Review Article

Hip Instability

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Abstract

Understanding of the etiology and pathology of hip instability has increased in recent years as new information has emerged regarding the disease processes of the hip. Hip instability, heretofore considered uncommon in clinical practice, is increasingly recognized as a pathologic entity. Instability may be classified as traumatic or atraumatic, and diagnosis is made based on patient history, physical examination, and imaging studies. Plain radiography, MRI, MRI arthrography, and hip instability tests (eg, posterior impingement, dial) can be used to confirm the presence of instability. Nonsurgical management options include physical therapy and protected weight bearing. Surgical intervention, whether arthroscopic or open, is required for large acetabular fractures and refractory instability. Knowledge of the etiology and evolving research of hip instability is essential to understand the spectrum of hip disease.

Historically, the hip has been considered one of the most stable joints. Hip instability has been assumed to be isolated to overt cases of high-energy trauma, and it may be missed in atraumatic cases. Research on hip arthroscopy, femoroacetabular impingement (FAI), hip dysplasia, labral tears, chondral defects, capsular disease, and other hip-related conditions has led to greater awareness of hip instability. Although controversy remains regarding diagnosis, imaging, and management, awareness of hip instability and evolving research on the topic is essential in understanding the spectrum of hip disease.

Anatomy and Biomechanics

Bony Anatomy

The normal hip is an inherently stable joint, and significant force is required to dislocate it. The bony acetabulum creates a quasi-hemisphere that allows 170° of femoral head coverage.[1] It is oriented with 48° of lateral cephalad tilt in the coronal plane and 21° of anterior tilt in the sagittal plane.[2] This orientation yields more posterior coverage than anterior coverage, which creates greater reliance on soft tissues for anterior stability while allowing more flexion than extension. Bony abnormalities of the acetabulum, as seen in patients with developmental dysplasia of the hip (DDH) or with a retroverted acetabulum and posterior insufficiency, may predispose to instability. The femoral neck axis is in 10° of anteversion from the transcondylar axis and 130° of superior inclination from the femoral shaft axis. Recently, there has been a resurgence of interest in proximal femoral anatomy because it has been theorized that subtle abnormalities in the head and neck anatomy can lead to damage of the soft-tissue stabilizers of the hip, resulting in potential instability and eventual osteoarthritis.[3-5]

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J Am Acad Orthop Surg 2011;19: 340-349

Soft-tissue Anatomy

The labrum is a triangular fibrocartilaginous structure that runs circumferentially along the acetabulum and attaches to the outer rim of the bony acetabulum. Longitudinal fibers run parallel to the acetabular rim and provide tensile stability similar to that of the meniscus in the knee.[6] Biomechanically, the labrum extends the acetabular coverage of the femoral head past its equator. Although this extension does not typically involve load transmission, the labrum augments stability by enhancing the negative intra-articular pressure, acting as a tension band, and participating in nociception and proprioception.[5,7] Most of the labrum is avascular and has limited ability to heal. A torn labrum that does not heal may contribute to instability.[5]

The iliofemoral, pubofemoral, and ischiofemoral ligaments are adjacent to the hip capsule and help to stabilize the hip. The iliofemoral ligament (ie, Y ligament, ligament of Bigelow) runs from the anteroinferior iliac spine to the femoral neck, forming a fan-shaped "Y" at its insertion proximally and distally along the intertrochanteric line.[8] The iliofemoral ligament is the strongest of the three ligaments and is taut in extension and external rotation of the hip as it resists anterior translation. The pubofemoral ligament arises from the pubis and inserts on the neck of the femur, providing resistance to hyperextension and hyperabduction.[9] Fibers from the medial arm of the iliofemoral ligament combine with the pubofemoral ligament to form the zona orbicularis (ie, annular ligament); however, whether the annular ligament encircles the entire femoral neck or has only a posterior contribution remains a matter of debate.[9,10] Posteriorly, the ischiofemoral ligament creates a spiral pattern around the neck of the femur. This ligament becomes loose with flexion and tight with extension (Figure 1). The hip is most stable in full extension.

The ligamentum teres arises from mesoderm originally associated with the transverse acetabular ligament and inserts on the fovea of the femoral head inferior and posterior to its center. The functional involvement of the ligamentum teres remains unclear; its role in mechanical stability is debated.[11-13] Arthroscopic evaluation shows this ligament to be taut in external rotation and lax in internal rotation[14] (Figure 2). Several studies have suggested the importance of the ligamentum teres in hip stability. Hypertrophy of this ligament has been reported in instances of acetabular dysplasia and osteonecrosis of the hip.[13,15] An animal study demonstrated decreased hip stability with sacrifice of the ligamentum teres.[16] Other studies have shown the presence of free nerve endings within the structure of the ligamentum teres.[17,18]

The iliopsoas is another extracapsular entity that provides structural support. It acts as a dynamic stabilizer of the hip as it crosses the anterior aspect of the hip capsule.[14]

Diagnosis

History

A thorough history is essential in determining the underlying etiology of suspected hip instability. The differential diagnosis is based primarily on events surrounding the onset of symptoms and activities that reproduce the complaints (Table 1). A history of clicking, locking, catching, giving way, or pain elicited by positions that reproduce instability should be investigated. Certain sports are associated with an increased incidence of hip dislocation, such as football, rugby, bicycling, skiing, dancing, and hockey, and athletes who participate in these sports should be evaluated accordingly.[19,20] Medical and family histories should be investigated for connective tissue disorders such as Ehlers-Danlos syndrome, arthrochalasis multiplex congenita, Marfan syndrome, and Down syndrome. Referred

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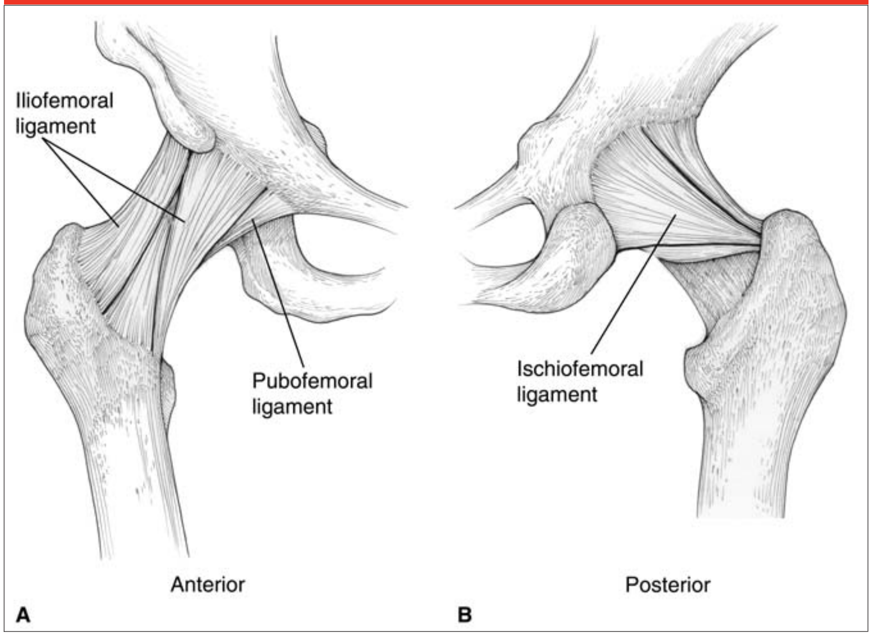
Figure 1



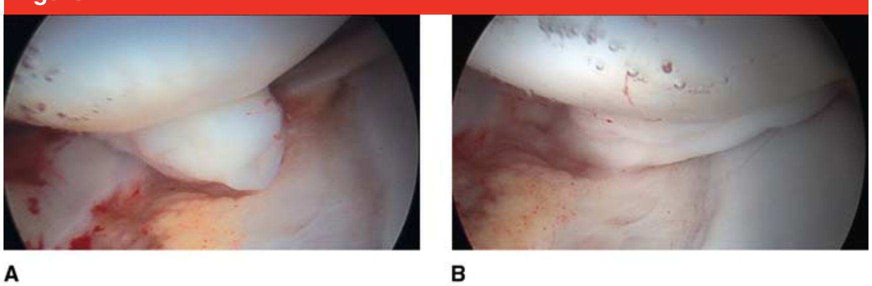
Illustrations demonstrating the anatomic constraints of the hip. **A,** The anterior ligamentous constraints of the hip include the iliofemoral and pubofemoral ligaments. **B,** The ischiofemoral ligament is the primary posterior restraint. (Redrawn with permission from Kelly BT, Williams RJ III, Philippon MJ: Hip arthroscopy: Current indications, treatment options, and management issues. *Am J Sports Med* 2003;31[6]:1020-1037.)

Table 1

Differential Diagnosis of Hip Instability

Traumatic instability
Major traumatic event
Fracture-dislocation of the acetabulum
Fracture-dislocation of the femoral head
Pure dislocation of the hip (± labral injury)
Minor traumatic event
Pure dislocation of the hip (± labral injury)
Subluxation of the hip (± osseous or labral injury)
Repetitive microtrauma
Atraumatic instability
Developmental dysplasia of the hip
Femoroacetabular impingement (± labral injury)
Congenital ligamentous laxity
Down syndrome
Ehlers-Danlos syndrome
Marfan syndrome
Arthrochalasis multiplex congenita
Acquired ligamentous laxity
Idiopathic ligamentous laxity
Iatrogenic ligamentous laxity

Figure 2



Arthroscopic images demonstrating a normal ligamentum teres with the hip in neutral position (**A**) and tightening of the ligament on external rotation (**B**).

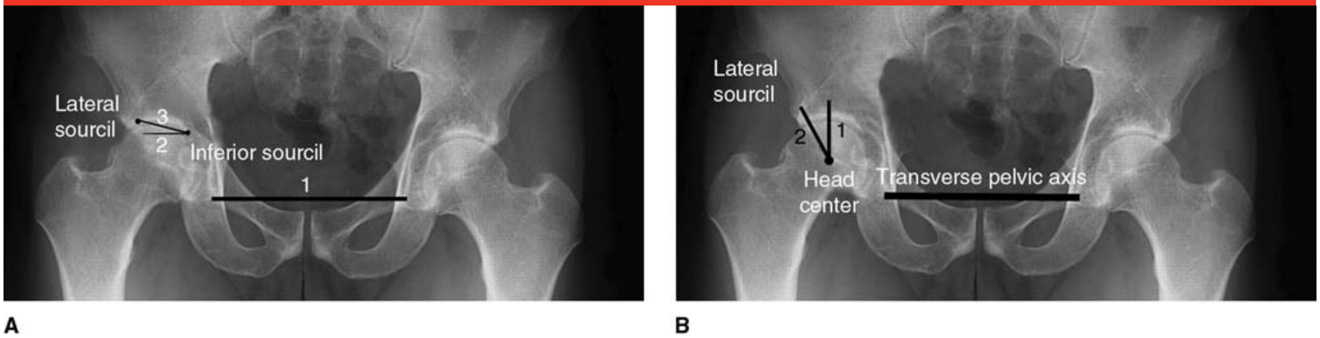
pain from the lumbar spine and radicular symptoms may be confused with primary hip pathology. Gastrointestinal, vascular, and genitourinary disease may masquerade as hip pain and should be considered, especially in the older patient.[21]

Physical Examination

The physical workup for hip instability includes evaluation of gait, posture, range of motion (ROM), and motor and neurovascular function. A full neurovascular examination of the limb and ROM should be undertaken, and pain should be noted. Increased ROM in the presence of capsular laxity is considered true instability only when the patient is symptomatic. Audible pops and other mechanical noises may be most notable as the hip is taken from flexion to extension. These sounds are indicative of labral pathology,[22] a loose body, or snapping of the iliopsoas tendon.[23] The patient also should be evaluated for signs of generalized ligamentous laxity, including the ability to bring the thumb to the radial aspect of the forearm, recurvatum of the knees, hyperextension of the elbows and metacarpophalangeal joints, and the ability to voluntarily dislocate or subluxate the hip.[1]

Specific tests are used to evaluate hip stability. For the posterior impingement test, the patient lies supine, and the examiner places the patient's hip in extension and external rotation. Discomfort or apprehension represents a positive finding. This implies posterior impingement, either with normal physiologic motion resulting from abnormal osse-

Figure 3



Technique for calculating acetabular inclination and the lateral center-edge angle of Wiberg on AP pelvic radiographs. **A,** A line is drawn connecting the inferior aspect of the left- and right-sided acetabular teardrops (1). A second line is drawn parallel to the first through the inferior aspect of the acetabular sourcil (2). A third line is drawn connecting the inferior and lateral aspects of the acetabular sourcil (3). The angle created by the intersection of lines 2 and 3 (ie, Tönnis angle) should measure 0° to 10°. **B,** The lateral center-edge angle of Wiberg is created at the intersection of a line drawn through the center of the femoral head, perpendicular to the transverse axis of the pelvis (1), and a second line drawn through the center of the femoral head that passes through the most superolateral point of the sclerotic weight-bearing zone of the acetabulum (2). Values <25° may indicate inadequate coverage of the femoral head. (Adapted with permission from Clohisy JC, Carlisle JC, Beaulé PE, et al: A systematic approach to the plain radiographic evaluation of the young adult hip. *J Bone Joint Surg Am* 2008;90[suppl 4]:47-66.)

ous anatomy (eg, coxa profunda) or with abnormal physiologic motion resulting from soft-tissue deficiencies (eg, anterior capsular laxity).[23] In the dial test, the patient lies supine in neutral extension, and the examiner internally rotates the affected limb. The limb is then released and allowed to externally rotate. The test is positive when the patient's limb passively rotates >45° from vertical in the axial plane and lacks a mechanical end point. The contralateral limb is tested for comparison. The senior author (M.J.P.) has demonstrated a correlation between a positive dial test and capsular laxity.[23,24] Traction on the affected limb may demonstrate apprehension, which is suggestive of mechanical instability. Instability may be difficult to identify on physical examination and may be confused with other hip pathology; therefore, imaging should be performed in patients with suspected instability.

Imaging

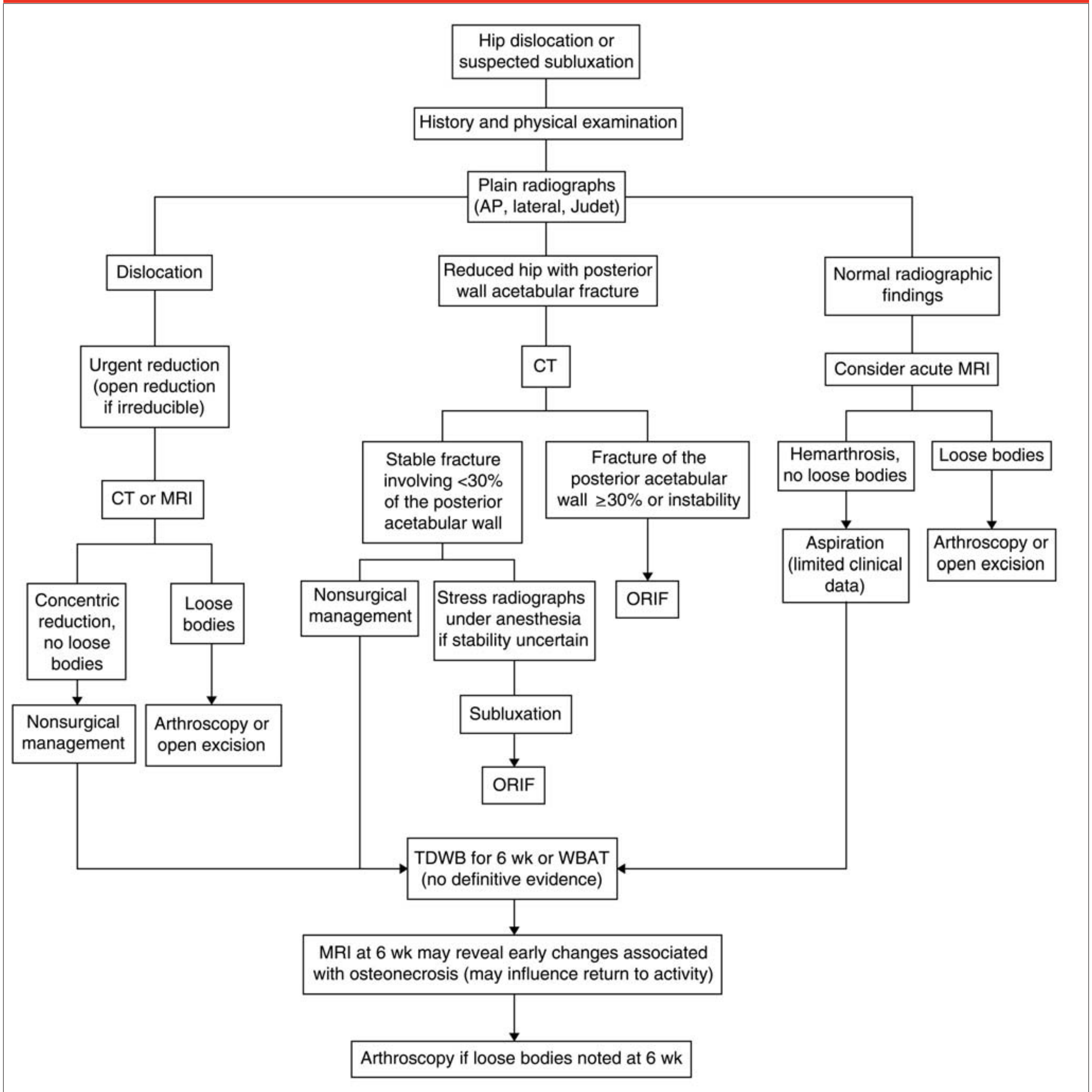
Radiographic evaluation begins with a standard AP pelvic radiograph and AP and lateral views of the affected side. Other views can be obtained to assess the acetabulum (eg, Judet), and the false-profile view can be obtained to evaluate anterior coverage of the femoral head. The lateral center-edge angle of Wiberg is commonly used to screen for acetabular dysplasia, which mitigates instability via compromise of the hip's bony foundation. The Tönnis angle measures lateral subluxation of the femoral head, which results in increased forces across the weight-bearing acetabulum (Figure 3). Abnormalities in femoral head-neck offset may imply impingement, which may lead to instability. In cases in which the diagnosis is unclear, traction views of the hip on plain radiographs or dynamic fluoroscopy may be used to identify hip subluxation.

CT is especially useful in the evaluation of traumatic instability. Small nondisplaced fractures of the acetabulum can be missed on plain radiography but are easily identified on CT. CT is routinely done following hip dislocation and subsequent reduction to assess for intra-articular loose bodies and adequacy of reduction. MRI and MRI arthrography allow detailed evaluation of the ligaments, labrum, capsule, and other soft-tissue structures. Although MRI arthrography is the most reliable means of identifying chondral lesions, labral pathology, and loose bodies, at some institutions, MRI has proved to be equally sensitive and specific for the identification of labral pathology.[25] Although traditional arthrography, ultrasonography, and bone scans have specific roles in the evaluation of acute and chronic hip pain, they typically are not useful in the evaluation of hip instability.

Etiology and Management

Hip instability can be categorized as traumatic or atraumatic. Traumatic instability is characterized by an acute event or series of repetitive events that lead to instability as a result of damage to the osseous or soft-tissue structures of the hip, or both. In contrast, atraumatic instability is

Figure 4



Treatment algorithm for hip subluxation and dislocation. ORIF = open reduction and internal fixation, TDWB = touch-down weight bearing, WBAT = weight bearing as tolerated. (Adapted with permission from Shindle MK, Ranawat AS, Kelly BT: Diagnosis and management of traumatic and atraumatic hip instability in the athletic patient. *Clin Sports Med* 2006;25[2]:309-326, ix-x.)

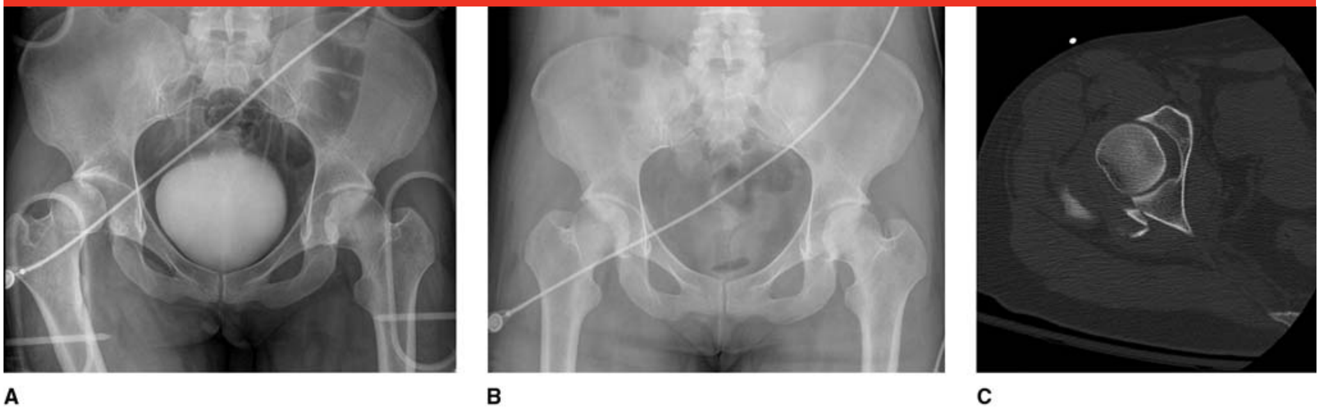
the result of underlying systemic disease, congenital bony or soft-tissue abnormalities, congenital laxity, or acquired abnormalities. A treatment algorithm for hip dislocation and subluxation is presented in Figure 4.

Traumatic Instability

Traumatic hip instability is best understood as a spectrum of injury that ranges from major trauma with osseous injury and hip dislocation, to minor trauma with pure dislocation or subluxation (eg, sports injuries), to microtrauma caused by repetitive motions of activities of daily living or exercise.

Dislocation of the native hip with or without fracture of the acetabu-

Figure 5



A, AP pelvic radiograph demonstrating right posterior hip dislocation and fracture of the posterior wall of the acetabulum. B, AP pelvic radiograph following reduction. C, Axial CT scan obtained following reduction demonstrating fracture of the posterior wall of the acetabulum.

lum or femoral head typically occurs when an axial load is applied to the femur with the hip joint in flexion and neutral adduction. In this scenario, the most common injury pattern is dislocation with associated posterior wall acetabular fracture, although pure dislocation without concomitant osseous injury may be seen.[26] The primary restraints in a pure dislocation are the soft-tissue stabilizers, that is, the ligamentum teres, capsule, extra-articular ligaments, labrum, and surrounding musculature. The resulting spectrum of injury to these structures has not been well-defined. Emergent reduction of the femoral head is indicated to reduce the risk of osteonecrosis.[14] Following hip reduction, passive limited ROM is evaluated to determine hip stability. A pelvic CT scan is obtained to evaluate for acetabular wall fracture, injury to the femoral head or neck, and intra-articular loose bodies. When doubt persists regarding stability, the hip should be examined and stress radiographs obtained with the patient under anesthesia.

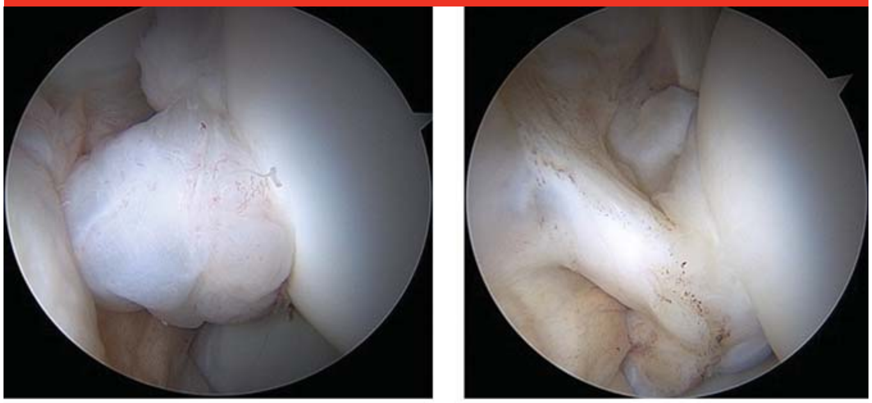
In certain posterior wall fractures and/or dislocation injuries, skeletal traction may be required to maintain reduction until definitive fixation can be achieved. Several authors advocate surgical fixation in patients with injuries involving >25% to 30% of the posterior wall and in those with hip subluxation on examination under anesthesia[14,27,28] (Figure 5). Mullis and Dahners[29] found loose bodies in 92% of patients (33 of 36) who underwent arthroscopy after closed reduction of a pure traumatic dislocation. In 78% of these patients, postreduction CT scans and radiographs did not elicit signs of chondral or osseous injury, which suggests that early arthroscopy after dislocation may be considered even with a negative result on imaging.

Weight bearing following dislocation remains controversial. Data in the trauma literature demonstrate that extended periods of protected weight bearing do not affect either the incidence of osteonecrosis[30,31] or outcomes.[32] MRI should be considered at 4 to 6 weeks postinjury because osteonecrosis is not detected on MRI until 4 to 6 weeks after posterior hip dislocation.[33]

Posterior subluxation of the hip is seen in the athlete who sustains a posteriorly directed force during sports participation, resulting in transient subluxation. This low-energy injury has been observed in a small series of American football players.[34] Although the hip may appear to be appropriately located on a plain radiograph, CT or MRI should be obtained in patients with a high suspicion of subluxation. Disruption of the iliofemoral ligament, acetabular lip fracture, and hemarthrosis are indicative of traumatic posterior hip subluxation. Intra-articular loose bodies may be noted, as well. In the patient with significant hemarthrosis visible on imaging, aspiration of the hip may be performed to provide pain relief and possibly protect the femoral head.[34,35] Intra-articular loose bodies must be removed, whether open or arthroscopically. Touch-down weight bearing for 6 weeks has been proposed for these patients;[34] however, there is a lack of clear evidence supporting a prolonged period of non–weight bearing versus early weight bearing as tolerated.[26]

Instability after traumatic dislocation attributed to capsular laxity with and without associated labral injury has been documented in the literature; however, the overall rate of instability following dislocation is thought to be low.[36-38] In a review of

Figure 6



A, Arthroscopic image demonstrating a loose ligamentum teres in a 17-year-old girl who experienced vague hip pain for 2 years. The patient was active in field hockey and lacrosse and had no history of a traumatic event. The ligamentum teres was notably lax and partially torn. **B,** Arthroscopic image following débridement with a shaver and radiofrequency probe to tighten the ligamentum teres. The patient also had mild femoroacetabular impingement and a partial labral tear.

264 dislocations, only 1 of 4 cases of instability involved a pure dislocation.[36] When discomfort persists following dislocation, capsular laxity should be considered. Physical examination should be performed, focusing on hip instability. In the patient with suspected capsular laxity, a trial of strengthening and physical therapy is warranted, focusing on the lumbar and abdominal core musculature, gluteus medius, and hip external rotators. Should instability persist, surgical intervention should be considered.

Acute traumatic labral tears and disruption of the ligamentum teres may occur with hip dislocation or subluxation resulting from high-, moderate-, and low-energy mechanisms. The ligamentum teres may become lax and redundant after injury, resulting in microinstability and pain (Figure 6). One case series involving complete and partial symptomatic tears to the ligamentum teres showed a spectrum of concomitant injury, including isolated ligamentum teres injury and associated labral tears and/or chondral injury.[39] The authors suggested the existence of a cycle of ligamentum injury associated with an increase in labral stress and potential further injury, but substantiating data were lacking.

Open capsular suture plication techniques have recently been described for instability.[37,40] Alternatively, arthroscopic techniques such as thermal capsulorrhaphy and capsular suture plication may be performed. These arthroscopic options may be associated with a lower risk of infection and heterotopic ossification than with open techniques. Arthroscopic thermal capsulorrhaphy uses heat created by radiofrequency to produce capsular shrinkage and stimulate the inflammatory cascade, leading to fibroplasia, angiogenesis, collagen resorption, and new collagen deposition.[41] In a series of 12 hips that underwent thermal capsulorrhaphy, all were stable at final follow-up and had improved Harris hip scores.[1] Risk of cartilage injury with thermal capsulorrhaphy has been documented in the arthroscopic shoulder literature;[42] however, we have found that with careful technique, injury to the cartilage of the femoral head and acetabulum can be avoided. Suture plication may be associated with a lower risk of thermal cartilage injury.

Sports such as golf, hockey, soccer, and ballet, as well as certain labor-intensive occupations, involve repetitive motion to or beyond the limits of normal physiologic ranges, which can lead to gradual breakdown of the labrum and subsequent microinstability. MRI or MRI arthrography may help to identify chronic capsulolabral changes.[43] Physical therapy is the first-line treatment option in the patient with chronic labral pathology. However, if 6 weeks have passed since the onset of symptoms and ROM and/or strengthening has offered no relief, then arthroscopic or open débridement may be considered. Débridement has been successful in alleviating pain and returning athletes to sport.[44]

Atraumatic Instability

In contrast to traumatic hip instability, which often involves an identifiable event or activity, atraumatic hip instability is associated with long-standing anatomic abnormalities and systemic conditions. Atraumatic instability in children and adults may be the result of bony dysplasia or ligamentous laxity; alternatively, the etiology may be iatrogenic or idiopathic. Patients with instability may present with apprehension, coxa saltans, pain, gait abnormality, or recurrent dislocation. True hip dislocation in adults is rare in the absence of trauma or an antecedent procedure, but in the pediatric population, true dislocation is associated with certain syndromes (eg, Marfan, Down).[1,45]

DDH is a common condition encompassing congenital and developmental abnormalities of the hip that cause an abnormal relationship between the femoral head and the acetabulum. DDH represents a continuum of abnormalities from dysplasia to subluxation to frank dislocation, and it is present in approximately 1% of the population.[46] With the advent of ultrasonography, most patients are treated early and achieve satisfactory results.[46] However, failure to recognize a subluxated or dislocated hip may eventually lead to dysplastic femoral and acetabular development as well as early arthritis. Typical changes in the dysplastic hip include a misshapen femoral head and increased anteversion of the neck with a shallow, anteverted acetabulum.[4] Insufficient acetabular coverage typically leads to anterior instability of the hip, with eventual pain and disability resulting from arthritis. The etiology of pain before the development of degenerative changes is not well understood, but it may be secondary to labral pathology, loose chondral tissue, chondral loss, ligamentum teres pathology, synovitis, or abnormal joint biomechanics. Tönnis and Heinecke[47] reported that patients with a low McKibbin instability index score (ie, sum of femoral and acetabular anteversion) had the lowest rate of pain and degeneration, whereas patients with a high McKibbin index score had a tendency toward a higher rate of pain, arthritis, and altered hip mechanics.

In patients without the classic pattern of hip dysplasia, abnormal morphology of the acetabulum and the femoral head and neck may cause FAI with secondary bony changes that can render the hip unstable.[4] For example, some patients with Legg-Calvé-Perthes disease develop significant impingement that leads to secondary acetabular dysplasia and subsequent instability. Cam impingement occurs when an abnormally shaped femoral head and neck collides with the acetabulum during normal motion, resulting in labral tearing and cartilaginous damage.[48] Pincer impingement occurs in the presence of overcoverage of the acetabulum in conditions such as coxa profunda and acetabular retroversion.[49] In a recent study, 9 of 14 patients with hip dislocation were found to have underlying FAI.[19]

Soft-tissue disorders that result in generalized ligamentous laxity may also be responsible for instability in the hip and other joints. Laxity of the iliofemoral, pubofemoral, and ischiofemoral ligaments predisposes patients to hip subluxation and dislocation.[1] Connective tissue disorders, including Down syndrome, Ehlers-Danlos syndrome, Marfan syndrome, and arthrochalasis multiplex congenita, may present as instability or frank dislocation.[1] Nearly 1 in 20 children with Down syndrome develops spontaneous dislocation of the hip before age 10 years.[45] Patients aged >2 years with any of these conditions may develop voluntary dislocation, that is, by definition, associated with ligamentous laxity.[50] Voluntary dislocation is posterior in most cases. True idiopathic instability may occur in the absence of trauma, bony dysplasia, overuse, or connective tissue disorder.[50]

Habitual dislocation of the hip has been described in children without laxity; this condition is not well understood.[51] It has been postulated that psychiatric conditions may contribute to habitual dislocation without documented anatomic aberrations. True idiopathic dislocation is rare in adults.[52,53] Bellabarba et al[54] reported on five patients with a snapping hip and gait disturbance as well as increased pain on flexion, adduction, and internal rotation. All patients were diagnosed with idiopathic dynamic atraumatic instability of the hip, possibly secondary to underlying laxity. None was diagnosed with a specific syndrome.

Iatrogenic instability of the hip is uncommon outside the setting of total hip arthroplasty, but it may be present after open procedures in which trochanteric osteotomy or capsulotomy is performed (eg, surgical dislocation).[55] Recently, there have been two case reports of anterior dislocation following arthroscopic hip procedures.[56,57] The incidence of such cases of instability may increase as these occurrences are better recognized and documented.

In general, atraumatic instability is initially managed nonsurgically with rest and activity modification followed by physical therapy. Should these measures prove to be unsuccessful, intra-articular anesthetic injection may be considered.[14] Nonsurgical management may be unsuccessful in patients with substantial instability or continued dislocation.[54] Surgical intervention should be considered when nonsurgical measures fail. Arthroscopic surgery may be undertaken for patients with idiopathic generalized ligamentous laxity or connective tissue disorders leading to redundancy of the capsule and instability. Arthroscopic surgery also may be advisable in patients with instability who respond to an anesthetic injection.[1] In the patient with concomitant labral pathology, arthroscopic débridement or repair of the acetabular rim may be appropriate with concomitant capsulorrhaphy or suture plication. Labral repair with reduction in capsular laxity has been reported.[38] Good results have been shown with labral débridement and thermal capsulorrhaphy in addition to capsular suture plication.[1,40]

In patients with atraumatic instability secondary to bony dysplasia, the role of arthroscopy is not as clearly defined. It has been proposed that altering the soft tissues alone without addressing the bony pathology may not lend stability; however, good results with arthroscopy have been achieved in patients with atraumatic instability and hip dysplasia.[58,59] In patients with advanced dysplasia, such as those with a center-edge angle of Wiberg <15°, an open procedure is required to address bony incongruity.[60] This can be accomplished with acetabular reorientation or proximal femoral osteotomy, or both.

Future Directions

Although traumatic hip instability has been relatively well defined, atraumatic hip instability remains a more difficult diagnosis. Future efforts should be directed at improving clinical diagnosis, standardizing management, and establishing reliable and valid hip-specific outcomes criteria. Dynamic imaging modalities may elucidate further subtleties in diagnosis and pathology. Caution should be exercised in extrapolating from experiences with shoulder instability because the anatomy of the hip joint is more constrained than that of the shoulder. The dynamic nature of hip instability requires a better understanding of the interaction of the soft-tissue and bony anatomy in both healthy and symptomatic patients. A better appreciation of the contribution of subtle collagen abnormalities and hormonal influences to the musculoskeletal system will help to identify hips at risk of symptomatic instability. The current body of evidence concerning hip instability continues to evolve, and further research is required.

Summary

The hip joint is one of the most stable articulations in the body. Until recently, hip instability was considered a rare clinical entity outside the setting of major trauma. Advances in the understanding of disease processes of the hip, including FAI, developmental disorders, and posttraumatic disorders, have brought more attention to the topic. Hip instability may be traumatic (eg, acetabular posterior wall fracture) or atraumatic (eg, DDH, connective tissue disorders). Pathology exists as a spectrum from complete dislocation to subluxation to microinstability. Diagnosis may be difficult. It is based on specific tests performed during the physical examination as well as on plain radiographs, CT, MRI, and MRI arthrography.

Nonsurgical management of hip instability includes protected weight bearing, physical therapy, and intra-articular injection. In refractory cases and those associated with a large acetabular fracture, surgical intervention should be performed. The underlying pathology dictates surgical repair, whether with open reduction and internal fixation of a fracture, open or arthroscopic labral repair, osteoplasty, capsulorrhaphy, or osteotomy. Dynamic imaging modalities and the development of hip-specific validated outcomes measures may greatly increase our understanding of hip instability.

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