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Developmental dysplasia of hip

(DDH)

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ABSTRACT

The term **"Developmental dysplasia of hip (DDH)"** refers to a spectrum of anatomical abnormalities of the hip joint in which the femoral head has an abnormal relationship with the acetabulum. The majority of studies report an incidence of 1 to 34 cases per 1,000 live births and differences could be due to different diagnostic methods and timing of evaluation.

Risk factors include first born status, female sex, breech presentation and oligohydramnios, positive family history.

Clinical manifestations of DDH vary depending on the age of the child. Newborns present with hip instability, on examination, infants have limited hip abduction, and older children and adolescents present with significant pain and dysfunction and is a leading cause of progressive hip osteoarthritis in affected patients.

To prevent late cases it`s important to repeat, careful examination of all infants from birth and throughout the first year of life until the child begins walking

Provocative testing includes the Barlow and Ortolani maneuvers. Other signs, such as femur shorting with hips and knees flexed (Galeazzi sign), asymmetry of the thigh or gluteal folds, and discrepancy of leg lengths are potential clues.

Plain radiographs are essential for making proper diagnosis, whereas 3D imaging such as MRI and/or CT detects intra-articular pathology and provides better characterization of hip morphology.

Treatment depends on age at presentation and results are much better when the child is treated early, particularly within the first six months of life.

Management of early, symptomatic DDH includes nonsurgical modalities and open joint preservation techniques. Arthroscopic management can be used as an adjunct for symptomatic treatment and for addressing intra-articular pathology,

but it alone does not correct the underlying osseous dysplasia and associated instability. The per acetabular osteotomy has become the mainstay of efforts to redirect the acetabulum and preserve the articular integrity of the hip; however, the proximal femur is also a potential source of pathology that should be considered. Open hip procedures are technically demanding yet provide the opportunity for pain relief, improved function, and preservation of the hip joint.

INTRODUCTION

The term ‘developmental dysplasia of the hip’ (DDH) includes a wide spectrum of hip alterations: neonatal **instability**; **acetabular dysplasia**; **hip subluxation**; and **true dislocation of the hip**[1][2][3].

Normal hip development involves a close association and congruency between the growing femoral head and the acetabulum in utero.

instability refers to acetabulum`s looseness or laxity [4].

In the case of **dysplasia**, some morphological changes in the acetabulum, proximal femur or both are present, but articular surfaces are concentrically in contact and the Acetabular dysplasia could be the initial abnormality or it could be secondary to in utero hip subluxation[5].In the subluxated hip, there is ***contact*** between both articular surfaces, but not concentrically. In a **true dislocation**, there is ***no contact*** between the articular surfaces of the proximal femur and acetabulum[5].it is essential to differentiate between these entities, because its clinical course, treatment and prognosis are also different. When facing a child with DDH, it is necessary to determine whether the hip is **concentrically reduced**. The popular terms like ‘congenital dislocation of the hip’ or ‘congenital dysplasia of the hip’ are less commonly used these days because they do not include the developmental aspect of the dysplasia, which is important from a medico-legal point of view[6].

Physiologic, mechanical, and genetic factors are implicated in DDH.

Physiologically, maternal hormones near delivery cause a temporary laxity of the hip joint.

Mechanically, in late gestation constant compression in utero with spatial restriction of movements is accentuated, with first pregnancy, breech presentation and oligohydramnios. Other conditions associated with in utero constraint, such as sternocleidomastoid torticollis (fibromatosis colli); foot deformities (ie, metatarsus adductus, clubfoot); and molding deformity of the skull, have an increased association with DDH

Genetically, DDH is more likely in people who have a strong family history and among Certain ethnic populations[7][8][9].

ANATOMY

The hip is formed by the acetabulum, the proximal femur and soft tissues joining them (capsule, teres ligament, transverse ligament and pulvinar). In the growing child, the acetabulum is a complex structure. It is formed by the joined pubis, isquion and ilion. This junction is called triradiate cartilage, which is responsible for acetabular development and growth. The external surface of the acetabulum is covered by a horseshoe-shaped articular cartilage. The transverse ligament joins both extremes of the articular cartilage inferiorly. Pulvinar fibroadipous tissue and teres ligament are at the floor of the external surface of the acetabulum. The labrum is attached to the acetabulum`s peripheral edge and plays an important role in maintaining hip stability [3].

Acetabulum and femoral head development are intimately related. Development of the acetabular cavity is influences by the presence of a concentrically reduced

femoral head[10].when the femoral head is not in contact with the acetabulum, the latter does not fully develop and it is flat-shaped[11].

At birth, the proximal femur is completely cartilaginous. The cephalic nucleus begins to ossify around the age of **six months** while the trochanteric nucleus starts to ossify **at five to six years**. Femoral ante version and cervico-diaphyseal angle decrease with age.

Anatomical changes in the dysplastic hip

With time and growth, several adaptive changes affect all the structures of the hip. A concentrically in-contact femoral head is required for acetabular cavity development. If the femoral head is not reduced, the acetabulum cavity flattens and the osseous wall widens.

Pulvinar fat, teres ligament, labrum, transverse ligament and capsule are hypertrophied. The hypertrophic labrum is the also known as limbus and can be everted (most frequently) or inverted (preventing hip reduction). The limbus should be differentiated from the neolimbus. The neolimbus is a crest of hypertrophic acetabular cartilage caused by the overloading of the subluxated femoral head against the acetabulum`s posterosuperior part. The neolimbus divides the articular cavity in two zones: the primary acetabulum is on the medial side, and the secondary acetabulum is on the lateral side. The neolimbus disappears when the hip is reduced[1].

Although acetabular anteversion has traditionally been thought to be increased in hip dysplasia,[12] other studies have found no differences in acetabular anteversion between affected and unaffected sides [13].

The proximal femur undergoes many changes as well. The dysplastic femur has a short neck and increased valgus and anteversion[14].However, some studies have found no variations in femoral anteversion between the affected and unaffected sides. In contrast to the contralateral side, the femoral head is deformed, and the ossification nucleus apparition is delayed. The medullar canal is narrow and straight[14].

EPIDEMIOLOGY

The overall prevalence of DDH is usually reported as approximately 1 case per 1000 individuals, though Barlow estimated that the incidence of hip instability during newborn examinations was as high as 1 case per 60 newborns [15].

According to Barlow's study, more than 60% of newborns with hip instability stabilized by age 1 week, and 88% became stable by age 2 months, leaving only 12% (of the 1 in 60 newborns, or 0.2% overall) with residual hip instability

Incidence

- most common orthopedic disorder in newborns
- dysplasia is 1:100
- Dislocation is 1:1000 [16].

Demographics

- more common in females (6:1)
- more commonly seen in Native Americans and Laplanders
    - due to cultural traditions such as swaddling with hips together in extension
    - rarely seen in African Americans

Location

- Most common in left hip (60%)
    - due to the most common intrauterine position being left occiput anterior (left hip is adducted against the mother's lumbosacral spine)

- bilateral in 20%

RISK FACTORS

First born

The uterus is compressed due to an unstretched uterus and rigid abdominal structures.

Female

Because of increased ligamentous laxity that transiently exists as the result of circulating maternal hormones and the estrogens produced by the fetal uterus

Breech

More commonly seen in female children, firstborn children, and pregnancies complicated by oligohydramnios

Higher risk of DDH with frank/single breech position compared to footling breech position

Family history

Oligohydramnios

Macrosomia

Limited hip abduction, Tulips, Swaddling.

ETIOLOGY

The etiology of DDH is multi factorial.

Initial instability thought to be caused by maternal and fetal laxity, genetic laxity, and intrauterine and postnatal malpositioning

Pathoanatomy

- initial instability leads to dysplasia
    - typical deficiency is anterior or anterolateral acetabulum
        - acetabular deficiency is posterosuperior in spastic cerebral palsy

- dysplasia leads to subluxation and gradual dislocation
    - Subluixation of the femoral head repeatedly results in the development of the limbus, a thickened articular cartilage ridge.
- chronic dislocation leads to
    - development of secondary barriers to reduction
        - ✓ pulvinar thickens
        - ✓ ligamentum teres thickens and elongates
        - ✓ transverse acetabular ligament hypertrophies
        - ✓ hip capsule and iliopsoas form hourgass configuration

- anatomic changes
    - increased femoral anteversion
    - flattening of the femoral head
    - increased acetabular anteversion
    - increased obliquity and decreased concavity of the acetabular roof
    - thickening of the medial acetabular wall

- Associated conditions

Associated with "packaging" deformities which include

  - Congenital muscular torticollis (20%)
  - Metatarsus adductus (10%)
  - congenital knee dislocation

Conditions characterized by increased amounts of type III collagen

CLASSIFICATION

Can be classified as a spectrum of disease involvement (phases)

Subsumable

Barlow-suggestive

Dislocatable

Barlow-positive

Dislocated

Portolan-positive early when reducible; Ortolani-negative late when irreducible [16].

SIGNS & SYMPTOMS

Since developmental dysplasia of the hip doesn't cause pain in babies, it can be difficult to notice. To look for signs of DDH, check the hips of all newborns and babies during well-child exams.

Parents could notice:

- The hips of the baby make a popping or clicking that is heard or felt.
- The legs of the baby are not the same length.
- One hip or leg doesn't move the same as the other side.
- The skin folds under the buttocks or on the thighs don't line up.
- The child has a limp when starting to walk.

Babies who exhibit any of these signs should see a doctor to have their hips checked. Finding and treating DDH early usually means there's a greater chance for a baby's hips to develop normally [17].

DIAGNOSIS

Most countries have routine newborn exams that include a hip joint exam screening for early detection of hip dysplasia. A "click" or more precisely "clunk" in the hip may be detected during an exam (although not all clicks indicate hip dysplasia) [18][19][20]. The child's hips are tracked with additional screening when a hip click (also known as "clicky hips" in the UK) is detected to determine if developmental dysplasia of the hip is caused [21] [22].

For physical examination, the child should be completely relaxed, on a smooth, warm, comfortable surface in a quiet environment.

Two maneuvers commonly employed for diagnosis in neonatal exams are the **Ortolani maneuver** and **the Barlow maneuver**[23][24][25].

In the Ortolani test, the baby should be supine with hips flexed to 90 degrees. The examiner’s index and long fingers are placed laterally over the child’s greater trochanter with the thumb positioned medially near the groin crease. In which the contralateral hip is held still while the thigh of the hip being tested is **abducted and gently pulled anteriorly**. If a "clunk" is heard (the sound of the femoral head moving over the acetabulum), the joint is normal, and that is

positive ortolani test but absence of the "clunk" sound indicates that the acetabulum is not fully developed.

For Barlow's, test the pelvis is stabilized and the patient is positioned similar to the Ortolani test position. The Barlow's test detects potential posterior subluxation or dislocation by **adducting the hip while pushing the thigh posteriorly**. The hip goes out of the socket it means it is dislocated, and the newborn has a congenital hip dislocation. After three to six months, soft tissue contractures limit motion of the hip even if it is dislocated[26].

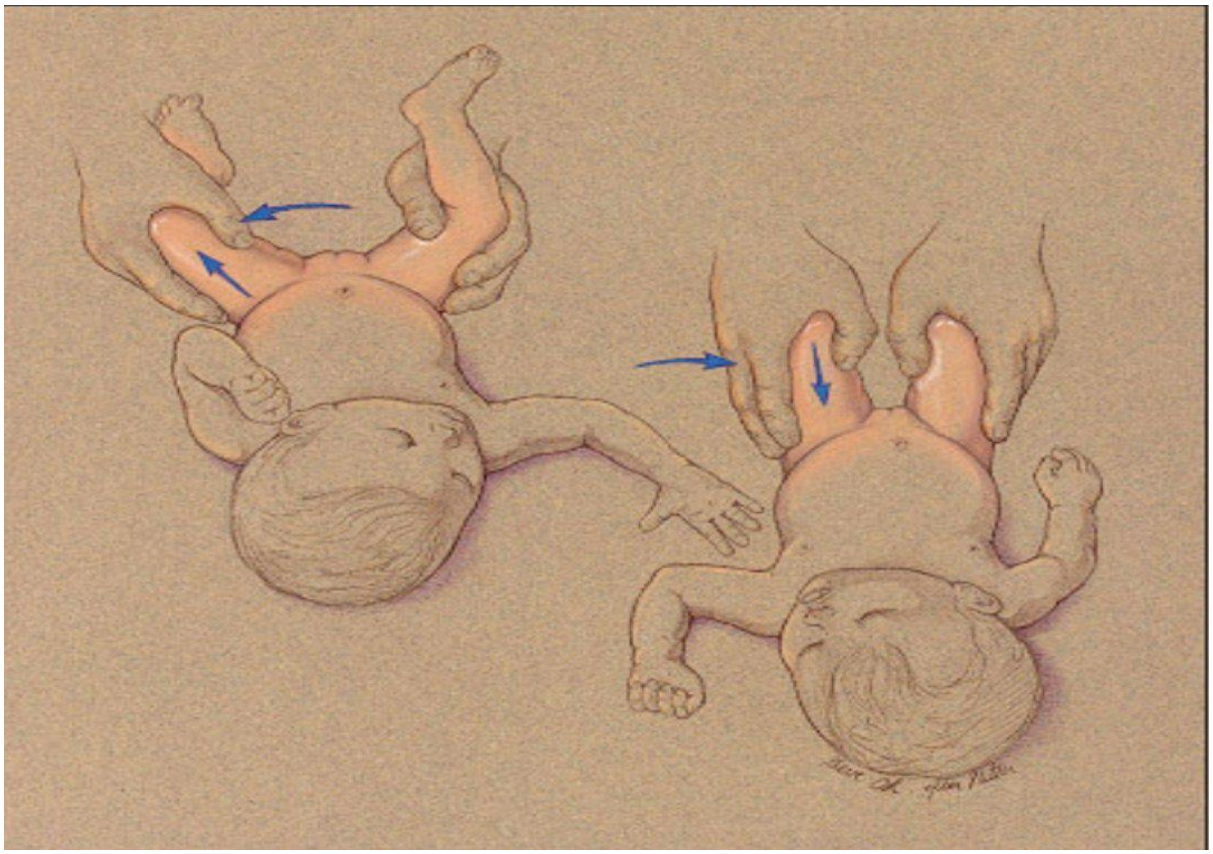


Fig. 1.Maneuvers used during the physical examination to assess the hips for dysplasia. (Left) Ortolani maneuver. (Right) Barlow maneuver. See text for explanation of the maneuvers.

Examination of an older child also needs careful assessment of extremities for asymmetric skin folds or leg length discrepancy in case of unilateral hip dislocation. Another indicator of hip dislocation is a positive Galeazzi sign. It is elicited by laying the child supine and flexing both hips and knees. A positive sign is indicated by a difference in the height of the knees. When the Galeazzi sign would be negative, Limited abduction may be particularly helpful in diagnosing children with bilateral hip dislocation. Maximal abduction of hips

Should be greater than 60°.

In neglected cases, DDH may be diagnosed when children approach walking age with a limp on the affected side (positive Trendelen - berg's sign) and hyperlordosis.

If a baby has signs of DDH or has a higher risk for it, the doctor will order tests.

Two tests help doctors check for DDH:

An ultrasound uses sound waves to make pictures of the baby's hip joint. This works best with babies under the age of 6 months. That's because most of a baby's hip joint is still soft cartilage, which won't show up on an X-ray.

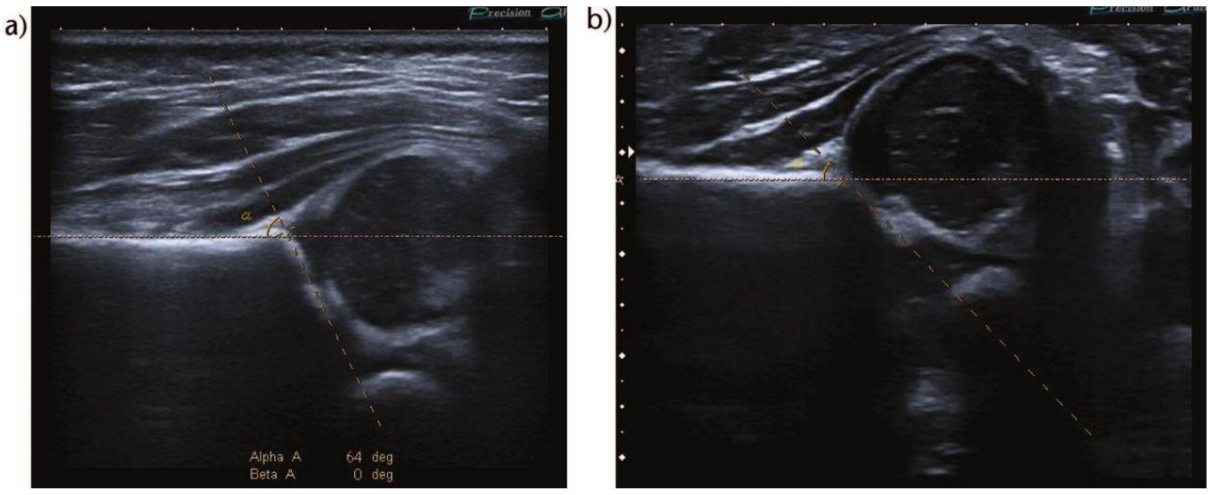


Fig. 2. (a) Normal sonography. (b) In contrast with (a), the angle is <60° and the hip is subluxated.

An X-ray is most effective in babies older than 4–6 months. At that age, their bones have formed enough to see them on an X-ray.

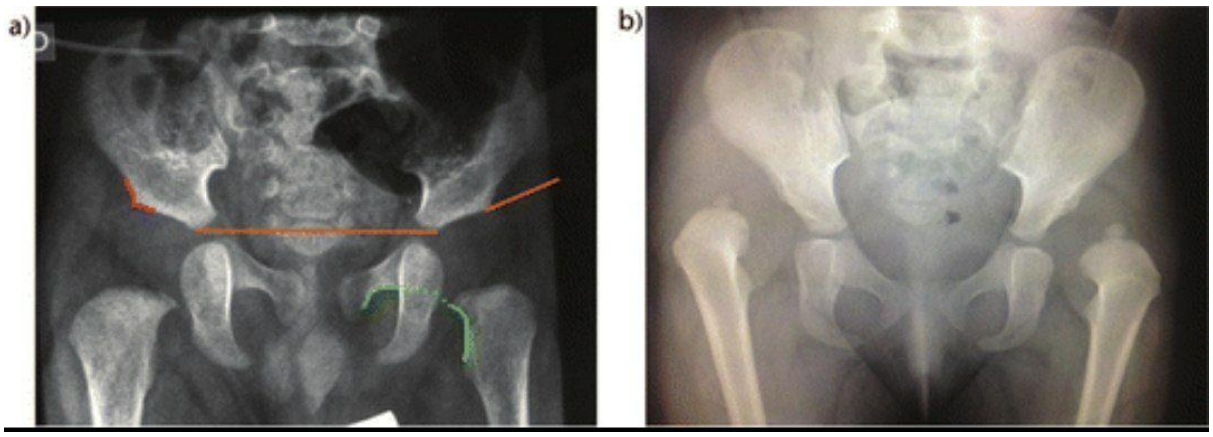


Fig. 3. (a) Radiograph studies are the reference in children older than 4 to 6 months of age. In the image, although the ossification nucleus of the head is absent, indirect signs of concentric reduction as the formed talus (red line) and Shenton line (green lines) are present. The acetabular index (orange line) is the main parameter to control acetabular development during the first years of age. (b) Bilateral dislocation of a two-year-old child.

TREATMENT

NON-operative

Splints

A special brace can be used to successfully treat babies with DDH. This holds the hip joint in the correct position, allowing it to develop properly. The patient may need to wear the brace for several months, until the hip is stable [27].

Indications

- < 6 months old and reducible hip

Contraindicated in teratologic hip dislocations and patients with spina bifida or spasticity

- Requires normal muscle function for successful outcomes [16].

Closed reduction procedure

If splinting fails, child may need a procedure called a closed reduction. Closed reduction means the hip joint is repaired without surgery. When the child is asleep under anaesthetic, the hip joint is moved into the correct position [27].

Indications

- 6-18 months old
- Failure of Pavlik treatment [16].

Operative

Sometimes, when the above treatments fails or DDH is diagnosed later than six months of age, the child may need open reduction surgery (when surgery is done through a cut in the body).

For DDH open reduction surgery, sleep the child under anaesthetic then move the hip joint into the correct position. The hip joint is made more stable by operating on the surrounding ligaments. This is all done through a small cut near the groin [27].

Open reduction and spica casting

Indications

- 18 months old
- Failure of closed reduction
- Open reduction and femoral osteotomy [16].

Open reduction and femoral osteotomy

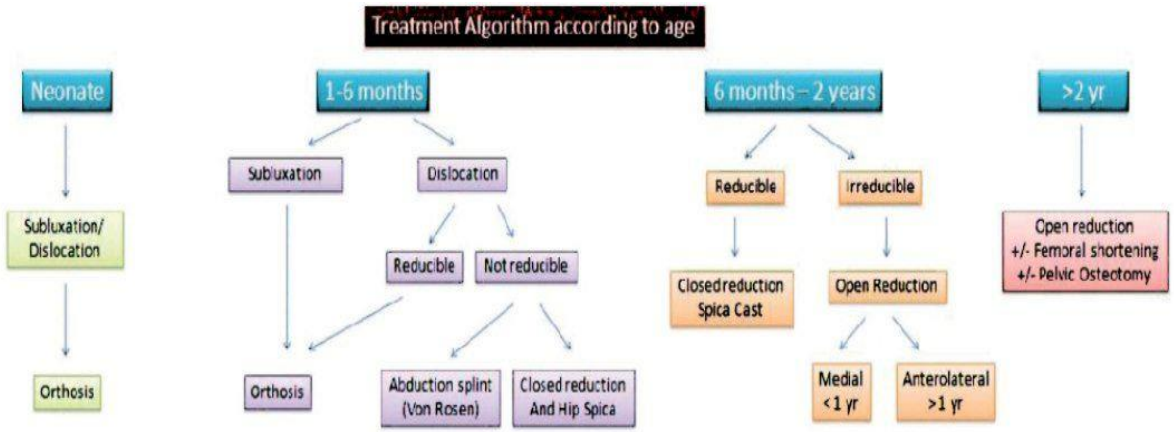
Indications

- 2 years old with residual hip dysplasia
- anatomic changes on femoral side (e.g., femoral anteversion, coxa valga)
- best in younger children (< 4 years old)
    - After 4 years old, pelvic osteotomies are utilized [16].

Open reduction and pelvic osteotomy

Indications

- 2 years old with residual hip dysplasia
- severe dysplasia accompanied by significant radiographic changes on the acetabular side (increased acetabular index)
- Used more commonly in older children (> 4 yr.)
    - Decreased potential for acetabular remodeling as child ages [16].



COMPLICATIONS

Redislocation, stiffness of the hip, infection, blood loss, and, possibly the most devastating, avascular necrosis of the femoral head, all of these are a complications that can occur. The rate of femoral head necrosis varies substantially; depending on the study, it may be range from 0% to 73%. Numerous studies show that extreme abduction, especially when combined with extension and internal rotation, results in a higher rate of avascular necrosis [15].

PROGNOSIS

Overall, children with hip dysplasia have very good prognosis, especially if the dysplasia is managed with closed treatment. If closed treatment is fails and open reduction is needed, the outcome may be less favorable, although the short-term outcome appears to be satisfactory. If secondary procedures are needed to obtain reduction, then the overall outcome is significantly worse. Some authors believe that patients with bilateral hip dysplasia have a poorer prognosis due to repeated diagnostic delays and greater treatment requirements. The radiographic outcomes were similar in a study comparing the outcomes of walking-age children with bilateral hip dislocations who underwent open reduction and pelvic osteotomy with or without femoral osteotomy with those of walking-age children with unilateral dislocated hips who underwent the same set of procedures. The rate of osteonecrosis was higher in the bilateral group in this study, but this difference was explained by older age at surgery and a greater degree of hip dislocation prior to surgery. The authors concluded that the clinical outcomes after surgery of the children with bilateral hip dislocations were worse mainly because of asymmetric outcomes [15].

CONCLUSIONS

Developmental dysplasia of the hip is a challenging condition. Formal training in the treatment of various age groups with DDH is mandatory.

The higher the age at presentation, the worse the outcomes after intervention for DDH. Therefore, the most significant factor affecting the outcome is early diagnosis. Instability maneuvers and hip abduction assessment should be done universally as a part of the physical examination of the newborn. Although they are classically referred to as isolated ‘clicks’ or inguinal fold asymmetry but have no clinical importance. When the neonatal physical examination is normal and the baby is breech or has a positive family history of DDH, there is a strong consensus to perform a hip ultrasound. Controversy remains around other risk factors that would indicate the necessity of a hip ultrasound. Universal ultrasound screening has not been shown to reduce the incidence of late dysplasia and, on the other hand, increases the rate of overtreatment. Radiographic evaluation is the main imaging study to assess the growth and development of the hip from four to six months of life until maturity.

The treatment within the first six months of life has a much better outcome than in late-diagnosed DDH.

Successful treatment with orthoses necessarily requires careful counseling of parents. Surgical management requires careful pre-operative planning and adequate follow up to ensure the best clinical outcomes.

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