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REVIEW

The patellar extensor apparatus of the knee

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Abstract The patellar extensor apparatus is composed of the quadriceps muscles that converge to a central tendon that inserts on and invests the patella. It continues by the patellar tendon to act on the tibial tuberosity and thereby extends the leg at the knee. The structure can be thought of as a chain with pathology able to occur at each level. Pathological processes are generally caused by the great force experienced at each level both chronically and acutely. The forces are, however, greatly modified by the particular geometries present at each level. The various pathological processes and factors that modify them are reviewed at each level, beginning with the quadriceps musculature and ending at the terminal point of action, the tibial tuberosity.

Keywords Patella · Extensor · Trochlea · Children

Introduction

The extensor mechanism of the knee is made up of a complex network of static and dynamic stabilizers that converge to surround the centrally located patella, the latter serving as an osseous pulley facilitating the efficient translation of force into action. This is achieved by the articulation of the anteriorly placed patella with the trochlear groove. The result is the production of a greater arc of motion and subsequently greater moment of force.

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Normal anatomy

The quadriceps muscle group provides the motive force for knee extension. The group is composed of four muscles, thus the quadriceps (*quadri* four, *ceps* from *capitis* head). The longest and most superficial of the quadriceps, and so the most prone to injury, is the rectus femoris. This muscle arises from the pelvis from two heads and spans two joints, the hip and the knee. The remaining three muscles comprise the vasti and include the vastus medialis, lateralis and intermedius. These take their origin directly from the femur.

The quadriceps musculature forms the quadriceps tendon a short distance above the patella. Each muscle contributes aponeurotic fibers to the tendon and the resultant multilayered structure is clear on an MR image. The rectus femoris contributes the most anterior layer, sending fibers to become the main body of the patellar tendon.

The vastus medialis merges with the lateralis and is the middle layer. Most posterior is the contribution from the vastus intermedius. An additional muscle bundle, the vastus medialis obliquus is either a wholly separate muscle or simply a part of the distal vastus medialis. Fibers of this bundle insert distally on the medial patella at a steeper angle relative to the main tendon of the vastus medialis proper. As such, the vastus medialis obliquus is an active restraint to patellar subluxation and serves to counterbalance the strong lateral forces on the patella.

The quadriceps tendon is continuous, with the patellar tendon inferior to the patella, making the patella (from the Latin for "little dish" *patina* or *patena*) the largest sesamoid bone in the body. The patella ossifies between 4 and 7 years of age. As with many ossific centers it ossifies in a fragmented manner, which should not be misinterpreted as a fracture. It slides in a trochlear groove on the anterior articular surface of the femur. Three articular facets are recognized on the posterior surface of the patella: lateral, medial and odd facets. The odd facet is sometimes present and is the most medial patellar facet. It is clearly separated from the medial facet by a change in the orientation of its surface (Fig. 1). The exact morphology of the patellar articular surface and the adjacent trochlea, unlike most joints, is highly variable.

The patellar tendon yokes the patella to the tibial tuberosity. This thick, flat tendon is derived from the fibers of the quadriceps. It has an apophyseal insertion on a tuberosity on the anterior surface of the proximal tibia. The tuberosity ossifies during adolescence either as a single or several ossific centers. The tendon also has a nonapophyseal insertion on the anterior cortical surface of the tibia just below the tibial tuberosity.

The course of the tendon commonly runs slightly lateral to its origin. It should be emphasized that the course itself varies with knee motion. In full extension the "screw home" mechanism rotates the tibia externally and helps lock the knee in position. As the tibia rotates externally, the tuberosity, and so the course of the tendon, becomes more lateralized. These factors serve to increase the lateral force on the patella and contribute to patellar subluxation. The "Q," or quadriceps, angle measures the course of the patellar tendon against the vertical. It is defined as the angle between a line drawn from the anterior superior iliac spine through the center of the patella and a second line from the patellar center to the tibial tuberosity. Women, in general, have a higher Q angle; their wider pelvis causes the anterior inferior iliac spine, and with it the proximal attachment of rectus, to be lateral [1].

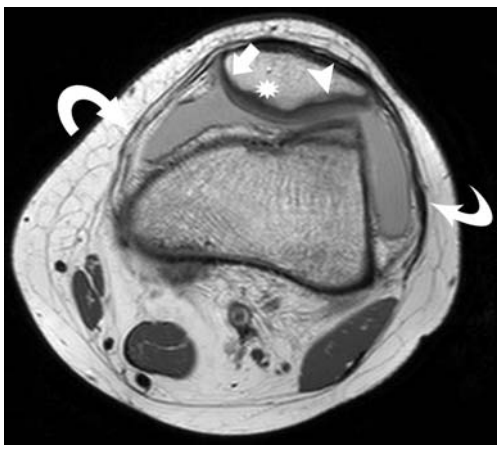

Fig. 1 Axial proton density (PD) image. The demarcation between the odd (*arrowhead*), medial (*asterisk*) and lateral (*arrow*) facets is indicated. Also shown is the medial and lateral patellar retinacula (*curved arrows*)

Various means have been used to measure the length of the patella tendon. The Insall-Salvati ratio is very commonly used, as it is accessible on any lateral film of the knee and is not affected by varying degrees of flexion. In general, the patellar tendon should be approximately equal to the greatest superoinferior dimension of the patella. In children, the patellar tendon is visualized as somewhat longer than the patella because the patella has not finished its ossification. Patella baja, or infera, is denoted as a tendon-to-patella ratio of below 0.79 in women and 0.74 in men. Patella alta is indicated as a ratio of over 1.32 in men and 1.54 in women [2]. From the foregoing it should be clear that women are more prone to patellofemoral joint disease because of geometric factors including higher lateral forces acting on a relatively longer patellar tendon.

Posterior to the patellar tendon is the infrapatellar, or Hoffa's, fat pad. The fat pad is useful as an arthroscopic port and can serve as a secondary shock absorber for frontal impact. It has its own intrinsic disease processes but also serves as an extrinsic indicator of dynamic dysfunction elsewhere in the extensor mechanism.

Disease processes of the patellar extensor apparatus are treated by level, beginning with the quadriceps tendon and ending with the tibial tuberosity.

Quadriceps tendon

Rupture

Rupture of the quadriceps tendon in children is very rare. Although penetrating trauma can cause transection of the tendon, rupture caused by chronic tendinosis as commonly occurs in adults is distinctly unusual in children. Steroids can weaken the tendon and cause rupture. Some chronic ailments for which steroids are given can also cause quadriceps rupture, including rheumatoid arthritis and systemic lupus erythematosus.

The tendon is easily observed on a lateral radiograph anterior to a thin fat pad. MR images show the tendon to have a clearly multilayered structure with the vasti and the contributions of the rectus separated by a thin stripe of fat signal (Fig. 2). Slightly increased signal within the tendon fibers at the patellar insertion is a normal finding.

With rupture, the patella is depressed inferiorly (patella baja). The patellar tendon might have a wrinkled appearance, as it can no longer be held under tension. Distention of the prepatellar bursa is frequently present as joint fluid passes through the ruptured tendon. In cases of rupture

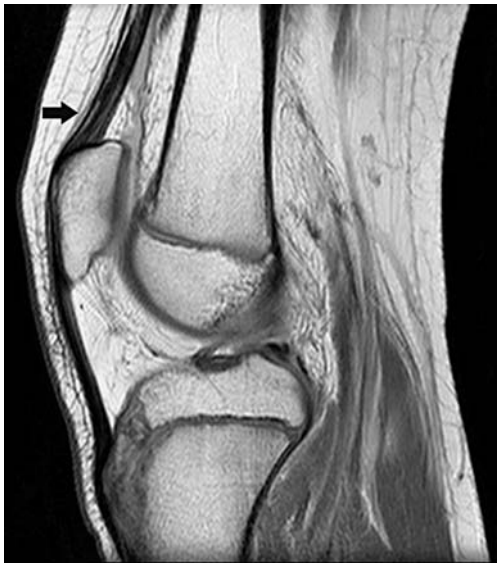

Fig. 2 Sagittal T1-W image, 13-year-old boy. The multiple layers of the quadriceps tendon (*arrow*), each derived from a different muscle, are clearly seen, each separated by slightly high signal

caused by chronic disease, overuse or steroid administration, the tendon shows heterogeneously increased signal with poor visualization of its lamellated architecture.

Patella

Dorsal defect and bipartite patella

The bipartite patella and the dorsal defect of the patella are usually thought of as normal variants. However, some investigators have questioned whether these two entities are actually the result of chronic traction on the patella by the vastus lateralis insertion at the superolateral patella [3, 4]. Histological support for this theory can be found in reported cases of bone necrosis at the site of the dorsal defect suggesting a pathological process. In addition, it is far more common in males, suggesting that the higher stress from male musculature is responsible for the nonfusion of the patellar ossific centers. The concept of an extensor chain of action in which the apparatus is conceived as multiple units functioning together with multiple levels of attachment lends itself to the philosophy that failure can occur at any level, the superolateral pole of the patella included. Despite the controversy as to the nature of the dorsal defect and bipartite patella, there are definite instances when both can be pathological.

The normal sequence of ossification in the patella is from two or three centers, with the superolateral corner usually representing one ossific center. In the bipartite patella, the superolateral center fails to fuse to the main body of the patella. It is commonly but not always bilateral. The accessory center takes at least some of the insertion of the vastus lateralis as noted above. It is separated from the main body of the patella by fibrocartilaginous tissue. The posterior surface is covered by a layer of articular cartilage in continuity with the articular cartilage of the main body of the patella. On MR images, the gap between the two patellar parts might be filled with bright T2 signal or dark T2 signal, depending on the relative amounts of fibrous or cartilaginous tissue [5]. In patients with anterior knee pain, edema might be present at either side of the opposed bony surfaces, indicating abnormal or dysfunctional motion between the bony moieties (Fig. 3). The patient could be tender to palpation directly over the area.

The dorsal defect of the patella also occurs at the superolateral pole of the patella. Although covered by articular cartilage, the defect is filled not by bone but by a cartilaginous matrix. Identified on plain film as a focal defect in the patella, the MR signal intensity of the defect usually mirrors that of the overlying cartilage. Areas of necrosis and fibrosis have been identified within the defect, which can cause some inhomogeneity of the signal intensity. The overlying cartilage should be closely inspected, as it might fissure and thin. The defect usually heals spontaneously or after surgery and is rarely noted in adults [6].

Patellar-trochlear dysplasia

The morphology of the patella and trochlea is of prime importance in providing a stable and smooth platform for patella motion along the trochlea. An analysis of the shapes of the patella and trochlea is crucial to understanding their healthy interaction and key to predicting future pathology.

The Wiberg classification is the most widely used classification to describe patellar articular morphology. Type

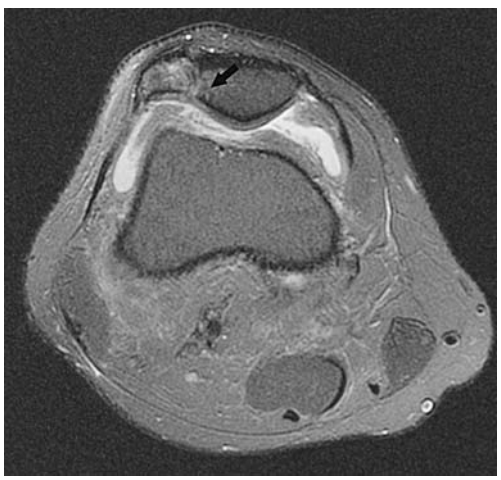

Fig. 3 Axial PD fat-saturated image, 22-year-old man. There is a bipartite patella. There is high signal in the interface between the two patellar parts, suggesting edema or granulation tissue. Increased signal is noted at the articular cartilage along this area, indicating cartilage abnormalities that might include malacia or fibrillation and frank fissuring (*arrow*)

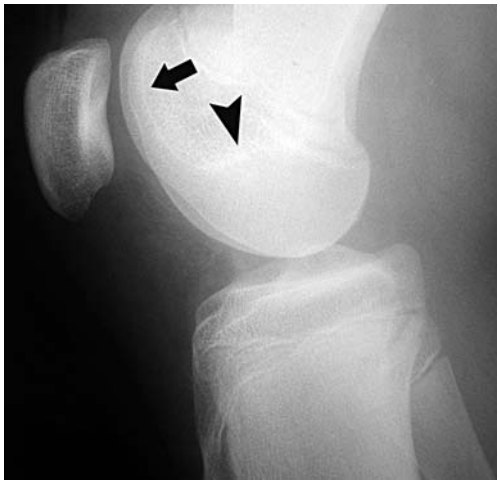

Fig. 4 Lateral radiograph. The roof of the intercondylar notch (*arrowhead*) and the white line indicating the trochlea (*long arrow*) are indicated. The two lines are continuous. The distance from the trochlear surface to the anterior extent of the femoral condyles represents the depth of the trochlea at each point. The top of the trochlea lies very close to the level of the distal femoral physis

1 is a patella with equal medial and lateral facets, both possessing concave articular surfaces. In type 2 patellae the medial facet is slightly smaller than the lateral but retains its articular concavity. Type 3 patellae have small medial facets with convex articular surfaces.

Less well-stressed in the medical literature is the morphology of the trochlea. The word "trochlea" means pulley, but the trochlea of the patellofemoral joint frequently does not provide a deep pulley surface over which the patella can ride. It might be flat or nearly so, which is frequently associated with type 2 or type 3 Wiberg patellae. The findings are apparent on plain film radiographs as well as cross-sectional images.

On standard lateral plain films the trochlear articular surface is identified by a white line deep to the anterior surface of either femoral condyle. Consideration of basic knee anatomy, which might not be immediately obvious, shows that the trochlear surface is continuous with the roof of the intercondylar notch posteriorly. The white and somewhat thick sclerotic line representing the roof of the notch posteriorly, and termed Blumensaat's line, is therefore continuous with the trochlear surface anteriorly (Fig. 4).

Trochlear dysplasia can be identified when the trochlear line lies anterior to the anterior extent of the lateral femoral condyle on a well-positioned lateral view. The finding known as the trochlear crossover sign indicates a dysfunctional situation because at the point at which crossover occurs the trochlea must be flat and will not restrict lateral patellar subluxation [7, 8]. Less-severe forms of trochlea flattening might still be abnormal. A simple nonquantitative gauge of the depth of the trochlea can be obtained by noting the distance from the sclerotic line of the trochlea to the anterior edge of the condyles. If desired, a quantitative assessment of trochlear depth can be obtained by measuring the depth of the trochlea on a standard lateral view approximately 1 cm inferior to the level of the physis, which corresponds to the most superior extent of the trochlear groove. A trochlear depth of 3–5 mm or less (as measured as the average distance from the anterior edge of the medial and anterior edge of the lateral femoral condyles to the white line indicating the trochlea) is associated with patellar instability because of an inappropriately shallow trochlea. A small nipple-like excrescence at the anterior superior margin of the trochlea might develop and is further evidence of trochlear dysplasia and patellar tracking abnormality [9] (Fig. 5).

Both findings are immediately evident on sagittal MR and MDCT images. Direct assessment of the trochlea and patella is easily performed with the use of axial sequences. The level for analysis should be carefully chosen, as the trochlea can appear spuriously flat; a point 3 cm above the weight-bearing surface of the condyles is an appropriate level [10]. On MR, midsagittal images might show a sharp step-like transition from the anterior femoral cortex to the trochlear articulating surface and that is indicative of trochlear dysplasia. The nipple-like excrescence seen on radiographs is well seen on non-fluid-sensitive images. On

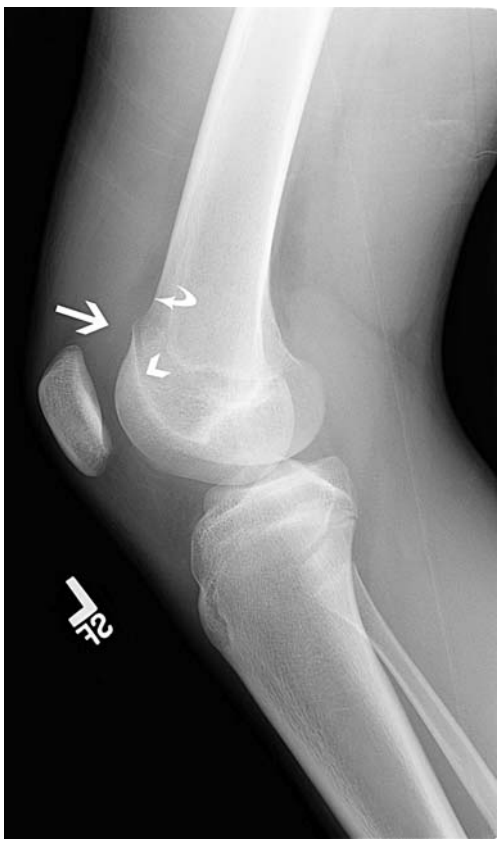

Fig. 5 Lateral radiograph in a 15-year-old boy. A positive trochlear crossover sign is present, with the white line representing the deepest part of the trochlea (*arrowhead*) crossing the anterior edge of the femoral condyles (*curved arrow*). A small excrescence is seen at the top of the trochlea, evidence of patellar trochlear dysplasia (*arrow*)

T2-W fat-saturated images the excrescence might merge in signal with the dark cartilaginous signal of the trochlea and might not be immediately obvious (Fig. 6).

Chronic patellar tracking abnormalities can cause several varied syndromes with either acute or chronic onset. It should be noted that because the pathological mechanisms causing these syndromes is shared among them, the resultant symptomatology and imaging findings have a certain amount of crossover.

Patellofemoral pain syndrome

This is an umbrella term to describe multiple causes of anterior pain associated with the patellofemoral articulation. These include impingement of the infrapatellar fat body (Hoffa's fat pad), plica syndrome, excessive lateral pressure syndrome, excessive lateral tension syndrome, and chondromalacia patellae. Common to many of these are chronic patellar tracking abnormalities.

Chondromalacia patellae

Although this is a problem more in the adult population, its nascence lies in childhood and is in many cases the result of chronic patellar tracking abnormalities. It is the result of a multivariable set of circumstances and as such should be construed not as a disease in itself but rather the manifestation of other disease processes. The findings centrally relate to softening of the patellar articular cartilage as the name implies, and progress to fissuring, subchondral cyst formation and ultimately articular cartilage loss and frank osteoarthritis. Chondromalacia patellae is an arthroscopic diagnosis and MR imaging findings of patellar articular cartilage heterogeneity in the absence of the more advanced stages of fissuring and cartilage loss can only suggest the diagnosis. Even so, MR imaging holds the greatest promise for advanced and noninvasive cartilage imaging. Although chondromalacia is not diagnosable on standard MR images, the factors that contribute to it are, and, as such, examination of the morphology of the patellofemoral joint should be performed on each MR examination of the knee.

Fat-pad impingement

The patella and patellar tendon must slide through an arc that includes some contact with Hoffa's fat pad. As the fat pad serves partially as a shock absorber, injuries to Hoffa's fat pad can be acute or chronic, primary or secondary. Acute injuries might occur because of direct impact on the patella with the patella compressing Hoffa's fat pad posteriorly on the trochlea. Alternatively, the fat pad might be injured during an internal derangement as the lateral tibial plateau, lateral femoral condyle or patella acutely compress the fat pad.

Chronically, fat pad impingement is the result of excessive lateral forces such as those observed in concert with abnormalities of patellar trochlear morphology or thickening of the lateral restraining structures [11] (Fig. 7). In addition, edema at the proximal fat pad might be related to chronic or acute avulsions of the inferior patellar pole. Increased signal in the fat pad inferiorly is most commonly related to chronic avulsion of the patellar tendon from its insertion on the tibial tuberosity.

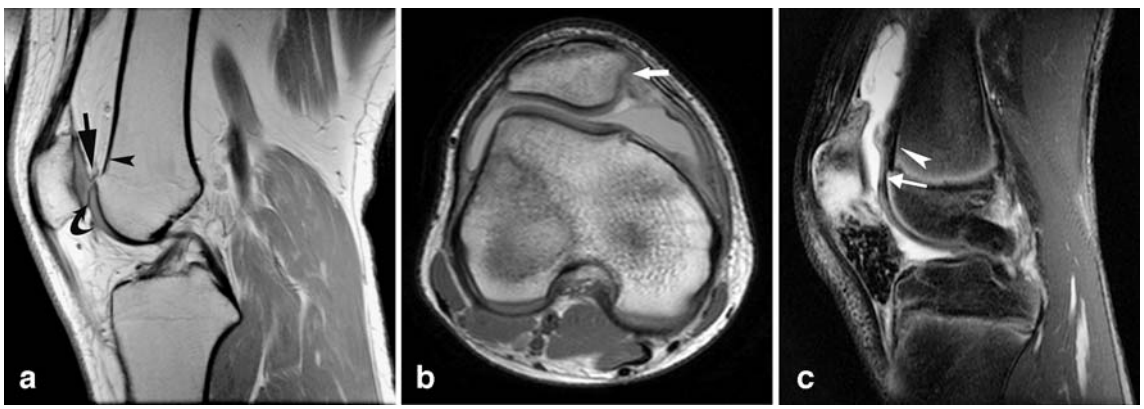

Fig. 6 MR images in a 16-year-old girl. **a** Sagittal PD image. There is a step-like transition from the anterior femoral cortex (*arrowhead*) to the trochlear articulating surface (*curved arrow*). Note the normal smooth transition seen in Fig. 2. The nipple-like excrescence at the anterosuperior margin of the trochlea (*arrow*) is well seen and is firm evidence of patellar trochlear dysplasia in this girl with a prior subluxation. **b** Axial PD image 3 cm above the joint line. The trochlea is flat and slopes laterally. Analysis of the image shows a steep medial patellar facet (*arrow*), which could not provide a good surface for articulation. **c** Sagittal fluid-sensitive image. There is edema in the patella caused by a patellar subluxation. Note that the anterior cartilaginous margin of the trochlea lies anterior to the anterior femoral cortex. There is a step-like transition from the anterior femoral cortex (*arrowhead*) to the trochlear articulating surface (*arrow*)

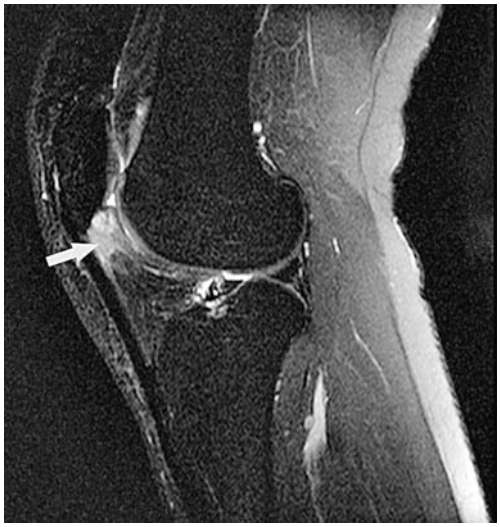

Fig. 7 Sagittal inversion recovery image. Edema is present in Hoffa's fat pad immediately inferior to the patella (*arrow*). This is a good example of a fat-pad impingement and was present without any other acute abnormalities

Excessive lateral tension syndrome, excessive lateral pressure syndrome

Both of these conditions relate to excessive lateral forces at the patellofemoral joint. In the excessive lateral pressure syndrome, pressure on the lateral patellar articular facet causes early cartilage loss at both the lateral patellar facet and the lateral aspect of the trochlea. The cause is unclear, but is seen in concert with abnormal tilting of the patella and probably relates to either diminished medial restraining forces or abnormalities of the shape of the articular surface of the patella or trochlea. It should be noted that as the lateral facet is involved, this differs from the classic chronic patellofemoral osteoarthritis, which occurs predominantly medially especially at the odd facet. A related syndrome is the excessive lateral tension syndrome that emphasizes progressive fibrosis of the lateral patellar retinaculum leading to thickening and progressive retinaculum shortening and cartilage abnormalities at the lateral patellar facet (Fig. 8). It is important to note that both the excessive lateral pressure and the excessive lateral tension syndromes have been described in the literature only in the adult population. Signs of articular cartilage injury, especially, might not manifest in the pediatric population. Little is known as to the nascence of this condition or its early appearance. It may be that thickening of the lateral patellar retinaculum precedes the articular cartilage injury and that thickening of the retinaculum may be the only apparent abnormality in the adolescent with pain.

Plica syndrome

The medial patellar plica is a thin fold of tissue, a remnant of fetal synovium, that runs from the medial wall of the knee joint superior to the patella and courses obliquely downward to an insertion on the synovium covering the infrapatellar fat pad. When irritated, it can become thickened and inelastic. The constant rubbing of the plica can cause erosive disease of the patellar articular cartilage. It is frequently palpable as a tender band medial to the patella. On MR images it is identifiable as a low-attenuation band of tissue posteromedial to the patella of varying thickness. Normal asymptomatic plica measure approximately 1–2 mm but when symptomatic can reach up to 1 cm in breadth. Even so, this situation is distinctly unusual and in general the agreement between MR imaging and actual symptomatology is poor [12].

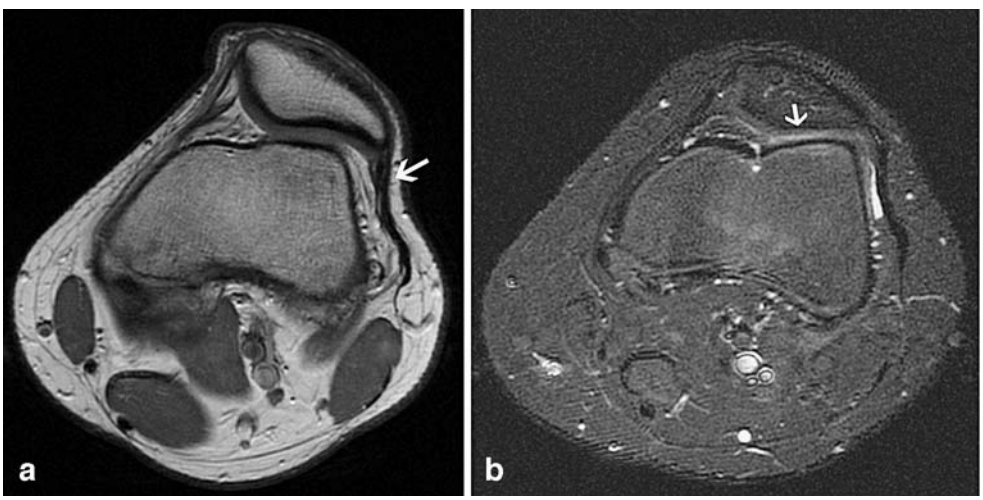

Fig. 8 Chondromalacia. **a** Axial PD image. The lateral patellar retinaculum (*arrow*) is markedly thickened and the patella is subluxed laterally, an example of the excessive lateral tension syndrome. **b** Axial T2-W fat-saturated image in the same patient shows well increased signal in the lateral patellar facet articular cartilage, suggesting chondromalacia (*arrow*), which was confirmed at surgery

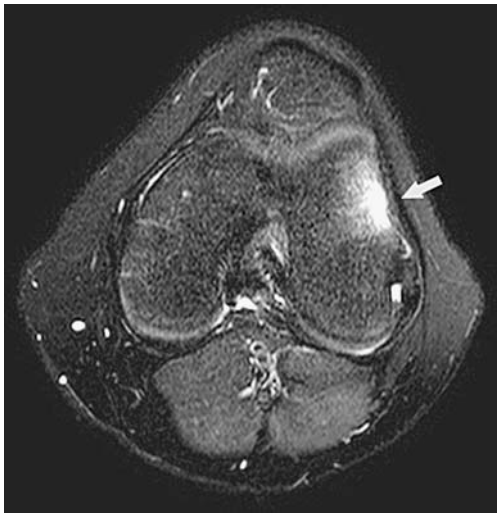

Fig. 9 Axial fluid-sensitive image in a 16-year-old girl. Note the focal edema at the anterolateral femoral condyle. There is also focal flattening of the anterolateral surface of the condyle caused by the impaction of the patella on this area (*arrow*) sustained during a patellar subluxation

Acute patellar subluxation

As patellar trochlear dysplasia is common and can be considered a developmental or congenital abnormality, it should not be surprising that patellar dislocation is a frequent occurrence in the adolescent. As the adolescent begins to be more active in sports and is able to exert greater force on an inherently unstable joint, he or she is predisposed to patellar dislocation.

The patient will complain of an acute onset of pain and might recognize the patella to be laterally dislocated. Most commonly, the dislocation reduces itself when the leg is straightened. The sequence of subluxation followed by reduction yields a characteristic MR pattern of edema and osteochondral defects that allows an accurate reconstruction of the mechanism of the internal derangement. The injury always occurs with flexion. Initially the patella subluxates laterally. Edema at the medial patellar pole is related to this phase as the medial patellar restraints are avulsed from their attachment. As the patella moves laterally the patella apex or medial facet impacts the trochlea or lateral femoral condylar articular surface, causing osteochondral defects on both the femoral and the patella side of the joints. Now with the patella dislocated, the patient reflexively extends the knee and reduces the patella to normal position. As the patella moves back medially it again impacts the femur. However, with the trochlea concavity shielding the articular surface of the femur, the patella impacts the femur along the anterolateral surface. Because this segment of the internal derangement occurs with the knee in extension, the edema on the anterolateral femur is just anterior to the osteochondral defect on the femur. The edema of the anterolateral femoral condyle is clearly anterior to that seen with anterior cruciate injuries during an anterolateral rotary instability injury, and that observation is a very helpful signpost to a complete and accurate interpretation of the MR examination (Fig. 9). Note that although the osteochondral injuries vary and might not be present on MR images, the edema pattern is a fairly constant feature on MR images of a patellar subluxation.

A careful analysis of the medial patellar passive restraints is of great importance to appropriate surgical therapy. In the overwhelming majority of cases there is rupture of some or all of the medial patellar restraining structures and a detailed description of these injuries might help guide the orthopedic surgeon to reconfigure the extensor apparatus to prevent a recurrence. Restraints to patellar subluxation are constructed of an apron of retinacular fibers and ligaments that hold the patella in place. The exact arrangement is very complex and is multilayered at multiple levels. Practically, however, the

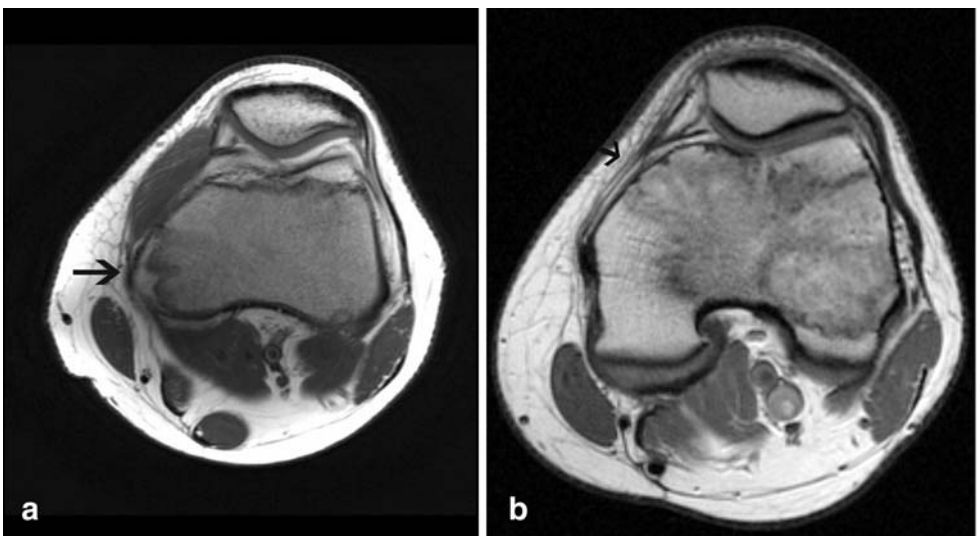

Fig. 10 Normal MPFL. **a** Axial PD image. The normal MPFL is seen emerging from the vastus medialis and attaching to the femur at the level of the adductor insertion (*arrow*). **b** The normal MPR complex is shown here as a double strand of dark signal running from the medial patellar pole to the superficial portion of the medial collateral ligament (*arrow*)

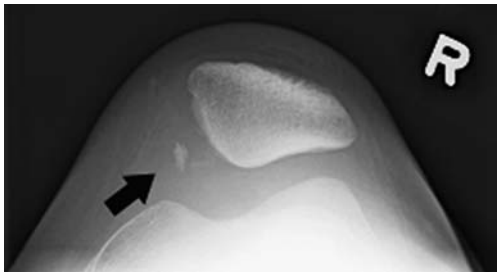

Fig. 11 Merchant view. A fragment of bone has been avulsed from the medial patellar pole (*arrow*) during an acute patellar subluxation

restraints can be divided into two functional groups with MR correlates. The most important restraint is called the medial patellofemoral ligament (MPFL). It is attached to the superior half of the medial patellar surface and inserts on the femur near the adductor tubercle intimate with the femoral attachment of the superficial medial collateral ligament. More inferiorly a group of fibers arises from the lower half of the patella and extends to multiple insertions on the medial meniscus, the tibia and the medial collateral ligament. This group taken as a unit can be called the medial patellar retinaculum (MPR).

Radiographically, in the acute setting a joint effusion is usually present. A fat fluid level might also be seen in a cross table lateral or axial view. The patellar tendon is frequently indistinct. The patella might be elevated (patella alta). An ossific fragment of varied size might be present, particularly on a Merchant view at the medial patellar pole. The fragment might have been avulsed from the medial patellar pole or it might have resulted from an osteochondral fracture sustained as the patella subluxed. Practically, on MR images, the MPFL can usually be identified at both its patellar origin and its femoral insertion using the insertion of the adductor tendon on its tubercle as a guide. The MPFL fibers can be seen emerging from the deep and superficial surfaces of the vastus as a fairly well-defined linear structure. The MPR is seen as a double- or triple-stranded dark signal structure arcing between the patella and the medial collateral ligament (Fig. 10).

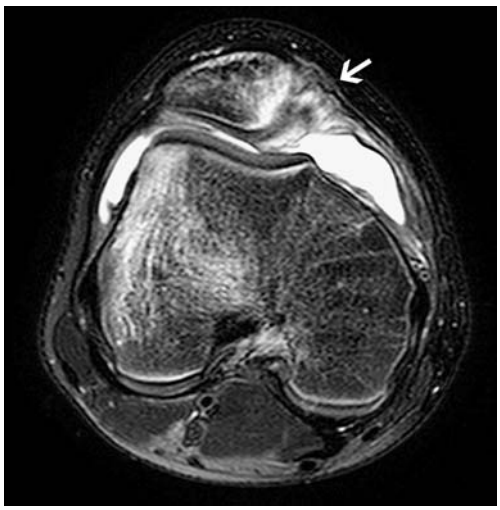

Fig. 12 Axial PD fat-saturated image. There is edema in characteristic locations for a patellar dislocation at the medial patellar pole and the anterolateral femoral condyle. The MPR is avulsed from its patellar attachment and its fibers have a wavy and poorly discernable course (*arrow*)

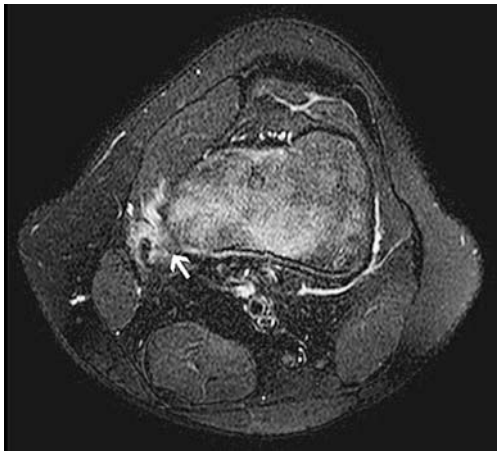

Fig. 13 Axial PD fat-saturated image. There is edema about the adductor tendon and vastus medialis. No MPFL is seen. Instead, edema fills the gap (*arrow*) and demonstrates a torn MPFL

Several key locations should be inspected for areas of discontinuity of the MPR and MPFL [13, 14]. First, the origin of these structures from the patella should be identified and

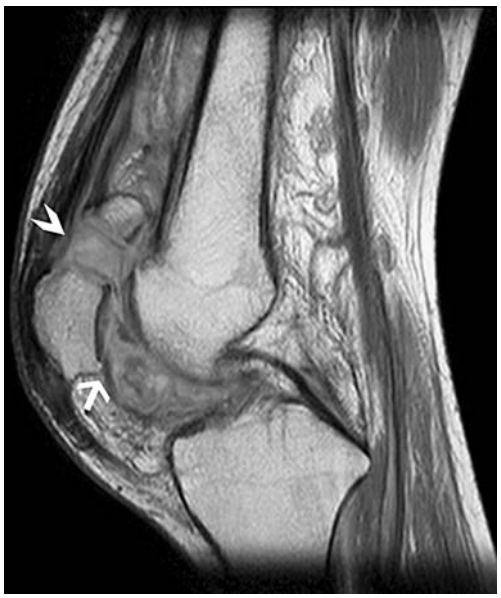

Fig. 14 Sagittal PD image in a 16-year-old boy. The patella has fractured (*arrowhead*) and the superior pole is displaced upward on one layer of the quadriceps tendon. More superficial layers of the quadriceps tendon remain attached to the distal fragment. The boy has hemophilia and has had multiple intraarticular hemorrhages, as evidenced by the synovial thickening and early degenerative changes, particularly at the inferior pole of the patella (*arrow*)

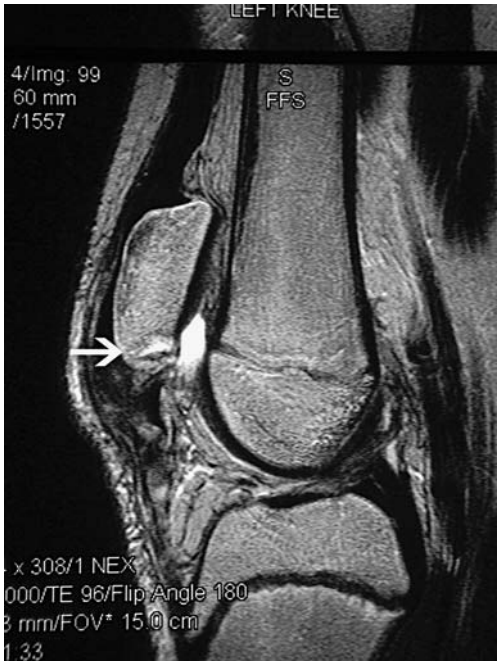

Fig. 15 Sagittal PD image in a 12-year-old boy. The inferior patellar pole has fractured, allowing fluid to extend between the superior and inferior fragments (*arrow*)

clearly visualized. There should be no bare areas on the medial aspect of the patella and areas of focal patellar edema deserve particular attention. An avulsed fragment of bone of varied size might be present (Fig. 11). As is the case with osteochondral injuries, the fragment containing fatty marrow might be obscured by the fat of Hoffa's pad and might show nearly complete saturation on fat-saturated or suppressed sequences and, in fact, the larger fragment might be more difficult to see. The attachments of the MPFL and the MPR on the patella should be carefully scrutinized. The fibers should attach cleanly on the patella. A wrinkled MPR with no clear attachment is torn (Fig. 12).

The insertion of the MPFL near the adductor tubercle is a very frequent location of rupture and in some reports it is the most frequent location (Fig. 13). The ligament as it emerges from the vastus should be followed to its insertion. Edema in the vastus itself is a clear indication that the rupture involves the MPFL, as the two structures are intimately related and indeed the MPFL is in large part derived from the components of the vastus medialis and medialis obliquus. In addition to the above two zones, rupture frequently occurs in a zigzag pattern running through the apron of retinacular fibers close to the insertion of the tibial collateral ligament. This path can frequently be visualized on successive transverse images. At times the MPR might be severely attenuated but intact, probably indicative of a chronic pattern of subluxation. Frequently, multiple facets of the patellar maltracking syndrome are present, including patellar-trochlear dysplasia, fat-pad impingement and thickening of the lateral patellar retinaculum indicative of the excessive lateral tension syndrome.

Fractures

Fractures of the patella are most commonly caused by direct impact. The patella most commonly is cleaved into upper and lower halves. Osteochondral defects at the patellar or opposing trochlear articular surface can also be the result of direct impacts. These cartilaginous injuries are well-diagnosed by MR imaging. Three-dimensional fat-saturated gradient sequences (GE 3D SPGR) portray cartilaginous injury well and have the advantage of multi-planar reconstructions [15], albeit with some loss of detail. More powerful 3-D imaging sequences are on the horizon that should be truly isotropic.

In addition to fractures of the normal patella, a patella weakened by osteopenia and chronic synovial inflammation can fracture with surprisingly little trauma (Fig. 14).

Before physeal closure, avulsions of tendon from the inferior patellar pole most commonly avulse the epiphyseal cartilage so that such an injury is a Salter-type fracture. The avulsion might go unrecognized until ossification of the avulsed cartilaginous fragments occurs. Such an injury is called a patellar sleeve fracture, as the fracture removes a sleeve of inferior patellar epiphyseal cartilage (Fig. 15). The injury pattern is not surprising when considering that before skeletal maturation physeal fractures rather than ligament or tendon ruptures are the rule rather than the exception.

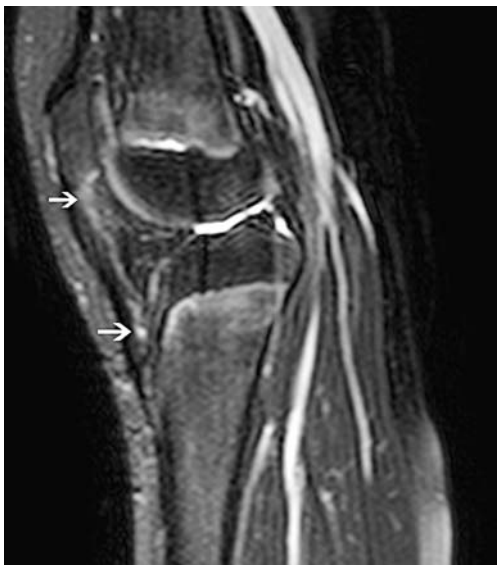

Fig. 16 Sagittal fluid-sensitive image in a 14-year-old boy. Edema is noted at the inferior patellar pole and the insertion of the patellar tendon at the tibial tuberosity (*arrows*). The boy was tender in both locations and was diagnosed with both Sinding-Larsen-Johansson and Osgood-Schlatter diseases

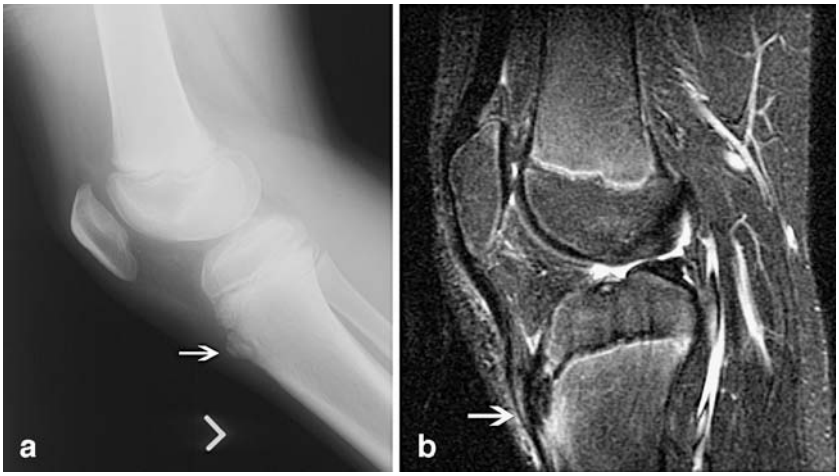

Fig. 17 Osgood-Schlatter disease. **a** The tibial tuberosity is enlarged and shows fragmentation (*arrow*). **b** Sagittal T2-W fat-saturated image. There is a mixture of bright and dark signal at the insertion of the patellar tendon (*arrow*), indicating the chronic avulsive context of Osgood-Schlatter disease

Sinding-Larsen-Johansson and Osgood-Schlatter diseases

Just as the inferior patellar pole can fracture, which in children causes a patellar sleeve fracture, as the physis matures chronic traction injuries can occur. Those injuries at the inferior patellar pole are called Sinding-Larsen-Johansson disease, or jumper's knee, and those involving the patellar tendon attachment at the tibial tuberosity are called Osgood-Schlatter disease. The two diseases are essentially the same process and pathology on both ends of the patellar tendon and can coexist (Fig. 16).

At the inferior pole of the patella, chronic avulsive traction can cause fragmentation of the patellar pole. Radiographically, multiple small ossific densities can be seen adjacent to the patella. In more acute cases the inferior pole cortex might be indistinct and rarefied. Hoffa's fat pad might be infiltrated with edema and the proximal patellar tendon thickened with a blurred demarcation between tendon and adjacent fat. Jumper's knee is the exact same disease in older athletes. On MR images, bright T2 signal is seen in and adjacent to the inferior patellar pole with associated changes in Hoffa's fat pad and the tendon. The fragments might be incorporated into the patella with a fairly normal appearance over time.

At the distal attachment of the patellar tendon on the tibial tuberosity, the same process yields a disease with the eponymous name Osgood-Schlatter disease. Here, as in the proximal tendon, chronic traction causes fragmentation of the tibial tuberosity, likely because of multiple small avulsions. Tenderness over the tibial tuberosity, especially on deep knee flexion, is a hallmark of the disease. Imaging is exactly analogous to changes at the inferior patellar pole in Sinding-Larsen-Johansson disease, with small avulsed

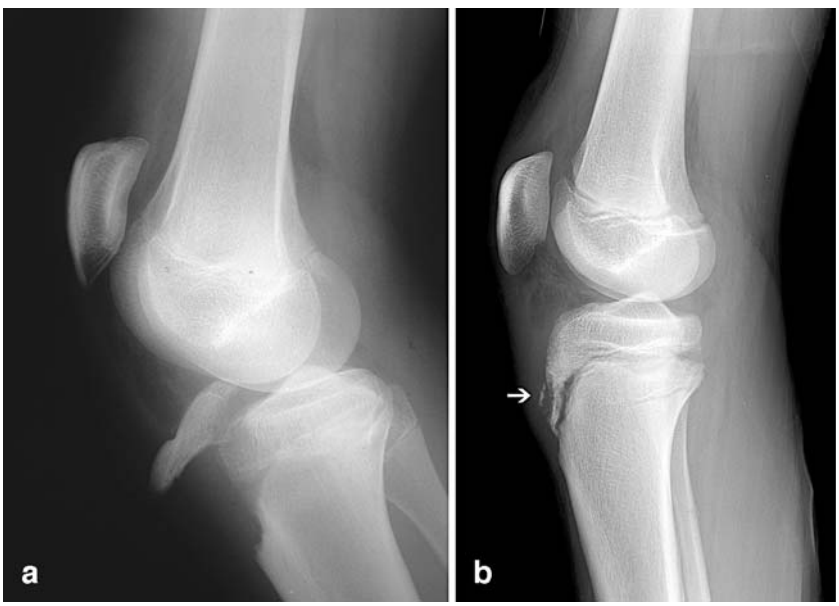

Fig. 18 Fractures in Osgood-Schlatter disease. **a** Lateral radiograph. The entire tibial tuberosity has fractured and is lifted superiorly. **b** In a different patient, there is a Salter 2 fracture of the proximal tibia. There are also chronic findings of Osgood-Schlatter disease with some fragmentation of the tibial tuberosity (*arrow*)

osseous fragments, blunting of Hoffa's fat pad, and thickening and poor demarcation of the tendon. On MR images, edema and thickening of the patellar tendon near its insertion can be seen. Edema of Hoffa's fat pad is noted inferiorly. The tibial tuberosity is enlarged with avulsed osseous fragments and a mixture of dark and bright signal on T2, indicative of the chronic nature of the disease (Fig. 17).

Although Osgood-Schlatter disease is usually considered a chronic repetitive injury, acute avulsions of the tibial tuberosity not only occur but might not be limited to small cartilaginous or osseous detachments. Large fragments can be torn off (Fig. 18). The entire proximal tibial epiphysis can detach.

The proximal tibial epiphysis and the tibial tuberosity share in common a continuous run of epiphyseal cartilage such that fracture planes might ramify along that cartilage boundary to affect both tibial tuberosity and proximal tibia. The treatment of these injuries is complex because of the differing functional anatomy at the two sites. Specifically, the patellar tendon has a long attachment that includes both the apophysis of the tibial tuberosity and the tibia proper.

Interestingly, proximal tibial Salter fractures are frequently preceded by Osgood-Schlatter-type abnormalities. Whether the chronic repetitive injury weakens the proximal tibial epiphysis until fracture occurs or the acute fracture as well as the chronic tuberosity injury is simply a manifestation of the magnitude and type of forces being exerted is unknown.

Conclusion

The patellar extensor apparatus is but one link in a long extensor chain. Injuries can be the result of excessive tractional forces such as in Osgood-Schlatter disease or abnormalities of patellar tracking such as in many instances of patellar subluxation. Whatever the cause, the morphology of the patellotrochlear joint and the geometry of the tendons that span it cause an unusual pattern of injury in children and especially in adolescents. These injuries and the morphological abnormalities that relate to them contribute to chronic degenerative disease in the adult. As our understanding of chronic articular disease advances, it becomes clearer that the pediatric radiologist must interpret images with an understanding that current morphology has an important effect on severe degenerative disease in the adult.

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