Pediatric and Adolescent Musculoskeletal MRI

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A Case-Based Approach

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Foreword

The number of new radiology texts that appears each year continues to grow, and each must compete with other available works to be successful. With this in mind, Drs. Herman Kan and Paul Kleinman have authored a book, *Pediatric and Adolescent Musculoskeletal MRI: A Case-Based Approach,* that will clearly prove a very useful addition to the literature. What sets it apart from other texts is its organization, its clinical utility, and, above all else, its readability.

The case-based organization is very user friendly. More than 100 cases dealing with the most important musculoskeletal conditions that affect children and adolescents are presented. Osseous, articular, and soft tissue disorders are covered, including neoplastic, traumatic, infectious, dysplastic, and vascular conditions. In fact, a survey of the cases indicates that virtually all of the important musculoskeletal disorders affecting the immature skeleton are included, such that upon reviewing the entire text, the reader is exposed to the imaging findings of all disorders that he or she probably will encounter in clinical practice.

Each case stands alone as a pragmatic review of the condition being covered. Initially, a history along with one or more appropriate images, including MRIs, is presented. The reader is able to survey these images and arrive at a diagnosis, thus testing his or her diagnostic acumen. Subsequently, the imaging findings are summarized and the correct diagnosis is given. This section is then followed by one or more questions about the entity being presented, followed by a focused discussion (appropriate in length) of this condition and any others that might have been considered. Finally, a section dealing with an orthopedic perspective of the entity, a list of what the clinician needs to know, and answers to the aforementioned questions follow. Completing the case are images of additional examples of the disorder and related conditions, a discussion of the findings and of pitfalls and pearls, and appropriate references. In this fashion, the organization of each case is superb and without fault, and the illustrations are of excellent quality with well-placed arrows.

Currently available texts dealing with musculoskeletal MR imaging are confined to a discussion of disorders affecting the adult skeleton. Thus, a book dealing with advanced imaging of those disorders that affect the immature skeleton, some of which are confined to children and adolescents, is welcome indeed. This text clearly fills a void and, further, presents material in such a clear and concise way that it is easy to remember. It presents practical material and is fun to read. There are not many books that actually lead to enjoyable reading!

I commend the authors for fulfilling their goals, and I encourage radiologists, orthopedists, and others involved in diagnosing and treating musculoskeletal disorders in children and adolescents to purchase this book, to consult it often, and even to read it in its entirety! I am honored, indeed, to have an opportunity to write this Foreword.

Donald Resnick, MD
Professor of Radiology
University of California, San Diego

Preface

MRI has transformed the field of pediatric and adolescent musculoskeletal imaging. When the more senior (and gray haired) of the two authors completed his pediatric radiology training, orthopedic radiology was a primarily plain film based discipline, occasionally supplemented by arthrography. Although much could be gleaned from the humble radiograph regarding the nature of orthopedic disorders, MRI has provided elegant depictions and insights of classic pediatric entities that would surely amaze the likes of John Caffey and Edward Neuhauser. With this technique, new challenges have arisen to comprehend the imaging findings in these classic disorders, and a wide array of newly appreciated entities has emerged with the wide utilization of MRI by pediatric, orthopedic and sports medicine specialists.

Despite these developments, a textbook devoted to MRI of pediatric and adolescent musculoskeletal diseases has been unavailable. Those with an interest in this area have had to rely upon published articles, as well as orthopedic, musculoskeletal and pediatric radiology texts. The goal of this work is to bring the literature of musculoskeletal MRI in children and adolescents together in an authoritative, but user friendly format. Cases are presented as "unknowns" in an effort to provide a dynamic learning process. The reader is given a brief history and initial images. A description of findings with appropriate annotated images and supplementary images follows. The diagnosis is then revealed and a discussion ensues. The discussion attempts to cover the salient features of the entity with related cases where appropriate. A differential diagnosis is given and, where appropriate, additional examples are illustrated. The result is a text that contains 315 pediatric and adolescent musculoskeletal MRI cases presented within the context of 102 unknowns.

To place this material more squarely in a clinical context, the authors invited two clinicians, Mininder Kocher, MD, MPH, a pediatric sports medicine orthopedic surgeon, and Mark Gebhardt, MD, a pediatric orthopedic oncologist, to join the effort. Sections entitled "Orthopedist's Perspective" and "What the Clinician Needs to Know" are provided to inform the radiologist about the important clinical issues and what information is required to plan a management strategy. A modest bibliography for each case guides the reader to further discussions in original articles, reviews and other texts. The authors hope that this unique combination of both the radiologic and orthopedic points of view will enrich the readers' learning experience and provide useful relevant information for the referring clinician.

Although the authors have sought to provide a solid and current presentation of both common and, where appropriate, unusual entities, space considerations have required exclusion of other entities. Like most first efforts, it is likely that this book will grow in scope and will undergo refinements in future editions, but for the present, we hope that this will be a useful instructional tool and reference source for radiologists and clinicians interested in pediatric and adolescent musculoskeletal disorders.

J. Herman Kan, MD
Paul K. Kleinman, MD

Acknowledgments

The authors would like to thank Susan A. Connolly, MD, staff radiologist, and Ilse Castro-Aragon, MD, pediatric radiology fellow at Children's Hospital Boston for their useful comments and criticisms during the preparation of this text. In addition, we would like to thank all the radiologists and clinicians of Children's Hospital Boston for referring us these interesting cases. Not least of all, we would like to thank Susan Ivey and Marllely Dewitz for their assistance in preparation of the manuscript. Rob Albano of Springer Science+Business Media has been behind this effort from its inception, and Merry Post has done a thorough and conscientious job of providing editorial support and guidance.

We would also like to graciously acknowledge the following radiology departments and hospitals for allowing us to publish useful images from their institutions:

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Children's Hospital Boston, Boston, MA
Brigham and Woman's Hospital, Boston, MA
Boston Medical Center, Boston, MA
Down East Community Hospital, Machias, ME
Falmouth Hospital, Falmouth, MA
Hospital Episcopal San Lucas, Ponce, Puerto Rico
Portsmouth Regional Hospital, Portsmouth, NE
Metrowest Medical Center, Framingham, MA
Morton Hospital and Medical Center, Tautum, MA
Massachusetts General Hospital, Boston, MA
Newton-Wellesley Hospital, Newton, MA
Rhode Island Hospital, Providence, RI
Shields Medical Center, Cape Cod, MA
Southern New Hampshire Medical Center, Nashua, NH
Vanderbilt Children's Hospital, Nashville, TN
West Suburban Imaging Center, Boston, MA

Dr. Kan would like to thank his wife, Shin-Mei, for her understanding and support. She provided unconditional encouragement and companionship as he worked on this text at various coffee shops all over Boston, evenings and weekends. He would also like to thank Paul Kleinman, MD, for his wisdom, patience, and mentorship during this incredible project.

Dr. Kleinman would like to thank his wife and colleague, Patricia, for her support and perseverance during the many evenings and weekends spent on this text. He offers special thanks to his coauthor, Herman Kan, MD, for his vision, industry, and willingness to accept guidance and criticism throughout the course of this challenging project.

Contents

Case 1

History

This is a 13-year-old boy who fell two days ago and has had persistent pain and swelling in his right knee. Radiographs of his right knee (not shown) demonstrated a large joint effusion only.

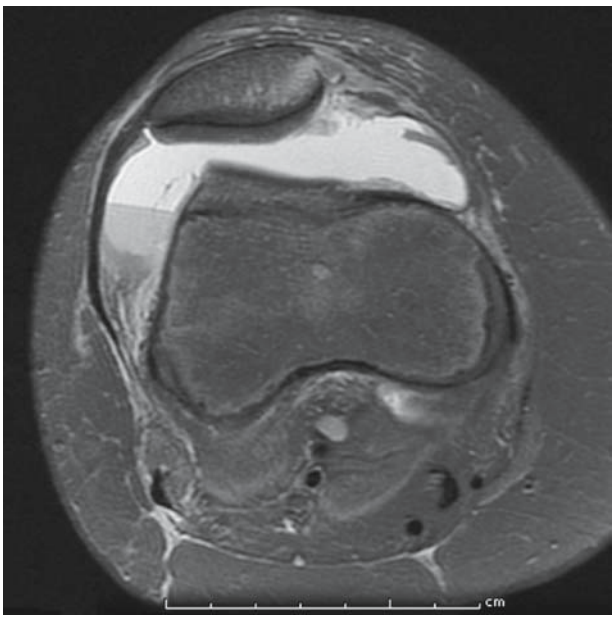

Figure 1A. Axial PD FS of the right knee.

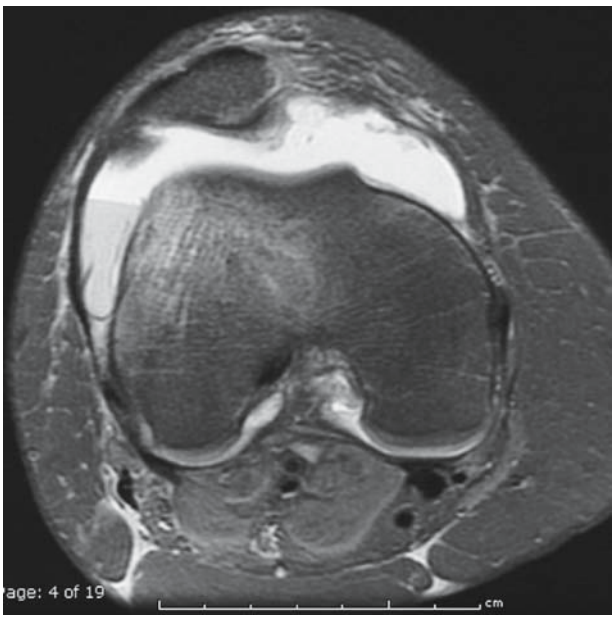

Figure 1B. Axial PD FS.

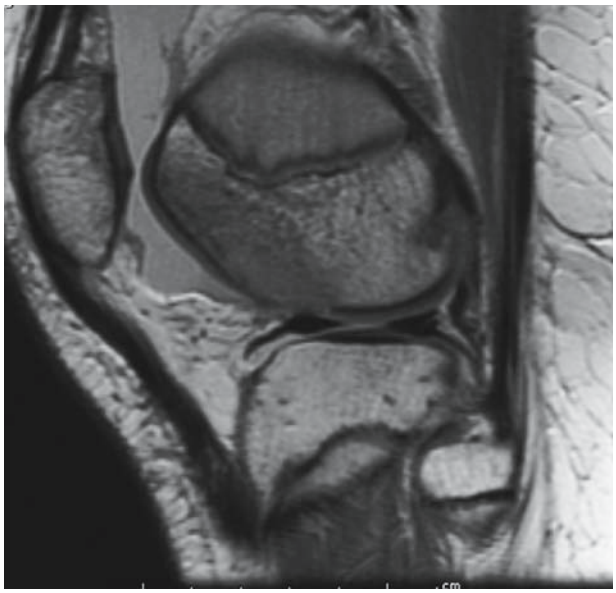

Figure 1C. Sagittal PD sections through the lateral femoral condyle.

Figures 1A (1A with annotations). There is a large joint effusion with fluid-fluid levels. Linear increased SI is present along the medial patella pole **(thin arrow)**. There is also edema within the medial retinaculum (*). A medial pole osteochondral fracture is present **(black arrowhead)**.

Figures 1B, 1C (1C with annotations). Edema is present within the anterior aspect of the lateral femoral condyle. An osteochondral fracture is better delineated on the sagittal view through the lateral femoral condyle **(thick arrows)**.

Figure 1D. A femoral condylar osteochondral loose fragment **(thick arrow)**, which originated from the donor site (Figure 1C with annotations), is identified. No internal derangement of the menisci, cruciate, or collateral ligaments was evident.

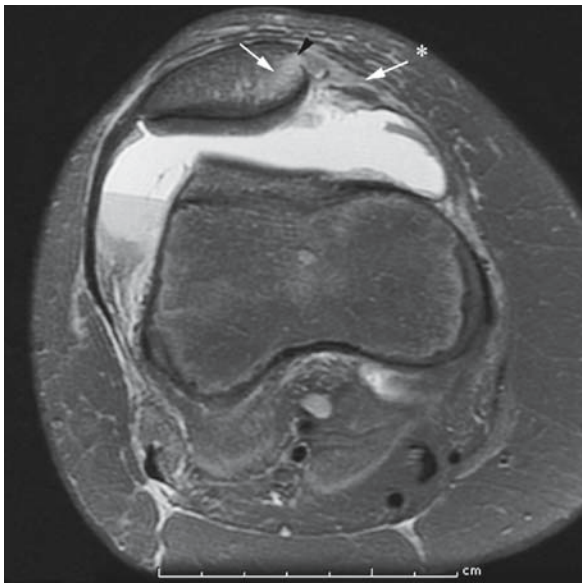

Figure 1A* Annotated.

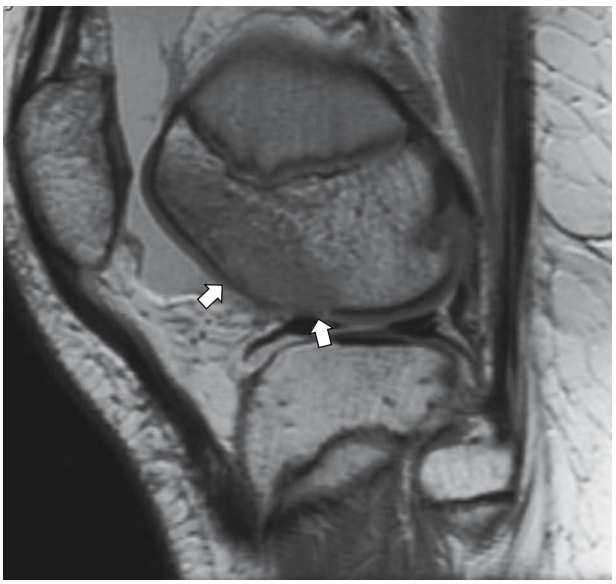

Figure 1C* Annotated.

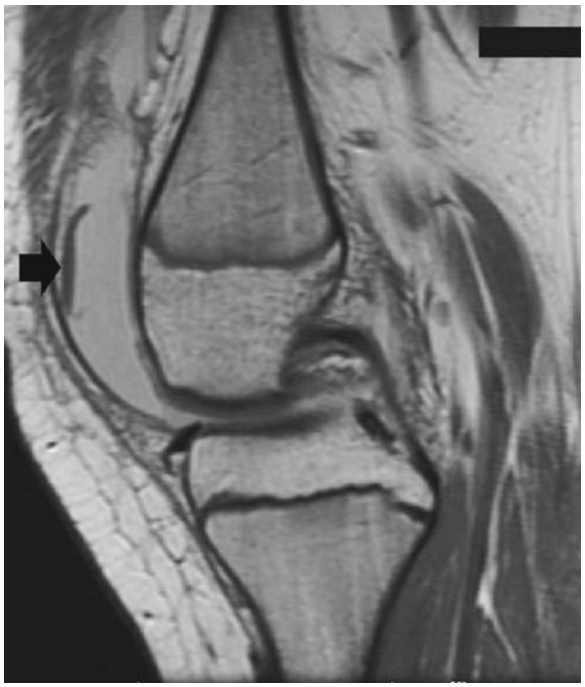

Figure 1D. Sagittal PD.

Diagnosis

Lateral patellar dislocation with displaced osteochondral fracture

Questions

1. What are two mechanisms leading to lateral patellar dislocation?
2. What pattern of bone bruising is seen with lateral patellar dislocation?

Discussion

Lateral patellar dislocation may occur in the setting of trauma with or without underlying patellofemoral dysplasia. The typical age of presentation is between 14 to 20 years with no gender predilection (1). A direct, medial blow or a valgus, twisting (external tibia rotation) injury while the knee is partly flexed are two traumatic mechanisms that may lead to lateral patella dislocation (Answer to Question 1). Anatomic causes that may predispose a patient to patellofemoral instability include patella alta (ratio of the patella tendon to the maximal diagonal length of the patella bone >1.2), a shallow femoral sulcus with hypoplasia of the femoral condyles (normal sulcus angles range from 134 to 155 degrees on a Merchant view), increased lateral patellar tilt, excessive knee valgus, increased femoral anteversion, increased Q-angle, and an abnormal extensor mechanism (2). In severe cases of patellofemoral dysplasia, the trochlear sulcus can even be convex. Causes of congenital patellar dislocation include, but are not limited to: Down's syndrome, Larsen's syndrome, Nail-Patella syndrome, diastrophic dysplasia, and arthrogryposis (3).

Osteochondral fractures and medial patella sleeve avulsion fractures are common occurrences in the setting of acute lateral patellar dislocation in children. In a study of 72 children with acute lateral patellar dislocation, 39% had fractures that were equally divided between osteochondral and medial capsular avulsion fractures (4). In another study, osteochondral injuries proven by arthroscopy were seen in 72% of cases of acute lateral dislocation in patients 12 to 19 years old (5). Osteochondral fractures and bone contusions usually occur when the patella reduces after lateral dislocation. Kissing bone contusions and osteochondral fractures may occur along the anterolateral femoral condyle, medial facet of the patella, and patella eminence (Figures 1E, 1F) (Answer to Question 2).

Medial patella sleeve avulsion fractures from lateral patellar dislocation preferentially occur in children because the patella is not completely ossified. The medial chondro-osseous junction is considered a relatively weak unit of the ligamentous-medial patella retinacular unit in children. The avulsion fragment may be missed entirely or underestimated on plain radiographs because the cartilaginous component is radiolucent. The medial retinaculum may also partially tear in the setting of medial patella sleeve avulsion fractures but usually does not completely tear. A complete medial retinacular tear should only be considered when no hypointense fibers are present and there are indirect signs of disruption such as lateral patella subluxation and/or a redundant and wavy medial retinaculum.

Intra-articular patellar dislocation is an entity that uniquely occurs in children (6). Intra-articular dislocation is when the patella dislocates and rests within the intercondylar notch. Intra-articular dislocations usually result from a quadriceps tendon tear at the chondro-osseous junction of the superior pole of the patella. The inferior pole of the patella is less commonly involved.

The objective of MRI in the assessment of acute lateral patellar dislocation is to suggest the diagnosis when it is unsuspected, to assess for surgical indications such as

osteochondral fragments, and to assess for internal derangement. Additional features that should be assessed in patients with recurrent lateral patellar dislocation include the degree of patellofemoral dysplasia and the presence or absence of premature degenerative changes.

Surgical intervention was performed in this patient because of the presence of an osteochondral fracture with loose body.

Orthopedic Perspective

Traumatic patellar dislocation is a common knee injury in the pediatric athlete. The diagnosis can usually be made by history and clinical examination. However, in the context of an acute traumatic hemarthrosis after a twisting injury, physical examination can be difficult and differentiation must be made from ACL injury. MRI is often ordered after traumatic patellar dislocation to evaluate for an osteochondral injury and loose body, and the findings are often used to guide treatment. Patellar dislocations with osteochondral loose bodies are usually treated surgically. Small loose bodies are excised. However, some osteochondral fragments can be very large involving nearly the entire medial patellar facet or lateral femoral condyle. Lateral patellar dislocations without loose bodies are usually treated nonoperatively with bracing and rehabilitation.

What the Clinician Needs to Know

1. The diagnosis in cases where physical examination is difficult.
2. Presence and location of an associated osteochondral fracture/ loose body.

Answers

1. Direct, medial blow or a valgus, twisting (external tibia rotation) injury while the knee is partly flexed.
2. Anterolateral femoral condyle and medial patella facet or the patella eminence.

Additional Example

Acute Lateral Patellar Dislocation with Osteochondral Fragment

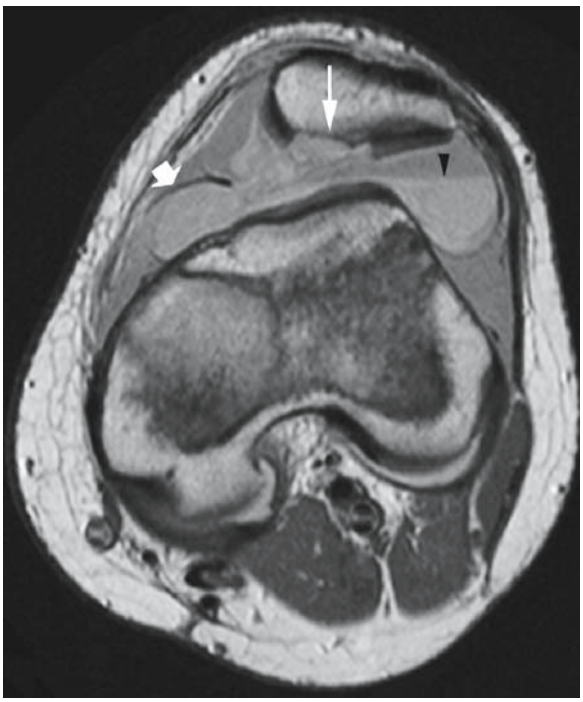

Figure 1E. Axial PD of the left knee.

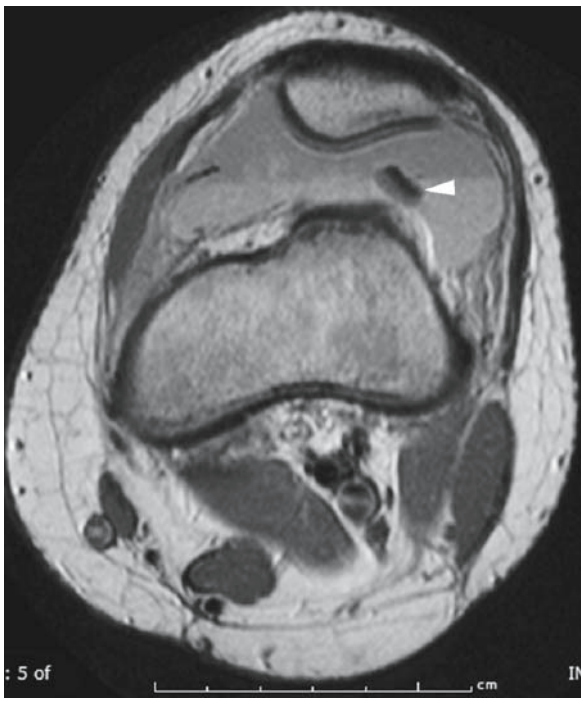

Figure 1F. Axial PD.

Findings

This is a 15-year-old girl with an acute left knee injury.

Figures 1E, 1F. A large hemarthrosis with a fluid-fluid level is present **(black arrowhead)**. There is a large osteochondral defect at the patella eminence **(white arrow)** and the daughter fragment is located laterally **(white arrowhead)**. The intermediate SI of the osteochondral fragment represents the patella articular cartilage and the hypointense SI represents the cortical bone fragment. Incidental note is made of a medial plica **(thick arrow)**.

Pitfalls and Pearls

1. Children and adolescents who dislocate their patella with trauma are often unaware that the dislocation has occurred. When the typical MRI findings are present, the diagnosis should be suggested—even in the absence of a history of dislocation.
2. Axial imaging at various stages of knee flexion may be able to demonstrate the dynamic nature of patella subluxation. The patella, when abnormal, tends to sublux with knee extension, and relocate with knee flexion.

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Case 2

History

This is a 13-year-old girl with chronic right knee pain. She is otherwise well, without fever or systemic symptoms. CBC and ESR were normal.

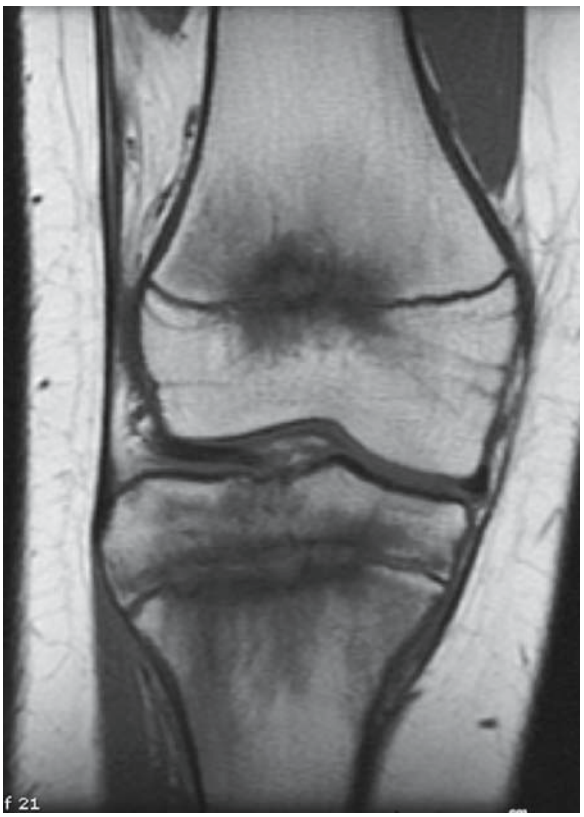

Figure 2A. Coronal T1 of the right knee.

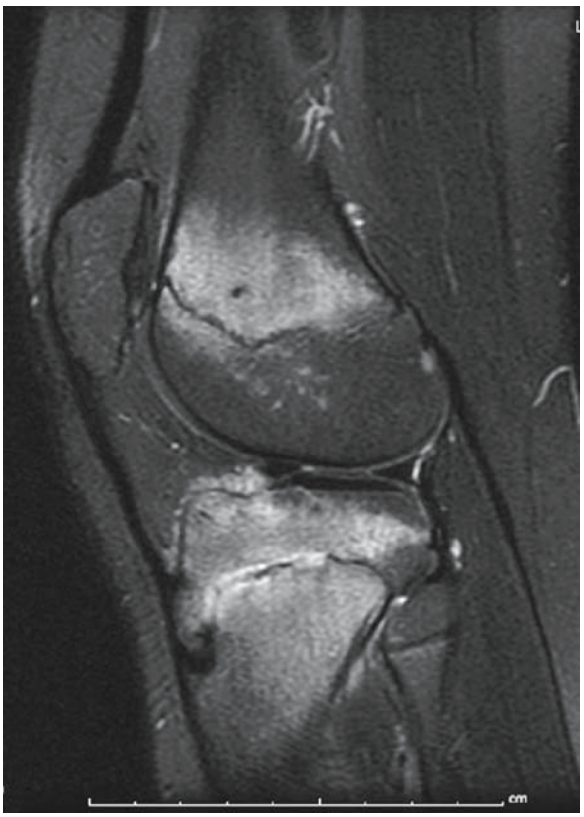

Figure 2B. Sagittal STIR.

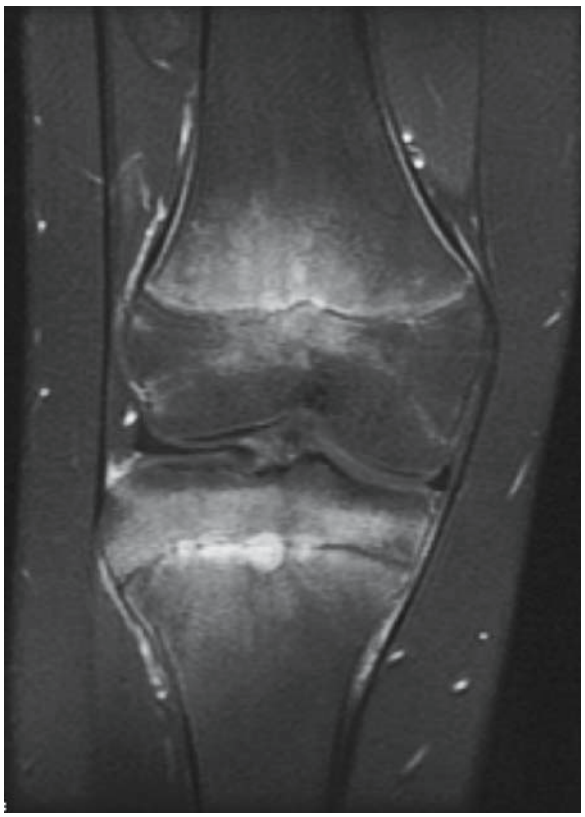

Figure 2C. Coronal T1 post-Gd FS.

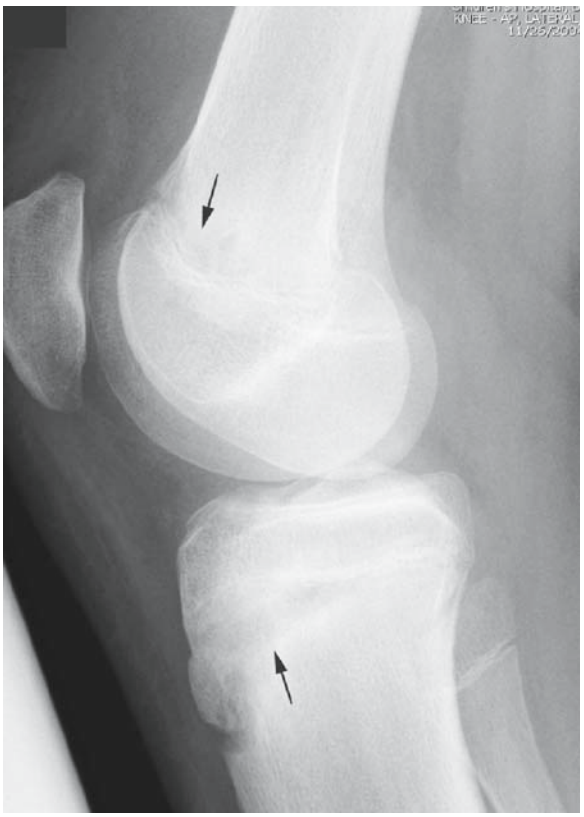

Figure 2D. Lateral plain radiograph.

Figures 2A, 2B, 2C. Focal ill-defined T1 hypointensity, STIR hyperintensity, and enhancement are present, centered at the physes of the distal femur and proximal tibia. There is minimal juxta-cortical edema in Hoffa's fat pad. There is also mild periosteal enhancement along the distal femoral and proximal tibial metaphyses. No intraosseous fluid collections or soft tissue abscess are seen.

Figure 2D. Osteolysis with marginal sclerosis involves the anterior aspects of the physeal margins of the metaphyses of the distal femur and proximal tibia **(arrows)**, corresponding to the signal abnormality on MRI.

Diagnosis

Chronic recurrent multifocal osteomyelitis (CRMO)

Questions

1. What MRI features are seen with pyogenic osteomyelitis and not CRMO?
2. What is the most common location for CRMO?

Discussion

Chronic recurrent multifocal osteomyelitis (CRMO) and pyogenic osteomyelitis share many features. Both may have osseous and adjacent soft tissue inflammation and they are typically located near the physis. Additional shared osseous findings include transphyseal spread, osteolysis or sclerosis, and varying degrees of periosteal reaction (1, 2). Pyogenic osteomyelitis is occasionally multifocal (Figures 2E, 2F), but unlike CRMO, there may be intraosseous and soft tissue abscesses, sequestra, and fistulous tracts (Answer to Question 1) (2). In the absence of these findings, CRMO and multifocal pyogenic osteomyelitis may be indistinguishable based on MRI features at individual sites.

CRMO is a diagnosis of exclusion after pyogenic osteomyelitis has been ruled out. By definition, an organism is not isolated by blood culture or biopsy. The term CRMO is a misnomer since it represents a non-pyogenic inflammatory disorder and is technically not considered osteomyelitis. CRMO is viewed as a seronegative arthropathy-like condition that occurs in children (3). Additional clinical features associated with CRMO include psoriasis, inflammatory bowel disease, recurrent arthritis, spondyloarthropathy, or sacroiliitis. CRMO is generally a self-limited disease and the majority of cases have no disability beyond childhood (4). SAPHO (synovitis, acne, pustulosis, hyperostosis, and osteitis) is considered the adult equivalent of CRMO.

CRMO has been observed most commonly in the tubular bones of the lower extremity (Answer to Question 2). The clavicles are next most common (see Case 11) (3). When the changes are restricted to the thorax and shoulder girdle, the term sternocostoclavicular hyperostosis is generally employed. Rarer locations include the spinal column and pelvic girdle (1, 5).

The principal differential diagnosis for CRMO is multifocal pyogenic osteomyelitis, and bone biopsy and culture are generally required for diagnosis. Less likely differential considerations include Langerhans cell histiocytosis, small round blue cell tumors, and trauma/stress reaction.

In this patient, the diagnosis of CRMO was invoked because of the multifocality and the absence of clinical or laboratory findings to suggest pyogenic osteomyelitis. A total body MRI showed no additional lesions. A biopsy showed changes of chronic osteomyelitis without bacterial organisms, and culture of the biopsy material showed no growth. Symptoms resolved with naproxen. She was not given antibiotics.

Orthopedic Perspective

Although patients with CRMO are usually not acutely ill, they may have erythema, low-grade fever, and abnormal laboratory values. The clinician relies on imaging to define the full extent of the process and identify a suitable site for open or percutaneous biopsy. Unlike pyogenic osteomyelitis, discrete fluid collections are not typical features of CRMO. Biopsy should target areas of granulation tissue and bone destruction, rather than bony sclerosis or nonspecific reactive edema to increase the diagnos-

tic yield. Patients are typically treated with anti-inflammatory medications and followed by a rheumatologist.

What the Clinician Needs to Know

1. Is the lesion pyogenic osteomyelitis, CRMO, or tumor?
2. Are there other lesions?
3. Which lesion is best suited for percutaneous biopsy?
4. Is the process subsiding on follow-up studies? Active lesions demonstrate juxtacortical soft tissue edema, whereas the SI within inactive lesions is confined to bone (2).

Answers

1. Intraosseous or soft tissue abscesses, sequestra, and fistulous tracts.
2. Lower extremity tubular bones.

Additional Example

Multifocal Staphylococcus aureus Osteomyelitis

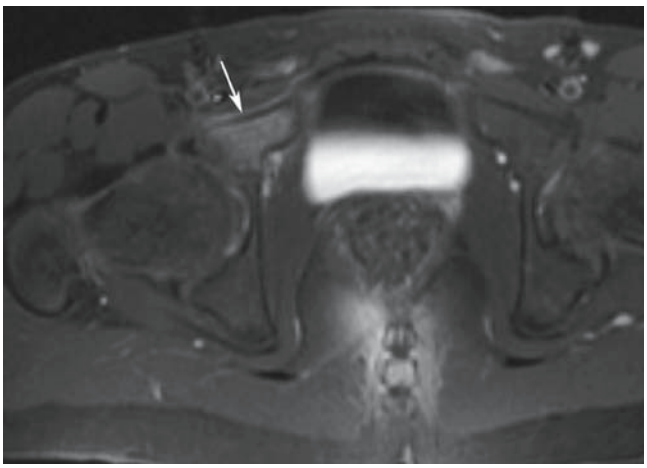

Figure 2E. Axial T1 post-Gd FS of the pelvis.

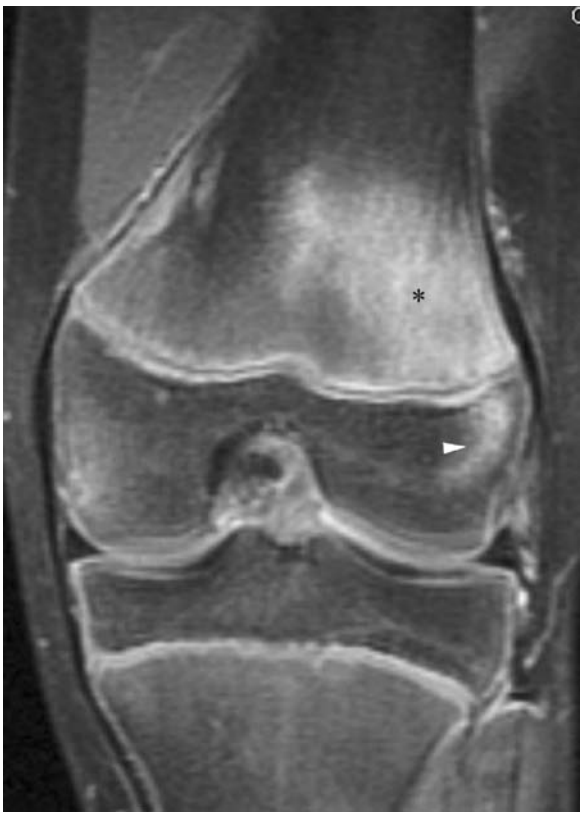

Figure 2F. Coronal T1 post-Gd FS of the left knee.

Findings

This is a 14-year-old boy with diffuse lower extremity pain and non-weight bearing. He had a severe upper respiratory tract infection 2 weeks prior to presentation. At the time of admission, he had a high fever and blood cultures were positive for *Staphylococcus aureus*.

Figures 2E, 2F. Two foci of abnormal enhancement are located in the right pubic ramus **(arrow)** and the left distal femoral metaphysis (*) and epiphysis **(arrowhead)**. Without the clinical history or positive blood cultures, the imaging findings do not allow differentiation of pyogenic osteomyelitis from CRMO, Langerhans cell histiocytosis, metastases, or stress reaction.

Pitfalls and Pearls

1. CRMO is a misnomer because it is not a bacterial infection of bone. CRMO represents a non-pyogenic inflammatory disorder that primarily affects bone.
2. The imaging features of pyogenic osteomyelitis and CRMO are similar in the majority of cases. Therefore, the diagnosis of CRMO should be made only if pyogenic osteomyelitis has been completely excluded.

3. If CRMO is a consideration, a bone scan is indicated to assess for other lesions, and to identify the optimal biopsy site.

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Case 3

History

This is a 10-year-old boy with right knee swelling following remote minor knee trauma. Radiographs demonstrated only a large joint effusion (not shown).

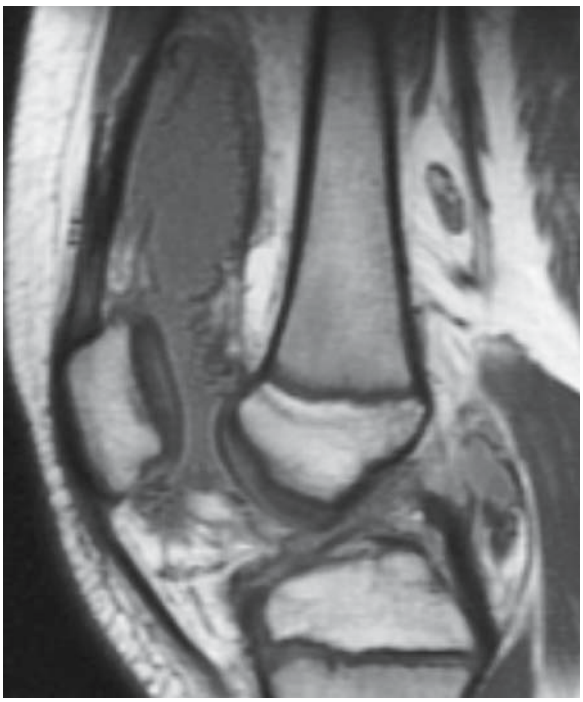

Figure 3A. Sagittal PD of the right knee.

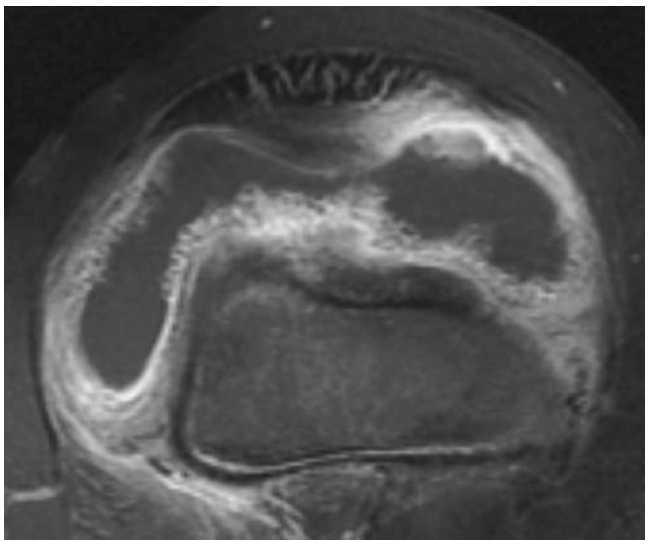

Figure 3B. Axial T1 post-Gd FS.

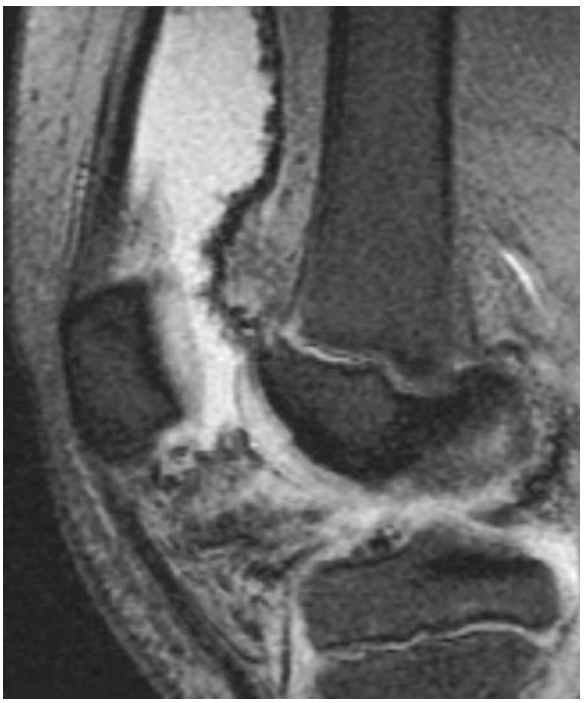

Figure 3C. Sagittal MPGR.

Figure 3A. A large joint effusion is present. The synovium is thickened with areas of hypointensity.

Figure 3B. Diffuse and nodular synovial enhancement is seen.

Figure 3C (3C with annotations). Marked susceptibility artifact **(arrows)** is present within thickened synovium and within Hoffa's fat pad **(arrows)**.

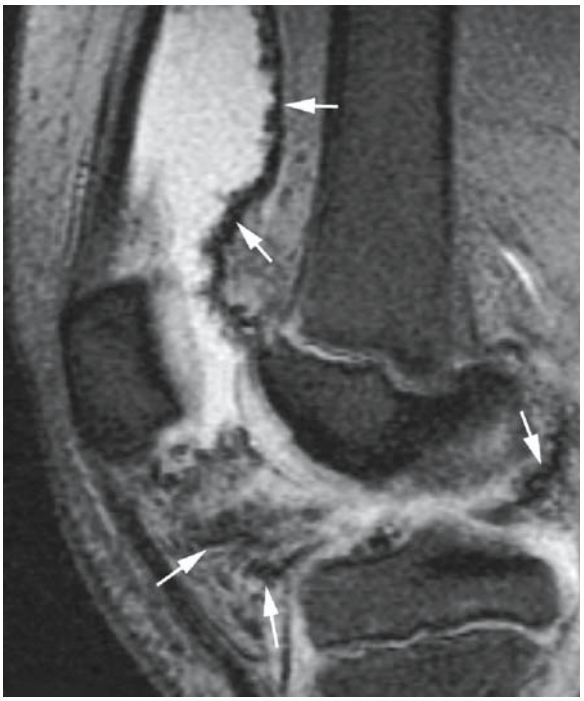

Figure 3C* Annotated.

Diagnosis

Pigmented villonodular synovitis (PVNS)

Questions

1. What are the MRI features of PVNS?
2. T/F: Post-arthroscopy blooming artifact on GRE sequences may be indistinguishable from that seen with recurrent PVNS.

Discussion

PVNS is a benign, synovial proliferation that may occur anywhere there is synovial tissue, including joints, bursae, and tendon sheaths (1). The two subtypes of PVNS are localized (most common) and diffuse. PVNS most commonly affects the knee followed by the hip. It usually is seen in the second and third decade of life and is less common in children (2). Giant cell tumor of the tendon sheath histologically resembles PVNS and may represent a localized extra-articular form of the disorder. It tends to occur in female patients in the third and fifth decade of life.

The histologic features of PVNS include synovial nodular hyperplasia associated with hemosiderin, variable lipid deposition, and fibrosis (1). On MRI, these lesions are generally hypointense on all imaging sequences and demonstrate blooming artifact on GRE sequences due to the presence of hemosiderin (Figure 3C) (3). Blooming artifact represents exaggerated signal loss artifact that may occur with hemosiderin or metal. Both the localized and diffuse forms of PVNS demonstrate variable enhancement (Answer to Question 1). Intense enhancement may be seen within these lesions related to active synovitis (3). The localized form of PVNS can be mistaken for a loose body or a mass (Figures 3D, 3E) (1). The diffuse form is often associated with a large joint effusion or hemarthrosis and may be a diagnostic challenge, since there is no normal synovial tissue on the image for comparison (Figures 3F, 3G). Although uncommon at the knee, juxta-articular bone erosions may occur in the less capacious joints, such as the hips and ankles (Figures 3H–3J).

The differential diagnosis for localized and diffuse PVNS includes: hematoma from acute or remote trauma, loose bodies, inflammatory arthritis with pannus, foreign body reaction, synovial venous malformations (AKA synovial hemangioma), hemophilic arthropathy, and synovial osteochondromatosis. All of these conditions may demonstrate hypointensity on T1 and T2W sequences due to the presence of blood products and, when focal, may suggest an intra-articular mass. Plain radiography or CT is often helpful to narrow the differential diagnosis, since PVNS rarely calcifies, whereas synovial osteochondromatosis, loose bodies, and synovial venous malformations may demonstrate calcifications (3).

The treatment for PVNS is synovectomy, but there is a high local recurrence rate. Postsurgical blooming artifact may be indistinguishable from recurrent PVNS on GRE sequences (Answer to Question 2) (2). Conventional SE post-Gd T1W sequences help distinguish PVNS recurrence from postsurgical changes (Figures 3K, 3L).

In this case, diffuse PVNS was pathologically confirmed. The patient underwent synovectomy and did well.

Orthopedic Perspective

There are very few tumors that affect the synovium, so the differential diagnosis from a tumor standpoint is short. Inflammatory arthropathies are the most common conditions in the differential diagnosis, but trauma, synovial venous malformations, and synovial chondromatosis should also be entertained. In developing countries, infection (tuberculosis) also enters the differential diagnosis. Of much more concern is a true intra-articular synovial sarcoma, which, although extremely rare, must be considered in the differential diagnosis. A biopsy is necessary to establish the correct diagnosis.

The nodular or localized form of PVNS is easier to treat, and complete excision is usually curative. Diffuse PVNS is an extremely frustrating disease for the patient and the treating surgeon. In the knee, complete synovectomy is difficult, often requiring anterior and posterior approaches, and in the hip it is almost impossible. Despite aggressive synovectomies, recurrence is common. Eventually, the destruction of the articular cartilage and adjacent bone will necessitate arthroplasty in unsuccessfully treated cases, and since these are generally young patients, this is often a poor alternative. External beam radiotherapy may control PVNS, but its use carries concerns, especially in children with open physes. The so-called radiation synovectomy, where radioactive agents are injected into the joint after synovectomy has some proponents, but its effectiveness has not been proven.

What the Clinician Needs to Know

1. Is this PVNS or an inflammatory arthritis?
2. Is this PVNS or an intra-articular sarcoma? This problem usually requires a tissue diagnosis, but the character and location of the lesion on MRI may affect the approach to biopsy.
3. Are the findings on a postoperative study due to recurrent/residual PVNS, or are they postoperative changes only?

Answers

1. Hypointensity on all imaging sequences, particularly with GRE sequences. Variable enhancement.
2. True.

Additional Examples

PVNS, Localized

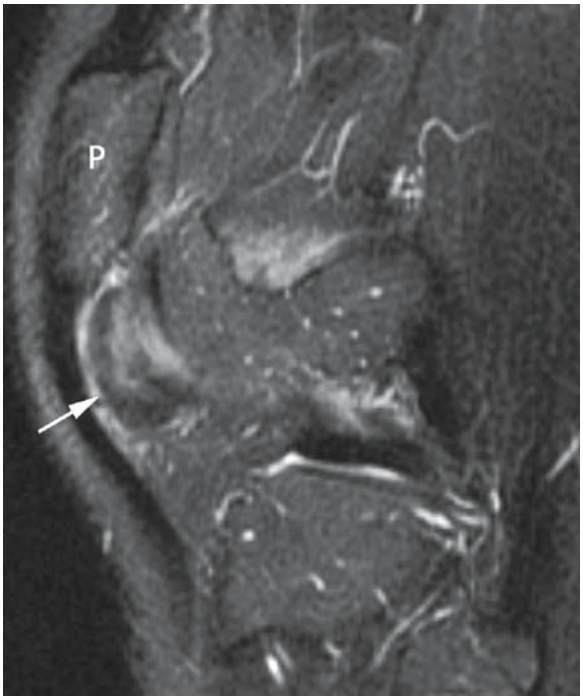

Figure 3D. Sagittal T2 FS.

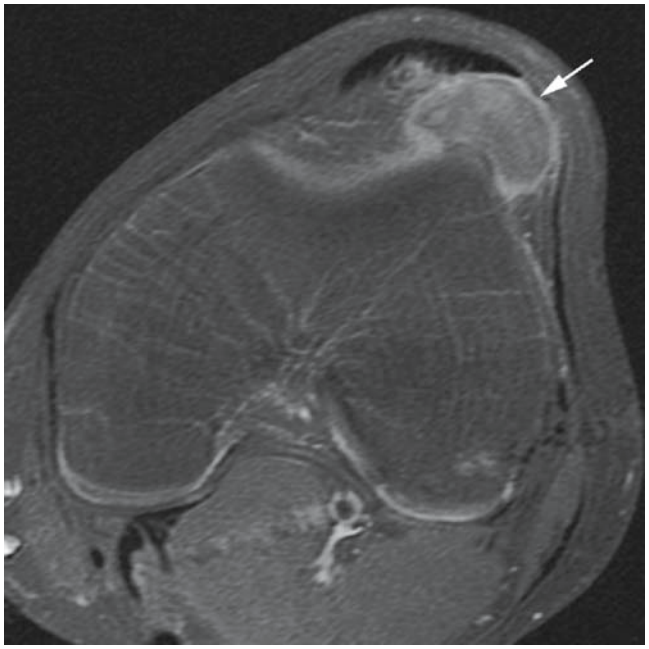

Figure 3E. Axial T1 post-Gd FS.

Findings

Figures 3D, 3E. This is a 13-year-old boy with a large, well-defined mass **(arrows)** that arises from the joint and extends inferiorly into Hoffa's fat pad. It is heterogeneously intermediate SI on T2 and demonstrates heterogeneous mild enhancement on post-Gd sequences. Blood products are inconspicuous. This lesion was pathologically confirmed localized PVNS.

Diffuse PVNS

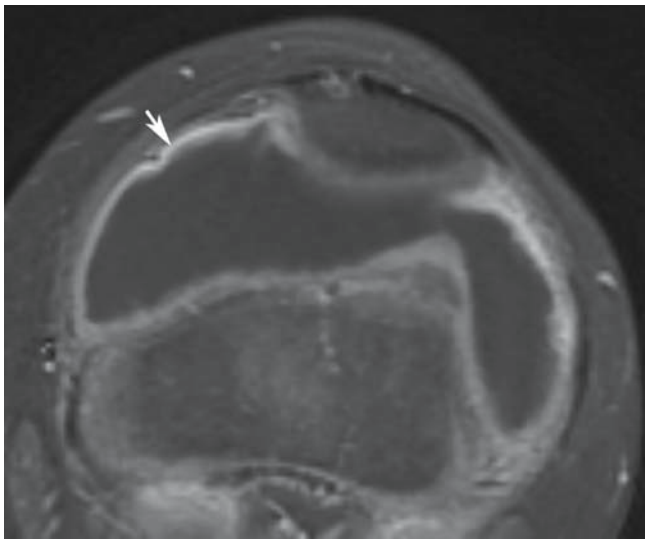

Figure 3F. Axial T1 post-Gd FS.

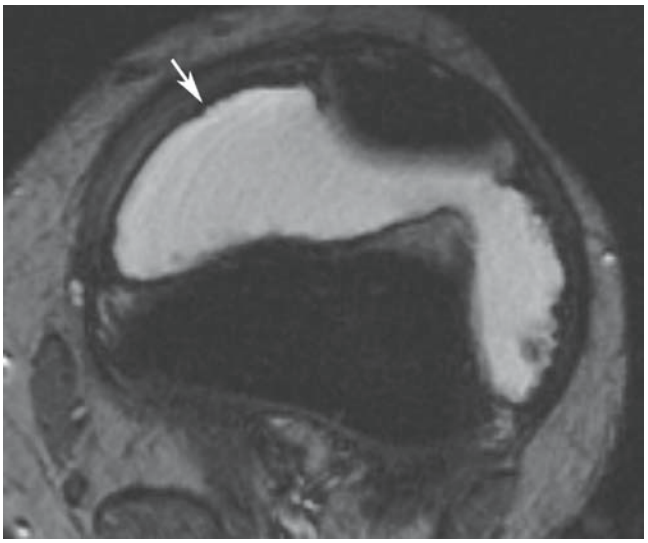

Figure 3G. Axial MPGR.

Findings

This is an 11-year-old boy who had a remote history of left knee trauma while skiing.
Figures 3F, 3G. There is a large joint effusion. There is diffuse synovial enhancement and hemosiderin deposition **(arrows)**. This was pathologically proven diffuse PVNS.

PVNS, Localized with Talar Neck Deformity

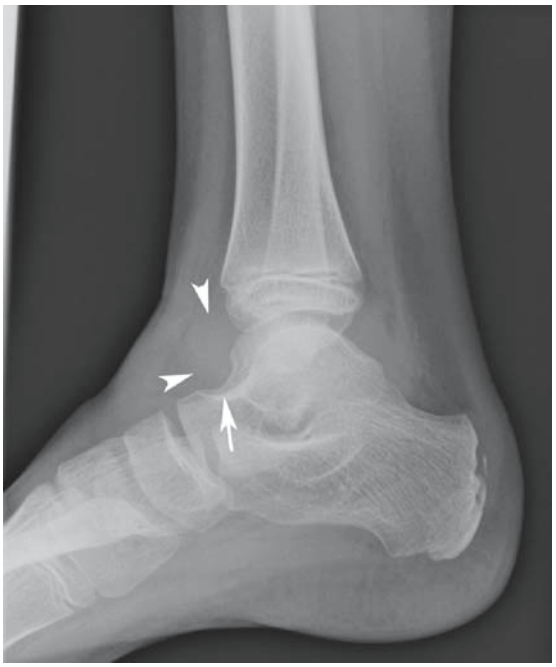

Figure 3H. Lateral radiograph of the left ankle.

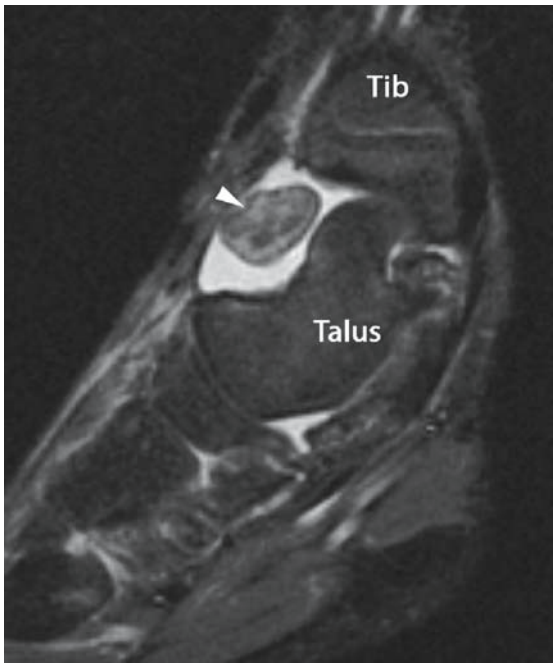

Figure 3I. Sagittal STIR.

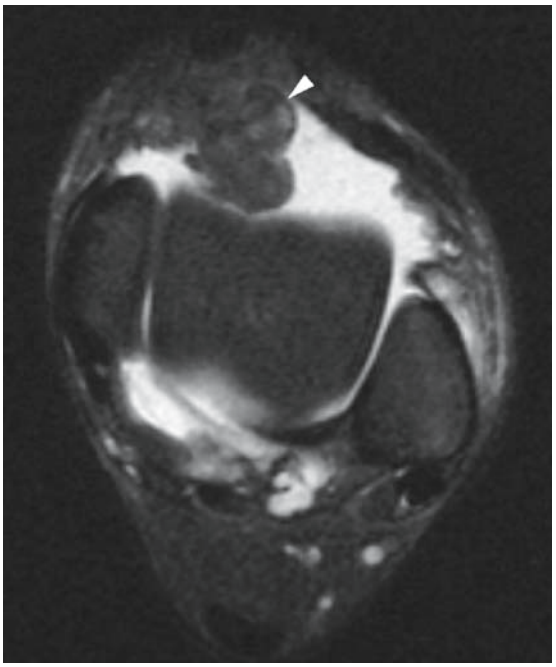

Figure 3J. Axial T2 FS.

Findings

This is a 9-year-old girl with chronic left ankle pain.

Figure 3H. There is an apparent intra-articular soft tissue mass **(arrowheads)** associated with concave deformity of the talar neck **(arrow)**.

Figures 3I, 3J. Fluid sensitive sequences demonstrate a moderate joint effusion and a hypointense lobulated mass with a hypointense rim **(arrowheads)** in the anterior joint space. This was pathologically confirmed localized PVNS. Tibia (Tib).

Recurrent Focal PVNS

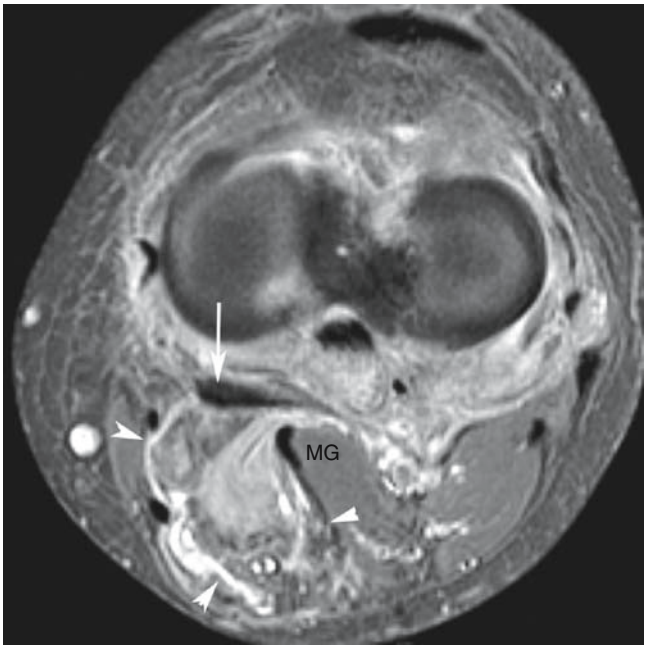

Figure 3K. Axial T1 post-Gd FS of the left knee.

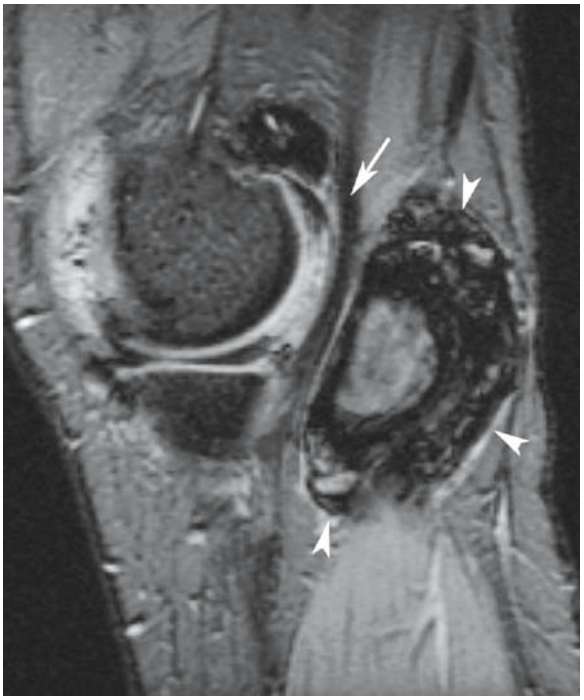

Figure 3L. Sagittal MPGR.

Findings

This 13-year-old girl with known left knee PVNS had undergone three prior synovectomies but continued to have recurrent knee pain.

Figure 3K. There is diffuse synovial enhancement with a more focal, heterogeneously enhancing mass **(arrowheads)** within the region of the semimembranosus bursa. Note location posterior to the semimembranosus tendon **(arrows)** and medial to the medial head of the gastrocnemius muscle (MG).

Figure 3L. This focal lesion shows central T2* hyperintensity and a thick peripheral rind of hemosiderin staining with blooming artifact **(arrowheads)** on this gradient echo sequence. The semimembranosus tendon is located anterior to this mass **(arrow)**. Recurrent PVNS was confirmed arthroscopically.

Pitfalls and Pearls

The absence of hemosiderin staining or blooming susceptibility artifact does not preclude the diagnosis of PVNS. Always include it in the differential diagnosis of a non-calcified intra-articular synovial-based mass.

References

1. Al-Nakshabandi NA, Ryan AG, Choudur H, et al. Pigmented villonodular synovitis. *Clin Radiol* 2004; 59:414–420.
2. Narvaez JA, Narvaez J, Aguilera C, De Lama E, Portabella F. MR imaging of synovial tumors and tumor-like lesions. *Eur Radiol* 2001; 11:2549–2560.
3. Masih S, Antebi A. Imaging of pigmented villonodular synovitis. *Semin Musculoskelet Radiol* 2003; 7:205–216.

Case 4

History

This is a 12-year-old boy with right hip pain and limp for 3 months after a minor fall.

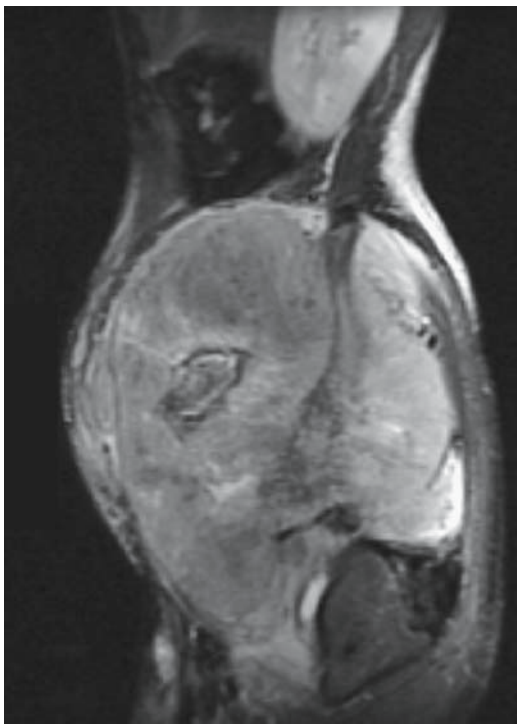

Figure 4A. Sagittal STIR through the right ilium.

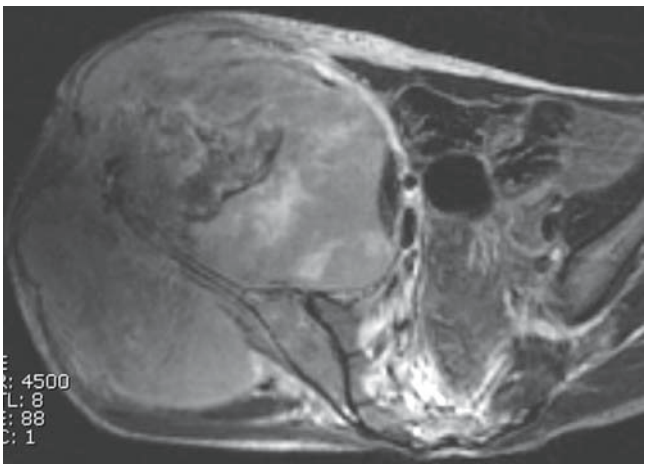

Figure 4B. Axial T2 FS.

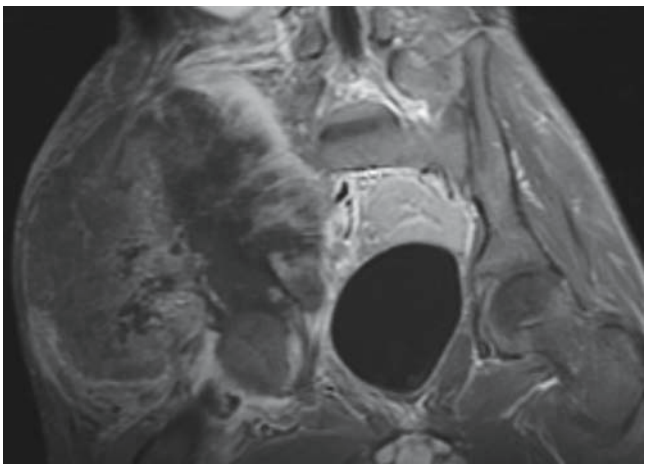

Figure 4C. Coronal T1 post-Gd FS.

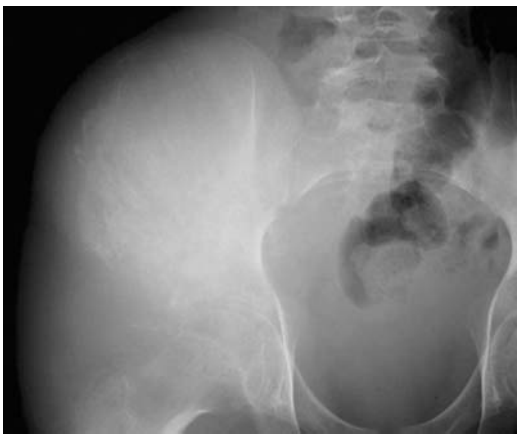

Figure 4D. AP radiograph.

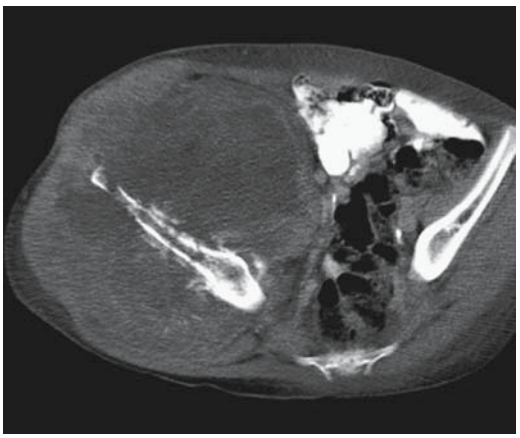

Figure 4E. CT.

Figures 4A, 4B (4A with annotations). A large heterogeneous mass is identified arising from the right ilium that is hypointense to intermediate SI on fluid-sensitive sequences. Centrally, there is a discrete area of heterogeneous SI that has a hypointense rim consistent with necrotic tumor **(arrowheads)**. Anteromedially, the mass extends into the pelvic space and displaces the iliac vessels medially. Posteriorly, the gluteal muscles are displaced and stretched over the mass.

Figure 4C. There is heterogeneous tumoral enhancement, with a sharply demarcated central zone of relatively decreased enhancement consistent with necrosis.

Figures 4D, 4E. Plain radiography and CT demonstrate moth-eaten destruction with a wide transition zone in the right ilium. Aggressive perpendicular periosteal new bone formation is seen on CT that correlates with hypointense regions on the MRI. There is some faint mineralization in the posterior extent of the lesion, but osseous tumor matrix is not evident.

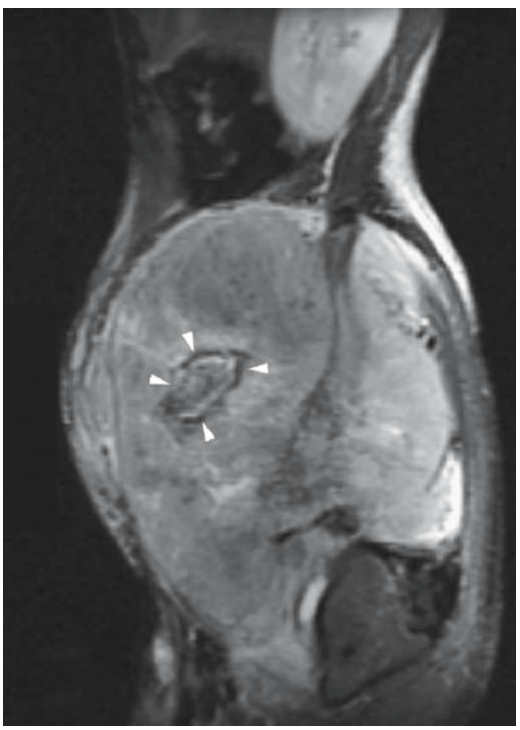

Figure 4A* Annotated.