



Hypermobile Hip Syndrome

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Hypermobile hip syndrome is defined as the triad of symptoms (pain and instability), physical examination findings (confirms the subjective history), and imaging findings (corroborates the subjective and objective examination) consistent with hip instability, manifest along a spectrum from microinstability to dislocation. Soft tissue (capsule and labrum) and osseous (coverage, rotational profiles) structures may contribute. Imaging may reveal unique findings, include an anterior or anterolateral hypermobile hip crevasse. Surgical management must ensure anatomical corrections of pathomorphology, labral preservation, and routine complete capsular closure, with variable degrees of plication and shift able to be achieved with modern instrumentation. Postoperative care with physical therapy and medical management optimizes outcomes. Oper Tech Sports Med 27:108-118 © 2019 Elsevier Inc. All rights reserved.

KEYWORDS femoroacetabular impingement, dysplasia, labral tear, microinstability, hypermobile hip, hip capsule

Introduction

Arthroscopic hip preservation surgery is one of the most rapidly growing and evolving fields within Orthopaedic Surgery. The learning curve of hip arthroscopy is significant and involves both surgical decision-making and surgical technique.¹ Arthroscopic surgeons must recognize (decision-making) and treat (technique) all soft tissue and osseous sources of pathology in order to optimize outcomes and avoid complications.² A significant complication following hip arthroscopy is hip instability, which exists across a wide spectrum from microinstability to macroinstability.³ Sources of postoperative instability may be native, iatrogenic, or both. The author believes that postoperative instability is

largely preventable in the carefully selected patient with skillful execution of the procedure.

Variable degrees of capsular incision are required for central and peripheral compartment access, visualization, and instrumentation. Although controversial, the type (interportal vs “T”), size, and location of capsulotomy determines the ability to correct the underlying reason for surgery (“if you can’t see it, you can’t treat it”). Capsular management requires an exceptionally detailed understanding of the pathomechanics of capsular closure vs intentional (or unintentional) capsular release (or tear). The titration of the optimal degree of capsular tightness (biomechanics) has not been elucidated in the lab or in the operating room. The interposition of capsular tissue between overlying musculotendinous and underlying intra-articular structures is an under-recognized component of the effect of capsular closure. Capsular management in patients with excessive motion demands the right amount of plication to avoid postoperative instability. In primary or revision surgery for patients with a hypermobile hip, capsular management includes side-to-side complete closure, plication, inferior capsular shift, augmentation, and interposition reconstruction.

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Hypermobile Hip Syndrome Definition

The native normal hip has a tendency toward stability due to the congruency of the femoral head in the depth of the

acetabulum, the suction seal of the acetabular labrum, the translational and rotational stability afforded by the capsule, and the contractile crossing musculotendinous units. The majority of motion at the hip joint should be rotational, with a relatively fixed center of rotation in the femoral head, rather than translational.⁴⁻⁷ When the center of rotation excessively translates, instability may occur. The spectrum of hip instability ranges from subtle microinstability to dislocation. Microinstability can be a cause or effect of several other pathologic conditions in the hip. These include osseous, chondrolabral, capsuloligamentous, musculotendinous, or kinetic chain neuromechanical causes.⁸

Laxity is defined as excessive motion in a specific joint in an asymptomatic individual. “Excessive,” relative to a joint, is defined as abnormally increased or suprphysiological motion, also known as hypermobility. Instability is defined as excessive motion in a specific joint in a symptomatic individual. The key distinction between laxity and instability is the absence (former) or presence (latter) of symptoms. Thus, “microinstability,” by definition, mandates the presence of symptoms.^{9,10} In the hip, the relevant planes of motion include sagittal (flexion, extension), coronal (abduction, adduction), and axial (internal, external rotation; distraction, compression). Hip hypermobility involves excessive motion in one or more of these planes. Hypermobile hip syndrome may be defined as a triad of symptoms (patient’s unwanted or undesired subjective complaints), signs (physical examination abnormalities with excessive motion that provoke the inciting symptoms), and imaging findings (plain radiographs, magnetic resonance imaging [MRI], computed tomography [CT], or ultrasound) (Table 1) consistent with instability. A patient with hypermobile hip syndrome may exhibit a constellation of symptom severity, from microinstability to frank dislocation.

Anatomy

The hip capsule is a quasicircumferential condensation of ligaments that functions as a checkrein to translational and rotational

motions.¹¹ The iliofemoral ligament (anterior), also known as the Y-ligament of Bigelow, composed of medial (inferior) and lateral (superior) limbs, is the most relevant part of the capsule to the arthroscopic hip preservation surgeon (Fig. 1).^{11,12} The ligament fibers are cut perpendicularly via an interportal capsulotomy and in parallel via T capsulotomy (Fig. 2) in order to optimally arthroscopically visualize the central and peripheral compartments of the hip joint.^{12,13} The iliofemoral ligament is a primary restraint to external rotation (lateral limb more than medial) and extension (medial limb more than lateral).^{11,14} The pubofemoral ligament, at the anteroinferior position of the joint, controls hip external rotation at less than 30° of hip flexion¹⁵ and controls hip internal rotation at greater than 30° of hip flexion with abduction of 20° and 40°.¹⁶ The ischiofemoral ligament, at the posterior-posteroinferior position of the joint, controls hip internal rotation at a minimum of 10° or 20° of abduction.¹¹ The zona orbicularis is the circumferential condensation of ligaments just distal to the head-neck junction that loosens as the hip is flexed and tightens as the hip is extended.^{17,18} The zona orbicularis is a key structure in the femoral head’s resistance to axial distraction.^{17,19,20} Once the labral suction seal is broken (at approximately 3 mm of femoral head distraction in neutral flexion, abduction, rotation), the capsule becomes the primary restraint to further distraction.²¹ In addition to the inert noncontractile capsule, certain musculotendinous units crossing the hip contribute to stability. The iliocapsularis is an important anterior stabilizer of the femoral head in both normal and dysplastic hips.^{22,23} Similarly, the conjoint tendon of the iliopsoas is an anterior stabilizer of the femoral head,²⁴⁻³⁰ affected by femoral version, lesser trochanteric version, and acetabular version.³¹⁻³³

Clinical Examination

Symptoms

The diagnosis of hypermobile hip syndrome is made with a thorough history and physical examination. Patients typically have a chief complaint of hip and/or groin pain and

Table 1 Hypermobile Hip Syndrome Triad of Symptoms, Signs, and Imaging Findings

Symptoms	Signs	Imaging Findings
<ul style="list-style-type: none"> • Pain • Instability • Apprehension • Fear • Weakness • Loose • Unstable • Snapping • Kinesiophobia 	<ul style="list-style-type: none"> • Positive axial distraction test • Positive external rotation recoil • Positive dial test • Positive hip pivot shift test • Positive Ludloff test • Positive iliopsoas test • Positive FADIR 	<ul style="list-style-type: none"> • Plain radiographs <ul style="list-style-type: none"> • Dysplasia • Coxa valga • Subluxation • Dislocation • MRI <ul style="list-style-type: none"> • Capsular tear • Capacious capsule (excessive volume) • Anterior labral tear (3 to 4 o'clock) • Thin capsule (iliofemoral ligament) • Hypertrophic iliocapsularis • CT <ul style="list-style-type: none"> • Excessive femoral anteversion • Excessive cranial and central acetabular anteversion • Decreased global coverage (surface area, volume)

CT, computed tomography; FADIR, flexion, adduction, internal rotation; MRI magnetic resonance imaging.

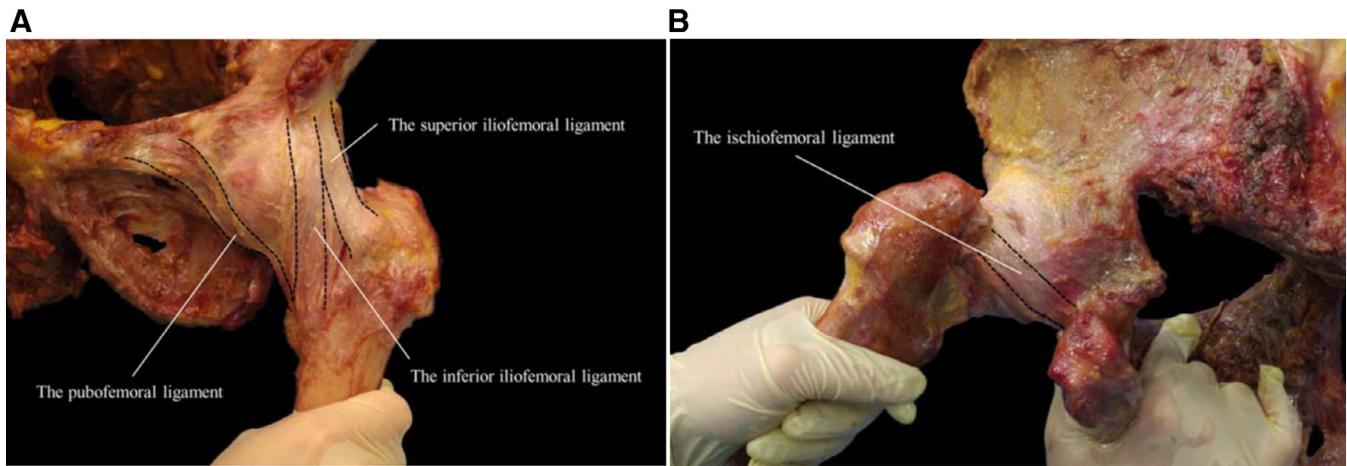


Figure 1 (A) Anterior view of left hip in cadaveric specimen, demonstrating the medial (inferior) and lateral (superior) limbs of the iliofemoral ligament and the pubofemoral ligament. Reproduced with permission from John Wiley and Sons, *Clinical Anatomy* 2014, Hidaka et al.¹¹ (B) Posterior view of left hip in cadaveric specimen, demonstrating the ischiofemoral ligament. Reproduced with permission from John Wiley and Sons, *Clinical Anatomy* 2014, Hidaka et al.¹¹

instability. Pain location is frequently demonstrated with a “C” sign or “between the fingers” sign (Fig. 3). Pain severity can range from mild to severe. Pain chronicity is usually insidious in atraumatic onset, waxing and waning based on activity levels with chronic pain being common. Patients tend to be frequently former (or current) flexibility athletes (eg, gymnasts, dancers, yogis, figure skaters, mixed martial artists, cheerleaders).³⁴ Complaints of instability manifest as “giving way,” “giving out,” “buckling,” instability,” “loose,” “weakness,” “popping,” “snapping,” “coming out of socket,” “scared of hip,” “fear,” “apprehension,” among others. A

history of instability of other joints should be sought, with shoulder (multidirectional instability, atraumatic; unidirectional, traumatic), patellar, finger, elbow, ankle (recurrent sprains). A history of connective tissue disorders should be explicitly ascertained (eg, Ehlers-Danlos, Marfan, benign joint hypermobility syndrome, among others), which can be inquired via multiple different evaluation scores (Table 2).³⁵⁻³⁷ Per the 2017 International Criteria for Ehlers-Danlos syndrome, terminology referring to joint hypermobility syndrome is now called Hypermobility Spectrum Disorder; similarly, previous terminology referring to Ehlers-Danlos-hypermobility

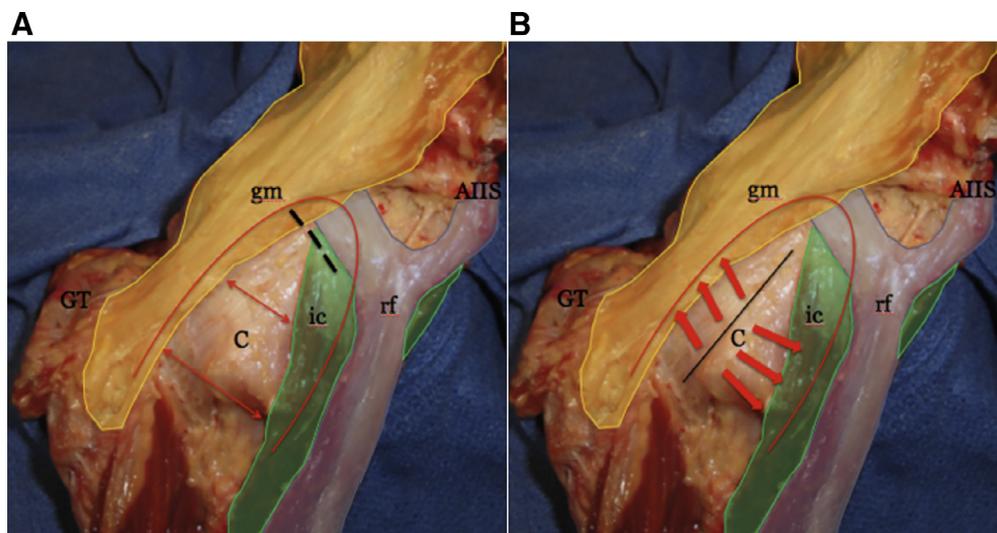


Figure 2 (A) Anterior view of right hip in cadaveric specimen, demonstrating the location of a standard interportal capsulotomy (dashed line), which makes a perpendicular incision in the iliofemoral ligament. AIIIS, anterior inferior iliac spine; C, capsule; gm, gluteus minimus; GT, greater trochanter; ic, iliocapsularis; rf, rectus femoris. Reproduced with permission from Elsevier, *Arthroscopy: The Journal of Arthroscopic and Related Surgery* 2014, Walters et al.¹² (B) Anterior view of right hip in cadaveric specimen, demonstrating the location of a standard T capsulotomy (straight line), which makes an incision in the ligament, parallel to the line of its fibers. AIIIS, anterior inferior iliac spine; C, capsule; gm, gluteus minimus; GT, greater trochanter; ic, iliocapsularis; rf, rectus femoris. Reproduced with permission from Elsevier, *Arthroscopy: The Journal of Arthroscopic and Related Surgery* 2014, Walters et al.¹²



Figure 3 (A) Patient exhibiting a “C” sign, using the hand to make the letter “C” wrapping from the front of the hip, around the side, to the back of the hip, typically felt deep inside the joint, not palpably tender to touch. (B) Patient exhibiting a “between the fingers” sign, using both hands to point between the front of the hip and the back of the hip, deep inside the joint, not palpably tender to touch.

type (EDS-HT/EDS III) is now called Hypermobile Ehlers-Danlos Syndrome.^{37,38}

In patients with a history of previous hip surgery, operative reports and operative photos should be critically scrutinized for surgical details relevant to potential postoperative instability. Regarding previous arthroscopy, there are several aspects of the technique that should be sought to determine if a potential contributing symptom source. Both hip and nonhip related

etiologies for native (hypermobile hip syndrome) and postoperative hips can be divided based on osseous and soft-tissue reasons via the layer concept.⁸ Osseous reasons (layer I) include dysplasia (native or iatrogenic),^{3,39} excessive femoral anteversion,^{39,40} excessive cam osteoplasty,⁴¹ coxa valga,⁹ ischiofemoral impingement,⁴² and trochanteric-pelvic impingement.⁴² Inert soft tissue reasons include capsular insufficiency (thin native capsule, increased native capsular volume, unrepaired capsulotomy, torn capsular repair, loose capsular repair),⁴³ labral deficiency (labral debridement, labral tear, small native labral size),⁴⁴ and ligamentum teres (tear, debridement).⁴⁵ Contractile soft tissue reasons include previous iliopsoas surgery (joint level fractional lengthening tenotomy, tendon release off lesser trochanter, ischiofemoral decompression lesser trochanterplasty).

Signs

Physical examination should be comprehensive and systematic. Inspection, palpation, range of motion, strength, and special testing should be assessed for characteristics of hypermobile hip syndrome. A Beighton score should always be obtained in patients suspected of having hypermobile hip syndrome. Inspection should especially critique gait (shortened stance phase [minimizes hip extension, which exacerbates anterior hip instability], Trendelenburg gait [weak abductors], intoeing [to avoid extended external rotation, which exacerbates anterior hip instability]). Muscle atrophy and cutaneous abnormalities (ecchymosis, erythema) are unusual in hypermobile hip syndrome. Palpation is usually relatively normal with intra-articular complaints, including hypermobile hip syndrome, due to their deep location. Range of motion, by the definition of hypermobility, is more than normal. This

Table 2 Scoring systems used to evaluate patients with hypermobile hip syndrome. For Beighton, a score of 4 or more (out of 9 total) suggests hypermobility. For Brighton, hypermobility is suggested in the presence of 2 major criteria, or 1 major and 2 minor, or 4 minor. For Hakim and Grahame, answer “yes” to 2 or more questions suggests hypermobility.

Beighton	Brighton	Hakim and Grahame
<ul style="list-style-type: none"> • Small finger MCP extension >90° <ul style="list-style-type: none"> ◦ Left ◦ Right • Thumb touch to volar forearm <ul style="list-style-type: none"> ◦ Left ◦ Right • Elbow hyperextension >10° <ul style="list-style-type: none"> ◦ Left ◦ Right • Knee hyperextensions >10° <ul style="list-style-type: none"> ◦ Left ◦ Right • Palms placed flat on floor from standing position, knees extended 	<ul style="list-style-type: none"> • Major criteria <ul style="list-style-type: none"> ◦ Beighton score of 4 or more ◦ Arthralgia >3 months in 4+ joints • Minor criteria <ul style="list-style-type: none"> ◦ Beighton score of 1, 2, or 3 ◦ Arthralgia >3 months in 1-3 joints or back pain, spondylolisthesis, spondylolysis, or spondylosis ◦ Dislocation/subluxation in more than one joint, or in one joint more than one time ◦ Soft tissue rheumatism, >3 lesions (eg, epicondylitis, tenosynovitis, bursitis) ◦ Marfanoid habitus (tall, slim, arachnodactyly, armspan: height ratio > 1.03) ◦ Abnormal skin (striae, thin skin, hyperextensibility, papyraceous scar) ◦ Eye signs (drooping lids, myopia) ◦ Varicose veins, hernia, rectal or uterine prolapse 	<ul style="list-style-type: none"> • Can you now (or could you ever) place hands flat on floor without bending knees? • Can you now (or could you ever) bend your thumb to touch your forearm? • As a child, did you amuse your friends by contorting your body into strange shapes or could you do the splits? • As a child or teenager, did your kneecap or shoulder dislocate on more than one occasion? • Do you consider yourself “double-jointed”?

MCP, metacarpophalangeal.

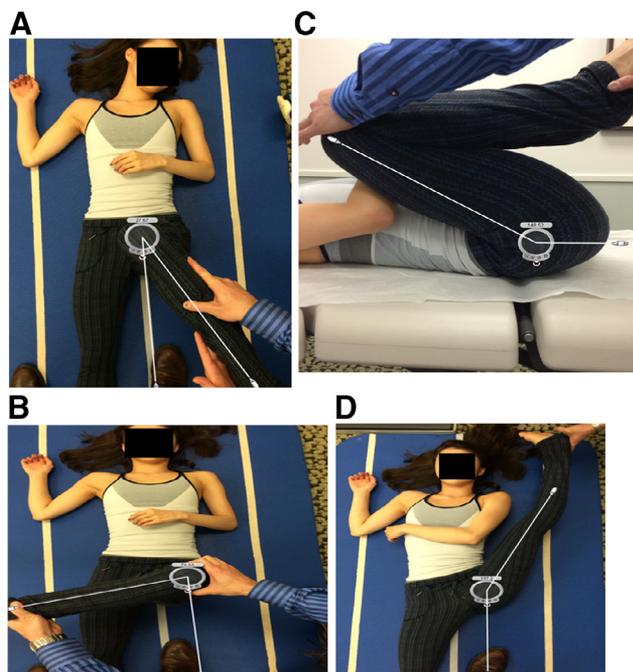


Figure 4 (A) Left hip supine internal rotation, measured via digital photography. (B) Left hip supine external rotation, measured via digital photography. (C) Right hip supine flexion, with contralateral hip extension, measured via digital photography. (D) Left hip supine abduction, measured via digital photography.

can be one or multiple planes (Fig. 4). Strength testing should evaluate core, pelvic, hip, and lower extremity to determine if weakness could be contributing cause of instability. Abductor fatigue is a common cause of deep anterolateral (AL) hip pain in dysplasia and hypermobile

hip syndrome. Special instability testing consists of an extension/external rotation apprehension test (patient either slid down to end of examination table, with gravity-induced hip extension and manual external rotation or patient moved to lateral edge of examination table, with gravity-induced hip extension and manual external rotation), hip distraction (manual axial distraction pull applied), external rotation recoil (assessment for symmetry of supine recoil with manual external rotation force applied, observing the recoil of internal rotation), hip dial test (assessment of symmetry of supine permissive external rotation after forced internal rotation), and hip pivot shift (standing test with planting the ipsilateral foot and then turning the body contralaterally away from the ipsilateral foot, forcing extension and external rotation). The key component of special instability testing is the reproduction of the patient's chief complaint of instability symptoms within the hypermobile hip syndrome. An extension of the office physical examination is the physical and fluoroscopic examination under anesthesia with muscle relaxation in the operating room.

Imaging

In patients with hypermobile hip syndrome, it is necessary that clinicians treat patients, and not just x-rays, due to the high prevalence of abnormal imaging in asymptomatic individuals.⁴⁶ This ensures treating subjective symptoms, objective physical examination findings, and corroborating these with imaging (plain radiographs, MRI, CT, and ultrasonography) to formulate a treatment plan. Plain radiographs are initially obtained with a checklist utilized to systematically evaluate all relevant osseous structures (Table 3). Special

Table 3 Imaging checklist for patients with hypermobile hip syndrome.

Femoral Side	Acetabular Side	Joint Checklist	Lumbopelvic Checklist
<ul style="list-style-type: none"> • Neck-shaft angle • Alpha angle • Head-neck offset • Head-neck offset ratio • Version • Mechanical axis • Triangular index • Head-neck sclerosis • Impingement cyst • Hypermobile hip crevasse • Omega angle* • Omega surface† • McKibbin index‡ • FEAR index§ • Splits view (IR, ER) • Glute max squeeze view 	<ul style="list-style-type: none"> • LCEA • ACEA • Tonnis angle • Femoral head extrusion index • Coxa profunda • Protrusio acetabulae • Crossover sign • AIIS • Posterior wall sign • Ischial spine sign • Shenton's line • Hip-center position • Version (1, 2, 3 o'clock) 	<ul style="list-style-type: none"> • Joint space width <ul style="list-style-type: none"> ◦ Medial sourcil ◦ Middle sourcil ◦ Lateral sourcil • Tonnis grade • Kellgren-Lawrence grade • Effusion • Iliocapsularis size • Labral size • Capsular thickness 	<ul style="list-style-type: none"> • Pelvic incidence <ul style="list-style-type: none"> ◦ Pelvic tilt ◦ Sacral slope • T1-pelvic angle <ul style="list-style-type: none"> ◦ Pelvic tilt ◦ T1-spinopelvic inclination

ACEA, anterior center edge angle; AIIS, anterior inferior iliac spine; AP, anteroposterior; ER, external rotation; IR, internal rotation; LCEA, lateral center edge angle.

*Omega angle indicates the radial extension of cam morphology (degrees);

†Omega surface is the sum of the alpha angle, neck-shaft angle, femoral version, acetabular version, and lateral center edge angle;

‡McKibbin index is the sum of femoral and acetabular version;

§FEAR (Femoro-Epiphyseal Acetabular Roof) index is a measure of hip stability in the setting of borderline dysplasia.

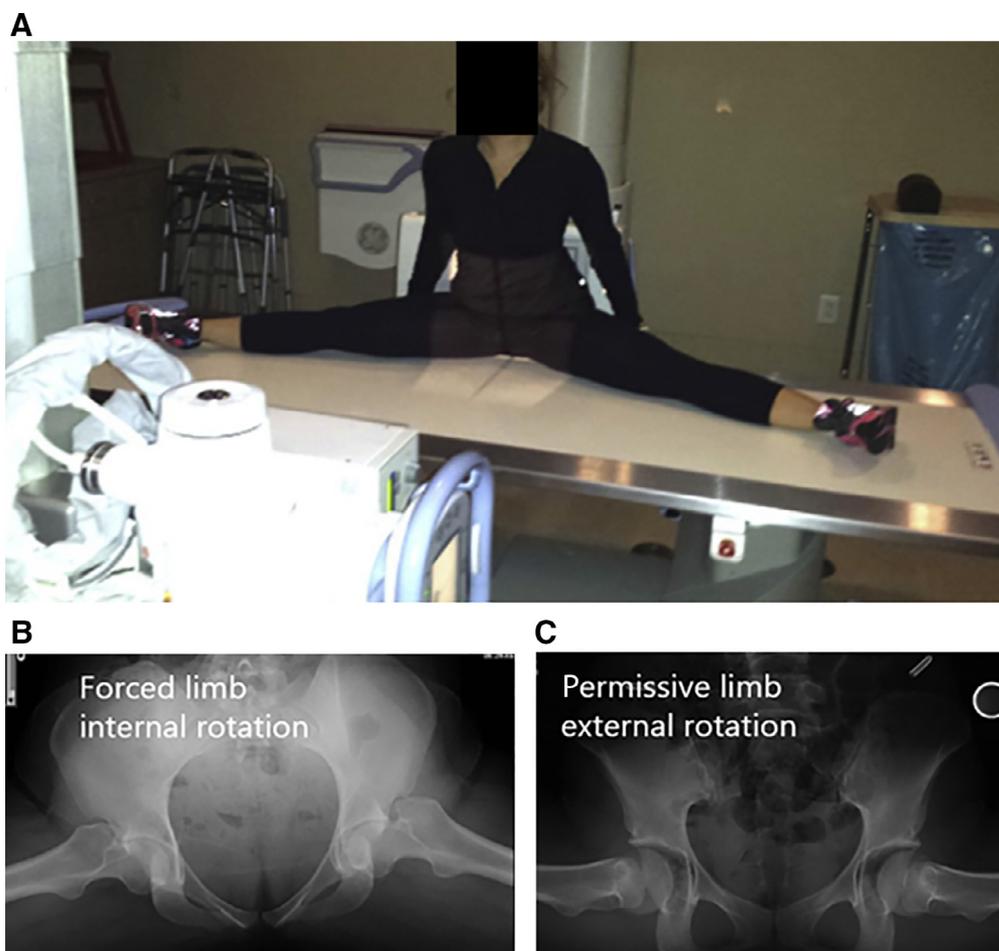


Figure 5 (A) Clinical photograph illustrating “splits” position with forced limb internal rotation in a 22-year-old professional dancer. There are 2 types of “splits” radiograph – (B) illustrates forced limb internal rotation, where the tip of the greater trochanter contacts above the superolateral rim (trochanteric-pelvic impingement). In patients with hypermobile hip syndrome, the pubofemoral and ischiofemoral ligaments are stretched in this position; (C) illustrates permissive limb external rotation, where the tip of the greater trochanteric contacts posterior to the posterior rim, levering the femoral head anteriorly, creating a loss of the suction seal and a “vacuum sign.”

radiographic views may be utilized in hypermobile hip syndrome – a “splits” radiograph (Fig. 5A-C) may illustrate femoral head translation (1.9 and 0.8 mm in males and females, respectively) and a vacuum sign (24% and 46% in males and females, respectively) in the grand ecart faciale “splits” position.^{9,42} A “glute max squeeze” radiograph may illustrate the ability to correct focal retroversion via posterior pelvic tilt with gluteus maximus voluntary activation (Fig. 6A,B). Additionally, any subspine component of hip pain would also be reduced with a “glute max squeeze” view secondary to posterior pelvic tilt.^{47,48} A hypermobile hip crevasse can also be observed with plain radiography (in addition to MRI, CT, and arthroscopy) (Fig. 7).⁴⁹ The pathomechanics of the crevasse is debatable, controversial, and, to date, not completely understood. A previous description has reported a posterior proximal head-neck junction postulated to result from a posterior translational motion of the femoral head leading to partial subluxation and posterior impaction between the head and the posterior acetabular rim.⁴⁹ The head essentially would perch on the posterior rim prior to reduction, similar to a Hill-Sachs with an anterior shoulder dislocation. The

presence of cam and/or pincer morphology would create a levering effect on the rim and posterior translation, predisposing the femoral head to dislocation.⁵⁰

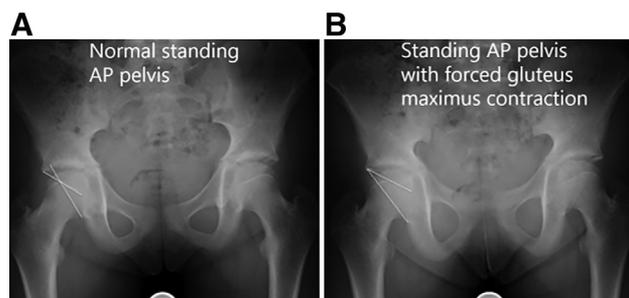


Figure 6 (A) Standing anteroposterior (AP) pelvis radiograph of a skeletally immature 13-year-old elite female dancer illustrating a crossover sign (dotted white lines) with the coccyx sagittally lined over the pubic symphysis, approximately 2 cm above it. (B) illustrates a “glute max squeeze” radiograph with the pubic symphysis overlying the coccyx and elimination of the crossover sign. Other measures of retroversion, posterior wall and ischial spine signs, are unchanged.

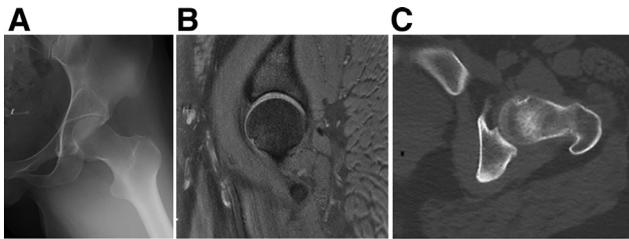


Figure 7 (A) Dunn 45° plain radiograph of left hip in 22-year-old female yogi, Beighton 9/9, illustrating a proximal anterolateral hypermobile hip crevasse; (B) sagittal T2-weighted MRI in the same patient; (C) axial CT in the same patient.

The hypermobile hip crevasse is a proximal anterior-AL linear femoral head lesion (Fig. 8). With hypermobile hip syndrome, femoral head instability in the acetabulum leads to excessive anterior translation with increasing hip flexion. Hypermobile hip syndrome, simulated in a laboratory model via sequentially increasing “pie-crusting” on the iliofemoral, pubofemoral, and ischiofemoral ligaments, leads to both excessive anterior femoral head translation and rotation (internal and external).^{4,5} As opposed to Philippon’s described crevasse,⁴⁹ the hypermobile hip crevasse is due to an anterior, rather than posterior, translation, with an anterior, rather than posterior, vertical chondro-osseous lesion. The anterior translation as the hip is flexed, leads to a more proximal location of “impingement.” Similarly, a more proximal impingement location has been observed in borderline dysplasia with

cam morphology.⁵¹ The anterior translation, even in the absence of dysplasia and/or cam or pincer morphology, causes a characteristic straight anterior chondrolabral injury.⁵² It is this anterior translation that prompts a compensatory iliopsoas activation, as the iliopsoas is a strong stabilizer to resist further anterior translation, frequently described as hip flexor symptoms in patients with hypermobile hip syndrome.^{10,53} The latter suggests that “iliopsoas impingement” is not truly “impingement,” but rather the iliopsoas attempting to stabilize the head in the acetabulum, similar to the long head biceps brachii stabilizing the humeral head in the glenohumeral joint.^{24,27-29} It must be recognized that hypermobile hip syndrome primarily describes an innate native hip condition, not an iatrogenic postoperative condition. However, in patients with pre-existing hypermobile hip syndrome, conditions that would exacerbate anterior instability (excessive femoral anteversion, native or iatrogenic dysplasia, coxa valga, iliopsoas release, capsular disruption, labral deficiency, ligamentum teres deficiency) may worsen hypermobile hip syndrome symptoms. Female ballet dancers may be the prototypical example of hypermobile hip syndrome, with a hypermobile hip crevasse previously described as a superior (proximal) femoral lesion observed with significant femoral head subluxations (translation) in the absence of significant osseous pathomorphology.⁵⁴⁻⁵⁶

Advanced imaging with MRI and CT is beneficial in the management of patients with hypermobile hip syndrome. MRI with gadolinium dye arthrography is effective in

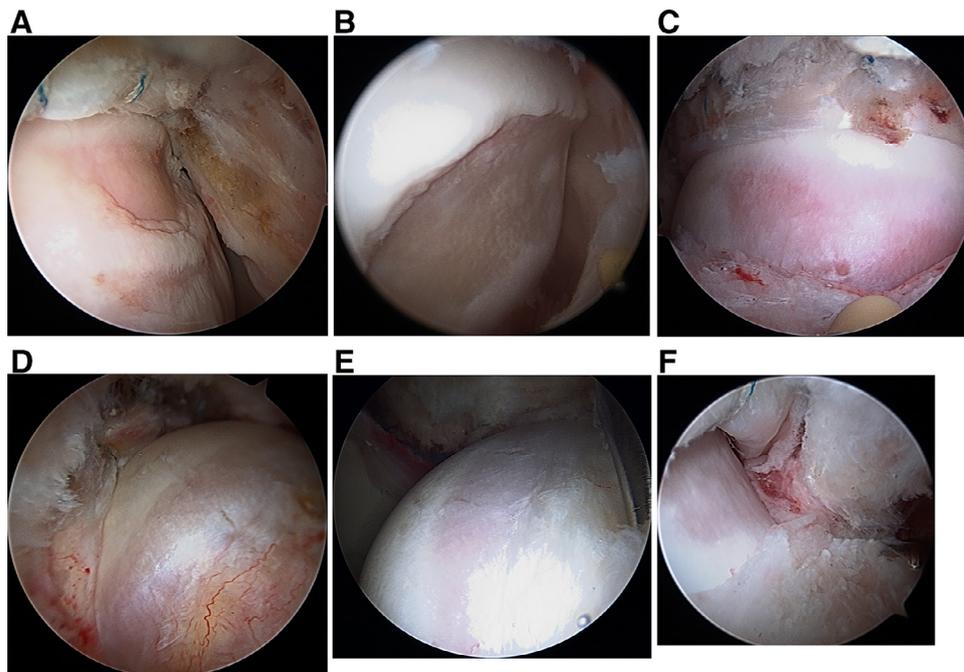


Figure 8 Multiple arthroscopic views of hypermobile hip crevasse. (A) viewing from modified midanterior portal in left hip arthroscopy in 25-year-old female Olympian powerlifter; (B) viewing from modified midanterior portal in right hip arthroscopy in 20-year-old male Division 1 collegiate football player; (C) viewing from modified midanterior portal in right hip arthroscopy in 18-year-old female cheerleader; (D) viewing from modified midanterior portal in right hip arthroscopy in 38-year-old female elite marathoner; (E) viewing from anterolateral portal in right hip arthroscopy in 16-year-old female volleyball player; (F) viewing from modified midanterior portal in left hip arthroscopy in 15-year-old elite ballet dancer.

evaluating the integrity of the labrum and capsule and can indirectly measure articular volume and detect the presence of a patulous capsule. In patients with hip instability without dysplasia or FAI (Femoroacetabular Impingement) Syndrome, a statistically greater incidence of straight-anterior and lateral chondrolabral injury has been observed.⁵² CT affords a complete osseous evaluation of the pelvis and the lower extremity rotational profile, including acetabular and femoral version. Additionally, it can show an osseous hypermobile hip crevasse clearly.

Surgical Decision-Making

Surgical indications in patients with hypermobile hip syndrome include a failure of initial nonsurgical treatment for 3 months minimum, with symptoms for 6 months minimum. However, there is limited evidence regarding the efficacy of nonsurgical treatment, in addition to evidence demonstrating short-term superiority of surgical treatment.⁵⁷⁻⁶¹ Mid- and long-term comparative prospective outcomes are currently unknown, including most subject patient-reported and objective clinician-measured (including radiographs, and osteoarthritis initiation and/or progression). Nonsurgical measures are similar to the triad for FAI Syndrome and include education, limited rest, activity modification (avoidance of provocative maneuvers [as hypermobile hip syndrome and FAI syndrome are both motion- and position-dependent entities, this includes avoidance of excessive deep flexion and rotational motions]), physical therapy, oral nonopioid, nonsteroidal anti-inflammatory medications, and diagnostic and therapeutic injections.^{62,63} In the situation of failure of nonsurgical measures and dissatisfaction with the current hip condition, arthroscopic hip surgery may be indicated. This consists of diagnostic hip arthroscopy, labral preservation (repair over debridement; reconstruction or augmentation in revision setting with deficient labrum), correction of morphology (cam, pincer, subspine), articular cartilage surgery (chondroplasty, microfracture, subchondral drilling, advanced cartilage restoration), and capsular plication with routine complete closure (reconstruction or augmentation in revision setting with deficient iliofemoral ligament). Surgical contraindications include advanced osteoarthritis (Tonnis grade 2 or 3; joint space less than 2 mm), more than mild dysplasia (combination of lateral and anterior center edge angles, Tonnis angle, head extrusion index, regional and global surface area and volumetric coverage), excessive femoral version disorder (gait abnormalities, excessive internal rotation, version greater than 25°-35°). In patients with genetic collagen-based disorders (eg, Ehlers-Danlos syndrome), it is currently unknown if capsular plication demonstrates durable success. As with multidirectional instability of the shoulder, the risk is that, despite skillful execution of the surgical technique, the ligament may stretch following surgery.

Surgical Technique

Patients undergo general endotracheal anesthesia with complete muscle paralysis to ensure safe distraction.



Figure 9 Fluoroscopic examination under anesthesia with gentle 2-finger distraction demonstrating a very easy loss of suction seal and femoral head distal translation, emphasizing the role of stability provided by labral preservation and capsular closure.

Patients are positioned supine on the operative table (Advanced Supine Hip Positioning System with 2 Universal Hip Distractors and 2 Active Heel Traction Boots; Smith & Nephew, Andover, MA) with a foam pad system to achieve postless distraction (Xodus Medical, New Kensington, PA). Range of motion (flexion, internal and external rotation, abduction), external rotation recoil and dial testing, and fluoroscopic examination (distraction at 0° and 30° abduction, trochanteric-pelvic impingement testing) under anesthesia are performed (Fig. 9). Trendelenburg position of 12°-14° is obtained, distraction pulled, Trendelenburg then eliminated back to flat table position, and adequate distraction (10 mm) ensured with fluoroscopy. Fluoroscopy is then generally removed for the remainder of the procedure.

Three portal diagnostic arthroscopy (AL, modified midanterior portal [MMAP], and distal anterolateral accessory) commences. The AL portal is created at approximately 12:30 on the clockface and MMAP at 2:00, to minimize the length of the interportal capsulotomy. Minimal to no proximal iliofemoral ligament is resected to ensure ample capsule for closure. The capsulolabral interval is exposed to evaluate and manage the acetabular rim and subspine. To assist with visualization under the capsule, a “suspension technique” of capsular suspension with traction sutures is advantageous (both central and peripheral compartments).⁶⁴ Importantly, the surgeon must always be cognizant of subspine impingement (to distal anterior-antemedial femoral neck) in hypermobile hip syndrome due to excessive flexion motion typically present.⁴⁷ Central compartment checklist is evaluated—acetabular rim, subspine, labrum, transverse acetabular ligament, articular cartilage, chondrolabral junction, fovea, and ligamentum teres.

Labral preservation (repair or reconstruction) includes suture anchor placement into the acetabular rim, with preanchor placement drilling and 100% confirmation of nonarticular and nonpsoas fossa penetration.⁶⁵⁻⁶⁷ For the articular side, close-up arthroscopic visualization during drilling and anchor placement can ensure safety. For the psoas side, slow advancement of the drill while tapping as the drill advances, ensuring solid bone contact, for the entirety of the drill

depth. If drill angle is incorrect, then the surgeon must stop drilling further at that point, change drill entry location, change drill entry portal, or change from straight to a curved guide to ensure proper anchor location.^{44,68} Smaller all-suture designs are preferred for acetabular labral repair.⁶⁹ For most anchors placed between 10 and 4 o'clock, the distal anterolateral accessory portal can be used with a straight guide for proper placement. Posterior to 10 o'clock (labral reconstruction, and very rarely, repair), a posterolateral portal can be necessary. Anterior to 4 o'clock, the MMAP is typically a better angle for placement. Biomechanical outcomes demonstrate superiority of a mattress labral base refixation technique over that of a simple loop.⁷⁰⁻⁷² However, there are no short-term clinical differences between the 2 techniques, including a hybrid mix of the 2.⁷⁰⁻⁷²

Once central compartment work is complete, traction is discontinued. The precapsular fat pad is debrided to expose the lateral and medial bands of the iliofemoral ligament. The medial border is the iliocapsularis and the lateral border is the gluteus minimus. A T capsulotomy is created but does not extend distally into the zona orbicularis. Traction sutures are placed medially and laterally to visualize the peripheral compartment. Cam morphology is comprehensively corrected. Dynamic arthroscopic (and fluoroscopic, if necessary, but infrequent) examination is performed to ensure no residual impingement. The most distal aspect of the T capsulotomy is closed first, and then sutures placed progressively proximally up to the interportal. High strength nonabsorbable #2 suture is used for the T using a simple stitch design. If zona integrity is compromised, or if volumetric reduction is desired, then a stitch placed distal to the T cut in the medial and lateral sides of the zona using a nonabsorbable tape suture may be used. Typically, 3 or 4 sutures are placed in the T. A variety of suture passing and retrieving devices are available for use. The interportal capsulotomy is then approached and prepared. Proximally, minimal fat pad is resected and indirect head of rectus femoris requires simple elevation to expose the proximal capsule for plication. Depending on the capsulotomy size, 2-5 sutures, in a variety of configurations (simple, mattress, figure-of-8, shoelace), may be placed. For the interportal, a nonabsorbable tape is preferred over a suture. An inferior capsular shift is biomechanically optimal. Both anteromedially and posterolaterally, the peripheral tape should overlap noncapsulotomy ligament, to ensure complete closure. The junction of the T is likely a weak point in the repair, which may be mitigated, using a figure-of-8 criss-crossing at the junction to ensure security of the closure (Fig. 10). A dynamic examination is then performed to assess the integrity of the closure to help guide rehabilitation, which should restrict extension and external rotation in the early protection phase.

Postoperative care includes brace and derotational boot placement. The brace limits hip flexion to 90°, no permissive extension, 10° of permissive abduction. Boots ensure no nocturnal external rotation. Crutch-assisted foot-flat 20 pounds weight-bearing commences immediately. The brace and crutches are for 3-4 weeks duration and boots for 2 weeks duration (compliance permitting). Formal

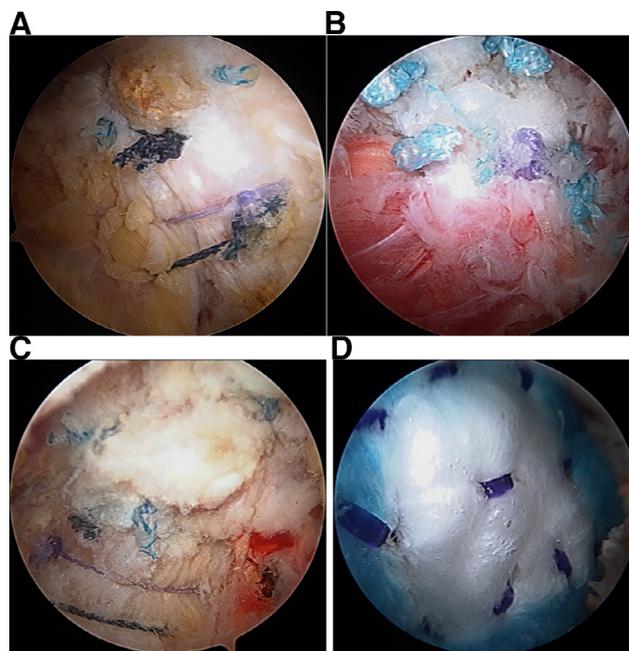


Figure 10 (A) Complete capsular closure using 3 nonabsorbable sutures in the T and 2 nonabsorbable sutures in the interportal repair; (B) complete capsular closure in a multirevision setting using 3 nonabsorbable tapes in the interportal, 3 nonabsorbable tapes and 1 nonabsorbable suture in the T; (C) complete capsular closure using 3 nonabsorbable sutures in the T and 3 nonabsorbable sutures in the interportal; (D) complete capsular repair, with superficial augmentation in the setting of thin ligament. Augmentation graft is Regeneten (Smith & Nephew, Andover, MA, USA), purified bovine Achilles tendon.

physical therapy begins preoperatively (education, teaching) and commences postoperatively on day 1 or 2. Passive motion, using therapist-guided circumductions, planar motion, continuous passive motion machines, and stationary bicycle, is key to success. A 6-week period of iliopsoas rest is strongly encouraged. Medical management includes heterotopic ossification prophylaxis using oral nonsteroidal anti-inflammatories; anti-fibrosis includes losartan in select cases (revision, known “scar-formers”, previous keloid); and chemical (and mechanical) thromboembolic disease prophylaxis.

Conclusions

Hypermobility hip syndrome is defined as the triad of symptoms (pain and instability), physical examination findings (confirms the subjective history), and imaging findings (corroborates the subjective and objective examination) consistent with hip instability, manifest along a spectrum from microinstability to dislocation. Soft tissue (capsule and labrum) and osseous (coverage, rotational profiles) structures may contribute. Imaging may reveal unique findings, include an anterior or AL hypermobile hip crevasse. Surgical management must ensure anatomical corrections of pathomorphology, labral preservation, and routine complete capsular closure, with variable degrees of plication and shift able to be achieved with modern instrumentation.

Postoperative care with physical therapy and medical management optimizes outcomes.

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