryotherapy, bracing, weight loss, aspiration if there is a large effusion present, and physical therapy. The goals of therapy are to decrease pain and swelling and increase range of motion, muscle strength, and endurance. Static strengthening along with electrical stimulation are used to combat quadriceps atrophy. With time, the addition of aerobic conditioning and open/closed kinetic chain exercises in all three planes with stretching of the lower limb may be utilized. Gradual resolution of symptoms within a period of 6 weeks and normal function by 3 months has been seen with some meniscal injuries [13].

The use of intra-articular injections of corticosteroids, analgesic medications such as lidocaine or bupivacaine, and viscosupplementation may be considered if there is associated underlying

osteoarthrosis. It is important to note that corticosteroids may hinder meniscal healing [13].

Orthobiologics are continuing to be studied in the treatment of meniscal injuries. These include platelet rich plasma (PRP), mesenchymal stem cells (MSCs), or micro-fragmented adipose tissue (MFAT) [14]. PRP, which is derived from autologous whole blood, is made up of numerous growth factors and cytokines that has been shown to promote healing [2]. Intraoperative PRP injections given to patients that underwent open meniscal repair has been shown to significantly improve pain and function [15]. Intraoperative PRP injections administered to patients that underwent index arthroscopy were also shown to significantly improve meniscus healing and functional outcome [16]. Studies examining the role of intrameniscal PRP injections without surgery have also demonstrated improvement in pain [17].

MSCs, a subdivision of stem cells, have been isolated from bone marrow (BM), periosteum, trabecular bone, adipose tissue, skeletal muscle, and deciduous teeth [14]. BM-derived MSCs have been shown to significantly improve knee pain after partial meniscectomy [18]. Adipose-derived stem cells (ASCs) are MSCs originating from adipose tissue with the ability to differentiate into numerous cell lines and have been shown to yield more stem cells when compared to BM-derived MSCs. In vitro studies have revealed the potential of ASCs to differentiate into chondrogenic and osteogenic cells with improved healing of articular cartilage in animal models, however, there are limited studies evaluating the use of ASCs in humans with meniscal injuries [14]. MFAT has been used in the treatment of knee osteoarthritis among other musculoskeletal conditions. A case report demonstrated the successful treatment of a degenerative meniscal tear in a triathlete with MFAT [19].

Operative management is considered when patients experience significant functional limitations without improvement following conservative management or those with large tears causing mechanical symptoms. The goal of surgery is to maximize the preservation of the meniscus. When able, a meniscal repair is preferred over a meniscectomy. As the outer 1/3 of the meniscus has good vascular supply, tears in this area can often be repaired.

However, the inner 2/3 of the meniscus is poorly vascularized, therefore meniscal tears in this area may require removal of tissue through meniscectomy [1]. Meniscal repair allows for the greatest preservation of tissue, however, it typically involves a longer rehabilitation process. In contrast, meniscectomy allows for a faster return to play, but increases the future risk of osteoarthritis. Lastly, meniscal transplant is considered when a patient continues to be symptomatic, wants to maintain an active lifestyle, and there is minimal viable meniscal tissue with high risk of progression to osteoarthritis. Ideally, the patient should have undergone total or near-total meniscectomy prior to the procedure [20].

Return to Activities

Rehabilitation for Nonoperative Patients

The goals of physical therapy include the maintenance of range of motion and improvement in strength and endurance. An athlete will advance through resistance exercises as tolerated and will subsequently slowly return to running. Nonimpact conditioning such as swimming and cycling is also recommended. Prior to returning to sports activity, athletes should exhibit at least 70–80% strength in the injured side compared to the uninjured side. The time course in which an athlete may return to sports activity is variable and depends on the patient's goals and severity of injury [21].

Rehabilitation Following Surgery

The patient advances through activities as tolerated. The athlete may return to competitive sports when he/she has achieved equal strength, full range of motion, and endurance in sports-specific activities. Accepted rehabilitation protocols are outlined in Table 3.3 [20]:

Table 3.3 Rehabilitation protocols following surgeries

	Meniscectomy	Meniscal repair	Meniscal transplant
Phase 1	*0–2 weeks:* WB and ROM exercises as tolerated. Hamstring, quadriceps, and core-strengthening exercises	*0–2 weeks:* Fully WB in extension, +/− brace, ROM 0°–90° NWB, isometric quadriceps strengthening	*0–2 weeks:* TTWB brace locked in extension, ROM 0°–90° without brace and NWB, isometric quadriceps strengthening
Phase 2	*2–4 weeks:* Sports-specific exercises, return to cardio training	*2–6 weeks:* Fully WB in extension, ROM 0°–90° NWB, closed chain exercises, terminal knee extensions	*2–8 weeks:* 50% WB at weeks 2–4, full WB at week 4, brace unlocked 0°–90° at weeks 2–6, ROM 0°–90° when NWB, closed chain exercises, terminal knee extensions, all activities with brace then discontinue brace at 6 weeks
Phase 3	*4–6 weeks:* Progress in sports-specific training, maintain strengthening program	*6–12 weeks:* Fully WB, discontinue brace, full ROM, hamstring and proprioception exercises, leg presses 0°–90°, stationary bicycle	*8–12 weeks:* Full WB w/o brace, full ROM, hamstring and proprioception exercises, leg press 0°–90°, stationary bicycle
Phase 4	N/A	*12–20 weeks:* Progress with exercises, start swim at 12 weeks, sports-specific exercises, run/jump protocol at 16 weeks	*12–20 weeks:* Progress with exercises, swim at 12 weeks, elliptical Run/jump protocol at 16 weeks
Phase 5	N/A	N/A	*More than 20 weeks:* Advance to sports-specific activities

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Osteoarthritis of the Knee

4

Aaron Bolds and Subhadra Nori

Introduction and Pathophysiology

Osteoarthritis (OA) remains the most common type of arthritis, with over 6% of U.S. adults aged 30 and over being clinically affected [1]. The prevalence of knee OA in the US is approximately 34%, with women representing roughly 10% more [2]. As populations continue to age, an increase in the prevalence of osteoarthritis is inevitable. There is a large economic burden associated with osteoarthritis secondary to the impact on disability. Many of the risk factors linked to osteoarthritis cause significant comorbidities as well, thus increasing risk for functional disability. The increasing prevalence of obesity continues to be of concern as it leads to increased "wear and tear" on large joints, specifically the knee. The knee is the largest synovial joint in the body and the most common site of OA associated with disability.

The joint is covered by an articular cartilage and surrounded by a synovial bursa. Extracellular matrix components such as type II collagen and other proteoglycans house receptors that allow chon-

A. Bolds
Mount Sinai Hospital, New York, NY, USA

S. Nori (✉)
NYC Health + Hospitals, New York, NY, USA
e-mail: NORIS@nychhc.org

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drocytes to detect mechanical stress. Metalloproteinases are key players in cartilage matrix degradation. Their upregulation is secondary to repeated mechanical or inflammatory stimulation [3]. Chondrocytes can undergo phenotypic transformation and express inflammatory mediators such as cytokines and chemokines, in response to repeated stimulation. During the remodeling process there is new blood vessel formation, known as vascular channels. These channels have sensory nerve endings and their articular cartilage innervation is a pain generator [3]. This cycle of cartilage damage and remodeling is the primary pathological feature of osteoarthritis.

Unique to the morphology of the knee joint is the infrapatellar fat pad, also referred to as Hoffa's fat pad. This structure is composed of a fibrous network, including a layer of adipose tissue. It is located beneath the patella and within close proximity of other structures including bone, articular cartilage and synovium [4]. Hoffa's fat pad contains many immune cells that are involved in producing inflammatory mediators. There are nociceptive nerve fibers present in Hoffa's fat pad. Inflammatory mediators, such as cytokines, may play a role in altering the sensitivity of nerve fibers and decreasing pain threshold [4]. Thus, the infrapatellar fat pad must be considered a significant factor in the pathology of knee OA.

Risk Factors

OA is more than just a "wear and tear" disease process. It is better defined as a multifactorial process influenced by, but not limited to, genetic history, inflammation, mechanical forces, immune-mediated cellular processes, biochemical processes, age, sex, and body composition. Risk factors are split into non-modifiable and modifiable. The most common modifiable risk factor is obesity which leads to increased joint loading and early disease progression of OA. For every pound of bodyweight gained, there are two to four pounds of load bearing pressure added to the knee joints [2]. The added load bearing is not the only contributing factor of obesity to knee OA. Obesity is also associated with negative

effects related to inflammation and psychological factors, such as sedentary lifestyle leading to loss of protective muscle mass surrounding the joint.

There is an association of estrogen deficiency and high incidence of OA in post-menopausal women. There are conflicting studies in regard to these mechanisms, as some studies show effects of estrogen, such as increased bone mass, may counteract the effect of estrogen on OA [1]. It has been established that there are sex differences in the incidence and severity of knee OA. Knee and hand OA appears to be more prevalent in women and African-American population. In addition to increased prevalence, women typically report higher pain burden and decreased overall function compared to men [5]. Understanding the mechanisms responsible for the above mentioned sex differences in OA will require further epidemiologic and pathophysiologic studies in the future.

There have been nutritional factors implicated in the disease process of OA. Chondrocytes are sources of reactive oxygen species (ROS), which can influence cartilage collagen and synovium degradation [1]. Therefore, antioxidant consumption may play a role in protection against OA, given its defense against tissue injury. Vitamin C has been found to decrease apoptosis and the expression of pro-inflammatory mediators, such as cytokines and metalloproteinases [6]. It also has antioxidative properties which can protect against reactive oxygen species created by chondrocytes during knee OA disease progression. Vitamin D deficiency can impact the bone response in OA and lead to increased severity. The Framingham cohort study on OA showed the risk for OA progression was increased threefold in persons with vitamin D deficiency, thus indicating the nutritional value of vitamin D in the knee OA disease process [1].

A small percentage of knee OA cases are genetically predisposed. Some of the genes previously studied for their up/down regulatory involvement in OA include, but are not limited to: insulin-like growth factor I genes, vitamin D receptor gene, neuronal growth factor, cytokine receptor-like factor 1, and tumor necrosis factor alpha-induced protein 6 [1]. Future studies are needed to understand the mechanisms by which these genes affect

disease occurrence. This will allow for proactive strategies to be utilized to prevent disease occurrence or progression.

There are also biomechanical risk factors associated with knee OA, such as varus–valgus laxity, knee–hip–ankle alignment, joint injury or alteration of anatomy, muscle weakness, job and sporting activities [1]. Increased joint instability secondary to articular surface fractures, ligamentous injury (ACL, PCL, MCL, and LCL), and menisci can lead to development of knee OA. High impact, high intensity sports have been shown to increase risk of knee OA. American football and soccer are common sports that involve repetitive pivoting and explosive horizontal and vertical forces. Decreasing risk of OA among sports participants is a growing topic. In conjunction with joint stability training; modifications to protocols, equipment, and playing surfaces can help lower risk [1]. It is vital that the aging population stays active and incorporates some level of physical activity at least 3–4 days per week. Including lower extremity strength training will help reduce obesity and also help maintain protective mass to muscles surrounding the knee joint, specifically the quadriceps.

Clinical Presentation

Patients with knee OA can present in various ways depending on the etiology. However, pain is the most common symptom, with various characters and/or temporal patterns. Patellofemoral area may be involved with anterior knee pain and joint line pain with activity could suggest meniscal involvement. Patients may experience crepitus, knee buckling, leg weakness, and locking. Swelling can also intermittently accompany pain, especially following high impact activities. Decreased functional ability can be a symptom, often reported as difficulty ambulating stairs, prolonged sitting, and performing activities of daily living (ADLs).

As a result of pain, patients may report difficulties with tolerating physical activity. This may lead to a sedentary lifestyle, exacerbating the modifiable risk factors associated with knee OA. As a clinician, it is important to screen these patients for depression, as a psychosocial component can be linked.

Physical Exam

Inspection should include evaluating for edema, skin changes, erythema, and deformities. Palpation should be used to locate any point tenderness proximal to the medial and lateral joint lines. Posterior and anterior aspects of the knee should be palpated to assess for baker's cyst or patellofemoral point tenderness, respectively. Range of motion should be tested, paying close attention to patellar maltracking, known as the J-sign. Instability of the joint can be detected by applying varus and valgus stress to the knee joint. Functional tests such as squatting, hopping, calf raises, and jumping can be used to see which mechanical movements illicit pain. Lower extremity strength should be tested bilaterally, being careful to differentiate true weakness versus pain limited weakness. Gait testing should be used to identify any malalignment and instability.

Special tests can be used to identify any superimposed ligamentous or meniscal pathology. There are many special tests with varying sensitivity and specificity. With respect to knee OA, the integrity of the patellofemoral and meniscal structures can be evaluated with some of the following tests: Meniscus (McMurrays, Apley's test, Thessaly test) and patellofemoral (Apprehension and Grind test/Clarkes sign).

Diagnosis

History taking and physical exams are utilized for diagnosis but definitive diagnosis is typically found radiographically. However, the American College of Rheumatology suggests that the diagnosis of knee OA can be made without radiographic evidence [2]. The clinical diagnosis must include at least 3 of the following: mechanical crepitus, morning stiffness lasting less than 30 min, age 50 and older, bony enlargement or tenderness on the knee exam, and no palpable warmth. The most frequently used grading system is the Kellgren and Lawrence system, including 4 grades. Grade 1 does not include joint space narrowing, but the initial formation of osteophytes on articular surfaces. Grade 2 is charac-

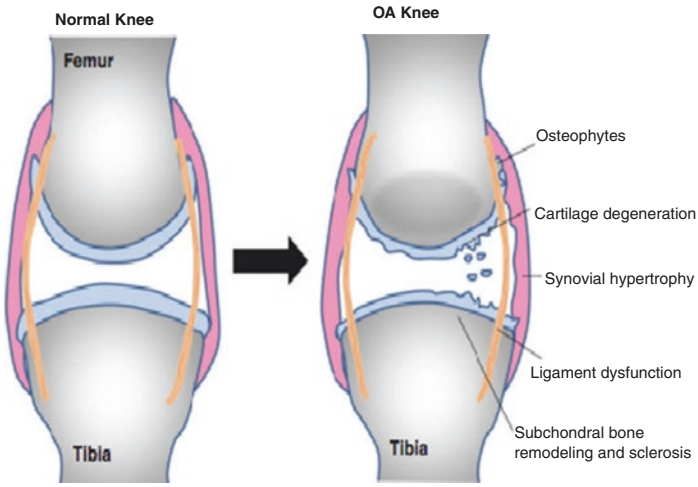

Fig. 4.1 Depiction of degenerative changes in knee OA [4]

terized by mild joint space narrowing and definite osteophytes. Grade 3 is similar to grade 2 except the joint space narrowing is worse and osteophytes are more abundant. Also, in grade 3 there may be sclerosis and bony deformity. Grade 4 reveals severe joint space narrowing and bony sclerosis, large osteophytes, and definite bony deformity at the tibial plateau [2]. Figure 4.1 depicts various pathological changes seen with knee OA.

Although plain radiographs and MRI are imaging techniques most frequently used for knee joint evaluation, ultrasound use is becoming more popular. There are advantages to ultrasound such as low cost, no radiation exposure, portable, and high sensitivity. It is also possible to evaluate structures dynamically. Limitations to ultrasound use include its user dependence, inability to display deep structures within the joint, and limited evidence on reliability [7]. Typically, ultrasound is not used for definitive diagnosis of knee OA and instead is used diagnostically or to guide the needle for therapeutic injections. However, as more research is conducted on the validity and reliability of ultrasound, development of standardized diagnostic methods may follow.

Treatment

Some of the conservative methods for treating OA are also methods for preventing OA. Exercising for weight loss and lower extremity muscle strengthening should be considered first-line. These exercise programs can be done solely by the patient or under supervision from a therapist or trainer. Supervised exercise programs may be more beneficial for those without significant experience with aerobic or strengthening exercises, in order to prevent injuries. A supervised program will allow for strength deficits, poor technique, and malalignment during dynamic exercises to be corrected. It is important that patients with knee OA participate in low impact exercises such as cycling, elliptical machines, and swimming. A systematic review and meta-analysis showed that stationary cycling exercise relieves pain and improves sport function in patients with knee OA, but may not be as clinically effective for improving stiffness, daily activity, and quality of life [8].

A structured physical therapy program will allow patients to participate in supervised aerobic and strengthening exercises, as well as utilize other noninvasive treatment modalities. Cold and hot compression, transcutaneous electrical nerve stimulator (TENS), and neuromuscular electrical stimulation are available. Structured programs will allow for the evaluation of gait and potential need for an assistive device or bracing.

Oral and topical NSAIDs are used as first-line medications in patients with knee OA. In a meta-analysis, diclofenac at a dose of 150 mg per day was found to be the most effective NSAID for pain and function [2]. Patients must be monitored for side effects of NSAID use such as gastrointestinal and renal issues. Opioids are usually not prescribed for knee OA but tramadol is sometimes used on a case by case basis, especially in patients with debilitating pain. Tylenol can be used safely at a dose of 3000 mg per day, typically taken as 1000 mg three times a day. A literature review showed that capsaicin has a good safety profile and efficacy in reducing knee OA, despite the studies having limitations [9].

Common interventions for treatment of knee OA include intra-articular injections with corticosteroids, viscosupplementation,

and platelet-rich-plasma (PRP). Corticosteroid injections help to disrupt the inflammatory cascade and reduce pain. There is often short-term pain reduction with corticosteroid injections, which is why they should be coupled with physical therapy to maximize overall benefit. Pain can be a limiting factor for patients not tolerating a physical therapy program. Viscosupplementation injections with hyaluronic acid are used to compensate for lack of cushion and lubrication in the knee OA joint. There are different brands of hyaluronic acid that can be used depending on insurance coverage, some including a series of injections. Platelet-rich plasma (PRP) is derived from the patient's blood and then injected into the knee joint. PRP is not currently FDA approved for the treatment of knee OA. However, studies have shown the benefits of PRP to include reduced pain, improved joint function, and potential cartilage repair, although more studies are required to validate the efficacy [10]. Corticosteroid and PRP injections are commonly performed under ultrasound guidance for visualization of the needle and to scan for any other pathology that may be associated with the knee OA. Figure 4.2 shows an intra-articular corticosteroid injection with effusion in suprapatellar recess.

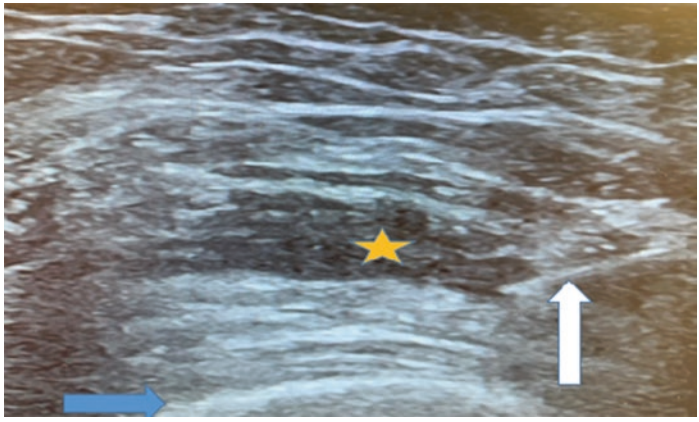

Fig. 4.2 Blue arrow—patella, yellow star—suprapatellar recess, white arrow—needle tip

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Patellofemoral Pathologies

5

Caroline Varlotta and Ian O'Connor

Introduction

Patellofemoral joint pain is one of the most common conditions presenting to a musculoskeletal physician's office. Pathology of this joint is common in athletes and other young active individuals. Due to its anatomical location, most pathology related to the patellofemoral joint is on the anterior aspect of the knee.

The posterior surface of the patella articulates with the femur and is divided into seven facets. Each facet is convex, allowing for articulation with the concave femoral surface. The distal femur will form an inverted U-shaped intercondylar groove to articulate with the patella. The lateral facet of the patella and femur are larger and extend proximally to improve patellar stability. The angle between the lateral and medial femoral condyles is the sulcus angle. Patellar subluxation is associated with a greater sulcus angle and can contribute to patellofemoral pain syndromes.

The orientation of the patella can affect biomechanics of the knee joint. When the patella's orientation is superior or inferior to the normal alignment, this is known as patella alta or patella baja, respectively. The patella can also have an inferior, superior, lat-

C. Varlotta (✉) · I. O'Connor
Mount Sinai Hospital, New York, NY, USA
e-mail: ian.oconnor@mountsinai.org

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eral, or medial tilt. It is also possible to have lateral and medial rotation of the patella. These different malalignments predispose patients to patellofemoral pathologies [1].

Biomechanics of the patellofemoral joint are dependent on the components' static and dynamic stability due to the shallow fit between the patella and femur. Static stability is supplied by the patellar tendon, joint capsule, medial ligaments (MPFL, medial patellofemoral ligament), lateral ligaments (IT band, lateral patellofemoral joint ligament, lateral retinaculum), and the joint capsule. The Q-angle is important for static and dynamic stability. To conserve patellar alignment in motion, the dynamic stabilizers such as the quadriceps, pes anserine muscles, and biceps femoris are necessary.

These components of stability enable the patella to act as a mechanical pulley for the quadriceps as it changes the direction of the extension force in the knee throughout range of motion. Contribution from the patella increases with progressive extension and is critical in the last 30 degrees of extension [2].

Patellar tracking describes the dynamic movement of the patella and depends on active contraction of the quadriceps and alignment of the patella in the trochlear groove. With open chain movements, the patella will glide superiorly with knee extension, as previously mentioned, and inferiorly with knee flexion. The contact surface area of the patella on the femur increases with increased knee flexion to disperse forces over a greater joint area. In addition to superior and inferior motion, the patella moves slightly laterally with extension and medially with flexion [3]. The patella will be restrained by the quadriceps in closed chain exercises, and therefore the femoral surface will glide posteriorly to the patella [1]. Maltracking of the patella may create narrow contact pressure points between the patella and the femoral groove, associated with anterior knee pain.

Anatomic and biomechanics are important to consider in any patient with patellofemoral pathology. Identification of malalignment or poor biomechanics may be crucial in determining appropriate treatment.

Patellofemoral Pain Syndrome

Patellofemoral pain syndrome (PFPS) is characterized by anterior knee pain around or behind the patella that cannot be attributed to another intra-articular or peripatellar pathology. Although no true consensus exists about the etiology of PFPS, the concept of overloading the knee's extensor mechanism is fundamental to all theories of the underlying pathophysiology. PFPS appears to be multifactorial with causative factors including overuse, malalignment, and trauma. Of these factors, overuse is the most common. PFPS is associated with periods of increased physical activity, high mileage in runners, and activities that overload the knee's extensor mechanism. The overload leads to subchondral bone degeneration, retinacular strain, and damage to small nerves [4–6].

Although the majority of patients with PFPS do not demonstrate malalignment during physical or radiographic examination, multiple authors suggest patellar malalignment, and therefore abnormal patellar tracking, is the primary etiology of PFPS. Static biomechanical risk factors for malalignment include leg length discrepancy, hamstring and hip muscle tightness, abnormal patellar mobility, abnormal trochlear morphology, angular and rotational deformities of the lower extremity, abnormal foot morphology, and hallux valgus. Dynamic biomechanical risk factors include muscle weakness or imbalance (ex. vastus medialis and hip abductors), knee abduction impulses, and excessive or insufficient foot pronation [7, 8]. The dynamic Q-angle is an important anatomical factor in PFPS. Females tend to exhibit a greater dynamic Q-angle than males when performing tasks associated with patellofemoral pain [9, 10].

Prior research has also indicated the subtalar joint of the foot may influence the positioning of the patella, potentially leading to PFPS. Tiberio suggests excessive pronation leads to tibia and femoral internal rotation, causing a relatively lateral patella, which may lead to PFPS [11]. Even though subtalar joint pronation may be a factor leading to PFPS, not all individuals with PFPS will have subtalar pronation during ambulation [12].

PFPS can also be caused by direct or indirect trauma to structures around the patellofemoral joint. For example, injury can occur secondary to falls, contact sports, or motor vehicle collisions [13].

PFPS is most common in active individuals accounting for nearly 25% of all identified knee injuries. The pathology occurs more often in patients participating in competitive sports than age matched controls. The ratio of women to men is nearly 2:1 and it occurs disproportionately in the second and third decades of life [13, 14].

Clinical Presentation

PFPS often presents as anterior knee pain poorly localized under or around the patella. Onset can be acute or gradual. Presentation can be unilateral or bilateral. Pain is often characterized as achy or dull but can be sharp. It is typically aggravated by loading the patellofemoral joint during weight bearing on a flexed knee, which occurs with activities such as squatting, running, prolonged sitting, or ascending and descending steps. History of overload or trauma should be obtained upon presentation [5].

Physical Exam

Physical exam should begin with observation of risk factors such as obesity, vastus medialis atrophy, or angular and rotational deformities. Assess for localized erythema, warmth, or effusion. Assess active and passive range of motion and observe any maltracking (J-sign). Patellar tendon tenderness may be present, though tenderness at the inferior border of the patella is more associated with tendinopathy. Test for risk factors such as quadriceps weakness, hip abductor and external rotator weakness, or strength discrepancies between affected and unaffected lower extremities. Patients usually have full range of motion of the knee. Observe gait for excessive varus or valgus knee movement and observe foot strike for excess supination or pronation. Of note, 80% of patients with PFPS experience pain with squatting [5, 6, 15].

Diagnostic Studies

PFPS is a clinical diagnosis and therefore imaging studies are unnecessary for initial management. Indications for imaging include history of trauma, overt instability, prior surgery, pain at rest, or failure to improve after a comprehensive rehabilitation program. When indicated, plain films of the knee (weight bearing PA, weight bearing lateral, sunrise view) are useful to rule out other sources of anterior knee pain such as bipartite patella, osteoarthritis, loose bodies, and occult fractures. Similarly, advanced imaging modalities (MRI, CT, US, radionuclide scanning) are not indicated in initial evaluation but can aid in diagnosis of pathology other than PFPS [16].

Treatment

In the acute phase (weeks 1–2), activity modification should be encouraged. Patients should avoid activities that cause pain such as running, jumping, squatting, and ascending or descending stairs. NSAIDs should be used acutely and then tapered off as pain decreases. Although evidence is lacking, applying ice to the anterior knee is reasonable for pain relief.

In the recovery phase (weeks 3–6), combined physical therapy and adjunctive therapy is recommended. Physical therapy should begin with a physical therapist or comparable professional and exercises should not cause pain. Exercise programs should include: quadriceps stretching, hamstring stretching, iliotibial band stretching, hip strengthening, quadriceps strengthening, core stability, and proprioceptive exercises. The lateral forces on the patellar are often stronger so a program that addresses muscle imbalance (ex: vastus medialis, hip abductors) would be appropriate. Adjunctive therapy can include foot orthoses to control excessive foot pronation or supination, patellar bracing, and patellar taping to allow pain-reduced range of motion and balance contractile forces [6, 15, 17].

Evidence is lacking for surgical intervention in PFPS and is considered a treatment of the last resort. Operative treatment should only be considered after 24 months of failed conservative management or when an obvious surgical lesion exists [16, 18].

Patellar alignment, patellar resurfacing, and patellar arthroplasty are the three principal categories of operative intervention for PFPS.

Currently there is no high-quality evidence supporting the treatment of PFPS with injectable therapies such intra-articular glucocorticoid, hyaluronic acid, platelet-rich plasma, glycosaminoglycan polysulfate, and botulinum toxin [19].

Return to Activities

Before returning to full activity, the patient should demonstrate equal range of motion and at least 80% strength to that of the uninvolved extremity. Mild pain that diminishes with activity is generally not concerning but severe or increasing pain should prompt termination of the activity and reassessment by a clinician. While recovering and awaiting returning to sport, athletes can maintain aerobic fitness with activities such as a stationary bicycle, upper body cycle, swimming, water running, or other painless exercises.

Idiopathic Chondromalacia Patellae

Chondromalacia patellae is often used synonymously with patellofemoral pain syndrome. However, there is consensus that patellofemoral pain syndrome applies only to individuals without cartilage damage. Chondromalacia patellae is defined as idiopathic articulate changes of the patella and may lead to anterior knee pain. The primary pain generator in this syndrome is not well understood and is thought to be multifactorial. The leading theory is the articulate changes along with roughening or damaging of the undersurface of the cartilage of patella lead to pain. Subchondral bone has weak potential to generate a pain signal. However, the anterior fat pad and joint capsule are sensitive areas with high potential for creating pain signals [20, 21].

Involved factors in chondromalacia patellae may include limb malalignment, muscle weakness, chondral lesions, and patellar maltracking. This pathology is more common in adolescent females.

Clinical Presentation

The patient will likely present with vague and diffuse knee pain in the peripatellar or retropatellar aspect of the knee, worsened with squatting, prolonged sitting, or ascending stairs. There may be pain on patellar compression when the knee is extended. Movie theater sign (pain with knee in flexed position for extended period of time) is also positive in these patients.

Anatomic characteristics to evaluate for in patients with suspected chondromalacia patellae include increased Q-angle, genu valgum, external tibial torsion, pronated feet or subtalus, and femoral anteversion.

Physical Exam

Findings are the same as those with PFPS, except these patients may also have palpable crepitus. Clark's test is a special test specific for chondromalacia. The examiner will compress the patella into the femur while the patient contracts their quadriceps. This maneuver is positive if there is a grinding sensation or pain.

Diagnostic Studies

Plain radiographs may show chondrosis, shallow sulcus, patella alta or baja, or lateral patella tilt. CT is indicated if there is suspicion of patellofemoral malalignment or fracture.

T2 weighted MRI is the best method to assess articular cartilage. Abnormal cartilage will appear with a higher signal compared to normal.

The Outerbridge Classification is used to describe the stages of degeneration. The first level is simple softening of the cartilage. Level two is classified by fibrillation of the hyaline cartilage, reflecting further degeneration. The third level is fissuring of the cartilage to the subchondral bone. The last and most severe level four refers to the area of bone devoid of articular cartilage covering [21].

Treatment

Conservative methods are the mainstay of treatment and should be trialed for a minimum of 1 year. In this condition, NSAIDs are superior to steroids. The patient should be advised to modify activities for relative rest and initiate a physical therapy program with focus on VMO, core, and hip external rotator strengthening, and closed chain short arc exercises. Orthotics may be used in patients with pronation of the foot. PRP (platelet-rich plasma) and prolotherapy may be used, but is not standard of care [22].

Surgery is indicated for patients who do not improve after 1 year of conservative management with extensive physical therapy or for patients with Outerbridge grade three or four. The patient may undergo an arthroscopic debridement or patellar realignment [21].

Return to Activities

Most patients recover in months to years depending on the severity of pain. Younger patients often achieve better long-term recovery.

Patellar Tendinopathy

Patellar tendinopathy, commonly known as "jumper's knee," is an overuse injury of the patellar tendon that develops after repetitive, forceful, eccentric contraction of the extensor mechanism [23].

This pathology of the knee was previously described as "tendonitis," which was a misnomer as the histology will show degeneration and microtears of the tendon, rather than inflammation [24, 25]. Inflammation is involved but is not the primary source of this pathology [26].

Clinical Presentation

Like PFPS, the patient will present with anterior knee pain. Most commonly, this will affect adolescent jumping athletes and males more than females, which distinguishes it from quadriceps tendinopathy that is more common in older adults. The pain will have an insidious onset. In addition to pain located in the anterior knee, it may be specifically localized to the inferior border of the patella. Movie theater sign may be positive, which is when the patient reports pain with prolonged knee flexion.

Patellar tendinopathy can be classified into four stages based on the Blazina et al. criteria [27]. Stage one is pain after sports or activity only. Stage two is pain at the beginning of sports or activity that disappears after warm-up, but reappears during fatigue. Stage three is constant pain at rest and with activity. Stage four is complete rupture of the patellar tendon [27].

Physical Exam

On inspection, there may or may not be swelling over the patellar tendon of the knee. The inferior border of the patella will be tender to palpation. Basset sign is a two-part provocative sign that can be used to identify patellar tendinopathy. The patient will be asked to flex and extend their knee. Basset sign is positive if the patient has no tenderness to palpation at the site of the patella insertion on the distal pole of the patella with full flexion, but does have tenderness when the knee is fully extended. This maneuver is relatively nonspecific [28].

Other factors identifiable on exam are intrinsic biomechanics of the knee joint. Individuals with patellar tendinopathy may also

have tight quadriceps, quadricep atrophy, hamstring tightness, weak VMO, increased Q-angle, patella alta, and genu varum or valgus [23].

Physical exam alone is unable to clearly diagnose patellar tendinopathy.

Diagnostic Studies

Plain radiographs will not usually reveal patellar tendinopathy but can rule out other potential causes of anterior knee pain. Recommended views are AP, lateral, and sunrise or Merchant. Merchant view can identify or rule out patellar subluxation [23]. In chronic cases, an enthesophyte, or inferior traction spur, can be present. Ultrasound will demonstrate thickening of the patellar tendon and hypoechoic areas within the tendon. However, ultrasound is user dependent [29]. MRI is indicated if the pain is chronic and without relief from conservative treatment, and for surgical planning. Findings on MRI include tendon thickening, increased signal on T1 images, and loss of the posterior border fat pad [30].

Treatment

Conservative treatment is first line to treat patellar tendinopathy, with the goal of returning the individual to their usual activity level. This includes a short period of relative rest and activity modification to reduce the repetitive stress on the patellar tendon, a short course of NSAIDs (no more than 14 days), and modalities, such as ice. In addition, the patient should participate in a formal physical therapy program with focus on quadriceps, hamstring, and core strengthening and stretching, in addition to eccentric exercises [23, 31]. Taping or a chopat's strap may be useful to reduce tension across the patellar tendon [32].

Sclerotherapy (also known as prolotherapy), iontophoresis, and extracorpeal shock wave therapy have few studies supporting their use. However, more research should be performed before recommending use of these modalities [33–35]. Steroid injections are contraindicated due to risk of possible patella tendon rupture [36].

Operative treatment is indicated in patients with symptoms refractory to conservative management or patients with Blazina stage III, stage IV, and partial tears. Open or minimally invasive surgery may be performed with the goal to resect the angiofibroblastic and mucoid degenerative area. Coleman et al. observed sympathetic benefit to a large percentage of patients regardless of approach type [37].

Return to Activities

Individuals who underwent conservative treatment may return to their sport once completing a physical therapy program, as described above, and when pain has become mild.

For patients who underwent operative treatment, there is a period of initial immobilization of the knee in extension, followed by progressive range of motion and mobilization exercises with weight bearing as tolerated. 80–90% of athletes will return to their sport. However, activity related aching can last 4–6 months postoperatively.

Patellofemoral Osteoarthritis

Knee osteoarthritis (OA) is a commonly diagnosed condition which can affect all three compartments of the knee. Up to 25% of cases involve the patellofemoral (PF) compartment and up to 40% of these cases are considered isolated PFOA [38]. PFOA typically results from the loss of articular cartilage of the patella and the trochlear groove from one or more of the following: overload, trauma, or instability. The lateral patellar facet is more often overloaded than the central or medial aspect of the patella so wear and tear of the lateral facet is most common. Incidence is higher in women and increases with age, primarily affecting those over 45 years of age. Additional risk factors include increased Q-angle, excessive hip anteversion, patellofemoral dysplasia, and history of patellar subluxation or dislocation [39].

Clinical Presentation

Patellofemoral osteoarthritis typically presents with anterior knee pain. Pain can be localized to the medial knee but this more commonly suggests medial-compartment tibiofemoral OA [40]. PFOA is usually bilateral but can be unilateral. The pain is aggravated by walking on inclined terrain, ascending/descending stairs, kneeling, squatting, and rising from a sitting position. Some patients report associated stiffness, locking, clicking, crackling, or crepitus from friction between exposed bones. Morning stiffness typically lasts less than 30 min. Widespread knee pain and distal radiation suggest moderate to severe knee OA. Persistent pain at rest or night pain interrupting sleep suggests more advanced OA [39, 41].

Physical Exam

Knee effusion is common though usually mild and correlates with severity of disease. Pain on palpation is usually localized to the patellar facets. Patellar grind test is often positive for crepitus [39].

Diagnostic Studies

Osteoarthritis in general may be diagnosed on clinical grounds alone with the presence of typical symptoms and risk factors. Additionally, it is important to note that patients with robust OA symptoms may have normal radiographs and vice versa. Diagnostic imaging should be considered, though, in the presentation of young patients, atypical pain location, severe pain at rest or night, severe locking or catching, weight loss, or constitutional symptoms. X-ray will show asymmetric narrowing of the joint space, osteophyte formation, subchondral sclerosis, and subchondral cysts. Isolated PFOA is diagnosed when characteristic findings are seen in the patellofemoral compartment on imaging and no other compartment of the knee is affected [39].

Treatment

Conservative treatment is the first line option for management of patellofemoral osteoarthritis. Activity modification should include avoiding stairs, squats, lunges, jumping, and impact sports. Physical therapy should focus on stretching and strengthening surrounding muscles such as quadriceps femoris. In obese patients, weight loss is helpful in reducing the load on the anterior knee. Evidence is lacking to promote the use of patella unload sleeves, braces, and taping. Pharmacotherapy with acetaminophen or non-steroidal anti-inflammatory drugs (NSAIDs) can be used for pain relief. If experiencing an acute flare or refractory PFOA, intra-articular injection of steroids or hyaluronic acid is recommended. Surgical intervention should only be considered after 3–6 months of failed conservative treatment [42].

For isolated PFOA, surgeons may recommend unicompartmental patellofemoral joint replacement, leaving the healthy medial and lateral compartments intact. Additional options include soft tissue realignment of the extensor mechanism (e.g., lateral retinaculum release), tibial tuberosity osteotomy to improve the Q-angle, and total knee replacement for severe or multicompartmental OA [39].

Return to Activities

While ceasing certain activities such as impact sports can be beneficial to patellofemoral osteoarthritis, patients should remain active during the recovery period. In addition to or following a physical therapy program, patients can gradually increase activity level provided it does not make symptoms worse. Patients can start with low-impact activities that do not stress the joint such as walking, bicycling, and water aerobics and increase from there to their desired activity level.

Prepatellar and Superficial Infrapatellar Bursitis

Bursitis refers to inflammation of a bursa, a synovial-lined fluid-filled sac which reduces friction between tissues of the body. The prepatellar and superficial infrapatellar bursae are commonly affected anatomical structures in the knee. Given their superficial location, these bursae are susceptible to microtrauma as well blunt and penetrating trauma. Septic bursitis can also occur secondary to local inoculation from trauma and accounts for nearly 1/3 of cases. After injury, synovial bursa cells thicken, granulation tissue and fibrous tissue develop, and the bursa fills with fluid [43, 44].

Clinical Presentation

Acute bursitis presents with a short history of localized pain, redness, warmth, and swelling at the anatomical location of the bursa. Chronic bursitis presents as a more indolent course, with pain and swelling elicited by prolonged periods of kneeling. Obtaining an accurate history upon presentation is critical to the diagnosis of prepatellar and superficial infrapatellar bursitis. Jobs requiring prolonged kneeling such as clergy, plumbing, gardening, etc. and sports with frequent direct blows to the knee such as wrestling are known as risk factors [45]. Nearly 80% of all cases occur in men aged 40–60 years. Chronic bursitis can also be due to gout, rheumatoid arthritis, or indolent infection [43, 44].

Physical Exam

Examination of acute bursitis may reveal localized tenderness, erythema, and edema. There may be evidence of skin abrasion or a puncture wound overlying the bursa. Knee effusion is observed in approximately 30% of cases. Chronic bursitis, however, presents as a soft, fluid-filled globular mass in front of the patella or the patellar tendon with typically little to no tenderness. Mild skin inflammation may be present. Strength and range of motion are often unaffected by bursitis but may be limited secondary to pain, particularly in the acute setting [43].

Diagnostic Studies

Prepatellar and superficial infrapatellar bursitis can be diagnosed with clinical history and physical examination alone but diagnostic testing can be helpful in determining the etiology. Often a blood cell count with differential, serum glucose, and serum uric acid are obtained. If there are signs of infection, blood cultures are also obtained. This underscores a key component of evaluation which is the exclusion of septic bursitis. Patients with severe knee pain, limited range of motion, or inability to beat weight should undergo aspiration of the inflamed bursa for evaluation of septic bursitis. Patients with symptoms such as fever, chills, night sweats, tachycardia, or tachypnea should undergo a full workup for sepsis [43]. Imaging studies of the knee are rarely necessary for cases of prepatellar and superficial infrapatellar bursitis, though the soft tissue edema can be detected on MRI [43, 44].

Treatment

Excluding gout and septic bursitis, most cases are self-limiting. Chronic microtraumatic is treated conservatively with addressing the underlying cause [43]. In cases where the damaging activity such as kneeling cannot be avoided, protective knee braces or pads are encouraged [46]. Oral or topical NSAIDs can be used for pain relief as needed. Glucocorticoid injections are not recommended for chronic bursitis due to increased risk of infection [43, 44]. Septic bursitis is managed with antibiotics, knee splint immobilization, and repeated aspiration if fluid reaccumulates [47]. Patients with bursa aspiration identifying monosodium urate crystals should be treated for gout. If conservative measures fail to resolve prepatellar and superficial infrapatellar bursitis, bursectomy is an effective treatment [44, 48].

Return to Activities

Patients should avoid activities that increase pain or inflammation but can re-incorporate activities of daily living provided they do

not cause trauma to the bursa. As mentioned above, patients in high risk occupations and those who cannot avoid activities such as kneeling can return to work/sport with the use of protective knee braces or pads. Traumatic effusions in general tend to resolve slowly over weeks to months. In cases requiring surgical bursectomy, 80% of patients have complete resolution of symptoms and return to pre-injury activity level.

Lateral Patellar Compression Syndrome

Lateral patellar compression syndrome (LPCS) is a disorder of overload and increased pressure on the lateral facet of the patella due to pathologic lateral soft tissue restraints and improper tracking of the patella in the trochlear groove. LPCS is usually caused by adaptive tightening and shortening of the lateral retinaculum. This leads to lateral tilt of the patella and chronic stress imbalance between the medial and lateral articular surfaces. The increased lateral stress on the patella leads to patellar maltracking, patellar cartilage injury, patellofemoral joint pain, and in the long-term osteoarthritis. Because pressure over the lateral patellar facet is increased with knee flexion, LPSC is sometimes referred to as lateral patellar pressure in flexion (LPIF) [49–51].

Clinical Presentation

LPCS typically presents with localized dull anterior knee pain. The pain is aggravated by activities that stress the patellofemoral joint such as climbing stairs, squatting, and prolonged sitting with flexion of the knee. Patients deny instability or crepitus [51, 52].

Physical Exam

Physical exam findings associated with LPCS include pain with compression of the patella, tenderness to palpation of the lateral facet, and inability to evert the lateral edge of the patella [51, 52].

Diagnostic Studies

The primary imaging modality to diagnose LPCS is X-ray. Sunrise knee radiographs often show patellar tilt in the lateral direction. However, the detection rate of patellar tilt by measuring the congruence angle (CA) and patellar tilting angle (PTA) is approximately 70–80% [53].

Treatment

Initial management of LPCS is conservative with NSAIDs, activity modification, and physical therapy focusing on quadriceps stretching and strengthening. Physical therapy should emphasize vastus medialis strengthening and closed chain short arc quadriceps exercises.

Operative intervention is indicated in cases refractory to a comprehensive rehabilitation program. No consensus exists on the gold standard surgical treatment for LPCS but arthroscopic lateral patellar retinaculum release (LPRR) is the most widely used in clinical practice. Other options include patellar realignment, extension of lateral patellar retinaculum or lateral patelloplasty [49, 54].

Intra-articular injection of hyaluronic acid can result in improvement of knee pain and functional outcomes after LPRR in patients with degenerative cartilage changes secondary to LPCS [52].

Return to Activities

Most patients successfully return to activity with conservative management. After arthroscopic lateral patellar retinaculum release though, patients are placed on knee immobilizer, educated on active range of motion and quadriceps strengthening exercises, and allowed to bear weight as tolerated with crutches for 2–4 weeks. The knee immobilizer is then removed when the patient demonstrates sufficient quadriceps control [51].

Patellar Instability

Patellar instability is defined as patellar subluxation or dislocation episodes because of injury, ligamentous laxity, or increased Q-angle. This condition is most common in young patients with patellar malalignment, Ehlers Danlos, or other ligamentous laxity. Osseous factors involved are patella alta, trochlear dysplasia, excessive lateral patellar tilt, and lateral femoral condyle hypoplasia [55].

Clinical Presentation

These patients will present with complaints of anterior knee pain associated with feelings of instability. The classification is based on whether the etiology was acute, chronic, or habitual. Traumatic or acute etiology is equal in male and female gender, and will occur from a direct blow, such as a helmet to knee collision [55, 56]. Chronic cases of patellar instability are more common in women with malalignment of the patella. These patients tend to have recurrent subluxation episodes.

Physical Exam

On inspection, a large hemarthrosis will be present if the etiology of the patellar instability is an acute process. The patient will have medial sided tenderness and increase in passive patellar translation. Lateral translation of the medial border to the lateral edge of the trochlear groove is considered an abnormal amount.

Patellar apprehension test, or passive lateral translation, is positive when it results in guarding. The patient may also have a positive J sign, which is excessive lateral translation in extension, popping the patella into the groove as the patella engages the trochlea early in flexion [57].

Diagnostic Studies

With chronic patellar instability, diagnosis is made with passive patellar translation and a positive J sign. Plain radiographs can be used to rule out fractures or loose bodies. The medial patellar facet is the most common site of fracture. Lateral X-ray can assess for patellar alta or baja and for signs of trochlear dysplasia, including flattened trochlear groove, hypoplastic medial condyle, and supratrochlear spur. Sunrise or merchant X-ray views can assess for lateral patellar tilt and sulcus angle. CT would be useful to assess tibial tubercle–trochlear groove (TT–TG) distance and MRI may rule out suspected loose bodies [57].

Treatment

Patients with first time dislocation without bony avulsion or presence of loose articular bodies should be treated with bracing, NSAIDs, activity modification, and physical therapy. These patients should undergo a short-term immobilization with a patellar stabilizing sleeve or J brace for comfort, then 6 weeks of controlled motion with emphasis on strengthening. Physical therapy should include closed chain short arc exercises, and quadriceps, core, and hamstring strengthening.

Operative treatment is indicated in patients with recurrent patellar instability and includes arthroscopic debridement with removal of loose bodies. Recurrent dislocation with nonoperative treatment is approximately 15–50% at 2–5 years, and recurrence rate is highest for those with a primary dislocation who are less than 20 years old [57].

Return to Activities

Patients undergoing nonoperative treatment may return to activities when asymptomatic or with mild symptoms after completion of a physical therapy program. After surgery, a patient must be cleared by their surgeon prior to returning to activities.

Patellar Tendon Rupture

Patellar tendon rupture is rare and caused by tension overload during activity in a patient at risk. It is more common in the quadriceps tendon than the patellar tendon. The mechanism is overload of the extensor mechanism of the knee through sudden quadriceps contractions with the knee in a flexed position, such as missing a step on the stairs, or jumping. Most ruptures occur with the knee in the flexed position, as the greatest forces on the patellar tendon are with knee flexion greater than 60°. The most common type of rupture is avulsion from the proximal insertion or inferior pole of the patella. The rupture can also occur midsubstance or from a distal avulsion on the tibial tubercle [58, 59].

Clinical Presentation

This pathology is most common in males in their third and fourth decade of life. Usually the injury occurs with a jumping event involving sudden quadriceps contraction in the knee flexed position. Patients will usually report feeling or hearing a pop and then immediate swelling, difficulty weight bearing, and infrapatellar pain. There are many risk factors for patellar tendon rupture. The collagen structure of the tendon can be weakened due to systemic disease, such as diabetes, lupus, rheumatoid arthritis, chronic kidney disease, or local injury from patellar degeneration, previous injury, or patellar tendinopathy [59, 60]. Corticosteroid injections in the area can also place individuals at risk for rupture.

Physical Exam

On inspection, patients with patellar rupture will have patellar elevation with large ecchymosis and/or hemarthrosis, localized tenderness to palpation below the patella, and a palpable gap between the inferior pole of the patella and the insertion site on the tibia. There will be reduced range of motion and difficulty

weight bearing of the affected knee. If only the patellar tendon is ruptured but the retinaculum is intact, then active extension is possible, but with extensor lag of a few degrees [61].

Diagnostic Studies

Physical exam and plain radiographs can confirm a full thickness tear. On X-ray, the patella will be elevated. MRI is the most sensitive modality and is required to diagnose partial tears or differentiate a partial from complete tendon rupture. Ultrasound can also be used to identify partial and full thickness ruptures (Fig. 5.1) [58, 61].

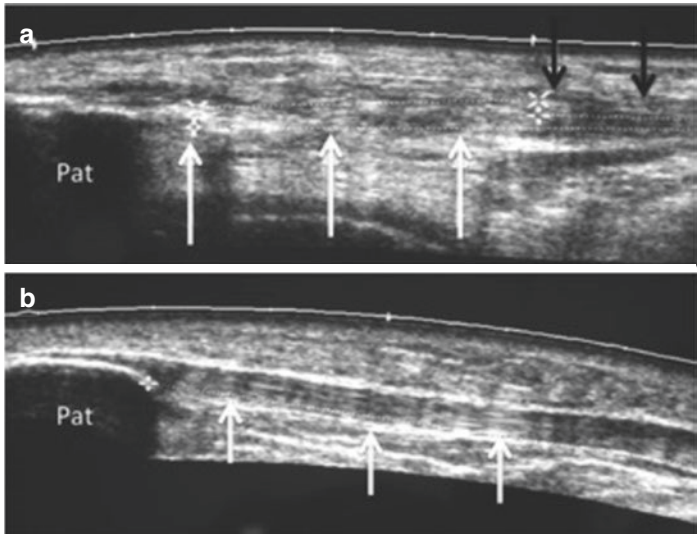

Fig. 5.1 Ultrasound of a normal patellar tendon and a chronic patellar rupture. (**a**) The proximal patella (Pat) is visualized on the left side of the image. The hypoechoic region (white arrows) indicate development of scar tissue. The remaining distal tendon is visible (black arrows). (**b**) Ultrasound image of a normal patellar tendon. (Source: Magnussen R.A., Demey G., Archbold P., Neyret P. (2014) Patellar Tendon Rupture. In: Bentley G. (eds) European Surgical Orthopaedics and Traumatology. Springer, Berlin, Heidelberg. https://doi-org.eresources.mssm.edu/10.1007/978-3-642-34746-7_137)

Treatment/Return to Activities

In patients with an intact extensor mechanism and partial tear on MRI, treatment begins with immobilization in full extension for 6 weeks with weight bearing in a hinged knee brace. After this 6 week period, rehab is a progressive active flexion and passive extension protocol [61].

Complete tears require timely operative repair. The most important prognosticating factor is timing of the repair of the tendon. Post-operative rehabilitation will begin after 6 weeks with a locked extension brace. Patients can immediately weight bear and then may undergo early motion protocols at 7–10 days post-op with a focus on passive extension and active flexion [58].

Patella Fracture

The patella is a trabecular bone with articular cartilage on the posterior aspect and is susceptible to fracture as it is superficial and unprotected by overlying soft tissue. As discussed, the patella is an important part of the extensor mechanism of the knee. The mechanism of fracture is usually direct trauma onto the anterior aspect of a flexed knee or a direct impact of an extended knee. When the knee is flexed on impact, the result is usually a complex comminuted fracture from impact and compression against the femoral condyles and trochlea. Impacts on the patella while extended usually cause nondisplaced fractures. Rarely, the extensor mechanism can result in an avulsed portion of the patella, more common in pediatric or athletic cohorts.

Clinical Presentation

The patient will usually present after a fall or direct trauma to the knee. The extensor mechanism may still be intact depending on the severity and location of the fracture. Often, there will be painful swelling of the knee and fractures may be palpable on exam.

Diagnostic Studies

Plain radiographs can confirm the diagnosis and analyze the fracture pattern. Fractures visualized may be classified in one of seven patterns—nondisplaced transverse fractures, displaced transverse fractures, longitudinal, or vertical, fracture, comminuted nondisplaced fracture, comminuted displaced fracture, osteochondral fracture, avulsion (sleeve) fracture (Fig. 5.2). These fractures were further classified by Duparc et al. into three types. Type I is

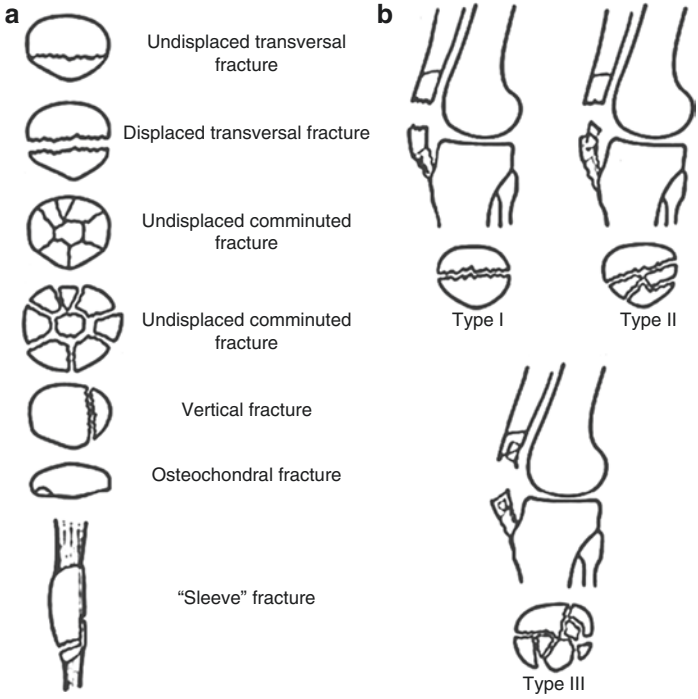

Fig. 5.2 (**a**) Morphologic classification of patellar fracture. (**b**) Duparc classification of patellar fractures. (Source: Weppe F., Demey G., Fary C., Neyret P. (2014) Fractures of the Patella. In: Bentley G. (eds) European Surgical Orthopaedics and Traumatology. Springer, Berlin, Heidelberg. https://doi-org.eresources.mssm.edu/10.1007/978-3-642-34746-7_230)

a transverse fracture located between the proximal two thirds and the distal one third. The etiology is usually pure knee flexion with a strong quadriceps muscle contraction. Type II is a transverse fracture with a comminuted distal fragment. The trauma etiology is a fall on a flexed knee causing sagittal compression of the distal pole of the patella, which compresses the patella against the condyles. Type III is the "stellate fracture," in which the patella fragment is comminuted. The mechanism is violent anterior top posterior compression, commonly seen when a knee undergoes a trauma against a solid object, just as a car dashboard (Fig. 5.2).

Treatment/Return to Activities

Nonoperative treatment, including physical therapy, is indicated if the extensor mechanism is preserved, the articular step off is less than 1 mm, and the fracture is stable. Mobilization should be initiated as early as tolerated. During the first 3 weeks post-injury, the patient should range the knee between 0° and 60°. After 6 weeks, the patient can begin to exceed 90° of flexion. Between physical therapy sessions, the patient should have the knee splinted at 30° to avoid patella baja. Weight bearing is allowed if the knee is in a full extension splint. Monitoring by plain radiograph should be performed at 1 week, 2 weeks, and 6 weeks to ensure no secondary displacement. Operative management is indicated if the extensor mechanism is not intact [62].

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Pediatric Knee Pain

6

Esha Jain and Elinor Naor

Introduction

Pediatric knee pain can be divided into several subcategories including life-threatening conditions, inflammatory conditions, traumatic injuries, and congenital abnormalities. The presentation of pediatric knee pain can often be variable, however, an extensive history of the presentation can be extremely helpful in determining the diagnosis. The components of presentation can be divided into systemic and localized symptoms. Systemic symptoms can clue the provider into whether the diagnosis is from an acute condition, such as an infection, or chronic condition, such as malignancy or rheumatologic condition. Localized symptoms can be present on different aspects of the knee which can further reveal the specific location of injury within the knee. This can then dictate diagnostic steps to help determine the specific pathology

E. Jain
Department of Physical Medicine and Rehabilitation, Icahn School of Medicine at Mount Sinai, Mount Sinai Health System, New York, NY, USA

E. Naor (✉)
PGY-3—Department of Physical Medicine and Rehabilitation, Icahn School of Medicine at Mount Sinai, Mount Sinai Health System, New York, NY, USA

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including laboratory studies and imaging. This will dictate treatment as well as rehabilitation to return to daily life activities.

Life-Threatening

In terms of life-threatening episodes, conditions that are life threatening include septic arthritis, osteomyelitis, and malignancies [1].

Septic Arthritis

What Is the Pathology, How, Why?

In the USA, septic arthritis can affect 1 in 100,000 children and has recently increased likely due to improvement in diagnostic technology. It is more common in males under 4 years of age and is found in the knee 37–54.5% of all cases [2]. Septic arthritis is an emergent condition in children that most commonly occurs in the knee [3]. Specifically, septic arthritis is an infection of the joint that can lead to destruction within hours of hematogenous inoculation. When bacterial pathogens are seeded into the blood it can quickly spread through the body's vessels and cause an extreme inflammatory response. This response causes high cytokine release which causes metalloproteinases and collagen-degrading enzymes to inflict great damage on joints. This process in combination with the bacterial toxins and lysosomal enzymes causes further destruction to the joint. To add, this condition leads to increased joint space pressure that can lead to avascular necrosis if untreated. Common bacterial pathogens include *Staphylococcus aureus* and *Streptococcus pneumoniae*. In children that are unvaccinated, *Haemophilus influenza* should be considered. In adolescents, *Neisseria gonorrhoeae* should be considered, and in children with sickle cell disease, Salmonella should be considered [4].

Clinical Presentation

Children with septic arthritis will present with acute onset of pain, very limited movement of the extremity, and fever. It is very com-

mon for the child to start limping, have signs of an effusion, and have swelling and warmth. In addition, it is typical for the child to hold their leg in a position that is most comfortable. For example, the child may hold the hip and knee in a flexed position, abducted, and externally rotated. Any movement will be painful to the child. Some children may present with the inability to bear weight and high fever [5].

In children, symptoms may present several days prior to presentation and start with generalized symptoms such as poor appetite. Some children may report injury to the affected knee, preceding illness, travel exposure, and missing immunizations [2, 6].

Physical Exam

On a physical exam, there may be swelling and effusion of the affected knee joint. There will be tenderness to palpation and warmth. In terms of physical exam maneuvers, active and passive knee range of motion should be examined. Skin findings, how the child is holding the knee, and the position that the knee is in are all clues in the physical exam that aid in diagnosis [2].

Diagnostic Studies

Initially, laboratory work up should include white blood cell count, differential cell count, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and blood cultures [5]. CRP is very helpful as compared to ESR as it may remain elevated. Lyme titers may also be beneficial to rule out lyme arthritis. In arthrocentesis, more than 50,000 white blood cells and more than 90% polymorphonuclear leukocytes are suggestive of septic arthritis. Based on clinical history culture specific media can be obtained [2].

Radiographic X-rays should be ordered of the involved knee joint and the surrounding bones to exclude other pathology such as osteomyelitis and malignancy. If possible, a bedside ultrasound should be obtained to detect a joint effusion. Immediately, with signs concerning for septic arthritis, an orthopedic surgeon should be consulted. In the emergency department, a synovial joint aspiration can be collected. This sample can then be sent for gram stain, cultures, and cell count [5].

Treatment

Antibiotics should not be administered until joint aspiration is taken. If septic arthritis is suspected and diagnosed, surgical drainage of the knee may be required. Therefore, a patient may urgently need to be transported to the operating room to wash out the infected space. Once this procedure is performed, empiric intravenous antibiotics are started. Once cultures and sensitivities result, antibiotics are adjusted, and the child is transitioned to oral antibiotics [5].

For empiric treatment, the provider should try to cover with penicillin or a first-generation cephalosporin to be effective against MSSA, *Streptococcus pyogenes*, and *Kingella kingae*. If a child is allergic to penicillin, then clindamycin should be used instead. MRSA coverage is not needed initially unless something in the history of the patient requires it. In terms of duration, studies have shown that there is no significant difference between 7.4 days versus 18.6 days in treatment. Another study, a randomized control study, showed no significant difference between 10 and 30 days of antibiotics [2].

In addition to antibiotics, some studies have shown the benefit of the use of corticosteroids. In one study, Folgel and colleagues found that a 4-day course of dexamethasone with antibiotics had better outcomes in terms of shorter fever, inflammatory, pain, and IV antibiotic duration [7]. In a Cochrane review to determine the advantages and disadvantages of corticosteroids, they found that corticosteroids improve pain and function [8]. However, this evidence is limited as there are no studies looking specifically at the knee [2].

Return to Activities

Prognosis of the disease is related to age the timing of acute surgical intervention and antibiotics. In other words, the quicker the diagnosis and treatment, the better the functional outcome [9]. In a study that looked at outcomes preparing arthroscopic and open methods for septic arthritis treatment, authors found that children with knees that underwent arthroscopy had faster recovery in terms of range of motion and weight bearing [10].

Osteomyelitis

What Is the Pathology, How, Why?

Osteomyelitis is a condition that similarly rises to septic arthritis through bacterial seeding through blood. In children, it can arise from otitis media, pharyngitis, and daily activities. This condition typically arises in the metaphyseal region of long bones where bacteria can gather in a highly vascularized area. Once an infection has seeded, it can quickly spread to other parts of the bone and adjacent joint spaces. It is important to note that osteomyelitis can occur and predispose the joint space to septic arthritis, at a rate of 33% [11].

Acute osteomyelitis in pediatric patients occur through occult bacteria spreading through the blood and a susceptibility in the bone that allows bacterial invasion. Similarly to septic arthritis, *Staphylococcus aureus*, MSSA, and MRSA are the most common organisms responsible for infecting the bone. Another organism, although less common, is *Kingella kingae*. The incidence of osteomyelitis in children is estimated to be about 10–80 in 100,000 children. In terms of bones, about 25–40% of osteomyelitis cases occur in the knee. This is followed by the hip and ankle [12].

Clinical Presentation

In terms of presentation, children may or may not be able to identify the specific site in the lower extremity that is painful. Some children will present with vague symptoms such as fever, pain with weight bearing, and non-specific history. It is important to ask about recent injury, trauma, travel history, sick contacts, immunization history, and pet exposure [12].

Physical Exam

In a possible case of osteomyelitis, the physical exam can be difficult due to pain but is extremely helpful in diagnosis. In some children, there may be point tenderness, warmth, and swelling. Patients will likely have fever, tachycardia, tachypnea, and

hypotension. If the child cannot speak, he or she may appear fussy with some adenopathy. If the patient cannot bear weight, use an extremity, or have their hip flexed and externally rotated, it may indicate osteomyelitis. If the child is in significant pain, it may be difficult to perform a thorough physical exam. However, if possible, it is helpful to test strength, reflexes, and sensation as it may aid in the diagnosis, especially when symptoms are vague [6, 12].

Diagnostic Studies

For a diagnosis suspicious of osteomyelitis, it is very important to obtain tests like those for septic arthritis, including a CBC, ESR, CRP, and blood cultures, prior to antibiotics. Kocher and colleagues found that patients with the criteria of fever, refusal to bear weight, leukocyte count of greater than 12,000, and ESR greater than 40 has a diagnostic sensitivity of 93–99%. In addition, providers can obtain serologies for specific bacteria, stool cultures, and joint fluid if possible.

Radiographic imaging is recommended, however, its yield is limited as 50% of bone mineral loss must be present for findings to show. More than X-ray, MRI is the most sensitive in seeing bone and soft tissue abnormalities. Ultrasound can also be very helpful in terms of speed and management. In children, because the risk of bacteremia is higher, it is important to not delay treatment due to imaging, especially if all signs and symptoms are present [12].

Treatment

Treatment of osteomyelitis is in two modalities: surgical intervention and antibiotics. In terms of surgical treatment, it is highly considered when there is a positive culture, need for source control, and need for preserving function. To prevent permanent damage such as avascular necrosis, surgery, if indicated, cannot be delayed.

In terms of antibiotics, empiric antibiotics include first-generation cephalosporins and penicillinase-stable penicillins.

Another alternative medication is clindamycin which can be used as an alternative medication for infection of the bone [12].

Return to Activities

Osteomyelitis can result in several complications such as damage to the growth plate or avascular necrosis. However, for the most part, patients are diagnosed and treated quickly. A total duration of therapy is still an area of study, however, children can likely return to activities several weeks after starting treatment [12].

Malignancies

Malignancies include but are not limited to osteosarcoma, Ewing sarcoma, primary bone lymphoma, soft-tissue malignancy, and leukemia [13]. Many malignancies that arise in bone affect adolescents in the time of growth spurts and thus can present as chronic pain that is worse at night or with activities [14].

Osteosarcoma

What Is the Pathology, How, Why?

Osteosarcoma is one of the most frequent malignancies in children that develop in bone and thus will be discussed in the next section. Its incidence is around 8 to 11 million per year in children between 15 and 19. Males are more affected than females. In terms of pathogenesis, rapid bone proliferation during puberty will incite growth in the cancer. Radiation may contribute to development of osteosarcoma. More often, osteosarcoma risk increases with heredity mutations in a tumor suppressor gene [15].

Clinical Presentation

Osteosarcoma is one of the most common malignant tumors that form in the knee of children. In terms of presentation, it will rap-

idly grow during puberty [16]. Children will often present with localized pain and limitation of movement of the joint, in this case, the knee. About 50% of osteosarcoma will be around the knee, specifically at the distal end of the femur or the proximal end of the tibia. Sometimes a fracture or shortness of breath can be seen as a first symptom, depending on the extent of the malignancy and its spread [6, 15].

Physical Exam

If the child has tenderness on palpation, difficulty with ambulation, or pain with passive and active range of motion of the knee, then osteosarcoma or malignancy of the knee can be on the differential diagnosis. Other systemic symptoms such as fatigue, weight loss or lack of growth, low-grade fever may signify malignancy over other pathology. Unfortunately, there are no specific exam maneuvers to identify pathology as malignancy [15].

Diagnostic Studies

In terms of imaging, on plain X-ray, osteosarcomas have mixed osteoblastic and osteolytic lesions. There may be areas of aggressive lesion growth that extend the periosteum off the bone, forming what is known as a Codman triangle. MRI is more detailed when evaluating tissues, vessels, and nerves and would be beneficial when diagnosing and directing management. If malignancy is suspected, a CT scan would be warranted to look for metastasis. In addition, laboratory work up includes lactate dehydrogenase, alkaline phosphatase, and blood count. If diagnosis is suspected and treatment is possible, pediatric patients may require baseline work up of their other organ systems to measure tolerance of chemotherapy, radiation, or surgery. The diagnosis then must be verified with biopsy. On biopsy, if proliferation of malignant mesenchymal cells are found with bone cells, this can confirm osteosarcoma [15] (Fig. 6.1).

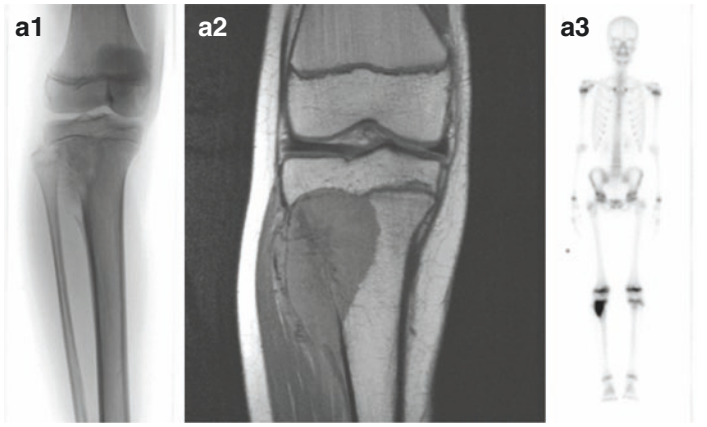

Fig. 6.1 (**a1**) Osteosarcoma on the lateral side in the proximal part of the tibia. Characteristic is the osteolysis with destruction of the cortex, subperiostal reaction (onion peel), Codman triangle and a Motheaten-away part. (**a2**) MRI image. (**a3**) Bone scan [17]

Treatment

In children, if left untreated, osteosarcoma will spread to the lungs. In about two thirds of patients, if treated with induction chemotherapy, surgery, then postoperative chemotherapy over the course of 6–8 months, remission can be achieved. Chemotherapy agents used include doxorubicin, cisplatin, methotrexate, and ifosfamide. Immunomodulatory agents include alpha-interferon and muramyl tripeptide phosphatidyl ethanolamine (MTP). Patients that have surgery or chemotherapy without remission may benefit from radiation. With surgery, the goal is to completely remove the tumor. Further surgery may be required for reconstruction or placement of a prosthetic device. Unfortunately, despite surgery, recurrence of osteosarcoma can be high [15]. In the past, surgeons would perform amputations to save the child from recurrence or metastasis. Currently, surgeons are employing "limb saving surgeries" which require extensive rehabilitation after [18].

Return to Activities

Physical and occupational therapy programs should be tailored to the patient and location of tumor. After resection, having a rehabilitation team that includes therapists, lymphedema specialists, and prosthetists or orthotists are appropriate to comprehensively rehabilitate the leg [19]. There is limited assessment and data on pediatric survivors. Outcomes vary based on the extent tumor growth, chemotherapy, and surgery. Therefore, there is no standard of care in regards to return to activities. In general, patients should have continuous surveillance every 3 months in the first year [20].

Inflammatory Conditions

There are certain inflammatory conditions that also cause pediatric knee pain including inflammatory and infectious arthritis. Inflammatory arthritis includes juvenile idiopathic arthritis and other systemic inflammatory conditions that can cause arthritis. Infectious arthritis can include Lyme and septic arthritis as discussed above.

Inflammatory and Systemic—Juvenile Idiopathic Arthritis (JIA)

What Is the Pathology, How, Why?

JIA is the most common rheumatologic disorder of childhood that can occur before age 16 and have an unknown cause. It is divided into several subtypes including systemic arthritis, oligoarthritic, rheumatoid positive and negative, psoriatic arthritis, and undifferentiated arthritis. However, the latter two tend not to affect larger joints such as the knee [21]. The pathology is not completely understood, but it is thought to be an immune mediated reaction where immune cells enter a new vascularization process. Then, host cells such as fibroblasts, chondrocytes, and osteoclasts mediate cartilage and bone destruction. In addition, immune cells release cytokines that cause inflammation in the area [22].

Clinical Presentation

In systemic arthritis, children will present with fever of at least 2 weeks with rash, hepatomegaly or splenomegaly, lymphadenopathy, or serositis. The arthritis in the child could be symmetrical and in multiple joints. In children with oligoarthritis, they will present with arthritis that affects four or fewer joints and with the knee mostly affected. In rheumatoid positive and negative polyarthritis, five or more joints will be positive. Typically, children will present with small joints first, then larger joints such as the knee [21].

Physical Exam

Vitals should be checked to see if the patient has a fever. Patients should be assessed for a rash, lymphadenopathy, and any visible signs of joint damage. Each joint should be looked at and reexamined in follow-up appointments [23]. Hansmann and colleagues compared children with unilateral knee joints with JIA to their opposite non-affected knee. They found the active range of motion during flexion and extension of the knee was significantly lower than the unaffected knee. Therefore, measuring and comparing the affected versus the unaffected knee can be very beneficial in physical exams [24].

Diagnostic Studies

Laboratory studies include complete blood count, ESR, CRP, and iron studies. ANA should be checked in presentations of oligoarthritis as 70–80% will be positive. If rheumatoid positive or negative subtype is suspected, titers of rheumatoid factor should be checked [21]. Recent studies are looking into different markers of JIA that may aid in its diagnosis. For example, Hilbert and colleagues found that high levels of "TRAP5b, NTX-1, and collagen" may be present compared to children without JIA [25].

Treatment

In terms of management, patients can benefit from NSAIDs such as naproxen, ibuprofen, and indomethacin. In patients with systemic JIA, corticosteroids could be used but should be reserved or

used a last resort due to potential endocrine side effects. Similarly, methotrexate has become a second line choice due to its side effect profile [21]. Overall, the standard treatment for children with JIA is anti-inflammatory medication and physical therapy. Some studies have looked at open and arthroscopic synovectomy and found that in patients with oligoarthritis JIA have early mobilization, shorter hospitalization, and good range of motion of the knee joint [26].

Return to Activities

Children diagnosed with any type of JIA will require frequent follow-up visits because throughout their lifetime they have periods of flare up of the disease. This can cause both physical and psychological distress. In general, children with JIA have been found to engage in less physical activity compared to their peers. However, this perhaps is changing with new medications and rehabilitation. Although there is no strong evidence for long-term outcomes from exercise training, children with JIA can participate in activities to try to decrease pain, improve their range of motion, and knee strength. This can include strength training, rope jumping, swimming, and general physical therapy [27].

Lyme Arthritis

What Is the Pathology, How, Why?

Lyme arthritis is caused by *Borrelia burgdorferi* and is very common in North America. B. burgdorferi can reside in tick saliva, and through a tick bite will bind to human cell receptors. Then, *B. burgdorferi* will reach the joint through the blood to the synovium causing arthritis. In some Lyme arthritis, molecular mimicry is thought to play a role [28].

Clinical Presentation

Often, Lyme arthritis will have early and late manifestations that make up the clinical presentation. In terms of early manifestations, symptoms may be self-limited and have no lasting effects.

In late manifestations, the infection is chronic and leads to permanent effects. In children, arthritis of the knee occurs in two thirds of patients. In the start of the disease, patients may complain of episodic arthritis that progresses to prolonged arthritis. It may even progress to multiple joints with eye involvement. Other key points in the presentation may include a report of a tick bite, rash, arthritis of the knee, myalgia, and age [28].

Physical Exam

Physical exams may vary; however, children will usually present with joint swelling, limited range of motion, and limping. Most patients will complain of morning stiffness as well as prolonged duration of symptoms. Therefore, a thorough musculoskeletal exam with gait observation is important.

Diagnostic Studies

Laboratory workup aids in the diagnosis of Lyme arthritis. This includes polymerase chain reaction (PCR) to detect *Borrelia*. Specific antibodies can also be found through enzyme immunoassay, immunofluorescence, hemagglutinin, and western blotting [28].

Treatment

Antibiotics are used for the treatment of Lyme and type will depend on how the disease is manifesting. In Lyme arthritis the choice is more difficult. In children, if treatment with ceftriaxone fails, then cotrimoxazole can be used. At least two antibiotic regimens should be used to treat before the diagnosis should be questioned. In children under nine, if they have erythema migrans, they should be treated with amoxicillin. In children above nine, they should be treated with doxycycline. For those with penicillin allergies, macrolides can be used [28].

Return to Activities

Some patients may develop chronic arthritis despite antibiotic treatment. Although not completely understood, this may be due to gender, age over ten, and steroids before antibiotic treatment [28].

Congenital or Developmental Abnormalities

When approaching pediatric patient with knee pain it is important to consider several anatomical differences from adults. One of the most significant differences is the growth plate and epiphysis. The physis is a particularly weak point in developing bones. Therefore, ligamentous injury in skeletally immature athletes may also be accompanied by fracture. Chronic knee pain will typically have a progressively worsening nature in children [29].

Osgood–Schlatter Disease

What Is the Pathology, How, Why?

Osgood–Schlatter disease first described in 1903, also known as tibial tubercle osteochondritis, is an injury affecting the point of insertion of the patellar tendon. The kinetic chain of the extensor mechanisms is formed by the quadriceps, quadriceps tendon, patella, and the patellar tendon. The tibial tuberosity apophysis is a secondary ossification center and is therefore in children the weakest point along the kinetic chain [30]. This injury is caused by traction on a tendon severe enough to affect the cartilage of secondary ossification centers. Osgood–Schaltter is typically a result of repetitive extension contraction done when playing sports that involve running and jumping or when suffering a fall on a flexed knee [31].

Clinical Presentation

Patients usually present with gradually worsening anterior knee pain that is localized to the tibial tubercle. This condition affects both boys and girls of growing age (8–15 years old). Typically, this condition affects males more than females [31]. Patients will usually deny hip pain, knee instability, or edema [30]. Young athletes are affected by this condition 20% of the time versus non-athletes who are affected 5% of the time. Patients will present with bilateral symptoms in 25–50% of cases. Pain is notably worse with kneeling, running, jumping, squatting, climbing stairs, or walking uphill. Patients report pain relief at rest [32].

Physical Exam

On physical exam patients have pain to palpation of their tibial tubercle. Some may experience pain with resisted knee extension [31]. Patients will also experience pain when hopping on the affected leg or when squatting [30]. In patients experiencing an acute exacerbation there maybe be swelling and warmth appreciated along the tibial tubercle. Of note, the range of motion of the knee is typically unaffected [32].

Diagnostic Studies

If Osgood–Schlatter is suspected after completing a focused history and physical exam, a plain AP and lateral X-ray is often sufficient to confirm the diagnosis. Radiographs will show the tibial tubercle fragmented on the lateral view (this can be a normal variant). In addition, X-rays may show ossicle formation. Tibial tubercle osteochondritis is visualized on MRI as patellar tendonitis (Fig. 6.2) [31].

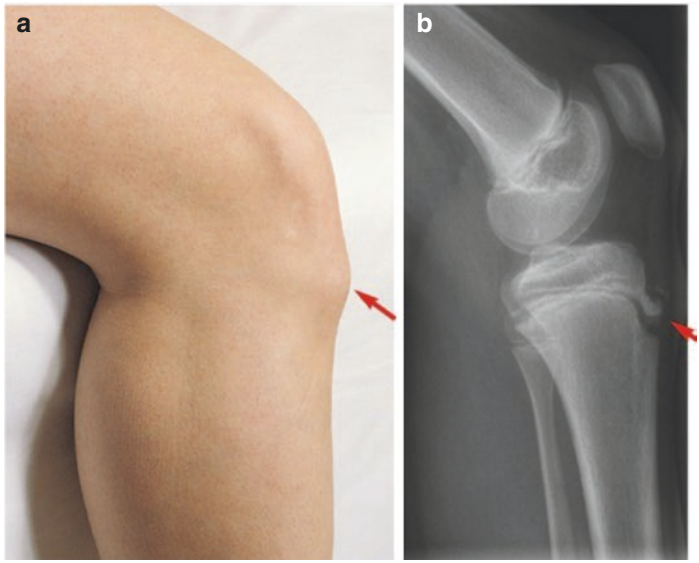

Fig. 6.2 Osgood–Schlatter disease. (**a**) Swelling around the tibial tuberosity (arrow). (**b**) Ossification disorder of the apophysis of the tibial tuberosity (arrow) [17]

Treatment

Osgood–Schlatter disease is typically treated conservatively. Patients should be instructed to perform activity as tolerated and treat their pain using ice and NSAIDs. Some patients may benefit from infrapatellar strap and protective padding with activity. Home exercises should focus on quadriceps and hamstring stretching. Pain that has persisted more than 3 months and is refractory to conservative measures may benefit from hyperosmolar dextrose injection distal to the patellar tendon [33]. Steroid injections are not recommended due to risk of tendon rupture. In patients with persistent debility, who failed conservative treatments and injections, may benefit from surgical removal of ossicles and tubercle-plasty [31].

Return to Activities

Osgood–Schlatter usually self-resolves once the growth plate ossifies which may take 6–18 months [34]. Complete rest is not typically recommended, and activity is encouraged to avoid deconditioning. Hence, athletes are advised to play their sport as tolerated as long as the pain is resolved within 24 h [35]. Patients who undergo surgical intervention can return to regular activity within 4–6 weeks [31]. If pain is severe, it may take months to a couple of years to resolve [30].

Osteochondritis Dissecans

What Is the Pathology, How, Why?

Osteochondritis dissecans (OCD) is osteonecrosis of the subchondral bone typically affecting the knee, elbow, or ankle. It may progress to cartilaginous resulting in destabilization. This condition is poorly understood and yet there are increasing incidents. OCD was found to affect boys more than girls (ratio 2:1).

One study showed the incidence typically occurs between the ages of 11 and 15. OCD is thought to be a result of genetics, trauma, or ischemic pathologies. Biological causes include ossification center deficits, endocrine disorders, or hereditary disorders. Mechanical causes of OCD include trauma, strain, tibial spine impingement, discoid meniscus, or other biomechanical issues [36].

Clinical Presentation

Adolescents presenting with OCD initially will complain of nonspecific knee pain that is worse with exercise. In more severe cases patients may complain of knee instability, and recurrent swelling [36].

Physical Exam

On physical examination of a knee affected by OCD may appear to be swollen and tender to the touch of the condyle. When observing the gait of a patient suffering from OCD, there is notable external rotation on the affected side. If the tibial spine impingement is reproduced by performing Wilson's test, OCD should be considered. This test is performed by internally rotation the knee while in an extended position [36].

Diagnostic Studies

The diagnosis of OCD can be confirmed using an X-ray of the affected knee (AP/lateral/tunnel/merchant). The staging of this condition is graded from 0 to 2. Stage 0 is a normal knee. Stage 1 shows a rim of sclerosis. Stage 2 shows a perifocal ring of sclerosis. MRI is a common diagnostic tool to identify OCD lesions (Fig. 6.3) and to assess knee stability. OCD on MRI will show the extent of marrow swelling, or a present fracture. The gold standard diagnostic tool is arthroscopy [36].

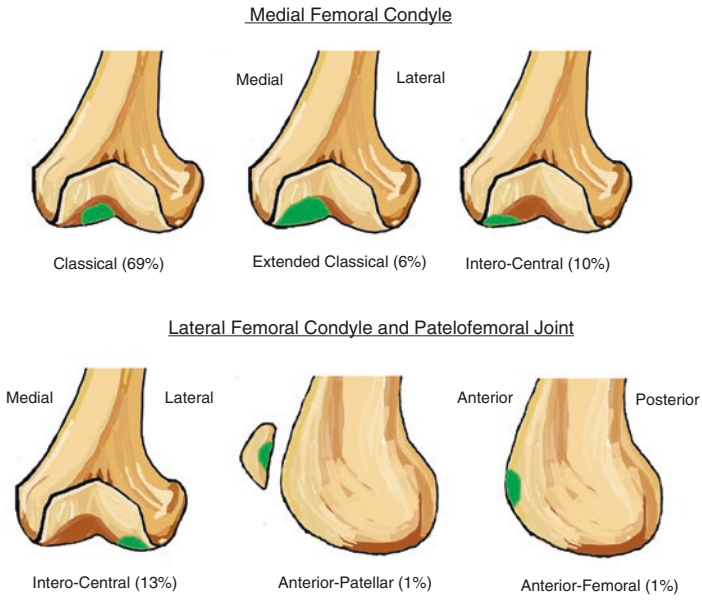

Fig. 6.3 Possible locations of an osteochondritis dissecans lesions

Treatment

In terms of management of OCD, there are not many randomized control trials to date comparing conservative intervention to surgical intervention. The best prognosis is found in athletes with wide open physes. The worst outcomes are found in patients with closed primary ossification centers, unstable lesions, or large injuries. Conservative treatment initially involves limiting activity for up to 6 months in a stable affected knee. Joint immobilization recommendations are inconsistent across multiple studies. Immobilization may be maintained for 4–6 weeks. Operative treatment is recommended when the ossification centers have fused (Fig. 6.4). Surgical interventions include arthroscopic drilling transarticularly or retroarticularly. In addition, surgery may include fixation of loose fibrous tissue or excision of unsalvageable fragments [36]. Once healing is observed on imaging, therapy can be initiated. Physical therapy exercises should be tailored to increase strength and mobility at the knee [37].

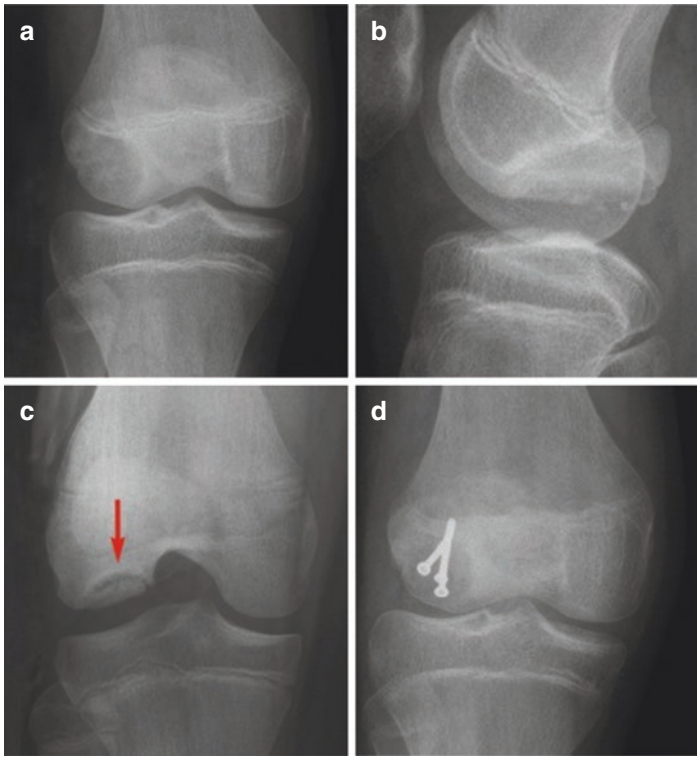

Fig. 6.4 (**a**) Anteroposterior, (**b**) lateral, and (**c**) tunnel view images of the knee. In contrast to the anteroposterior and lateral images the osteochondritis dissecans lesion can be clearly seen on a tunnel view X-ray (arrow). (**d**) Fixing the osteochondral lesion with three screws [17]

Return to Activities

The initial treatment is limiting physical activities, therefore athletes and children are advised to refrain from vigorous play until pain has improved and healing is shown on follow-up X-ray. Completion of therapy and return to activities pain-free is a process that on average takes 6 months but can be as soon as 4 months [37]. In patients requiring surgical interventions, one study found that with post-retroarticular drilling, most patients returned to activity within 2.1 months. However, another study found that the average return time was 8.5 months. If patients suffer from unsalvageable

lesions, the return to activity time is prolonged, averaging, in one study, 14.1 months. Of note, a number of studies have shown that patients may not return to their baseline activity level. The stage and severity of the OCD lesion are also associated with the rate of healing and more severe lesions may take longer to recover [36].

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7 Outpatient and On-Field Evaluation of Sports Trauma in the Knee

Dustin C. Buller, Esha Jain, and Alexis Colvin

Introduction

Sports trauma is frequently observed in the USA and globally. One study estimates 90% of sports injuries occur in the hip and below [1].

In a study performed looking at emergency department visits from sports and recreation from 1997 to 2007, it was found that one million of these four million cases were for lower extremity injuries. From these one million cases, 169,000 were knee specific. Thus, knee related sports injuries demonstrate a large presence in the USA [2]. As athletes continue to push themselves daily, they are prone to injury and acute sports trauma [3].

D. C. Buller (✉) · A. Colvin
Leni and Peter W. May Department of Orthopaedic Surgery, Icahn School of Medicine at Mount Sinai, Mount Sinai Health System, New York, NY, USA
e-mail: Dustin.Buller@mountsinai.org; alexis.colvin@mountsinai.org

E. Jain
Department of Physical Medicine and Rehabilitation, Icahn School of Medicine at Mount Sinai, Mount Sinai Health System, New York, NY, USA

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The Scope of This Chapter

The intended use of this particular chapter, with its content and organization, is different from the others within this book. Throughout the other chapters of this book, the authors have endeavored to present the pathophysiology, anatomy, clinical presentations, evaluation, and overall management of various specific pathologies of the knee in great detail. Alternatively, in this chapter we will set out to provide something of a concise but comprehensive and broad reference for the physiatrist, sports medicine, or family physician, who may be required to evaluate a variety of sporting knee injuries in the outpatient setting, either during sports coverage and on-field evaluation or, more commonly, amidst a subacute presentation in the office.

The chapter will be in two primary parts. First, a section on "general principles" of acute and subacute management of sports injuries of the knee will provide helpful principles and algorithms in managing various broader categories of knee injuries. These principles will guide a provider to a specific diagnosis, after which they may refer to a more detailed reference on said diagnosis, such as in one of the other chapters of this text. The second section will serve as a quick reference for sport-specific mechanisms of injuries. This section is meant to allow a provider to form an organized differential diagnosis of knee injuries by sport in which the injury occurred, and to proceed forward with next steps in management based on the likely diagnoses.

For each sport addressed, the sub-sections will consist of common mechanisms and the specific pathologies related to these mechanisms, a sub-section with comments for on-field evaluation for those providers who may participate in sports team coverage, assessment in the outpatient clinic setting, and finally, management, with comments on differences between amateurs and elite athletes, and a focus on return-to-play.

Many different sports and their injury mechanisms are analogous. The focus in this chapter will be for injury mechanisms which are unique to a sport due to specific positions or activities required of it.

Common injuries are common, and as such, injuries such as ACL and/or meniscal injuries are possible with any number of sports. We will address these injuries earlier on within this chapter, within the sub-section for a sport in which the injury is quite common. We will mention the injury again where necessary and/or relevant. Where there may be any injury common to a sport which has been previously addressed, we will mention it and provide a reference to where it has already been addressed.

The authors hope you may find this chapter to be a useful standalone reference to aid in the outpatient diagnosis and management of your patient athletes.

Principles in the Management of Sports Trauma in the Knee

Categories of Musculoskeletal Injury

This section sets out some general guiding principles in the management of sporting injuries of the knee. Organizing the spectrum of musculoskeletal injuries into "buckets" which require similar treatment algorithms allows the provider to work efficiently and competently within the space of the unknown diagnosis.

Musculoskeletal injuries can be divided broadly into bony injuries and soft tissue injuries. There are exceptions to every rule, but speaking generally, for example, bony injuries are more often than not going to require non-weight bearing and immobilization initially. On the other hand, for the vast majority of soft tissue injuries it is safe to allow weight bearing as tolerated, and while immobilization may provide comfort, it is less frequently necessary and carries with it a risk of greater future stiffness of any involved joints. Furthermore, both bony and soft tissue injuries exist on a spectrum of severity which may often be recognized quickly and can provide a diverging point in management, examples including an incomplete fracture as opposed to a displaced fracture, or a sprain versus a tear.

We find it helpful to think of musculoskeletal injuries in terms of the following: (1) chronic injuries, such as tendinopathies or enthesopathies, of which the management varies greatly, and which will not be specifically addressed except in instances where a sports trauma may "complete" the injury (such as a patellar tendinitis leading to a patellar tendon rupture during sporting activity); (2) skin injuries, such as abrasions, lacerations, and burns; (3) the "incomplete" injuries, such as bony contusions, stable incomplete fractures, ligamentous sprains or strains (terms which we will use interchangeably), or muscle strains; (4) "complete" soft tissue injuries such as tendinous, ligamentous, or muscular "tears" or "ruptures"; and (5) and "complete" bony injuries, namely, fractures.

Each will be addressed in slightly more detail below, and then in much more detail by specific injury throughout the chapter. Generally, for contusions, sprains, and strains, the patient may be allowed to weight bear as tolerated, does not require immobilization, usually does not require further imaging in the subacute setting, does not require surgical management, and appropriate return-to-play timing varies depending on the specific injury, severity of symptoms, and function. For complete soft tissue injuries, the patient may be allowed to weight bear as tolerated, may or may not require immobilization, often requires MRI evaluation to determine definitive management, may or may not require surgery, and appropriate return-to-play timing varies by injury but generally will be at least 6 weeks, very commonly about 3 months, and sometimes up to 9 months after definitive management. For fractures, the patient is usually immobilized—most commonly in a splint or cast, but occasionally a brace, depending on fracture type—and made non-weight bearing with few exceptions (to be addressed), requires radiographic evaluation with X-rays and possibly advanced imaging (usually computed tomography), may or may not require surgery, and return-to-play will commonly be at about 3 months for non-contact sports and 6 months for contact sports.

Initial Management of the Suspected Acute Musculoskeletal Injury

Whenever an athlete is injured during sport, the first and most important step is an immediate on-field primary assessment, focused physical examination, and triage. Generally, the most life threatening and life-altering sports injuries are those involving the head, neck and spine, and so it is important to assess for and to be able to recognize these injuries. However, as the focus of this book is on knee injuries, we will assume from here on out that the patient's primary complaint is that of knee pain.

Mechanism

It is not always possible to observe the exact mechanism that led to the patient's injury. Perhaps the provider's attention was elsewhere, or the view was obscured by other players or equipment, or the injury happened so quickly as to make an interpretation of the provider's visual inputs difficult or impossible. However, if it is possible to deduce the mechanism either by direct observation or by the patient's description, this can provide a significant clue towards the patient's diagnosis, or can at least help to narrow the differential substantially. For each injury discussed in this chapter, common inciting mechanisms will be addressed. For example, a non-contact pivoting or twisting injury can frequently lead to an ACL injury with or without concurrent meniscus injury, and is less likely to lead to a fracture. A direct impact to the front of the knee may lead to a patella fracture. If the mechanism is hyperextension, one might consider a PCL injury. Varus and valgus impacts could lead to LCL and MCL injuries, respectively.

While a full knee examination should be conducted regardless of the injury mechanism, the mechanism can lead the provider to perform uncommon examination maneuvers or to guide their special attention throughout a general examination.

Initial Examination of the Knee [4]

A thorough examination of the knee and the surrounding area is necessary for any complaints of knee pain or suspicion of any knee injury whatsoever. A good knee examination can be both

efficient and thorough, narrowing the differential diagnosis and often leading to a fairly specific suspected diagnosis in a matter of minutes. In one series, an accurate primary diagnosis of knee pathology was made by history and physical exam alone in 83% of patients (as confirmed by diagnostic arthroscopy) [5]. We suggest the timeless examination order of inspection, palpation, and manipulation. Here we will briefly discuss a suggested order of examination of the knee and specific examination maneuvers which should test for the vast majority of possible injuries. Specifically, less commonly performed maneuvers will be discussed later with the injuries for which they test.

Beginning with inspection, the provider should carefully observe the knee as it lies. Depending on the required uniform and/or equipment for the sport of the patient in question, it may be more or less feasible to remove any coverings and directly observe the knee, but this should be done when reasonable. Any overlying clothing should be removed without exception in the outpatient setting. The provider should inspect for any focal edema, global edema, or knee effusion, which can be difficult to differentiate by inspection alone, but can be differentiated by palpation and patellar ballottement either during the course of observation or later as the provider begins the palpatory examination. The provider should inspect for any discoloration, ecchymosis, abrasion, or laceration. Finally, the provider should inspect for any gross deformity of the knee, which may imply a displaced fracture, or most concerningly, an acute knee dislocation.

After careful inspection, the provider should palpate the knee. We prefer to palpate each of the major structures in and about the knee in order, so as not to miss any unsuspected areas of tenderness. Additionally, it is prudent to leave any area at which the patient has indicated pain to be palpated last, so as not to increase their anxiety about being examined and to allow for easier and more comfortable palpation of other sites before that at which injury is already suspected by history or inspection. Providers should palpate the medial and lateral condyles of the distal femur, the quadriceps tendon, the patella, the patellar tendon and tibial tubercle, the medial and lateral joint lines anteriorly, the medial and lateral joint lines posteriorly, the MCL, the LCL, the pes

anserine bursa/insertion, the IT band, and the medial and lateral condyles of the proximal tibia.

If there is a fracture, the presence of fracture and its location can often be determined or suspected by a focal area of severe tenderness. Joint line tenderness indicates an internal derangement or at the very least synovitis. In isolation, this can narrow the differential to only intra-articular diagnoses, though this differential remains broad and could include anything from an osteoarthritis flare to an acute ACL tear. If joint line tenderness is isolated to the medial or lateral joint line in the context of the right history and injury mechanism, it can lead one to specifically suspect a medial or lateral meniscus injury. Tenderness at the tibial tubercle can indicate a patellar tendinitis. Tenderness at the pes anserinus is indicative of pes bursitis. In addition to focal tenderness at the quadriceps or patellar tendon, a palpable defect in the tendon substance can suggest injury of that structure.

Active range of motion of the knee should be attempted. In the setting of some acute injuries, the patient may be unable to move the knee or unable to tolerate active motion secondary to pain. Active range of motion can provide valuable information. For example, active extension with a straight leg raise should be performed. If the patient is unable to fully extend their knee and/or maintain a straight leg raise after assistance and removal of that assistance, one should be suspicious of extensor mechanism disruption by injury to the quadriceps tendon, patellar tendon, or the patella itself.

Passive range of motion should then be performed by the examiner. Non-mechanical limitation secondary to intolerance of pain is quite common, but in the vast majority of knee injuries, a nearly full passive range of motion should persist. A gradual, soft, mechanical block may occur and limit the range of motion by varying amounts when there is a large effusion and the intra-articular fluid volume resists extreme flexion, however, the range of motion usually remains supple through the mid-range in these cases. A severely mechanically limited passive range of motion of the knee typically only occurs with extreme knee effusions, a displaced bucket-handle meniscus tear, or in the setting of a traumatic knee dislocation. Large knee effusions may indicate septic arthri-

tis, which is rare in the setting of an acute trauma. Septic arthritis should remain on the differential since it may present in the outpatient setting in at-risk patient, masking as a mechanical injury due to a recent confounding history of a minor trauma.

During examination of passive range of motion of the knee, a few specific knee examination maneuvers can be performed concurrently. For example, medial and lateral McMurray tests for meniscus injury can be done while flexing and extending the knee.

Further manipulation of the knee must then be performed to test ligamentous structures. An efficient and systematic ligamentous knee exam may be performed by testing each of the following structures in succession: ACL (anterior drawer and/or Lachman test), PCL (posterior drawer), MCL (valgus stress), and LCL (varus stress). The order does not matter as long as it is consistently performed.

A dial test may be performed, if indicated, but since this requires a patient to lay prone, it may be most helpful to perform this test last.

Finally, it is important to perform both distal neurologic and vascular examinations, as injuries at the level of the knee may infrequently cause concurrent neurovascular injury. Palpation of the dorsalis pedis and posterior tibial artery pulses should be performed, and motor function for the peripheral nerves of the lower extremity, as well as sensation of the distal dermatomes, should be tested.

Return-To-Play

Return-to-play recommendations for specific injuries and pathologies will be discussed on an individual basis as they are addressed below. In this introductory section we will briefly discuss return-to-play decisions in generalities, with the frame of reference of on-the-field evaluation in mind.

The "primary responsibility" of return-to-play guidelines is "to cause no harm to the athlete, while enabling him or her to participate at the highest level possible" [6]. As such, the most important guiding principle of deciding when an athlete may return to competitive play, or even to "participation" in their sport, is to

determine when the return will not further harm them, or provide a risk of harm, above a pre-injury baseline level. Thus, the index of suspicion for serious injury susceptible to worsening by continued play must be high, and the threshold for removing an athlete from play must be relatively low.

On the field, in general an athlete may immediately return-to-play in the setting of minor abrasion, laceration, bruise, bony contusion, strain, or sprain. The injury must be minor enough so as not to limit the athlete's function or to cause them undue pain. Skin injuries may require cleaning and dressing. However, some leagues may not allow return-to-play if an athlete is bleeding or has an open wound, in order to decrease the risk of contaminating others. If a laceration will require stitches to heal, generally immediate return-to-play would not be advised, for risk of rupturing a suture repair. Contusions, strains, and sprains vary greatly in severity, and may be so minor that a patient barely notices and can function at near baseline, or may be so severe as to limit the patient's ability to bear weight (secondary to pain) for weeks. A good general rule of thumb is that if a patient can bear weight on their knee without pain, has full active range of motion, and has no instability with a quick test of required knee movements for their respective sport, it is likely safe for them to return-to-play.

If there is any doubt during on-the-field evaluation, it is prudent to remove the athlete temporarily for a more thorough evaluation. A few minutes to allow the athlete to recover from their initial shock, to assess for concurrent injuries, and to perform a complete examination with functional testing specific to the athlete's sport can provide useful information, and possibly a strong reassurance that it is safe to return-to-play, in a matter of minutes.

Dislocations and Acute Reduction

If in the course of an on-field evaluation, it becomes clear that a patient has a joint dislocation, as evidenced by deformity and/or decreased passive range of motion at a joint, it is often beneficial to reduce the dislocation in the acute setting, if possible.

The two kinds of knee dislocations (which are both commonly encountered in sport) are the patellofemoral dislocation and the tibiofemoral dislocation. Each of these will be covered in greater

detail below, in addition to elsewhere within this text. A patellofemoral or "patellar" dislocation should have a closed reduction attempted on the field. If successful, patients do not necessarily require an emergency room visit and may follow-up in the outpatient setting. A tibiofemoral or true "knee" dislocation should be immediately evaluated with a distal neurovascular exam. This is a true emergency and potentially a limb-threatening injury with rates of vascular and neurologic injury in approximately 14–18% and ~25% of patients, respectively [7–9]. If the neurovascular exam is within normal limits, on-field reduction may be attempted if the provider has experience, but it is also reasonable to splint the knee where it lies and call an ambulance for expeditious transfer to a hospital for orthopedic evaluation and reduction. It is possible to cause or worsen a neurovascular injury by prolonged unsuccessful attempts at reduction. If the neurovascular exam is abnormal, especially if the distal pulses are decreased, reduction should be attempted immediately, as the reduction may sometimes restore distal circulation or relieve traction on vessels and nerves about the knee, preventing worsening injury. If reduction is not initially successful, prolonged attempts are not necessary and the patient should be emergently transferred to an emergency room. Regardless of whether or not a tibiofemoral dislocation is successfully reduced, the knee should be splinted for transfer, and the patient should be evaluated in the emergency room with orthopedic and vascular consultations, as a significant number of popliteal artery injuries are intimal injuries or thromboses which may or may not be detected by pulse exam, Doppler, or ankle-brachial indices, yet still result in serious distal morbidity, if not amputation [8].

Principles of Immobilization

Immobilization plays an important role in the early management of many knee injuries. Broadly, immobilization may serve either or both of two primary purposes: 1) the limitation of painful movements for the comfort of the patient and 2) to stabilize the injury prevent its worsening either during motion or weight-bearing.

It is crucial to develop a good understanding of when immobilization is necessary and when it is not, and for how long. It is

equally important to be able to counsel a patient regarding the benefits and risks of immobilization, especially when there is a plan for temporary immobilization and the possibility of difficulties with follow-up. Unnecessary immobilization, or unnecessarily long immobilization, can result in joint stiffness which causes a patient significant long-term limitations in their function.

The vast majority of the time, immobilization is unnecessary in the setting of isolated skin injuries such as abrasions or small lacerations, contusions, strains, or sprains. Exceptions may be made, for example, if a laceration repair is performed and temporary immobilization is desired in order to protect the suture repair. Additionally, immobilization may always be provided for patient comfort in the setting of these more minor injuries. Generally, it is best to provide a removable form of immobilization such as a knee brace or knee immobilizer in these settings. A good general rule is that if a patient is able to get up, ambulate, and range the knee without much pain immediately after a knee injury, it is not necessary to immobilize the knee.

Any time a patient is unable to bear weight immediately following an injury to the knee, acute immobilization is very likely to benefit the patient. Patellar or knee dislocations are more likely to be grossly appreciated at the time of injury than an isolated fracture, and should be immobilized either following reduction or as they lay if reduction is unsuccessful or not attempted. Fractures are not so often definitively diagnosed by examination alone, so in general if there is any clinical suspicion whatsoever of fracture, the patient should be immobilized both for comfort and to prevent any further displacement of a possible fracture.

If splinting supplies or pre-fabricated splints are available at the time of evaluation in the acute setting, these may be applied on the field or on the sidelines after careful transport of the patient. Otherwise, if EMS personnel are called upon, it is important to communicate the suspicion of fracture. EMS teams are equipped with prefabricated lower extremity splints which may be applied to the leg on the field to stabilize the patient for transport and further evaluation at the hospital.

Follow-Up and Continuing Evaluation

Except in the most minor of injuries, every traumatic knee injury should be followed in the outpatient setting after acute management to ensure a good recovery, proper rehabilitation, and a safe return to sport.

For any sports injury which does not require hospital level care on the day of injury, the patient should be seen by a physician in the office within 1–2 days after the injury occurs. This allows for the ability to perform diagnostic tests such as X-ray, the establishment of a symptom trajectory, a careful and thorough examination in a controlled and comfortable setting, and the ability to provide prescriptions for medications and further testing or referrals to any necessary specialists for further evaluation.

Injuries which are evaluated in the hospital should of course have a plan for expeditious follow-up with an appropriate provider in place before the patient is discharged either from the emergency department or the inpatient setting.

Any fracture or complete soft tissue injury should be referred to an orthopedist for a surgical evaluation. Generally, knee fractures should be evaluated by an orthopedic traumatologist (or a general or sports orthopedist who is known to take care of fractures), and complete soft tissue injuries about the knee should be evaluated by an orthopedic sports surgeon (or a general orthopedist).

General Principles in Outpatient Management

Contusions and Sprains

A contusion is a compressive injury which results in hemorrhage without the complete disruption of the organization of the tissue unit (as opposed to fracture or a complete soft tissue tear or rupture), typically due to a direct blow to the structure. A sprain or strain (for our purposes considered synonymous) on the other hand is a tensile or "overstretch" injury which results in hemorrhage without complete disruption of tissue architecture. As such, bony prominences and muscles are most susceptible to contusions

as they are the most exposed anatomic structures. Strains occur only in muscles, tendons, and ligaments as bone is relatively inelastic and will fracture before it stretches or plastically deforms. These kinds of injury are exceedingly common in sports, much more common than tears or fractures, and accounting for up to 95% of sports injuries in some cohorts [10].

Because these are inherently stable injuries without a complete structural disruption (and thus largely not amenable to surgical treatment), they can be thought of as a group of more "minor" or "incomplete" injuries and can all be treated similarly. They share a similar clinical presentation and examination findings.

Typically a patient with a contusion or strain will present with a history of a moment of sudden, acute-onset, focal pain in the area of a specific anatomic structure. In a contusion this is in the setting of some impact, and in a strain this is in the setting of a sudden stretch or exertion of the injured structure. The pain often resolves almost immediately initially (though it may not), and gradually worsens over the course of hours to days as the injured structure continues to hemorrhage internally and incites an inflammatory response. Though it may be painful to bear weight through or move a contused or strained knee, it should be physically possible were the patient to be made comfortable enough. Examination in the outpatient setting will reveal a focal area of tenderness and edema, without any identifiable bony or soft tissue instability. Radiographic evaluation will be negative for fracture, and MRI will show increased fluid signal intensity at the area in question without complete disruption due to the inflammatory response, and possibly partial tearing of the muscle, tendon or ligament in question. A partial tear is still considered a sprain or strain for our purposes of developing general treatment principles here.

Contusions, sprains, and strains in general can be allowed to weight bear as tolerated, and immobilization or bracing is not necessary, though it may be used initially for patient comfort until pain improves, and may protect against reinjury during a short initial time period. They are treated with "RICE therapy." "RICE" is an acronym for rest, ice, compression, and elevation. Ice, compression, and elevation all work to slow bleeding acutely,

and subsequently prevent local edema, which in turn decreases pain due to decreased stretch and mechanoreceptor activation. These injuries typically heal in approximately 4–6 weeks, though they can sometimes take up to 3 months. Rehabilitation and physical therapy are focused on gradual strengthening as pain decreases over time, as well as stretching and range of motion exercises to prevent stiffness.

Because no limitation to weight bearing or range of motion is required in order for a contusion or sprain to heal, return to sport is dependent upon symptoms (e.g., pain) and function. It important not to return to full activity too early, as there is evidence that some strains and sprains may increase the risk of a future complete injury. A partial ACL tear, for example, progressed to a complete tear in 39% of patients under 30 years old within 2–8 years in one cohort, with a 6.29× odds ratio for those participating in pivoting sports [11]. These numbers do not observe a cohort which returned to sport too early (i.e., with activity-associated pain) and therefore are likely an underestimate.

Once an athlete is able to do all of the activities required of them in their position in their sport without pain, they should return. This return may be graduated, allowing the athlete to return partially and limiting them to participation only in activities which they can perform pain free. For example, sometimes pivoting is most painful for an athlete recovering from a ligamentous knee sprain, but running in a straight line becomes comfortable much earlier on in the recovery. In such a situation it is appropriate to allow the athlete to return to practice and conditioning drills prior to returning to pivoting drills or competitive play, in the meantime having them work with a physical therapist on gradual strengthening and pivoting activities in a guided return-to-sport program.

Complete Soft Tissue Injuries About the Knee

For our purposes, we will consider the "complete" soft tissue injuries of the knee to be complete ligamentous tears, complete tendon ruptures or tears, meniscus tears, cartilage injuries, and complete muscle tears. While the ultimate treatment of the pathologies in this family varies widely, we find it helpful to think of them together because of the similarities in initial management.

These injuries will present with a variety of mechanisms and histories, though they are all injuries which occur acutely under a momentary stress to the anatomic structure. Some may have more subacute or chronic preceding symptoms leading up to the acute injury, such as in a patellar tendinitis which goes on to rupture.

Though these injuries may be in some way "displaced," no reduction is performed as the soft tissue structures about the knee may not be finely manipulated in the way that a bony injury sometimes can be, and are not so inherently stable once reduced. As such, they are usually initially managed at the time of injury with bracing and crutches, often made non-weight bearing initially prior to the diagnostic investigation, as there is always the initial possibility of fracture.

Examination in the outpatient setting will often reveal a focal area of tenderness and edema, either directly over the injured structure, if superficial, or with a knee effusion and some degree of joint line tenderness if intra-articular. The other positive examination findings vary widely by pathology, so the knee examination and the specific maneuvers performed should be guided by the clinician's leading suspected diagnoses and remaining differential.

It is extremely important to obtain an X-ray early on in the diagnostic process, as this differentiates between fracture, (which will require immobilization and likely non-weight-bearing), and a complete soft tissue injury, (which may require immobilization but is permitted to weight bear in the majority of cases).

There are exceptions which will be addressed specifically later in the chapter, but in general, if there is a negative X-ray and physical exam suggests a complete soft tissue injury, with gross ligamentous instability, palpable tendon defect, positive meniscus testing, or otherwise, an MRI to evaluate the extent of the injury is indicated.

Radiographic evaluation in these injuries will be negative for fracture, except in the event of avulsion fractures at the origin or insertion of a ruptured soft tissue structure. The MRI will show the complete tear of the structure in question, in addition to the amount of displacement or retraction of the limbs on either side of

the injury. It will show increased fluid signal intensity at the area in question, often with surrounding hematoma, or if intra-articular, hemarthrosis.

These injuries may require surgical fixation and the surgical indications vary by pathology. Therefore, a referral to an orthopedist for surgical evaluation should be made. In the meantime, once fracture is ruled out, most of these injuries can be made weight bearing as tolerated, often placed in a knee immobilizer or knee brace for comfort, and instructed to rest, ice, compress, and elevate the extremity. They may require crutches to ambulate initially. A non-exhaustive list of notable exceptions includes the following: for quadriceps or patellar tendon ruptures, immobilization of the knee in full extension at all times is mandatory, as flexion will displace the tendon limbs. For conditions in which weight bearing may cause extreme pain or further damage, such as in a displaced bucket-handle meniscus tear or a displaced cartilage lesion, non-weight bearing is appropriate.

Here, return to sport guidelines are more complicated, and vary by the injury and the way it was treated. Whether treated operatively or not, return to sport for any complete soft tissue injury is likely to be at least 3 months, in order to allow for at least 6 weeks healing followed by a period of rehabilitation.

Fractures

The definitive treatment of a fracture varies enormously by an equally large number of different contributing factors including location, fracture pattern, degree of comminution, articular involvement, degree of displacement, patient age, bone quality, medical comorbidities, and patient preference. As such, every fracture should be referred to an orthopedist for a surgical evaluation. Fortunately, the initial workup and management is largely similar no matter the fracture location or type.

Open Fractures

"Open fracture", sometimes called "compound fracture," is the preferred terminology for any fracture which communicates with an opening in the skin, whether from the "inside-out" when the fracture

tears open the skin, or from "outside-in" which is usually the result of a high-energy force or crush injury to the affected area, or a coincidental laceration near to the fracture site during the same injury event. The opening is usually overlying the fracture site, but rarely there may be an opening in the skin far distal or proximal to a fracture which still is in open communication due to soft tissue planes. Therefore, it is generally safest to assume a fracture should be treated as "open" if there is any opening through the dermal layer within the same limb segment. Superficial abrasions are not considered "open." In summary, if a fracture and a laceration are on the same side of a joint, it is safest to treat the fracture as open.

It is important to carefully evaluate for open fracture at the time of injury, and also to be observant for a missed or developing open fracture in the outpatient setting. Not infrequently, open fractures can be missed at the time of initial evaluation, especially smaller openings and posterior openings which would have required the patient to be moved to have been seen [12]. It is important to recognize these injuries early, as they have a high rate of subsequent deep infection and the expeditious administration of an IV first-generation cephalosporin has been shown repeatedly to decrease the infection rate [13, 14]. Ideally antibiotics should be administered as soon as possible, and have been demonstrated to make a significant difference even up to as late as 24 h after the injury. Open fractures should always be sent to the hospital for IV antibiotics, tetanus prophylaxis, and evaluation for the need for any possible surgical irrigation and debridement [12].

If an open fracture is seen in the acute setting with any gross contamination, the wound should be irrigated, gross contaminants removed, and the wound covered with sterile materials such as gauze sponges or a sterile wrap before being splinted for transport to the hospital [12].

Initial Management of Closed Fractures

Closed fractures, on the other hand, do not necessarily require hospital evaluation. If evaluating a closed fracture at the time of injury, it is important to rule out any associated emergencies first. A distal neurovascular examination must be performed, and if there are

decreased pulses, paresthesia, or motor weakness, the patient should be evaluated in an emergency room. One should inspect for an obvious deformity indicating an underlying displaced fracture. If significant displacement is suspected, this patient also should be evaluated in an ED for possible need for reduction.

Evaluation in the emergency room provides the easiest and most efficient way to diagnose a fracture and receive proper early care. However, if the distal neurovascular exam is intact, a fracture is suspected by physical exam but significant displacement is not, and the patient prefers to undergo further evaluation and workup in the outpatient setting, this is not unreasonable. The patient's knee should be braced or splinted and the patient should be given crutches and instructed not to bear weight through the injured extremity.

It is fairly common for fractures to present for the first time in the outpatient setting, especially in sports where a team may have a "team physician" with whom their athletes can get expeditious outpatient follow-up for injuries incurred on the field. Examination of a fracture about the knee in the outpatient setting will reveal a focal area of tenderness and edema directly over the fracture site. If the fracture extends intra-articularly there may be a knee effusion from resultant hemarthrosis. If one is evaluating a suspected fracture within 24–48 h after injury it is important to ensure that there is no acute or developing compartment syndrome. The soft tissue compartments around the fracture should be examined to ensure that they are soft and compressible, and again a distal neurovascular examination should be performed.

The first step in the diagnosis of a fracture is to obtain an X-ray. The X-ray will reveal the fracture location, pattern, and any degree of displacement. Non- or minimally-displaced fractures can sometimes be treated definitively in a brace or cast, and displaced fractures generally will need a reduction or surgery. However, the decision-making can be complex and once diagnosed, fractures should be evaluated by an orthopedist either in the outpatient setting or in the hospital for a surgical evaluation. Generally, an MRI is not needed in the evaluation of a fracture, though some knee fractures are associated with concomitant soft tissue injuries. However, even in these cases it is common to obtain an MRI at a later date after the fracture has been treated.

These patients should be properly immobilized and made non-weight bearing to prevent any fracture displacement and for comfort. Generally, a knee immobilizer is sufficient, though if a patient is still uncomfortable in a knee immobilizer, a long leg splint or cast can be applied for better stability.

Return to sport guidelines are fairly uniform. Whether treated operatively or not, return to sport for any fracture is likely to be approximately 3–4 months. All fractures require at least 6 weeks to heal following either surgery or immobilization for non-surgical management, and some peri-articular fractures about the knee require something more like 10 weeks. After the fracture has healed, the patient is allowed to bear weight and gradually rehabilitate to their prior level of function, which usually takes approximately an additional 6 weeks.

Exceptions

There are two notable exceptions to the above guidelines on the initial management of fractures about the knee:

The first are patella fractures. Because the body weight is transmitted through the tibiofemoral joint and not through the patella, patella fractures may be allowed to weight bear as tolerated without fear of this displacing the fracture, and they often can do so fairly comfortably within a few days of injury. However, patella fractures must be immobilized, and it is especially important to wear a knee immobilizer when weight bearing, because flexion of the knee may displace a patella fracture.

The second are avulsion fractures. Avulsion fractures are usually the result of a ligamentous or tendinous injury avulsing a small piece of bone with the soft tissue as it tears. As such, they do not usually result in an unstable fracture pattern and so should be thought of more as a complete soft tissue injury and allowed to weight bear.

Both patellar and avulsion fractures can sometimes be surgical, so they should still undergo orthopedic surgical evaluation.

Conclusion

This concludes the section on general principles in the management of traumatic sports knee injuries in the outpatient setting. We hope it provides guiding principles which allow for the efficient and competent evaluation, triage, early management, and workup for these injuries. We now turn to our overview of specific sports, injury mechanisms, and specific injuries.

The Outpatient Management of Traumatic Sports Knee Injuries

Introduction and Scope

The following section of this chapter is meant to serve as a quick reference to guide the initial outpatient management for a number sport-specific mechanisms of injuries. The injuries discussed in this chapter are given a more detailed treatment of their etiologies, pathoanatomies, management options, outcomes, and relevant literature. Here we focus specifically on the important examination maneuvers, imaging studies, and initial steps in management relevant to these injuries in their initial evaluation in the outpatient setting.

In order to decide which sports, mechanisms, and injuries to include, we researched the frequencies of various knee injuries in sports in general, as well as the frequencies of different kinds of knee injuries by sport, and opted to include those that were most common. Where there was a sport with an unusually strong correlation with a specific injury, we have included the sub-section for that injury with that sport. If the distribution of the injury among sports was relatively even, we have placed the injury within the sport in which it most commonly occurs [15–17].

Sports and Injuries Included

Some very common sports (both in America and globally) such as baseball, tennis, or rowing are notably not included in this chap-

ter, as they are primarily upper-extremity-driven sports and most of their unique injury mechanisms and pathologies are related to the upper extremity. Although knee injuries do occur—as in any sport played from a standing position or requiring running or pivoting—the injuries are not unique to the sport and will have been covered elsewhere.

Organization

We have chosen to organize this chapter by sport (in alphabetical order) for ease of use.

American Football

American football has the highest injury rate amongst all American sports, with knee injuries comprising the majority and the most frequently career ending injuries [18].

Mechanisms and Injuries

Direct Impact, Collateral Ligament, and Posterior Cruciate Ligament Injuries

By far, the most common mechanism for knee injuries in general in football is direct impact. Due to the frequency of tackling and the contact nature of the sport, the knee is frequently put through sudden changes in the amount and direction of force imposed upon it throughout the course of a game. Almost any knee injury could theoretically occur from a direct blow in football, which is a point common to all contact sports. As such, the differential of a knee injury amongst contact sports is uniquely broad, and football is no exception. Common injuries to consider as part of one's differential diagnosis are fractures, collateral ligament injuries (both lateral and medial), cruciate injuries (both anterior and posterior), cartilage injuries, and even rarely knee dislocations.

The exact described mechanism, or observable if recorded, can be helpful in elucidating the likely diagnosis. For example, a medial collateral ligament (MCL) injury is going to result from a

valgus force, i.e. a direct blow to the lateral knee. Conversely, a lateral collateral ligament (LCL) injury occurs when the mechanism is a varus force, i.e. a direct blow to the medial knee. Fractures can occur with a direct blow to the area of bone in question.

Here we specifically discuss PCL injuries (and later MPFL, which are sometimes due to direct impact), due to their disproportionate frequency in American football as opposed to other sports. The other injuries which may commonly occur due to direct force in American football will be briefly alluded to with reference made to where they are highlighted within this chapter.

Knee Dislocation

Please see the sub-section on knee dislocation where it is addressed under section "High-Energy Collision, Knee Dislocations, and the Multi-Ligamentous Knee Injury".

Collateral Ligament Injuries

MCL injury is discussed elsewhere. Please see the sub-section on MCL where it is addressed under section "Alpine Skiing and the Collateral Ligaments".

Posterior Cruciate Ligament (PCL) Injury

Posterior cruciate ligament injury is a relatively rare phenomenon, accounting for a small minority of sports knee injuries, variously quoted between 1 and 5%. However, football is the sport with the highest rate of PCL injury [18]. This is thought to be because a PCL injury typically requires extreme knee hyperflexion or a higher-energy posterior-directed force direct to the tibia in a flexed knee. Examples of relevant mechanisms in American football are a low tackle from the front, direct impact to the front of the leg while a player is mid-air making a catch or hurdling over another player, or a fall onto a flexed knee.

Because PCL injury occurs in higher-energy mechanisms, it is very rarely injured in isolation. In fact, >75% of PCL injuries occurs in association with one or more of either posterolateral corner, ACL, or MCL injuries [19]. It is very important to care-

fully examine patients who present with a mechanism of injury and symptoms consistent with PCL injury and have a high index of suspicion for these other knee injuries. Finally, PCL injury is predominantly an injury found in males, with at least a 3:1 male: female ratio in studied cohorts [19].

Patellar Dislocation and Medial Patellofemoral Ligament (MPFL) Injury

Patellar dislocations occur in two kinds. First, there are those with more chronic patellar instability and repeated subluxations and/or dislocations over months to years. This can be due to connective tissue disease, sequelae of a distant prior injury, baseline global ligamentous laxity, or normal anatomic variants which destabilize the patellofemoral joint. In patients with chronic patellar instability, the patella may dislocate without a contact mechanism. As such, this sort of patellar dislocation has a somewhat even distribution among various sports. The second kind, on the other hand, is the acute patellar dislocation.

Usually an acute patellar dislocation is a first time dislocation and occurs via a contact mechanism. We elected to discuss this injury here due to the unique amount of knee contact injuries seen in American football. In most sports the majority of patellar dislocations seen are in repeat dislocators, but in football, over half of patellar dislocations are acute injuries from contact force. When the patella is forcibly dislocated laterally, the vast majority of the time the medial patellofemoral ligament (MPFL) is injured, due to its role as being the primary medial stabilizer of the patella [18].

One should be particularly suspicious if history provides a mechanism of a medial blow to the anterior knee with a described patellar dislocation event. When patients present in the outpatient setting following a patellar dislocation they will usually relate the sensation of their “kneecap going out to the side” and someone performing a reduction or “putting it back in place.”

Twisting Mechanisms, Anterior Cruciate Ligament and Meniscal Injuries

One of the most common mechanisms of knee injury during sport is that of a pivot or twist while weight bearing. This mechanism is the etiology for the vast majority of ACL and meniscal injuries in nearly all sports that are played while standing. Because these injuries and this mechanism are so common among sports, we will briefly mention them throughout the various sports and refer to the sub-section where they are primarily addressed.

Anterior Cruciate Ligament (ACL) Injury

Please see the sub-section on ACL where it is addressed under section "Pivoting and Cutting: ACL and Meniscal Injuries".

Meniscal Injuries

Please see the sub-section on meniscal injuries where they are addressed under section "Pivoting and Cutting: ACL and Meniscal Injuries".

On the Field

In general, due to the contact mechanisms and the size and speed of football players leading to potential for relatively high-energy injuries, a careful and thorough on-field or sideline knee examination should be performed for any suspected knee injury. If a player can immediately stand up and walk or run, there is unlikely to be a serious injury necessitating hold from play or any temporizing management. However, if the player is unable to bear full weight on the leg due to pain, they should not be allowed to return-to-play and a serious injury should be suspected.

If an intra-articular injury is suspected, the athlete may be placed in a knee immobilizer, given crutches for comfort, and allowed to weight bear as tolerated until they follow-up with a sports physician. Follow-up should be expeditious.

If a patellar dislocation occurs, it may be reduced on the field by applying a medial force to the dislocated patella while the knee is fully extended. Having the patient sit up during the maneuver can help as the hip flexion takes tension off of the quadriceps muscles. If a successful reduction is unable to be performed, the

patient should be sent to the emergency room for reduction. If, however, the reduction is successful, the athlete should be placed in a knee immobilizer, given crutches for comfort, allowed to weight bear as tolerated, and made to follow-up with a sports physician as soon as possible for repeat evaluation.

Outpatient Evaluation

As neither PCL injury nor patellar dislocation typically will require emergent evaluation (unless the patella is unable to be reduced acutely), it is common for these injuries to present in the outpatient setting 1–2 days after injury, after being referred by a covering on-field physician or athletic trainer.

Knee Examination

The complete examination of the knee should be performed as reviewed in section "Initial Examination of the Knee". If PCL or patellar dislocation is suspected, there are a few specific tests which can be helpful.

For patellar dislocation, the patella can be assessed for continued instability utilizing the lateral patellar apprehension test. The patient's knee is flexed to 45° and a lateral pressure is applied to the patella. If the patient experiences apprehension, or the sensation that their patella will dislocate, the test is positive. One may also detect tenderness over the origin (medial femoral condyle), insertion (medial patella), or along the course of the MPFL, which can indicate a concomitant MPFL injury.

The PCL is most commonly assessed with posterior drawer test, and the extent of the injury can be graded based on the amount of posterior tibial translation on examination with the knee flexed to 90°. A Grade 1 injury is 1–5 mm translation, Grade II is 6–10 mm, and Grade III is >10 mm. There are a number of other PCL tests, but one which is quick, easy, and has a relatively high sensitivity with a very high specificity is to observe for the posterior sag sign. With patient's hips flexed to 45°, and knees flexed to 90°, the posterior sag sign is present if the tibia of the suspected injured side is contoured or sags posteriorly at the proximal aspect of the anterior tibia as compared to the contralateral, unaffected side [19].

Due to the high rate of associated injuries with the PCL injury, it is important to also carefully examine the ACL, MCL, and posterolateral corner.

Radiographic Evaluation

An X-ray of the knee including an axial patellar view such as a merchant or sunrise view should be obtained for any knee injury to rule out fracture.

After a reported lateral patellar dislocation, one may see an MPFL avulsion fracture either at the medial femoral condylar origin or an avulsion from the insertion on the medial patella. Additionally, one may see an associated osteochondral fracture from the articular surface of the patella or femur.

A PCL injury can also result in avulsion fracture, most commonly at the distal insertion, which will appear as a bony fragment at the posterior tibia on the lateral view of the knee radiograph.

For both injuries, an MRI can be helpful. For a PCL injury, MRI can assist in grading the severity of the injury and ruling out any of the common associated injuries. After a patellar dislocation MRI can confirm a suspected MPFL injury and assess for any concurrent chondral or osteochondral injury.

Management

When to Refer to a Surgeon

Any Grade III (by examination) PCL injury or a complete tear as confirmed on MRI is potentially operative and should be referred to an orthopedic sports surgeon for surgical evaluation.

Any MRI-confirmed acute MPFL tear, or a confirmed MPFL tear in a chronic, repeat dislocator with refractory pain or instability should be referred to an orthopedic sports surgeon for surgical evaluation.

Initial Non-Surgical Management

Grade I and II PCL injuries can typically be treated non-operatively with a brief period of immobilization for comfort, followed by physical therapy (PT) 2–3 times per week, focusing on knee stabi-

lization and strengthening, especially quadriceps strengthening. For significant PCL injuries, especially in recreational athletes, the rehabilitation phase may be longer. Suggested rehabilitation of athletes can be divided into different phases, "protective phase, translational phase, and functional phase" [20]. In the protective phase, the goal is about controlling effusion, preserving or improving knee range of motion, and restoration of a normal gait and normal quadriceps strength. In the first 2 weeks, the athlete will have the knee immobilized. Athletes in this phase will transition out of crutches. Weight bearing will go from partial to full depending on toleration. Range of motion exercises will first be performed in the prone position. Flexion will be limited for the first 2 weeks then progress to full motion. Strengthening is performed through straight leg raises, stationary bikes with no resistance, squats, and leg press with no greater than 70 degrees of flexion.

The second phase is the transitional phase, which can last 6–12 weeks in non-elite athletes. The point of this phase is to strengthen the lower extremity to let the athlete perform low-impact activities pain free. With certain exercises, it is important at this phase to ensure there is no compensation from the non-injured leg. For example, in squats, it is important that the player is not shifting their weight on the non-injured leg. This could potentially cause a higher risk of reinjury if not controlled.

The third phase is the functional phase, which can last 12–16 weeks in non-elite athletes. Bracing at this point should have been stopped and the athlete can start exercises with core strengthening, proprioception, and neuromuscular control. The athlete should be pain free without activity during these activities.

After a full course of at least 6 weeks of PT, if the patient has persistent symptoms, they should be referred to a surgeon.

First time patellar dislocations, if they remain stable after reduction, are typically treated non-operatively initially, as some athletes are able to return to sport without surgery. Non-operative management consists of a short period of immobilization in a knee brace, followed by PT for strengthening and range of motion. However, if a second dislocation occurs, or an MPFL tear is confirmed and the patient continues to have symptoms despite conservative management, a surgical referral is warranted.

Return to Sport

If either of these injuries is treated non-operatively, the patient may return to sport as soon as they are functionally capable without pain. If pain improves quickly and physical therapy progresses well, both more minor PCL and MPFL injuries may be able to return to sport as early as 2–4 weeks after injury, although it would not be uncommon to require closer to 6 weeks of PT before returning. PT prior to return to sport should include dynamic drills that build power, strength, and speed. Full-strength exercises for endurance, sport-specific agility, and quality activities to prevent reinjury should also be included [20].

If the patient undergoes surgery, return to sport will be at the discretion of the surgeon, but will likely not be until 3–4 months after surgery, in order to allow for plenty of time for healing of the ligamentous reconstruction, followed by a careful and thorough rehabilitation program.

Basketball

While many of the common sports knee injuries are certainly just as common in basketball, the sport provides an opportunity to explore knee injuries unique to (or at least more frequent with) jumping. In the grand scheme of injuries in basketball, acute trauma to the knee is relatively infrequent, making up anywhere from <5% of all injuries in one NBA and WNBA cohort [21] (the majority of which were contusions or sprains), to 10% of total basketball injuries. Overuse injuries of the knee such as patellofemoral syndrome and "jumper's knee" (patellar tendinopathy) are much more common than traumatic injuries, and ankle sprains are particularly common in basketball, in some cohorts accounting for approximately 30% of acute basketball injuries alone [22].

Mechanisms and Injuries

Jumping, "Jumper's Knee" and Quadriceps and Patellar Tendon Rupture

One injury mechanism unique to jumping sports is that of the quadriceps or patellar tendon rupture as the end stage of what has

historically been called "Jumper's Knee"—namely, repetitive microtrauma to either the quadriceps insertion at the superior pole of the patella, or to the patellar tendon origin at the inferior pole of the patella, from overuse of the extensor mechanism. While a tendon rupture represents the very rare end-stage complication of a common problem, we thought it worth discussing due to the unique presentation and reliable outcomes with proper management.

It is important to consider these diagnoses when an athlete in a jumping sport such as basketball or volleyball presents with acute knee pain and a history of more chronic antecedent anterior knee pain. Typically, the athlete will relate a history of having had anywhere from months to years of waxing and waning pain about the extensor mechanism of the knee, usually having been previously managed with anti-inflammatory medications, rest, physical therapy, and sometimes local corticosteroid injections. Acutely, they will report a popping or tearing sensation during a sudden contraction of the extensor mechanism, usually while jumping or landing, with pain, swelling and difficulty or inability to fully extend the leg afterward.

Pivoting and Ligamentous or Meniscal Injury

The rate of acute ligamentous or meniscal knee injury in basketball is surprisingly low, each less than 1% of all injuries in a professional cohort. As such, they will be addressed elsewhere. However, it is worth noting here that in basketball, as in some other sports, the risk of complete ACL tear has been found to be higher in female athletes as compared to males—in basketball approximately 1.6-fold [21].

Anterior Cruciate Ligament (ACL) Injury

Please see the sub-section on ACL where it is addressed under section "Pivoting and Cutting: ACL and Meniscal Injuries".

Meniscal Injuries

Please see the sub-section on meniscal injuries where they are addressed under section "Pivoting and Cutting: ACL and Meniscal Injuries".

On the Field (Court)

Similarly to football, a careful and thorough on-field or sideline knee examination should be performed for any suspected knee injury sustained during basketball, due to the wide variety of potential injuries.

If encountering a suspected patellar or quadriceps tendon tear, both can be managed on the court by placing the athlete in a knee immobilizer and allowing them to bear weight in the immobilizer with crutches until they follow-up with a sports physician.

Outpatient Evaluation

As neither patellar tendon nor quadriceps tendon rupture necessarily require emergent evaluation, they may also present for the first time in the outpatient setting 1–2 days after injury, after being referred by a covering on-field physician or athletic trainer.

Knee Examination

The complete examination of the knee should be performed as reviewed in section "Initial Examination of the Knee". Here we will specifically discuss examination for quadriceps or patellar tendon injury.

When beginning to assess for a suspected quadriceps or patellar tendon injury, one may start with examination of the extensor mechanism as a whole by testing the patient's ability to sustain a straight leg raise. With the patient lying supine, elevate their leg at least 6 inches above the ground with the knee fully extended, ask them to keep it elevated, and let go. If the patient is unable to keep their heel off of the ground, their extensor mechanism is not intact, and either a quadriceps tendon injury, a patella fracture, or a patellar tendon injury is implied.

Once it has been demonstrated that the extensor mechanism is dysfunctional, palpation for a defect or focal tenderness can help delineate the location of the injury. A caveat to keep in mind is that the ability to maintain a straight leg raise can be impaired by pain and possibly resultant quadriceps inhibition. As such, inability to straight leg raise is often seen with a large knee effusion of any etiology. If the patient is limited by pain, an aspiration followed by intra-articular lidocaine injection can be given into the

knee in order to make the patient more comfortable, sometimes revealing that the extensor mechanism is in fact intact.

Radiographic Evaluation

An X-ray of the knee including an AP, lateral, and axial patellar view such as a merchant or sunrise view should be obtained for any knee where extensor mechanism injury is suspected. The key view on plain radiographs is the lateral, where one can appreciate either "patella alta" (meaning high patella), seen with patellar tendon injury, or "patella baja," seen with quadriceps injury.

An MRI can confirm the diagnosis in any case where the exam and X-ray have been ambiguous, such as in a partial tearing. The other utility of MRI is to assess for retraction of the two limbs of the tendon for late-presenting injuries, which also helps in surgical planning.

Management

When to Refer to a Surgeon

Both acute patellar tendon and acute quadriceps tendon injuries should be referred to an orthopedic sports surgeon for operative evaluation.

Initial Non-Surgical Management

A small partial tearing or a non-displaced avulsion fracture of either tendon can often heal without surgery with a brief period of immobilization for comfort (weight bearing as tolerated in a knee immobilizer), followed by physical therapy (PT) 2–3 times per week, focusing on knee stabilization and strengthening, especially quadriceps strengthening. After a full course of at least 6 weeks of PT, if the patient has persistent symptoms they should be referred to a surgeon.

Return to Sport

If either of these injuries is treated non-operatively, the patient may return to sport as soon as they are functionally capable without pain. If pain improves quickly and physical therapy progresses well, partial tears of either the quadriceps or the patellar tendon may be able to return to sport within 5–8 months [23].

If the patient undergoes surgery, they will require at least 3–4 months following the surgery in order to be cleared to return to sport, and only after a functional assessment and return to sport training program in physical therapy is completed. A suggested rehabilitation program may include the following. The goal of the first 2 weeks is to protect the repair, keep the knee in a brace, and limit the range of motion. In the second to sixth week, the goal is normalized gait with a brace and crutches. Athletes can increase range of motion at this point. From 6 to 12 weeks, the athlete can allow flexion and quadriceps training. The athlete can do more light squats, core strengthening, and other physical therapy exercises. The goal of weeks 12–16 are to get out the brace, have full range of motion, and squat to 70 degrees of flexion. After this time, the goal is transition from light sports to full sport activity without pain or swelling [24].

Ski

Skiers in general are unfortunately especially prone to knee injuries. Prior to the 1970s, skiing was performed in leather alpine boots and had alarming rates of injury as compared to present day. Though a large portion of these injuries were ankle sprains and fractures, because of the relative flimsiness of the old boot designs and the stress risers created by the bindings, tibial shaft fractures were not uncommon.

Thankfully after the 1970s, sturdier, taller plastic boots have been designed, cutting the rate of ski injuries in half overall with dramatic declines in ankle and leg injuries. However, these improvements had an inverse relationship with knee injuries, which increased dramatically now that under modern ski boot designs, the forces of any lower extremity injury concentrate most frequently at the knee [25].

Mechanisms and Injuries

Anterior Cruciate Ligament Injury

ACL injury is exceedingly common in skiing for the reasons mentioned above, however, it is not quite as common as in soccer or

American football according to some sources. This section discusses two injuries more unique to skiing mechanisms, and we address ACL injuries elsewhere under section "Pivoting and Cutting: ACL and Meniscal Injuries".

Alpine Skiing and the Collateral Ligaments

The medial collateral ligament (MCL) is the most commonly injured ligament in skiing, and is injured in at least 20% of all ski injuries in at least one cohort. The MCL is frequently injured in conjunction with the ACL in skiing. The rate of MCL injuries is decreasing while the rate of ACL injuries is increasing year by year. Overall, MCL injuries remain more common [25, 26].

These injury patterns are thought to be due to the frequency of valgus stresses on the knee during skiing. Each time one pushes a ski, the knee receives a valgus force, while typically in flexion. In accidents or while landing from a jump, these forces may be applied through the knee at a higher momentum.

These athletes will typically present with medial knee pain and report feeling a stretch or hearing a pop after exerting a sudden force while skiing, from landing a jump or quickly attempting to stabilize one's self during a near fall. They may be able to ambulate without difficulty but this should not lead the provider to disregard the injury.

High-Energy Collision, Knee Dislocations, and the Multi-Ligamentous Knee Injury

While knee dislocation is an exceedingly rare injury as compared to the other knee injuries discussed in this chapter, it is more likely to occur in extreme sports, where injuries may occur at high speeds and the kinetic energy transferred in injury may be significantly higher than lower-speed impact sports or non-contact sports. Beyond that, due to the force distribution and stress rising of ski boots as discussed above, knee dislocation is uniquely relatively common in skiing. In fact, alpine skiing has the highest rate of knee dislocation of any extreme sport [27].

Knee dislocation in skiing usually occurs from a high-energy impact to the skier's leg below the knee—such as high-speed crash into a rock, branch, or other skier, or from landing on the leg

at high speed with a hyperflexion or hyperextension moment. Any of these mechanisms along with a reported dislocation event should raise the suspicion of a tibiofemoral or "true knee" dislocation and resultant multi-ligamentous knee injury.

On the Slopes

As there is no "covering physician" "on the field" on a ski slope, there are unique safety precautions which physicians may counsel their skiing patients to take. Skiers should always take a cell phone and/or a flare in order to signal for help in the event of an emergency. They should stick to known courses and not venture from standard slopes, so that they will be within sight of the ski patrol should something happen. It may even be prudent for the skier to bring some minimal first aid supplies which could be life-saving, such as a tourniquet.

The most important step in the emergent treatment of acute knee injuries from skiing is to be able to signal for help. It is difficult for any evaluation or treatment to begin in any useful or meaningful way on the side of the mountain, and the sooner the skier is able to be transported down the slope to a treating facility, the better.

If evaluating a knee dislocation in the acute setting, closed reduction may be immediately attempted, as discussed above in section "Dislocations and Acute Reduction". Once reduced, both the previously dislocated knee and the MCL-injured knee may be temporized in the acute setting by placing within a knee immobilizer and weight bearing as tolerated with a plan for expeditious follow-up. As mentioned in section "Dislocations and Acute Reduction", knee dislocations may require admission to the hospital and thorough workup for vascular or neurologic injury [28].

Outpatient Evaluation

While it is exceedingly unlikely for a multi-ligamentous knee injury from a knee dislocation to present undiagnosed to an outpatient visit, it is very possible that a patient who was evaluated in the emergency room or the hospital after a knee dislocation and discharged may not carry specific diagnoses for their likely multiple knee injuries. The multi-ligamentous injury is often assumed

and ignored during the more important, potentially-limb-saving workup of any vascular injury. A knee MRI is often purposefully delayed for a short time after the injury to allow for the resolution of some edema and potentially a high-quality image.

Knee Examination

The complete examination of the knee should be performed as reviewed in section "Initial Examination of the Knee". Here we will specifically discuss examination for MCL and multi-ligamentous knee injury.

The MCL is best examined with the valgus stress test (also called medial stress test), in which a valgus force is applied to the knee at 30 degrees of flexion, and the knee is assessed for valgus laxity. MCL injuries are classified according to the result of this test. A first degree injury has a firm endpoint and no laxity (i.e., a sprain); a second degree injury has slight laxity and a firm endpoint (i.e. a partial tear); and a third degree injury is a complete tear with no endpoint, which can be sub-divided by the amount of gross laxity appreciated.

In the examination of the knee which was previously dislocated, one should ensure that a careful vascular and neurologic examination is performed, taking care to confirm that strong distal pulses are present, and the common peroneal nerve and its branches are functioning.

Ultimately, the examination and diagnosis of the multi-ligamentous knee injury can be performed as the careful and thorough combination of the examination of each individual possible knee injury. As such, examination of the MCL, LCL, ACL, PCL, and posterolateral corner should be performed with the valgus stress, varus stress, Lachman, posterior drawer, and Dial tests, respectively.

Radiographic Evaluation

An X-ray of the knee including at least AP and lateral should be obtained for any knee where MCL injury, knee dislocation, or its sequelae are suspected. MCL injury may not be obviously seen on plain radiographs, though avulsion fractures at the origin or inser-

tion may be present. Knee dislocation can typically be easily appreciated on a lateral view, with either anterior or posterior dislocation of the tibia relative to the femur.

An MRI can confirm the diagnosis of MCL sprain, though this is not often necessary, since an MRI will not change management of a lower degree MCL injury, and third degree injury can be detected by exam. The greatest utility of MRI in the setting of an MCL injury is the diagnosis of other suspected injuries. Similarly, an MRI is very useful after knee dislocation when multi-ligamentous injury is suspected and the physical exam is unclear or complex, even more so because knowing the exact injuries in a multi-ligamentous knee injury can change the surgical procedure ultimately performed.

Management

When to Refer to a Surgeon

Any multi-ligamentous knee injury should be referred to an orthopedic sports surgeon, as they are often operative injuries. MCL injuries, on the other hand need not necessarily be referred, as nearly all MCL injuries may at least undergo a trial of non-operative management. If a third degree MCL injury has persistent pain and/or instability it should then be referred.

Initial Non-Surgical Management

A multi-ligamentous knee injury can be temporized in a knee immobilizer and allowed to weight bear as tolerated until the time of (likely) surgery. Crutches may be provided to support weight bearing. In some cases, reconstructive surgery may be purposefully delayed in order to "prehab" the injury, prevent stiffness, and optimize knee function post-operatively. In these cases, the immobilizer should be removed as soon as is comfortable and the athlete should begin formal physical therapy to work on stretching and range of motion prior to planned delayed surgery.

The vast majority of MCL injuries may be treated non-operatively. All MCL injuries may be treated with NSAIDs for pain relief and anti-inflammatory effect, rest, ice, and elevation,

with eventual formal physical therapy for strengthening once the patient is able to tolerate it. Second and third degree injuries should be immobilized or braced in order to prevent re-tearing and instability during recovery. First degree injuries do not require any bracing as they are stable injuries [29].

Return to Sport

The majority of multi-ligamentous knee injuries after knee dislocation (if the extremity survives any vascular complications) require surgical treatment and an extensive and careful rehabilitation process following surgery. Ultimately return to sport should be at the discretion of the treating surgeon and physical therapist based on functional return to sport criteria after a standardized rehab protocol for the patient's injuries, and will average from 9 to 12 months following surgery [30].

On the other hand, MCL injuries often have a surprisingly quick return-to-play. First degree injuries may return as soon as pain resolves, which could be as soon as 1–2 weeks after injury. Second degree injuries may often return 2–4 weeks after injury with proper rehabilitation, and for third degree injuries, return after 4–8 weeks is achievable [31]. In the rare case that an MCL injury requires surgery, the return to sport is of course much longer, on the order of 6–9 months.

Soccer

While cutting and pivoting injuries are certainly not unique to soccer, it is possible that they are most common there.

Mechanisms and Injuries

Pivoting and Cutting: ACL and Meniscal Injuries

The majority of ACL and meniscal injuries are non-contact injuries. Soccer is uniquely suited to these injuries as they are both prone to occur with injury mechanisms of a sudden pivot or twist on a weight bearing knee, a mechanism which occurs frequently

in the sport due to the nature of kicking and the frequency of pivoting and cutting required in competitive play. Non-contact injuries result from a rapid change in direction that results in momentary deceleration. This causes strain on the ACL from the anterior shearing force of the proximal tibia. This force increases as the angle of flexion decreases [32]. This force is then increased with the addition of a varus or valgus load. In soccer, when the player rapidly changes directions on the field through pivoting, the player adds a valgus force, quadriceps loading, and a foot placement with internal rotation of the tibia. As a result, there is now contusion of the lateral femoral condyle and the posterior aspect of the lateral tibial plateau. The subsequent varus stress during pivoting causes internal rotation of the tibia that causes ACL injury.

The rates of ACL and meniscal injuries in soccer are both relatively high even when compared with other sports. Both injuries in conjunction or in isolation can be suspected after a history of a sudden pain, possibly an audible pop, and the onset of knee swelling. It is notable that in soccer (similar to other sports) ACL injuries occur in females at a rate nearly 3× that of males [33].

On the Pitch

Similarly to football and basketball, a careful and thorough on-field or sideline knee examination should be performed for any suspected knee injury sustained during soccer, due to the wide variety of potential injuries.

If encountering a suspected ACL or meniscal injury, both can be acutely temporized on the field by placing the athlete in a knee immobilizer and allowing them to bear weight in the immobilizer with crutches until they follow-up with a sports physician.

Outpatient Evaluation

As neither ACL nor meniscal injury necessarily requires emergent evaluation, they may frequently present for the first time in the outpatient setting 1–2 days after injury, after being referred by a covering on-field physician or athletic trainer.

Knee Examination

The complete examination of the knee should be performed as reviewed in section "Initial Examination of the Knee". Here we will specifically discuss examination for ACL and meniscal injury.

As both are intra-articular injuries, a knee effusion can be noted in the acute setting.

The ACL can be evaluated by a number of provocative tests, including the Lachman test, the anterior drawer test, and the pivot shift. The Lachman test is performed by attempting anterior translation of the tibia relative to the femur with the knee flexed at 30° and is the most sensitive physical examination test for ACL injury. The pivot shift is more specific, but is often intolerable to the patient due to pain and requires muscle relaxation, so is more often performed in the OR under anesthesia.

Similarly, there are many tests for meniscal injury. Commonly the McMurray test (can be performed for lateral or medial meniscus), Thessaly test, and Apley test are performed, along with an assessment for joint line tenderness along the meniscal periphery.

Radiographic Evaluation

An X-ray of the knee including at least AP and lateral views should be attained when ACL injury or meniscal injury is suspected. One should look for radiographic effusion, which can be present with either of these injuries. In the event of ACL tearing, one can sometimes see what is called a "Segond fracture" on the AP knee radiograph. This is an avulsion fracture of either the anterolateral ligament (ALL) or lateral knee capsule from the proximal lateral tibia. Regardless of which structure the avulsion fracture is attached to, it occurs in common with a complete ACL injury the vast majority of the time. Unfortunately, acute meniscal injury is unlikely to be visible on X-ray except for a knee effusion.

An MRI can confirm the diagnoses of both ACL injury and meniscal injury, in addition to identifying any additional concurrent knee injuries. However, it has been shown that a careful physical examination is equal to if not better than MRI in identifying acute meniscal injuries, particularly in isolation, so if the examiner is confident in this diagnosis, MRI is not strictly necessary [34].

Management

When to Refer to a Surgeon

Both complete ACL injuries and meniscal injuries should be referred to an orthopedic sports surgeon for surgical evaluation. If an ACL sprain is diagnosed by clinical evaluation with a stable Lachman test, with or without confirming MRI, referral to a surgeon is not necessary.

Initial Non-Surgical Management

In the outpatient setting, a complete ACL injury can be temporized in a knee immobilizer, and allowed to weight bear as tolerated until the time of surgery. Crutches may be provided to support weight bearing. If the injury is only an ACL sprain, this treatment may be continued along with formal physical therapy for rehabilitation with range of motion and gradual strengthening over the course of the following weeks.

In some cases for the complete ACL injuries, reconstructive surgery may be purposefully delayed in order to "prehab" the injury, prevent stiffness, and optimize knee function post-operatively. In these cases, the immobilizer should be removed as soon as is comfortable and this athlete too should begin formal physical therapy to work on stretching and range of motion prior to planned delayed surgery.

Meniscal injuries should be managed similarly, with weight bearing as tolerated in a knee immobilizer or brace and crutches for comfort, until it is determined by the treating surgeon whether the meniscal tear requires surgery or not. If it is determined, the meniscal tear may be treated non-operatively, similarly the patient should continue with the same treatment, gradually weaning the brace and crutches as tolerated and working formally with physical therapy to strengthen the injured knee.

Return to Sport

If either of these injuries is treated non-operatively, the patient may return to sport as soon as they are functionally capable without pain. If pain improves quickly and physical therapy progresses

well, for ACL sprain or non-operative meniscal injury, this could be within 1–2 months or earlier.

After meniscectomy, patients have a similarly quick return to sport, as they often have good initial pain relief after surgery, are allowed to bear weight immediately, and need physical therapy simply to regain strength and prevent stiffness.

After meniscal repair, however, there is often a period of non-weight bearing or partial weight bearing to allow for healing of the repair, and so the recovery course is longer, with return-to-play only after return to sport testing, usually >5 months post-operatively [35]. Once an athlete has been cleared to weight bear, the first 2 weeks of a rehabilitation program would focus on progressing weight bearing as tolerated and improving range of motion to about 90° while non-weight bearing, and with or without a brace. Then, there is phase two which ranges from 2 to 6 weeks where the athlete can tolerate full weight bearing in extension, continue range of motion 0°–90°, and can engage in exercises with the knee. From 6 to 12 weeks, the athlete should be in full weight bearing, discontinue wearing a brace, full range of motion, begin hamstring and proprioception exercises, and add a stationary bike. Lastly, the last phase is between 3 and 5 months where the athlete can progress with exercises and activities, swim, and enter a run and jump protocol [36].

A suggested rehabilitation program after ACL surgery can be divided into three phases: "the early postoperative stage, strengthening and neuromuscular control stage, and return to activity and sports stage" [37]. In the first stage, which lasts 4–6 weeks after surgery the main purpose is to control pain, swelling, and protect the healing graft. This would involve preserving muscle strength and progression to full weight bearing as well as knee extension and at least 100 degrees of knee flexion. At the end of this stage, the athlete should be able to walk without crutches. In this stage, the use of "ICE" or "ice, compression, and elevation" is extremely helpful. Also, cryotherapy has been found to decrease pain as well in this stage. Depending on the injury, meniscal tear versus ACL tear, braces and crutches should be used. If there is a meniscal tear, however, the brace should remain locked in extension in this

stage versus in an ACL tear it can be unlocked. A highly important aspect of this stage is redeveloping the range of motion. There are a series of exercises done to achieve this, including active knee flexion, gastrocnemius stretches, hamstring stretches, and straight leg raises. Interventions such as a stationary bike, patellar mobilization, and early initiation of quadriceps exercises are beneficial.

The second stage is about obtaining balance and preparing to return to activity. At this point, the athlete should be able to tolerate strength and flexibility movements without joint pain, swelling, or injury exacerbation. To get to this point, the athlete must participate in activities that encompass weight bearing and non-weight bearing. Several studies have compared weight bearing and non-weight bearing activities and have found no significant difference between them after ACL reconstruction. However, there is still a debate in the literature of the pros and cons of each type of exercise. Despite this, both can combine in effective healing and avoiding stress to the joints. For non-weight bearing knee extension exercises, it is suggested to perform knee extension between 9° and 60°. It can also include partial squats, wall slides, and single leg extension activities thus not relying on the non-surgical leg to compensate. Weight bearing exercises can include lunges, stepping exercises, and double or single leg squats. Hip exercises are also helpful at this stage in combination with pelvic and abdominal muscle exercises. Techniques to regain neuromuscular control include the use of a roller board and tilt board. These techniques can help athletes gain back their learned responses, reflex activities with stress on the ACL, and balance. Towards the end of this stage, an athlete should be able to progress to a jogging stage in which they can run at a slow pace several days of the week without swelling or pain.

The last stage is achieving actual return to activity and sports. At this point in the rehabilitation atmosphere, athletes should have achieved functional progression to the point of being able to fully return to their sporting activities with pivoting, jumping, and range of motion. It is expected that this will depend on the individual in terms of what the sport demands, tolerance, and graft healing. However, if the athlete has satisfied all the previous stages including range of motion, strength, neuromuscular activ-

ity, then they can be integrated back into sporting activities. The athlete should go slowly in terms of integration to ensure safety of return. Opposition by other players in this sense should also be introduced slowly. Finally, although evidence does not support return-to-play at a specific time after ACL reconstruction [38], it has been strongly suggested that it is safest (i.e., the risk of re-rupture is lowest) if the patient does not return to sport until at least 9 months after surgery [39].

Summary and Conclusions

In this chapter, we have presented an introduction to the evaluation of traumatic sports knee injuries in the outpatient setting. We first placed injuries into broad categories in order to provide general guiding principles on management of the undiagnosed or incompletely-diagnosed knee injury, including an efficient but thorough physical examination of the knee. Finally, we described mechanisms of injury which are unique to a handful of popular sports and presented a concise summary of the outpatient management of the example injuries including examination maneuvers, and next steps in radiologic evaluation and management.

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Knee Fractures

8

Seth Probert and Aditya Raghunandan

Basics of Fractures

Description of Fractures

In this chapter, we will discuss common fractures that occur around the knee. A fracture occurs when external stress on a bone exceeds the bones strength.

In general, displaced and/or open fractures will require surgery and non-displaced and/or closed fractures can be managed non-operatively.

Fracture Healing

There are three stages of fracture healing; inflammation, repair, and remodeling (factors that influence healing). The whole pro-

S. Probert (✉)
PGY-4, Physical Medicine and Rehabilitation, University of Texas Health San Antonio, San Antonio, TX, USA
e-mail: probert@uthscsa.edu

A. Raghunandan
Department of Rehabilitation Medicine, Long School of Medicine at the University of Texas Health Science Center at San Antonio, San Antonio, TX, USA
e-mail: raghunandan@uthscsa.edu

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cess of fracture healing takes anywhere from 4 to 12 weeks. The inflammatory stage starts from the time of initial injury to 2 weeks, when callus formation has started. The fracture callus ossifies in weeks 2–4 which denotes the repair stage. Finally, the remodeling stage is when woven bone replaces trabecular bone and starts during the repair stage but can last up to 12 weeks [1].

Fracture/Displacement Types

The two most important aspects of any fracture are the fracture location and fracture morphology. To be able to fully assess the location and morphology of any fracture, an X-ray of the suspected region is imperative. For fracture location, you will need to identify which bone is fractured, if it is distal or proximal and what part of the bone is affected. For long bone fractures, it is important to identify what region of the bone is effected; the epiphysis, diaphysis or metaphysis.

After identifying the location of the fracture, the morphology of the fracture will help determine the severity of the fracture. The morphology of the fracture is often used to determine whether the fracture is operative or non-operative. For long bone fractures along the diaphysis, there are three types of morphology: simple, wedge, and multifragmentary. Simple fractures are classified as either spiral, oblique or transverse; wedge fractures consist of an intact wedge or fragmented wedge fracture; and multifragmentary fractures consist of multiple fracture lines and consist of either intact segmental or fragmented segmental fractures. Fractures that involve the metaphysis or epiphysis have different morphology. The most important factor before determination of morphology is whether the fracture is intra- or extra-articular. Extra-articular will consist of avulsion, simple, wedge, and fragmented fractures. Partial and complete intra-articular fractures are further classified as simple, split, depressed and multifragmented [1].

What Needs Immediate Orthopedic Surgery Referral

Compartment Syndrome

There are four different compartments in the leg that have rigid fibro-osseous borders. Compartment syndrome is caused by increased pressure in one or more of these compartments. Compartment syndrome can occur with any fracture in or around the knee discussed in this chapter [1]. The most common involved compartment is the anterolateral compartment. Compartment syndrome requires immediate surgical referral for emergent fasciotomy. It is important to understand how to assess for compartment syndrome to help get the patient a surgical referral as quickly as possible.

The most commonly used method to diagnose compartment syndrome is commonly remembered as the five "P's" [2]:

1. Pain with passive stretch.
2. Paresthesia.
3. Pallor.
4. Pulselessness.
5. Paralysis.

Concern for Arterial Damage

It is imperative to check pulses distal to the fracture. The dorsalis pedis pulse is the most common pulse evaluated, but you can also palpate for the posterior tibialis artery as it courses through the tarsal tunnel just about the ankle.

Concern for Nerve Damage

If there is decreased or no movement in the lower extremity with the associated fracture and/or decreased/no sensation, this will require immediate orthopedic surgery referral to evaluate for possible nerve damage.

Physical Examination for All Fracture Types

Evaluate the skin to look for abrasions or tears that may indicate an open fracture. Evaluate for effusion, if this is present, this could indicate osteochondral or ligamentous injury [3].

Evaluate for compartment syndrome above. Distal pulses (tibialis posterior and dorsalis pedis) must be tested. If not present, may consider Doppler to evaluate for blood flow or immediate orthopedic surgical referral. Sensation to the ipsilateral lower extremity, if decreased, there is likely nerve damage that will require immediate surgical intervention.

Phases of Rehabilitation

1. *Resolve pain and inflammation*: This first stage of rehabilitation is focused on recovery and minimizing further damage done to tissues. Different modalities that will help in this process include ice, limiting aggravating activities, NSAIDs, ultrasound, and electrical stimulation.
2. *Restore range of motion*: After the patient has shown recovery from initial injury, it is time to start restoring function. The first part of restoring function will be focusing on returning to pre-injury range of motion. In this stage, it is important to perform gentle range of motion exercises and stretching to not overexert the injured body part but also to help limit decreased range of motion long term.
3. *Strengthen*: Muscle strength is often lost during the recovery phase due to disuse of the injured body part. Once full range of motion is obtained, the focus turns to strengthening the muscles that were affected from the injury. Weight machines are

incredibly beneficial in this stage as they help patients focus on strength training exercises of specific weakened muscles while limiting riskier strength training activities that can aggravate injuries.

4. *Restore function*: The last stage of rehabilitation involves returning patients to their prior level of function. This phase will focus on coordination, balance, agility, change-of-direction capability, and rate of force development. Each of the aforementioned qualities will vary greatly depending on if you are returning a patient to a sport-specific activity versus returning them to performing their activities of daily living.

Common Fractures

For the rest of this chapter, we will discuss the different types of knee fractures, mechanism of injury, physical exam findings, diagnostic imaging, treatment, and return to activities.

Patellar Fractures

Overview

Patella fractures are uncommon and typically occur in patients aged 20–50 years of age. The most crucial aspect when diagnosing patella fractures is determining the integrity of the quadriceps tendon. Non-displaced fractures and those with intact knee extension heal well with immobilization and rehabilitation. As is common in most pediatric cases, patella fractures in children are typically avulsion or osteochondral fractures [3].

Types of patellar fractures include osteochondral, transverse polar, vertical comminuted, medial avulsion, lateral shear, central shear, and non-displaced avulsion fractures of the patella which may occur after a lateral patellar dislocation [3]. Transverse fractures are the most common type of patellar fracture [3]. Most patella fractures are due to direct forces such as a fall on the anterior aspect of the knee or striking the knee on the dashboard in a motor vehicle accident [3]. The quadriceps muscle can also contract forcefully causing a patellar fracture.

Clinical Presentation

Typically occurs from a direct blow to the knee but can occur from excessive quadriceps force. There will be immediate tenderness and swelling over anterior knee.

Physical Exam

If fracture is displaced, a defect can be felt if no significant swelling is present.

Patient will be unable to actively extend the affected knee; to test, the patient will lay supine and actively extend the affected leg 6 inches. If unable to do so secondary to pain, provider may consider injection of local anesthetic to test full knee extension capabilities.

Diagnostic Studies

- X-rays: AP, lateral, and sunrise view of the knee. The lateral view is the most useful view in delineating fracture lines and determining displacement [3].
- MRI and CT scan are rarely used for patellar fractures but can be utilized to evaluate for suspected soft tissue injury or injury to the quadriceps extensor mechanism.

A separation of more than 3 mm between fragments or an articular step off of more than 2 mm constitutes a displaced fracture [3]. This is important as it dictates whether the fracture is non-operative vs. operative.

Treatment

Urgent: Any avulsion fractures that involves the quadriceps and patellar tendon and any open fracture [3].

Operative: Severely comminuted fractures and displaced patellar fractures >3 mm between fragment or >2 mm articular step off.

Non-operative: Non-displaced patellar fractures that have a smooth articular surface with an intact quadriceps mechanism, and who are capable of extending the knee against gravity [3].

For immediate management, the knee should be placed in an immobilizer in full extension with compression and ice. Non-weight bearing status should be maintained for 5–7 days until

follow-up. Once swelling has stabilized (generally 5–7 days), a cylinder cast should be utilized for 4–6 weeks. The cast will need to go from the groin to just above the ankle malleoli with the knee in extension [3].

Non-displaced marginal vertical fractures do not have to be immobilized and can be treated with reduced activity for 3–6 weeks and progressive ROM and strengthening exercises [3].

Return to Activities

Work:

Sedentary jobs: Patients can return within a couple days for non-operative management but may be best to wait 5–7 days for initial follow-up.

Active jobs: May return after cast/brace removed and have begun functional range of motion (4–6 weeks).

Sports:

Non-operative management will be a minimum of 4–6 weeks until immobilizing cast/brace is removed. At this point, patients will gradually progress with therapy focusing on range of motion and strength training. Patients may return to full activity participation when asymptomatic.

Patellar Dislocation

Overview

Patella dislocation occurs when the patella is displaced outside of its position in the vertical groove of the knee.

Predisposing factors to patellar dislocation include obesity, young female, shallow femoral groove, joint laxity, high riding patella, genu valgum, external tibial torsion, and atrophy of the vastus medialis [4].

Dislocation of the patella typically occurs with an external pivotal motion on a partially flexed knee followed by a forceful contraction of the quadriceps that pulls the patella laterally (e.g., swinging a baseball bat, quick lateral change of direction when running) [3]. Patellar dislocation can also occur from a direct blow to the medial patella, forcing it laterally, though this is less common [3].

Clinical Presentation

Patients often present post-dislocation reduction with anterior knee pain. If patella is still dislocated on presentation, patient will maintain knee at 20–30 degrees of flexion with an obvious deformity and palpable patella laterally [3].

Physical Exam

Most of the exam findings will be along the medial aspect of the knee. Tenderness to palpation is typically found along the medial patella edge or proximal to the medial femoral condyle. A hemarthrosis can often be appreciated as well; a larger hemarthrosis volume (50 mL) suggests a more major injury to the medial stabilizers or osteochondral injury and is associated with a lower recurrence rate [3].

Diagnostic Studies

X-rays should be obtained to rule out an associated fracture even if the patella spontaneously reduced. The AP and lateral views should be examined, particularly for an osteochondral fracture of the patella, which occurs in up to 40% of cases [5].

Treatment

Urgent: Those that cannot be reduced or rare patellar dislocations (superior, inferior, or medial) require prompt orthopedic referral.

Non-operative: Initial treatment includes limitation of knee flexion with a knee immobilizer, icing, and elevation of the knee. The patient should remain non-weight bearing until seen for the initial follow-up visit in 2–3 days [3]. Early rehabilitation with emphasis on range of motion is imperative. Patients should be provided with a patellofemoral brace to be worn in the initial post-injury phase. Patients should be referred for physical therapy to focus on quadriceps strengthening. Patients should limit walking, standing, and repetitive bending.

There is conflicting reports on the benefits of operative versus non-operative management for first time dislocators [6].

Return to Activities

Work:

Sedentary: Patient is able to return to work within days as tolerable.

Active jobs: Similar to return to sport as below but will be adjusted depending on the level of activity at work.

Sports:

The criteria for return to sport is now based on clinical criteria versus weeks to months. The following must be obtained before return to sport; (1) no pain; (2) no effusion; (3) no patellofemoral instability; (4) a full range of motion; (5) nearly symmetrical strength compared to uninjured side (85–90%); and (6) excellent dynamic stability. The typical timeline to return is 6 weeks after a dislocation with non-operative management, and 3 months with operative management [7].

Tibial Plateau Fractures

Overview

The tibia is the most frequently fractured long bone. Many tibial plateau fractures are a result of a car-pedestrian accident where the bumper strikes the outside of a patient's leg with a force directed medially. Because the lateral plateau is weaker than the medial plateau, the majority of tibial plateau fractures will result in a depressed or split fracture of the lateral tibial plateau. Fractures of the medial plateau suggest a higher energy force (varus) because of the greater strength of the medial plateau. Elderly, osteoporotic patients are more likely to sustain a fracture of the tibial plateau than a ligamentous or meniscal tear after a twisting injury to the knee. Associated injuries include intercondylar eminence fractures and ACL tear as a result of hyperextension or rotatory forces [3].

It is important to get near anatomic alignment of these fractures due to the increased risk of nonunion and malunion in these fractures. Minimal displacement in tibial fractures require referral to an orthopedic surgeon [3].

Clinical Presentation

Patients with tibial plateau fractures will have a painful swollen knee and are unable to bear weight on the ipsilateral leg.

Physical Exam

Tenderness over the proximal tibia and limited flexion and extension of the knee. To ensure a more thorough knee exam, aspiration of effusion and injection of local anesthetic are indicated. Test the stability of the knee by stressing the knee in varus and valgus throughout range of motion; a stable knee will have less than 10 degrees of joint widening on varus and valgus stress from full extension to 90 degrees of flexion. The ACL should also be examined as it is commonly injured in this type of fracture [3].

Diagnostic Studies

- X-ray*:* AP, lateral, and intercondylar notch views of the knee.
- Fracture types: [2]
  - Type 1: Lateral plateau split fracture.
  - Type 2: Lateral split/depression fracture.
  - Type 3: Lateral plateau depression.
  - Type 4: Medial plateau split fracture.
  - Type 5: Bicondylar plateau fracture.
  - Type 6: Fracture with metaphyseal-diaphyseal separation.
  - Type 4–6 Typically result from high-energy trauma.
- CT Scan:

  If plain radiographs above are equivocal but there is still high clinical suspicion for a fracture, additional imaging using a CT scan is warranted. MRI has become more advantageous due to its superior ability to detect associated ligamentous and meniscal injuries [3].

Treatment

Urgent: A significantly comminuted, open fracture associated with vascular injury, compartment syndrome or knee dislocation require emergent surgery [3].

Operative: X-rays with greater than 3 mm step off and less than 5 mm gapping.

Non-operative management: Patient's knee should be immobilized in a long-leg splint from the thigh to the metatarsals in full extension and the ankle at 90°. This can be transitioned to hinged brace at full extension with gradual flexion adjustment from 0 to 4 weeks. At 4 weeks, 90 degrees of flexion is allowed; if not obtained at 4 weeks, physical therapy is indicated [3].

Along with the above weekly hinge adjustments, weekly knee X-rays should be obtained to ensure correct bone healing and alignment (if malalignment, patient should be referred to specialist immediately for correct alignment).

Non-weight bearing status is maintained for at least 6 weeks, until bone healing is appreciated, and partial weight-bearing with crutches until bone healing appears complete on radiographs (8–12 weeks).

Hinged brace use should continue until 8–12 weeks, whenever union of the bone is achieved.

Operative management: Any displaced or depressed tibial plateau fractures with associated ligamentous or meniscal injuries should be referred to an orthopedic surgeon [3].

Return to Activities

Work:

Sedentary jobs: Once a patient begins flexing the knee and becomes partial weight bearing (around 6 weeks) working full time should be manageable. Returning to work earlier can be achieved depending on pain tolerance.

Active jobs: These may be gradually resumed after a solid union is present and full weight bearing is well tolerated [3].

Sports:

Athletes should be able to safely resume non-weight bearing aerobic activities such as swimming or biking after full weight bearing is started. Sports specific activity will then gradually progress based on the patient's tolerance.

Tibial Shaft Fractures

Overview

Tibial shaft fractures occur as a result of three types of mechanisms: 1) Low-energy forces such as a sports injury, 2) Rotational forces on a fixed foot 3) High-energy forces such as falls from a significant height [3]. Low-energy injuries typically lead to distal fractures, whereas high-energy injuries typically lead to proximal fractures. Oblique and spiral fractures occur as a result of indirect forces, whereas transverse and comminuted fractures occur as a result of direct trauma. The degree of comminution is usually proportional to the amount of energy that caused the fracture. The anteromedial aspect of the tibia is at higher risk of fracture from direct blow due to its relatively superficial position [3].

Clinical Presentation

Inability to bear weight and significant pain over the fracture site.

Physical Exam

Swelling and obvious deformity of tibia typically seen (unless non-displaced).

Open fractures are common in these types of fractures so thorough skin evaluation is imperative. Dorsalis pedis and tibialis posterior pulses should be identified and examined. Tibial shaft fractures are one of the most common causes of compartment syndrome, so prompt evaluation is a must (please see beginning of chapter for more detail on compartment syndrome).

Diagnostic Studies

- *X-rays:* AP and lateral views of the entire tibia, including the knee and ankle joints, should be obtained.
- *MRI and CT* are typically not indicated for tibia shaft fractures unless arterial damage is suspected [3].
- Fracture type: [2]
  - Transverse fracture (fibula intact).
  - Spiral fracture with shortening.
  - Comminuted fracture with marked shortening.
  - Segmental fracture with marked shortening.

A tibia shaft fracture is considered non-displaced if it meets the following criteria: less than 5 mm of displacement and less than 10° of angulation in both the AP and mediolateral planes, and less than 10° of rotation [3].

Treatment

For all treatments of tibial shaft fractures, patient will need to be immobilized and placed in a long-leg splint.

Urgent: Any fracture that is open has neurovascular injury, or knee/ankle dislocation requires immediate orthopedic surgery referral.

Operative: Comminuted and segmental fractures.

Non-operative: Non-displaced fractures as described above. The leg should be immobilized in a long-leg posterior splint from the metatarsals to the upper thigh, with the knee in 10–15 degrees of flexion and the ankle at 90° [3]. Patient must remain non-weightbearing and consideration for hospitalization for observation may be warranted in cases with higher concern for compartment syndrome.

Circumferential cast applied after swelling has decreased (typically 3–5 days). To prevent excess motion above and below the fracture, the cast should extend from the thigh to the metatarsal heads [3].

Return to Activities

Return to work or sports depends on the severity of the injury.

Work:

Sedentary jobs: Able to return as soon as the long-leg cast is discontinued.

Active jobs: Similar to return to sport as below but will be adjusted depending on the level of activity at work.

Sports:

Non-weight bearing activities initially when out of the long-leg cast followed by progressive supervised rehabilitation

for several months to restore motion and strength. Return to full activity depends on the severity of the fracture and any associated injuries.

Proximal Fibular Fractures

Overview

Isolated proximal fibula fractures are uncommon and are typically associated with more serious injuries. Fibular fractures are caused by a direct blow to the lateral leg but can also occur from an avulsion of the lateral collateral ligament or significant external rotational force about the ankle (The Maisonneuve fracture). In order to distinguish complicated from uncomplicated fractures, the practitioner will have to recognize compartment syndrome or any neurovascular and ligamentous injuries [3].

Clinical Presentation

Patient will have lateral knee pain along the fracture site that may span the length of the fibula with diffuse swelling and effusion on initial assessment. Lateral compartment syndrome uncommon but can occur with crush injuries [3].

Physical Exam

Testing the actions of the peroneal nerve including dorsiflexion and plantarflexion of the ankle. Evaluate for paresthesias over the lateral aspect of the middle to distal leg and dorsum of the foot. Dorsalis pedis and posterior tibial artery will need to be palpated. Will also need to investigate the integrity of the interosseous membrane.

Diagnostic Studies

- *X-rays*: AP and lateral views of the entire fibula.
- MRI and CT are only utilized if associated arterial or ligamentous is suspected.

Treatment

Initial immobilization in a long-leg knee immobilizer in near full extension should be used until the patient is seen by the orthopedic surgeon. Isolated fractures of the fibula shaft heal well with minimal treatment and are treated symptomatically.

Urgent: Crush injuries, open fractures, associated tibial fracture, compartment syndrome, or peroneal nerve injury involvement all require immediate orthopedic surgery consultation.

Operative: Patients with displaced or comminuted fractures, fractures with concomitant knee or ankle joint instability, and proximal head or neck fractures must be referred to an orthopedic surgeon [3].

Non-operative: Non-displaced fractures, a patient with an isolated fibular fracture should be placed in a stirrup splint with the ankle at 90°. Patients will need to remain non-weight bearing until follow-up, typically around 3–5 days.

A short-leg walking cast or cast boot is indicated for relief of moderate-to-severe pain and should be applied for 3–4 weeks. At this point patients can move to an as-needed splint with full healing of the fracture expected around 6–8 weeks [3].

Return to Activities

ROM and calf strengthening exercises should be started after immobilization period (3–4 weeks).

Work

Sedentary jobs: May return to work within 1-week post-injury.

Active jobs: Return to work after rigid immobilization cast removed (3–4 weeks).

Sports:

Athletes can return to high-level sports as soon as they have near-normal ankle motion and lower extremity strength [3].

Stress Fractures

Overview

Stress fractures are relatively common overuse injuries seen in athletes, particularly in running athletes. Stress fractures account for 0.7–20% of all sports medicine clinic injuries [8]. Tibial stress fractures are more common than fibular stress fractures.

Most studies of stress injuries cite some alteration in the training program as the most significant factor in producing the injury. It has been well documented that there is an increased injury rate with increasing distance beyond approximately 32 km/week. Hard training surfaces play a role in acceleration of stress fractures [8].

Clinical Presentation

Patient presents with pain after or toward the end of physical activity at the location of the stress injury, which is in contrast to ligamentous injuries which occurs at the beginning of physical activity and lasts longer [8].

Physical Exam

The most pertinent and typically only physical exam finding is focal tenderness and swelling at the site of the stress fracture.

Diagnostic Studies

- *X-rays*: Typically stress fracture is not evident within the first 2–4 weeks after fracture. Localized periosteal thickening, cortical sclerosis, or a true fracture line are positive findings of tibial shaft stress fractures [3].
- *MRI*: This is the gold standard for identifying stress fractures. Useful in distinguishing soft tissue injuries and shin splints from stress fractures [3].

Treatment

Stress fractures are non-urgent and non-surgical unless the stress fracture leads to a full tibial shaft fracture (refer to above section for this scenario).

Operative: As above, these injuries are non-surgical.

Non-operative: The initial treatment is rest and immobilization in a long air splint or hinged knee brace for 1–2 weeks. The patient should remain non-weight bearing as long as there is pain with walking. Once the patient can walk with little to no pain, only partial use of crutches is needed followed by progression to unassisted ambulation [3].

Return to Activities

Work:

Sedentary jobs: May return as soon as pain is controlled.

Active jobs: May return to work within a few weeks with an air splint.

Sports:

May return to full activity around 8-weeks post-injury.

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Rheumatic and Infectious Causes of Knee Pain

9

Mark Riley and Michelle Leong

Rheumatic Knee Pain

This chapter will focus on some of the more common autoimmune and autoinflammatory conditions which can cause knee pathology. Autoimmune diseases result from abnormalities of the adaptive immune system and usually involve the generation of autoantibodies. Examples of autoimmune disease include rheumatoid arthritis and systemic lupus erythematosus (SLE). Autoinflammatory diseases result from abnormalities of the innate immune system and include disorders like Adult-onset Still's disease and familial Mediterranean fever.

Autoimmune or autoinflammatory causes for knee pain should be considered in the differential diagnosis of knee pain particularly if there is evidence of inflammatory arthritis. Inflammatory arthritis is characterized by synovitis with warmth,

M. Riley (✉)
Division of Rheumatology, Rush University Medical Center, Chicago, IL, USA
e-mail: mark_l_riley@rush.edu

M. Leong
Department of Rehabilitation and Human Performance, Icahn School of Medicine at Mount Sinai, New York, NY, USA

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swelling, pain, and reduced range of motion. A joint effusion may also be present, often in the suprapatellar region. Patients may also report prolonged stiffness with inactivity or upon awakening that improves with physical activity. This is in contrast to the symptoms often reported in patients with osteoarthritis with relatively short periods of stiffness and pain which worsens with physical activity.

Knee joint involvement in autoimmune or autoinflammatory disease can present as monoarticular arthritis or in the context of polyarticular arthritis. Extra-articular structures around the knee joint may also be affected. Many of the autoimmune and autoinflammatory diseases are systemic, warranting a comprehensive approach to history, and physical examination. Extensive serologic testing may be tempting when considering these disorders, but this approach is costly and can easily lead to confusing results, further clouding the clinical picture. It is best to use the history and physical examination to hone a differential diagnosis and consider serologic testing if a specific diagnosis is suspected.

History

When considering autoimmune or autoinflammatory etiologies of knee pain, one should tailor their history and examination to evaluate for evidence of a systemic autoimmune or autoinflammatory process in addition to the typically taken history (Table 9.1).

Additionally, pay particular attention to a family history of autoimmune or autoinflammatory disease, especially among first degree relatives.

Crystalline Arthropathies

Crystals can deposit in and around joint tissues, and the resulting immune response to these crystals may lead to inflammatory arthritis. The mere presence of crystals does not always equate to

Table 9.1 History and examination key points in evaluating for rheumatic diseases

Constitutional	Changes in weight, particularly unintentional weight loss which may signal an underlying inflammatory process. Recurrent fevers often reflect systemic inflammation and can be a symptom of infectious or rheumatic disease
Ocular	Assess for symptoms or a history of episodic eye redness associated with pain, photophobia or vision changes. These features may suggest a history of uveitis or scleritis
Ear, nose, throat (ENT)	A history of episodic swelling, redness, and pain of the ears or nose may suggest relapsing polychondritis or granulomatosis with polyangiitis (GPA). Recurrent epistaxis or nasal perforation can also suggest GPA. A history of oral and/or nasal ulcers can be symptoms of systemic lupus erythematosus (SLE) or systemic vasculitis
Cardiovascular	Chest pain with pleuritic features may be a sign of serositis. Chest pain which worsens with supine positioning which improves with seated position is classically a feature of pericarditis which can be a component of numerous rheumatic diseases including SLE, vasculitis and rheumatoid arthritis
Respiratory	Symptoms of new onset dyspnea with exertion or rest. Also querying about hemoptysis, which may be a symptom of systemic vasculitis
Gastrointestinal (GI)	If considering inflammatory bowel disease (IBD) associated arthritis or reactive arthritis, a GI review of systems is essential. Assess for symptoms or a history of recurrent abdominal pain, nausea, vomiting, diarrhea, melena and hematochezia
Genitourinary (GU)	Assess for symptoms or a history of genital ulcers or recent GU infection. Genital ulcers in the setting of knee inflammatory arthritis may suggest Behçet's disease. Knee inflammatory arthritis in the setting of a recent GU infection suggests reactive arthritis or gonococcal arthritis
Neurologic	Assess for symptoms or a history of sensory and/or motor neuropathy. For example, mononeuritis multiplex in the setting if knee inflammatory arthritis would suggest a systemic vasculitis

(continued)

Table 9.1 (continued)

Vascular	Assess for Raynaud's phenomenon which can be associated with a variety of rheumatic diseases
Musculoskeletal	Assess for pain or swelling of other joints beyond the knee. This includes asking about back pain. Inflammatory back pain is classically characterized by pain that is worse with inactivity, worse at night, and improves with physical activity
Integumentary	Assess for a history of skin rash, including those that are photosensitive, or skin ulcers. Many rheumatic diseases present with articular and skin involvement including psoriatic arthritis, reactive arthritis, adult-onset Still's disease, sarcoidosis, SLE, and systemic vasculitis

active inflammatory arthritis. The most common crystals which deposit in joints are monosodium urate (MSU) and calcium pyrophosphate. Other crystals include hydroxyapatite and calcium oxalate. The presence of crystals in synovial fluid does not rule out infection, and gout/pseudogout may present concurrently with septic arthritis. Conversely, acute crystalline arthropathy alone may present with fever and leukocytosis, mimicking infection. This underscores the importance of arthrocentesis and synovial fluid analysis.

Gout

Gout is the most common cause of crystalline arthropathy and commonly affects the knee in addition to other joints including the first metatarsophalangeal joint and ankle. Gout is caused by long-standing hyperuricemia which leads to deposition of MSU crystals. Crystal deposits may eventually form tophi and can progress to chronic, erosive arthritis. Acute gout may be triggered by a variety of etiologies including infection, trauma, sur-

gery, and stress including hospitalization. Medications including hydrochlorothiazide, loop diuretics, calcineurin inhibitors, angiotensin converting enzyme inhibitors, and pyrazinamide may lead to hyperuricemia. Other factors include consumption of purine-rich foods including red meat, seafood, beer, and other alcoholic beverages are also associated with hyperuricemia.

Clinical Presentation Gout frequently presents with acute onset joint pain with associated warmth, swelling, and decreased range of motion. Pain from acute crystalline arthropathy can be excruciating and patients will frequently complain of discomfort even with light palpation of the affected joint. When the knee is affected, a suprapatellar joint effusion is often present. Collections of uric acid precipitate, known as tophi, may be present in patients with long-standing, untreated gout. Tophi manifest as subcutaneous nodules that can be found around the knee, but are also commonly found on the medial aspect of the first MTP, extensor surfaces or even the pinna [1].

Diagnostic Studies Gout is diagnosed with arthrocentesis which often demonstrates inflammatory synovial fluid with white blood cell counts frequently exceeding 50,000/μL. Occasionally, white blood cell counts may be greater than 100,000/μL, mimicking septic arthritis, but gram stain will show no organisms and fluid culture will be negative. MSU crystals can be identified as needle-shaped, negative birefringent crystals under polarized light microscopy. Visualization of MSU crystals within neutrophils signifies acute gout. Serum urate levels can be checked, but may be normal during an acute gout attack. A high serum urate level may be helpful in establishing a history of hyperuricemia, but a normal or even low serum urate level does not rule out acute gout. Inflammatory markers like erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels are typically elevated in acute gout.

Imaging Radiographs of the knee may show a joint effusion, tophi, or erosions with overhanging edges sometimes referred to as "rat bite" lesions [2].

Treatment Treatment of gout depends on whether the presentation is acute or chronic and if one or multiple joints are affected. For acute monoarticular gout of the knee, arthrocentesis can be both diagnostic and therapeutic. Intra-articular glucocorticoid injection, usually with triamcinolone or methylprednisolone, is very effective, but it is important to rule out infection before this is done. Other treatment options include a course of non-steroidal anti-inflammatory drugs (NSAIDs), colchicine, systemic glucocorticoids, or anakinra. Systemic glucocorticoids or anakinra are generally reserved for polyarticular flares. Treatment is often dictated by the patient's comorbidities, including diabetes, renal impairment or recent surgery, and current medications.

Long-term management of gout consists of urate-lowering therapy with the goal of reduced number of flares and progression of erosive disease. Commonly used medications include the xanthine oxidase inhibitors like allopurinol or febuxostat, and uricosuric agents such as probenecid. Pegloticase, a recombinant uricase enzyme, may be indicated in refractory cases of gout. These medications can paradoxically be associated with increased risk of gout flare within the first 3–6 months of initiating therapy. As such, low-dose colchicine, low-dose prednisone or NSAIDs are commonly prescribed during this period to mitigate flare risk [3].

Return to Activity There are no specific recommendations regarding return to activity, so return is based on level of pain and the patient's ability to tolerate physical activity. There is growing evidence that exercise is beneficial and may have anti-inflammatory properties in gout and other rheumatic diseases [4].

Calcium Pyrophosphate Deposition Disease (CPPD)

Acute calcium pyrophosphate deposition disease (CPPD) is also known as pseudogout. CPPD arthropathy is most often seen in older adults over the age of 60, and age is considered a risk factor for the development of the disease. Other risk factors include hemochromatosis, hypomagnesemia, and hyperparathyroidism. These etiologies should be considered particularly in patients with CPPD who are less than 60 years of age [5].

Clinical Presentation CPPD presents similarly to gout with severe pain, swelling, and limited range of motion. A moderate to large joint effusion may be present. The knee joint is the most commonly affected. CPPD is often associated with osteoarthritis and chondrocalcinosis, the deposition of calcium pyrophosphate in cartilage. CPPD may also present as a chronic inflammatory polyarthritis which can mimic rheumatoid arthritis.

Diagnostic Studies As with gout, synovial fluid analysis is usually inflammatory with white blood cell count ranging from 5000 to 100,000/μl. The presence of intracellular rhomboid shaped, positively birefringent crystals under polarized light is diagnostic for CPPD arthropathy. Patients can have both gout and CPPD concurrently, indicated by the presence of intracellular MSU and calcium pyrophosphate crystals in synovial fluid. Testing serum magnesium, parathyroid hormone, and iron studies should be particularly considered in patients with CPPD who are less than 60 years of age. ESR and CRP are usually elevated.

Imaging Radiographs of the knee showing chondrocalcinosis of the knee cartilage can be a clue that CPPD is present. However, chondrocalcinosis may be found incidentally on radiographs without overt CPPD. Isolated patellofemoral joint space narrowing is also a common, unique feature of knee CPPD [2].

Treatment Acute CPPD is treated much like acute gouty arthritis with either NSAIDs, colchicine or glucocorticoids. If the knee is the only joint affected, glucocorticoid injection can be considered. Treatment options for chronic CPPD can include colchicine or hydroxychloroquine [6].

Return to Activity As with gout, there are no specific recommendations regarding return to activity. Return is based on the level of pain and the patient's ability to tolerate physical activity.

Rheumatoid Arthritis

Rheumatoid arthritis (RA) typically presents as an erosive, inflammatory polyarthritis, but oligoarthritis or monoarthritis may occur as well. RA most frequently affects the metacarpophalangeal and proximal interphalangeal joints of the hands, the feet, and yv wrists, but large joints like the knee are also commonly affected [7]. RA usually develops with a gradually increasing number of involved joints over a period of weeks to months.

Clinical Presentation Rarely, monoarthritis of the knee may be the presenting feature of RA. In this setting, the knee can be acutely inflamed with warmth, swelling, tenderness to palpation, and reduced range of motion. A joint effusion may be present in this case along with synovial thickening. Conversely, an acutely swollen knee in a patient with known rheumatoid arthritis with otherwise well-controlled disease should raise suspicion for septic arthritis. Patients with long-standing, uncontrolled RA may develop flexion contractures at the knee and valgus deformity. Other physical exam features may be boutonniere or swan neck deformities of the fingers, deformities of the toes including hammertoes, and subcutaneous nodules on the extensor surfaces known as rheumatoid nodules.

Diagnostic Studies Common laboratory features of active rheumatoid arthritis include elevated inflammatory markers, anemia of chronic disease, and thrombocytosis. Serology may be positive

for rheumatoid factor (RF) and antibodies to cyclic citrullinated peptides8 (anti-CCP). Antinuclear antibodies may also be present. It is important to recognize that RF and anti-CCP are absent in a significant subset of patients, termed seronegative rheumatoid arthritis. Seropositive RA tends to be more severe and have more extra-articular manifestations including interstitial lung disease, scleritis, and rheumatoid vasculitis [8].

Imaging Radiographs of an affected knee in rheumatoid arthritis may demonstrate a joint effusion. Other common radiographic features include periarticular osteopenia, erosions, and symmetric joint space narrowing, particularly in long-standing and untreated disease [2].

Treatment Treatment of knee involvement in rheumatoid arthritis is generally managed in a similar fashion to RA affecting other joints. If infection has been ruled out, corticosteroid injection into an acutely swollen knee can provide significant pain relief. Systemic steroids may be warranted for a polyarticular RA flare. Methotrexate is the cornerstone of long-term management of rheumatoid arthritis as a conventional synthetic disease-modifying antirheumatic drug (csDMARD). Other csDMARDs including leflunomide, hydroxychloroquine, and sulfasalazine are options, the latter two of which may be combined with methotrexate. If moderate to high doses of methotrexate are unable to adequately control disease activity, biologic DMARDs (Table 9.2) or targeted synthetic DMARDs are considered in addition to methotrexate. Patients with end-stage joint damage of the knee from RA should be evaluated for joint replacement [7].

Return to Activity Physical activity and exercise is encouraged in patients with rheumatoid arthritis, and are associated with lower disease activity and pain scores. This applies to aerobic exercise, resistance training, and a combination of the two [9]. As with other rheumatic diseases, rheumatoid arthritis patients have an increased risk of cardiovascular disease. Exercise may help to mitigate this risk. Patients with damage and secondary osteoarthritis from RA benefit from targeted physical therapy to address

Table 9.2 Biologic DMARDs used in rheumatoid arthritis

Biologic DMARDs used in rheumatoid arthritis
Anti-tumor necrosis factor-α inhibitors (adalimumab, infliximab, etanercept, certolizumab pegol, golimumab)
IL-6 inhibitors (tocilizumab, sarilumab)
Rituximab: Binds to CD20 leading to B lymphocyte depletion
IL-1 receptor antagonist (anakinra)
Abatacept: CTLA-4 IgG, interferes with T cell co-stimulation
Targeted synthetic DMARDs
Janus kinase inhibitors (tofacitinib, baricitinib, upadacitinib)

specific muscle strength imbalances. Unfortunately, many rheumatoid arthritis patients are physically inactive, underscoring the importance of physicians discussing its benefits and encouraging patients to exercise.

Systemic Lupus Erythematosus (SLE)

Arthralgia is the most common musculoskeletal manifestation of SLE [10]. While tendonitis and tenosynovitis are common in SLE, true tendon rupture such as at the infrapatellar tendon is unusual [11]. When inflammatory arthritis is present, it is usually polyarticular and may involve the knee joint. Unlike rheumatoid arthritis, SLE usually causes non-erosive inflammatory arthritis. However, there is a subset of SLE patients whose joint disease behaves like rheumatoid arthritis and leads to significant erosions and joint deformities. These patients often have positive anti-CCP antibodies and are considered to be a lupus-rheumatoid arthritis overlap sometimes referred to as "rhupus" [12]. Arthralgia and arthritis can also be features of drug-induced lupus. Common drugs associated with drug-induced lupus include hydralazine, procainamide, isoniazid, and minocycline. TNF-α inhibitors have also been associated with drug-induced lupus [13].

Clinical Presentation In a lupus patient with knee pain, physical examination may simply demonstrate joint line tenderness to palpation. If inflammatory arthritis is present, the knee will be warm,

tender to palpation, have reduced range of motion, and often swelling with joint effusion. Poorly localized extra-articular pain, particularly if associated with pain in other body regions, may indicate myofascial pain from fibromyalgia which commonly occurs with SLE.

SLE can have a wide array of clinical manifestations with varying severity. Some specific extra-articular manifestations that can help aid in the diagnosis include skin rash, oral ulcers, nasal ulcers, serositis, nephritis, cytopenias, and secondary antiphospholipid antibody syndrome. SLE patients are also at risk of developing avascular necrosis, particularly due to chronic steroid use. The femoral heads are most commonly affected, but other sites like the distal femur may be affected as well [10].

Diagnostic Studies Laboratory testing in patients with SLE may show cytopenias including leukopenia, lymphopenia, anemia, and thrombocytopenia. Elevated serum creatinine, proteinuria, and hematuria may signal underlying lupus nephritis.

Serology in SLE almost always includes a positive antinuclear antibody (ANA). Other antibodies that may be seen include anti-Smith, anti-double stranded DNA, ribonuclear protein (RNP), anti-Ro, and anti-La. Low serum C3 and C4 complement levels are also common, and may be a sign of active disease if they are lower than a patient's established baseline. Rising double stranded DNA antibody titers can also be a sign of active disease, particularly lupus nephritis [10]. Thus, it is common for an SLE patient's rheumatologist to routinely monitor double stranded DNA antibody, C3 and C4 levels. Titers of other antibodies, including ANA, do not correlate with SLE disease activity and thus do not need to be repeated if they have been previously tested. Abnormal coagulation studies, especially a prolonged activated partial thromboplastin time, may signal the presence of a lupus anticoagulant and should prompt a workup for antiphospholipid antibody syndrome.

Anti-histone antibodies are seen in the majority of drug-induced lupus cases, but are not specific for the diagnosis as they can be seen in patients with SLE as well [13].

Treatment Treatment of SLE depends upon which clinical manifestations are present and their severity. Hydroxychloroquine is standard background therapy for nearly all SLE patients, and can be used for mild SLE-associated arthritis. Monoarthritis of a joint like the knee is amenable to steroid injection. For more severe polyarticular disease, methotrexate, azathioprine, or mycophenolate mofetil can be efficacious. If response to methotrexate is inadequate, addition of biologics such as belimumab or rituximab may be considered [14].

Return to Activities There are no specific recommendations regarding exercise modifications or return to activity in patients with SLE. Therapeutic exercise programs have not been found to worsen disease activity in SLE and instead may help improve levels of fatigue and depression [15].

Spondyloarthropathies

Spondyloarthritis (SpA) is categorized into inflammatory bowel disease (IBD)-associated SpA, psoriatic arthritis (PsA), reactive arthritis (ReA), and axial spondyloarthritis (AxSpA). While these are considered distinct disorders, the spondyloarthropathies can have phenotypic overlap.

Clinical Presentation All forms of SpA present with inflammatory arthritis. SpA may also present with enthesitis, inflammation of the tendinous insertion onto bone, which usually presents with swelling and/or tenderness over the entheses. Commonly affected areas include the plantar fascia, patellar, and Achilles tendon insertions [16]. Infrapatellar enthesitis presents with tenderness to palpation at the tibial tubercle. Localized swelling may also be present. Dactylitis, swelling of an entire digit, may also occur with any form of SpA. The presence of either enthesitis or dactylitis can help distinguish SpA from other causes of inflammatory arthritis like RA or SLE.

Diagnostic Studies Inflammatory markers like erythrocyte sedimentation rate and C-reactive protein are usually elevated in SpA, but normal levels do not rule out these diagnoses. Unlike many other autoimmune diseases, autoantibodies are typically absent in the spondyloarthropathies. The presence of HLA-B27 is associated with the spondyloarthropathies with AxSpA having the highest association [17]. However, HLA-B27 positivity is not a prerequisite for the diagnosis of SpA.

Imaging Radiographs of the knee in all forms of SpA may show symmetrical joint space narrowing, erosions, and joint effusion which reflect inflammatory arthritis. Bony proliferation or periostitis is a common radiographic feature of SpA. It may occur next to areas of erosion, along the bone shaft, across joints and at entheses [2]. This may be seen in the lateral view with bony proliferation at the superior and inferior surfaces of the patella.

Return to Activity No specific recommendations exist regarding return to activity for the various forms of SpA. In general, during acute manifestation of disease, weight bearing is as tolerated and as symptoms resolve, activities can progress. The European League Against Rheumatism (EULAR) guidelines strongly recommend the promotion of regular physical activity including aerobic activity, muscle strength, flexibility, and neuromotor performance for patients with osteoarthritis, rheumatoid arthritis, and spondyloarthritis [18].

Inflammatory Bowel Disease-Associated Spondyloarthritis

Clinical Presentation Inflammatory bowel disease (IBD)-associated spondyloarthritis (SpA) is a common extraintestinal manifestation of IBD. It is seen with Crohn's disease, ulcerative colitis, and microscopic colitis. IBD-associated arthritis may

present as peripheral SpA, sacroiliitis, axial SpA or a combination of the three. The knee is one of the most commonly affected joints [19]. While most IBD-associated SpA develops after IBD diagnosis, a significant proportion of patients may develop joint disease before intestinal manifestations. IBD-associated peripheral SpA is typically categorized into one of two types.

Type I peripheral SpA: often an oligoarticular arthritis which tends to occur early in the disease course of IBD. Like other extraintestinal manifestations of IBD, type I parallels bowel disease activity. This form is non-erosive, usually resolves within several months and does not typically require immunosuppressive therapy.

Type II peripheral SpA: Less common than Type I. This form of IBD-associated arthritis is most commonly polyarticular and affects the small joints of the hands although the knee joints may be involved as well. Type II is a chronic, erosive inflammatory arthritis that progresses independently of bowel disease activity. Immunosuppression is usually needed [19].

IBD-associated SpA should be considered in any patient with a known history of IBD who presents with knee pain. If inflammatory arthritis of the knee is present, the knee will be warm, tender to palpation with reduced range of motion. Swelling with joint effusion may be present. Localized tenderness or swelling at the tibial tubercle may indicate enthesitis. If IBD is present, patients will frequently complain of recurrent abdominal pain. They may also have varying degrees of diarrhea and hematochezia. Crohn's disease patients may develop oral ulcers and anal fissures. Fistula formation may also occur.

Diagnostic Studies Laboratory abnormalities in IBD-associated SpA may include elevated inflammatory markers like ESR and CRP. Anemia may reflect iron deficiency or chronic inflammation. Crohn's disease patients with small intestinal involvement may have features of malabsorption. As mentioned previously,

knee radiographs may show symmetrical joint space narrowing, erosions, periostitis, and joint effusion.

Treatment Many of the treatments used for IBD are effective for peripheral SpA, including sulfasalazine, methotrexate, and TNF-alpha inhibitors with the exception of etanercept. When considering treatment for IBD-associated SpA, it is important to identify the presence of peripheral SpA, axial SpA, or both, as certain medications which are effective for peripheral SpA may be ineffective for axial SpA. For example, sulfasalazine and methotrexate are effective for peripheral SpA but ineffective for axial disease. Vedolizumab, which is effective for bowel disease, is not effective for either peripheral or axial SpA. Conversely, the IL-17 inhibitors secukinumab and ixekizumab are effective for SpA but ineffective for IBD [20].

Reactive Arthritis

Reactive arthritis (ReA) is characterized by inflammatory monoarticular or oligoarticular arthritis that occurs within days to weeks of a recent infection, usually gastrointestinal or urogenital. *Chlamydia trachomatis* causing urethritis in men and cervicitis in women is one of the most commonly-associated pathogens with ReA. Causes of gastrointestinal infections with associated ReA include *Salmonella* and *Shigella* species, *Campylobacter jejuni*, and *Yersinia* species. More recently infections of other organisms including *Clostridiodes difficile* and *Chlamydia pneumoniae* have been recognized [21].

Clinical Presentation ReA primarily affects large joints of the lower extremities, with the knee being the most commonly involved [22]. If inflammatory arthritis of the knee is present, the knee will be warm, tender to palpation, and with reduced range of motion. Swelling with joint effusion may be present. Enthesitis is a common feature of ReA and may manifest as

patellar tendinopathy as described previously. ReA can have extra-articular manifestations as well. Ocular manifestations include conjunctivitis and uveitis. Cutaneous manifestations like keratoderma blennorrhagicum or circinate balanitis may occur. Historically the triad of arthritis, conjunctivitis and urethritis was referred to as Reiter syndrome, but this term has fallen out of favor.

Diagnostic Studies HLA-B27 is positive in up to 50–80% of patients with ReA, but is not a prerequisite for the diagnosis. Patients who have HLA-B27 appear to be at increased risk for a more severe disease course, are more likely to have axial involvement and have extra-articular manifestations [22].

Treatment Reactive arthritis is usually self-limited, resolving within several months. NSAIDs are considered a first line treatment. Monoarticular involvement of a lower extremity joint like the knee may be managed with intra-articular steroid injection. A short course of systemic glucocorticoids may be warranted for oligoarticular or polyarticular disease. Some patients may develop a chronic disease course lasting greater than 6 months. In these cases, consideration may be given to conventional synthetic DMARDs like sulfasalazine or methotrexate, although these medications are not effective for enthesitis or dactylitis. If these therapies are ineffective, TNF-alpha inhibitors may be considered. Research studies have evaluated whether a prolonged course of antibiotics is beneficial in patients with *Chlamydia*-associated ReA, but with mixed results [22].

Psoriatic Arthritis

Psoriatic arthritis (PsA) is an inflammatory arthritis associated with psoriasis. Estimates in the literature report PsA occurs in up to 30% of patients with psoriasis [23]. The knee may be affected in PsA, but is not the most commonly affected joint. PsA has historically been categorized into one of five subtypes [24].

1. Asymmetric oligoarticular arthritis: Knee and ankle joints may be involved, but most commonly affects the distal interphalangeal (DIP), proximal interphalangeal (PIP), metacarpophalangeal (MCP), and metatarsophalangeal (MTP) joints.
2. Predominant DIP involvement.
3. Polyarticular: can resemble rheumatoid arthritis in its distribution affecting the PIPs, MCPs, and wrists.
4. Arthritis mutilans: Variant with severe erosions and joint deformities with telescoping of digits.
5. Isolated axial spondyloarthritis: sacroiliac and vertebral involvement. Classically, sacroiliac involvement in PsA is unilateral.

Psoriasis develops about 10 years before the onset of inflammatory arthritis in about 70% of cases. Roughly 15% of patients will develop psoriasis and inflammatory arthritis together. This group of patients will often experience simultaneous flares of their skin and articular disease [23].

If inflammatory arthritis of the knee is present, the knee will be warm, tender to palpation with reduced range of motion. Swelling with joint effusion may be present. As with other forms of SpA, enthesitis is a common feature. When considering PsA in the differential diagnosis for inflammatory arthritis of the knee, it is important to both query and closely examine the patient for the presence of psoriatic plaques or nail changes. Psoriasis does commonly affect the skin over extensor surfaces, but small plaques may only affect skin behind the ears, the intertriginous regions of the groin, or gluteal cleft. At the groin site, lesions may appear as plaques without scale, sometimes called inverse psoriasis.

Diagnostic Studies Laboratory studies may show elevated ESR and CRP, but these are non-specific findings. HLA-B27 may be positive but is not a prerequisite for diagnosis.

Imaging Radiographic findings of the knee may show symmetric joint space narrowing with marginal erosions with adjacent bony proliferation, and additional bony resorption [2]. X-rays may also show periostitis and bony spurs at areas of enthesitis.

Table 9.3 Treatments for psoriatic arthritis

Conventional synthetic DMARDs for PsA
NSAIDs
Methotrexate
Leflunomide
Sulfasalazine
Glucocorticoids
Intra-articular steroids can be beneficial for patients with knee inflammatory arthritis due to PsA
Systemic steroids may be considered in severe disease, but there is a chance of significant worsening of skin disease if steroids are withdrawn
Biologic DMARDs for PsA
Anti-tumor necrosis factor-α inhibitors (adalimumab, infliximab, etanercept, certolizumab pegol, golimumab)
IL-12/23 inhibitor (ustekimumab)
IL-17 inhibitors (ixekizumab, secukimumab)
IL-23 inhibitors (guselkumab is FDA approved for PsA)
Abatacept: CTLA-4 IgG, interferes with T cell co-stimulation
Targeted synthetic DMARDs for PsA
JAK inhibitors: Tofacitinib is approved for PsA
Phosphodiesterase (PDE) 4 inhibitor (apremilast)

Treatment There are numerous treatments available for PsA, many of which are also effective for skin disease [25] (Table 9.3).

Axial Spondyloarthritis

As the name implies, axial spondyloarthritis (AxSpA) primarily affects the spine and bilateral sacroiliac joints, but may have a peripheral component. Ankylosing spondylitis refers to axial SpA with radiographic evidence of disease, which differs from non-radiographic AxSpA in which spondyloarthritis or sacroiliitis may only be visible on magnetic resonance imaging [17].

Clinical Presentation As with other forms of SpA, knee involvement in AxSpa often presents as inflammatory arthritis. Enthesitis of the patellar tendon may be present, manifesting as tenderness over the tibial tubercle sometimes with localized swelling.

Treatment csDMARDs like methotrexate, leflunomide, and sulfasalazine, which are effective for peripheral SpA, are ineffective for axial disease. In this case, NSAIDs are considered the first line disease-modifying agents, however they are often inadequate to control disease. With both axial and peripheral SpA (of the knee, for instance), consideration must be given to medications which will have activity for both. Biologic DMARD classes which are effective for axSpA include anti-TNFα inhibitors and IL-17 inhibitors. Research is ongoing regarding the efficacy of JAK inhibitors in AxSpA. Systemic glucocorticoids are considered ineffective for AxSpA and generally should not be used.

Return to Activities Physical activity and exercise are essential components in the management of AxSpA. Many of the current recommendations focus on spinal mobility, and no specific recommendations exist regarding return to activity with regard to knee joint involvement. Generally, most exercise modalities are safe in patients with ankylosing spondylitis. However, in patients with advanced disease, balance or mobility issues, osteoporosis, or have cardiac and pulmonary complications, certain forms of exercise may be contraindicated. These exercises include high impact exercise such as contact sports and certain forms of martial arts, high velocity or strongly resisted exercise, particularly if truncal flexion or rotation is involved, exercise that excessively challenges posture, balance or cardiorespiratory function, or exercises which excessively challenge mobility of a specific joint which is affected by ankylosis [26].

Sarcoidosis

Sarcoidosis is an inflammatory disease that is histologically characterized by non-caseating granuloma formation in tissues. Sarcoidosis most commonly affects the lungs with manifestations including hilar lymphadenopathy and parenchymal disease. Other commonly affected organs include the eye, often manifesting as uveitis, and skin, but essentially any organ can be affected. Musculoskeletal involvement occurs in about 25–30% of sarcoid-

osis and may manifest as inflammatory arthritis, inflammatory tenosynovitis or periarticular inflammation [27].

Clinical Presentation Inflammatory arthritis in sarcoidosis has either an acute or chronic presentation. Acute sarcoid arthritis usually affects large joints of the lower extremities, particularly the ankles, but the knee is also commonly involved. It is typically an oligoarthritis, but monoarthritis can occur [28, 29]. If inflammatory arthritis of the knee is present, the joint will be warm, tender to palpation, and with reduced range of motion. Swelling with joint effusion may be present. Swelling and pain with intact range of motion is more suggestive of periarticular inflammation [30]. Acute arthritis may be a part of Lofgren's syndrome which comprises bilateral hilar lymphadenopathy, arthralgia/arthritis, and erythema nodosum. The majority of cases of Lofgren's syndrome resolve spontaneously. Chronic sarcoid arthropathy has varied presentations but may present as symmetrical oligoarthritis. Sarcoidosis tends to declare its organ involvement within the first 3–5 years of the disease. After that, it is uncommon for new organ involvement to occur [29].

Diagnostic Studies Serum angiotensin converting enzyme (ACE) and lysozyme levels are often checked to evaluate for sarcoidosis. Either or both may be elevated in sarcoidosis, however neither is sensitive nor specific [29]. Additionally, both ACE and lysozyme do not correlate with disease activity. Hypercalcemia and hypercalciuria may be present, reflecting increased 1,25-dihydroxy-vitamin D levels. Rheumatoid factor can be positive in sarcoidosis as well. If a diagnosis of sarcoidosis is being considered, a chest radiograph should be obtained to evaluate for evidence of hilar lymphadenopathy or pulmonary parenchymal disease. Except in cases with classical presentation like Lofgren's syndrome for example, tissue biopsy is usually needed for diagnosis. A biopsy showing non-caseating granulomas in the appropriate clinical setting is diagnostic.

Treatment First line treatment for most manifestations of sarcoidosis is low to moderate dose glucocorticoids. Lofgren's syndrome can often be managed with NSAIDs. However, if the patient continues to be symptomatic or has a contraindication to NSAIDs,

low to moderate dose glucocorticoids may be used. In patients whose disease is either refractory to steroids, or in situations where steroids cannot be tapered due to persistent disease activity, steroid sparing agents like methotrexate and azathioprine may be considered. Anti-TNF-α inhibitors, particularly infliximab and adalimumab, may be considered in refractory sarcoidosis [29].

Return to Activities There are no limitations of range of motion or activity in musculoskeletal involvement in sarcoidosis. Weight bearing and activity should be encouraged as tolerated.

Sjögren's Syndrome

Sjögren's syndrome (SjS) is an autoimmune sicca syndrome characterized by inflammatory infiltration of exocrine glands. It may be primary or secondary in the setting of other autoimmune diseases like rheumatoid arthritis or SLE. SjS can have numerous extraglandular manifestations.

Clinical Presentation Musculoskeletal involvement can include arthralgias as well as non-erosive inflammatory arthritis, both of which can involve the knee. Joint symptoms are usually polyarticular [31]. When inflammatory arthritis of the knee is present, there is usually tenderness to palpation, warmth, swelling, and reduced range of motion. A joint effusion may be present. Generalized periarticular and myofascial pain may indicate underlying fibromyalgia which may occur with SjS.

Diagnostic Studies Laboratory features of SjS can include elevated inflammatory markers like ESR and CRP when the disease is active. Autoantibodies to Ro (SSA) and La (SSB) are usually positive in high titers, although they may be seen in other autoimmune disorders. Rheumatoid factor is commonly positive. A positive anti-CCP antibody is rare in primary SjS and its presence in a patient with known SjS should suggest underlying rheumatoid arthritis with secondary SjS.

Treatment Joint symptoms, particularly inflammatory arthritis, may be treated initially with hydroxychloroquine or methotrexate. Low-dose glucocorticoids may be used as well [31]. Monoarthritis of the knee is amenable to intra-articular glucocorticoid injection.

Return to Activity Weight bearing and activity is as tolerated.

Adult-Onset Still's Disease

Adult-onset Still's Disease (AOSD) is an autoinflammatory disorder that represents the adult-onset equivalent of systemic juvenile idiopathic arthritis.

Clinical Presentation AOSD is characterized by high daily fevers, evanescent pink rash, and arthralgias and arthritis. The rash classically appears with fever and abates as fever resolves. Between febrile episodes, patients appear and feel otherwise healthy. Other features may include hepatosplenomegaly, lymphadenopathy, and sore throat. Serositis, often manifesting as pericarditis sometimes with a pericardial effusion, is sometimes a feature. AOSD tends to follow one of three disease patterns [32]:

1. Monocyclic: Solitary episode occurs and then resolves completely with no further flares.
2. Polycyclic: Recurrent flares with periods of remission.
3. Chronic: Disease is persistently active.

Inflammatory arthritis is most associated with the chronic form of AOSD and typically affects the small joints of the hands but may also affect larger joints like the knee. Knee examination is consistent with findings of inflammatory arthritis including warmth, tenderness to palpation, range of motion reduction and potentially an effusion. If examined during a febrile episode, the patient may appear unwell, diaphoretic, and tachycardic. Erythematous to pink macules and patches may be present, particularly on the trunk and proximal extremities. The rash may resemble urticaria. A palpable liver or spleen may be present reflecting hepatosplenomegaly [32].

The most severe complication of AOSD is macrophage activation syndrome (MAS) which is a hyperinflammatory state characterized by persistent high fevers, extreme hyperferritinemia, cytopenias, and liver dysfunction with coagulopathy. Some similarities have been drawn to MAS and the hyperinflammatory state sometimes associated with severe SARS-CoV-2 infection (COVID-19). Unabating fevers in a patient with known AOSD should prompt consideration for developing MAS [33].

Diagnostic Studies Laboratory features of AOSD include a neutrophilic predominant leukocytosis, elevated serum ferritin, and elevated liver enzymes. Given that these laboratory features are not specific to AOSD, infection and malignancy need to be excluded.

Treatment Treatment includes oral glucocorticoids initially, usually at doses of 0.5–1 mg/kg/day, and tapered over weeks to months depending on clinical response [32]. Steroid sparing agents that have been demonstrated to be effective include methotrexate, anakinra, rilonacept, and canakinumab.

Return to Activities Patients will likely not feel well enough to exercise during febrile episodes, but may feel well enough to do so between episodes or when the disease is well-controlled or in remission. Weight bearing and activities are as tolerated.

Polymyalgia Rheumatica

Clinical Presentation Polymyalgia rheumatica (PMR) is an inflammatory disorder primarily characterized by pain and stiffness of the neck, shoulder girdle, and hip girdle. Patients may also report constitutional symptoms such as fever, malaise and fatigue. The symptoms of PMR are primarily due to periarticular inflammation, including bursitis. Peripheral inflammatory arthritis is a less common feature but when present, may affect the knees or wrists [34]. In this scenario the knee will be warm, tender to palpation and exhibit reduced range of motion. Knee pain without these features in a patient with PMR may indicate hip pathology.

PMR is associated with giant cell arteritis, and patients should be questioned about symptoms of recurrent fever, headache, jaw or tongue claudication, visual disturbances, and balance issues.

Diagnostic Studies Laboratory features of PMR typically include elevated ESR and CRP.

Treatment Symptoms of PMR respond rapidly to low-moderate dose glucocorticoids, the equivalent of prednisone 10–20 mg per day [34]. A lack of response to this therapy should prompt consideration of alternative diagnoses.

Return to activities Weight bearing and activities are as tolerated.

Other Causes of Knee Pain (Table 9.4)

Tenosynovial Giant Cell Tumor

Previously known as pigmented villonodular synovitis, tenosynovial giant cell tumor is a rare cause of knee pain. The vast majority of tumors are benign, although a small number of malignant tenosynovial giant cell tumors have been reported. The knee is the most commonly involved joint and usually presents as monoarticular arthritis. Within the knee itself, the tumor often affects the intercondylar region of the femur [35].

Clinical Presentation Symptoms include pain with reduced range of motion. Recurrent episodes of swelling may occur.

Diagnostic Studies If a joint effusion is present, synovial fluid analysis is classically bloody and non-inflammatory.

Radiographs of the knee may show periarticular cystic lesions. MRI is particularly helpful in this diagnosis and may show a soft tissue mass with hemosiderin deposits.

Table 9.4 Other causes of knee pain

Other autoinflammatory syndromes	Familial Mediterranean Fever, TNF-receptor-associated periodic syndrome (TRAPS), cryopyrin-associated periodic syndrome (CAPS)
Primary systemic vasculitis	• Large vessel vasculitis: Takayasu arteritis, giant cell arteritis • Medium vessel vasculitis: Polyarteritis nodosa, Kawasaki disease • Small vessel vasculitis: ANCA-associated vasculitis, cryoglobulinemic vasculitis, IgA vasculitis Mixed vessel size: Behçet's disease Inflammatory arthritis affecting the knee can be a manifestation of these disorders but is less prominent than many other distinguishing disease features
Mixed connective tissue disease	A specific overlap syndrome with varying features of lupus, systemic sclerosis and myositis associated with high titer anti-U1-RNP antibodies. Nearly all patients have Raynaud's phenomenon. Inflammatory arthritis is a common feature and can be erosive. Most commonly involved joints are MCPs, PIPs, MTPs but the knee joint may be involved as well.

Treatment Treatment may include total synovectomy with adjuvant radiation therapy [36]. Unfortunately, tumor recurrence may occur, particularly if there is residual synovium present after surgery.

Synovial Chondromatosis

Synovial chondromatosis is characterized by the formation of multiple nodules of hyaline cartilage in subsynovial connective tissue. Nodules may become calcified. The knee is affected in over 50% of cases [37]. Synovial chondromatosis is almost always benign, but may rarely develop into synovial chondrosarcoma [35].

Clinical Presentation Patients may present with knee pain, swelling, and with a locking or clicking sensation. Physical examination may reveal swelling, reduced range of motion, and crepitus.

Imaging Plain radiographs of the knee usually show multiple intra-articular, oval-shaped, calcified loose bodies.

Treatment Treatment of synovial chondromatosis involves synovial excision and removal of loose bodies [38].

Infectious Knee Pain

This section will cover common causes of infectious knee pain. Organisms that can infect a knee joint include bacteria, viruses, fungi, mycobacteria, with bacteria being the most common. The term septic arthritis encompasses joint infections of any of the above organisms. Early diagnosis is important as infection can lead to degradation of joint cartilage as early as 48 hours [39, 40].

Bacterial Arthritis

Specific Organisms and Population (Adults) [41]

- *Staphylococcus aureus* (*S. aureus*): general population, pre-existing joint disease, prosthesis, open skin or other infection. Most common overall.
- Coagulase-negative staphylococci: prosthetic joint infection.
- Streptococcal species (i.e.: *Streptococcus pneumoniae*): general population, splenic dysfunction.
- *Neisseria gonorrhoeae*: most common in sexually active general population.
- Aerobic gram-negative (i.e.: *Pseudomonas* species): immunocompromised, gastrointestinal infection.

- Anaerobic gram-negative: immunocompromised, gastrointestinal infection, human bite, decubitus ulcers, intra-abdominal abscess.
- Brucellosis: general population with animal exposure.
- *Borellia burgdorferi*: general population with exposure to ticks.
- *Mycoplasma*: immunocompromised.

Specific organisms (children): group B streptococci, group A streptococci (*streptococcus pyogenes, Kingella kingae*) gram-negative enteric bacilli, *S. aureus.*

Risk Factors Bacterial arthritis of the knee can be as a result of other infections that cause bacteremia and hematogenous seeding or direct inoculation of the joint. Risk factors that predispose a patient include pre-existing joint disease (including gout, CPPD or autoimmune diseases like rheumatoid arthritis), skin or soft tissue infection, recent joint surgery or injection, trauma, animal bite, advanced age, intravenous (IV) drug use, indwelling ports or catheter, and immunosuppression.

Clinical Presentation A patient with bacterial arthritis will typically present with acute (around one to five days) joint pain, swelling, redness, warmth, and stiffness, muscle spasm, with or without accompanied fever. Larger joints such as the knee are more often affected. Majority of the cases are monoarticular. Bacterial monoarthritis is more common, however, oligo- or polyarticular septic arthritis is more likely in patients with rheumatic or other systemic connective tissue disease [42].

Physical Exam Examination of the affected knee joint will demonstrate erythema, edema, warmth, diffuse tenderness to palpation. An effusion is palpable. Range of motion, both passive and active, is typically restricted and painful, which leads to dysfunctional ambulation and difficulty weight bearing.

Outside of the focused knee exam, evaluate all other joints to determine if polyarthritis is present. Evaluate for a source of infection such as open wounds or abscesses, indwelling ports or catheters. Examine cardiopulmonary systems. Vitals may show elevated temperature. Hypotension and tachycardia may be present in the setting of bacteremia and hematogenous seeding.

Diagnostic Studies Synovial fluid should be aspirated from the knee joint prior to administration of antibiotics. Analysis involves volume (typically >3.5 mL), gross appearance (cloudy, opaque, yellow, purulent), viscosity (variable), crystals (none), cell count and differential (WBC >50,000/μL, polymorphonuclear leukocytes >75%), gram stain (large number of neutrophils and organisms), and bacterial culture, including Neisseria gonorrhoeae culture (positive if bacterial). Certain bacterial organisms such as Neisseria may have lower WBC counts at >2000/μL. If the leukocyte count is >50,000/μL and mostly neutrophils, but a negative gram stain and culture, it is still presumed to be bacterial septic arthritis. Synovial fluid should be checked for crystals as crystal-induced disease can mimic bacterial septic arthritis clinically and can also occur concomitantly [43].

A CBC will likely show leukocytosis with left shift, however, this is non-specific. ESR and CRP may also be elevated, but is another non-specific finding. Blood and wound cultures should be obtained to determine the source of infection in hematogenous spread and for identification of pathogen [41].

Imaging Imaging is not required to diagnose bacterial septic arthritis but can be used in conjunction with synovial fluid analysis. Knee X-rays may show soft tissue swelling, joint space widening or narrowing, joint effusion, erosions, osteomyelitis, subcutaneous emphysema, foreign body, or trauma. Ultrasonography may show joint effusion, cortical irregularities, or separate fluid collection.

An echocardiogram may be necessary to evaluate for endocarditis especially in an IV drug user.

Treatment In order to appropriately diagnose bacterial septic arthritis, synovial fluid must be collected prior to administration of antibiotics. At the time of synovial fluid collection, the knee joint should be fully drained with large-bore needle aspiration with or without ultrasonography. Indications for arthroscopic or open surgical drainage and irrigation include persistent effusions despite serial aspirations, inadequate drainage via needle, loculated effusions, penetrating trauma, or retained foreign body. If the infection is severe, repeated aspiration or drainage may be required. If the aspiration and drainage is adequate, often the patient improves clinically and lab values return to normal [41].

Antibiotics When there is a high suspicion of bacterial septic arthritis, empiric antibiotics should be broad spectrum and cover the most likely pathogen based on age and risk factors.

If the gram stain shows gram-positive organism:

- Gram-positive cocci → empiric vancomycin.
- Methicillin-susceptible S. aureus (MSSA) → beta-lactam agent.
  - Examples: Cefazolin, nafcillin, oxacillin
  - If allergies to penicillins → vancomycin
- Methicillin-resistant S. aureus (MRSA) → vancomycin.
  - If allergy or intolerance → Daptomycin or linezolid or clindamycin

If the gram stain shows gram-negative organism:

- Gram-negative bacilli → cephalosporin.
- Ceftriaxone, cefotaxime, ceftazidime, cefepime
- Pseudomonas aeruginosa: initial empiric therapy with two anti-pseudomonal agents.
  - Cephalosporin + ciprofloxacin or aminoglycoside
  - If allergy to cephalosporin: Aztreonam 2 g IV every 8 h
- Negative initial Gram stain → vancomycin.

- If trauma, add a third-generation cephalosporin
- If immunocompromised and/or IV drug user, add anti-pseudomonal cephalosporin

In all instances, await susceptibility data and narrow antibiotic therapy for definitive treatment and to avoid antibiotic resistant bacteria.

The duration of antibiotic therapy should be tailored to each case. In general, uncomplicated cases typically require a shorter course of IV therapy (~7 days) with additional 14–21 day oral therapy. For bacterial septic arthritis with bacteremia, duration of IV therapy is at least 14 days and additional oral therapy for at least 14–21 days [41].

Return to Activities There is no indication for immobilization of the joint. Passive range of motion should be done to preserve mobility. Weight bearing can be avoided until signs of inflammation have decreased. In addition, comorbidities and the source of bacteria in bacterial septic arthritis contribute significantly to the outcome. Uncomplicated cases are likely to be discharged home with progressive therapy and return to regular function as tolerated. Those with pre-existing conditions or complicating factors may require discharge to a facility for rehabilitation and longer term stay. Return to baseline functional status in this case is slower and likely diminished. Complete antibiotic therapy and clearance of infection, stabilization of comorbidities, along with appropriate physical therapy is vital to recovery.

Gonococcal Arthritis

Neisseria gonorrhoeae can lead to disseminated gonococcal infection which can cause arthalgias or purulent arthritis, most commonly in sexually active young adults. There may be asymptomatic gonococcal mucosal colonization of genitourinary or pharyngeal systems that lead to bacteremia and disseminated gonococcal infection [40, 41, 44].

Risk Factors A sexually active population is most at risk. Women during menstruation and pregnancy and those who are immunocompromised (complement deficiency) can also be at increased risk.

Clinical Presentation Typical symptoms include migratory polyarthralgias, tenosynovitis, dermatitis, fever (all in combination is called arthritis-dermatitis syndrome), genitourinary symptoms, and purulent arthritis in one or more joints. Purulent arthritis typically involves knees, wrists, ankles.

Diagnostic Studies Diagnostic testing includes blood cultures, mucosal site specimen cultures, and synovial fluid analysis. The average synovial fluid leukocyte count for gonococcal arthralgia is lower at ≤20,000/μL. Because the arthralgia is as a result of immune response and immune-complex deposition, gram stain, synovial fluid cultures, and even blood cultures may return negative. If the joint has developed gonococcal purulent arthritis, the synovial fluid would show leukocytes >50,000/μL and a positive gram stain [43].

Treatment Initial treatment is with ceftriaxone (alternatively, cefotaxime or ceftizoxime) along with presumptive treatment for chlamydia with doxycycline or azithromycin. Depending on susceptibility data, treatment may be changed to fluoroquinolones or penicillin if purulent arthritis is not present. Purulent arthritis should be treated with ceftriaxone for at least 7–14 days along with aspiration or drainage. Those with penicillin allergies typically can use cephalosporins safely. Those with beta-lactam allergies should undergo desensitization [44].

Imaging X-rays are typically benign in polyarthralgias. In purulent arthritis, knee X-rays may show soft tissue swelling, joint space widening or narrowing, joint effusion, erosions, osteomyelitis, subcutaneous emphysema, foreign body, or trauma. Ultrasonography may show joint effusion, and cortical irregularities.

Return to Activities With appropriate antibiotic management, patients typically make a full recovery without residual deficit. There is no indication for immobilization of the joint and range of motion should be preserved passively and actively. With polyarthralgias, weight bearing is as tolerated. With purulent arthritis, weight bearing can be avoided until signs of inflammation are decreased. Return to activity is progressive and as tolerated.

Lyme Arthritis

Infection with the spirochete *Borrelia* leads to Lyme disease. *Borrelia burgdorferi* (*B. burgdorferi*) and *Borrelia mayonii* are the most common species in the United States. The bacteria is transmitted to humans through the *Ixodes* tick bite. Early localized disease has the characteristic erythema migrans (EM) skin lesion, with a central clearing and bulls-eye appearance. Early disseminated disease has multiple EM skin lesions occurring days to week after infection along with intermittent arthralgias and myalgias, and neurologic or cardiac findings that present weeks to months after infection. Late Lyme disease has associated mono- or oligoarthritis of large joints. The knee is a commonly affected joint. The arthritis can be migratory, intermittent, or persistent. Onset is variable and can present months or more after initial infection [40].

Clinical Presentation While technically Lyme arthritis is an infectious arthritis, the presentation is more akin to inflammatory arthritis since it creates an inflammatory synovial response. Fever is less common. The knee joint will have pain and swelling. Oftentimes, symptoms will wax and wane. If untreated, about 10% of patients will develop chronic inflammatory synovitis leading to erosions and destruction of the joint.

Despite months of treatment, a small portion of patients will develop post-infectious persistent Lyme arthritis. This arthritis is characterized by smaller knee joint effusions. A significant increase in synovial proliferation is likely associated with the recurrent effusions. The persistent arthritis can last several years [41].

Physical Exam Knee joint examination is overall less painful with range of motion and weight bearing. There may be pain along the joint lines along with a palpable effusion.

Diagnostic Studies Serological testing positive for serum immune globulin G (IgG) to *B. burgdorferi* is used to confirm the diagnosis of Lyme disease. Synovial fluid analysis of the joint fluid will portray an inflammatory arthritis. Fluid will be translucent to opaque, yellow in color, low viscosity, average WBC count 10,000–25,000/μL, polymorphonuclear leukocytes >50%. Synovial fluid culture is typically not positive. Imaging is not required for diagnosis [43].

Treatment Initial treatment starts with an oral antibiotic such as doxycycline or amoxicillin for 28 days. If prior treatment is ineffective or if there is persistent arthritis, IV ceftriaxone for 14–28 days or another 28 day course of oral antibiotics can be used. In post-infectious Lyme arthritis where both oral and IV antibiotic therapy is ineffective, consider management by a rheumatologist for DMARDs, or arthroscopic synovectomy. Overall, without treatment, post-infectious Lyme arthritis typically resolves spontaneously, though it may take a significant amount of time and the risk of damage to bone and cartilage increases.

Return to Activities Restrictions in persons with Lyme arthritis include decreasing high levels of activity and high impact activities while the knee joint inflammation is active. Rest followed by range of motion directed physical therapy can be helpful for quicker recovery.

Prosthetic Joint Septic Arthritis

There is a higher incidence of prosthetic knee joint infections over other joints such as the hip and shoulder. There is increased risk in those who have comorbidities such as diabetes, malignancy, rheumatic diseases, kidney disease, immunosuppression through

infection or medication, prior injection at the same joint, wound infections, and bacteremia. Organisms include *S. aureus*, coagulase-negative staphylococci, beta-hemolytic streptococci, gram-negative bacilli, enterococci, or anaerobes. Fungal and mycobacterial infections are also possible. The presence of hardware can lead to formation of biofilm and increases the risk of infection [45].

Clinical Presentation If infection is present less than 3 months after surgery, it is considered early onset, 3–12 months is considered delayed, and greater than 12 months after surgery is considered late onset. Early onset infection can present as pain, erythema, effusion, induration of incision, wound drainage or dehiscence, and fever and is usually as a result of implantation. It is also possible as a result of superficial wound spread. Delayed onset infection symptoms can be more subtle and often are attributed to failure or loosening of the prosthesis. Delayed onset of symptoms may be as a result of infection during implantation with less virulent organisms, including coagulase-negative staphylococci. There can be persistent joint pain in weight bearing and range of motion which can be attributed to hardware loosening instead of infection. In addition, fever and mild effusion may be present along with a sinus tract with drainage. Late onset is usually as a result of hematogenous seeding. Symptoms will be similar to early onset infection [46].

Diagnostic Testing Diagnosis via culture can be missed due to quality of swab or tissue culture, slow growing bacteria, antibiotic use prior to culture, or difficult to culture organisms due to lower virulence or media type. Positive culture with prominent organisms such as *S. aureus*, or a sinus tract that connects with the prosthesis is assumed to be true prosthetic joint infection. Two or more cultures positive for a less common or lower virulent organism can also be assumed to be true infection over contamination. Laboratory data is non-diagnostic with leukocytosis and elevated inflammatory markers such as ESR and CRP. X-ray of the joint can show soft tissue swelling, fracture, or lucency of bone sugges-

tive of hardware loosening but may be non-diagnostic in early onset infection. Other imaging modalities such as CT, MRI, or bone scans are not typically used for diagnosis. Arthrocentesis and synovial fluid analysis can be categorized as septic during early onset infection. However, in delayed and late onset infection, fluid leukocyte count can be much lower and have other fluid characteristics that fit both inflammatory and septic synovial fluid. Synovial fluid should be cultured. It is important for appropriate aseptic technique during aspiration to avoid contamination. Sinus tracts should be aspirated and drained and sent for culture. During removal of an infected prosthesis, multiple samples from different areas should be obtained for culture.

Treatment Antimicrobial treatment should be started after cultures are obtained and should be tailored to culture results and susceptibilities. Empirically, vancomycin in combination with a third- or fourth-generation cephalosporin can be used. Surgical options include debridement and removal of prosthesis with or without reimplantation, or amputation. The prosthesis can be retained, however, it is more common to explant the prosthesis, treat with antimicrobial therapy, and reimplant the prosthesis. Reimplantation should be delayed as recurrence of infection is common within several weeks to months. Without removal of the prosthesis, chronic antibiotic suppression may be required [47].

Return to Activity Patients are often non-weight bearing during acute stages of infection and also after explant of a prosthesis and/or amputation. Physical and occupational therapy is important to prevent muscle wasting and to preserve other joint mobility. As a result of the infection, patients may have some chronic pain in addition to limited use of the joint. Successful return to near-baseline activity is seen more with patients who explant and reimplant the prosthesis. Rehabilitation after reimplantation allows for immediate weight bearing as tolerated and progressive increase in activity over the course of weeks. The overall functional outcome may be diminished compared to those who did not have a prosthetic joint infection [48].

Viral Arthritis

Enterovirus (i.e.: coxsackie virus and echovirus) Overall, arthritis is rare in enterovirus infections. However, for non-specific viral arthritis, enterovirus is the most common. Enterovirus arthritis can affect both small and large joints, including the knee [49].

Clinical Presentation Joint symptoms such as generalized knee pain and stiffness are typically self-limited. Acute viral symptoms will likely be present, such as fatigue, fever, sore throat, rhinorrhea, cough, rash, and pleuritic chest pain.

Diagnostic Studies Non-specific laboratory findings may be present such as elevated ESR and leukocytosis. Aspiration of joint fluid may show 2000- > 10,000 WBC/'μL.

Treatment Supportive care.

Hepatitis A

Arthritis is rare. Knee arthralgias may occur. The infection is self-limited and treatment for the joint pain is supportive care [50].

Hepatitis B (HBV)

Knee or other joint pain can be the first manifestation of acute hepatitis B infection and occurs a few weeks before jaundice. The joint pain can be symmetric or migratory and typically signal the prodrome phase of infection. It is theorized that deposition of immune complexes into joints is what causes the arthritis.

Clinical Presentation Joint stiffness is present, particularly in the morning. Typically, fever, skin urticaria or a maculopapular rash will manifest along with the joint pain. Joint symptoms can last for days up to weeks. Joint symptoms can resolve at the time

jaundice develops. If arthralgias continue, they regress as jaundice regresses. Acute HBV joint arthritis is less likely to become chronic arthritis or cause permanent damage to the joint. However, if a person has a chronic HBV infection, joint arthritis can be prolonged and be more inflammatory in nature as seen in joint fluid analysis.

Treatment Supportive therapies for joint manifestation. Treat the HBV with supportive therapies or in the case of chronic HBV with antivirals such as interferon and nucleos(t)ide analogs [51].

Hepatitis C (HCV)

Acute HCV infection is more associated with arthralgias and myalgias. Chronic HCV infection can be associated with chronic arthralgias and arthritis.

Clinical presentation Chronic HCV arthritis is non-erosive and does not cause deformities. Presentation can be monoarticular, like the knee, or oligoarticular. Common complaints include joint pain and stiffness.

To distinguish hepatitis from other viral illnesses, laboratory values will show abnormal liver function tests, decrease in complement levels, increased circulating immune complexes, and positive specific hepatitis surface antigen (i.e.: HBsAg) [52].

Treatment Supportive care for the joint manifestations. Treat the HCV with appropriate antiviral medications [53].

Parvovirus B19

This virus can affect both adults and children. In children, it is also known as fifth disease or erythema infectiousum. Arthritis more commonly happens in adults and women over men.

Clinical Presentation Joint symptoms can occur concurrently with skin rash (erythematous malar rash, "slapped cheek") or soon after eruption. Fever is generally present. In adults, arthritis can occur without fever or rash. The affected joints in adults are typically in the hand, while in children the knee is more commonly affected. The arthritis can be monoarticular, or asymmetric oligoarticular. Overall, joint pain and stiffness is more likely to occur over joint swelling. Symptoms can last for a prolonged period of time from weeks to months. The infection is usually self-limited, however, there is a potential for recurrence or chronic arthritis [54].

Diagnostic Studies Parvovirus B19 can present like a systemic autoimmune rheumatic-like disease with positive laboratory values such as antinuclear antibodies, rheumatoid factor, elevated ESR and CRP. The arthritis is non-erosive and despite the rheumatic-like syndrome, it has not been found to progress to rheumatoid arthritis. To distinguish this from other viruses, a serologic test for parvovirus b19-specific IgM antibody can be performed. Parvovirus DNA can be found in inflamed joints and in other tissues.

Treatment Supportive care for joint manifestations.

Alphaviruses

These viruses are mosquito-borne RNA viruses that appear in countries across the globe. These viruses appear in cycles and re-emerge from time to time as a result of continuous transmission from mosquito to human and other mammals or birds. Transmission is highest during the rainy season. Notable alphaviruses include Ross River virus, Chikungunya virus, Mayaro virus, and Sindbis virus. The incubation period can be for days to weeks. Out of the above, Chikungunya virus is more persistently common with outbreaks in multiple countries. In the USA, it is usually found in travelers [55, 56].

Clinical Presentation In general, infection with an alphavirus will cause fever, arthritis, and rash. The arthritis is typically polyarticular with occasional periarticular and tendinous involvement. Symptoms can last anywhere from weeks to months. The joint symptoms will typically occur prior to a skin rash and may even manifest prior to fever. The rash itself may be short lived (7–10 days) compared to the joint symptoms.

For Chikungunya virus specifically, the arthritis can cause significant, incapacitating pain as the virus invades joint tissues such as synovium and surrounding muscles and replicates causing a release of inflammatory cytokines. Joint symptoms may present in one joint first but will typically have bilateral, symmetric, polyarticular involvement. Symptoms include morning stiffness and edema with decreases in strength due to pain. The virus may be cleared from the joint in a few weeks, but viral RNA has been found to persist in tissues which can lead to chronic arthritis as a result of persistent viral replication, inflammatory or autoimmune response to viral RNA. Older age, prior existing osteoarthritis, and severe acute Chikungunya virus can predispose someone to chronic inflammatory polyarthritis [57].

Treatment Treatment of alphavirus related arthritis should start off with medications such as tylenol and NSAIDs, and other conservative measures such as physical therapy. There are no specific antiviral therapies available. For Chikungunya virus treatment, if conservative medications are ineffective, oral glucocorticoids are the next step. If steroids continue to be ineffective, DMARDs such as methotrexate or sulfasalazine can be considered.

Epstein–Barr Virus (EBV)

Also known as infectious mononucleosis. EBV is transmitted through saliva and stays latent in B-cells. The majority of the population will remain asymptomatic despite infection [49].

Clinical Presentation Of those that become symptomatic, most will have simple arthralgia. However, for those that develop arthritis, there will be large joint swelling along with pain and stiffness. Symptoms are self-limited. Arthrocentesis findings will show inflammatory findings in the synovial fluid.

Treatment Supportive care for joint manifestations.

Varicella

The varicella virus causes chickenpox. Varicella is a rare cause of arthritis, but when present, happens most often in the knee. Despite presentation like a septic bacterial joint, the pain from varicella arthritis is more likely related to nerve pain than pain from the joint itself [49].

Clinical Presentation Symptoms include swelling, pain, stiffness, and decreased range of motion. Varicella typically involves only one joint and will present a few days after the onset of rash. Symptoms are self-limited.

Diagnostic Studies Arthrocentesis should be performed to rule out concomitant bacterial septic arthritis. Synovial fluid analysis will often be non-diagnostic with up to 6000 WBCs per μL and all mononuclear cells.

Treatment Supportive care for joint manifestations.

Human Immunodeficiency Virus (HIV)

HIV infection has a few musculoskeletal syndromes that are related specifically to HIV, but also has syndromes associated with rheumatic diseases. The rheumatic diseases are typically separate from HIV infection but their expressions are affected by

the HIV infection. Some examples include reactive arthritis, septic arthritis, and psoriatic arthritis. The expression of rheumatic diseases may be related to immunodeficiency, immune hyperactivity, dysregulated production or activity of cytokines, or molecular mimicry [49].

HIV-Associated Syndromes

Painful articular syndrome: This presents asymmetrically, normally in lower extremity joints. The pain is typically out of proportion to clinical findings which are usually benign. This syndrome is self-limited and can last for only a day without treatment. This does not progress to an active inflammatory joint process.

HIV-associated arthritis: This typically lower extremity symmetric oligoarticular arthritis is non-erosive and non-destructive. Symptoms can last for weeks and are self-limited without treatment. Arthrocentesis is usually non-diagnostic and diagnostic imaging is normal.

Diffuse infiltrative lymphocytosis syndrome: Causes salivary and lacrimal gland enlargement and can mimic Sjögren's syndrome. This syndrome causes arthralgias but not arthritis [49].

Rheumatic Diseases with HIV Infection

Reactive arthritis: The typical presentation of conjunctivitis, urethritis, and arthritis is less likely to occur. HLA-B27 antigen may be present, but axial involvement is rare. There will likely be enthesopathies and mucocutaneous manifestations. The reactive arthritis will be chronic and relapsing.

Septic arthritis: Staphylococcus aureus is the most common organism, along with others such as *Streptococcus*, *H. influenzae*, and *Salmonella* species. The risk of septic arthritis is higher with CD4 < 200 cells/μL and substance abuse. HIV-associated septic arthritis typically manifests as monoarticular.

Psoriatic arthritis: Severity of the disease can be worse with concomitant HIV infection. Feet and ankle joints are the most severely affected, but there can be knee involvement. It may present with bony deformities and osteolysis, without obvious skin lesions.

Other diseases: Rheumatic and inflammatory diseases will usually not manifest in HIV infection if the CD4 count is low. However, once HIV treatment has begun and immune system function has improved, these syndromes can manifest [49].

Return to Activity Overall, physical and occupational therapy is an important part of a long-term treatment plan for chronic HIV and its comorbid conditions. There is no joint restriction for HIV-associated or related arthritis and maintaining general mobility and functional activity is ideal. Activities and weight bearing are as tolerated and should be progressive [58].

SARS-CoV-2 Infection (COVID-19)

COVID-19 musculoskeletal manifestations include diffuse malaise, arthralgias, and myalgias, which can mimic rheumatic diseases. Manifestation may be oligo- or polyarticular. It has not yet been shown to target specific joints or have specific findings in joint analysis or imaging [40, 59].

Other viruses that can cause knee arthralgias or arthritis not included in this chapter: Rubella, rubella vaccine virus, flaviviruses like dengue and zika, mumps, adenovirus, herpes simplex, and cytomegalovirus.

Return to Activity Since in general, the majority of viral disease is self-limited or treated completely with antivirals, there is no restriction of activity or joint mobilization with viral arthritis. Activity and weight bearing is as tolerated and progressive.

Fungal

Fungal etiologies are rare causes of infectious arthritis and osteomyelitis but need to be kept in the differential diagnosis, particularly in endemic areas. Patients who are immunocompromised either from chronic infection, like HIV, or iatrogenically, are at risk for disseminated fungal infections. One important group are those taking TNF-α inhibitors. Common fungal organisms include blastomycosis, coccidioidomycosis, cryptococcosis, and candida. Other etiologies can include histoplasmosis, and sporotrichosis. Fungal arthritis is usually as a result of hematogenous spread or direct extension of a bony lesion from disseminated disease. Arthrocentesis and synovial fluid analysis can show leukocytes ≤40,000/μL and about 70% neutrophils. Diagnosis of these disorders often requires tissue biopsy with culture or direct visualization of yeast forms on histology. In the context of the knee joint, biopsy of synovium may be needed. Treatment involves aspiration of joint fluid along with lavage. Identification of the pathogen and systemic antifungals are required. In some instances, amphotericin B has been used intraarticularly [60].

Return to Activity Appropriate and complete antifungal therapy generally leads to full recovery without residual joint deficits. Recovery may be prolonged if surgery is required. In general, activity and weight bearing is progressive and as tolerated.

Blastomycosis

Blastomycosis is endemic to the North-Central and Southern United States. It is caused by *Blastomyces dermatitidis*. Musculoskeletal manifestations are most commonly bone pain, swelling and soft tissue abscesses. Infectious arthritis is a less common manifestation, but when it occurs it most commonly affects large joints of the lower extremity including the knee [61].

Radiographs of the knee may show "punched-out" bone lesions.

Coccidioidomycosis

Classically endemic to the southwestern United States (including Arizona, New Mexico and Southern California) and Central America. *Coccidioides immitis* is the most common. The primary site of infection is usually the lungs through inhalation of spores, manifesting as pneumonia. Infection of bones or joints is the result of hematogenous spread and happens weeks to months after exposure. Joint manifestation is typically monoarticular. The knee joint is the most commonly affected joint. Symptoms include pain and stiffness with or without effusion. If infection progresses, imaging may show soft tissue and joint effusion along with bony involvement due to osteomyelitis. Arthrocentesis analysis may have infectious cell count qualities and positive culture. Despite treatment, there is a possibility of recurrence of infection at sites of prior infection.

Valley Fever: This syndrome include infectious pneumonia, arthralgias, and erythema nodosum.

First line treatment for coccidioidomycosis septic arthritis is fluconazole or itraconazole. Amphotericin B may be another option. Depending on severity of joint or bony infection, surgical debridement may be required [62].

Cryptococcosis

Cryptococcosis is generally as a result of inhalation but can also be from direct inoculation from trauma or through the gastrointestinal tract. Classically, it can be found in pigeon droppings. Cryptococcal arthritis is rare and is most frequently a chronic monoarticular arthritis affecting the knee as a result of extension of osteomyelitis. Treatment includes amphotericin B, flucytosine, and fluconazole. In severe or persistent cases, surgery for bony debridement or resection may be necessary [63].

Candida

Musculoskeletal manifestations of candida infections are often due to hematogenous spread to bones or joints. However, exogenous exposure due to trauma, surgery, or intravenous drug use is also possible. Those at risk are typically immunocompromised or suppressed, IV drug users, have chronic indwelling catheters or have used broad spectrum antibiotics. The most recognizable organism is Candida albicans. The knee is a common joint affected by Candida, in both native and prosthetic joints [64].

Clinical Presentation One or more joints may be affected. If a joint is infected, there should be suspicion for surrounding osteomyelitis. Presentation can occur weeks or months after inoculation. Symptoms may be less prominent than bacterial septic arthritis of the knee. There can be pain, tenderness, erythema, effusion, stiffness, along with fever. However, tracts from joint to skin can also occur in severe infections.

Diagnostic Studies Laboratory studies may yield mild leukocytosis and elevated ESR and CRP. X-ray imaging upon initial presentation may be benign but can show erosions and destruction if infection persists. MRI bony signal changes can signify osteomyelitis. Arthrocentesis may show purulent fluid with infectious cell qualities (WBC >20,000 cells/μL, >75% polymorphonuclear leukocytes). Cultures will be positive on standard media.

Treatment Native joint fungal septic arthritis can be treated with oral fluconazole for at least 6 weeks or IV echinocandins for 2 weeks followed by oral fluconazole for 4 weeks. Amphotericin B can also be used. Prosthetic joints will usually need to be surgically removed prior to treating with antifungals as above for 12 weeks. A new prosthesis may be implanted 3–6 months after with antifungals should be given for at least 6 weeks after.

Treatment of osteomyelitis requires a longer course of antifungals for 6–12 months. Susceptibilities of candida species should be performed as fluconazole-resistant species should be treated with echinocandins and voriconazole, or amphotericin B [61, 64].

Mycobacteria

Mycobacterium tuberculosis (TB) can manifest in extrapulmonary structures, more often in the spine (Pott's disease) than in the extremities. Tuberculosis arthritis accounts for about 10% of extrapulmonary TB cases. Those at risk for TB joint infection include immunocompromised state, malnutrition, or advanced kidney disease. Joint manifestation of TB is typically reactivation of disease and can present months to years after initial infection. Current active pulmonary tuberculosis is not usually present. Pathogenesis is related to hematogenous spread. Joint involvement is typically monoarticular in large weight-bearing joints. Of note, HIV co-infection is common. If there is a femur or tibial metaphyseal lesion, it can move through the growth plate and affect the adjacent joint [65, 66].

Clinical Presentation TB arthritis include progressive joint pain and stiffness and will often lack constitutional symptoms classic with other septic arthritis, such as fever and chills. Swelling may or may not be present, and overall is usually absent. Symptoms can progress over a time period of months to years. In osteomyelitis, bone pain is common, and can be associated with soft tissue erythema, edema, sinus tract, or abscess formation.

Poncet disease: This is an acute reactive symmetric polyarthritis affecting both small and large joints. There will be inflammation of the joint but mycobacteria is not found in the joint. Joint symptoms are treated once anti-TB therapy is started and will not have residual joint destruction.

Diagnostic Studies Arthrocentesis and synovial fluid analysis can be done. Synovial fluid will have an average leukocyte count

of 20,000/μL and about 50% neutrophils. Acid-fast stain is positive in only about one-third of cases. Synovial fluid culture is positive about 80% of the time. Synovial tissue biopsy should be performed and is more likely to be positive over fluid culture. Histopathology can show the more classic caseating, or non-caseating granulomas. TB cultures can be delayed due to the slow growth of the organism.

Imaging Imaging studies may show osteolytic lesions in multiple sites, including peripheral erosions at sites of synovial attachment, and joint space narrowing if late in the disease. There can also be soft tissue swelling or calcification, or periarticular osteopenia. MRI can show if the musculoskeletal TB is spreading to adjacent structures.

Treatment Anti-tuberculosis therapy includes rifampin, isoniazid, pyrazinamide, and ethambutol and treatment length is typically longer than for pulmonary TB due to poor penetration of medication. Treatment courses are also longer if the patient is immunosuppressed. Surgical debridement may be required for abscesses or sinus tracts. Surgical stabilization of bony lesions, synovectomy, and joint replacement may be performed depending on severity of disease.

Return to Activities If treated in the early stages, patients typically make a full recovery without residual deficits. Activities and weight bearing are progressive and as tolerated. If joint destruction has occurred, patients will likely require surgery in order to return to baseline level of function.

Atypical Mycobacteria Atypical mycobacteria such as *M. marinum, M. avium-intracellulare, M. terrae, M. kansasii, M. fortuitum, M. chelonae* are found in soil and water and through trauma and direct inoculation can cause chronic arthritis. Diagnosis can be made as above with biopsies and cultures. Treatment depends on susceptibilities to antimicrobials.

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Knee Pain Case Studies

10

Daniel Weng, Caroline Lewis,
and Miguel Escalon

Acute Burning Knee Pain

History

A 34-year-old male with history notable for severe factor VIII deficiency (on factor VIII prophylaxis, 3 times a week) presented with new bilateral knee pain, right greater than left, during a follow-up visit. The pain is deep within the knee joint, stiff and burning in quality, and keeps the patient awake at night. It is unalleviated by position changes. He has tried NSAIDs 1–2 times a day for the past few weeks with minimal relief. The patient has had chronic right ankle pain in the past attributed to hemophilic arthropathy, worsened by walking and training. He has taken NSAIDs and oral steroids in the past with minimal to moderate pain relief, and underwent ultrasound guided corticosteroid injec-

D. Weng (✉)
Mount Sinai Rehabilitation and Human Performance,
New York, NY, USA

Johns Hopkins School of Medicine, Baltimore, MD, USA

C. Lewis · M. Escalon
Department of Rehabilitation and Human Performance, Icahn School of Medicine at Mount Sinai, New York, NY, USA
e-mail: miguel.escalon@mountsinai.org

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tion with moderate improvement. He also has had occasional hemarthroses in various joints (including the knee) that eventually resolved with factor VIII injection.

Review of systems:
No fever, chills, cough, paresthesia, weakness, bowel/bladder changes or falls

Past medical Hx/surgeries:
- Severe FVIII deficiency
- Right ankle corticosteroid injection

Medications:
- Factor VIII solution
- Diclofenac gel
- Celecoxib
- Lidocaine patches
- Meloxicam
- Emicizumab (monoclonal antibody for factor VIII)

Family history:
None

Social history:
The patient is a professional table tennis player and trains 3–4 times per week

Physical exam findings:
BMI of 23 kg/m^2
General: Not in acute distress, well developed
Skin: No rash, swelling, ecchymoses, erythema, or warmth

Knee:
Inspection: No bony abnormalities, no muscular atrophy
Palpation:
- No effusion or hemarthrosis
- No pain with manipulation or palpation to joint lines, bursa, tendons, bony prominences

Range of motion:
- Bilateral knees unable to fully extend with hard stop, non-painful
- Positive crepitus bilaterally

Strength: 5/5 in bilateral lower ext.
Sensation: Intact to light touch in lower ext.

Gait:
- Antalgic with right toe walk
- Left lower limb discrepancy

Provocative maneuvers:
Negative Lachman/anterior or posterior drawer
Negative Thessaly
Negative McMurray
Negative varus/valgus stress or laxity

Differential Dx (Table 10.1)

Imaging

Bilateral Knee X-Ray

See Fig. 10.1.

Diagnostic Discussion

In this patient with acute, bilateral knee pain in the setting of chronic intra-articular bleeds due to severe hemophilia, and severe degenerative arthropathy seen on imaging at a young age (Fig. 10.1), the most likely diagnosis is hemophilic arthropathy. While the patient may have an episode of acute hemarthrosis, the lack of swelling or warmth on physical exam, and the bilateral nature of his pain makes this less likely. Patellofemoral pain syndrome may secondarily contribute to his pain from a rigorous training schedule. Crystal arthropathies such as gout and pseudogout would present with more severe pain on manipulation and

Table 10.1 Differential diagnosis and associated key findings

Differential	Key findings
Hemophilic Arthropathy	Osteoarthritic changes on imaging, pain worsened with activity
Acute hemarthrosis	Swelling, warmth, pain, and stiffness in the setting of recent trauma or a known bleeding disorder
Crystal arthropathy	Joint inflammation, erythema, and intense pain with a history of similar episodes in other joints (toes, ankles, fingers)
Patellofemoral pain syndrome	Common among athletes due to overuse, aching pain in front of the knee
Bursitis	Swelling, warmth, and pain on palpation localized to a bursa
Ligament tear	Knee trauma accompanied with swelling and a "popping" sensation, leading to joint instability on exam, non-weight bearing

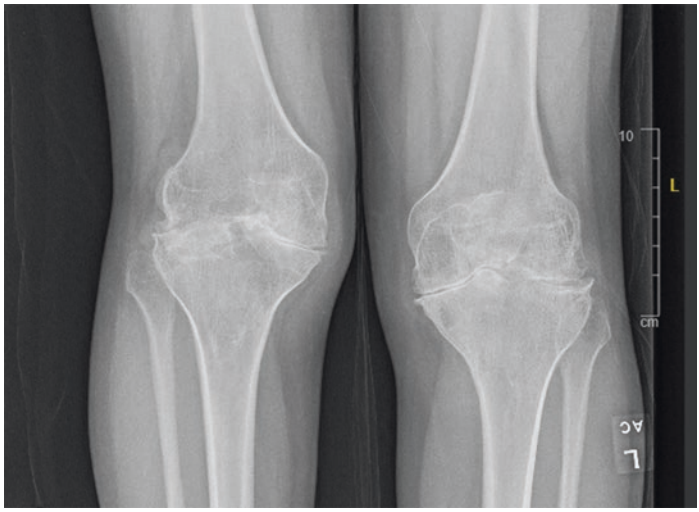

Fig. 10.1 Bilateral Knee X-Rays: Both knees show advanced arthritic changes with chronic erosions and loss of articular surfaces. No substantial effusions on either side

localized erythema. Bursitis or a ligamentous tear are less likely, as the patient does not have any effusions, lacks any pain on manipulation, and has not experienced recent trauma to the knee, or evidence of joint instability.

Hemophilic arthropathy (HA) is a degenerative joint disease (DJD) caused by repeated intra-articular bleeding events. Initial bleeding episodes have been proposed to cause toxicity for articular chondrocytes due to iron overloading, leading to impaired chondrocyte function and survival. Chronic bleeding has also been shown to cause synovial hypertrophy, leading to an increase in the number of fibroblast-like synoviocytes and macrophages promoting a cycle of further inflammation. The early phases of HA have been likened to the synovial hypertrophy and inflammation seen in rheumatoid arthritis (RA), with later stages resembling degenerative arthropathies such as osteoarthritis (OA) [1]. Patients with severe Factor VIII or IX deficiency will have spontaneous joint bleeding in childhood, leading to a high percentage

of adults (30–50% of patients) with hemophilia presenting with clinical arthropathy despite access to prophylactic factor replacement since childhood [2].

MRI has been the preferred modality for diagnostic imaging in children due to its direct visualization of hemosiderin deposits, joint effusions, marrow edema, and therefore detection of potentially reversible pathology, while radiography can only capture irreversible late-stage disease.

Stages of Hemophilic Arthropathy

This patient already has evidence of advanced stage arthritis (Fig. 10.2) from chronic hemophilic arthropathy, making MRI less consequential in informing disease management. Musculoskeletal ultrasound (MSKUS) has also been used in clinical practice for the imaging of hemophilic joints: comparison of MSKUS to MRI has shown comparable detection of soft tissue and osteochondral abnormalities, cartilage destruction, and effusions. Additionally, MSKUS modalities such as power doppler allow for more dynamic assessments of synovial blood flow and demarcation of synovitis from fluid [2].

Management

Management of hemophilic arthropathy can be separated into prevention of further bleeding events (beyond factor replacement) and treatments that decrease further joint inflammation and degeneration, as well as pain control.

Bleeding Reduction

- Radioactive synoviorthesis is a procedure in which radioactive isotopes are injected into the synovium to fibrose sub-synovial blood vessels and reverse synovial hypertrophy, leading to a reduction in the incidence of hemarthrosis (Fig. 10.3). This is currently the standard of care in patients with recurrent bleeds, is effective in more than 90% of patients, and can be performed in the ambulatory setting.

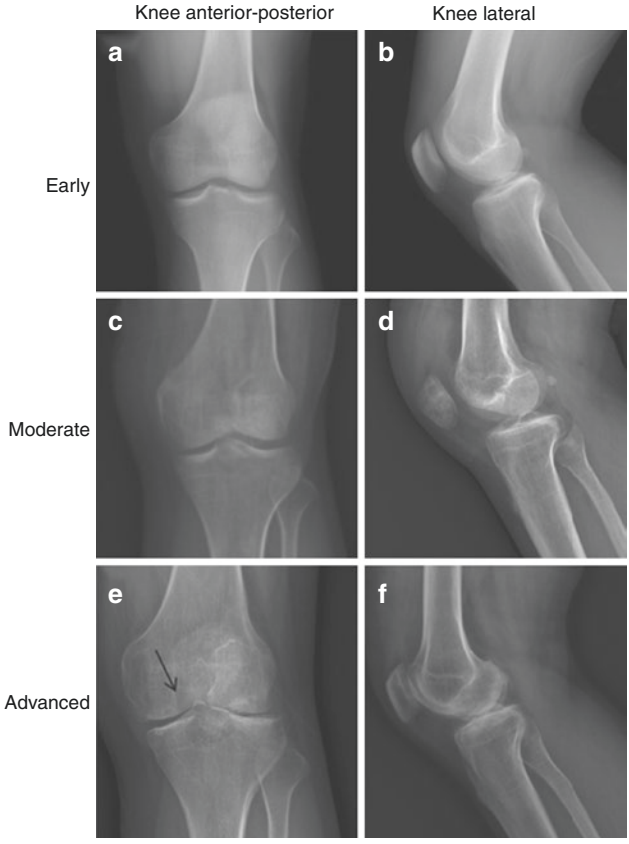

Fig. 10.2 Figure (**a**, **b**) show early stage of hemophilic arthropathy in knee joint: AP and lateral view. Note no osteoporosis, no enlarged epiphysis, no irregular subchondral surface, no narrowing of joint space, no subchondral cyst formation, no joint margin erosion, no gross incongruence of articulating bone ends, and no joint deformity. Figure (**c**, **d**) show moderate stage of hemophilic arthropathy in knee joint: AP and lateral view. Note the osteoporosis, no enlarged epiphysis, no irregular subchondral surface, narrowing of joint space (>1 mm), no subchondral cyst formation, no joint margin erosion, no gross incongruence of articulating bone ends and no joint deformity. This is Patterson grade 2. Figure (**e**, **f**) show advanced stage of hemophilic arthropathy in knee joint: AP and lateral view. Note the osteoporosis, no enlarged epiphysis, partially irregular subchondral surface, narrowing of joint space (<1 mm), 1 subchondral cyst formation (black arrow, e), no joint margin erosion, slight gross incongruence of articulating bone ends and no joint deformity. This is Patterson grade 6 [3]

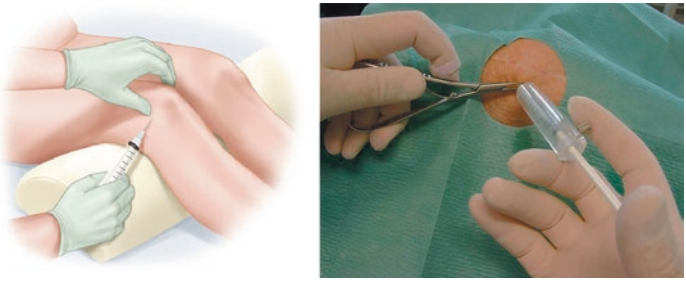

Fig. 10.3 Knee synoviorthesis (lateral suprapatellar approach) [4]

Inflammation Reduction, Tissue Regeneration, and Pain Control

- Selective Cox-2 Inhibitors such as celecoxib have been found to be effective in children with hemophilic arthropathy in a small study, showing a reduction in chronic synovitis and pain. Because these selective NSAIDs avoid exacerbating platelet dysfunction, they function as a safer option compared to traditional NSAIDs in reducing joint inflammation, especially among those with hemophilia [5].
- Tramadol has often been used in OA to diminish pain severity, leading to functional improvement without complications such as gastrointestinal bleeding or renal impairment seen in NSAIDs. Its use in late stage hemophilic arthropathy may be appropriate if other medications are contraindicated. Data regarding their efficacy and safety is still scant in this patient population, therefore they should not be regularly utilized even with severe, chronic articular pain [6].
- Intra-articular hyaluronic acid injections have been shown to have some efficacy in reducing chronic articular pain and improving functional status, usually lasting between 6 months and 1 year. However, there is a lack of robust evidence demonstrating its clinical benefits over placebo in randomized control trials. These injections are typically considered as second-line interventions after selective cox-2 inhibitors [7].

- Intra-articular corticosteroids injections (most often methylprednisolone or triamcinolone) have also been shown to relieve joint pain refractory to NSAIDs, albeit over several weeks to a month, compared to hyaluronic acid injections [7, 8]. Although the rate of adverse effects for corticosteroid injections in the short term are low, longer-term use (every 3 months for 2 years) may lead to structural damage such as cartilage loss as seen in its use for OA [9]. Therefore, corticosteroids are typically not recommended for the chronic management of hemophilic arthropathy.

Consideration 1: Comparing Intra-Articular Injections

It is important to note that strict comparisons between hyaluronic acid and corticosteroid injections regarding efficacy and side effects is challenging. First, their mechanisms of action are different: While corticosteroids help reduce the inflammatory processes associated with bleeding and synovitis, hyaluronic acid improves the viscoelastic properties of synovial fluid. Second, there is little to no research looking at these interventions in specific patient populations. Until more robust RCTs are conducted, the efficacy of these injections may vary widely across a heterogenous patient cohort.

Consideration 2: Safety of Intra-Articular Injections in Hemophilic Patients

First, it is necessary to assess whether a patient's knee pain is caused by an acute hemarthrosis (swelling, warmth, pain) or due to a chronic hemarthropathy. Patients with acute hemarthrosis require IV infusion of coagulation factor preferably within 2 h from the start of articular bleed (most patients will have factor concentrates at home), local cryotherapy to induce vasoconstriction, and joint aspiration within 2 days of bleeding to reduce joint impairment [6]. For those with chronic hemarthropathy, patients should have previous hematologic factor coverage to be consid-

ered for intra-articular injections to reduce bleeding risk [8]. Intra-articular injection of corticosteroids or hyaluronic acid may be considered after joint aspiration for acute hemarthrosis or afterwards from a safety perspective.

Knee Replacement

For those with severe hemophilic arthropathy refractory to the treatments listed above, total knee arthroplasty (TKA) should be considered for patients optimized for the procedure. Both the short- and long-term outcomes of TKA in these patients are favorable, with a high prosthesis survival rate (10-year survival achieved in 97.1% of procedures among 56 patients) and significant increases in the range of motion and functional scores [10].

Areas of Research

- Intra-articular injections of platelet-rich plasma or mesenchymal stem cells are hypothesized to aid in tissue regeneration through the addition of platelet derived growth factors in the former, and multipotent stem cells in the latter. However, they currently lack robust evidence in reversing hemolytic arthropathy and are not currently recommended [7].
- The role of certain biologics such as TNF-α is currently being investigated in addressing the early inflammatory stages of HA, as they have shown efficacy in treating rheumatoid arthritis and other inflammatory arthropathies [1].

Key Takeaways

1. Hemophilic arthropathy is a degenerative joint disease caused by repeated intra-articular bleeds often seen in patients with hemophilia, with initial presentation similar to rheumatoid arthritis and chronic degeneration leading to OA.
2. MRI and MSKUS are both modalities that can help assess the degree of early pathology, while radiographs will be sensitive only to late-stage disease.

3. Radioactive synoviorthesis is the standard of care for reducing intra-articular bleeds, while selective cox-2 inhibitors are first line in reducing inflammation. Intra-articular hyaluronic acid and corticosteroid injections are second line, while TKA is the definitive treatment for late stage arthropathy.

Non-traumatic Knee Pain with Effusion

History

The patient is a 55-year-old female with a history of thyroid disease who presented to the clinic with right-sided knee pain. The pain is localized to the right anterolateral knee, is characterized as an intermittent tingling pain graded 4/10 in severity and began a few weeks ago with no inciting event. At the beginning of the year, she started a new job that required more walking. The pain is aggravated by walking, bending the knee, going down hills and is alleviated by rest and sitting. She has tried turmeric tea, ibuprofen, and ice packs with some alleviation and has not attended physical therapy.

The patient reports having similar symptoms in the past and is not sure if she was diagnosed with arthritis at the time. The last time she had knee pain was 4 years ago, which improved with physical therapy. The pain is significantly impacting the patient's ability to perform activities of daily living.

Review of systems:
No fever, chills, cough, paresthesia, weakness, bowel/bladder changes or falls

Past medical Hx/surgeries:
- Thyroid disease
- History of thyroidectomy

Medications:
- None

Family history:
- None

Social history:
- Drinks alcohol once a week
- Recently started a new job requiring more walking

Physical exam findings:
BMI of 28 kg/m^2
Gen: Not in acute distress, well developed

Knee:
Inspection: No bony abnormalities, no muscular atrophy
Palpation:
- Right knee with effusion and warmth
- Tender to palpation to lateral joint line along area of effusion, mildly tender to distal quadriceps tendon attachment to patella
- Nontender to palpation over the prepatellar bursa, infrapatellar bursa, pes anserine, or patellar tendon

Range of motion:
- Right knee limited in flexion with pain, has full extension, no crepitus

Strength: 5/5 in lower extremities
Sensation: Grossly intact to light tough
Reflexes: 1+ bilateral patellar and Achilles
Tone: Normal

Gait: Antalgic gait

Specialized exam maneuvers:
- Pain on right Thessaly test
- Pain on right McMurray laterally
- Negative Lachman, ant. and post. drawer test
- Negative for varus/valgus laxity or stress pain

Differential Dx (Table 10.2)

Work Up: Imaging

Right Knee X-ray (AP View and Lateral View)

See Fig. 10.4.

Diagnostic Discussion

In this patient with anterolateral knee pain with associated effusion, the differential can be narrowed through the presence or absence of inciting factors, the location and severity of pain and effusion, and exam maneuvers that help pinpoint specific struc-

Table 10.2 Differential diagnosis and associated key findings

Differential diagnoses	Key findings
Meniscal tear	Caused by forceful twisting of the knee, catching sensation, knee swelling, joint line tenderness, positive McMurray and Thessaly tests
Crystal arthropathy	Joint inflammation, erythema, and intense pain with a history of similar episodes in other joints (toes, ankles, and fingers)
Quadriceps or patellar tendinopathy	Occurs over weeks to months due to overuse, pain along the superior pole (quadriceps) or inferior pole (patellar) of the knee joint, minor swelling, calcification may be present
Patellofemoral pain syndrome	Associated with joint overuse or onset of new strenuous activity involving the knee joint. Presents as an aching pain in front of the knee
Intra-articular fracture	Often caused by valgus stress on the knee, associated with rapid swelling and pain, lateral joint line tenderness and instability
Osteoarthritis	Depending on the stage of osteoarthritis, pain can be intermittent and sharp with associated stiffness, with or without chronic aching pain. Worsened by joint use and relieved by rest. Joint deformity and swelling can also be present

tural damage. The absence of notable trauma on history makes a significant ligament or tendon tear unlikely. Absence of joint laxity on anterior and posterior drawer test further supports the integrity of the ACL and PCL, while absence of varus and valgus laxity shows preservation of the MCL and LCL. Bursitis is also unlikely as the patient endorses pain along the lateral joint line as opposed to the knee bursae. An intra-articular fracture is also unlikely without significant trauma to the knee, however, could be considered due to the presence of an intra-articular body on radiograph along with joint line tenderness.

Degenerative joint disease such as osteoarthritis (OA) should also be considered in post or peri-menopausal females with activity induced joint pain and stiffness. Radiography can help discern the degree of joint space loss as well as osteophyte formation. This patient has minor OA affecting her medial joint space more than her lateral, unlikely to be the cause of her recent symptoms (Fig. 10.4). Tenderness along the distal quadriceps tendon associated with increased ambulation at work may reveal some degree of quadriceps or patellar tendinopathy.

Based on patient history, a crystal arthropathy such as gout or pseudogout should be strongly considered in the setting of acute to subacute joint pain with swelling, without any inciting trauma.

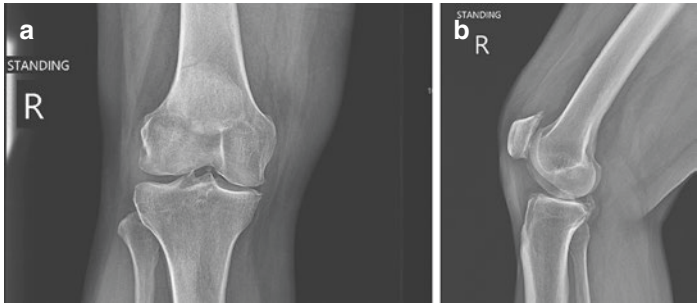

Fig. 10.4 Right knee X-rays: Medial joint compartment narrowing with small osteophytes. The lateral compartment is preserved. There are small patellofemoral osteophytes and small to moderate knee joint effusion. There is an intra-articular body in the posterior aspect of the knee. (**a**) Right knee X-ray (AP View). (**b**) Right knee X-ray (Lateral View)

Confirmatory testing is done through joint aspiration to evaluate for the presence of uric acid or calcium pyrophosphate crystals.

Procedures and Lab Tests

Ultrasound guided joint aspiration was performed at the prepatellar space. 30 cc of clear yellow joint fluid was aspirated without evidence of blood. The joint fluid was analyzed under polarized light without any crystals visualized.

The absence of crystals rules out a crystal arthropathy from our differential.

A minor meniscal tear is the most likely cause of this patient's knee pain and effusion. Pain on Thessaly and McMurray tests point towards a meniscal pathology that may have occurred during an unnotable event involving sudden pivoting or kneeling (Fig. 10.5). Joint line tenderness and effusion due to reactive synovitis area are also common findings [12].

While meniscal injuries often occur in sports and areas of profession that involve rapid changes in direction, older patients may induce minor tears with little to no trauma. Confirmatory testing for meniscal pathology can be done through MRI, especially for small defects. Radiograph and ultrasound are appropriate initial imaging modalities but are limited by their inability to visualize deeper structures. In this patient with a probable small meniscal tear, it is unlikely that obtaining an MRI would change management unless symptoms continued to persist after conservative treatment.

Management

Conservative, nonoperative management of meniscal tears is preferred in patients who do not have severely restricted range of motion, locking, or instability of the knee. A regimen for this patient may include the following, with gradual resolution of symptoms over 6 weeks [13].

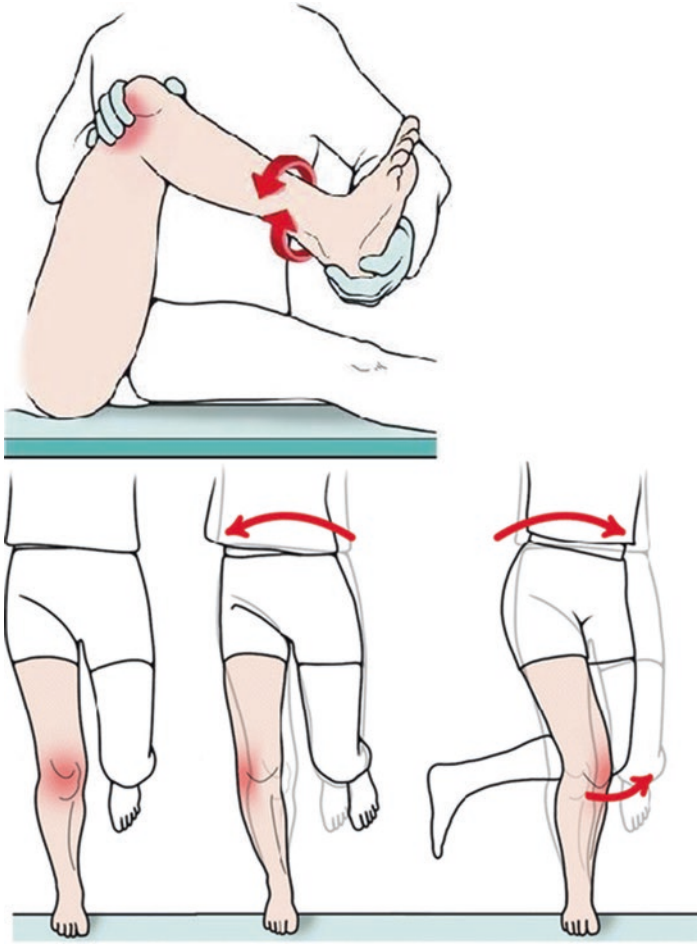

Fig. 10.5 McMurray and Thessaly tests for evaluation of meniscal injury [11]

- Rest, ice, and compression of the knee.
- Offloading until fully weight bearing.
- NSAIDs for pain relief.
- Physical therapy.

- Controlling and managing swelling while maintaining range of motion.
- Quadriceps and hamstring strengthening.
- Proprioceptive training.

If there is no improvement in symptoms after undergoing conservative therapy, it is reasonable to obtain an MRI of the knee to further assess the degree of meniscal injury, or the presence of a separate concomitant injury.

Operative management could be considered with failure of conservative therapy and meniscal lesions on MRI amenable to partial meniscectomy or direct repair. Regarding clinical characteristics attributed to successful repair, this patient's older age (>40) is a negative prognosticator while most positive prognosticators depend on the nature of the tear (acute tears, vertical tears, red-red zone tears, no mechanical misalignment, and tears longer than 1 cm but shorter than 4 cm) [13].

While partial meniscectomies are a common orthopedic procedure, no RCTs have supported its superiority compared to nonsurgical management, with several studies showing an increased risk of future OA. If the patient has a meniscal tear not appropriate for direct repair, then meniscectomy could be discussed regarding risks, benefits, and variability in success [13].

Intra-articular steroid injections can also be performed in patients who have concomitant mild OA, such as in this patient [12].

Areas of Research

- Platelet Rich Plasma and Mesenchymal Stem Cells have shown promising results in promoting meniscal recovery and healing with and without surgical intervention. These orthobiologics may address at treatment gap for patients who fail conservative

management and are not candidates for surgical intervention [13].

Key Takeaways

1. Meniscal tears are often caused by sudden changes in direction that cause forceful pivoting of the knee joint. However, minor meniscal tears can occur in older patients with little to no trauma, and can present with joint line tenderness, effusion, and pain on ambulation.
2. Physical exam maneuvers such as the Thessaly and McMurray tests are useful in diagnosing a meniscal pathology. Confirmatory testing with MRI is indicated in patients who fail conservative management over 4–6 week period.
3. Conversative management is preferred in patients who do not have joint instability or severe restrictions in range of motion. Operative management (direct repair vs. partial meniscectomy) should be decided based on the patient's age, degree and location of meniscal injury, and other concomitant conditions such as OA.

Left Knee Swelling for 1 Month

History

Patient is an active 37-year-old female with no significant past medical history, who presents to the orthopedics clinic for evaluation of left knee swelling for 1 month. She had a motorcycle accident 1 month ago, in which she was traveling at about 50–60mph at the time of the accident and was wearing a helmet. She was seen at an ER and was found to have a left shoulder dislocation, which was reduced at this time. She also had left knee pain and swelling after the accident; however, patient states no imaging of the knee was done at the time. She continues to have persistent swelling of the left knee, which is exacerbated by deep bending and alleviated with extension of the knee. When she

bends her knee, she feels "pressure within the knee." She denies any trauma prior to the accident, surgery, or injection to the left knee. She denies numbness, tingling, decreased ROM, or buckling.

Review of systems: No fever, chills, cough, paresthesia, weakness, bowel/bladder changes or falls *Past medical Hx/surgeries:* No significant past medical history No past surgical history *Medications:* None *Family history:* No significant family history *Social history:* Patient works as a carpenter. She enjoys playing yoga, cycling, and playing tennis. She smokes cigarettes 1–2× weekly and drinks alcohol socially	*Physical exam findings:* **Wt**: 75.8 kg (167 lb) **BMI** 22.65 kg/m^2 **General:** In no acute distress *Musculoskeletal exam*: ***Left knee*:** **Inspection**: Large anterior effusion, no deformity, no erythema, skin intact **Palpation**: Non-tender to palpation. **Movement**: ROM: 0–135, within normal limits. Negative crepitus **Strength**: 5/5 strength **Sensation**: Intact to light touch, well perfused **Special exam maneuvers:** Lachman: Positive compared to other side but with endpoint Negative McMurrays Negative Thessalys Negative valgus/valgus stress Negative patellar grind Negative patellar apprehension Positive dial test at 90, negative at 30 **Gait**: Normal gait **Right knee exam unremarkable**

Differential Dx (Table 10.3)

Work Up: Imaging

The patient obtained an MRI of the left knee with the following results (Fig. 10.6):

Table 10.3 Differential diagnosis and associated key findings [14]

Differential	Key findings
Anterior cruciate ligament (ACL) tear	Sudden change in direction or landing from a jump (usually audible "pop" at time of injury), positive Lachman and/or anterior drawer test, history of direct trauma to knee, rapid swelling, instability with weight bearing, decreased ROM
Morel-Lavallee lesion	Caused by rotational (shearing) force, often from a motor vehicle collision. Localized swelling, contusion, abrasion, and/or decreased sensation may also be present
Prepatellar and infrapatellar bursitis	Recurrent trauma to anterior knee, seen in individuals who kneel frequently ("housemaid's knee"). Can also be caused by infection, gout, or arthritis. Relief with flexion of knee. Presents with swelling and tenderness over anterior knee
Lipoma	Present as soft, painless subcutaneous nodules of mature fat cells enclosed by thin fibrous capsules. Rarely, may involve fascia or deeper muscular planes. Most frequently occur on trunk and upper extremities, and usually patients have multiple
Subcutaneous hematoma	Presents with pain, drop in hemoglobin, or fluid collection on imaging Studies. May see enlarging mass on exam
Soft tissue sarcoma	Presents as gradually enlarging, painless mass, most commonly in extremities. May have paresthesias or edema. Rarely symptoms of fever and/or weight loss

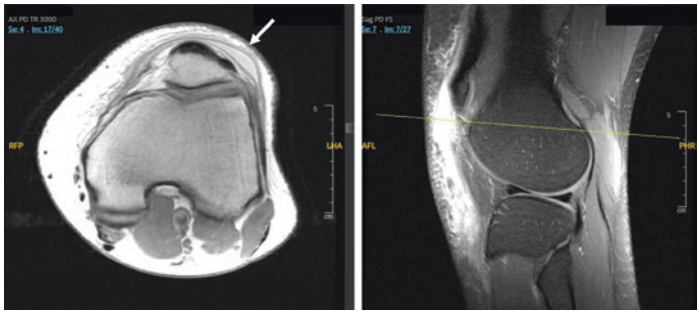

Fig. 10.6 MRI of left knee

Impression:

1. Small peripheral undersurface tear of the body of the medial meniscus.
2. Mild to moderate chondromalacia of the patellar articular cartilage, greater laterally than medially.
3. Mild patella alta and findings of mild soft tissue secondary to abnormal patellar tracking.
4. Joint fluid/cysts: Small complex Baker's cyst. Subcutaneous edema predominantly in the prepatellar and infrapatellar soft tissues with more confluent fluid collection in the prepatellar space. Thin prepatellar fluid collection extends proximally, superficial to the quadriceps tendon and vastus medialis, and distally superficial to the patellar tendon, beyond expected confines of pre-patellar bursa, and likely represents sequela of prior trauma with possible hemorrhage into and rupture of the prepatellar bursa with subsequent loculation (Fig. 10.7).
5. No fracture or bone contusion.

Diagnostic Discussion

Degloving soft tissue injuries occur when the skin and subcutaneous fatty tissue are abruptly separated from the underlying deep fascia in the setting of severe trauma, most commonly motor vehi-

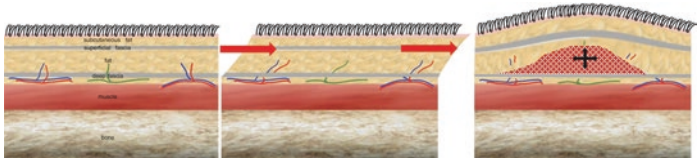

Fig. 10.7 Shearing between the subdermal plane and the fascia leading to distended cavity

cle collisions. As a result, there may be cavity formation following disruption of the lymphatics and subdermal capillaries; the cavity may consist of lymphatic fluid, debris, and subdermal fat (Fig. 10.7) [15]. This collection of fluid may spontaneously resolve or persist if it becomes encapsulated [16]. When degloving injuries do not involve a break in the skin (closed injury), they are called Morel-Lavallee lesions. These lesions classically occur in the hip (i.e. greater trochanter of the femur) and proximal thigh, but can occur in any area in which there is severe mechanical force such as over the lumbar spine, the scapula, or the pre-patellar knee [17].

The typical presentation is an enlarging, painful lesion following trauma, which usually occurs within hours to days of the event [18]. Contusions, abrasions, and/or decreased sensation may also be present [17, 18]. A small study showed Morel-Lavallee lesions can form as early as a few hours after trauma, or as late as 13 years from the time of injury, with a median time of presentation of 1.4 years [19]. Data shows one-third of acute lesions are missed or considered insignificant, especially when more severe injuries are present [17].

MRI is the modality of choice for diagnosis, which can clearly demonstrate fluid collection and the associated underlying fascia [18]. CT scan can be useful at showing fluid–fluid levels and capsule formation, while ultrasound can be used to identify fluid, internal debris such as fat globules, and capsule formation (Fig. 10.8) [15]. Morel-Lavallee lesions are typically compressible and have no vascularity on ultrasound [16, 18]. The Mellado-Bencardino classification system characterizes Morel-Lavallee lesions based on shape, signal and enhancement, and the presence or absence of a capsule observed on MRI [20].

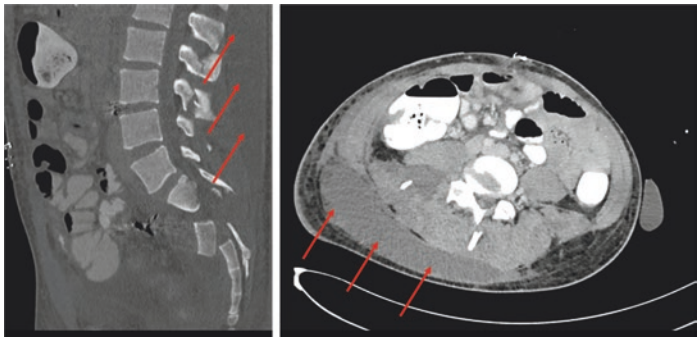

Fig. 10.8 CT demonstrating Morel-Lavellee lesion after high-energy fall

Management

Prompt identification of Morel-Lavallee lesions is essential, as delay in diagnosis or a missed lesion can lead to infection, extensive soft tissue necrosis, loss of limb, or death [20, 21]. There is currently no universally accepted treatment algorithm for the management of Morel-Lavallee lesions, however, studies have shown for small lesions without an identified capsule, conservative treatment with compression is recommended [20, 22]. Data shows treatment with compressive elastic bandages or corsets for patients with small fluid accumulation had successful healing within an average of six weeks [23]. However, other data has suggested that healing time is significantly less for patients who receive surgical intervention over those where compression is used alone [24].

Percutaneous aspiration is another option for treatment; however, majority of lesions recur following aspiration. Studies have shown if there is percutaneous aspiration of more than 50 mL of fluid from Morel-Lavallee lesions, there is a higher likelihood of recurrence, and surgical intervention would be recommended [17]. The use of sclerosing agents to close off pathological cavities has also been applied to the treatment of these lesions and is often recommended when percutaneous aspiration alone fails. Doxycycline is the most used agent, in addition to erythromycin,

bleomycin, vancomycin, absolute ethanol, tetracycline, and talc. Most of these agents work by causing cell destruction within the periphery of the lesion, which obliterates the cavity and induces fibrosis [24]. Studies show sclerodesis has an efficacy of 95.7% in treating Morel-Lavallee lesions and is recommended as first line treatment for acute lesions or chronic lesions up to a volume of 400 mL [24, 25].

In circumstances where patients have lesions with overlying skin that is necrotic, then debridement of the dead tissue is required with reconstruction. If the overlying skin is viable, open drainage is another option for treatment, which involves curettage of the cavity to induce fibrosis. Quilting sutures, fibrin sealant, and low suction drains have also proved to be successful adjuncts to this method. Lastly, mass resection is typically indicated for lesions with an intact capsule and when other measures have failed and/or fluid volume is greater than 400 mL [24].

Patient Conclusion

The patient above was referred to physical therapy for her left knee and left shoulder and instructed to start a home exercise program. An MRI of the left knee was ordered due to concern for ACL/PCL injury given the ligamentous laxity and large effusion of the left knee, and she was advised to ACE wrap her left knee during the day and ice as needed until her MRI results came back. Patient returned to the clinic 1 month later and stated her left knee swelling had significantly improved and noted that she was back to most of her normal activities. On exam of the left knee, there was mild residual swelling and the Morel-Lavallee lesion diagnosed on MRI was resolving without any additional therapies.

Key Takeaways

1. Degloving soft tissue injuries occur when the skin and subcutaneous fatty tissue are abruptly separated from the underlying deep fascia in the setting of severe trauma, creating a collection of fluid.

2. When degloving injuries do not involve a break in the skin (closed injury), they are called Morel-Lavallee lesions.
3. They present as an enlarging, painful lesion following trauma, which usually occurs within hours to days of the event.
4. MRI is the modality of choice for diagnosis, which can clearly demonstrate fluid collection and the associated underlying fascia; however, CT scan and ultrasound can also aid in identification of fluid accumulation and capsule formation.
5. Prompt identification of Morel-Lavallee lesions is essential, as delay in diagnosis or a missed lesion can lead to infection, extensive soft tissue necrosis, loss of limb, or death.
6. Conservative treatment includes compressive elastic bandages or corsets for patients with small fluid accumulation.
7. While percutaneous aspiration may also be used for treatment, sclerodesis has shown the greatest efficacy in treating Morel-Lavallee lesions and is recommended as first line treatment for acute lesions or chronic lesions up to a volume of 400 mL.
8. Open drainage with the adjunctive use of quilting sutures, fibrin sealant, and low suction drains has also proved to be effective.
9. Mass resection is indicated for lesions with an intact capsule, and when other measures have failed and/or fluid volume is greater than 400 mL.

Diffuse Knee Pain and Decline in Ambulation

History

Patient is a 62-year-old female with past medical history of hypertension, diabetes mellitus, multiple sclerosis, polymyositis, morbid obesity, who presents to the orthopedics clinic for follow-up of worsening bilateral diffuse knee pain and loss of mobility. She states her knee pain has limited her ability to use the toilet or shower, and she often uses a rolling office chair to get around her

home due to painful standing and walking. She has received seven bilateral steroid knee injections and four bilateral hyaluronic acid knee injections over the past 3 years with moderate relief lasting a few months.

Review of systems:
No fever, chills, cough, paresthesia, weakness, bowel/bladder changes or falls.
Past medical Hx/surgeries:
Hypertension
Diabetes mellitus
Multiple sclerosis
Polymyositis
Morbid obesity
Chronic osteoarthritis
No significant surgical history
Medications:
Amlodipine 5 mg tablet
Lopressor 50 mg tablet
Gabapentin 600 mg tablet
Tramadol 50 mg tablet
Lidocaine patch
Voltaren 75 mg tablet
Voltaren topical gel
Tylenol 500 mg
Family history:
No significant family history
Social history:
Works as a schoolteacher (on disability). Former smoker (quit date: 09/27/1992), 0.5ppd, 8 pack years. Does not drink alcohol

Physical exam findings:
Ht: 5′5″
Wt: 151 kg (333 lb)
BMI: 55.41 kg/m^2
Gen: Not in acute distress, morbidly obese
Musculoskeletal exam:
Inspection:
Bilateral knees: Joint effusion observed. No obvious bony deformities or varus/valgus alignment, however limited by body habitus
Palpation:
Bilateral knees: No tenderness to palpation over medial or lateral joint lines.
Movement:
Bilateral knees: ROM of the knees restricted by body habitus. Pain in both flexion and extension. Crepitus bilaterally.
Strength: 5/5 in bilateral lower extremities
Sensation: Grossly intact to light touch
Special exam maneuvers:
Negative straight leg raise
Negative anterior and posterior drawer test
Negative Lachman's test
Negative McMurray's test
Gait: Sit to stand time increased. Antalgic gait, only able to tolerate a few steps

Differential Dx (Table 10.4)

Work Up: Imaging

See Fig. 10.9.

Diagnostic Discussion

Osteoarthritis (OA) is the most common form of arthritis in the knees, hips, and hand joints, and is one of the most prominent causes of pain, functional limitation, and reduced quality of life worldwide. Knee OA accounts for approximately 80% of the dis-

Table 10.4 Differential diagnosis and associated key findings [26]

Differential	Key findings
Osteoarthritis	Middle-aged or older patients. Usually bilateral knees. Pain in joints typically worse after activity. Radiographs show joint space narrowing, osteophytes, and subchondral erosions. Absence of RF and CCP (see below)
Rheumatoid arthritis	Middle-aged or older patients. Stiffness of joints typically worse after rest. Presence of rheumatoid factor (RF), antibodies to cyclic citrullinated peptide (CCP), and elevated ESR, CRP
Infectious arthritis	Single joint with tenderness, redness, warmth, swelling, and restricted ROM. Inability to weight bear. Patients often febrile. Synovial fluid with >20,000 WBCs
Crystalline arthritis or gout	Severe pain, redness, warmth, swelling, and restricted ROM. Flares often occur at night. Synovial fluid with monosodium urate (MSU) crystals. Tophi on exam and radiographs
Ligamentous or meniscal tear	Typically involves trauma to the knee, such as sudden change in direction, landing, twisting or direct force. Positive special tests (i.e., Lachman or anterior drawer test for ACL tear). Rapid swelling, instability with weight bearing, decreased ROM

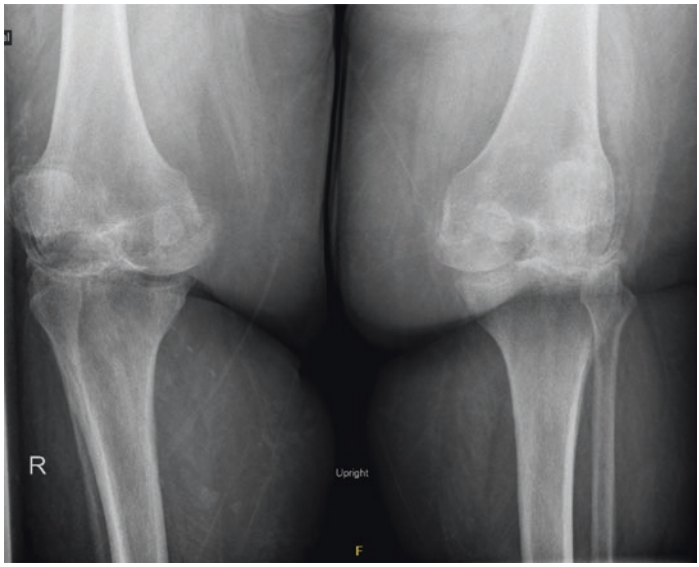

Fig. 10.9 Bilateral knee X-rays: Severe end stage tricompartmental OA and osteophytes

ease and most commonly affects the patellofemoral and medial tibiofemoral joints [26, 27].

It is caused by localized loss of cartilage in the joint, subsequent remodeling of adjacent bone, and associated inflammation [28]. The most notable risk factor for OA is age, where the mechanism of joint damage is poorly understood but thought to be multifactorial (oxidative damage, thinning cartilage, muscle weakening, reduction in proprioception, etc.) [29]. "Chondrosenescence" describes the age-dependent deterioration of chondrocytes, the cells responsible for cartilage formation, and this can lead to the cartilage dysfunction seen in OA. The term "inflammaging" has also been used to describe the low-grade inflammation that occurs as people age and is a major contributing factor to the progression of OA [30]. Obesity also plays an important role in the development of OA, especially in weight-bearing joints such as the knees and hips. Data shows that indi-

viduals with BMI > 30 kg/m^2 were 6.8 times more likely to have knee OA than controls with normal-range BMIs [31]. Modifiable risk factors such as obesity, comorbidities (i.e., diabetes, cardiovascular disease), and sedentary lifestyles have been shown to heavily influence the course of disease progression [30].

Prevalence of OA is also associated with previous joint injury and abnormal loading of the joints. Studies have shown that anterior cruciate ligament (ACL) tears are associated with early-onset knee OA in 13% of cases after 10–15 years. When ACL tears also involve injury to cartilage, subchondral bone, collateral ligaments and/or menisci, the prevalence of OA increases to 21–40% [29]. Furthermore, data suggest that people with occupations that involve frequent squatting, kneeling, stair climbing, crawling, and/or bending had increased risk of developing knee OA. Such occupations include workers in construction, firefighting, agriculture, fisheries, forestry, and mining [32].

Osteoarthritis usually presents as joint pain (often at the end of the day), stiffness, limited motion, swelling, and in some cases joint deformity depending on advancement of disease [26]. Diagnosis of osteoarthritis is typically clinical; however, imaging can be useful when the diagnosis is unclear. Radiographs are the most widely used form of imaging and can identify key features of OA such as osteophytes, joint space narrowing, cysts, and subchondral sclerosis [33] (Fig. 10.9). One limitation of radiographs, however, is that they can be insensitive in early disease. While an MRI is not typically necessary for diagnosis, it can identify OA at earlier stages and rule out OA when there is suspicion for other pathologies, such as ligamentous and/or meniscal tears [34].

Management

Management of knee osteoarthritis varies depending on the severity of the disease. For patients with mild to moderate OA, symptoms are usually controlled with nonpharmacologic therapies such as exercise therapy, muscle strengthening, and weight loss. Exercises that strengthen the quadriceps femoris are thought to be the most effective method both for the treatment and prevention of

knee OA, as this muscle maintains the stability of the knee joint and allows for its extension [35]. Studies show that exercise therapy has the same pain-relieving effect as NSAIDS and 2–3× the effect of acetaminophen in patients with knee OA, without the side effects or risk of dependence associated with pharmacologic therapies. Furthermore, exercise therapy is essential in the management of OA, not only for its positive impact on joint pain and function, but because of its role in the prevention and/or improvement of chronic conditions that are often associated with OA. Studies show 2/3 of patients with knee and hip OA have one or more comorbidities, including type 2 diabetes, hypertension, and depression, and physical activity can help these patients maintain good health [36]. Recreational or moderate intensity exercises such as walking, long-distance running, swimming, biking, and Tai chi are recommended for patients with OA. Elite level activities, particularly contact sports, which can lead to injury or post-traumatic OA are not recommended [30].

If symptoms persist, it is recommended to add topical therapies (i.e., diclofenac gel, capsaicin cream or patches) or oral analgesics as needed [37]. Studies have also shown that some nutritional supplements, such as curcumin and *Boswellia serrata*, can be beneficial for the treatment OA due to their anti-inflammatory and analgesic properties, however, research on their efficacy is limited [38, 39]. For moderate to severe OA, treatment also starts with nonpharmacologic treatment as above, with emphasis on aquatic over land-based exercise due to reduced load on the knees [40]. Other supportive measures include knee braces, specialized footwear, assistive walking devices (i.e., canes), and cognitive behavioral therapy for pain coping.

Alternatives for treatment include the addition of oral NSAIDS, intra-articular steroids, duloxetine, and eventually surgery if indicated [37]. For patients with inadequate response to oral NSAIDS or concomitant comorbidities in which oral NSAIDS are contraindicated, duloxetine is the next suggested treatment. Intra-articular glucocorticoids are not recommended unless patients have moderate to severe pain and there has been failure of relief with the other treatment options. Triamcinolone or methylprednisolone are most commonly used, although betamethasone can

also be used. Studies show that serial injections every 3 months is not recommended due to findings of increased cartilage volume loss measured on MRI and no effect on knee pain long-term compared to controls with saline injections [41]. Other studies have shown that intra-articular glucocorticoid injections do provide short-term relief for up to 6 weeks post-injection, however, after this period the effects on knee pain and function were similar to the placebo injections [42]. Additionally, physical therapy was shown to be more effective for pain relief and improvement in functional debility compared to glucocorticoid injections after 1 year [43].

Lastly, surgery is recommended for patients after significant symptoms persist despite nonsurgical interventions. Total knee replacement is the most common type of surgery, and osteotomy is an option for younger patients with varus or valgus deformity [36, 44].

Patient Conclusion

The patient above had bilateral methylprednisolone injections on the day of her visit. Physical therapy had been recommended; however, she did not feel comfortable going out frequently on her own and is wheelchair bound. It was determined by the clinician that this patient requires bilateral total knee replacement, however, she was not a good candidate due to her body habitus. Patient was encouraged to increase physical activity and to initiate a weight loss program.

Key Takeaways

1. Osteoarthritis (OA) is the most common form of arthritis in the knees, hips, and hand joints, and knee OA accounts for approximately 80% of the disease.
2. The most notable risk factors of OA are age, obesity, previous joint injury, and abnormal loading of the joints.

3. Osteoarthritis of the knee usually presents as joint pain (often at the end of the day), stiffness, limited motion, swelling and in some cases joint deformity.
4. Diagnosis of osteoarthritis is typically clinical. Radiographs are the most widely used form of imaging and can identify key features of OA such as osteophytes, joint space narrowing, cysts, and subchondral sclerosis.
5. For mild OA, symptoms are usually controlled with nonpharmacologic therapies such as exercise, muscle strengthening, weight loss, topical therapies, and nutritional supplements.
6. Exercise therapy has been shown to be safer and more effective than pharmacologic treatments and can help with prevention and/or improvement of chronic conditions that are often associated with OA.
7. For moderate to severe OA, persistent symptoms involve the addition of supportive measures such as knee braces, specialized footwear, and assistive walking devices, as well as oral NSAIDS, duloxetine, and intraarticular steroids.
8. Surgery is recommended for patients after significant symptoms persist despite nonsurgical interventions. Total knee replacement is the most common type of surgical intervention.

Chronic Knee Pain in a 12-Year-Old

History

A 12-year-old male presents to clinic with complaint of anterior left knee pain and popping that was first noted roughly 1 year ago. The pain has gradually worsened over time from a dull ache to moderate pain after physical activity causing him to limp. The pain is exacerbated by jumping, running, and climbing stairs. He has no recent trauma or injury.

Review of systems: No fever, chills, cough, paresthesia, weakness, bowel/bladder changes or falls. *Past medical Hx/surgeries:* ADHD *Medications:* Concerta *Family history:* None *Social history:* Patient lives at home with his mother and brothers. He attends seventh grade and enjoys basketball, baseball, and football	*Initial physical exam findings:* **BMI:** 19.6 **Height:** 4′ 11″ **Gen:** Not in acute distress, well developed *Left knee:* **Inspection:** Mild swelling of the left knee located anterior and inferior to patella **Palpation:** • No tenderness to palpation over medial or lateral joint lines, or over pes anserine • Mild tenderness to palpation over the tibial tubercle • General tenderness to palpation over anterior knee, including patellar tendon **Movement:** 0–140 degrees of flexion without significant pain **Strength:** 5/5 in bilateral lower extremities **Sensation:** Grossly intact to light touch *Special exam maneuvers:* Negative Lachman, anterior/posterior drawer test Negative valgus stress or laxity test Negative varus stress or laxity test Negative McMurray's test

Differential Dx (Table 10.5)

Work Up: Imaging

Left Knee X-ray: AP Lateral View

See Fig. 10.10.

Table 10.5 Differential diagnosis and associated key findings

Differential	Key findings
Patellofemoral pain syndrome	Associated with joint overuse or onset of new strenuous activity involving the knee joint. Presents as an aching pain in front of the knee
Osgood-Schlatter disease	Osteochondrosis caused by traction of the proximal tibial tubercle secondary to repetitive contractions of the quadricep muscle. Often presents as pain and swelling at the tibial tubercle
Sinding-Larsen-Johansson disease	Osteochondrosis caused by traction of inferior pole of the patella due to repetitive quadricep contractions, similar mechanism to Osgood-Schlatter. Associated with pain at the inferior border of the patella
Quadriceps/patellar tendinopathy	Occurs over weeks to months due to overuse, pain along the superior pole (quadriceps) or inferior pole (patellar) of the knee joint, minor swelling, calcification may be present
Prepatellar/infrapatellar bursitis	Swelling, warmth, and pain on palpation anterior to the patella or patellar tendon. Often caused by direct, repetitive trauma to the knee such as kneeling
Fat pad impingement	Impingement of the infrapatellar fat pad between the inferior poles of the patella and femur, leading to diffuse pain over the patella exacerbated by knee extension

Bilateral Knee X-ray: AP Merchant View

See Fig. 10.11.

Diagnostic Discussion

Chronic knee pain in a pediatric patient (lasting longer than 6 weeks) should be evaluated for the following etiologies: pain onset related to excessive activity, impaired healing after an acute injury, congenital/anatomical anomalies, and systemic causes.

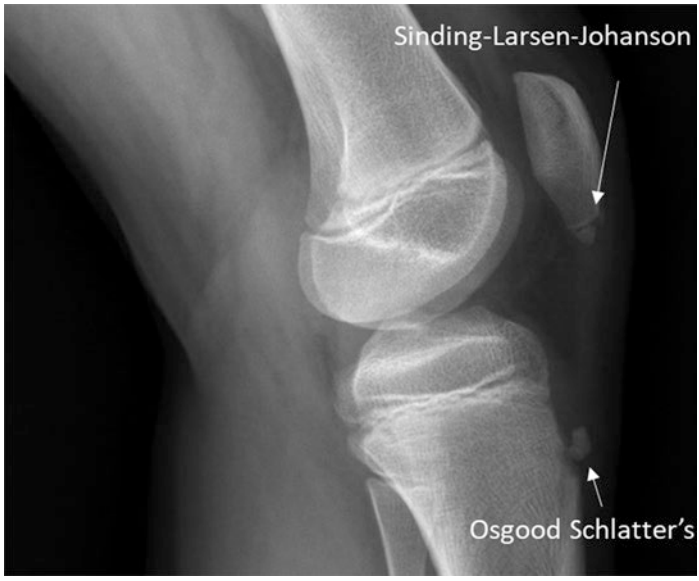

Fig. 10.10 Enthesopathy of left knee seen on the distal aspect of the patella

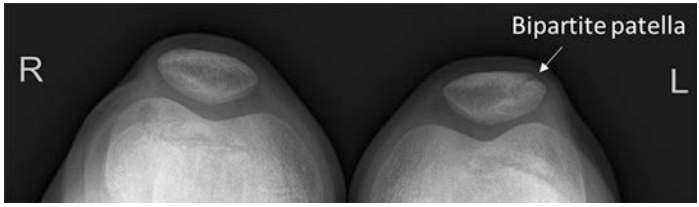

Fig. 10.11 Bipartite patella on the left

This patient's worsening knee pain over the past year is associated with physical activity without any notable trauma. The physical exam does not show any evidence of joint laxity or decreased range of motion which would point more towards a ligamentous or meniscal injury. As such, the differential can be narrowed to several pathologies relating to joint overuse injury, tendinopathy, and bursitis. Localization to the anterior knee is more reflective of patellar or quadricep involvement, or bursitis in the anterior com-

partment. Iliotibial band injury (typically localized to the lateral knee) or pes anserine bursitis (typically localized to the medial knee) are less likely.

Patellofemoral pain syndrome is one of the most common causes of anterior knee pain among physically active patients, often associated with ascending, descending stairs, squatting, and jumping. Overuse of the knee joint can lead to tightening and weakening of surrounding connective tissue, causing inflammation between the femoral and patellar joint space secondary to misalignment or excessive compressive forces on the synovial space.

Osgood-Schlatter disease and Sinding-Larsen-Johansson disease are also common overuse injuries in the pediatric population due to traction on the proximal tibial tubercle and inferior pole of the patella respectively. Symptoms typically arise in adolescents (ages 12–15) during their growth spurt and within highly active children [45]. Radiographic evaluation can be helpful in elucidating the primary contributor to this patient's knee pain.

Quadriceps or patellar tendinopathy is also associated with overuse but is typically associated with localizable pain to either the superior or inferior pole of the knee joint. Prepatellar or infrapatellar bursitis is less likely due to lack of erythema or warmth. Fat pad impingement is also unlikely as the patient's pain is not exacerbated by knee extension.

This patient has radiographic evidence of osteochondrosis both at the proximal tibial tubercle (Osgood-Schlatter) and the inferior pole of the patella (Sinding-Larsen-Johansson), as well as a bipartite patella seen on merchant view which may contribute to the patient's anterior knee pain (Figs. 10.10 and 10.11). Repetitive strain on the insertion sites of the patellar tendon can result in small avulsion fractures which result in sclerosis and fragmentation of the tibial tubercle or patella [45].

Management

Both Osgood-Schlatter and Sinding-Larsen-Johansson syndrome are usually self-limited with most adolescents responding well to

conservative treatment over several months. This includes rest from sports-related/strenuous activity, anti-inflammatory medications, and physical therapy geared towards hamstring and quadricep stretches, range of motion exercises, and core stability. Symptoms typically resolve at skeletal maturity with closure of the apophysis, which may range from several months to 1–2 years [46].

For patients who continue to have disabling symptoms after closure of the apophysis, surgical intervention may be warranted. Persistent pain can usually be attributed to the presence of free ossicles and enlargement of the anterior portion of the tibial tubercle (Fig. 10.12). Both endoscopic and open operative approaches are available to remove these ossicles or reduce the enlarged tubercle with beneficial outcomes [48].

Areas of Research

While corticosteroid injections are not recommended in the pediatric population due to increased risk for atrophy and tendon rup-

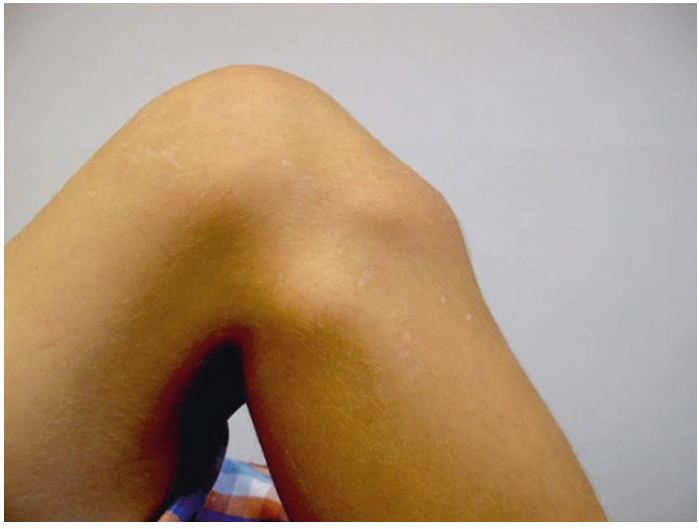

Fig. 10.12 Typical clinical appearance of Osgood Schlatters with marked tibial tubercle swelling [47]

ture, joint injections with hyperosmolar dextrose and lidocaine have been explored in improving symptoms, especially during sports-related activity [49, 50].

Key Takeaways

1. Chronic knee pain in the pediatric population can often be categorized by the following considerations: pain onset with activity, impaired healing after trauma, congenital anomalies, and systemic conditions.
2. Anterior knee pain worsened by physical activity can often be attributed to one or more of the following conditions: Osgood Schlatter disease, Sinding-Larsen-Johansson disease, patellofemoral pain syndrome, or quadriceps/hamstring tendinopathy.
3. Both Osgood Schlatter disease and Sinding-Larsen-Johansson disease are characterized by joint overuse leading to traction apophysitis of the tibial tubercle or inferior patella respectively. Radiographic imaging may show associated osteochondrosis with small avulsion fractures and free ossicles.
4. These conditions are typically self-limiting and improve with conservative management. While most cases resolve with closure of the apophysis, refractory cases may warrant surgical intervention.

Posterior Knee Pain

History

A 88-year-old-female with history notable for a right nondisplaced femoral neck fracture (with closed reduction and percutaneous pinning) presented with worsening chronic bilateral knee pain. The pain is localized to her posterior knees and is worsened with flexion and prolonged standing. She has difficulty with ambulation and takes acetaminophen four times a day for pain alleviation. She reports no recent history of knee trauma. She reports her knee was drained four times and she has received four steroid injections in the past 2.5 years with initial improvement in pain lasting for 1–2 weeks.

Review of systems:
No fever, chills, cough, paresthesia, weakness, bowel/bladder changes or falls.
Past medical Hx/surgeries:
Coronary artery disease
History of cardiac stent placement
Type 2 diabetes mellitus
Hypertension
Deep vein thrombosis
Right nondisplaced femoral neck fracture (with closed reduction and percutaneous pinning)
Medications:
- Apixaban
- Furosemide
- Gabapentin
- Diclofenac gel
- Aspirin (81 mg)
- Atorvastatin
- Hydrochlorothiazide
- Metoprolol succinate
- Metformin
- Quinapril

Family history: Non-contributory

Physical exam findings:
BMI: 35.5
General: Not in acute distress, well developed
Cardiovascular: Well perfused
Knees:
Inspection:
Left knee varus deformity
Diffuse bony overgrowth of bilateral knees
Palpation:
No erythema/edema
Tenderness to palpation to posterior left knee; crepitus in bilateral knees
Movement:
Right knee flexion 100 degrees
Left knee flexion 120 degrees
Bilateral knee extension 0 degrees
Strength: 5/5 in lower extremities
Sensation: Grossly intact to light touch
Gait: Slowed cadence, antalgic, ambulates with walker
Special exam maneuvers:
Positive anterior drawer test bilaterally
Negative McMurray's test
Negative for varus/valgus laxity or stress pain

Differential Dx (Table 10.6)

Work Up: Imaging

Left Knee MRI (Fig. 10.13)

Left knee MRI results: Tricompartmental osteoarthrosis, most pronounced in the medial joint compartment. Moderate joint effusion with synovitis. Intra-articular body in the posterior aspect of the knee. Degenerative tearing of the medial and lateral menisci. Chronic complete rupture of the ACL. Mild degenerative change of the PCL and MCL. Small Baker's cyst.

Table 10.6 Differential diagnosis and associated key findings

Differential	Key findings
Anterior cruciate ligament tear	Sudden change in direction or landing from a jump (usually audible "pop" at time of injury), positive Lachman and/or anterior drawer test, history of direct trauma to knee, rapid swelling, instability with weight bearing, decreased ROM
Popliteal Baker's cyst	Joint fluid contained in a cyst localized to the posterior capsule, worsened by activity typically involving repetitive knee flexion. Presents as posterior knee pain and a palpable cyst in the popliteal fossa (with knee in extension)
Osteoarthritis	Depending on the stage of osteoarthritis, pain can be chronic or intermittent with associated stiffness and reduced range of motion. Worsened by joint use and relieved by rest. Joint deformity and swelling can also be present
Popliteus/biceps femoris tendinopathy	Can be caused by acute or chronic trauma, most associated with downhill activities. Pain to palpation at the tendon insertion site, anterior to the lateral femoral condyle
Popliteal artery aneurysm	Seen in patients with cardiovascular risk factors. Claudication leading to chronic or acute posterior knee pain, associated with a pulsatile mass in the popliteal fossa

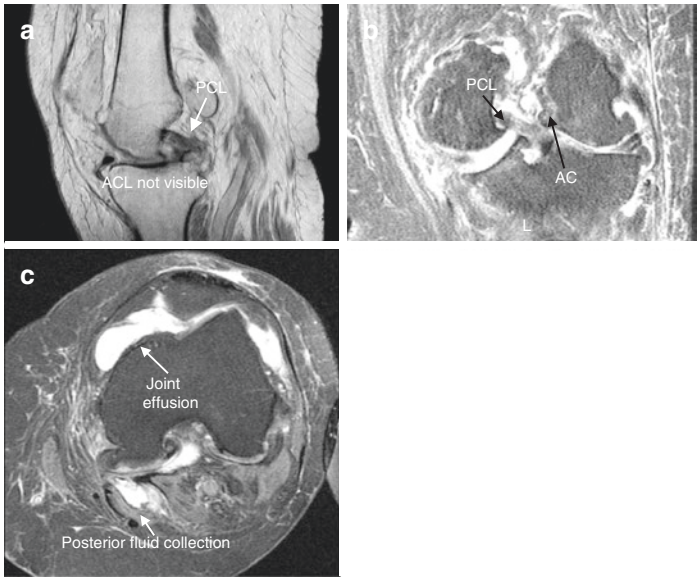

Fig. 10.13 (**a**) Sagittal View (**b**) Coronal View, (**c**) Axial View

Right Knee MRI (Fig. 10.14)

Right knee MRI results: Tricompartmental osteoarthrosis, most pronounced in the medial and lateral joint compartments. Small joint effusion with synovitis. Degenerative tearing of the medial and lateral menisci. Chronic complete rupture of the ACL. Partial tear of the MCL. Degenerative change of the LCL. Small Baker's cyst.

Along with the patient's severe osteoarthritis, she has several degenerative changes (meniscal, PCL, MCL) including complete tears of her ACL bilaterally. ACL tears are commonly seen more in younger populations engaged in sports-related activity (deceleration, jumping, pivots, direct trauma), but many other mechanisms can cause ACL damage leading to an eventual tear [51]. Considering the patient's advanced age, her ACL tears are most likely due to non-contact injuries throughout her lifetime that were not intervened on. These chronic ligament tears have contributed to this patient's advanced osteoarthritis, leading to chronic joint effusion and recurrent Baker's cysts.

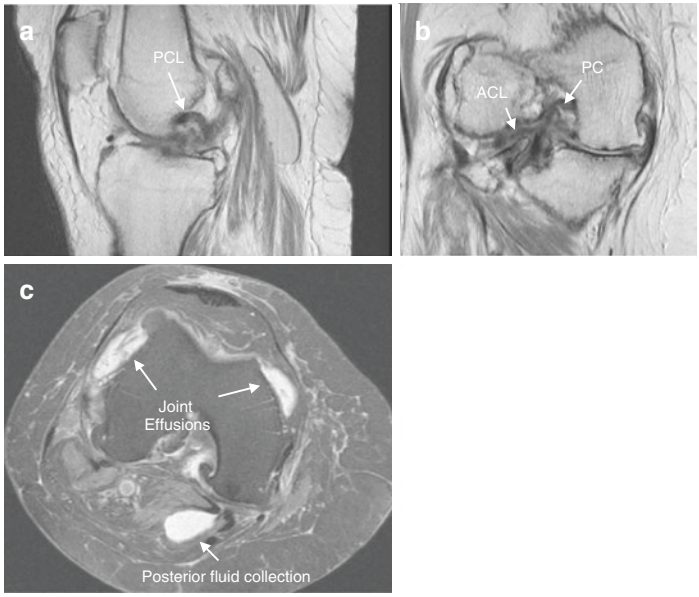

Fig. 10.14 (**a**) Sagittal View (**b**) Coronal View, (**c**) Axial View

Diagnostic Discussion

Baker's cysts typically develop due to a communication between the cyst and joint space, with sequestration of fluid caused by repeated knee flexion/extension. These cysts are typically found between the semimembranosus and medial gastrocnemius muscles. Popliteal cysts are relatively common even in asymptomatic individuals—anywhere from 4.7% to 37% depending on the population. They are often found in association with other intra-articular or inflammatory conditions such as osteoarthritis, rheumatoid arthritis, and meniscal or ligament tears [52]. For patients where the diagnosis is uncertain on physical exam and history, ultrasound and plain radiographs are recommended. While ultrasound can identify cysts and adjacent effusions, radiographs can help identify other pathologies associated with cyst formation and maintenance. MRI is often used to evaluate soft tissues structures and areas of fluid collection (Fig. 10.15).

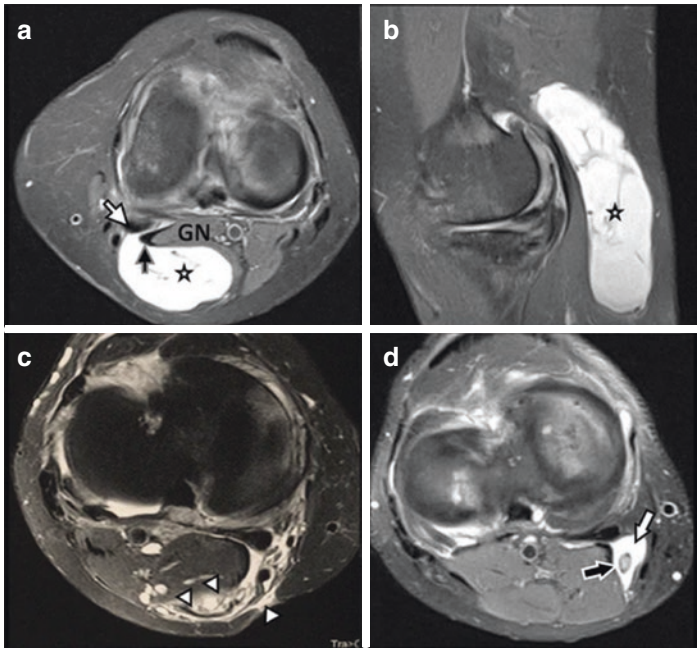

Fig. 10.15 Popliteal cysts. The axial (**a**) and sagittal (**b**) fat saturated proton density weighted images show a large multiseptated popliteal cyst (asterisks) emerging between the medial gastrocnemius tendon (black arrow) and the semimembranosus tendon (white arrow) and abutting the medial gastrocnemius muscle belly (GN). The axial (**c**) fat saturated proton density weighted image shows a ruptured popliteal cyst (arrowheads). The axial (**d**) fat saturated proton density weighted image demonstrates a Baker's cyst (white arrow) with a single loose osteocartilaginous body inside the cyst (black arrow) [53]

In this older patient with chronic bilateral knee pain, recurrent joint effusion and Baker's cysts, and a history of femoral neck fracture, osteoarthritis should be highly suspected as a primary cause for this patient's recurrent cysts. However, it is important to rule out other reversible causes. This patient's positive findings on anterior drawer test bilaterally pointed towards ACL pathology and warranted further imaging.

Other diagnoses to consider include popliteal or biceps femoris tendinopathy, which are localized to the posterior knee due to their attachment sites, though less likely in this patient who has not endorsed any aggravating activities. And in a patient with several cardiovascular comorbidities (coronary artery disease, hypertension, prior stent placement), vascular pathologies such as a popliteal artery aneurysm should also be evaluated for.

Management

This patient has severe osteoarthritis with bilateral, chronic ACL tears presenting with recurrent Baker's cysts. Baker's cysts are typically managed by identifying a source of cyst formation as well as treating the cyst if symptomatic. In this case, the patient's bilateral ACL tears, ligament degeneration, meniscal degeneration, and severe osteoarthritis are all potential contributors.

ACL tears can be managed conservatively or surgically depending on a patient's activity level, extent of injury, and the presence of arthritic changes. ACL reconstruction in active, older patients (ages 40+ and 60+) without arthritis have been shown to restore function and knee stability with similar complication rates compared to younger patients [54]. While this patient ultimately deferred surgical treatment, her eligibility would depend on the surgeon's judgment regarding expected return of function and pain relief in the setting of existing severe osteoarthritis and ligament/meniscal degeneration. Non-operative management includes rehabilitation and bracing that helps control knee range of motion (such as a hinge knee brace) to avoid further joint instability.

Management of symptomatic Baker's cysts include knee joint arthrocentesis and intra-articular injection with glucocorticoids for symptom relief and to reduce the rate of recurrence. Direct aspiration of the cyst can also be attempted with ultrasound guidance. The same management can be followed for ruptured cysts [55]. For cysts refractory to repeat aspirations and injections, further imaging and evaluation should be performed to evaluate reversible causes as mentioned prior, with few patients requiring surgical excision.

Areas of Research

While the frequency of surgical intervention remains low, more options have been explored for patients with refractory cysts. For patients with or without one-way valve lesions contributing to cyst maintenance, arthroscopic cystectomy has been shown to provide good outcomes in reducing pain and recurrence [56]. New techniques such as arthroscopic communication enlargement (valvular opening between the semimembranosus and medial gastrocnemius) may produce better outcomes in future studies [57].

Key Takeaways

1. Popliteal (Baker's) cysts are often asymptomatic and are commonly found incidentally on imaging. Patients with symptomatic cysts typically present with posterior knee pain and palpable cyst in the popliteal fossa made more detectable on knee extension.
2. Osteoarthritis, rheumatoid arthritis, and meniscal/ligamentous pathology can contribute to symptomatic Baker's cysts, which can be managed through cyst aspiration, steroid injection, and less commonly cystectomy in refractory cases.
3. ACL tears in older individuals are often managed conservatively with rehabilitation and bracing due to lower physical demands. ACL reconstruction is still a viable option for all active patients regardless of age, after assessing the extent of injury and severity of arthritis.

Knee Trauma After Motor Vehicle Collision

History

A 35-year-old female with no significant past medical history, presents with right knee pain and weakness after a motor vehicle collision. She was riding a moped going 5–10 mph, hit an open car door, and fell onto her right lower extremity. She had immediate pain localized to the right knee. She attempted to ambulate after the fall but was unable to bear weight due to weakness in the right knee. She denies numbness or tingling in the right lower extremity. She denies head injury or loss of consciousness.

Review of systems:
No fever, chills, cough, chest pain, SOB, paresthesia, dizziness, loss of consciousness, or lightheadedness
Past medical Hx/surgeries:
Abnormal pap smear
Gonorrhea
Ovarian cyst (ruptured)
Anemia
No past surgical history
Medications:
None
Allergies:
Sulfa
Family history:
No relevant family history
Social history:
Patient is a social worker.
Denies smoking or drug use.
Drinks alcohol socially

Physical exam findings:
Constitutional: Alert. Oriented to person, place, and time. No distress
HEENT:
Head: Normocephalic and atraumatic
Cardiovascular: Normal rate. 2+ dorsalis pedis pulses bilaterally
Pulmonary/chest: Effort normal and breath sounds normal. No respiratory distress
Skin: Skin is warm and dry. Non-diaphoretic
Musculoskeletal exam:
Inspection:
Right lower extremity: Mild effusion superior and medial to patella. No visible ecchymosis or bony deformities. Hip, calf, and ankle without abnormalities
Left lower extremity: No abnormalities noted
Bilateral upper extremities without abnormalities
Palpation:
Right lower extremity: Limited right knee passive ROM 3–80 degrees due to pain. Mild right knee medial joint line tenderness. Right hip, calf and ankle non-tender to palpation
Left lower extremity: Non-tender to palpation
Bilateral upper extremities non-tender to palpation
Sensation:
Bilateral upper and lower extremities neurovascularly intact
Strength:
Right lower extremity: Unable to assess due to pain
Full strength of the left lower extremity and bilateral upper extremities
Gait:
Unable to bear weight on the right lower extremity due to weakness secondary to pain
Special exam maneuvers:
Right lower extremity:
Straight leg raise with minimal lag
Grade 2 posterior drawer
Soft endpoint with Lachman
Mild laxity with valgus stress
Negative McMurray
Negative dial test
Left lower extremity: Negative

Differential Dx (Table 10.7)

Work Up: Imaging

See Fig. 10.16.

X-rays of the right knee were obtained (Fig. 10.16) which showed:

Fracture fragments detected at the lateral femoral condyle and posterior to the fibular head, highly suspicious for comminuted traumatic fractures of the lateral femoral condyle and/or lateral tibial plateau. No significant suprapatellar effusion.

Table 10.7 Differential diagnosis and associated key findings

Differential	Key findings
Tibial plateau fracture	High energy trauma, most often motor vehicle collisions, also seen in sports injuries. Acute pain, swelling. Ligamentous and meniscal injuries often accompany due to mechanism of injury
Fibular head or neck fracture (Maisonneuve fracture)	High energy trauma with direct blow to fibula; also seen with severe medial ankle sprains or medial malleolar fractures; lateral knee pain that may present with or without swelling; focal bony tenderness at fibular head or neck
Knee (tibiofemoral) dislocation	High energy trauma with direct blow to anterior knee causing hyperextension; often involves multiple knee ligaments, and can compromise vascular supply to lower leg; trauma often related to motor vehicle collisions and sports; swelling, pain, and instability
Bone contusion	Commonly associated with PCL injuries, in which there is direct knee trauma to tibia and hyperextension. Can cause significant pain, disability, and restricted ROM. Negative posterior drawer test when an isolated injury
Patellar fracture	Caused by direct trauma to the anterior knee; presents with pain, swelling and ecchymosis localized to directly over patella; PCL tears often accompany due to mechanism of injury

Table 10.7 (continued)

Differential	Key findings
Patellar dislocation	Usually caused by twisting force to planted, flexed knee. Presents as acute swelling, tenderness to palpation on medial edge of patella. May be audible "pop" or tear at time of dislocation. Often instability with weight bearing followed by severe pain. Patella typically palpable laterally if not reduced
Anterior cruciate ligament (ACL) tear	Sudden change in direction or landing from a jump (usually audible "pop" at time of injury), positive Lachman and/or anterior drawer test, history of direct trauma to knee, rapid swelling, instability with weight bearing, decreased ROM
Posterior cruciate ligament (PCL) tear	History of direct trauma to knee (usually direct blow to proximal anterior tibia), positive posterior drawer test, posterior knee pain, instability with weight bearing, knee may feel like it hyperextends
Medial/lateral collateral ligament strain	MCL: Twisting of leg or direct blow to medial knee, Medial knee pain; medial knee feels unstable with lateral movements; focal tenderness over MCL; positive valgus stress test; often accompanied by medial meniscus tear LCL: Twisting of leg or direct blow to lateral knee, lateral knee pain; lateral knee feels unstable with lateral movements; focal tenderness over LCL; positive varus stress test; often accompanied by ACL and PCL injury due to greater required force
Meniscus tear	Sudden, forceful twisting of knee with foot planted, tearing/popping sensation, pain/swelling, effusion, joint line tenderness; positive McMurray and/or Thessaly test

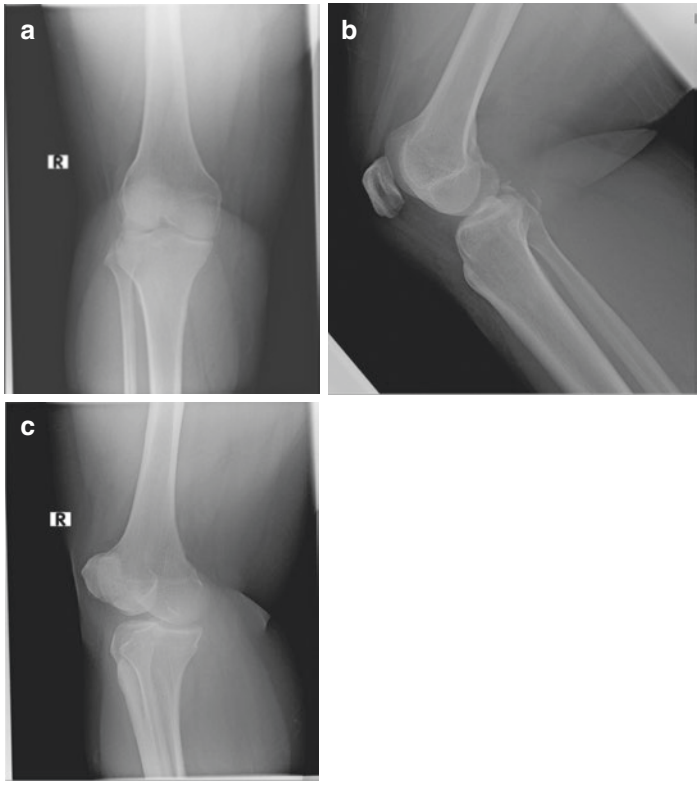

Fig. 10.16 (**a**) AP View, (**b**) Lateral View, (**c**) Oblique External View

CT right knee w/o contrast was obtained which showed (Fig. 10.17):

Comminuted traumatic fracture of the posterior mid and medial tibial plateau. Lateral subluxation of the patella.

MRI Right Knee was obtained which showed (Fig. 10.18):

1. Comminuted posterior tibial plateau fracture.
2. Large osteochondral defect weightbearing lateral femoral condyle as above.

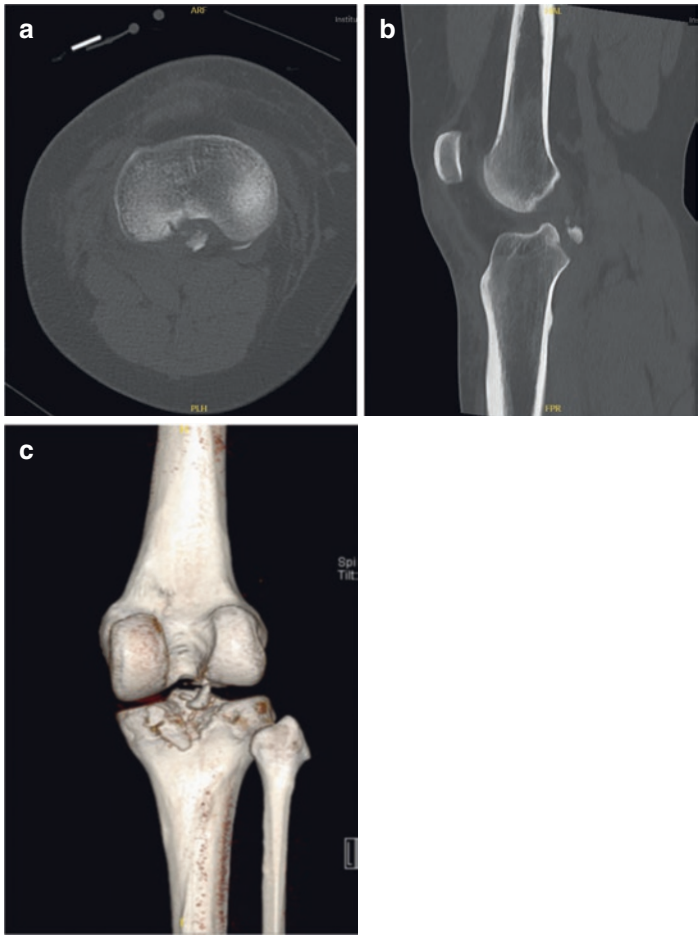

Fig. 10.17 (**a**) Axial View, (**b**) Sagittal View, (**c**) 3D Spin View

3. Complete avulsion posterior root lateral meniscus. Avulsion of posterior meniscocapsular attachments.
4. Complete tear ACL and distal PCL.
5. Complete tear superficial and deep MCL.

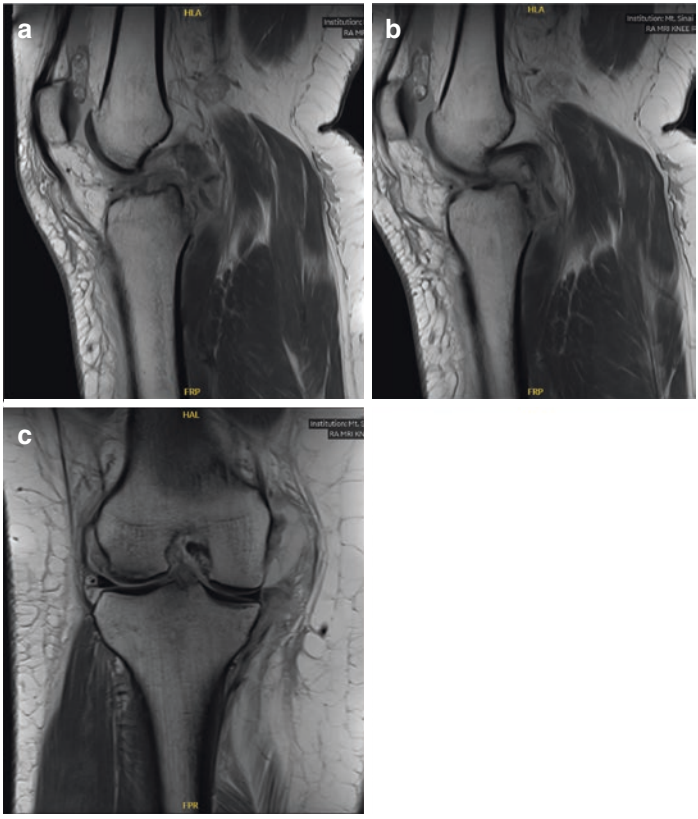

Fig. 10.18 (**a**) Sagittal view, notable for ACL tear, (**b**) Sagittal view, notable for PCL tear, (**c**) Coronal View

6. High-grade sprain proximal LCL with associated lateral femoral epicondyle avulsion fracture.
7. Mild popliteus muscle strain.
8. Large joint effusion containing fracture fragments/loose bodies posteriorly.

Diagnostic Discussion

The proximal portion of the tibia, also known as the tibial plateau, is the most common site of tibial fractures compared to the distal and midshaft tibia [58]. Tibial plateau fractures account for about 1% of all fractures and occur most often in situations that involve significant direct trauma to the knee, such as motor vehicle collisions, vehicle-pedestrian collisions, and contact sport injuries [59]. The lateral tibial plateau is affected in 60% of fractures, likely because most injuries involve a lateral to medial force. Medial tibial plateau fractures occur 15% of the time, while bicondylar lesions occur in 25% of cases. Injury often presents on exam as swelling and tenderness over the proximal tibia, as well as skin abrasions and/or contusions. Complications such as compartment syndrome, peroneal nerve dysfunction, and ligamentous or meniscal instability may also be seen [60].

Ligamentous and meniscal injuries often occur with tibial plateau fractures, likely because the proximal tibia forms the lower surface of the knee joint and serves as an attachment site. A recent study which evaluated MR scans of 29 patients with acute tibial plateau fractures, showed that tibial (medial) collateral ligament tears (55%) and lateral meniscal tears (45%) were the most common. 41% of patients had anterior cruciate ligament injuries, compared to 28% of posterior cruciate ligaments. Furthermore, medial meniscal tears were noted in 21% of cases, while lateral collateral ligament tears were seen 34% of the time [61].

The Schatzker classification system divides tibial plateau fractures into six types: lateral plateau fracture without depression (type I), lateral plateau fracture with depression (type II), lateral plateau fracture with central depression (type III), medial plateau fracture (type IV), bicondylar plateau fracture (type V), and plateau and proximal shaft fracture (type VI). CT and MRI are the most accurate method for Schatzker classification of tibial plateau fractures (Fig. 10.19) [62–64].

AP and lateral radiographs are usually sufficient for the diagnosis of proximal tibial fractures. CT scans can better define the fracture when radiographs are unremarkable but clinical suspi-

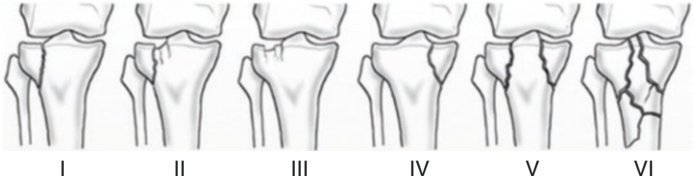

Fig. 10.19 Schatzker classification system of tibial plateau fractures [62]

cion is high, while MRI is most useful for displaying meniscal or ligamentous injuries [61]. Ultrasound may be useful for showing knee effusions or soft tissue injury [65].

Management

Initial management of tibial plateau fractures involves compression, icing, splinting of knee in extension, elevation, non-weight bearing, and analgesics. If there is an uncomplicated fracture without displacement, or ligamentous/meniscal injury, management may be non-operative. This usually includes weekly radiographs to monitor healing, non-weight-bearing for about 6–8 weeks and knee immobilizer for 8–12 weeks until radiographs show complete healing. Patients usually regain full function of the lower extremity after 16–20 weeks with physical therapy [66].

Orthopedic referral is indicated within 48 h in the setting of open or displaced tibial plateau fractures, signs of vascular compromise, acute compartment syndrome, or involvement of ligamentous or meniscal injury. Most of these fractures will require surgical intervention with open reduction and external fixation (ORIF) [66]. Total knee arthroplasty may be indicated in older patients with significant osteoporosis or osteoarthritis and younger patients with severe tibial plateau destruction [67]. Return to full sporting activity is usually attainable in about 6 months, if indicated [68].

Studies have shown that severe open fractures of the tibia often require amputation due to considerable soft tissue injuries; how-

ever, proximal tibia fractures comprised the lowest percentage of amputation (19.3%) compared to midshaft tibial (46.8%) and distal tibial (34.0%) fractures [69].

Outcomes and Complications

Overall, the outcome following treatment of tibial plateau fractures is good. However, because tibial plateau fractures often occur with high impact force such as motor vehicle collisions, the patient usually sustains other severe injuries or complications. These include acute compartment syndrome (ACS), infection, nonunion, malunion, and this may cause a delay in their return of daily function and cause long-term morbidity [66].

Gait abnormality is also a significant long-term complication that has been observed following tibial plateau fractures. In one study, 22 patients were evaluated 3 years after high energy tibial plateau fractures. Findings showed these patients walked slower by 18%, and had a shorter step length by 11% in the affected leg and shorter step length by 12% in the unaffected leg. This study also assessed for quality of life in patients with tibial plateau fractures, which showed a Physical Health Score 65% lower compared to healthy controls and Mental Health Score 40% lower compared to healthy controls [70].

Another study analyzed the long-term functional results following surgical repair of tibial plateau fractures using ORIF. It was found that the most significant factor for variation in functional outcome was age, where patients under 40 had a high rate of functional outcome (>92%) regardless of fracture type. Patients over 40 showed a much poorer functional outcome, and still experienced lower extremity pain, stiffness, and decreased physical function compared to the younger age group [71]. Lastly, studies have shown that patients with tibial plateau fractures that required surgery had a 5.3× increase in the likelihood of also needing a total knee arthroplasty after 10 years [72].

Patient Conclusion

The patient above underwent surgical repair of the ACL, PCL, medial and lateral meniscus, and medial collateral ligament. Post-op instructions included instructions for home exercises, and a physical therapy referral.

Key Takeaways

1. Tibial plateau fractures occur most often after significant direct trauma to the knee, such as motor vehicle collisions, vehicle-pedestrian collisions, and contact sport injuries.
2. Ligamentous and meniscal injuries are often associated with tibial plateau fractures. Therefore, physical exam and imaging should rule out these potential pathologies when a patient presents with a tibial plateau fracture.
3. Orthopedic referral is indicated within 48 h in the setting of open or displaced tibial plateau fractures, signs of vascular compromise or acute compartment syndrome, or involvement of ligamentous or meniscal injury. Most of these fractures will require surgical intervention with open reduction and external fixation (ORIF).
4. For uncomplicated fractures, initial management involves compression, icing, splinting of knee in extension, elevation, and non-weight bearing, and analgesics. Regaining full function of the lower extremity usually takes 16–20 weeks with physical therapy and return to full sporting activity usually takes about 6 months.
5. While the outcome following treatment of tibial plateau fractures is typically good, there are often other severe injuries or complications that may cause a delay in a patient's return of daily function and cause long-term morbidity.

Knee Trauma from a Ski Crash

History

A 14-year-old female presented to the urgent care 2 days prior after crashing while competing at a ski race. She could not describe moments leading up to the event but endorsed acute swelling of the right knee and was unable to bear weight immediately. At urgent care, she was diagnosed with a Salter 1 distal femur fracture of the right leg and was placed in a knee immobilizer. Patient would like to return to skiing and playing soccer.

Review of systems: No fever, chills, cough, paresthesia, weakness, bowel/bladder changes or falls. *Past medical Hx/surgeries:* None *Medications:* None *Family history:* None *Social history:* Eighth grade in middle school Lives at home with her mom, dad, and sister Plays soccer and participates in ski races	*Initial physical exam findings:* **Evaluation while on ski hill** **Gen:** Not in acute distress, well developed *Right knee:* **Inspection:** Mild swelling of the right knee with associated erythema **Palpation:** Tenderness to the right distal lateral femur and lateral aspect of proximal tibia No tenderness to palpation of the patellar tendon, quadricep tendon, medial joint line, popliteal fossa, or hamstring tendon *Gait*: Unable to bear body weight on right leg Antalgic gait while using crutches

Follow-Up Physical Exam Findings

Evaluation at outpatient clinic.

BMI: 19.5.

Height: 5′ 3″.

Right Knee

Inspection:

Mild swelling of the right knee.
Patella in appropriate position.

Movement:

Right knee flexion and extension limited to 30* due to pain.
Able to do straight leg raise with both legs.

Strength: 5/5 in bilateral hips and dorsi & plantar flexors.

Sensation: Grossly intact to light touch.

Special Exam Maneuvers

Positive Lachman test.

Negative posterior drawer test.

Negative valgus laxity or stress test.

Negative varus laxity or stress test.

Unable to perform McMurray due to pain.

Differential Dx (Table 10.8)

Diagnostic Discussion

Acute, traumatic injuries to the knee can cause multiple pathologies (fractures, ligament tears, dislocations, and subluxations) necessitating a thorough workup to dictate proper management. While imaging such as MRI is often needed, the patient's initial history and physical exam findings are useful in guiding treatment following initial follow-up. In this patient with a knee effusion after collision during a ski race, an isolated femur fracture would

Table 10.8 Differential diagnosis and associated key findings

Differential	Key findings
Anterior cruciate ligament tear Posterior cruciate ligament tear	Associated with a popping sensation localized to the knee at time of injury followed by acute swelling and joint instability **ACL:** Commonly caused by a direct lateral impact to the knee, or deceleration of the knee causing valgus stress. Positive on anterior drawer test and Lachman's test **PCL:** Can be caused by a direct impact to the proximal anterior tibia but often presents as subacute/chronic symptoms not associated with trauma. Positive on posterior drawer test
Medial collateral ligament tear Lateral collateral ligament tear	**MCL:** Can be caused by direct (blow to lateral aspect) or indirect valgus stress (abduction/rotation) to the knee. Positive on valgus stress test **LCL:** Least commonly injured. Can be caused by direct (blow to medial aspect) or indirect varus stress to the knee. Positive varus stress test
Meniscal tear	Caused by forceful twisting of the knee; catching sensation, knee swelling, joint line tenderness, positive McMurray and Thessaly tests.
Patellar dislocation	Associated with a popping sensation at the knee followed by acute swelling and joint instability. Lateral displacement of the patella is most common, palpable on physical exam with knee flexed to 30 degrees
Quadriceps or patellar tendon tear	Can also be associated with a popping sensation and acute swelling with sharp pain at the knee. Focal tenderness at the anterior knee just above the patella (quadriceps tendon tear) or below the patella (patellar tendon tear). Patients cannot maintain a straight leg or extend their knees
Intra-articular fracture	Often caused by valgus stress on the knee, associated with rapid swelling and pain, lateral joint line tenderness, and instability

unlikely be the only cause for her pain and lack of weight bearing. In addition to her inability to bear weight due to the femur fracture, a knee ligament sprain or tear should be highly suspected: a positive Lachman's test indicates ACL pathology that warrants further imaging. Other injuries such as a concomitant ligament tear, meniscal injury, or intraarticular fracture should also be evaluated for as they often occur concurrently with an ACL tear [73, 74].

Patellar dislocation or subluxation is less likely in this patient, as the patella is appropriately positioned, and the patient does not endorse localized pain to palpation on manipulation. While limited by pain, the patient is able to flex and extend their knee to 30 degrees, making a quadriceps or patellar tendon tear less likely.

Work Up: Imaging

Right Knee Radiograph from Urgent Care (Fig. 10.20)

Right Knee MRI (Fig. 10.21)

Management

In this adolescent with an ACL tear seen on MRI, the acute management involves pain control, reducing inflammation, and achieving full range of motion. A hinged knee brace and crutches are also indicated if there is joint instability, evidenced by excessive range of motion beyond a joint's intended movement. Deciding on an operative versus non-operative approach depends on several key factors, the following of which would lean towards operative:

1. Patient desires to resume athletic, high impact activity.
2. Presence of other concomitant injuries: meniscal tears, collateral ligament injuries.
3. Presence of severe joint instability.

In a young adolescent who is interested in returning to athletic activity, an operative approach such as ACL reconstruction is considered standard of care. Preparation for surgery depends primarily on the condition of the injured knee, which should exhibit absence of effusion, adequate strength, and full range of motion [75]. In this patient with only a slight effusion and full strength and range of motion on subsequent follow-up, ACL reconstruc-

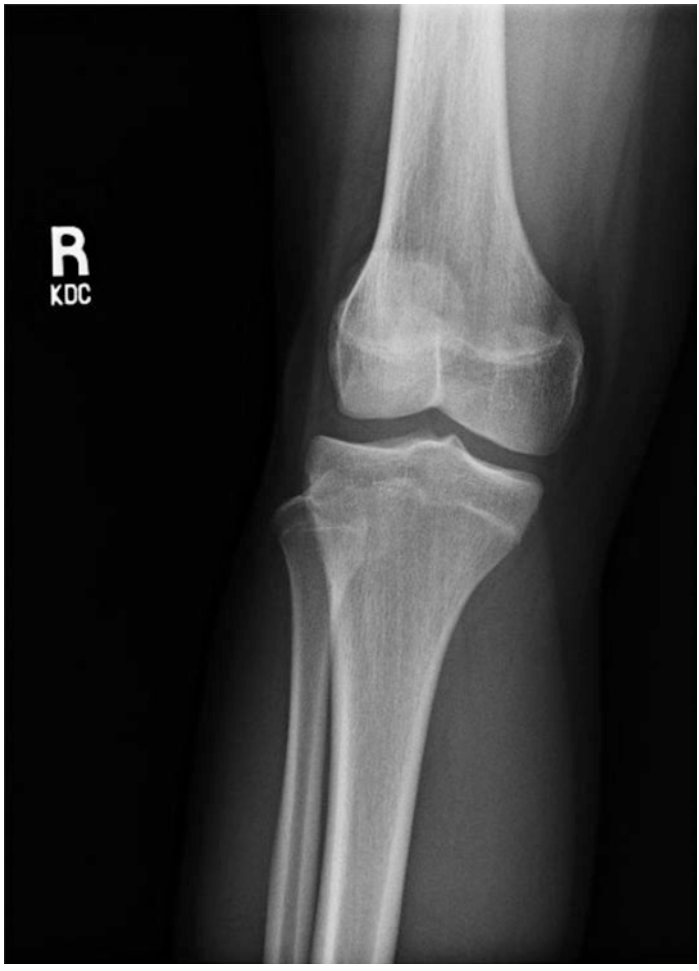

Fig. 10.20 Right knee X-ray: No definitive fracture seen. Growth plates visible but nearly closed

tion was appropriately planned for 1–2 weeks after the incident using a quadricep tendon autograft.

For other patients who may require longer periods of preparation, these considerations should be tempered by the conse-

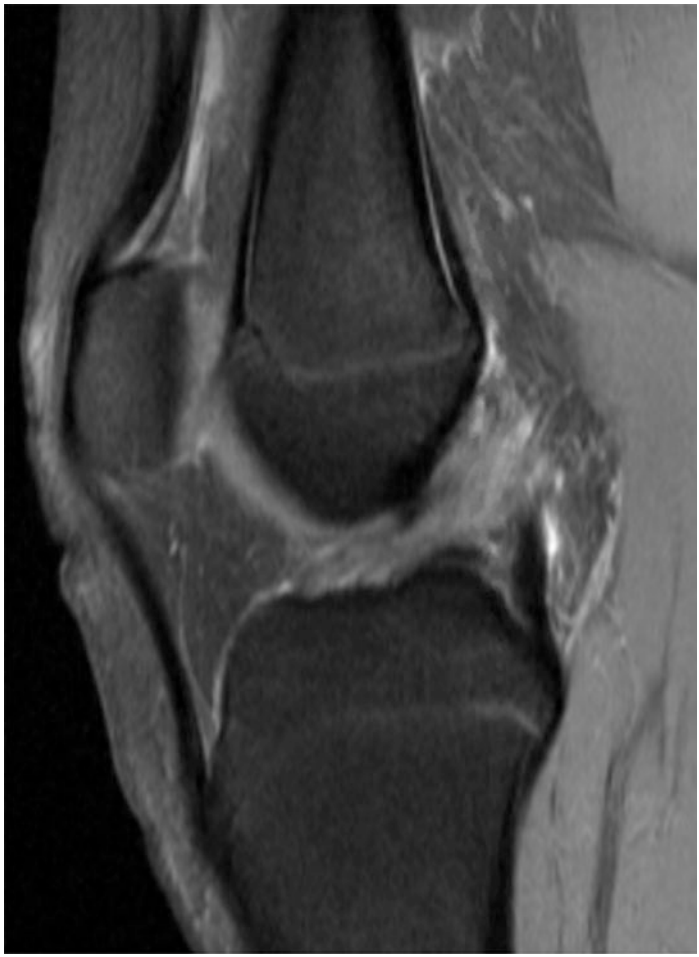

Fig. 10.21 Right knee MRI: Grade 3 full thickness tear of the junction of the proximal and middle thirds of the ACL. The distal ACL exhibits an atypically horizontal spatial orientation. Secondary findings suggestive of ACL instability

quences of extended delays in surgery. Studies have shown that delaying ACL reconstruction in pediatric or adolescent patients for greater than 12 weeks significantly increased the risk of meniscal injuries, and nonoperative management leads to higher rates of residual knee instability, increased risk of meniscal tears, and comparatively lower rates of return to sports. Both early and delayed operative treatment have been shown to achieve satisfactory knee stability [76]. Therefore, young active patients who elect for nonoperative care may still undergo delayed reconstruction within 3 months and have similar outcomes.

Consideration 1: Pediatric Vs. Adult Patients

In comparison to pediatric patients, adult patients have been shown to have more flexibility regarding timing of ACL reconstruction. Active adults who delayed ligament reconstruction for 6 months showed similar functional outcomes at 5 year (compared to adults who had early reconstruction and rehab) [77]. For patients who are not participating in athletic activity or are overall less active (such as geriatric patients), non-operative management (strength training, activity modification, stability training) can be a reasonable alternative.

Following ACL surgery, a personalized rehabilitation program is essential in improving treatment results and return-to-play. While the phases of rehabilitation may vary, all should incorporate the following: pain control, early weight bearing and range of motion exercises, reducing joint effusion, and increasing quadriceps muscle activation [75]. Closed kinetic chain exercises (squats, lunges) are initially emphasized to strengthen the quadriceps and hamstring muscles while maintaining joint stability, followed by open kinetic chain exercises [78].

The large majority of young individuals who undergo ACL reconstruction are able to return to sports: 81–92% return to any sport, 65–79% return to their preinjury level, and 55–81% return to competitive sports [75, 79]. A patient's return-to-play timeline can vary widely depending on the timing and type of surgery, patient symptoms, functional testing, properly structured rehabilitation, and psychologic readiness. Across this heterogeneity, bet-

ter outcomes typically occur after 9 months post-reconstruction [75]. Patients who return to sports prematurely significantly increase their risk for reinjury and graft failure [79], as well as increased risk for osteoarthritis [80]. Therefore, return to full activity sports should depend on return of normal strength, ability to achieve full range of motion, adequate performance on functional tests (strength tests, hop tests, movement quality) and sports specific movements [81].

Areas of Research

- While ACL reconstruction has been considered the gold standard for operative intervention, more surgeons are revisiting primary ACL repair due to improvements in imaging, arthroscopic modalities, and techniques that augment the mechanical/biological environment to allow for primary healing [82].

Key Takeaways

1. High impact trauma to the knee (as seen in athletic activity, sports, and motor vehicle accidents) can often lead to several concomitant injuries, requiring a thorough history, physical exam and follow-up imaging. Injuries may include ligament tears, tendon tears, meniscal tears, fractures, and dislocations.
2. ACL tears are often accompanied by a popping sensation, acute pain, and joint swelling. Tears are best visualized on MRI, along with any other concomitant ligament or tendon injuries.
3. Patients have the option of choosing either operative or non-operative intervention, depending on their desired activity level, presence of other injuries, and extent of joint instability. Most young athletes will elect to undergo ACL reconstruction, which has been shown to decrease residual knee instability, decrease risk of meniscal tears, and lead to higher rates of return to sports compared to physical rehabilitation only.
4. The success of ACL reconstruction depends heavily on a personalized rehabilitation regimen post-procedure, which should include pain control, early weight bearing and range of motion

exercises, reducing joint effusion, and increased quadriceps muscle activation. The large majority of athletes will be able to return to their desired sport, with large heterogeneity between individuals regarding length of rehab.

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Index

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