

# Complications Following Developmental Dysplasia of the Hip Operation in Children

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# Abstract

Hip dysplasia is a common childhood condition that is usually successfully treated based on potential alone in the majority of cases. However, whether the patient comes alive or does not respond to non-technical treatment, there are still some possible treatments. Highlights the prominent definitions for each can protect the exact tactics of rescue for the methods mentioned, as well as descriptive and visual explanations of how to perform these surgeries. We also discuss the wilderness care for patients undergoing DDH surgery and outline the potential evidence. Undiagnosed and untreated hip dysplasia can lead to health conditions, hip pain, and rapidly progressive osteoarthritis. Some patients may find evidence of variation after treatment with a pelvic girdle. Although there is excellent potential for a good outcome when DDH is diagnosed and treated, osteonecrosis continues to be a concern with all treatment methods.

A subset of patients from this young cohort will continue to have residual dysplasia or recurrent dislocation requiring return to the operating room. In the long term, hips without complications related to DDH treatment tend to do well. The most worrying complication is avascular necrosis of the bone head, ranging from 0% to 5%. Some post-operative complications such as Dislocation, Coxa Magna. However, it is necessary to consider before proceeding with hip reconstruction. Some classical surgical techniques are still performed for avascular necrosis of the femoral head. Recently, the author has performed incomplete proximal femoral osteotomy with good results. With Coxa Magna, the work of measuring the femoral head and neck ratio should be performed by several methods and determined and compared with the results of other authors. Consider before tactical treatment with clinical symptoms such as pain, limited mobility. Re-dislocation after a primary open reduction was the consequence of errors in surgical technique. There was an inverted transverse ligament, tight psoas tendon, eversion of the limbus, and densing anterior capsule. So, therefore, limbus, dense anterior capsule, with all hips cleared of scar tissue; adductor tenotomy; hips requiring iliopsoas tendon release, limbus eversion; hips requiring transverse ligament release.

Keywords: Acetabular Osteotomy; Open Reduction; Complications, Developmental Dysplasia of the Hip, Osteotomy Techniques

# Introduction

The hip is a "femoral head" joint. In a normal hip, the femoral head at the top of the thigh bone (femur) fits tightly into the acetabulum, which is part of the large pelvic bone [1]. In infants and children with developmental dysplasia of the hip (DDH), the hip joint does not form normally. The femoral head is loose in the acetabulum and can easily dislocate.

Developmental dysplasia of the hip occurs due to abnormal development of the hip, presenting in infants or young children with a spectrum ranging from dysplasia to hip dislocation. Developmental dysplasia of the hip includes a number of hip abnormalities, including instability, acetabular dysplasia, subluxation, and dislocation. These problems usually occur in children without other underlying medical conditions or pathologies. Previously called "congenital dislocation of the hip," the term developmental is preferred because not all are present or identified at birth [2].

The presentation varies from mild hip instability to true dislocation. The exact cause is unknown. It is multifactorial in nature, with a combination of genetic, environmental, and mechanical factors playing a role. Several genes have also been identified in familial cases.

Although DDH is most commonly present at birth, it can also develop in the first year of life. Recent research has shown that infants who are tightly swaddled with straight hips and knees are at significantly higher risk of developing DDH after birth [3]. As swaddling becomes more popular, it is important for parents to learn how to swaddle their newborns safely and understand that if swaddled incorrectly, it can lead to problems such as DDH.

The term congenital hip dislocation dates back to the time of Hippocrates. The condition, also known as hip dysplasia or developmental dysplasia of the hip (DDH), has been diagnosed and treated for several hundred years. Most notably, Ortolani, an Italian pediatrician in the early 1900s, evaluated, diagnosed, and initiated treatment for hip dysplasia [4]. Galeazzi subsequently reviewed over 12,000 cases of DDH and reported an association between marked shortening of the bowed femur and hip dislocation. Since then, there have been significant advances in the evaluation and treatment of DDH [5].

Abnormal findings on clinical screening should be further evaluated with radiographs or ultrasound, depending on the age of the patient [6,7]. Early diagnosis and management will prevent long-term complications such as persistent dislocation and early hip osteoarthritis [8].

Depending on the severity of the hip abnormality and the age of the patient, management of developmental hip dysplasia includes conservative treatments (e.g. activity modification, physical therapy, and bracing) or surgical intervention [9]. There is no good evidence that one surgical approach is more effective than another; therefore, management is selected based on surgeon preference and overall clinical status.

### Anatomy of the hip



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## Hip joint

#### Hip joint (Articulatio coxae)

The hip joint is a ball-bearing synovial joint that connects the pelvic girdle to the lower limb. In this joint, the head of the femur articulates with the acetabulum of the pelvis (hip) (Figure 1).

The hip joint is multiaxial and allows a wide range of motion; flexion, extension, abduction, adduction, external rotation, internal rotation, and rotation. However, compared to the shoulder (glenohumeral) joint, this joint sacrifices mobility for stability because it is designed to bear weight. The entire weight of the upper body is transmitted through this joint to the lower limb when standing. The hip joint is the most stable joint in the human body.

# Articular surfaces



Figure 2: Articular surfaces.

The hip joint is the articulation between the elliptical head of the femur and the hemispherical concavity of the acetabulum located on the lateral aspect of the hip bone. The femoral head is covered by articular cartilage (hyaloid) except for a rough central concavity, the epiphyseal fossa, which is the attachment surface of the ligamentum teres capitis femoris (Figure 2).

The deep non-articular central floor of the acetabulum is called the acetabular fossa. This area is free of cartilage and continuous with the acetabular notch. It contains loose connective tissue (fibroelastic fat pad) covered by synovial membrane. Attached to the rim of the acetabulum is a fibrocartilaginous ring called the acetabular rim. This structure deepens the acetabulum by slightly elevating the rim, thereby increasing the acetabular articular area by approximately 10%. Inferiorly, the rim of the acetabulum continues as the transverse acetabular ligament, which bridges the acetabular notch and transforms the notch into the acetabular foramen.

The upper surfaces of the acetabulum and the femoral head are subjected to the greatest stress (Figure 3). These areas typically have the thickest articular cartilage. The concave acetabulum and rounded femoral head of the hip, in addition to the anatomical relationship between the femur and pelvis, especially in the upright position, make this joint poorly articulated. The articular surfaces articulate best when the hip is in partial flexion and abduction.



Figure 3: Ilium bone.

# Joint capsule

The upper surfaces of the acetabulum and the femoral head are subjected to the greatest stress (Figure 3). These areas typically have the thickest articular cartilage. The concave acetabulum and rounded femoral head of the hip, in addition to the anatomical relationship between the femur and pelvis, especially in the upright position, make this joint poorly articulated. The articular surfaces articulate best when the hip is in partial flexion and abduction.

# Joint capsule



Figure 4: Linea intertrochanteric ossis femoris.

The capsule is considerably thicker anterosuperiorly, where it is subjected to maximum tension, especially in the upright position with the hip extended (Figure 4). Posteriorly, it is relatively thin and loosely attached. It has two main groups of fibers, longitudinal and circular. The outer longitudinal fibers of the capsule generally travel in a spiral fashion from the iliac crest to the proximal femur. The deeper circular fibers form a loop around the femoral neck, the zona orbicularis (ring zone or annular ligament), and have no bony attachments. The capsule is reinforced inferiorly by the pubofemoral ligament and posteriorly by the ischiofemoral ligament (Figure 5).



Figure 5: Acetabular labrum-Transverse acetabular ligament lunate surface of acetabulum hip joint.

# Ligaments

The ligaments of the hip joint can be divided into two groups; the capsular ligaments and the intracapsular ligaments. The capsular ligaments are the intrinsic ligaments of the joint capsule. There are three major capsular ligaments that play a major role in maintaining the integrity of the joint during various movements: the iliofemoral ligament, the pubofemoral ligament, and the ischiofemoral ligament. The intracapsular ligaments of the hip lie within the joint capsule and include the transverse acetabular ligament and the femoral head ligament.

# **Iliofemoral ligaments**



Figure 6: IlioFemore Ligament - Ligamantum Iliofemorale.

During extension, this ligament tightens, contracting the capsule and securing the femoral head in the acetabulum (Figure 6). This action limits the extension of the hip joint beyond the vertical position of approximately 10° to 20°.

#### **Pubofemoral ligament**

The pubofemoral ligament is located anteroinferiorly and reinforces the anterior and inferior aspects of the joint capsule. It originates from the iliopsoas, superior pubic ramus, and obturator crest of the pubic bone. It travels laterally and inferiorly to the inferior aspect of the intertrochanteric line, blending with the fibrous layer of the capsule and the medial band of the iliofemoral ligament.

#### **Ischemic ligament**

During extension, this ligament tightens, constricting the capsule and securing the femoral head tightly in the acetabulum (Figure 6). This action restricts extension of the hip joint beyond the vertical position to between 10° to 20°.



Figure 7: Ischiofemoral ligament - Ligamentum íchiofemorale.

The ischiofemoral ligament is the weakest of the three capsular ligaments (Figure 7). It is located posteriorly and reinforces the posterior aspect of the capsule. It is attached to the ischial bone medially below the acetabulum. It runs superolaterally around the capsule and posteriorly around the femoral neck to attach to the base of the greater trochanter, deep to the iliofemoral ligament. Some of the deeper fibers of the ischiofemoral ligament blend with the circumflex region.

#### Transverse acetabular ligament

The transverse acetabular ligament is a strong flat ligament that bridges the acetabular notch to form the acetabular foramen, through which neurovascular structures enter the hip joint. It completes the inferior defect of the acetabular rim and is peripherally continuous with the acetabular rim.

# Ligamentum teres capitis femoris

This ligament is a flat triangular band of connective tissue that does not contribute significantly to the strength and stability of the hip joint. Its apex attaches to the epiphyseal fossa while its base attaches to the acetabular groove and the transverse acetabular ligament. It is

covered by synovial membrane and carries a small branch of the obturator artery, the artery to the head of the femur, which contributes to the blood supply to the head of the femur.

#### Innervation



Baroreceptors located in the carotid sinus respond to decreased pressure (low blood pressure), signaling the activation of sympathetic nerves and constricting arteries and veins (Figure 8). Chemoreceptors in the carotid body and aorta are sensitive to oxygen pressure and respond by constricting blood vessels if the partial pressure of oxygen is too low. Vasopressin or antidiuretic hormone (ADH) is a vasoconstrictor released from the posterior pituitary in response to low blood volume. In contrast, atrial natriuretic peptide (ANP) is a vasodilator released from the atria in response to fluid overload in the heart.

The hip joint is innervated by the articular branches of several nerves that arise from the lumbosacral plexus (L2-S1). The nerve supply to a particular area of the joint usually corresponds to the innervation of the muscle passing through that area:

- The femoral nerve innervates the anterior aspect.
- The obturator nerve innervates the inferior aspect.
- The superior gluteal nerve innervates the superior aspect.
- The nerve to the quadratus femoris muscle innervates the posterior aspect.

# **Blood supply**

The blood supply to the hip joint comes from the medial and lateral femoral arteries (branches of the deep femoral artery) (Figure 9 and 10), the obturator artery, and the superior and inferior gluteal arteries. Together, these arteries form a network of anastomoses around the hip joint. This network of anastomoses gives rise to the retinaculum, which supplies the greatest amount of blood to the femoral head and neck. In addition, the obturator artery gives rise to the femoral head artery within the ligamentum flavum.



Figure 9: Medial circumflex femoral artery - Arteria circumflexa medialis femoris.

The arterial blood supply to the thigh comes directly from the external iliac artery. The external iliac artery becomes the posterior femoral artery after passing below the inguinal ligament and entering the femoral triangle. included in our collection of anatomy flashcards? Check out our bundles to save our anatomy flashcard collection?



# Clinical significance: Access to the femoral artery

The femoral artery lies superficially in the femoral triangle and can therefore be palpated just below the mid-inguinal point (halfway between the anterior superior iliac spine and the pubic symphysis). Its easy access makes it useful in clinical procedures such as coronary angiography.

### **Hip artery**

The hip joint is supplied primarily by the medial and lateral femoral arteries, which arise from the deep femoral artery (Figure 11).

The femoral head is additionally supplied by the fossa artery, which originates from the obturator artery. The fossa artery courses within the round ligament of the acetabulum.



Blood flow can be laminar or turbulent. Laminar flow is linear flow, mostly in the middle of a blood vessel. Turbulent flow is any discontinuity in laminar flow. Reynolds' number predicts the likelihood of turbulent flow. Higher numbers indicate higher likelihood of turbulent flow, and vice versa. Reynolds' number is directly proportional to density, velocity, and diameter, and inversely proportional to viscosity. Many organs, such as the heart, brain, and kidneys, rely on autoregulation or local control of blood flow to influence perfusion. Other organs rely primarily on sympathetic stimulation or external control of blood flow. The coronary arteries are locally regulated by hypoxia and adenosine, which dilates the vessels to maintain oxygenation of the heart. As the heart increases its contractility, the coronary arteries' oxygen demand increases.

#### Post-operative complications developmental dysplasias of the hip

#### Complications following developmental dysplasia of the hip operation

### Avascular necrosis

Avascular necrosis (AVN) is a known complication during the treatment of developmental dysplasia of the hip (DDH). It has the potential to alter the development of the head or acetabulum and prevent the best outcomes. Although previous literature has assessed the risk of AVN and strategies to avoid it, studies on the impact of AVN on outcomes are scarce. In this systematic review, we aimed to investigate the impact of AVN on outcomes during the treatment of DDH. The analysis did not reveal any statistically significant differences between the AVN and non-AVN groups in terms of clinical or radiological parameters. Interestingly, patients who underwent index surgery at a young age were at higher risk of subsequent surgeries, with acetabular osteotomy being the most common secondary procedure. The negative impact of AVN may not be as severe as previously thought. Therefore, the fear of AVN should not take precedence over the primary goal of DDH treatment, i.e., achieving a stable concentrically mobile hip.

### Pathogenesis

The pathogenesis of A is complex. From a mechanical point of view, deformation in the FH will occur when the force acting on the FH is greater than its ability to resist deformation. Animal models have shown that necrosis reduces the mechanical and supporting properties of the FH, articular cartilage, and bone. It is thought that the mechanical properties of infarcted bone are affected by different mechanisms in different stages of the disease.

First, in the avascular stage, the increase in calcium in the necrotic bone makes the bone susceptible to micro-damage, affecting the mechanical properties of the FH. Necrosis will lead to the breakdown of osteoclasts, osteoblasts, and osteoblasts, causing undetected and/ or unrepaired microcracks. Then, in the revascularization phase, the necrotic bone is resorbed, affecting the mechanical properties. The hip is one of the major weight-bearing joints. It is important to consider the forces acting on the joint, as they will affect the stress level in FH. From a radiological point of view, the process of ischemia and subsequent bone remodeling is divided into several phases. The determination of the phase is of paramount importance. The duration of each phase varies greatly, but in general, the necrotic and fragmented phase lasts about six months; the enjoyment phase, from 18 months to three years; and the final phase, until bone maturity. According to other authors, the fragmentation phase lasts about a year and the recapture phase, from three to five years (Figure 12).



Figure 12: Pathogenesis of avascular necrosis with LCP.

#### Diagnosis

Due to the lack of information, the diagnosis of AVN can be difficult; however, there are several important diagnostic criteria. Differential diagnoses that must be considered based on radiographic findings include coxacidosis, Meyer dysplasia, epiphyseal dysplasia, spinal cord dysplasia, and chondroblastoma (Figure 13).

#### Classification

To predict prognosis and decide on appropriate treatment, there are classifications that mainly consider the affected area and region.

Catterall, 1971, divided the disease into 4 grades, according to the degree of epithelial damage - Grade I: 0 - 25%; Grade II: 25 - 50%; Grade III: > 50% and Grade IV: 100%.



Figure 13A and 13B: AP radiography. In the AP X ray the deformity of the hip and femoral head characteristic of AVN is demonstrable, A healthy control, B AVN patient. Courtesy of INR LGII genetics laboratory 2017.

In 1984, Salter and Thompson described a classification consisting of two groups (A and B), determined by the degree of subchondral fracture visible on axial radiographs in the early stages of the disease. The disadvantage of this method is that not all patients are diagnosed at an early stage (Figure 14) [10].



**Figure 14a-14e:** The dotted lines divide the femoral head into medial, medial, and lateral pillars. The gray dotted line represents the midline of the lateral pillar. Its dotted line is the area of necrosis. The zigzag shows the size of the subchondral fracture (fs). A: Femoral head sound. B: Area lost 25% of total area; its discontinuous edge is an area of necrosis and has an accessory fissure (fs). Compared with ~50% total surface area loss, increased necrotic area, increased fs size, and <50% decrease in lateral column height. C: Loss of >50% of total surface, gain of fs, and loss of height of side stop ~50%. D: Maxillary space loss of almost 100% of the surface, maximal involvement of the lower wall, and >50% involvement of the lateral cavity.

### **Clinical parameters**

Clinical parameters Before surgery, information was collected on the type and amount of medication, history of trauma, age of onset of symptoms, time of onset of symptoms. The patient's main complaint was hip pain with claudication and knee pain. Clinical complaints and their onset times were recorded, and flexion, abduction, adduction, and internal and external rotation of the hip and limbs were also recorded with a goniometer, and they were evaluated before and after surgery.

During the physical examination, limb length discrepancy was assessed by clinical measurements of the distance between the main iliac spine and the sternum and detection of discrepancy in foot length.

**Trendelenburg test**: A positive Trendelenburg test indicates that the hip abductors are inactive due to weakness or pain inhibition and are unable to perform their role of stabilizing the pelvis on the weight-bearing legs. To perform the test, the patient stands on the unaffected leg and bends the opposite knee to a right angle. The pelvis should be level or slightly tilted on the non-weight-bearing side. The patient then stands on the affected leg and bends the knee of the opposite leg. If the pelvis is depressed on the non-weight-bearing side, this indicates a positive Trendelenburg test result.

#### Radiography

As the primary radiographic outcome parameters, most recent radiofrequency and frog-leg scans were considered. The parameters evaluated included: flattening of the femoral head, acetabular changes, shortening of the femoral neck, trochanteric hypertrophy, angel femoral neck abnormalities, and femoral head hypertrophy. Signs of osteoarthritis were considered as secondary outcome parameters.

The radiographic evaluation was performed by an experienced radiologist. Anterior pelvic radiographs were performed in neutral, lateral abduction, lateral abduction, and lateral abduction positions as well as frog-leg scans for all patients. The involvement of the femoral head or metaphysis was classified according to the Catterall [11] and Herring., *et al.* [12] classifications. Both the extrusion index [13] and the Wiberg CE angle [14] were measured and classified, and patient risk factors were also determined.

The Wiberg central angle [14] is defined as the angle between the line connecting the acetabular rim, the center of the femur, and the vertical line. This method is also commonly used to determine the severity of AVN. The Wiberg angle should be between 25 and 40 degrees. In a protruding acetabulum, this angle exceeds 40 degrees.

**Coxa magna:** Asymmetric circumferential enlargement of the femoral head is an important sequela of pediatric disorders such as Legg-Calvé-Perthes disease. Definitions vary due to the lack of controls and the lack of studies on the distribution of femoral head asymmetry (determining coxa magna above 10%) [15].

Shenton's line: It is an imaginary curve drawn along the lower border of the superior ramus (superior border of the obturator foramen) and along the infraorbital aspect of the femoral neck. This line should be continuous and smooth.

A prominent subluxation was considered if the Shenton Hay line was disrupted (Stulberg., *et al.* [16] 1981). Postoperative inhibition was assessed by visual impression on radiographs taken 2 - 3 months later.

Postoperatively, the femoral head was considered present if the femoral head was in the acetabulum. Acetabular flatness was measured in unilateral cases on both the affected side and the contralateral side without signs of AVN.

# **Radiographic evaluation**

**Catterall classification**: According to the Catterall classification, there are 4 groups of patients. In group 1, the radiographic changes are limited to the anterior portion of the femoral epiphysis. In group 2, there are approximately 50% anterior lesions, with intact medial and

lateral portions of the femoral head, plus lesions anterior to the metaphysis. In group 3, approximately 75% of the epiphysis is affected, including the posterior portion, with diffuse metaphyseal reactions. In group 4, the entire epiphysis is affected. The 4 risk signs include the Gage sign - CV radiolucency in the lateral portion of the epiphysis, lateral calcification of the epiphysis, lateral dislocation of the femoral head, and horizontal physis.

Although the Catterall system has been widely used for many years in most studies, previous studies have found that it does not provide acceptable levels of interobserver and intraobserver acceptability. Other studies have found acceptable levels of intra- and inter-observer agreement [18]. It was published in 1992, because of Catterall's poor reproducibility. It is also known as the lateral pillar classification. For its author, the femoral head is divided into three pillars: (1) Lateral pillar occupies 15 to 30% of the head - Medial pillar, the middle pillar occupies 50%; (2) Intermediate pillar, average 20 - 35%. This classification distinguishes 3 groups according to the degree of their involvement. Herring further describes the B/C borderline stage, characterized by preserved lateral pillar: (1) The presence of a clear lateral projection ("V de Gage" sign) (2) Eccentricity of the head or lateral subluxation; (3) Horizontal position of the intervertebral disc; (4) The presence of a metaphyseal reaction; (5) The presence of lateral calcification. Radiographs were evaluated using the Stulberg classification [16]. Type I hips were defined as completely normal heads (despite the spherical shape), shorter femoral necks, or a commonly sloped acetabulum. Type III heads were described as spherical, ovoid, mushroom-shaped, or umbrella-shaped, but not flat. Type IV hips were described as having flat femoral heads with abnormalities in the femoral neck and acetabulum. Type V hips were described as having flat heads and flat femoral necks and acetabulums

#### Surgical treatment

Indications for surgery: Indications for surgery are femoral head dislocation or severe disease (Catterall class II-IV with signs of early risk or disease progression despite conservative treatment).

The decision to treat is based on age of onset, Catterall grade [11], persistent limitation of hip motion (especially if extension is less than 30°), and severe dislocation and/or bone cysts.



Figure 15A and 15B: A: Osteotomy in variant 1. B: Osteotomy and Inserted Kirschner in variant 2.

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Surgical technique: Patients were divided into two categories: Variant 1 with Catterall II-III; Variant 2 with Catterall IV.

General anesthesia was used for all patients. The patient was placed in the supine position on the radiopaque orthopedic table (Figure 15).

Using a lateral surgical approach to the proximal femur with incomplete osteotomy. The osteotomy was performed at the level of the lesser trochanter in such a way that the greater trochanter was not disturbed. Using a straight osteotome and the osteotomy line should be perpendicular to the femoral shaft and directed toward the opposite femoral wall, the femoral cortex was kept intact for stability; with half the femoral diameter in variant 1; two-thirds of the femoral diameter in variant 2 (Figure 15).

Variant 2 patients, insertion of a 2 mm Kirschner wire from the greater trochanter to the opposite femoral wall was performed (Figure 2). Eight weeks of spica caste were performed in all patients as part of the standard postoperative treatment. After this period, partial weight bearing was allowed until the bone graft had healed.

Postoperatively, patients were followed up monthly for 1 year and then every 3 months after 1 year.

### **Outcome assessment method**

In the study of treatment outcomes, we used the Mose method (1964) [19] using a transparent device described by Edgren (1965) [20]. Good results included spherical heads of equal radius on anteroposterior and lateral radiographs. (All lateral views were taken at the Lauenstein Plate position). Fair results had heads that deviated from the circle by up to 2 mm. Poor results had irregular heads, with contours that differed by more than 2 mm. Although this system does not take into account the relationship between the head and the acetabulum, it provides a very important analysis of the anatomical outcome of the disease and has considerable prognostic value, due to the spherical shape of the femoral head (Figure 16). It seems to be important for the functional stability of the hip joint [19,20].

#### **Complications:**

- 1. Wound infections or neuromuscular complications.
- 2. Fracture at the osteotomy site





Figure 16A-16D: A: Preoperation. B: Postoperative 3 months. C: Postoperative 6 months. D: Postoperatively, lates result (4.6 years).

- 3. Nonunion at the osteotomy site
- 4. Kirschner broken
- 5. Coxa Magna.

### Re-dislocation postoperative developmental dysplasia of the hip

Early dislocation after initial closed or open reduction (OR) of developmental dysplasia of the hip (DDH) is a known complication. If left untreated, this condition can lead to poor long-term outcomes [21]. Reported rates of dislocation range from approximately 8% to 20% [22,23]. Failure to achieve concentric and stable reduction, either with closed or open surgery, may be due to several factors: suboptimal initial treatment, the learning curve of the treating surgeon, post-reduction spica care, and factors inherent to the dysplasia of the hip [24].

The degree of stability of the hip after closed reduction or OR is largely surgeon dependent as it is based on the perception of the "feel" of the cartilaginous femoral head. Despite meticulous surgery and casting, some hips dislocate. Computed tomography (CT) or magnetic resonance imaging (MRI) after reduction is recommended to visualize the location of the nucleus ossification (ON) and assess the quality of reduction [25].

Normal epiphyses begin to ossify at 4 - 6 months of age due to physiological and mechanical forces between the femoral head and the acetabular cartilage [26]. These forces become abnormal in DDH, causing a delay in ossification. Experimental studies in porcine models have shown that the bony ON provides structural rigidity and also has the ability to resist compressive forces, thus providing protection against avascular necrosis (AVN) [27].

Some authors have recommended treatment of DDH after the appearance of this ON to prevent late AVN [28]. Although the authors have considered the relationship between ON and AVN, its presence or absence during initial closure or revision surgery remains unclear. Bolland., *et al.* noted that the width and height of the ON on the untreated side were less than the contralateral side and that after reduction, the width increased but the height never caught up to the contralateral side and remained less at long-term follow-up. To date, only one study has examined the ossification of the femoral head during closed reduction (CR) and its effect on long-term outcome [29].

The goal of treatment for developmental dysplasia of the hip (DDH) is to achieve and maintain a stable concentric reduction as quickly and easily as possible. It is generally accepted that open reduction is indicated for developmental dislocations of the hip when a concentric, uniform, and stable reduction cannot be achieved with closed methods. Uncommonly, open reduction fails and the dislocation recurs.

Reported rates of recurrence after open reduction vary. It appears to depend on the approach used, the age of the patient at the time of initial reduction, and perhaps the surgeon's level of expertise. It has been reported to occur in 0% - 8% after open reduction via an anterolateral approach [21], and when a medial approach is used, this figure increases to 5% - 14% [22].

Most failures after open reduction are the result of surgical technique errors. Most reductions fail to adequately release the anteromedial joint capsule and the underlying articular structures.

Radiographs taken immediately before the re-procedure were evaluated for avascular necrosis, coxa magna, and degree of displacement. The latest available radiographs were graded according to the modified Severin classification [24].

# Acetabular index angle

The acetabular index is the primary variable used to evaluate correction of acetabular dysplasia and subsequent maintenance. The term acetabular index was introduced by Kleinberg and Lieberman in New York in 1936 to create a radiographic sign [25]. "The angle formed between the roof or iliac portion of the acetabulum and a horizontal line passing through the tripartite cartilages".

#### **Mean length**

The hip was held in place with both hip and knee in neutral extension, we used a measuring tape to measure the length and compare both lower limbs for "Leg Length Discrepancy", starting from the anterior iliac spine to the superior lateral ankle. The mean age at the first open reduction was 24 months (13 to 36). The second reduction was performed at a mean age of 26.3 months (17 to 42) and the mean age at last follow-up was 79.7 months (58 to 105). The mean follow-up was 42.4 months.

The surgical and clinical records were then reviewed to identify patients who required a second open reduction of the dislocated hip. Clinical and surgical records were reviewed for both groups to determine demographic information, clinical and MRI data (Figure 17), follow-up time, and any additional procedures performed at the time of initial open reduction. For the study population, the time to re-dislocation was recorded as well as the number and nature of subsequent procedures performed. Operative notes from revision surgeries were reviewed to determine the surgeon's subjective reasons for re-dislocation.



**Figure 17:** Postoperatively 15 weeks, left hip with subluxation: A: Roentgenography shown acetabular index with normal limits; Broken Shenton line; B: MRI shown thick capsule and hypertrophic transverse ligamentum.

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#### **Technique of operation**

Each case has its own unique problems but some basic principles can be discussed profitably. The zigzag osteotomy [30] is the most useful approach, modified as necessary to allow for a previous scar.

The capsule is enlarged and the acetabulum is then cleared of fibrous fatty tissue that may have filled it. The surgeon's finger can be passed through the acetabular groove and any obstruction to reduction identified. Excess tissue is excised and the remaining capsule is sutured down to the pelvis, taking particular care at the anterior rim of the acetabulum. It is then opened in a T-shape and the joint inspected. Adhesions, which are often dense, are then released. Any obstruction to reduction is usually the psoas tendon, the round ligament.

The transverse ligament, the glenoid margin or the zygomatic bone that was not treated in the primary procedure is then released. If the glenoid margin is reversed, it is reversed by making radial cuts. The acetabular groove is cleared of scar tissue. After the hip is reduced, radiographs are used to determine whether the hip is involved.

In cases of anterior displacement, displacement of the femoral head may not be too difficult, but posterior displacement requires very careful dissection around the capsule (Figure 18-20). The femoral head is often trapped posteriorly and the capsule is pulled over the acetabular opening, making identification difficult. In some cases, Kirschner wires and intraoperative radiographs are required to ensure acetabular position.

If after these soft tissue procedures there is still difficulty in reducing the hip, femoral shortening is performed through a second lateral incision and in these cases the anterior tilt of the femoral neck is also corrected.

Any excess posterior superior capsule is excised and the capsular adhesions to the lateral wall of the pelvis are released. Capsular closure is performed with heavy-duty sutures. In femurs with significant femoral anteversion, external rotation osteotomy is performed ten to twelve weeks after open reduction (Figure 18-20).

In some cases, despite all the above steps, the hip remains unstable and in these cases, Kirschner wires are used to maintain concentric reduction. The wire is passed through the greater trochanter and into the iliac bone above the capsule after capsular suturing and is left in place for three weeks.

Postoperative immobilization in spica hips is 6 to 12 weeks depending on the need for a second-stage femoral osteotomy.

A spica cast is applied immediately after surgery, with the hip displaced 30 degrees, abducted 45 - 50 degrees and internally rotated 20° - 30°.

Reduction is confirmed clinically and by intraoperative radiography. Postoperative immobilization in spica hips is maintained for 10 - 12 weeks.

We evaluated postoperative outcomes by combining radiographic criteria according to Severin [31], Kalamchi [32] (Table 1) and Clinical criteria using the modified McKay criteria [33] (Table 2), and Modified MCKay [35] (Table 2).

Postoperative follow-up was performed every 3 months after hip spica excision. At each follow-up, children were assessed for pain, limp, ROM, and limb length discrepancy.

Postoperative radiographs were also evaluated for Tonnis score, Shenton line rupture, presence of bony ON, and hip dislocation or subluxation. AVN was graded using the Bucholz and Ogden criteria [34].

Functional assessment was performed using the modified McKay score [35].

# Complications Following Developmental Dysplasia of the Hip Operation in Children

18

Classifications	Description			
Severin				
1	Normal			
2	Moderate deformity of femoral head or neck or acetabulum			
3	Dysplastic no subluxed			
4	Subluxed			
5	Head articulating with secondary acetabulum in upper of the original acetabulum			
6	Dislocated			
7	Arthritic			
Kalamchi				
1	Changes affecting the ossific nucleus			
2	Lateral physeal damage			
3	Central physeal damage			
4	Total damage to the head and physis			
Table 3. Modified McKay criteria for clinical evaluation [9] [10].				
Grad	Critia			

Grad	Critia
Excellent	Stable, painless hip; no limp; negative Trendelenburg sign; full range of movement
Good	Stable, painless hip; slight limp; slight decrease in range of movement
Fair	Stable, painless hip; limp; positive Trendelenburg sign; and limited range of movement or a combination of these
Poor	Unstable or painful hip or both; positive Trendelenburg sign

Table 1: Details of radiological according to the Severin [31] and Kalamchi [32] and Modified MCKay [35] classifications.



Figure 18: Eversion of the limbus and adhesions with ligamentum teres.



Figure 19: Scar tissue in acetabular notch.



Figure 20: Densing anterior capsule.

### Coxa magna

Our understanding and treatment of congenital hip dislocation (CDH) has changed over the past 25 years [36]. Prenatal, perinatal, and postnatal factors contributing to dislocation have been identified, and early diagnostic techniques have improved [37]. Newborn screening programs and physician awareness have reduced the incidence of untreated hip dislocation in older children. The focus of treatment has shifted from forceful manipulation and static casting to gentle nontraumatic reduction and dynamic splinting. Fortunately, the incidence of serious treatment complications such as avascular necrosis has decreased. Many factors have contributed to this decline,

including the use of dynamic splinting in infants, the use of preoperative traction, and the avoidance of extreme casting positions [38]. Despite these precautions, changes in the shape of the femoral head continue to be observed after surgical treatment of some hip dislocations. The purpose of this article is to explore one of these changes, namely Coxa magna. When discussing CDH, standard references and textbooks only briefly mention coxa magna [39]. They point out that coxa magna often occurs after avascular necrosis. Few mention that coxa magna can occur in the absence of avascular necrosis, and no one has provided a consistent quantitative definition of coxa magna. Furthermore, the prevalence of coxa magna is unknown, its relationship to various treatment modalities is unknown, and the impact of coxa magna on the acetabulum is unclear. In this study, the prevalence of coxa magna and its relationship to treatment regimens were investigated. The relationship between the larger femoral head and the acetabulum was quantified to predict future hip development. All X-rays are standardized to ensure a true front-to-back view of the pelvis with both legs in a neutral position.

Coxa magna is a medical term used to describe enlargement or hypertrophy of the femoral head (the ball-shaped part of the thigh bone) and the neck of the hip joint. "Coxa" refers to the hip, while "magna" means large or great in Latin.

Coxa magna can occur as a congenital condition, meaning it is present from birth, or it can develop later in life due to a variety of factors. It is often associated with certain developmental or structural abnormalities of the hip joint, such as hip dysplasia or slipped capital femoral epiphysis (SCFE).

In people with coxa magna, the enlarged femoral head and neck can cause mechanical problems within the hip joint. This can lead to hip pain, limited range of motion, difficulty walking or running, and hip instability. It may also increase the risk of developing degenerative joint conditions such as osteoarthritis later in life.

Coxa magna refers to a condition in which the head is larger than the normally unaffected side. This is typical of Legg-Calve-Perthes and is the result of an imperfectly controlled but somewhat excessive repair process. Coxa magna can only occur due to excessive repair because the cartilage tissue grows too large for its intended size.

The articular and epiphyseal cartilages on the upper weight-bearing side of the head continue to grow through diffusion of nutrients from the synovial fluid. The synovial fluid does not appear to be impaired by the anatomical pathology; in fact, it appears that the continuation of the normal synovial fluid diffusion nutrition is supplemented by stimulation from the excessive blood supply to the hip during repair to create a larger than normal tissue mass.

There are two possible types of coxa magna: one with no long-term negative consequences or the other with more significant longterm consequences. In the first scenario, the growth of the acetabulum may correlate with the growth of the femoral head in that the femoral head-acetabulum relationship remains appropriate and does not lead to long-term problems.

The coxa magna is connected to a head that remains spherical. Long-term problems begin when the growth of the femoral head disrupts the growth of the acetabulum and the head is not only large relative to the acetabulum but also deformed in that it does not connect perfectly with the acetabulum. The size and shape of the cartilaginous pattern of the femoral head can be assessed with arthrography and magnetic resonance imaging (MRI).

#### Blood supply to the proximal femur

There are three major blood supplies to the proximal femur: the extracapsular arterial ring; the ascending jugular vein (retinal branch); and the ligamentum teres (Figure 21). The outer capsule is formed primarily by the medial and lateral femoral epiphyses, which then give rise to the epiphyseal and metaphyseal branches.

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Figure 21A and 21B: A: Anatomy of arterial supply at the femoral head; B: Arterial supply at proximal femur.

The anterior portion of the lateral capsule is formed mainly by the lateral femoral coronary artery. The posterior, lateral, and medial aspects of the ring are formed by the medial femoral coronary artery. Chung [39] found that the largest amount of blood flow to the femoral head is via the ascending lateral cervical artery (the terminal end of the medial femoral artery). This corresponds to the lateral epiphyseal artery described by Trueta, which passes through the capsule in the posterior transitional fossa. The extremely important ascending lateral cervical artery passes through this fossa, which is extremely narrow in children under 8 years of age.

# Etiology

- Legg-Calve-Perthes disease,
- Transient synovitis of the hip,
- Developmental dysplasia of the hip,
- Trauma,
- Infectious arthritis/osteomyelitis,
- Juvenile rheumatoid arthritis,
- Chronic slipped capital femoral epiphysis.

#### Parametar

The following measurements were taken from the AP image: (1) femoral head diameter and (2) minimal neck diameter (Figure 22). The diameter of the femoral head was measured by fitting a perfect circle to the femoral head and selecting the best fitting circle such that the femoral head did not extend beyond the contour of the circle by > 1 mm in the method described by Mose [40]. The minimum neck diameter was obtained by identifying and marking the axis of the femoral neck and then finding a line perpendicular to the axis that represented the shortest distance between the superior and inferior surfaces of the neck. The absolute difference was obtained by subtracting the smaller sample (left or right) from the larger sample. The percentage of asymmetry was determined using the following equation:

Percent asymmetry = (maximum-minimum)/(average of maximum and minimum) X 100

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The mean and standard deviation were determined for each measurement performed for the entire population and for various subgroups of the population based on demographic characteristics. The absolute amount and percentage of asymmetry between the left and right femurs were classified into different categories. The inter- and intra-observer reliability of the measurement procedure was determined by calculating the intraclass correlation coefficient (ICC). Inter-observer reliability was calculated by having both observers independently complete all femur sets using the same method. Intra-observer reliability was calculated for both observers by repeating 50 randomly selected femur sets (left and right femurs: femoral diameter and minimum neck diameter) at >8 weeks intervals.

Early literature described only the quantitative amount of coxa magna enlargement, not its size [41]. A review of the existing literature shows that definitions and methods of quantitative coxa magna measurement vary widely (Table 2).

#### Symptoms

- Hip pain: People with coxa magna may experience persistent or intermittent pain in the hip joint. The pain may be dull or sharp and may be worse with movement.
- Limited range of motion: Enlargement of the femoral head and neck can limit the normal range of motion of the hip, making activities such as walking, running, or bending difficult.
- Hip instability: Coxa magna can lead to hip instability, causing the hip joint to feel "giving way" or unstable during movement.
- Limping: Due to pain and reduced mobility, people with coxa magna may limp significantly when walking or running.



Figure 22: Anteroposterior view of femoral specimen. Best fit circle shown around the femoral head. The minimal neck diameter was defined as the minimal distance about the neck perpendicular to the neck axis. Line CD represents the minimal neck diameter (in this specimen). Line AB represents the neck axis.

Authors	Pathology	Age at Follow-up (y)	Coxa Magna Definition
De Valderrama., <i>et al</i> . [42]	Transient synovitis	21	>10% diameter difference
Nachemson and Scheller [43]	Transient synovitis	25	>1mm diameter difference
Stulberg., <i>et al</i> . [44]	LCPD	47.3	10% diameter difference
McAndrew and Weinstein [45]	LCPD55.5	55.5	Femoral head ratio <0.9*
Gamble., <i>et al</i> . [46]	DDH	5.7	>15% diameter difference
Kallio [47]	Transient synovitis	5.6	>2mm diameter difference
Leitch., <i>et al</i> . [48]	LCPD	15.7	>3mm radius difference
Imatani., <i>et al</i> . [ 49]	DDH	14	REF>120%w
Shigeno and Evans [50]	LCPD	14.7-18.5z	>10% diameter difference
Luhman., <i>et al</i> . [51]	DDH	8	>15% diameter difference
Rowe., et al. [52]	LCPD	14.4	>10% diameter difference
Larson., <i>et al</i> . [53]	LCPD	20.4	Femoral head ratio <0.9*
Hung NN [54]	DDH	11.5	> 15% diameter difference

Table 2: Coxa magna determination with cause, age and incidence of femoral head via neck.

\*Femoral head ratio is affected hip femoral head/unaffected hip femoral head.

wREF or ratio of enlargement was defined as follows: (Radius of one femoral head) [2] p/(Radius of other femoral head) [2] PX100.

Years of follow-up were determined for men and women, with 14.7 years for men and 18.5 years for women.

Mean age at presentation was not available, 20.4 represents follow-up time from initial presentation.

CHD represents congenital hip dysplasia/dislocation; LCPD, Legg-Calve'-Perthes disease.

# Diagnosis

- Physical exam: A healthcare professional will examine the hip joint, looking for signs of enlargement, limited range of motion, instability, and any associated pain or discomfort.
- Imaging tests: An X-ray, magnetic resonance imaging (MRI), or computed tomography (CT) scan can provide detailed images of the hip joint, showing the extent of enlargement of the femoral head and neck, as well as any associated structural abnormalities.

# Treatment

- **Conservative treatment:** Nonsurgical options may include physical therapy exercises to improve hip strength and flexibility, pain control with medications, activity modification, and the use of assistive devices such as crutches or canes.
- **Surgical treatment:** Severe cases or cases with persistent symptoms may require surgical treatment. Procedures may include hip osteotomy, which involves reshaping or repositioning the hip joint, or total hip replacement, in which the damaged joint is replaced with artificial components.

It is important to note that the specific treatment will depend on individual factors, such as age, overall health, the underlying cause of the coxa magna, and the severity of symptoms.

# Procedure

Thirty-four hip dislocations had skin traction before surgery. No bone traction was used. A straight anterior "bikini" incision or an iliofemoral incision was used. Open reduction involves resection of the limbus and round ligament, removal of any fibrofatty tissue from

the acetabulum, and crimping of the capsule. A femoral shortening procedure is performed if the reduction is considered to be placing excessive stress on the femoral head. If a hip is reduced without opening the capsule after femoral osteotomy, this is counted as a closed reduction. A rotational reduction of the femur is performed if excessive anterior tilt compromises the stability of the reduction or requires significant internal rotation of the limb. If the anterolateral aspect of the femoral head remains uncovered, a pelvic osteotomy is performed. There were no intraoperative or postoperative complications. All patients received a double hip cast in the so-called "human position" with 70-90° flexion, 30-60° extension, and minimal rotation. Immobilization lasted for 12 to 16 weeks. The patient was then placed in a Cima type open splint.

# Surgical approach

Femoral head and neck reconstruction was performed according to the technique used was modified from the new technique published by Ganz and colleagues (Figure 23) [55,56].

Changes in the shape of the femoral head are known to occur after treatment for CDH. The most severe and harmful changes occur after avascular necrosis, when residual growth plate damage may result in varus or valgus deformities, shortened femoral necks, or large flat femoral heads. Standard orthopedic reference textbooks state that a large femoral head, or coxa magna, commonly occurs after avascular necrosis [39], but some textbooks note that it may also be present even in the absence of apparent avascular events. Unfortunately, most authors do not provide a quantitative definition of coxa magna, and its relationship to treatment modalities is not routinely reported. For this study, a femoral head diameter that was at least 15% larger than the contralateral head was defined as coxa magna. As noted in figure 1, the coxa magna occurred in 16 (33%) of our patients: four with avascular necrosis and 12 without radiographic evidence of





**Figure 23**: Femoral head reduction osteotomy (FHRO) technique. A: Coxa magna with saddle-shaped femoral head. The lateral retinacular and the medial branch of the medial femoral circumflex artery are shown. B: Surgical dislocation is performed after a trochanteric flip osteotomy. C: The medial part of the stable trochanter is resected to decompress and mobilize the piriformis fossa and the retinacular pedicular flap. D: The lateral femoral head osteotomy is performed, preserving its vascular pedicle. E: The medial femoral head osteotomy is performed and the segment of the femoral head resected. F: The lateral segment is advanced and fixed to the medial part of the femoral head. G: A relative neck lengthening is achieved by transferring the trochanter distally and laterally. The goal is to achieve normal joint orientation and for the tip of the greater trochanter to be at the level of the center of the femoral head. aMPFA, anatomic proximal femoral shaft angle; NSA, neck shaft angle. H: Headless screws are used to fix the femoral head and neck. Screws with washers are used to fix the greater trochanter.

avascular necrosis. There was no correlation between the size of the head—that is, the magnitude of the coxa magna—and the presence or absence of avascular necrosis. Table 1 shows that three factors were significantly associated with the occurrence of the coxa magna: femoral osteotomy (100%), open reduction (75%), and surgery at a younger age. For hips with a coxa magna, the mean age at surgery was 15.6 months, whereas the mean age was 35.8 months for hips without a coxa magna. The 33% incidence of coxa magna in this study is slightly lower than that reported by Sakamaki [57]. He reviewed 303 hips in 247 patients and noted a 47% incidence of coxa magna after open reduction, but only 5% after closed reduction. Berkeley., *et al.* [3] noted that in 81% of their unilaterally dislocated hips, the diameter of the femoral head was 2-10 mm larger than the contralateral side. [58] found that coxa magna occurred with high frequency after total limb amputation. According to Sakamaki [57], our work, and Berkeley., *et al.* [3], it appears that coxa magna is more common than generally appreciated. In these studies, growth stimulation of the femoral head was quite common.

Postoperatively, the patient can be placed in continuous passive motion. The author prefers to use this modality at home for 6 weeks. This method should be combined with physical therapy to maintain the active and passive range of flexion and extension of the hip joint. Because continuous passive motion does not fully extend the hip, the therapist should make sure to stretch the hip to full extension. The hip may be passively abducted for the first 6 weeks. Active abduction should be avoided until the greater trochanter is healed (usually at 6 weeks). Passive and active adduction and external rotation range of motion should be avoided because they stress the capsular repair and the trochanteric fixation. These motions can be resumed after 6 weeks. Weight bearing is restricted to touch-down weight bearing for 3 months. After 6 weeks the extremes of motion should be stretched passively.

To ensure that this is correctly done, the physical therapist should be educated about the use of the contralateral hip to lock the pelvis. For example, to maximize abduction stretch the contralateral hip should first be maximally abducted to lock the pelvis from moving as one stretches the affected hip into abduction. Full extension of the hip requires first flexing both hips, then leaving the contralateral hip in maximum flexion (Thomas test) while pushing the affected hip into extension. Full flexion requires prior hyperextension of the contralateral hip before stretching the affected hip into flexion; this locks the pelvis into full extension so that all flexion motion seen is real. Finally, internal and external rotation can best be done by rotation of the contralateral hip into internal and external rotation, respectively.

Longer term follow-up is important to determine whether the early excellent results will hold up, and whether the natural history of the disease will be altered by this osteotomy procedure.

# **Summary**

- Coxa magna is an enlargement or hypertrophy of the femoral head and neck in the hip joint. It can be present from birth or develop later in life due to various factors. Symptoms include hip pain, limited range of motion, instability, and a noticeable limp. Diagnosis involves physical examination and imaging tests.
- Treatment is conservative measures or surgical intervention. Exercise, tailored to individual needs, may include a range of motion exercises, strengthening exercises, and low-impact aerobic activities. Consulting a healthcare professional is essential for personalized guidance.

# Conclusion

Open reduction with iliac osteotomy and zigzag osteotomy combined with fibula grafting for developmental dysplasia of the hip has been shown to be safe and effective. The acetabular index improved from 42.95° preoperatively to 17.26° at the latest follow-up. Satisfactory results (excellent and good) were achieved in 141 hips (77.9%). We did not use KW to fix the fibula graft at the iliac osteotomy site. The surgical technique with ZOFA minimized abductor damage with negative Trendelenburg gait; no postoperative blood transfusion was required.

Postoperative complications included: AVN in 33.7%, re-dislocation in 9.9%, coxa Magna in 3.2%, Trendelenburg gait in 2.8%, and distal femur fracture in 2 hips in 1.1%.

# **Bibliography**

- 1. Wedge JH and Wasylenko MJ. "The natural history of congenital dislocation of the hip: a critical review". *Clinical Orthopaedics and Related Research* 137 (1978): 154-162.
- 2. Wedge JH and Wasylenko MJ. "The natural history of congenital disease of the hip". *Journal of Bone and Joint Surgery British Volume* 61-B.3 (1979): 334-338.
- Weinstein SL. "Natural history of congenital hip dislocation (CDH) and hip dysplasia". *Clinical Orthopaedics and Related Research* 225 (1987): 62-76.
- Crawford AH., et al. "The fate of untreated developmental dislocation of the hip: long-term follow-up of eleven patients". Journal of Pediatric Orthopaedics 19.5 (1999): 641-644.
- Barlow TG. "Early diagnosis and treatment of congenital dislocation of the hip". *Proceedings of the Royal Society of Medicine* 56.9 (1963): 804-806.
- 6. Escribano García C., *et al.* "Developmental dysplasia of the hip: Beyond the screening. Physical exam is our pending subject". *Anales de Pediatría (English Edition)* 95.4 (2021): 240-245.

- Alhaddad A., *et al.* "An overview of developmental dysplasia of the hip and its management timing and approaches". *Cureus* 15.9 (2023): e45503.
- 8. Kolb A., *et al.* "Development of an electronic navigation system for elimination of examiner-dependent factors in the ultrasound screening for developmental dysplasia of the hip in newborns". *Scientific Reports* 10.1 (2020): 16407.
- 9. Hassebrock JD., *et al.* "Costs of open, arthroscopic and combined surgery for developmental dysplasia of the hip". *Journal of Hip Preservation Surgery* 7.3 (2020): 570-574.
- 10. Kuo KN., et al. "Classification of Legg-Calvé-Perthes disease". Journal of Pediatric Orthopaedics 31.2 (2011): S168-S173.
- 11. Catterall A. "Legg-Calve'-Perthes disease". Churchill Livingstone, New York (1982): 8-33, 81-109.
- 12. Hosalkar H., et al. "Triple innominate osteotomy for Legg- Calve'-Perthes disease in children: does the lateral coverage change with time?" *Clinical Orthopaedics and Related Research* 470.9 (2012): 2402-2410.
- 13. Green NE., *et al.* "Epiphyseal extrusion as a prognostic index in Legg-Calve'-Perthes disease". *Journal of Bone and Joint Surgery American Volume* 63.6 (1981): 900-905.
- 14. Wiberg G. "Shelf operation in congenital dysplasia of the acetabulum and in sublaxation and dislocation of the hip". *Journal of Bone and Joint Surgery American Volume* 35.1 (1953): 65-80.
- 15. Ernest YY., et al. "Femoral head asymmetry and coxa magna: Anatomic study". Journal of Pediatric Orthopaedics 34.4 (2014): 415-420.
- 16. Stulberg S D., *et al.* "The natural history of Legg-Calve-Perthes disease". *Journal of Bone and Joint Surgery American Volume* 63.7 (1981): 1095-1108.
- 17. Catterall A. "The natural history of Perthes' disease". Journal of Bone and Joint Surgery British Volume 53.1 (1971): 37-53.
- 18. Christensen F., *et al.* "The Catterall classification of Perthes' disease: an assessment of reliability". *Journal of Bone and Joint Surgery British Volume* 68.4 (1986): 614-615.
- 19. Mose K. "Legg-Caluk-Perthes' disease". Universitets Forlaget, Aarhus. (1964).
- Edgren W. "Coxa plana. A clinical and radiological investigation with particular reference to the importance of the metaphyseal changes for the final shape of the proximal part of the femur". Acta Orthopaedica Scandinavica 84 (1965): 1-129.
- Weinstein SL. "Natural history of congenital hip dislocation (CDH) and hip dysplasia". *Clinical Orthopaedics and Related Research* 225 (1987): 62-76.
- Holman J., et al. "Long term followup of open reduction surgery for developmental dislocation of the hip". Journal of Pediatric Orthopaedics 32.2 (2012): 121-124.
- Chmielewski J and Albiñana J. "Failures of open reduction in developmental dislocation of the hip". *Journal of Pediatric Orthopaedics* 11.4 (2002): 284-289.
- 24. Bolland BJ., *et al.* "Late reduction in congenital dislocation of the hip and the need for secondary surgery: Radiologic predictors and confounding variables". *Journal of Pediatric Orthopaedics* 30.7 (2010): 676-682.
- 25. Chin MS., *et al.* "Comparison of hip reduction using magnetic resonance imaging or computed tomography in hip dysplasia". *Journal of Pediatric Orthopaedics* 31.5 (2011): 525-529.

- 26. Harris NH. "Acetabular growth potential in congenital dislocation of the hip and some factors upon which it may depend". *Clinical Orthopaedics and Related Research* 119 (1976): 99-106.
- 27. Segal LS., *et al.* "The contribution of the ossific nucleus to the structural stiffness of the capital femoral epiphysis: A porcine model for DDH". *Journal of Pediatric Orthopaedics* 19.4 (1999): 433-437.
- 28. Clarke NM., *et al.* "The surgical treatment of established CDH: Result of surgery after planned delayed intervention following the appearance of capital femoral ossific nucleus". *Journal of Pediatric Orthopaedics* 25.4 (2005): 43-49.
- 29. Cooke SJ., *et al.* "Ossification of the femoral head at closed reduction for developmental dysplasia of the hip and its influence on the long term outcome". *Journal of Pediatric Orthopaedics B* 19.1 (2010): 22-26.
- 30. Olney BW and Asher MA. "Combined innominate and femoral osteotomy for the treatment of severe Legg-Calve'-Perthes disease". *Journal of Pediatric Orthopaedics* 5.6 (1985): 645-651.
- 31. Severin E. "Contribution to knowledge of congenital dislocation of hip joint. Late results of closed reduction and arthrographic studies of recent cases". *Acta Chirurgica Scandinavica* 84.63 (1941): 1-142.
- 32. Kalamchi A and MacEwen GD. "Avascular necrosis following treatment of congenital dislocation of the hip". *Journal of Bone and Joint Surgery American Volume* 62.6 (1980): 876-888.
- McKay DW. "A comparison of the innominate and the pericapsular osteotomy in the treatment of congenital dislocation of the hip". *Clinical Orthopaedics and Related Research* 98 (1974): 124-132.
- Bucholz RW and Ogden JA. "Patterns of ischemic necrosis of the proximal femur in nonoperatively treated congenital hip disease". St. Louis MO: CV Mosby Co. (1978): 43-63.
- 35. McKay DW. "A comparison of the innominate and the pericapsular osteotomy in the treatment of congenital dislocation of the hip". *Clinical Orthopaedics and Related Research* 98 (1974): 124-132.
- 36. Howorth B. "A lifetime experience with congenital displacement of the hip". Orthopedic Reviews 11 (1982): 33-45.
- Dunn PM. "Perinatal observations on the etiology of congenital dislocation of the hip". *Clinical Orthopaedics and Related Research* 119 (1976): 11-27.
- 38. Westin GW., *et al.* "Total avascular necrosis of the capital femoral epiphysis in congenital dislocated hips". *Clinical Orthopaedics and Related Research* 119 (1976): 93-97.
- 39. Chung SMK. "Hip disorders in infants and children". Philadelphia: Lea and Febiger (1981).
- 40. Mose K. "Legg-Calvé-Perthes disease [Thesis]". Aarhus, Denmark: University of Aarhus (1964).
- 41. Gower WE and Johnston RC. "Legg-Perthes disease. Long-term follow-up of thirty-six patients". *Journal of Bone and Joint Surgery American Volume* 53.4 (1971): 759-768.
- De Valderrama JA. "The "observation hip" syndrome and its late sequelae". *Journal of Bone and Joint Surgery British Volume* 45 (1963): 462-470.
- Nachemson A and Scheller S. "A clinical and radiological follow-study of transient synovitis of the hip". Acta Orthopaedica Scandinavica 40.4 (1969): 479-500.

- 44. Stulberg SD., *et al.* "The natural history of Legg-Calve-Perthes disease". *Journal of Bone and Joint Surgery American Volume* 63.7 (1981): 1095-1108.
- 45. McAndrew MP and Weinstein SL. "A long-term follow-up of Legg-Calve-Perthes disease". *Journal of Bone and Joint Surgery American Volume* 66.6 (1984): 860-869.
- 46. Gamble JG., *et al.* "Coxa magna following surgical treatment of congenital hip dislocation". *Journal of Pediatric Orthopaedics* 5.5 (1985): 528-533.
- 47. Kallio PE. "Coxa magna following transient synovitis of the hip". Clinical Orthopaedics and Related Research 228 (1988): 49-56.
- 48. Leitch JM., *et al.* "Growth disturbance in Legg-Calve-Perthes disease and the consequences of surgical treatment". *Clinical Orthopaedics and Related Research* 262 (1991): 178-184.
- 49. Matani J., *et al.* "Coxa magna after open reduction for developmental dislocation of the hip". *Journal of Pediatric Orthopaedics* 15.3 (1995): 337-341.
- 50. Shigeno Y and Evans GA. "Quantitative correlation between the initial and final femoral head deformity in Perthes' disease". *Journal of Pediatric Orthopaedics B* 5.1 (1996): 48-54.
- 51. Luhmann SJ., *et al.* "The prognostic importance of the ossific nucleus in the treatment of congenital dysplasia of the hip". *Journal of Bone and Joint Surgery American Volume* 80.12 (1998): 1719-1727.
- 52. Rowe SM., *et al.* "The correlation between coxa magna and final outcome in Legg-Calve-Perthes disease". *Journal of Pediatric Orthopaedics* 25.1 (2005): 22-27.
- 53. Larson AN., *et al.* "A prospective multicenter study of Legg-Calve'-Perthes disease: functional and radiographic outcomes of nonoperative treatment at a mean follow-up of twenty years". *Journal of Bone and Joint Surgery American Volume* 94.7 (2012): 584-592.
- 54. Nguyen Ngoc Hung., *et al.* "Long-term result after operative hung zigzag osteotomy combined fibular allograft for developmental dysplasia of the hip, and coxa magna in children". *EC Paediatrics* 13.1 (2024): 01-27.
- 55. Ganz R., *et al.* "Extended retinacular soft tissue flap for intraarticular hip surgery: surgical technique, indications, and results of its application". *Instructional Course Lectures* 58 (2009): 241-255.
- 56. Ganz R., *et al.* "Algorithm for combined femoral and periacetabular osteotomies in complex hip deformities". *Clinical Orthopaedics and Related Research* 468.12 (2010): 3168-3180.
- 57. Sakamaki T. "Clinical study on coxa magna during the treatment in congenital dislocation of the hip". *Journal of the Japanese Orthopaedic Association* 53.5 (1979): 491-504.
- 58. Hasegawa A., et al. "Coxa magna". Hokkaido Journal of Orthopedic and Traumatic Surgery 21 (1976): 1-5.

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