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REVIEW ARTICLE

MRI of anterior knee pain

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Abstract Anterior knee pain is the most common knee complaint. It may be due to a variety of soft tissue or osseous abnormalities. Knowledge of the radiologic appearance of the abnormalities allows more accurate diagnosis of the cause of the pain including chondral abnormalities, patellar instability and dislocation, femoral trochlear dysplasia, abnormal patellar location, bipartite patella, various tendinopathies, bursal inflammation, traction apophysitis in pediatric and adolescent patients, and miscellaneous diseases including mediopatellar plica syndrome and Hoffa's disease. Radiographs are often obtained to exclude acute osseous abnormalities, such as fractures. Magnetic resonance (MR) imaging offers superior soft tissue contrast resolution and allows for more accurate evaluation of the underlying etiology and therefore may improve treatment and possible surgical planning.

Keywords Anterior knee · Extensor mechanism · Pain · MRI

Introduction

Anterior knee pain (AKP) is the most common knee complaint, usually occurring in adolescents and young adults [1]. It is more common in athletic individuals, with the incidence rate as high as 9 % in young active adults and comprises up to a quarter of all knee problems treated at sports injury clinics [2, 3]. AKP may cause chronic disability, limited sports participation, and may affect quality of life. Despite its prevalence, AKP remains poorly understood, as it has not been well studied in the literature, making its treatment one of the most complex among the various pathologies affecting the knee [4].

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Explanations for the poor understanding suggested by the International Patellofemoral Study Group include the complex biomechanics of the patellofemoral joint, less clinical interest in patellofemoral compartment abnormalities, various causes for AKP, and the lack of correlation between symptoms and physical or radiologic findings [5].

AKP is a nonspecific symptom that can result from various etiologies, including chondromalacia patella, patellar instability and dislocation, trochlear dysplasia, patella alta or baja, patellar tendinitis, bipartite patella, anterior knee bursitis, quadriceps or patellar tendon tear, plica syndromes, Hoffa's disease, and traction apophysitis syndromes. Familiarity with diseases of the anterior knee is important in understanding the source of symptoms.

This article reviews several of the common causes of AKP, with emphasis on the clinical, radiographic, and MR imaging findings with the goal of allowing for a more accurate diagnostic understanding and improved treatment of this common complaint.

Discussion

Chondromalacia patella (CP)

Chondromalacia patella is a term applied to cartilage loss involving one or more portions of the patella that leads to patellofemoral pain. It was first described in 1908 by Budinger, who drew attention to the importance of cartilage lesions on the undersurface of the patella, which are characterized by fissures and softening [6]. Also known as "runner's knee", CP is more common in adolescents and young adults. The term chondromalacia is used by arthroscopists probing cartilage and finding regions of slight surface depression and/or surface fraying [7]. The possible relationship of CP and osteoarthritis of the patellofemoral compartment has not been clearly defined, with the later more common in the elderly population.

CP of the lateral facet appears to be secondary to prolonged patellar tilt and compression with eventual arthrosis, while CP of the medial facet may be secondary to deficient contact or a combination of compression and shearing forces, which occurs more commonly at the time of patellar dislocation and relocation [8].

Closed CP, which consists of simple softening of the cartilage with an intact surface, is common and may not be symptomatic. Once the surface layer has been interrupted, it is termed open CP. Importantly, the cartilage lesion in open chondromalacia would appear to be irreversible, as surface lesions do not heal [9].

Symptoms and findings in patients with CP are similar to those of other patellofemoral pain problems; namely, AKP worsened by climbing or descending stairs [10].

The radiograph may be normal or may demonstrate signs of articular cartilage loss, patellar malalignment, subchondral sclerosis, or other evidence of arthrosis. The extent of articular cartilage loss can be best determined using MRI. CP can be divided into four grades using the Outerbridge grading system (Table 1), which was originally devised for arthroscopy but has become the foundation of modified MRI grading [11, 12] (Figs. 1, 2, 3, 4).

Reports indicate that the overall MRI sensitivity for detection of chondral lesions of the patellae ranges between 30 and 100 %, with a specificity between 50 and 94 % and a diagnostic accuracy between 77 and 90 % [13, 14]. In a study by Mattila et al., MRI scans detected grade I articular cartilage lesions of the patella with a sensitivity of only 20 % [15]. However, sensitivity was significantly higher for higher-grade lesions with 72 % sensitivity for grade 2 lesions and 100 % sensitivity for grade 3 and 4 lesions. The study by Mattila et al. showed that the grading of articular cartilage lesions of the patella based on arthroscopy and MRI poorly correlated with the clinical symptoms of AKP or the duration of the symptoms before arthroscopy. In a different study, only 60 % of the patients with clinically diagnosed anterior knee pain with retropatellar crepitus had articular cartilage lesions of the patellae [16].

Patellar instability and dislocation

Patellar instability is usually secondary to a morphologic abnormality in patellofemoral joint, which leads to recurrent patellar dislocations. Patellar dislocation occurs when the patella is completely displaced from the femoral trochlea so that there is no longer any articular surface apposition. It may occur as a result of either direct trauma or twisting injury in a patient with normal patellar alignment, or it may occur in the patient with a preexisting malalignment. Regardless of the mechanism of injury, acute articular cartilage injury is common [9]. When dislocations occur as a result of activity or direct trauma, patients may mistakenly think that the patella has dislocated or subluxed medially, when in fact they are feeling the medial femoral condyle that is normally medial relative to the patella.

The patella can be physiologically displaced slightly laterally; however, this tendency is normally balanced by two main stabilizer mechanisms: active stabilizers, which consist of the extensor muscles and orientation of the distal vastus medialis fibers, and passive stabilizers, comprised of osseous (i.e., buttress effect of the lateral femoral trochlea and deep femoral sulcus) and ligamentous structures (i.e., medial patellofemoral retinaculum and ligament) [9, 17].

Some predisposing bony abnormalities for patellar instability are trochlear dysplasia, trochlear facet asymmetry,

Table 1 Grading of chondromalacia patella

Chondromalacia patella grading, MRI, and arthroscopic appearance [11, 12]	
I	• MRI: Focal areas of hyperintensity with normal contour
	• Arthroscopically: softening or swelling of cartilage
II	• MRI: Blister-like swelling/fraying of articular cartilage extending to surface with less than 50 % thickness cartilage loss
	• Arthroscopically: fragmentation and fissuring within soft areas of articular cartilage
III	• MRI: More than 50 % thickness cartilage loss with focal ulceration
	• Arthroscopically: partial thickness cartilage loss with fibrillation
IV	• MRI: Full-thickness cartilage loss with underlying bone reactive changes
	• Arthroscopically: cartilage destruction with exposed subchondral bone

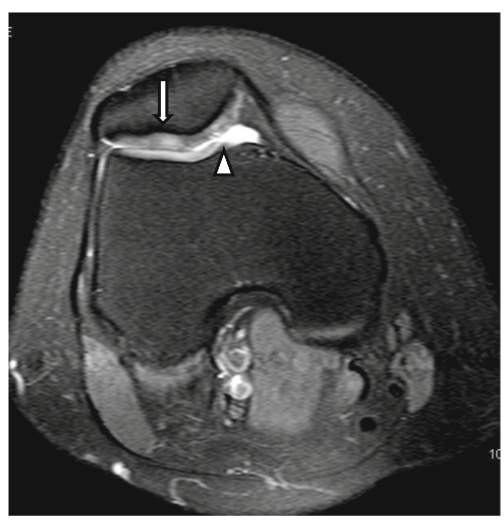

Fig. 1 Chondromalacia patella grade I (19-year-old patient with knee pain). Axial fat-suppressed proton density-weighted image (PDWI) demonstrating increased signal in patellofemoral cartilage of the lateral patellar facet (*arrow*). Additionally, there is cartilage fraying at the medial patellar facet (*arrowhead*)

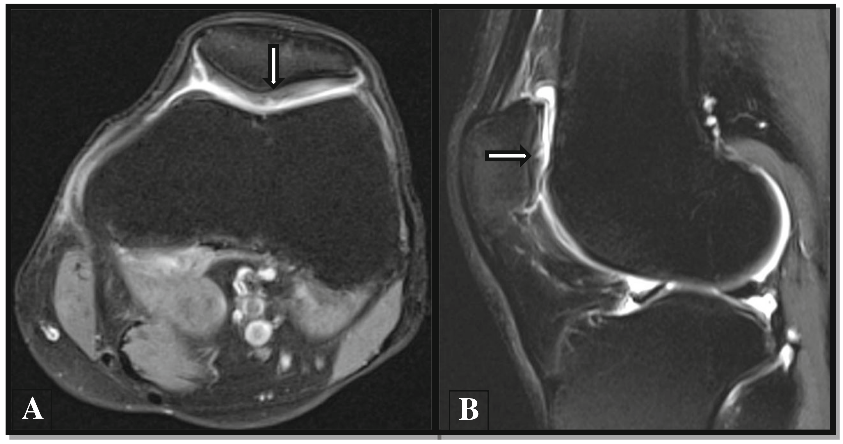

Fig. 2 Chondromalacia patella grade II (50-year-old patient with knee pain). **a** Axial fat-suppressed PD and **b** sagittal T2-weighted images demonstrating chondromalacia at the median ridge of the patella with fraying of articular cartilage extending to surface (*arrows*), with less than 50 % thickness articular cartilage involvement

lateralization of the tibial tuberosity, and femorotibial malrotation. Soft tissue abnormalities that may predispose individuals to patellar instability include patella alta, abnormal medial stabilizers, tight lateral retinaculum, and syndromes leading to generalized ligamentous laxity such as Ehler–Danlos, Marfan, and Down syndromes [9, 18].

Imaging is commonly obtained after the patella has relocated (Fig. 5). Therefore, the initial radiograph may be unremarkable, but the trauma of dislocation generally leaves some residual lateral placement of the patella, either as an indication of baseline malalignment or as a result of medial retinacular disruption. This is noted as lateral patellar subluxation or lateral patellar tilt on axial images. Subluxation is defined as partial lateral dislocation of the patella from the femoral groove and is usually evaluated subjectively. Patellar tilt is diagnosed by determining the lateral patellofemoral angle, which is calculated from lines drawn along the lateral joint surface patella and along the anterior aspect of the condyles [19]. A patellofemoral angle opening laterally is considered normal. An angle opening medially indicates patellar tilt.

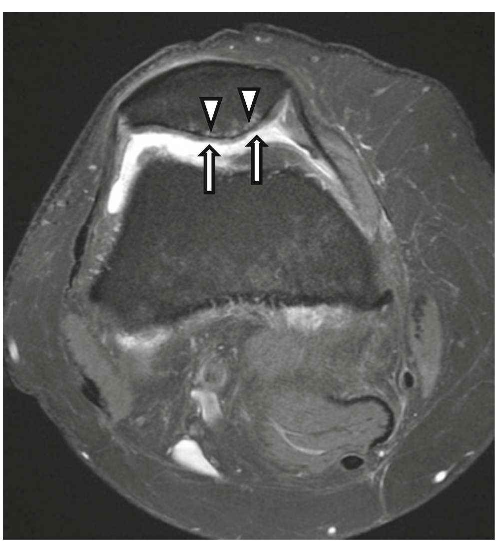

Fig. 3 Chondromalacia patella grade III. Axial fat-suppressed PDWI demonstrating greater than 50 % partial-thickness articular cartilage loss (*arrows*) and scattered foci of subchondral bone marrow edema (*arrowheads*)

As a result of the impaction forces, bone contusions along the medial facet of the patella and the lateral aspect of the lateral femoral condyle are common, with or without an impaction fracture line (Figs. 6, 7). The bone contusions may be associated with osteochondral injuries involving the articular cartilage with or without involvement of the subchondral bone plate [9, 20]. The osteochondral injury may be stable, unstable, or displaced; therefore a loose body is always looked for on MRI examinations after patellar dislocation. In addition to showing the bony and cartilaginous injuries, MRI would reveal the associated tearing of the medial patellar retinaculum (Figs. 5, 6), and commonly hemarthrosis [21, 22]. MRI can also show predisposing malalignment of the extensor mechanism as patella alta or a defect in medial border of patella (Fig. 8) [9, 23]. In cases of old patellar dislocation, X-rays may show soft tissue calcification in the traumatized medial patellofemoral ligament.

Femoral trochlear dysplasia

The femoral trochlear groove provides a mechanical restraint that helps to stabilize and serve as a guide for the patella as it articulates during joint flexion. The normal femoral trochlear shape has a deep sulcus with well-defined medial and lateral facets that are either equal in size or has a mildly larger lateral facet [24]. Trochlear dysplasia or a shallow trochlea is quantified as a maximum trochlear depth of 3 mm or less measured 3 cm above the femorotibial joint space [25, 26]. Trochlear dysplasia has been identified as one of the main factors contributing to chronic patellofemoral instability [27]. Dejour et al. proposed a classification distinguishing four morphologic types of trochlear dysplasia [28]. In type A, the normal

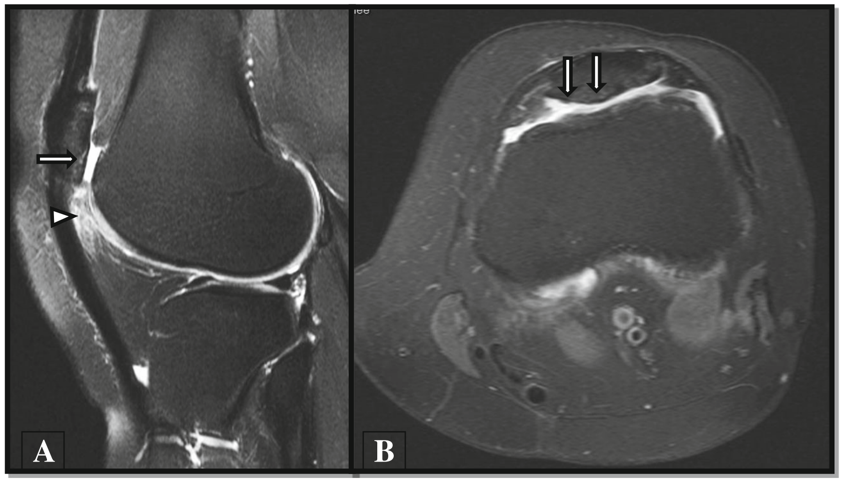

Fig. 4 Chondromalacia patella grade IV. **a** Sagittal and **b** axial fat-suppressed T2-weighted images demonstrating diffuse full-thickness articular cartilage loss associated with subtle subchondral bone marrow edema (*arrows*). There is patella alta and edema in superolateral aspect of Hoffa's fat pad (*arrowhead*), suggestive of patellar maltracking

shape of the trochlea is preserved but the trochlear groove is shallow (Fig. 9); in type B, the trochlea is markedly flattened or even convex with loss of the trochlear groove and facet anatomy resulting in a horizontally oriented flattened joint surface (Fig. 10); in type C, asymmetric trochlear facets are noted, with the lateral facet being too high and the medial facet being hypoplastic, resulting in an obliquely oriented flattened joint surface. In type D, in addition to the features of type C, a vertical link between medial and lateral facets leading to small bony bump is present on sagittal images.

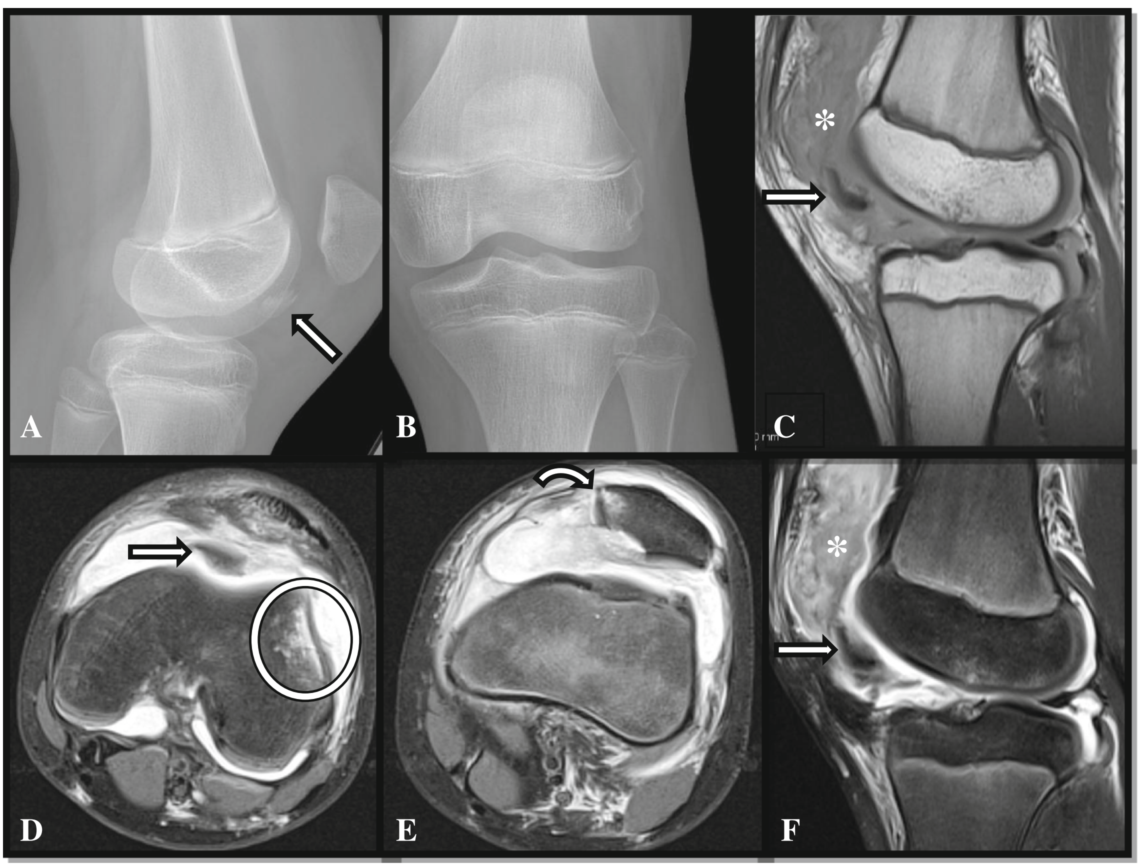

Fig. 5 Patellar dislocation (relocated) (15-year-old patient with recent acute patellar dislocation). **a** AP and **b** lateral radiographs show a large displaced bony fragment anterior to the trochlea (*arrow*). **d**, **e** Axial fat-suppressed PD, **f** sagittal fat-suppressed PD, and **c** sagittal T1-weighted images confirm the large, displaced, and rotated osteochondral body interposed between the patella and femoral trochlea (*arrow*). There is hemarthrosis (*asterisk*), avulsion of the MPFL from its patellar insertion (*curved arrow*), and bone contusions in the medial facet of the patella and lateral femoral condyle (*circle*)

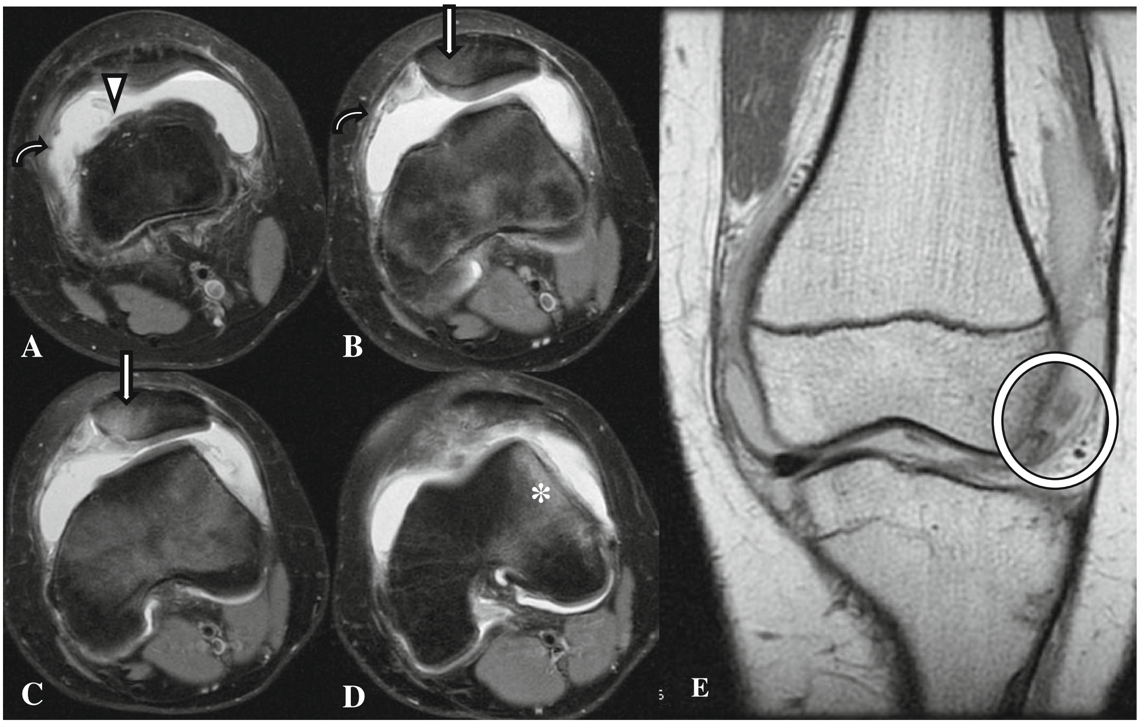

Fig. 6 Patellar dislocation (relocated) (15-year-old patient with knee pain after skiing). **a–d** Axial fat-suppressed PD and **e** coronal PDWI shows evidence of recent lateral patellar dislocation with bone contusions in the medial patella (*arrow*) and lateral femoral condyle (*asterisk*). There is irregularity of the articular cartilage at the median ridge and medial facet of the patella (*arrowhead*) with intra-articular bodies in the lateral joint space (*circle*). The patellar attachments of the medial patellofemoral ligament (MPFL) and medial retinaculum are markedly thickened and edematous likely reflecting partial tear or disruption of the fibers (*curved arrow*)

Patella alta (PA)

PA, or high-riding patella, is an abnormal superior location of the patella relative to the trochlear fossa due to an effectively lengthened patellar tendon [29]. In PA, the patella engages the trochlea with higher degrees of flexion, which in turn leads to reduced patellar contact area and decreased bone stability with more shallow degrees of flexion [23, 30]. In the presence of genu varus deformity, a PA configuration can be accentuated by shortening the distance between the quadriceps myotendinous junction and the tibial tuberosity [23]. This configuration may predispose to lateral patellar subluxation or dislocation. Patellar and quadriceps tendonitis and Osgood-Schlatter disease are also associated with PA [31].

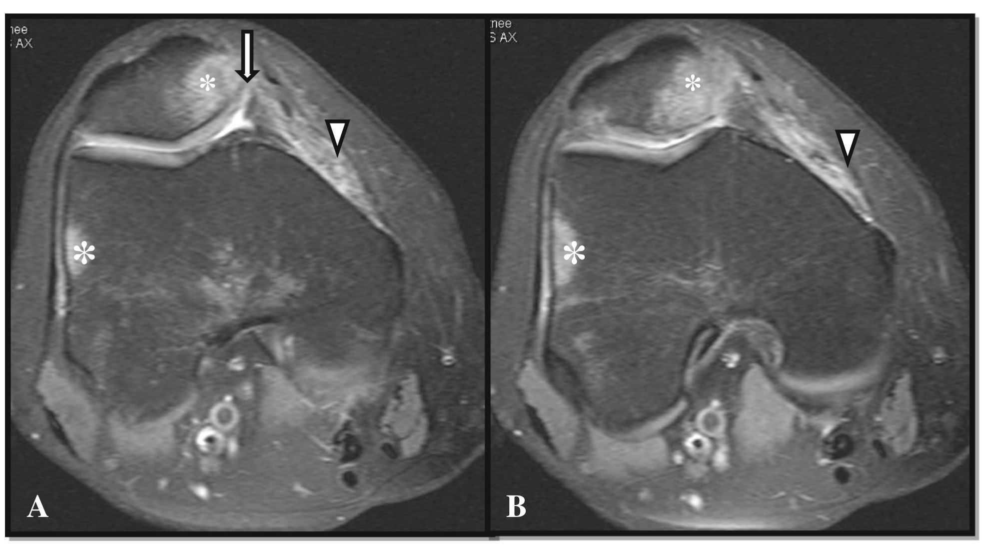

Fig. 7 Recurrent patellar dislocation (25-year-old patient with recent lateral patellar dislocation). **a**, **b** Sagittal PD-weighted images demonstrating avulsion of the MPFL at its patellar insertion (*arrow*) and tearing of its midportion (*arrowhead*). There are corresponding bone contusions in the medial facet of the patella and lateral femoral condyle (*asterisks*), so-called "kissing contusions"

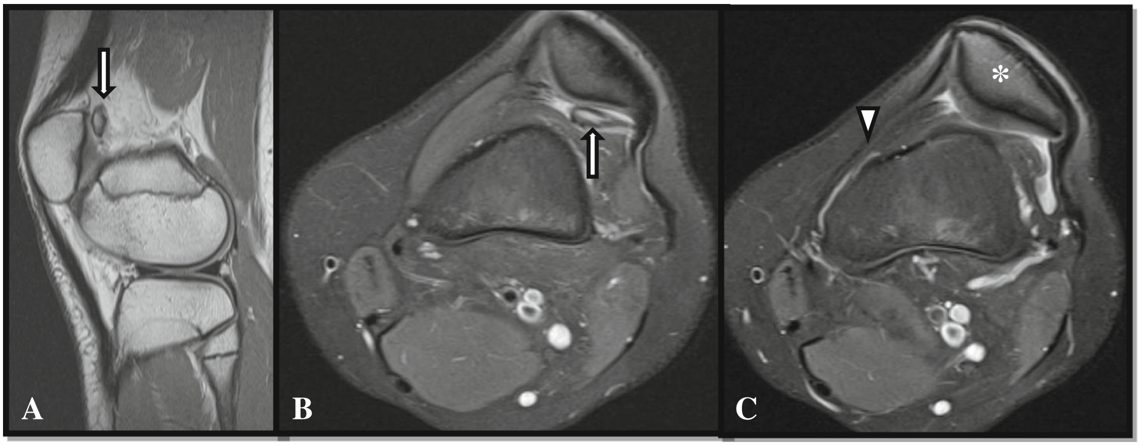

Fig. 8 Recurrent patellar dislocation (14-year-old patient with history of several patellar dislocations). **a** Sagittal PD-weighed images demonstrating patella alta and a loose body in the suprapatellar recess (*arrow*). **b**, **c** Axial fat-suppressed and PD-weighted images demonstrating attenuation of the midportion of the MPFL (*arrowhead*), denoting a remote tear/injury. There is lateral subluxation of patella (*asterisk*)

PA is a normal anatomic variant and most commonly causes no symptoms [32]. However, it is still an important finding, as it may increase the risk of patellar dislocation.

The most widely accepted method of evaluating patellar height is the Insall–Salvati index, which is calculated as the length of the patellar tendon measured posteriorly from the apex of the patella to its attachment to the tibial tuberosity, divided by the longest superoinferior diameter of the patella [33] (Fig. 11). Patella alta is defined as a patellar height ratio of more than 1.3 (normal is 0.8 to 1.1) [23, 30].

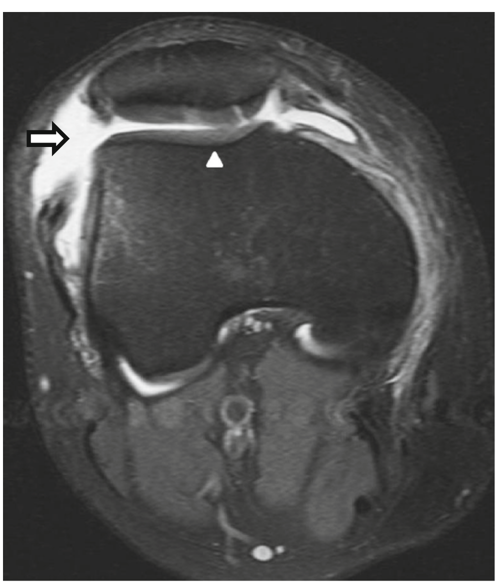

Fig. 9 Trochlear dysplasia Dejour type A (30-year-old with history of patellar dislocation and lateral retinaculum release). Axial proton–density image with fat saturation shows the preserved normal shape of the trochlea with medial and lateral facets, but with the shallow groove (*arrowhead*). Note released lateral retinaculum (*arrow*) and small fissure with some degeneration of patellar articular cartilage

Patella baja (PB) or infera

PB, or a low-riding patella, refers to an abnormal inferior location of the patella relative to the femoral trochlea. As a consequence, the patella remains engaged within the trochlear groove in full knee extension. It may rarely occur spontaneously in otherwise normal children [9]; however, it can also be seen in achondroplasia, neurological disorders, as a sequela of prior trauma, or as a complication of previous knee surgery such as harvesting of the patellar tendon for an anterior cruciate ligament reconstruction [34, 35]. It has been associated with Osgood Schlatter disease; however, it is unclear whether the association is cause or effect [36].

PB may be associated with restricted range of motion, crepitation, and retropatellar pain. It is defined as Insall–

Fig. 10 Trochlear dysplasia Dejour type B (25-year-old male with recent lateral patellar dislocation). Axial proton-density image with fat saturation demonstrating complete flattening of the femoral trochlea with absence of discrete medial and lateral facets (*arrowheads*). Bone contusions on lateral femoral condyle and medial patella are noted (*stars*)

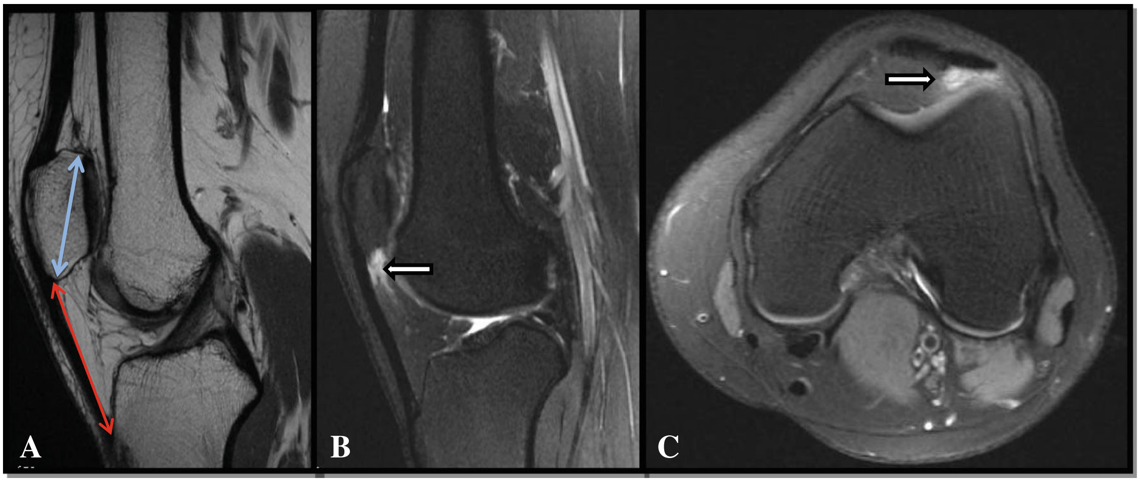

Fig. 11 Patellar alta and maltracking (25-year-old woman with chronic anterior knee pain). **a** Sagittal T1, **b** sagittal, and **c** axial fat-suppressed PD-weighted images demonstrating patella alta (Insall–Salvati index > 1.3), and edema in the superolateral aspect of Hoffa's fat pad (*white arrow*), suggestive of patellar maltracking

Salvati index less than 0.8 (Fig. 12) [9, 32]. Variants in patellar morphology and previous patellar surgery may affect the ratio, therefore, it is important to note the patient's symptoms before coming to a definitive diagnosis [37].

Bipartite patella

Bipartite patella represents failure of the fusion of the secondary ossification centers of the patella. They have been classified by Saupe into three types [38]. The first type involves the inferior pole of the patella, the second type involves the lateral margin of the patella, and the third type, the most common type, involves the superolateral pole. Patients with bipartite patella are generally asymptomatic [39]. Rarely, patients with bipartite patella may become symptomatic, complaining of anterior knee pain worse with squatting and climbing stairs with localized tenderness over the patella on exam.

A radiograph shows a well-corticated non-united fragment, commonly along the superolateral pole of the patella (Fig. 13). On MRI, asymptomatic bipartite patella is characterized by intact but thinned articular cartilage along the border between the patella and the fragment, fluid between the cleft, and a lack of any

Fig. 12 Patella baja (40-year-old man with knee pain). **a** Lateral radiograph and **b** sagittal T1-weighted image demonstrate patella baja (Insall–Salvati index < 0.8). Note the incidental enchondroma (*arrowhead*) in the proximal tibia

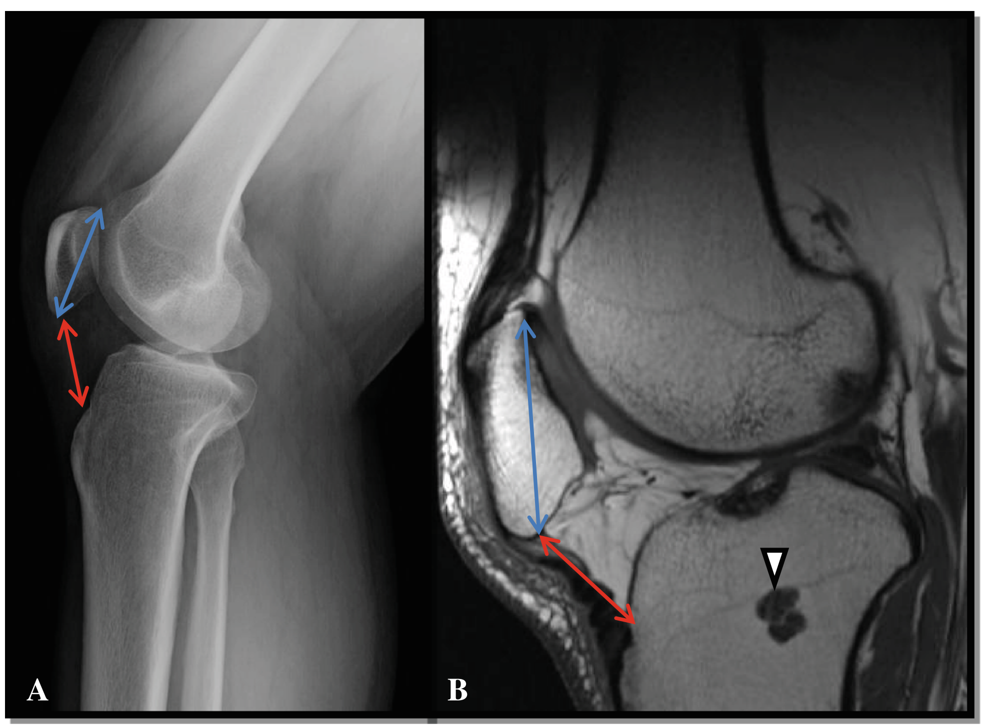

Fig. 13 Saupe type 3 bipartite patella. Frontal radiograph demonstrating the non-fused corticated bone fragment along the superolateral pole of the patella (*arrow*)

bone marrow edema or high signal within the patella or its fragment (Fig. 14) [40].

Symptomatic bipartite patella has been associated with the presence of bone marrow edema in the bipartite fragment. In a study by Kavanagh et al., bone marrow edema within the bipartite fragment was the sole finding in 49 % of the patient with bipartite patella and knee pain [41]. A symptomatic bipartite patella may be treated with surgical excision or lateral retinacular release [42].

Patellar tendinitis (PT) or Jumper's knee

PT is one of the most common tendon abnormalities in athletes, particularly in jumping athletes such as basketball and volleyball players. It is well established that the mechanism of disease in PT is that of degenerative tendinopathy rather than an inflammatory tendinitis, as absence of inflammatory cells has been shown in analyzed tissue samples excised from the patellar tendons of patients with acute symptoms [43, 44].

Fig. 14 Saupe type 3 bipartite patella. Axial fluid-sensitive image showing the non-fused ossific fragment with intact articular cartilage along the patella and fragment (*arrow*) and lack of bone marrow edema

Clinical symptoms may include pain, perceived swelling or fullness, and a sensation of "weakness" or "giving way". It may result in partial or full-thickness tears with and without avulsion of the patellar apex in more advanced cases. PT may be classified into four clinical stages of injury ranging from the presence of pain after activity without functional impairment to complete tendon rupture [45, 46].

Radiographs may be unremarkable in the more acute phase or occasionally demonstrate localized calcification in the region of the patellar tendon or rarely intrasubstance calcification or ossification [47].

On MR imaging, the normal patellar tendon demonstrates homogenous low signal intensity, should not exceed 7 mm in anteroposterior (AP) diameter, may have a convex anterior border, and should always have a well-defined posterior border [44, 48]. MR features of PT include focal thickening of the proximal one-third of the tendon, an AP diameter greater than 7 mm, and focal T2 hyperintensity within the proximal tendon, most commonly involving the medial one-third of the tendon [44, 48], but may extend to involve the central third of the tendon. Other imaging findings include an indistinct posterior tendon border and edema in the adjacent Hoffa's fat pad. The degenerative changes of the tendon, if allowed to progress, may cause full-thickness tearing of the tendon (Fig. 15).

Patellar tendon tear

Patellar tendon tears are less common than disruption of the QT as a cause of extensor mechanism dysfunction [49]. The major predisposing factors include chronic microtrauma and tendinopathy (e.g., Jumper's knee), prior interventions such as after tendon graft harvest for ACL repair or steroid injection, and systemic diseases such as chronic renal failure, diabetes, and rheumatoid arthritis [10, 50]. The most common site of tendon rupture is at the tendo-osseous junction at the patellar apex followed by its insertion upon the tibia [49, 51].

Patellar tendon tears are more common in athletes who are exposed to repetitive violent contraction of the quadriceps musculature, such as basketball or volleyball players. Patellar tendon rupture can be the end stage of "Jumper's knee" due to the cumulative effect of repetitive trauma and microtearing of the tendinous fibers (Fig. 15) [50, 52, 53]. Clinically, patients present with anterior knee pain, swelling, and a mobile patella with loss of extensor function.

On radiographs, complete rupture can manifest as significant patella alta. An avulsion fracture may be present.

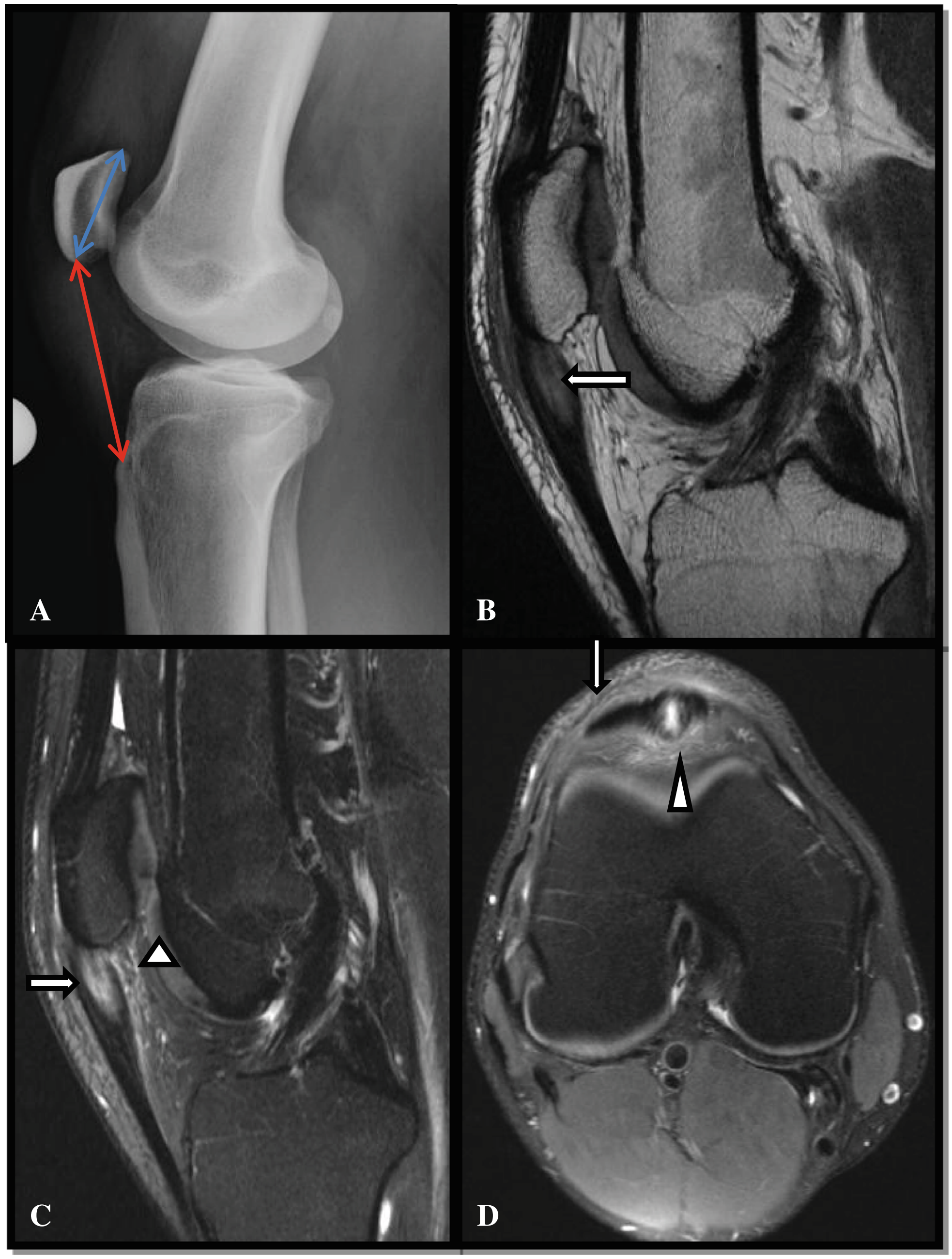

Fig. 15 Patellar tendinitis with partial thickness tear (27-year-old patient with non-traumatic knee pain localized to the patellar apex). **a** Lateral radiograph showing patella alta (Insall–Salvati index > 1.3). **c**, **d** Sagittal and axial fat-suppressed PD and **b** T1-weighted images demonstrating longitudinally oriented increased T2 signal and thickening involving the proximal patellar tendon consistent with a partial tear (*arrows*). There is edema within the superior aspect of Hoffa's fat pad (*arrowhead*), presumably reactive in nature

MR imaging demonstrates a discontinuity of the patellar tendon and increased T2 signal intensity due to hemorrhage and edema, which can result in blurring of the posterior margin of the patellar tendon extending to the region of Hoffa's fat pad (Fig. 16) [50].

Quadriceps tendon (QT) tear

QT tear is the second most common injury to the extensor mechanism after patellar fracture [49]. It occurs most commonly at the tendo-osseous junction at the patellar base [49, 54]. Other potential areas of injury may include the muscle belly, musculotendinous junction, or the tendon itself.

Most patients are in their 6th or 7th decades of life and have predisposing conditions, such as diabetes mellitus, that contribute to the injury [50].

Tendon tears can be partial or full thickness. Clinically, partial-thickness tears may be more difficult to diagnose as extensor function may be intact [50, 55]. The quadriceps femoris consists of three layers, which if visible on sagittal images, are the rectus femoris, combined vastus lateralis and medialis, and vastus intermedius tendons [47]. Discontinuity within any of the layers is consistent with a partial tear, while transection of all layers is consistent with a complete tear [50].

Radiographs may demonstrate patella baja with a complete tendon tear, an avulsed fragment arising from the superior

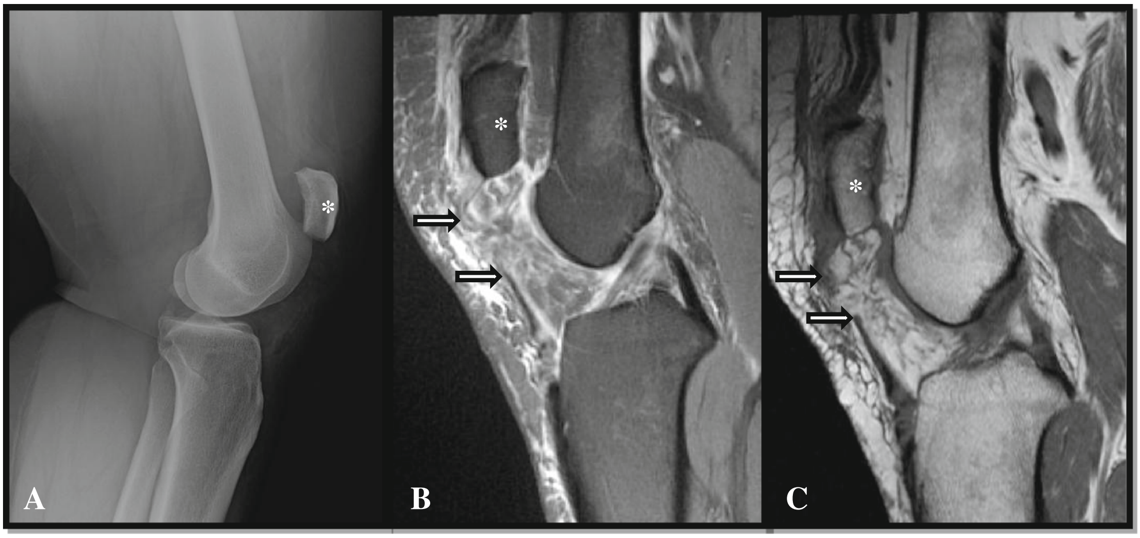

Fig. 16 Full-thickness patellar tendon rupture (57-year-old patient with anterior knee pain and swelling after a fall with twisting mechanism). **a** Lateral radiograph showing patella alta (*asterisk*). **b** Sagittal fat-suppressed PD and **c** sagittal T1-weighted images demonstrating a ruptured patellar tendon with proximal retraction and patella alta, creating a gap of approximately 3 cm (*arrows*)

patella, suprapatellar soft tissue swelling, and calcification in QT.

On MR imaging, hematoma and surrounding edema at the site of injury manifests as an area of increased signal intensity on T2-weighted images. Partial tendon tearing may be best appreciated on axial fluid-sensitive sequences. In a complete tendon tear, important points to note in the report include the distance between the torn tendon edges, characteristics of the torn tendon edges, and presence of hematoma, as these findings may affect surgical planning (Fig. 17) [47].

Osgood–Schlatter disease (OSD)

OSD was first described over 100 years ago [56], yet different etiologies have been postulated in the literature such as osseous avulsions involving portions of the tibial tuberosity secondary to forceful contraction of the extensor mechanism, or insult to the distal patellar tendon at its insertion up on the tibial tubercle with more emphasis on the soft-tissue aspect of the disease [57]. The most popular etiology is suspected to be a chronic avulsion injury related to repetitive microtrauma and traction on the tibial tubercle by the patellar tendon [58], hence it is considered traction apophysitis [59].

OSD is most common among adolescent male athletes in sports that require jumping and kicking. It is bilateral in up to 50 % of cases [60]. Patients clinically present with pain and swelling over the tibial tuberosity that is aggravated by exercise.

In patients with active OSD, knee radiographs can be normal or may demonstrate osseous fragmentation of the tibial tubercle, soft tissue swelling, and obliteration of the inferior angle of the Hoffa's fat pad [60]. If the tibial surface is not yet ossified, radiography may not reveal any abnormalities [47].

MRI may reveal enlargement of the patellar tendon with increased signal intensity at its distal insertion on both T1- and T2-weighted images denoting tendinosis or tendinitis (Fig. 18), a fluid distended deep infrapatellar bursa (Fig. 19), edema of the surrounding soft tissue, and bone marrow edema subjacent to the tibial tubercle [61].

In its chronic form, OSD may leave an ossicle under the distal patellar tendon adjacent to the tibial tuberosity or a prominent tibial tubercle, known as the unresolved Osgood–Schlatter lesion (Fig. 20) [62]. These may lead to pain and disability, due to recurring injuries or athletic exercises. Knee radiographs show a non-fused corticated ossicle adjacent to the tibial tuberosity or a prominent tuberosity without significant soft tissue swelling. MRI shows the ossicle along the distal patellar tendon or prominence of the bony tuberosity without the soft tissue edema. Infrapatellar bursitis or bone marrow edema can be seen in active OSD. Slight residual thickening of the distal patellar tendon may be seen in chronic cases. When symptomatic, the unresolved Osgood–Schlatter lesion may be treated with resection of the ossicle and adjacent bursa or scar tissue [63].

Sinding–Larsen–Johansson syndrome (SLJ)

SLJ is traction apophysitis of the inferior patellar pole [59], which represents a chronic traction injury of the immature tendo-osseous junction and has been considered as an equivalent to jumper's knee in a pediatric setting by some authors [64]. The etiology is believed to be related to forceful contraction of the QT against resistance [60]. SLJ is seen in athletes typically between 10–14 years of age [65]. Patients may present with pain at the inferior pole of the patella associated with swelling.

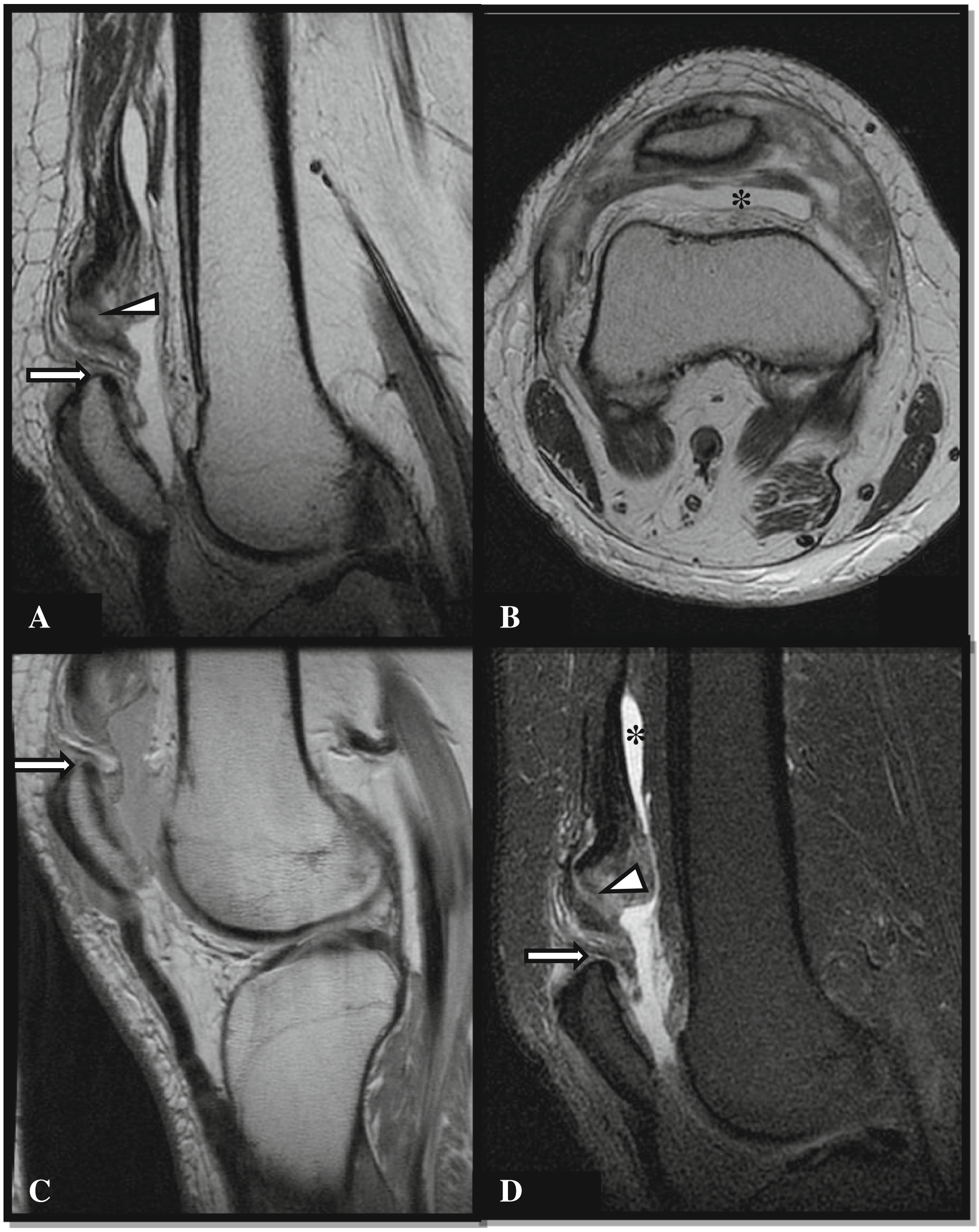

Fig. 17 Distal quadriceps tendon rupture. **a** Sagittal and **b** axial T2, **c** sagittal T1, and **d** sagittal fat-suppressed PD-weighted images demonstrating a complete tear of the quadriceps tendon at its attachment to the patellar base (*arrows*) with no significant retraction. The tear also involves the medial and lateral expansions of the patellar attachment. Note the degenerated appearance of the torn quadriceps tendon (*arrowheads*) and associated effusion (*asterisk*)

Radiographs may reveal patella alta and tiny osseous fragments adjacent to the inferior pole of the patella [60]. Although it may be difficult to distinguish SLJ from patellar sleeve avulsions on radiographs, the latter typically involves an injury to spherical growth plate at the patellar apex in the pediatric population; however, small osseous fragments are usually present in both conditions [57].

MRI demonstrates an osseous avulsion injury at the proximal patellar tendon insertion without extensive cartilaginous injury, thereby differentiating SLJ from a patellar sleeve avulsion (Fig. 21) [60]. It is important to distinguish between the two entities as they may require different treatment strategies, with conservative management for SLJ versus possible internal fixation for displaced patellar sleeve avulsion fractures [57]. MRI in the acute phase also shows thickening and edema of the patellar tendon along the inferior pole of the patella and adjacent soft tissue edema. In the chronic healed phase of SLJ, an ossific protuberance from the inferior pole of the patella may be present.

Anterior knee bursitis

There are 12 named bursae about the knee, out of which four bursae are more susceptible to inflammation: the prepatellar, superficial, deep infrapatellar, and pes anserine bursae [10]. Trauma, infection, overuse, and hemorrhage may result in inflammation of a bursa [66].

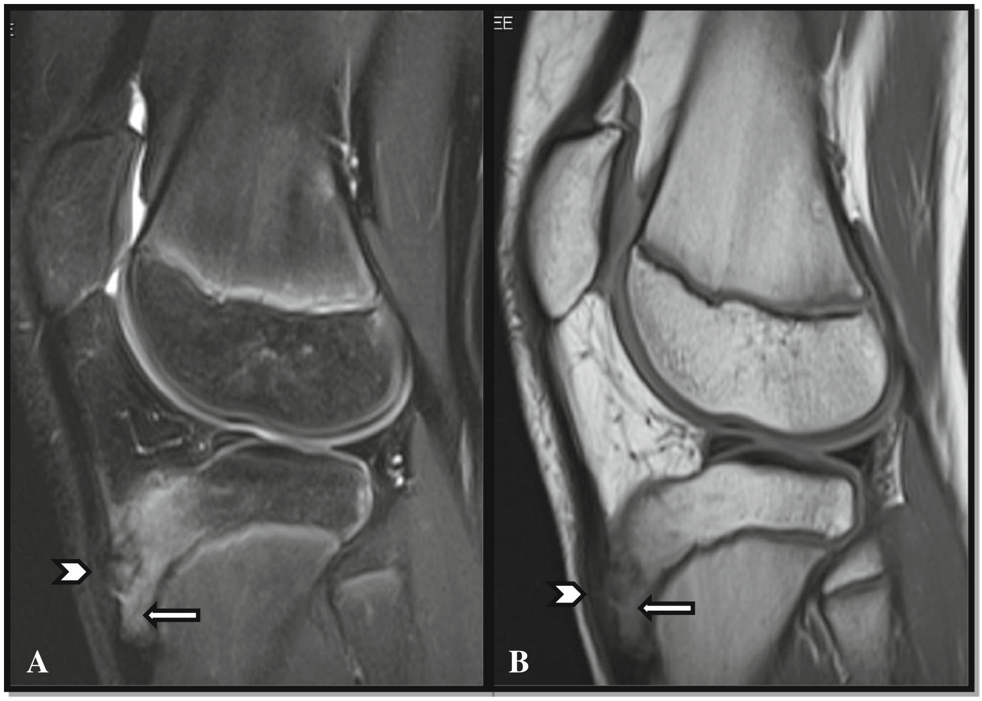

Fig. 18 Osgood–Schlatter disease (14-year-old patient with anterior knee pain). **a** Sagittal fat-suppressed T2 and **b** sagittal PD-weighted images demonstrating bone marrow edema and osseous fragmentation of the tibial tuberosity (*arrows*), with overlying distal patellar tendon and soft tissue edema (*arrowhead*)

Prepatellar bursitis (PPB) or housemaid's knee

The prepatellar bursa is located between the patella and overlying subcutaneous tissue. Inflammation or hemorrhagic bursitis can occur as a result of acute injury, such as falling directly onto the patella, or from recurrent minor injuries in the form of prolonged or repetitive microtrauma [67]. Pyogenic PPB is also common, especially in children [10]. *Staphylococcus aureus* is the most commonly isolated organism. Clinically, patients may present with pain and swelling over the patella.

Radiographs may appear normal or may demonstrate thickening of the prepatellar soft tissues. In chronic cases, calcification may be seen in the region of the prepatellar soft tissues.

On MR imaging, PPB appears as an oval-shaped fluid signal intensity collection between the subcutaneous tissue and the patella (Fig. 22), which may reflect either an effusion and/or hematoma (Fig. 23). The effusion and edema may

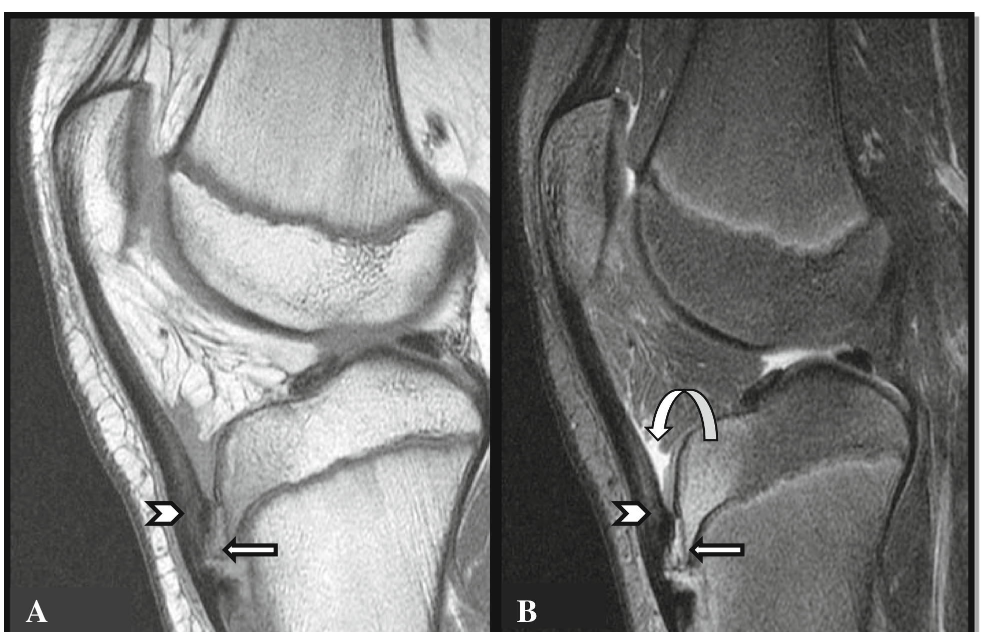

Fig. 19 Osgood–Schlatter disease (11-year-old boy with knee joint "giving out"). **a** Sagittal PD and **b** sagittal fat-suppressed PD-weighted images demonstrating bone marrow edema and osseous fragmentation of the tibial tuberosity (*arrows*), overlying distal patellar tendon and soft tissue edema (*arrowhead*) and fluid in deep infrapatellar bursa (*curved arrow*)

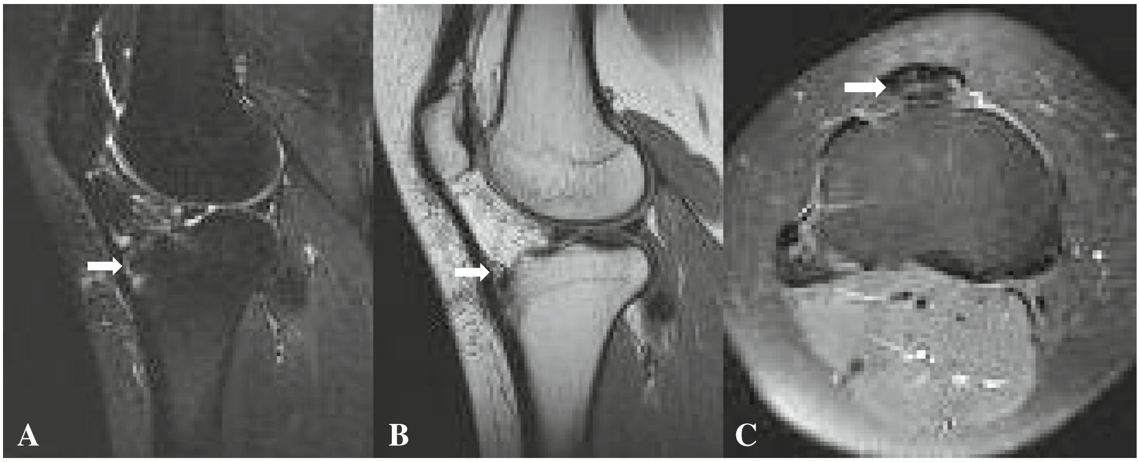

Fig. 20 Non-resolved Osgood–Schlatter lesion (17-year-old female with pain and tenderness over tibial tuberosity). **a** Sagittal T2 fat-suppressed, **b** sagittal, and **c** axial PD images demonstrating small non-fused bone fragment adjacent to tibial tuberosity along patellar tendon insertion (*arrow*). Notice mild bone marrow edema within non-fused bone fragment and adjacent tibial tuberosity

extend and communicate with the superficial infrapatellar bursa or pretibial bursa [47].

Infrapatellar bursitis (IPB)

There are both superficial and deep infrapatellar bursae. The superficial infrapatellar bursa is located between the tibial tubercle and the overlying skin, whereas the deep infrapatellar bursa is between the posterior aspect of the patellar tendon and the anterior aspect of the proximal tibia [68].

Superficial IPB, also known as "clergyman's knee", is due to inflammation and fluid accumulation resulting from chronic stress. It may be part of an overuse spectrum as seen in jumpers and runners [47]. The inflamed bursa may communicate with the prepatellar bursa superiorly and with the pretibial bursa inferiorly [47]. IPB-related symptoms are remarkably similar to those of patellofemoral arthralgia [9]. Soft tissue swelling inferior to the patella may be palpable on physical examination.

On radiographs, soft tissue thickening or a large collection presenting as a soft tissue density/mass may be seen.

On MRI, superficial IPB presents as a loculated, exophytic collection anterior to the distal patellar tendon. Contrary to pretibial subcutaneous edema, bursitis appears as a localized collection with well-defined borders [67]. Deep IPB appears as a triangular fluid collection posterior to the inferior patellar tendon (Fig. 24). When deep IPB presents in younger patients, it can be differentiated from Osgood–Schlatter disease by the lack of a thickened and increased intrasubstance T2 hyperintensity in the patellar tendon [69].

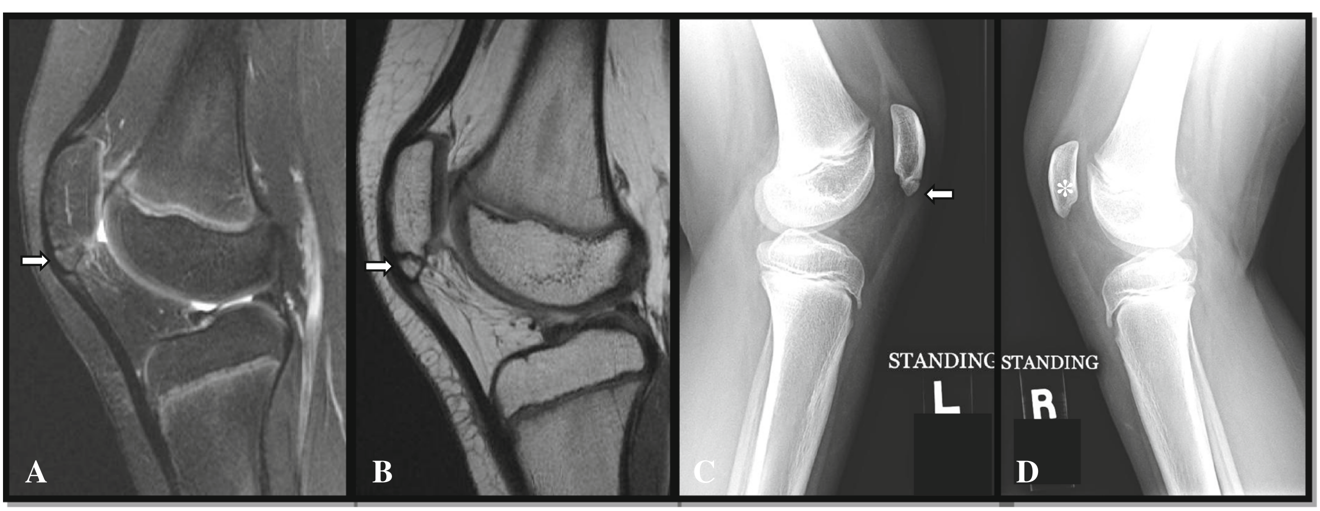

Fig. 21 Sinding–Larsen–Johansson disease (8-year-old girl with recurrent left knee pain). **a** Sagittal PD and **b** sagittal fat-suppressed PD-weighted images of the left knee demonstrate fragmentation at the patellar apex with associated edema (*arrows*). **c, d** Note the normal appearing right patella on plain radiograph (*asterisk*)

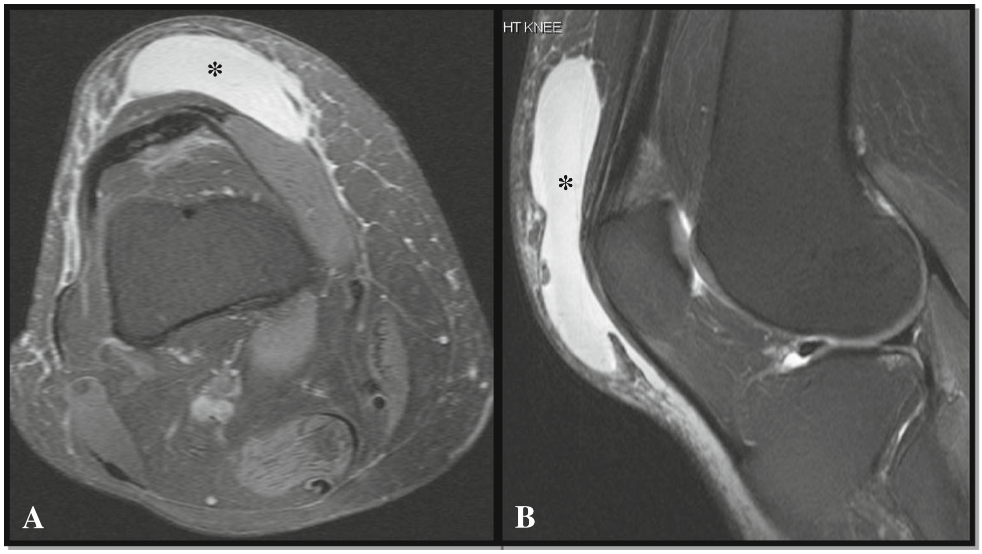

Fig. 22 Prepatellar bursitis (70-year-old patient with anterior knee pain and swelling). **a** Axial fat-suppressed T2 and **b** sagittal PD-weighted images demonstrate oval-shaped fluid signal intensity between the subcutaneous tissue and the patella (*asterisk*)

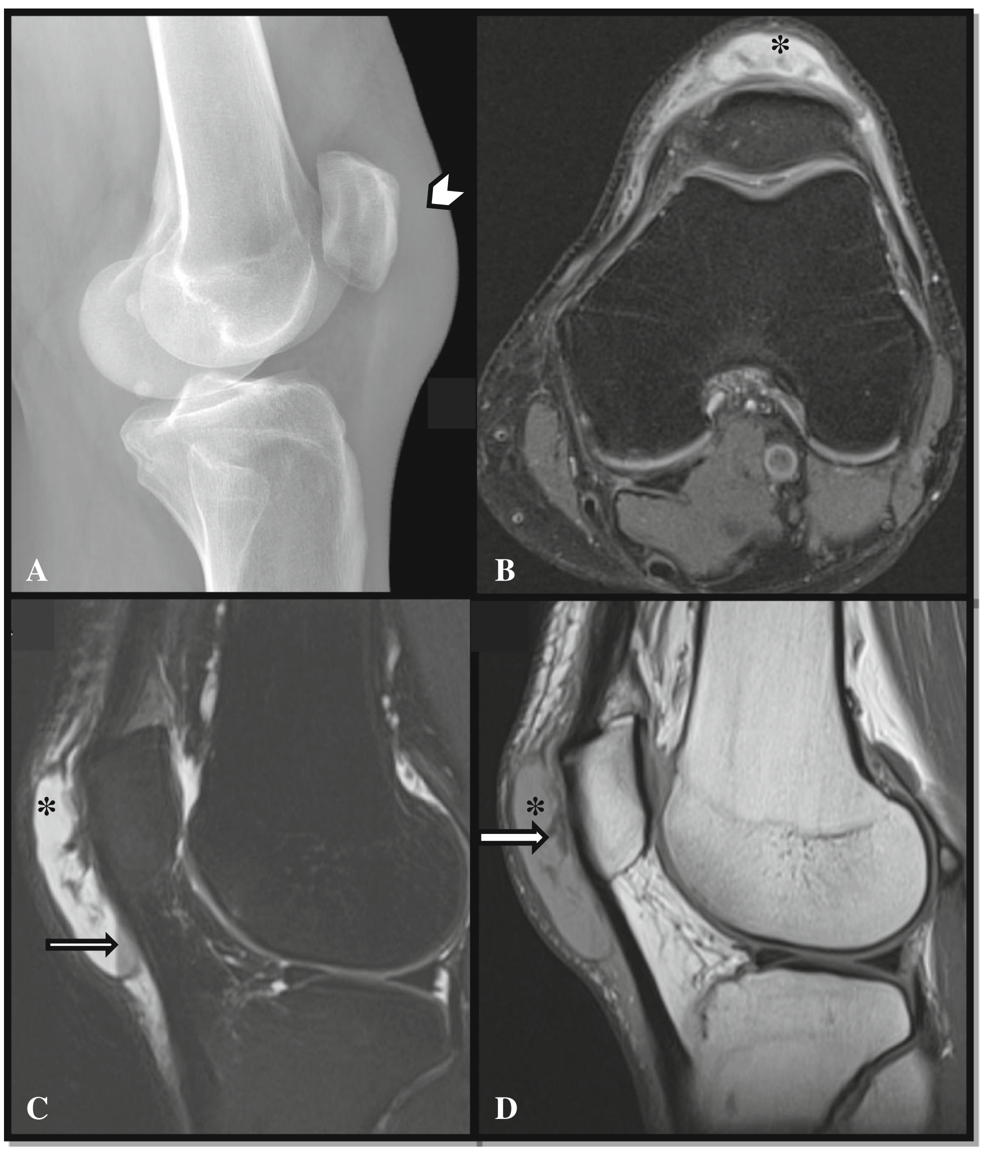

Fig. 23 Hemorrhagic prepatellar bursitis (60-year-old patient with anterior knee pain and swelling). **a** Lateral radiograph shows prepatellar soft tissue thickening (*arrowhead*). **b** Axial and **c** sagittal fat-suppressed T2 and (D) sagittal PD-weighted images demonstrating an oval-shaped fluid signal intensity collection in the prepatellar soft tissue (*asterisk*), containing debris and fluid–fluid level (*arrow*) due to hemorrhage

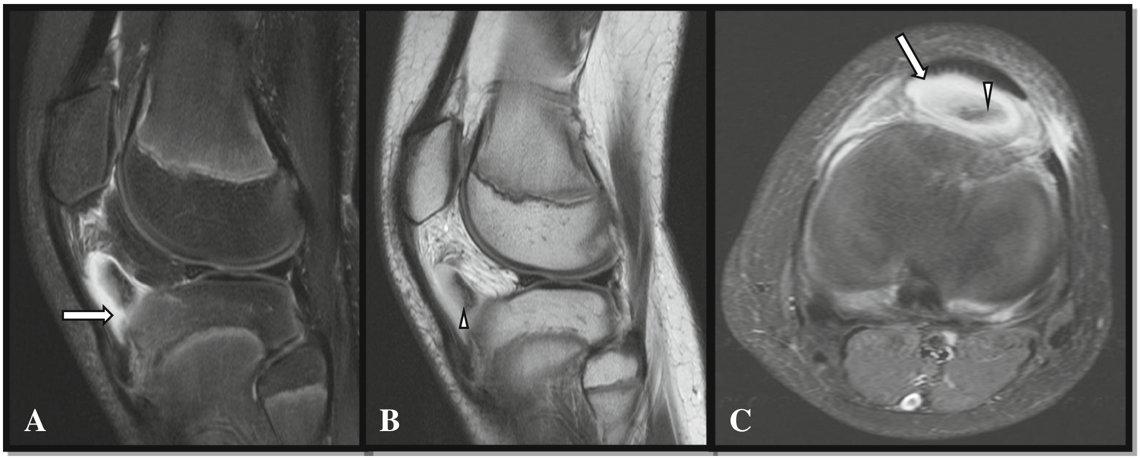

Fig. 24 Hemorrhagic deep infrapatellar bursitis (13-year-old boy with knee pain after an unspecified injury). **a** Sagittal fat-suppressed T2, **b** sagittal, and **c** axial fat-suppressed PD-weighted images demonstrating a complex fluid collection in the deep infrapatellar bursa (*arrows*) with low signal fluid–fluid level denoting blood degradation products (*arrowheads*)

Pes anserine bursa

The pes anserine bursa is located along the medial aspect of the proximal tibia separating the pes anserine tendons (the distal sartorius, gracilis, and semitendinosus tendons) from the tibial insertion of the medial collateral ligament and the bony surface of the medial tibial condyle [70]. Pes anserine bursitis may occur as a result of overuse, especially in runners, or following medial collateral ligament injury [47, 71]. The patient may complain of medial knee pain isolated below joint line.

On MR imaging, pes anserine bursitis classically appears as an oblong, multiloculated fluid collection at the medial aspect of the proximal tibia subjacent to the pes anserine tendons, best appreciated on T2-weighted axial images (Fig. 25). It is important to differentiate pes anserine bursitis from semimembranosus bursitis and a popliteal cyst, the latter two entities are typically located more posteriorly, and the neck of popliteal cysts classically originates between the medial head of the gastrocnemius and semitendinosus tendons [67].

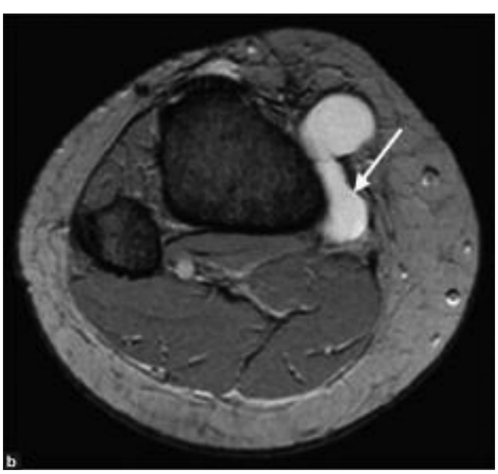

Fig. 25 Pes anserine bursitis. Axial fluid sensitive image shows fluid collection (*white arrow*) along the pes anserine tendons and the proximal anteromedial tibia, just below the joint line

Mediopatellar plica syndrome (MPS)

A plica is a normal synovial fold found in the lining of a joint that sometimes becomes symptomatic [72]. The most commonly encountered plicae of the knee are the infrapatellar, suprapatellar, and mediopatellar plica [73].

Direct trauma, repetitive sports activities, or other pathologic knee conditions may result in chronic inflammation and a thickened fibrotic plica, which in turn can irritate the synovium at the condylar margin with motion of the knee and lead to synovitis and articular cartilage loss [72].

The mediopatellar plica is the most likely plica to cause symptoms when it becomes inflamed, thickened, and fibrotic [74]. The mediopatellar plica originates from the medial aspect of the joint capsule, inserts into the synovium, and covers the infrapatellar fat pad [72] with a cranio-caudal course along the medial aspect of the joint. MPS can cause anterior knee pain particularly in the medial infrapatellar region [75, 76] that may be associated with a palpable tender band in the medial peripatellar area [9].

On MR imaging, the normal plica has low signal on both T1- and T2-weighted images and is easily identified with some degree of joint distention. Increased T2 signal intensity may be seen in symptomatic plica. When the articular fluid is not enough to delineate the plica, MR arthrography is a useful technique to better delineate the plica [72]. Mediopatellar plica has been classified into four types, types A through D, on the basis of size and the extent of coverage over the anterior surface of the medial femoral condyle [77]. Types C and D can be trapped between the medial condyle and the patella, become thickened, and even cause chondral damage (Fig. 26) [72].

Infrapatellar and suprapatellar fat pad

The infrapatellar (Hoffa's) fat pad is an intracapsular-extrasynovial structure, bordered by the inferior pole of the patella superiorly, the joint capsule and patellar tendon anteriorly, the proximal tibia and deep infrapatellar bursa inferiorly, and the joint cavity posteriorly. It has an attachment with the anterior horns of the menisci inferiorly and to the periosteum of the tibia [78, 79].

Hoffa's disease, also known as infrapatellar fat pad impingement syndrome [80], is secondary to trauma or joint space narrowing [78], with overuse as the most likely mechanism. Initially, the repetitive microtrauma causes hemorrhage in the fat pad [80, 81], with chronic mechanical irritation either between the patellar tendon and lateral femoral condyle or the lateral aspect of Hoffa's fat pad and lateral femoral condyle. Hoffa's disease also has high association with patellar maltracking or malalignment including patella alta and lateral subluxation of the patella [82]. Diffuse edema of Hoffa's fat pad has also been associated with HIV [83].

Symptoms of anterior knee pain are generally accompanied by tenderness and induration of the infrapatellar region [9]. On MR images, an ill-defined area of T2 hyperintensity is present at the inferolateral aspect of the patellofemoral joint and the lateral portion of the Hoffa's fat pad. In other cases, the edema may involve the entire fat pad (Fig. 27). With chronic disease, hypointense T2 signal intensity may be present secondary to fibrosis and hemosiderin deposition [47]. Other associated findings include lateral patellar subluxation and patella alta [82].

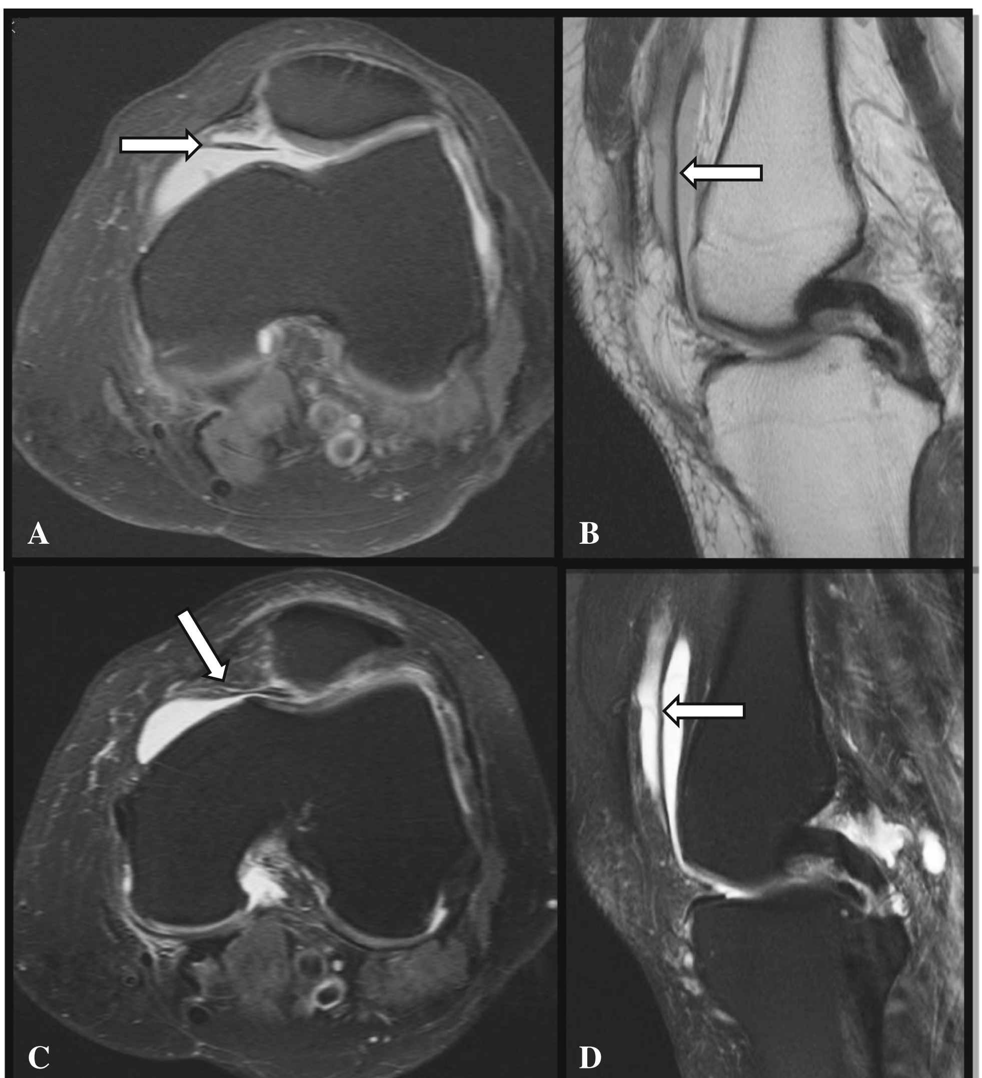

Fig. 26 Mediopatellar plicae syndrome (60-year-old patient with chronic anterior knee pain). **a**, **c** Axial, **b** sagittal fat-suppressed T2, and **d** sagittal PD-weighted images demonstrating mediopatellar plica that has a shelf-like appearance and covers the anterior aspect of the medial femoral condyle (*arrows*)

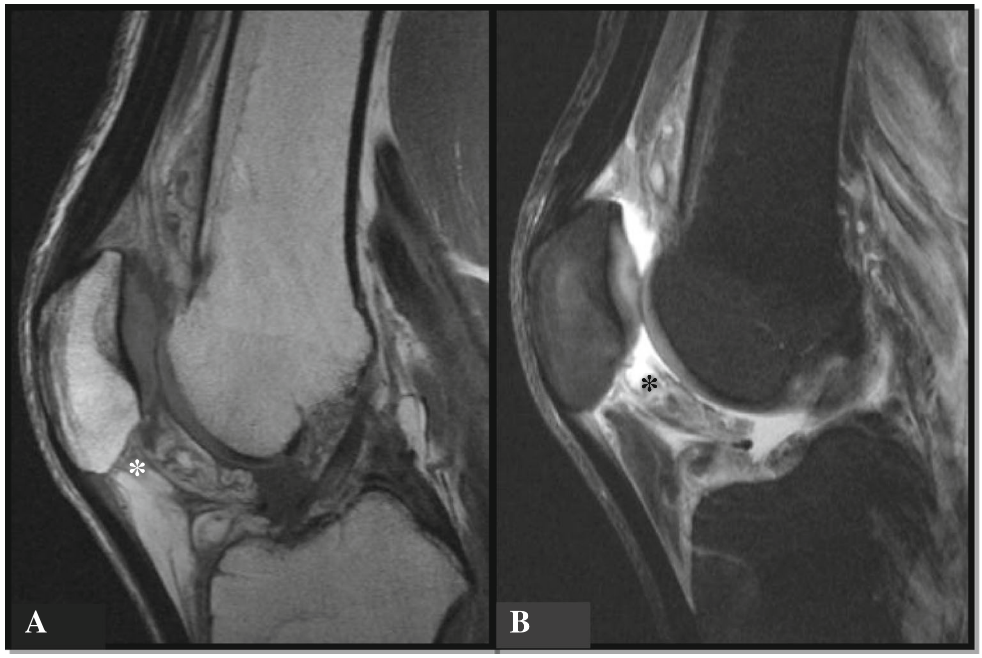

Fig. 27 Hoffa's disease. **a** Sagittal T1 and **b** sagittal fat-suppressed T2-weighted images demonstrating edema involving a significant portion of the infrapatellar fat pad (*asterisks*). Please note the edema within the anterior and posterior suprapatellar fat pad

The suprapatellar fat pad lies posterior to the quadriceps tendon insertion on the patellar base and anterior to the suprapatellar recess, which separates it from prefemoral fat pad. Suprapatellar fat pad edema and enlargement might result from repetitive microtrauma or overuse injury with resultant hemorrhage and inflammation [84]. On MR, the convex appearance of the posterior border of the suprapatellar fat pad has been suggested as a criterion for its edematous enlargement (Fig. 28) [84], however its association with anterior knee pain is controversial [85, 86].

Conclusions

AKP is the most common complaint of the knee joint, which can be secondary to a variety of etiologies. We have reviewed several of the most common causes of AKP, falling into seven broad categories of diseases including chondral abnormalities, patellar instability and dislocation, bipartite patella, abnormal patellar location, various tendinopathies, bursal inflammation, chronic traction avulsion injuries in pediatric and adolescent patients, and miscellaneous diseases including MPS and

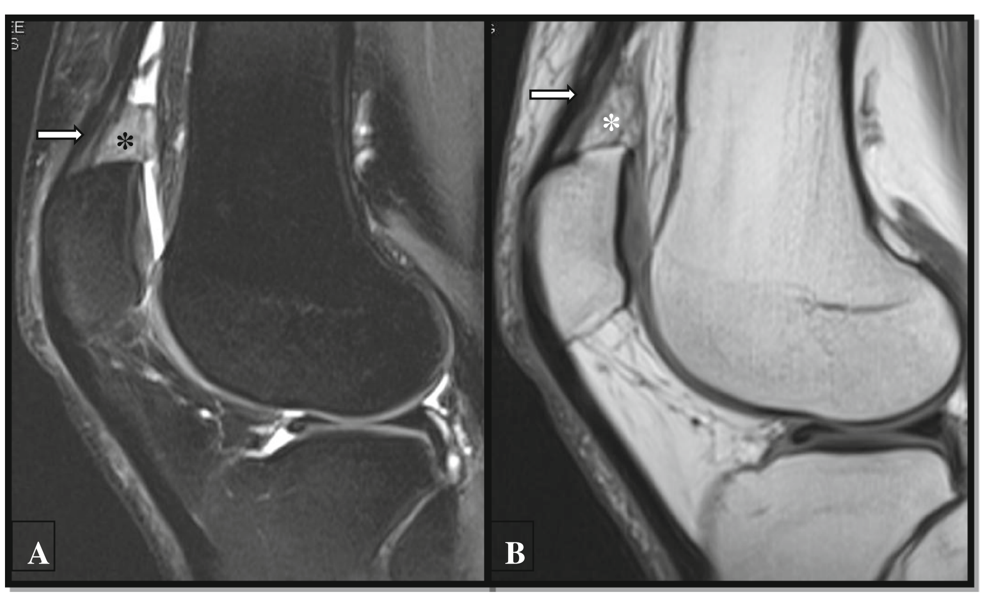

Fig. 28 Inflammation of the suprapatellar fat pad (60-year-old patient with knee pain). **a** Sagittal fat-suppressed T2 and **b** sagittal PD-weighted images demonstrating inflamed and edematous suprapatellar fat pad posterior to the quadriceps tendon (*asterisk*). Note the posterior convexity of the fat pad better seen on sagittal PD image. There is associated mild tendinosis of adjacent quadriceps tendon (*arrow*)

Hoffa's disease. An improved understanding of AKP with an increased awareness of the common abnormalities and their appearance, particularly on MR imaging, allows for a more accurate diagnosis, which may facilitate improved treatment and pre-operative planning.

Conflict of interest There are no conflicts of interest to disclose.

Disclosure None.

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