

Abstract:

Nontraumatic pediatric hip pain and related hip pathology have a broad differential and often present a diagnostic dilemma. The age of the child; history and physical examination; and, if needed, laboratory and imaging studies can guide diagnosis. This article reviews the common etiologies for hip complaints occurring in the absence of trauma in children. The clinical presentation, evaluation, and management will be discussed as well as relevant existing literature to assist the physician in distinguishing between hip pathologies.

Keywords:

non-traumatic hip pathology; pediatric hip; transient synovitis; toxic synovitis; hip effusion; hip imaging; osteoarticular infections; septic arthritis; osteomyelitis; Lyme arthritis; Legg-Calve-Perthes disease; slipped capital femoral epiphysis; limp

Division of Pediatric Emergency Medicine, Department of Pediatrics, Children's Hospital of Pittsburgh of UPMC, Pittsburgh, PA.

Reprint requests and correspondence: Desireé Noel Wagner Neville, MD, Division of Pediatric Emergency Medicine, Department of Pediatrics, Children's Hospital of Pittsburgh of UPMC, 4401 Penn Avenue, AOB 2nd Floor, Suite 2400, Pittsburgh, PA 15224.

desiree.neville@chp.edu (D.N.W. Neville), zuckerbraunn@upmc.edu (N. Zuckerbraun)

1522-8401

© 2016 Elsevier Inc. All rights reserved.

Pediatric Nontraumatic Hip Pathology

**Desireé Noel Wagner Neville, MD,
Noel Zuckerbraun, MD, MPH**

The hip is a marvelously complex joint capable of movement in all planes while simultaneously supporting the entire weight of the body. The hip is a ball-and-socket synovial joint enclosed in a fibrous capsule. It is formed by the articulation between the femoral head, and the acetabulum of the pelvis (Figure 1).

While the hip is often injured, nontraumatic problems are common as well. These problems can present with significant distress to both the child and family, and the underlying disease can range from benign to quite serious. A complete history, thorough physical examination (often), imaging, and laboratory studies (sometimes), are the tools needed to differentiate among these disease processes.

Nontraumatic hip pathology may present as pain in the hip, thigh, or knee; altered gait; or refusal to bear weight. Eliciting the presence or absence of fever is important. Although nontraumatic hip pathology by definition does not result from an injury, at times, the patient or family may recall a recent, typically mild, trauma that is not significant enough to explain the clinical presentation.

HIP EXAMINATION

A complete hip examination begins with observation of patient's resting position, which is a useful way to assess the involvement of the hip in any patient presenting with a lower extremity complaint. The patient with a hip effusion, hemarthrosis, or hip fracture will often present with the hip resting in flexion, abduction, and external rotation.¹ Assessment of the hip is difficult because hip effusions are often not clinically apparent, and the hip joint can be difficult to isolate. Palpation over bony prominences, the pelvis, hip joint space, and shaft of the femur

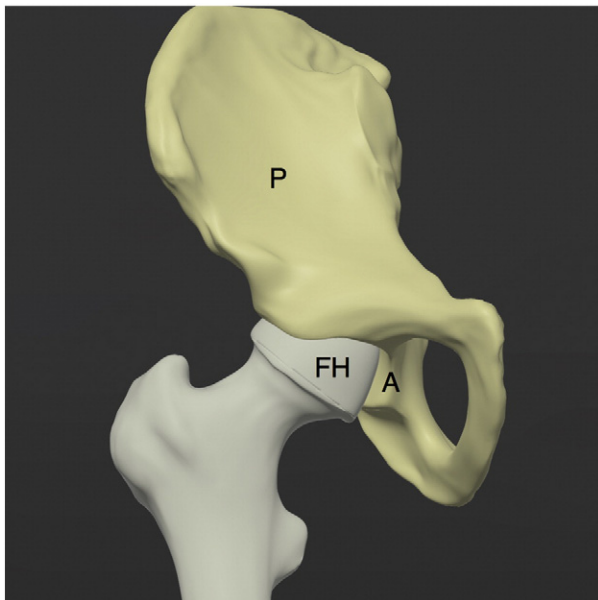


Figure 1. Hip joint anatomy: composed of the capital femoral epiphysis or femoral head (FH) within the acetabulum (A) of the pelvis (P). This ball-and-socket joint is surrounded by a fibrous capsule and contains synovial fluid.



Figure 2. Assessment of internal rotation of the hip. Shown is testing with the hip in flexion, adduction while internally rotating. The examiner is distracting the patient with an electronic device to make the child less fearful and increase the chances of a successful examination.

can be helpful to isolate areas of tenderness. Next, the clinician should evaluate the hip joint's passive and active range of motion. Internal rotation, external rotation, hip flexion, extension, abduction, and adduction should be assessed. An indicator of hip joint space disease is limitation of internal rotation (Figure 2). Internal rotation can also be tested with both of the patient's legs straight and knees extended; gentle internal rotation of the leg in this position may not elicit fear, which can skew the examination. The back should also be examined for tenderness and range of motion. Finally, both the willingness to bear weight and any gait abnormalities should be noted.

IMAGING

Radiographs

Radiographs are often the first imaging modality used to evaluate the hip. When obtaining hip radiographs, it is important to obtain a comparison view (full pelvis with view of both hips) and specifically, to always obtain both anterior-posterior (AP) and frog-leg views (Figure 3).

Radiographs are not always required. Of note, a study of 310 children with acute (<2 weeks) nontraumatic hip pain found that 1% of radiographs were positive in children younger than 9 years,

suggesting that there is limited utility of radiographs in young children with acute hip pain.²

Ultrasound

The primary application of hip sonography in nontraumatic hip pathology is for detection of an effusion. An ultrasound cannot distinguish between sterile and pyogenic effusions. Radiology ultrasound has long been known to be superior to radiographs for the detection of hip effusions.³ Although it is traditionally performed in the radiology department, the emergency provider can diagnose hip effusions with point-of-care ultrasound care ultrasound (POCUS).^{4,5} It is rapid, is easily accessible, lacks ionizing radiation, and does not require sedation.

The following is a brief description of the POCUS technique for the hip. The leg should be positioned in slight abduction and external rotation. The linear (high frequency) transducer is placed parallel to the long axis of the femoral neck, which can be found just inferior to the inguinal ligament and lateral to the femoral vessels. The transducer is positioned with the indicator pointing superomedially on an imaginary line extending from the greater trochanter toward the umbilicus.⁶ (Figure 4)

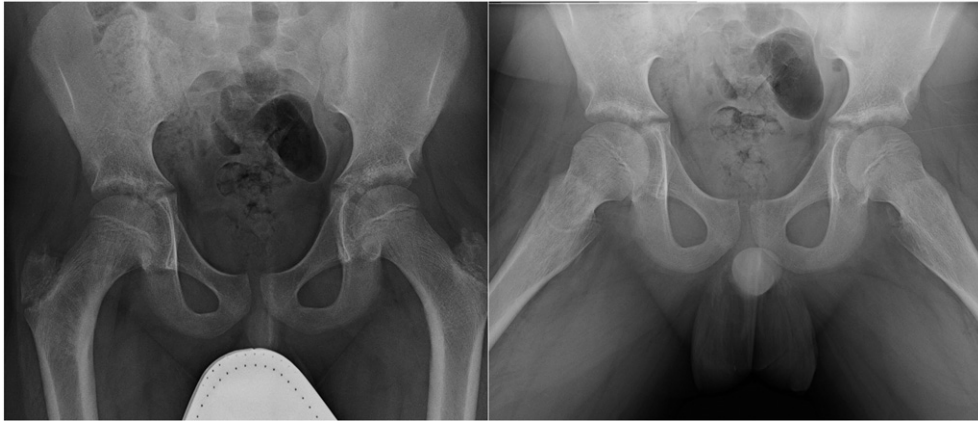


Figure 3. Anterior-posterior and frog-leg hip radiographs.

The femoral head can be identified as a curved, hyperechoic line. The femoral neck can be identified as a hyperechoic line distal to the femoral head. In the normal hip joint, the joint capsule appears as a hyperechoic band that runs anterior to the anterior surface of the femoral neck and posterior to the posterior surface of the iliopsoas muscle⁶ (Figure 5). A hip effusion displaces the joint capsule resulting in a hypoechoic or anechoic fluid collection just distal to the femoral head and anterior to the femoral neck.

Criteria for the diagnosis of a hip effusion include AP fluid collection of greater than 5 mm or a fluid collection difference of greater than 2 mm when compared to the contralateral hip.^{6,7} The effusion can deform the typically concave anterior joint capsule to convex.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) can be helpful in cases where more detailed assessment of the hip is necessary for diagnosis or management planning, particularly to distinguish which deep structures are involved, such as assessing for bone or muscle involvement surrounding a septic hip joint. Although MRI is increasingly used, it is not always readily available. In addition, MRI is time, cost, and resource intensive, as children frequently require sedation to acquire images. Because of these constraints, it is not a routine imaging study for all patients with a hip complaint. The specific indications for performing an MRI in the evaluation of individual hip pathologies will be discussed in each respective section.

LABORATORY STUDIES

History and clinical examination dictate whether laboratory studies are necessary. The primary

indications for obtaining these studies are when infection and/or malignancy are being considered.⁸ Useful laboratory tests in these cases may include complete blood count with differential, C-reactive protein (CRP), erythrocyte sedimentation rate, and for some, a blood culture. Additional laboratory tests may be helpful based on the specific clinical scenario, such as Lyme testing (in areas where Lyme is endemic) and synovial fluid analysis (synovial fluid culture and Gram stain and cell counts). In the following sections, we will review specific indications for other laboratory studies. It is worthwhile to note that antinuclear antibody, rheumatoid factor, and antistreptolysin O are not helpful in the workup of isolated hip pain or limp.⁹

SPECIFIC NONTRAUMATIC HIP DISORDERS

Transient Synovitis

Transient synovitis is a self-limiting inflammation and effusion of the hip joint space of unknown etiology without serious sequelae and is confirmed by excluding serious hip pathology.¹⁰

Epidemiology

Transient synovitis is the most common diagnosis among patients with nontraumatic pediatric hip complaints.^{9,11-13} The mean patient age at presentation is 4.7 years with a typical range of 3 to 8 years.^{2,12} A study in Sweden calculated a lifetime risk for transient synovitis at 3%, with a 0.2% annual incidence and a 4% incidence of recurrence.¹⁴ Transient synovitis is usually unilateral, but in a minority of patients, bilateral involvement may occur.¹³ There is a male predominance.¹⁴ A history of preceding upper respiratory, gastrointestinal, or urinary tract infection or minor trauma may be

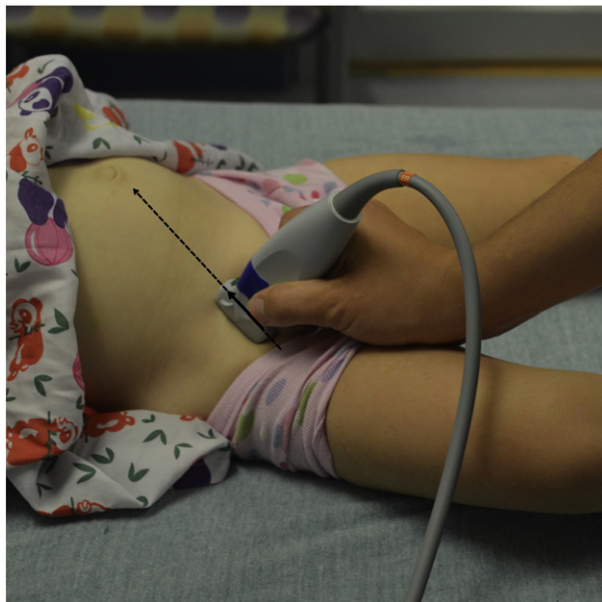


Figure 4. Hip ultrasound position. Ultrasound of the hip with the probe positioned parallel to the femoral neck with the indicator pointing superomedially toward the umbilicus.

described.¹⁴ In a series of 383 children with transient synovitis, 40% had an upper respiratory infection within 2 weeks preceding symptoms onset.⁹

Clinical History and Examination

Children with transient synovitis characteristically present with an acute onset of groin or thigh pain and a limp or unwillingness to bear weight¹¹ (Figure 6). They may hold their hip in flexion, abduction, and external rotation and have limited range of motion on examination, particularly limited internal rotation.¹⁵ Some authors describe patients with the presentation of nontraumatic hip pathology of unknown origin as having an acutely irritable hip. In a series of 417 patients with “acutely irritable hip,” 383 were diagnosed with transient synovitis. In those with transient synovitis, limping was present in 94%, with 16% unable to bear weight.⁹ Children with transient synovitis are typically well appearing, nontoxic, and afebrile or may have a low-grade temperature elevation. In the above series, only 1% of those with transient synovitis had a reported oral temperature greater than 38.5°C.⁹

Diagnosis

The diagnosis of transient synovitis is largely clinical. It is diagnosed in a child with acute onset of hip pain, limp, or refusal to bear weight in 1 leg, in the correct age group (3-8 years) who is without

trauma, high fever, or concerning examination. During the acute evaluation, observing improved weight bearing and ambulation after a dose of nonsteroidal anti-inflammatory drugs (NSAIDs) is additionally reassuring against serious underlying pathology. A concerning examination would include toxic appearance, high fever ($>38.5^{\circ}\text{C}$), or continued severe limitation of movement or weight bearing despite symptomatic treatment (NSAIDs).

Without the presence of trauma, prolonged duration of symptoms (>1 -2 weeks), or concerning examination in a young patient (<9 years), hip radiographs are often unnecessary.⁹ In these cases, the diagnosis of transient synovitis can be made based on history and physical examination alone.

Hip sonography can identify an effusion, but as stated above, it cannot distinguish between sterile and septic joint effusions, and thus, is of minimal utility in a patient who fits criteria for transient synovitis in the young age group.^{16,17}

If a high fever or other signs and symptoms that are not characteristic of transient synovitis are present, the clinician may choose to obtain laboratory studies and joint aspiration to differentiate transient synovitis from septic arthritis.

Clinical Course

An understanding of the clinical course of transient synovitis is important for anticipatory guidance and to assure return to medical care if the child is not improving as expected. Transient synovitis is self-limited and typically resolves over a period of 3 to 10 days without specific treatment.¹⁸ A study of patients with transient synovitis followed with serial examinations and ultrasound found full symptom and effusion resolution by 7 days in 60% of patients and 100% symptom resolution with 84% effusion resolution by 14 days.¹⁹ A randomized clinical trial in children with transient synovitis showed a decrease in symptoms from 4.5 days in the placebo group to 2 days the ibuprofen group.²⁰ If there is a recurrence of transient synovitis, it typically takes place within the first year but can occur more remotely in a small portion of patients.²¹ A literature review of 455 patients with transient synovitis evaluated in 10 studies found that 4% were later diagnosed with Legg-Calve-Perthes disease (LCPD).²²

Management

After diagnosis, discharge with plan for clinical follow-up in 5 to 7 days is appropriate. Scheduled NSAIDs should be prescribed. Finally, review of anticipatory guidance and reasons to urgently return to care including high fever, progressive and continual inability to bear weight, or toxic

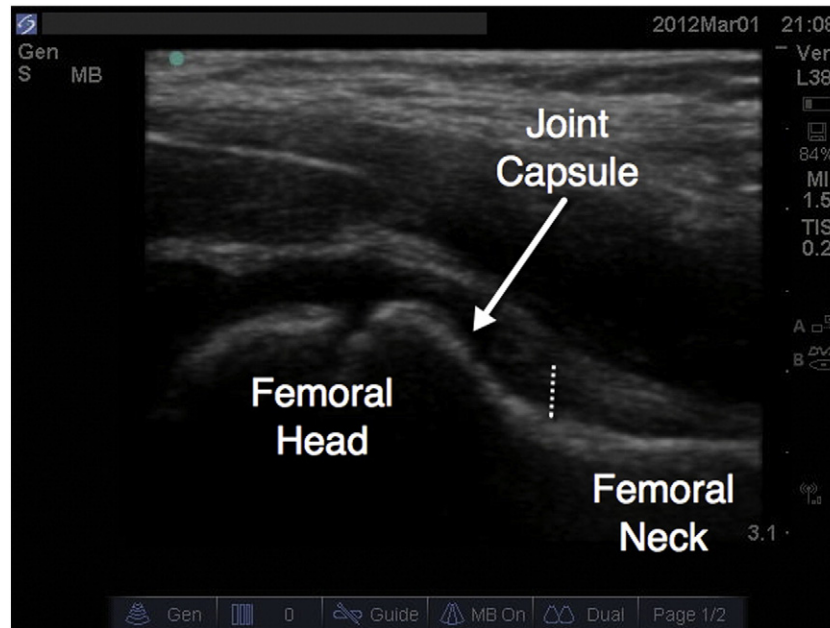


Figure 5. Normal hip ultrasound showing the femoral head, femoral neck, and the hypoechoic area representing the joint capsule. The dotted line is where the distance in the joint capsule is measured for a hypoechoic effusion. The measurement here is 3 mm and normal; abnormal will be discussed in subsequent sections.

appearance with systemic symptoms should be provided.

Legg-Calve-Perthes Disease

Legg-Calve-Perthes disease (LCPD) is an aseptic, noninflammatory, self-limiting, idiopathic avascular necrosis of the capital femoral epiphysis in children.²³ After initial devascularization, blood supply to the femoral head restores to normal within 1 to 2 years.²⁴

Epidemiology

Legg-Calve-Perthes disease mostly affects children between 2 and 12 years of age.^{23,24} It is 4 times more common in males and 10 to 15% of cases have bilateral hip involvement.²⁵ Incidence rate is 0.2 to 19.1 per 100 000 children.²⁶ Obesity and hypercoagulability can predispose patients to develop LCPD.^{27,28}

Clinical History and Examination

Children with LCPD present subacutely with weeks to months of painless or painful limp or groin, hip, thigh, or knee pain.²⁴ Given the insidious nature of the limp, patients may actually seek medical attention when the limp is noted after a minor trauma and then on thorough history taking, the limp is found to be more longstanding. Alternatively, limp may be noted only in the review of symptoms for another complaint. Examination

findings depend on timing in the disease course. Hip range of motion may be affected with abduction and internal rotation being limited first, followed by Trendelenberg gait (trunk shift over the affected lower extremity during ambulation), and in severe cases adduction contracture.²⁵



Figure 6. Toddler with refusal to put leg down.

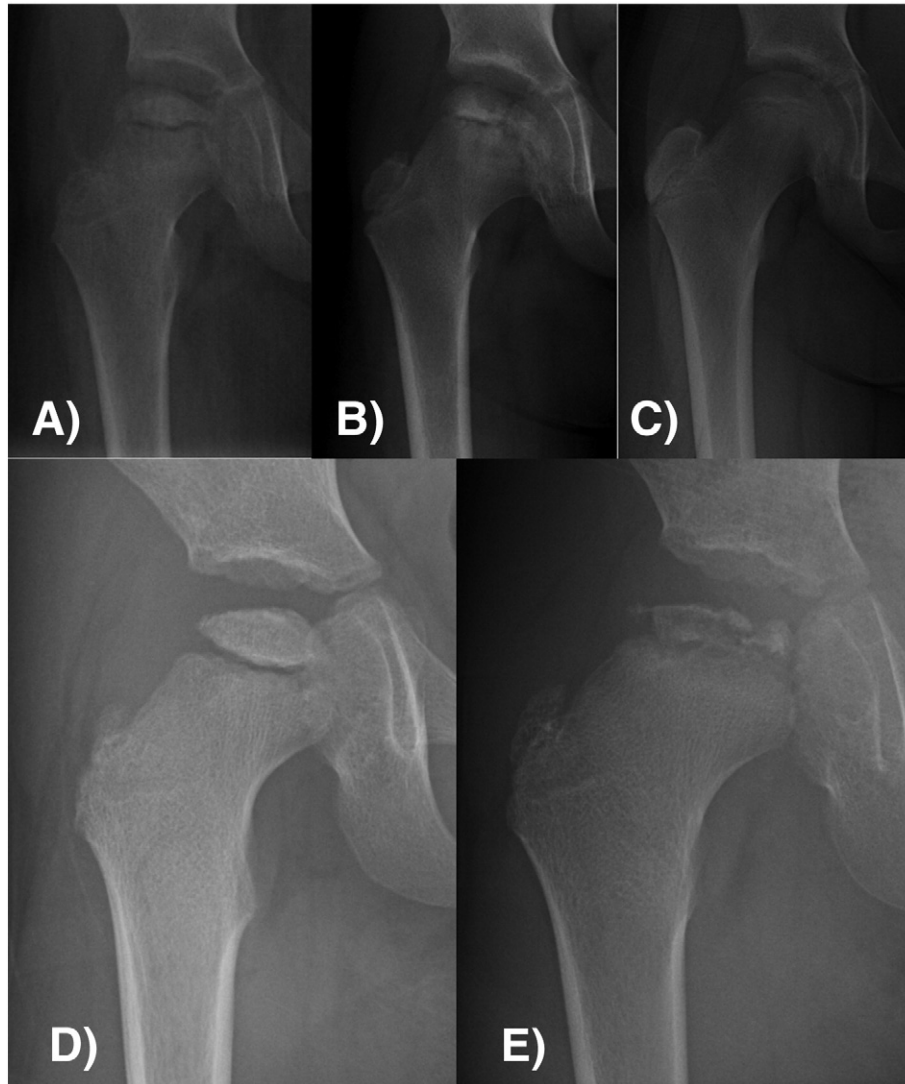


Figure 7. Evolution of Legg-Calve-Perthes disease: femoral head flattening (A), femoral head fragmentation (B), healing (C). Panels D and E show femoral head flattening and fragmentation, respectively, in better detail.

Diagnosis

Hip radiographs can establish the diagnosis of LCPD, but early radiographs may be normal as it can take months after disease onset for diagnostic radiographic signs.²⁵ The radiographic findings are changes in the size and shape of the femoral head and congruency with the acetabulum²⁵ (Figure 7). There are several classifications to describe the severity of the deformity on radiographs, all of which classify the femoral head as it progresses from spherical to elliptical to irregular to fragmented.²⁹ If radiographs are normal and suspicion is high based on age and duration of symptoms, MRI can be used to identify early disease vs referral to an orthopedic surgeon for further clinical evaluation.³⁰ Gadolinium-enhanced MRI can allow for early assessment of

femoral head perfusion deficits. Less perfusion on this type of MRI correlates with greater femoral head deformity.³¹

Clinical Course

Although the altered blood supply of LCPD is self-limited lasting 1 to 2 years, irreversible deformation of the hip joint occurs when LCPD has progressed to femoral head fragmentation or soon after.²³ The age of the child at presentation, hip range of motion, shape of the femoral head, and whether there is extrusion of the femoral head are all factors that affect the prognosis and management decisions. The sequelae from LCPD are degenerative changes and subsequent arthritis as an adult.²⁵

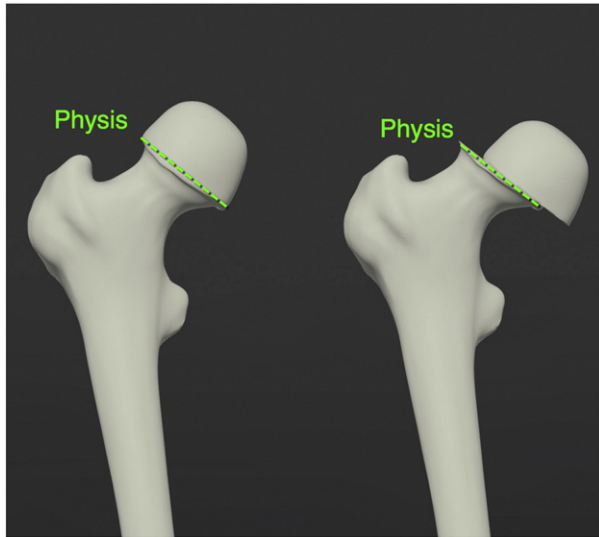


Figure 8. Diagram of the normal proximal femoral anatomy on the left and slippage of the femoral head or capital epiphysis through the physis in an SCFE on the right.

Management

Patients diagnosed with LCPD should be referred to an orthopedic surgeon for ongoing outpatient management. The goal of therapy is to prevent secondary degenerative arthritis of the hip as adults by maintaining sphericity of the femoral head and congruency with the acetabulum.^{24,32} Depending on both patient and femoral head anatomic factors, treatment may range from decreased physical activity and close observation to surgical intervention.³²

Slipped Capital Femoral Epiphysis

Slipped capital femoral epiphysis (SCFE) is a displacement of the femoral head from the femoral neck through the epiphyseal plate (Figure 8). This pathology results from mechanical overload to the proximal femoral physis causing slippage. The proximal femoral physis is a vulnerable region as it is nourished by a fragile blood supply and is an area of rapid cellular proliferation vulnerable to instability, particularly during the hormonal changes of puberty.³³

Epidemiology

The incidence of SCFE is estimated at 10 per 100 000 children with a higher proportion of males (4:3, male:female).³³ Patients with SCFE present at a mean age of 12 years, with most patients presenting between 10 and 16 years of age.³⁴ The blood supply to the femoral neck coupled with a rapid change in the angle of the physis during

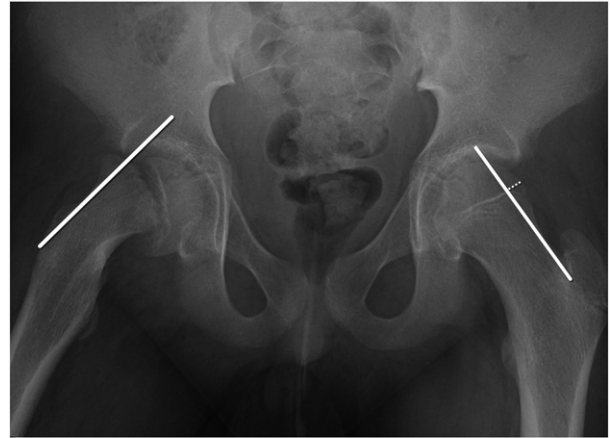


Figure 9. Anterior-posterior hip radiographs of a patient with right-sided SCFE. Klein lines are drawn in solid white. The line on the left hip intersects the femoral head (epiphysis), where the right line does not intersect the femoral head due to its slippage. The dotted line on the left hip shows the measurement of the femoral head that is lateral to the Klein's line as discussed in the text. On the right, there is no mass of the femoral head lateral to Klein's line as there is a significant slip. In mild slips, a difference of 2 mm between the distances the femoral head extends laterally to the Klein's line may be an early indicator.

growth, particularly at 9 to 12 years of age, contributes to the occurrence of SCFE in adolescence.³³ A higher weight load on the femoral epiphysis in obese children and adolescents also predisposes to SCFE.³⁵ Most patients outside of the classic age range represent atypical SCFE; for example, those patients with endocrinologic conditions such as hypothyroidism and growth hormone deficiency.³⁴ Of note, 6 to 22% of patients have bilateral SCFE at presentation, and up to 24% of patients with unilateral SCFE have subsequent contralateral slip.³⁵⁻³⁸ These numbers are highest in patients with endocrinopathies.^{39,40}

Clinical History and Examination

Patients with SCFE commonly present subacutely with months of symptoms. The most common symptoms include limp or groin, hip, thigh, or knee pain.³³ Patients may report a recent minor trauma that does not explain the symptoms. Of note, 15 to 50% of patients are reported to have knee pain at presentation. Knee pain presents a challenge for SCFE diagnosis and can lead to higher rates of misdiagnosis, more radiographs, and more severe SCFE by the time of treatment.^{41,42} Patients with SCFE often have an altered gait, which can be antalgic, waddling, or Trendelenburg.⁴³ The altered gait is rarely painless.⁴⁴ On examination, patients may have external foot rotation, weak hip

abduction, decreased hip flexion, and decreased internal rotation and can develop a flexion contracture over time.

Diagnosis

The classic way to diagnose SCFE is by drawing a “Klein's line” on an AP radiograph.⁴⁵ Klein's line is a line that is drawn along the superior aspect of the femoral neck, which in normal subjects should intersect the epiphysis of the femoral head. Without that intersection, SCFE is present (Figure 9). A 2009 study in which radiologists reviewed 60 SCFE radiographs found a low sensitivity of the Klein's line alone missing 60% of cases and suggested using an additional measurement of the epiphyseal width lateral to Klein's line. If the difference between the epiphyseal widths lateral to Klein's line of the 2 hips is greater than 2 mm, the side with the smallest width represents the SCFE with a sensitivity of 79%⁴⁶ (Figure 9). This measurement helps to detect an early or mild slip by assessing the amount of epiphysis that remains lateral to the Klein's line. Earlier radiographic findings that suggest SCFE include widening and irregularity of the physis, sharpening of the metaphyseal border of the head, loss of the anterior concavity to the head-neck junction, and subtle periosteal elevation.⁴⁵ Use of ultrasound, MRI, and computed tomography have been described for the diagnosis and assessment of SCFE; however, most can be diagnosed by plain radiographs.³³

Management

Patients diagnosed with SCFE should be made non-weight bearing and have immediate orthopedic consultation. The treatment of SCFE is to stabilize the epiphysis and prevent progression and complications including osteonecrosis of the femoral head.^{33,47} The most accepted treatment method to stabilize SCFE per a recent systemic review is single in situ screw fixation, which has a high success rate (91%).^{48,49}

Osteoarticular Infections (Septic Arthritis and Osteomyelitis)

Septic arthritis is an inflammation of the joint space due to infection. It is an emergency, as delayed diagnosis and treatment can lead to irreversible joint damage.⁵⁰ Osteomyelitis is an inflammation of bone due to infection and may be an isolated problem or coexist with septic arthritis.

Epidemiology

Septic arthritis and osteomyelitis can occur in any age group. The most common etiology of septic



Figure 10. Resting position with hip joint pathology: slight flexion, abduction, and external rotation.

arthritis and osteomyelitis is hematogenous spread from a distant source.^{43,51} Septic arthritis may have other sources including penetrating trauma or spread from adjacent infection (cellulitis/osteomyelitis). In children younger than 2 years of age, blood vessels cross the proximal femoral physis and allow spread of infection into the epiphysis and contiguous hip joint.⁴³ This is cited as one reason septic arthritis is found most commonly in the youngest age groups.

The occurrence of coexisting osteomyelitis with septic arthritis has been reported in a wide range of 20 to 70% of cases and occurs in all age groups.⁵²⁻⁵⁴ Of all osteoarticular infections in children, those involving the pelvis or hip represent 49% of isolated septic arthritis, 20% of isolated osteomyelitis, and 31% of combined septic arthritis and osteomyelitis.⁵² The hip, similar to the knee, ankle, and shoulder joints, possesses an intracapsular metaphysis, which makes it more susceptible to coexisting infections.⁵⁴

Clinical History and Examination

The classic clinical examination of a patient with septic arthritis is a high-grade fever and toxic appearance with a hot and swollen joint of short duration, typically less than 1 week.^{53,55} Clinically, the hip joint is difficult to assess given its deep anatomical position, and it is often impossible to visibly detect a hip effusion. The patient with septic arthritis of the hip may hold their hip in a position of

comfort with the hip in slight flexion, abduction, and external rotation⁴³ (Figure 10). Patients may have severe resistance or even complete inability to allow their joint to be moved through any range of motion, as opposed to more minimal to moderate resistance to internal rotation with transient synovitis.⁴³ Refusal or complete inability to bear weight is also a frequent feature. Like other hip pathologies, a septic hip can present with referred pain to the knee. Although septic arthritis most often involves only a single joint, involvement of multiple joints is possible, especially in the immunocompromised patient.⁵⁶

In general, the diagnosis of osteomyelitis is primarily clinical with the main features pain and tenderness over the affected bone. However, osteomyelitis of the proximal femur within the hip joint or osteomyelitis of the acetabulum or pelvis can be indistinguishable on examination from septic arthritis. Of note, neonatal presentation of any osteomyelitis may be more subtle and nonspecific.⁵⁷

Diagnosis

The standard for the diagnosis of osteoarticular infections is isolation of a pathogen from the site of infection or isolation of a pathogen from the blood with imaging demonstrating evidence of inflammation at the site. There are a significant number of cases of presumed septic arthritis when purulent fluid is obtained from the joint space, but pathogenic bacteria are not isolated. This number has decreased as diagnostic techniques such as polymerase chain reaction (PCR) improve to detect difficult to isolate organisms, primarily *Kingella kingae*.⁵⁸⁻⁶⁰

Although they have a low yield, blood cultures should be obtained as they can confirm the diagnosis and guide management in some cases. Blood cultures in septic arthritis and osteoarthritis are reported to be positive in 14 and 30% of cases, respectively.⁶⁰⁻⁶² Joint fluid cultures in septic arthritis are positive in up to 30 to 50% of cases.⁶⁰⁻⁶² Utilization of PCR methods report a 20 to 40% increase in bacteria isolation from joint fluid in septic arthritis with higher yield in younger children in which *K kingae* may be more prevalent.^{58,61} In contrast to the yield in blood and joint fluid cultures, site cultures in osteomyelitis are positive in 90-95% of cases.⁶²

For septic arthritis, a combination of clinical presentation, laboratory testing, and imaging can help in determination of the need for joint aspiration and diagnosis. The classic criteria used to differentiate septic arthritis from transient synovitis in children are the Kocher criteria, which use a combination of fever, refusal to bear weight, erythrocyte sedimentation rate greater than 40 mm/h, and

white blood cell greater than 12000/ μ L.⁶³ In the initial report, patients who met all 4 criteria were reported to have a 99.6% probability of septic arthritis, those with 3 criteria had a 93.1% probability, and those with 1 criterion had less than 3% probability. Some subsequent validation studies did not duplicate as high of sensitivity and specificity.⁶⁴ More recently, CRP has been used to aid in diagnosis. Caird et al⁶⁵ added CRP to the Kocher criteria and found patients with 5, 4, and 3 criteria had a 98%, 93%, and 83% probability of septic arthritis, respectively. Sultan and Hughes⁶⁶ reported fever greater than 38.0°C was the most sensitive feature associated with septic arthritis followed by a CRP greater than 20 mg/L (2 mg/dL). Alternatively, to determine low risk, Singhal et al⁶⁷ found a combination of CRP less than 20 mg/L (2 mg/dL) and ability to weight bear identified children in their series with less than 1% chance of septic arthritis. All of these diagnostic criteria combining history, physical examination, and laboratory findings have reported to minimize unnecessary joint aspirations in patients with transient synovitis.

There are times in which joint aspiration may be warranted regardless of laboratory findings. In patients with concerning exam findings (toxic appearing, high fever, or refusal of weight bearing despite NSAID treatment), these criteria should be used with caution. This is especially true in children younger than 4 years who are known to be more susceptible to *K kingae* osteoarticular infection, which can be present despite normal laboratory values.^{58,68}

Synovial Fluid

When joint aspiration is warranted based on clinical examination and/or laboratory studies, the synovial fluid analysis should include Gram stain, synovial culture, and synovial cell counts. While synovial counts more than 50 000 have been reported to be concerning for septic arthritis, recent analyses have shown difficulty differentiating the etiology of hip effusions based on cell counts alone.⁶⁹ A study of 46 children who underwent joint aspiration for hip effusions and had synovial cell counts between 25 and 75 000 found septic arthritis in 33%, Lyme infection in 28%, and transient synovitis in 17%. In another study, septic arthritis was diagnosed in 48% of patients with cell counts more than 50 000 and in 17% of patients with cell counts less than 17 000.⁷⁰ Thus, although cell counts are not definitive, they can be helpful.

Imaging

Radiographs. Septic arthritis will typically yield normal hip radiographs, and if anything, a widened

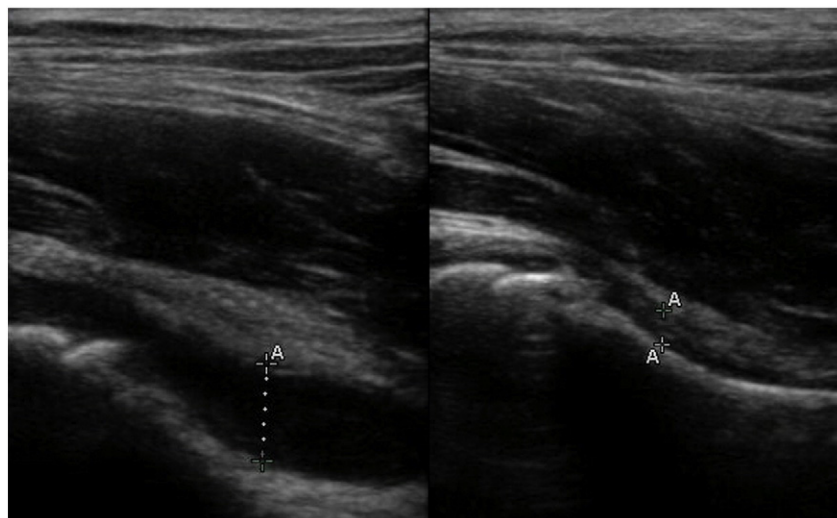


Figure 11. Ultrasound of a patient with septic arthritis showing effusion and normal contralateral hip. On the left is an ultrasound of a hip with an effusion, and the picture on the right is the contralateral side of the patient. The side with the effusion measures greater than 5 mm and is greater than 2 mm larger than the contralateral side, both of which alone indicate an effusion.

joint space may be appreciated. Radiographs do not detect early osteomyelitis. The first signs of osteomyelitis may be visualized on radiographs between 7 and 21 days and include soft tissue swelling, bone lucency, and periosteal reaction.^{71,72}

Ultrasound. As described previously, ultrasound is helpful to confirm a hip effusion but cannot

differentiate between sterile and septic effusions^{16,17} (Figure 11). Prior reports have shown that US findings should be interpreted with caution because there may be false negative ultrasounds early in the course of septic arthritis.^{73,74}

Magnetic Resonance Imaging. Magnetic resonance imaging is both sensitive and specific for septic arthritis and can differentiate it from the other hip pathologies including osteomyelitis, which will have similar laboratory values⁷⁵⁻⁷⁹ (Figure 12). The drawbacks of MRI include cost and potential difficulty obtaining due to availability and need for sedation. Magnetic resonance imaging is also less feasible for patients with polyarticular disease. Magnetic resonance imaging can detect osteomyelitis in early stages and can help to guide surgical management.⁸⁰ Two studies recommend preoperative MRI and suggest that failing to recognize adjacent infections in septic arthritis risks inadequate antimicrobial or surgical treatment, repeated surgical procedures, and longer hospital stays.^{52,55} Given the difficulty of obtaining an MRI in young children at some facilities and the small size of studies recommending universal application, a decision about preoperative MRI can be made in conjunction with an orthopedic consultation.

Management

Surgical Management. Emergent orthopedic consultation and surgical intervention are necessary. The long time paradigm has been that septic arthritis of the hip should be immediately surgically drained

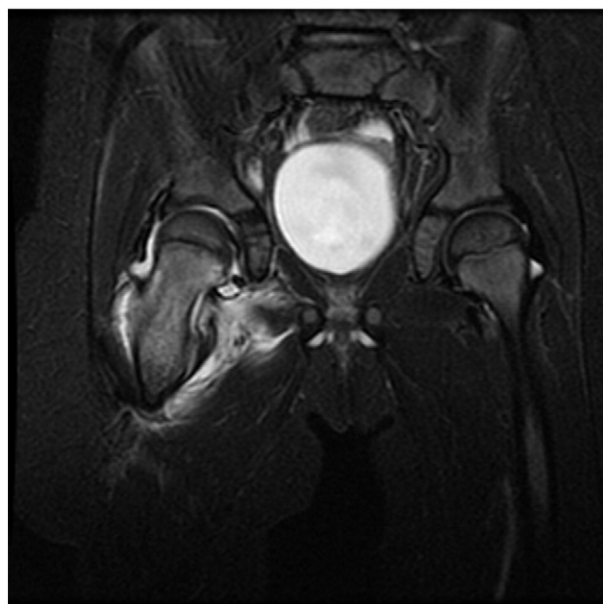


Figure 12. Magnetic resonance imaging of a patient with a right-sided septic hip joint. Short T1 inversion recovery. The image showed a moderate right hip effusion as well as bone marrow edema and surrounding myositis.

through open arthrotomy.⁸¹⁻⁸⁷ Recent systematic review suggests that arthroscopic management of the hip may be an acceptable alternative.^{88,89}

Antibiotic Therapy. One recommendation in patients with osteoarticular infections is to delay antibiotics until tissue cultures (either synovial fluid in septic arthritis or bone aspirate in osteoarthritis) are obtained.^{90,91} If antibiotics are thought to be immediately required preoperatively due to overt sepsis or patient instability, concurrent orthopedic consultation is recommended.

Current guidelines for septic arthritis recommend a short course of intravenous antibiotics followed by an oral course.^{43,92} Reduction in CRP is often used as indication to allow transition from intravenous to oral antibiotics.⁹³

The chosen antibiotic should have adequate tissue penetration as well as be effective against the isolated or presumed microorganism. *Staphylococcus aureus*, *Streptococcus pyogenes*, and *K kingae* are the most commonly isolated bacteria from osteoarticular infections. *S aureus* is typically the most prevalent organism cultured.⁴³ Recent studies have shown that, with PCR analysis, *K kingae* is the most common cause of osteoarticular infections in children younger than 4 years.^{60,94} Methicillin-resistant *S aureus* has been increasingly implicated in osteoarticular infections and often portends a more severe and complicated course.⁹⁵ Other organisms can occur particularly in specific populations, such as *Salmonella* osteomyelitis in patients with sickle cell disease.⁹⁶ *Shigella*, *Salmonella*, *Campylobacter*, and *Yersinia* have been isolated in cases of septic arthritis following episodes of infectious diarrhea.⁹⁷ *Neisseria gonorrhea* has high selectivity for the synovium and has been implicated in monoarthritis and polyarthritis in adolescents.⁴³

An empiric antibiotic regimen for an osteoarticular infection is nafcillin or oxacillin or cefazolin to cover methicillin-sensitive *S aureus*, *S pyogenes*, and *K kingae*. If covering methicillin-resistant *S aureus*, the antibiotic of choice is clindamycin or vancomycin. Neither vancomycin nor clindamycin is effective against *K kingae*, so cefazolin would need to be added.⁵⁷ Empiric antibiotics would be different in the special situations described above. Any antibiotic regimen should be narrowed once the species is identified.

Lyme Arthritis

There are several case reports of hip monoarthritis due to Lyme (*Borrelia burgdorferi*) infection. One study of almost 400 patients in a Lyme-endemic

area noted that 5% of the patients who presented with acute nontraumatic hip pain had a diagnosis of Lyme arthritis.⁹⁸ The authors concluded that routine testing of patients thought to have transient synovitis is unlikely to be helpful; however, if the patient is thought to have septic arthritis, sending serum Lyme serologies is warranted.

Epidemiology

The Centers for Disease Control and Prevention estimates that there are more than 35 000 cases of Lyme disease in the United States each year as of 2013, with cases concentrated in the northeast, but occurring throughout the country.⁹⁹ Lyme disease is most frequent in children 5 to 15 years of age.¹⁰⁰

Clinical History and Examination

More than a third of children with Lyme disease present with a sign of dissemination as the presenting manifestation, including erythema migrans, arthritis, facial palsy, meningitis, or carditis. Lyme disease can cause brief intermittent attacks of swelling and pain in 1 or more joints—primarily large joints.¹⁰¹ The mean incubation after a tick bite is 3.4 months with a wide range (2 weeks to 2 years).¹⁰² Lyme arthritis can present at any time of the year.

Like septic arthritis of the hip, pain of the hip joint can be elicited with range of motion. In most cases, however, the hip pain in Lyme is not severe enough to debilitate or prevent weight bearing, in contrast to the complete refusal to bear weight in septic arthritis.¹⁰³ Of note, lack of history of the classic erythema migrans rash does not preclude diagnosis of Lyme arthritis, as a significant number of patients never have the rash.

Diagnosis

Lyme disease can be a challenging diagnosis to make, and Lyme arthritis of the hip can be particularly difficult. There are no laboratory criteria that have been reported to help differentiate Lyme from septic arthritis of the hip. Patients have a wide range of presentations, from mild hip discomfort, to fever and ill appearance. It is therefore a difficult diagnosis to make without other Lyme manifestations on examination or history. It is also difficult to separate septic arthritis from Lyme arthritis based on synovial fluid analysis alone. In a recent study of 46 children with hip synovial fluid white blood cell values of 25 000 to 75 000 cells/mm³, 28% had Lyme arthritis.⁷⁰ If a patient is being evaluated for septic arthritis in a Lyme-endemic area, Lyme serologies should be sent. The laboratory test to confirm the diagnosis of Lyme disease is a serum Lyme enzyme-linked immunosorbent assay

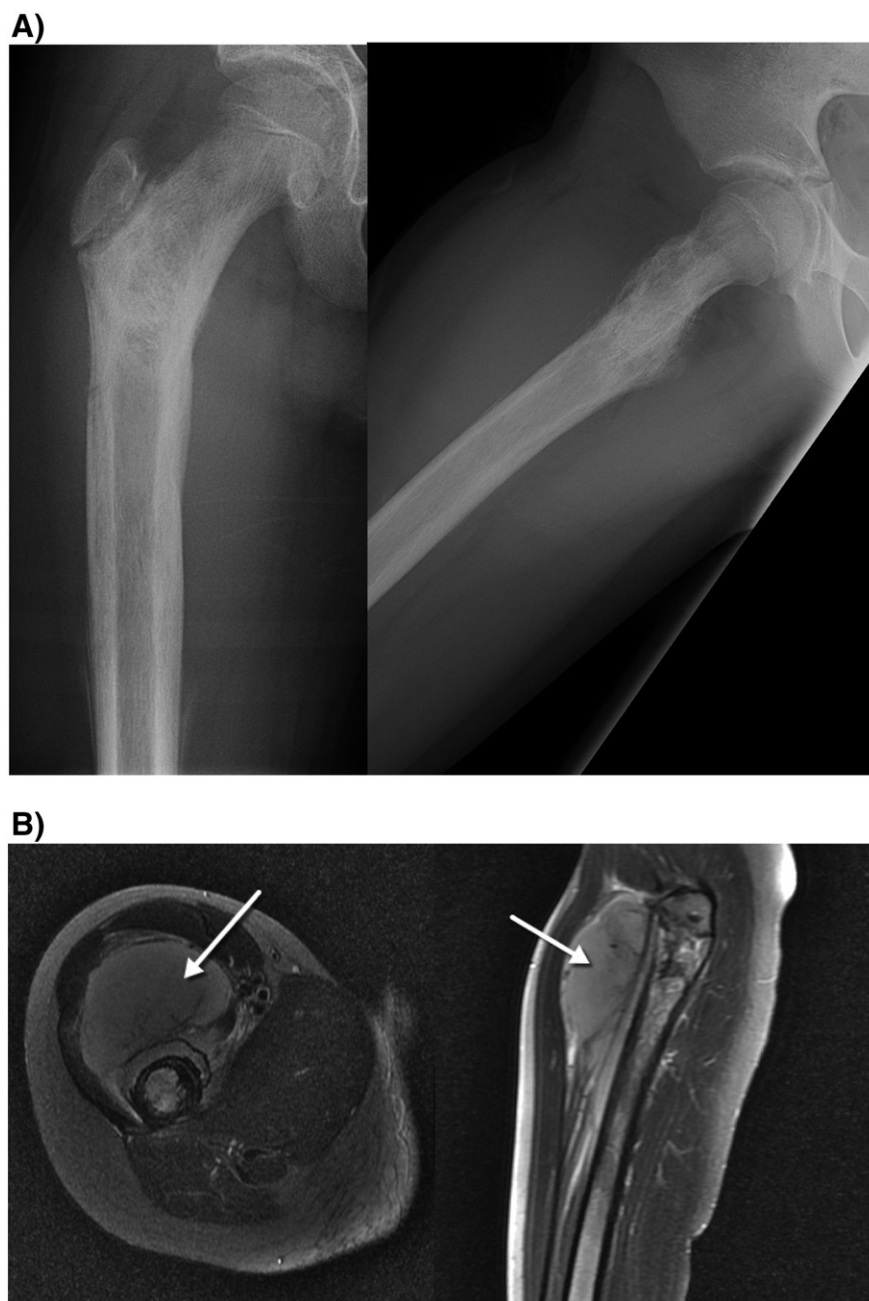


Figure 13. Ewing sarcoma in an adolescent with limp, radiographs, and corresponding MRI demonstrating Ewing sarcoma of the proximal femur, with a destructive pattern, mainly at the intertrochanteric and proximal shaft. Periosteal reaction is interrupted and aggressive. A large soft tissue mass is also present.

with a reflex Western blot performed if the enzyme-linked immunosorbent assay is positive.

Management

Lyme disease management is composed of antibiotic treatment and assessment for other manifestations of Lyme disease infection such as carditis and facial nerve palsy. Appropriate antibiotics for Lyme arthritis are amoxicillin in children younger

than 8 years and doxycycline in patients 8 years and older.¹⁰⁴

Malignancy

Malignancy is an infrequent cause of nontraumatic hip pathology. Osteosarcomas are the most common primary bone cancer, and Ewing sarcomas are the second most common. Both peak in

adolescence and can occur in the hip region.¹⁰⁵ Leukemia can present with leg pain and limp due to bone marrow expansion.

Clinical Examination and History

With osteosarcoma or Ewing sarcoma of the hip, the patient may present with tumor-related pain with or without a mass, a painless mass, or a pathologic fracture. The most common presenting symptoms are pain (70%), a palpable mass (5%), or both (25%). The pain tends to be intermittent and exacerbated by activity.¹⁰⁶ Of note, patients may report their pain began at the time of minor musculoskeletal injury (approximately 25% of patients report this), and it may delay the diagnosis.¹⁰⁶ Constitutional symptoms, such as fever and weight loss, are uncommon at presentation.

Leukemia can present with lower extremity complaints. A group presented a series of 9 patients with a chief complaint of limp found to have leukemia. They also retrospectively reviewed their leukemia population of 77 patients and reported that 11.6% had limp and hip or knee pain at diagnosis.¹⁰⁷ Contrary to the other patients with hip complaints, however, most of them had systemic symptoms including bruising, lymphadenopathy, and hepatosplenomegaly.

Diagnosis

Plain radiographs are usually the first line. The classic radiologic findings in osteosarcoma are an irregular boundary between the tumor and normal bone and a sunburst pattern (linear new bone growth perpendicular to the bony cortex).¹⁰⁸ In Ewing sarcoma, the typical finding is an onionskin pattern (linear new bone growth parallel to the cortex)¹⁰⁹ (Figure 13).

Laboratory Studies

Laboratory studies are often normal in osteosarcoma or Ewing sarcoma. Basic laboratory studies, in particular the complete blood count, are helpful to diagnose leukemia when bone marrow suppression or peripheral blasts are present.

Management

If there is any suspicion for malignancy, immediate referral to a pediatric oncologist is warranted for discussion of further workup recommendations and family counseling.

SUMMARY

Most children with a nontraumatic hip complaint are diagnosed with transient synovitis; however,

there are serious conditions that must be differentiated including osteoarticular infections (septic arthritis and osteomyelitis), Legg-Calves-Perthes disease, slipped capital femoral epiphysis, and rarely malignancy. The common hip pathologies were reviewed in detail, along with distinction of the most common and those that require emergent intervention.

ACKNOWLEDGEMENTS

Jennifer Marin, MD, MSc, for her contributions of ultrasound images and to the content of the ultrasound section.

REFERENCES

1. Zitelli GJ, McIntire S, Nowalk AJ. Zitelli and Davis' atlas of pediatric physical diagnosis. Philadelphia, PA: Saunders/Elsevier; 2012.
2. Baskett A, Hosking J, Aickin R. Hip radiography for the investigation of nontraumatic, short duration hip pain presenting to a children's emergency department. *Pediatr Emerg Care* 2009;25:78–82.
3. Adam R, Hendry GM, Moss J, et al. Arthrosonography of the irritable hip in childhood: a review of 1 year's experience. *Br J Radiol* 1986;59:205–8.
4. Plumb J, Mallin M, Bolte RG. The role of ultrasound in the emergency department evaluation of the acutely painful pediatric hip. *Pediatr Emerg Care* 2015;31:54–8.
5. Vieira RL, Levy JA. Bedside ultrasonography to identify hip effusions in pediatric patients. *Ann Emerg Med* 2010;55:284–9.
6. Riera A, Chen L. Orthopedics: extremity fractures, reductions, and arthrocentesis. In: Doniger SJ, editor. *Pediatric emergency and critical care ultrasound*. Cambridge, England: Cambridge University Press; 2014. p. 285–6.
7. Tsung JW, Blaivas M. Emergency department diagnosis of pediatric hip effusion and guided arthrocentesis using point-of-care ultrasound. *J Emerg Med* 2008;35:393–9.
8. Chasm RM, Swencki SA. Pediatric orthopedic emergencies. *Emerg Med Clin North Am* 2010;28:907–26.
9. Dubois-Ferriere V, Belaieff W, Lascombes P, et al. Transient synovitis of the hip: which investigations are truly useful? *Swiss Med Wkly* 2015;145:14176.
10. Fabry G. Clinical practice: the hip from birth to adolescence. *Eur J Pediatr* 2010;169:143–8.
11. Nouri A, Walmsley D, Pruszczyński B, et al. Transient synovitis of the hip: a comprehensive review. *J Pediatr Orthop B* 2014;23:32–6.
12. Krul M, van der Wouden JC, Schellevis FG, et al. Acute non-traumatic hip pathology in children: incidence and presentation in family practice. *Fam Pract* 2010;27:166–70.
13. Ehrendorfer S, LeQuesne G, Penta M, et al. Bilateral synovitis in symptomatic unilateral transient synovitis of the hip: an ultrasonographic study in 56 children. *Acta Orthop Scand* 1996;67:149–52.
14. Landin LA, Danielsson LG, Wattsgard C. Transient synovitis of the hip. Its incidence, epidemiology and relation to Perthes' disease. *J Bone Joint Surg (Br)* 1987;69:238–42.
15. Kastrissianakis K, Beattie TF. Transient synovitis of the hip: more evidence for a viral aetiology. *Eur J Emerg Med* 2010;17:270–3.

16. Miralles M, Gonzalez G, Pulpeiro JR, et al. Sonography of the painful hip in children: 500 consecutive cases. *Am J Roentgenol* 1989;152:579–82.
17. Craig JG. Infection: ultrasound-guided procedures. *Radiol Clin North Am* 1999;37:669–78.
18. Do TT. Transient synovitis as a cause of painful limps in children. *Curr Opin Pediatr* 2000;12:48–51.
19. Skinner J, Glancy S, Beattie TF, et al. Transient synovitis: is there a need to aspirate hip joint effusions? *Eur J Emerg Med* 2002;9:15–8.
20. Kermond S, Fink M, Graham K, et al. A randomized clinical trial: should the child with transient synovitis of the hip be treated with nonsteroidal anti-inflammatory drugs? *Ann Emerg Med* 2002;40:294–9.
21. Uziel Y, Butbul-Aviel Y, Barash J, et al. Recurrent transient synovitis of the hip in childhood. Longterm outcome among 39 patients. *J Rheumatol* 2006;33:810–1.
22. Mukamel M, Litmanovitch M, Yosipovich Z, et al. Legg-Calve-Perthes disease following transient synovitis. How often? *Clin Pediatr* 1985;24:629–31.
23. Joseph B, Varghese G, Mulpuri K, et al. Natural evolution of Perthes disease: a study of 610 children under 12 years of age at disease onset. *J Pediatr Orthop* 2003;23:590–600.
24. Shah H. Perthes disease: evaluation and management. *Orthop Clin North Am* 2014;45:87–97.
25. Mazloumi SM, Ebrahimpzadeh MH, Kachooei AR. Evolution in diagnosis and treatment of Legg-Calve-Perthes disease. *Arch Bone Jt Surg* 2014;2:86–92.
26. Perry DC, Machin DM, Pope D, et al. Racial and geographic factors in the incidence of Legg-Calve-Perthes' disease: a systematic review. *Am J Epidemiol* 2012;175:159–66.
27. Lee JH, Zhou L, Kwon KS, et al. Role of leptin in Legg-Calve-Perthes disease. *J Orthop Res* 2013;31:1605–10.
28. Woratanarat P, Thaveeratitharm C, Woratanarat T, et al. Meta-analysis of hypercoagulability genetic polymorphisms in Perthes disease. *J Orthop Res* 2014;32:1–7.
29. Stulberg SD, Cooperman DR, Wallensten R. The natural history of Legg-Calve-Perthes disease. *J Bone Joint Surg Am* 1981;63:1095–108.
30. Milani C, Dobashi ET. Arthrogram in Legg-Calve-Perthes disease. *J Pediatr Orthop* 2011;31:S156–62.
31. Du J, Lu A, Dempsey M, et al. MR perfusion index as a quantitative method of evaluating epiphyseal perfusion in Legg-Calve-Perthes disease and correlation with short-term radiographic outcome: a preliminary study. *J Pediatr Orthop* 2013;33:707–13.
32. Joseph B, Price CT. Consensus statements on the management of Perthes disease. *Orthop Clin North Am* 2011;42:437–40.
33. Georgiadis AG, Zaltz I. Slipped capital femoral epiphysis: how to evaluate with a review and update of treatment. *Pediatr Clin North Am* 2014;61:1119–35.
34. Loder RT, Starnes T, Dikos G. Atypical and typical (idiopathic) slipped capital femoral epiphysis. Reconfirmation of the age-weight test and description of the height and age-height tests. *J Bone Joint Surg Am* 2006;88:1574–81.
35. Nasreddine AY, Heyworth BE, Zurakowski D, et al. A reduction in body mass index lowers risk for bilateral slipped capital femoral epiphysis. *Clin Orthop Relat Res* 2013;471:2137–44.
36. Riad J, Bajelidze G, Gabos PG. Bilateral slipped capital femoral epiphysis: predictive factors for contralateral slip. *J Pediatr Orthop* 2007;27:411–4.
37. Larson AN, Yu EM, Melton LJ, et al. Incidence of slipped capital femoral epiphysis: a population-based study. *J Pediatr Orthop B* 2010;19:9–12.
38. Baghdadi YM, Larson AN, Sierra RJ, et al. The fate of hips that are not prophylactically pinned after unilateral slipped capital femoral epiphysis. *Clin Orthop Relat Res* 2013;471:2124–31.
39. Loder RT, Skopelja EN. The epidemiology and demographics of slipped capital femoral epiphysis. *ISRN Orthop* 2011;2011:486–512.
40. Loder RT, Wittenberg B, DeSilva G. Slipped capital femoral epiphysis associated with endocrine disorders. *J Pediatr Orthop* 1995;15:349–56.
41. Matava MJ, Patton CM, Luhmann S, et al. Knee pain as the initial symptom of slipped capital femoral epiphysis: an analysis of initial presentation and treatment. *J Pediatr Orthop* 1999;19:455–60.
42. Kocher MS, Bishop JA, Weed B, et al. Delay in diagnosis of slipped capital femoral epiphysis. *Pediatrics* 2004;113:e322–5.
43. Tanwar YS, Jaiswal A, Singh S, et al. Acute pediatric septic arthritis: a systematic review of literature and current controversies. *Pol Orthop Traumatol* 2014;79:23–9.
44. Cowell HR. The significance of early diagnosis and treatment of slipping of the capital femoral epiphysis. *Clin Orthop Relat Res* 1966;48:89–94.
45. Klein A, Joplin RJ, Reidy JA, et al. Roentgenographic features of slipped capital femoral epiphysis. *Am J Roentgenol Radium Ther* 1951;66:361–74.
46. Green DW, Mogekwu N, Scher DM, et al. A modification of Klein's line to improve sensitivity of the anterior-posterior radiograph in slipped capital femoral epiphysis. *J Pediatr Orthop* 2009;29:449–53.
47. Peck K, Herrera-Soto J. Slipped capital femoral epiphysis: what's new? *Orthop Clin North Am* 2014;45:77–86.
48. Aronson DD, Carlson WE. Slipped capital femoral epiphysis. A prospective study of fixation with a single screw. *J Bone Joint Surg Am* 1992;74:810–9.
49. Loder RT, Dietz FR. What is the best evidence for the treatment of slipped capital femoral epiphysis? *J Pediatr Orthop* 2012;32:S158–65.
50. Fabry G, Meire E. Septic arthritis of the hip in children: poor results after late and inadequate treatment. *J Pediatr Orthop* 1983;3:461–6.
51. Steer AC, Carapetis JR. Acute hematogenous osteomyelitis in children: recognition and management. *Paediatr Drugs* 2004;6:333–46.
52. Monsalve J, Kan JH, Schallert EK, et al. Septic arthritis in children: frequency of coexisting unsuspected osteomyelitis and implications on imaging work-up and management. *Am J Roentgenol* 2015;204:1289–95.
53. Montgomery CO, Siegel E, Blasier RD, et al. Concurrent septic arthritis and osteomyelitis in children. *J Pediatr Orthop* 2013;33:464–7.
54. Perlman MH, Patzakis MJ, Kumar PJ, et al. The incidence of joint involvement with adjacent osteomyelitis in pediatric patients. *J Pediatr Orthop* 2000;20:40–3.
55. Rosenfeld S, Bernstein D, Daram S, et al. Predicting the presence of adjacent infections in septic arthritis in children. *J Pediatr Orthop* 2016;36:70–4.
56. Dubost JJ, Fis I, Denis P, et al. Polyarticular septic arthritis. *Medicine (Baltimore)* 1993;72:296–310.
57. Agarwal A, Aggarwal AN. Bone and joint infections in children: acute hematogenous osteomyelitis. *Indian J Pediatr* 2015 [epub ahead of print].
58. Ceroni D, Cherkaoui A, Ferey S, et al. *Kingella kingae* osteoarticular infections in young children: clinical features and contribution of a new specific real-time PCR assay to the diagnosis. *J Pediatr Orthop* 2010;30:301–4.

59. Williams N, Cooper C, Cundy P. *Kingella kingae* septic arthritis in children: recognising an elusive pathogen. *J Child Orthop* 2014;8:91–5.
60. Ilharreborde B, Bidet P, Lorrot M, et al. New real-time PCR-based method for *Kingella kingae* DNA detection: application to samples collected from 89 children with acute arthritis. *J Clin Microbiol* 2009;47:1837–41.
61. Carter K, Doern C, Jo C, et al. The clinical usefulness of polymerase chain reaction as a supplemental diagnostic tool in the evaluation and the treatment of children with septic arthritis. *J Pediatr Orthop* 2016;36:167–72.
62. Section J, Gibbons SD, Barton T, et al. Microbiological culture methods for pediatric musculoskeletal infection: a guideline for optimal use. *J Bone Joint Surg Am* 2015;97:441–9.
63. Kocher MS, Zurakowski D, Kasser JR. Differentiating between septic arthritis and transient synovitis of the hip in children: an evidence-based clinical prediction algorithm. *J Bone Joint Surg Am* 1999;81:1662–70.
64. Uzoigwe CE. Another look: is there a flaw to current hip septic arthritis diagnostic algorithms? *Clin Orthop Relat Res* 2014;472:1645–51.
65. Caird MS, Flynn JM, Leung YL, et al. Factors distinguishing septic arthritis from transient synovitis of the hip in children. A prospective study. *J Bone Joint Surg Am* 2006;88:1251–7.
66. Sultan J, Hughes PJ. Septic arthritis or transient synovitis of the hip in children: the value of clinical prediction algorithms. *J Bone Joint Surg (Br)* 2010;92:1289–93.
67. Singhal R, Perry DC, Khan FN, et al. The use of CRP within a clinical prediction algorithm for the differentiation of septic arthritis and transient synovitis in children. *J Bone Joint Surg (Br)* 2011;93:1556–61.
68. Yagupsky P, Porsch E, St Geme JW. *Kingella kingae*: an emerging pathogen in young children. *Pediatrics* 2011;127:557–65.
69. Nade S. Acute septic arthritis in infancy and childhood. *J Bone Joint Surg (Br)* 1983;65:234–41.
70. Heyworth BE, Shore BJ, Donahue KS, et al. Management of pediatric patients with synovial fluid white blood-cell counts of 25,000 to 75,000 cells/mm³ after aspiration of the hip. *J Bone Joint Surg Am* 2015;97:389–95.
71. Blickman JG, van Die CE, de Rooy JW. Current imaging concepts in pediatric osteomyelitis. *Eur Radiol* 2004;14:L55–64.
72. Capitanio MA, Kirkpatrick JA. Early roentgen observations in acute osteomyelitis. *Am J Roentgenol Radium Ther Nucl Med* 1970;108:488–96.
73. Zamzam MM. The role of ultrasound in differentiating septic arthritis from transient synovitis of the hip in children. *J Pediatr Orthop B* 2006;15:418–22.
74. McGoldrick F, Bourke T, Blake N, et al. Accuracy of sonography in transient synovitis. *J Pediatr Orthop* 1990;10:501–3.
75. Kothari NA, Pelchovitz DJ, Meyer JS. Imaging of musculoskeletal infections. *Radiol Clin North Am* 2001;39:653–71.
76. Hopkins KL, Li KC, Bergman G. Gadolinium-DTPA-enhanced magnetic resonance imaging of musculoskeletal infectious processes. *Skeletal Radiol* 1995;24:325–30.
77. Kanal E, Burk DL, Brunberg JA, et al. Pediatric musculoskeletal magnetic resonance imaging. *Radiol Clin North Am* 1988;26:211–39.
78. Lee SK, Suh KJ, Kim YW, et al. Septic arthritis versus transient synovitis at MR imaging: preliminary assessment with signal intensity alterations in bone marrow. *Radiology* 1999;211:459–65.
79. Mazur JM, Ross G, Cummings J, et al. Usefulness of magnetic resonance imaging for the diagnosis of acute musculoskeletal infections in children. *J Pediatr Orthop* 1995;15:144–7.
80. Pugmire BS, Shailam R, Gee MS. Role of MRI in the diagnosis and treatment of osteomyelitis in pediatric patients. *World J Radiol* 2014;6:530–7.
81. Shaw BA, Kasser JR. Acute septic arthritis in infancy and childhood. *Clin Orthop Relat Res* 1990;257:212–25.
82. Sucato DJ, Schwend RM, Gillespie R. Septic arthritis of the hip in children. *J Am Acad Orthop Surg* 1997;5:249–60.
83. Chen CE, Ko JY, Li CC, et al. Acute septic arthritis of the hip in children. *Arch Orthop Trauma Surg* 2001;121:521–6.
84. Betz RR, Cooperman DR, Wopperer JM, et al. Late sequelae of septic arthritis of the hip in infancy and childhood. *J Pediatr Orthop* 1990;10:365–72.
85. Choi IH, Pizzutillo PD, Bowen JR, et al. Sequelae and reconstruction after septic arthritis of the hip in infants. *J Bone Joint Surg Am* 1990;72:1150–65.
86. Vidigal Jr EC, Vidigal EC, Fernandes JL. Avascular necrosis as a complication of septic arthritis of the hip in children. *Int Orthop* 1997;21:389–92.
87. Hallel T, Salvati EA. Septic arthritis of the hip in infancy: end result study. *Clin Orthop Relat Res* 1978;132:115–28.
88. de Sa D, Cargnelli S, Catapano M, et al. Efficacy of hip arthroscopy for the management of septic arthritis: a systematic review. *Arthroscopy* 2015;31:1358–70.
89. Fernandez FF, Langendorfer M, Wirth T, et al. Treatment of septic arthritis of the hip in children and adolescents. *Z Orthop Unfall* 2013;151:596–602.
90. Al-Mayahi M, Cian A, Lipsky BA, et al. Administration of antibiotic agents before intraoperative sampling in orthopedic infections alters culture results. *J Infect* 2015;71:518–25.
91. MacLean SB, Timmis C, Evans S, et al. Preoperative antibiotics for septic arthritis in children: delay in diagnosis. *J Orthop Surg* 2015;23:80–3.
92. Keren R, Shah SS, Srivastava R, et al. Comparative effectiveness of intravenous vs oral antibiotics for post-discharge treatment of acute osteomyelitis in children. *JAMA Pediatr* 2015;169:120–8.
93. Chou AC, Mahadev A. The Use of C-reactive Protein as a Guide for Transitioning to Oral Antibiotics in Pediatric Osteoarticular Infections. *J Pediatr Orthop* 2016;36:173–7.
94. Afghani B, Kong V, Wu FL. What would pediatric infectious disease consultants recommend for management of culture-negative acute hematogenous osteomyelitis? *J Pediatr Orthop* 2007;27:805–9.
95. Sarkissian EJ, Gans I, Gunderson MA, et al. Community-acquired methicillin-resistant *Staphylococcus aureus* musculoskeletal infections: emerging trends over the past decade. *J Pediatr Orthop* 2015 [epub ahead of print].
96. da Silva Jr GB, Daher Ede F, da Rocha FA. Osteoarticular involvement in sickle cell disease. *Rev Bras Hematol Hemoter* 2012;34:156–64.
97. Fryden A, Bengtsson A, Foberg U, et al. Early antibiotic treatment of reactive arthritis associated with enteric infections: clinical and serological study. *Br Med J* 1990;301:1299–302.

98. Bachur RG, Adams CM, Monuteaux MC. Evaluating the child with acute hip pain ("irritable hip") in a Lyme endemic region. *J Pediatr* 2015;166:407–11 [e1].
99. Centers for Disease Control and Prevention. Reported cases of Lyme disease by year, United States, 1995-2013. Available at: <http://www.cdc.gov/lyme/stats/chartstables/casesbyyear.html>. Accessed: 8–24-2015.
100. Mead PS. Epidemiology of Lyme disease. *Infect Dis Clin North Am* 2015;29:187–210.
101. Sood SK. Lyme disease in children. *Infect Dis Clin North Am* 2015;29:281–94.
102. Szer IS, Taylor E, Steere AC. The long-term course of Lyme arthritis in children. *N Engl J Med* 1991;325:159–63.
103. Davidson RS. Orthopaedic complications of Lyme disease in children. *Biomed Pharmacother* 1989;43:405–8.
104. Kimberlin DW, Brady MT, Jackson MA, et al, editors. *Red Book*. 30th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2015.
105. Bleyer A, O'Leary M, Barr R, et al, editors. *Cancer epidemiology in older adolescents and young adults 15 to 29 years of age, including SEER incidence and survival: 1975-2000*. Bethesda, MD: National Cancer Institute; 2006.
106. Widhe B, Widhe T. Initial symptoms and clinical features in osteosarcoma and Ewing sarcoma. *J Bone Joint Surg Am* 2000;82:667–74.
107. Tuten HR, Gabos PG, Kumar SJ, et al. The limping child: a manifestation of acute leukemia. *J Pediatr Orthop* 1998;18: 625–9.
108. Gross M, Stevens K. Sunburst periosteal reaction in osteogenic sarcoma. *Pediatr Radiol* 2005;35: 647–8.
109. Reinus WR, Gilula LA, Shirley SK, et al. Radiographic appearance of Ewing sarcoma of the hands and feet: report from the Intergroup Ewing Sarcoma Study. *Am J Roentgenol* 1985;144:331–6.