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Developmental dysplasia of the hip: update of management

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- 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.
- DDH alters hip biomechanics, overloading the articular cartilage and leading to early osteoarthritis. DDH is the main cause of total hip replacement in young people (about 21% to 29%).
- Development of the acetabular cavity is determined by the presence of a concentrically reduced femoral head. Hip subluxation or dislocation in a child will cause an inadequate development of the acetabulum during the remaining growth.
- Clinical screening (instability manoeuvres) should be done universally as a part of the physical examination of the newborn. After two or three months of life, limited hip abduction is the most important clinical sign.
- Selective ultrasound screening should be performed in any child with abnormal physical examination or in those with high-risk factors (breech presentation and positive family history). Universal ultrasound screening has not demonstrated its utility in diminishing the incidence of late dysplasia.
- Almost 90% of patients with mild hip instability at birth are resolved spontaneously within the first eight weeks and 96% of pathologic changes observed in echography are resolved spontaneously within the first six weeks of life. However, an Ortolani-positive hip requires immediate treatment.
- When the hip is dislocated or subluxated, a concentric and stable reduction without forceful abduction needs to be obtained by closed or open means. Pavlik harness is usually the first line of treatment under the age of six months.
- Hip arthrogram is useful for guiding the decision of performing a closed or open reduction when needed.
- Acetabular dysplasia improves in the majority due to the stimulus provoked by hip reduction. The best parameter to predict persistent acetabular dysplasia at maturity is the evolution of the acetabular index.
- Pelvic or femoral osteotomies should be performed when residual acetabular dysplasia is present or in older children when a spontaneous correction after hip reduction is not expected.
- Avascular necrosis is the most serious complication and is related to: an excessive abduction of the hip; a force closed reduction when obstacles for reduction are present; a maintained dislocated hip within the harness or spica cast; and a surgical open reduction.

Keywords: DDH; hip dysplasia; congenital dislocation of the hip

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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.[1–3] Looseness or laxity within the acetabulum is called instability.[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.[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 important to differentiate between these entities, because its clinical course, treatment and prognosis are also different. When facing a child with DDH, it is very important to determine whether the hip is concentrically reduced. Classical terms such as 'congenital dislocation of the hip' or 'congenital dysplasia of the hip' are used less

often these days because they do not include the developmental aspect of the dysplasia, which is important from a medico-legal point of view.[6]

Anatomy

The hip is formed by the acetabulum, the proximal femur and soft tissues joining them (capsule, teres ligament, transverse ligament and pulvinar). The acetabulum is a complex structure in the growing child. It is formed by the joined pubis, isquion and ilion. This junction is called triradiate cartilage, which is responsible for acetabular 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 peripheral edge of the acetabulum and plays an important role in maintaining hip stability.[3]

Acetabulum and femoral head development are intimately related. Development of the acetabular cavity is determined by the presence of a concentrically reduced femoral head.[7] When the femoral head is not in contact with the acetabulum, the latter does not develop adequately and it is flat-shaped.[8]

The proximal femur is completely cartilaginous at birth. The cephalic nucleus of ossification appears at about six months of age while the trochanteric nucleus starts to ossify at five to six years. Femoral anteversion and cervicodiaphyseal angle decrease with age.

Anatomical changes in the dysplastic hip

With time and growth, several adaptive changes affect all the structures of the hip. Acetabular cavity development needs a concentrically in-contact femoral head. 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 so-called 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 overload of the subluxated femoral head against the posterosuperior part of the acetabulum. The neolimbus divides the articular cavity in two zones: the medial part is the so-called primary acetabulum and the lateral part is the secondary acetabulum.[8] The neolimbus disappears when the hip is reduced.[1]

Regarding orientation, while classically acetabular anteversion was thought to be increased in hip dysplasia,[9] other studies do not detect differences in acetabular anteversion between affected and unaffected sides.[10]

Several changes also occur in the proximal femur. The dysplastic femur has increased valgus and anteversion, and a short neck.[11] However, some studies do not report differences in femoral anteversion in comparison with the unaffected side.[9] The femoral head is deformed and the ossification nucleus apparition is retarded in comparison with the contralateral side. The medullar canal is narrow and straight.[11]

Ethiology and pathogenesis

Adequate growth and development of the hip depends on two main (and necessary) factors: concentric positioning of femoral head into the acetabular cavity and adequate balance in growth between triradiate and acetabular cartilage.[8,12,13] Any alteration in these two conditions leads to a hip dysplasia. As stated by Dunn et al,[14] based on the findings that there was no incidence of hip dysplasia among fetuses aborted below 20 weeks of gestation, it seems that most of the changes that lead to a DDH appear in the last months of intrauterine life.

Diverse theories and risk factors have been proposed as the origin of DDH. Hormonal theory is based on a misbalance between oestrogens and progesterone. It has been demonstrated experimentally that oestrogens are protective against dislocation while an environment with higher concentrations of progesterone can facilitate dislocation.[15] However, no relationship between DDH and serum concentration of beta-estradiol and relaxine has been demonstrated.[16–18] More important than hormone environment, however, seems to be gender.[19]

Mechanical theory sustains that persistent mechanical stimulation can provoke a deformity, especially in periods of high growth. The human fetus accomplishes these criteria, because of its plasticity and rapid rate of growth. All the circumstances where the fetus is exposed to increased deforming forces are suitable for producing a DDH. Oligoamnios, macrosomy or breech presentation with extended knees are some of the risk factors based on the mechanical theory (Table 1).[14,15,20] The maintained posture with forced hyperflexion of the hip and knee extension associated with breech presentation could lead to hip dysplasia and dislocation.[20,21] The left side is more commonly affected, because most of non-breech newborns have this hip against the mother's spine, limiting abduction of that hip.[6]

The normal newborn has hip and knee flexion contractures, which resolve spontaneously within the first weeks after birth. Cultures that carry children with the lower extremities wrapped tightly together have a higher risk of hip dysplasia compared with those that carry the baby in a jockey position.[4]

Familial predisposition has been well documented in the literature. First-grade familiars have an increased risk

Table 1. Risk factors for developmental dysplasia of the hip

- Breech presentation
- Familiar history
- Female
- Oligohydramnios
- Elevated weight at birth
- Multiple pregnancy
- Left hip
- Hyperlaxity
- Clubfoot

of 12-fold of developing a DDH while relative risk is only 1.7 in the second grade.[22] A higher incidence of hip osteoarthritis (OA) and implantation of total hip arthroplasty in the parents and grandparents of patients diagnosed with DDH in comparison with the general population has also been reported.[23] Some alterations in genes such as *CX3CR1* have been observed in cases of familial aggregation of DDH.[24,25] Genetic characterization and screening will probably be developed in next years.

Some conditions of the neonate have been associated with DDH. Torticollis is one of them and DDH should be discarded in all newborns with congenital torticollis.[14] Other conditions classically associated with DDH are clubfoot and metatarsus varus; however, data supporting this association are controversial.[26–29]

In a meta-analysis leaded by Hundt et al,[30] only familial aggregation, breech presentation, females and clicking hips in exploration demonstrated an increased risk for DDH. The most important risk factors associated with an altered echography in the newborn are breech presentation, familial aggregation, female infants and hip instability,[31] but even they are moderately supported by the literature.[32]

Despite the abovementioned factors, most patients with DDH and most of those patients that need treatment do not present any risk factors except for the female sex (80% of cases).[31]

Natural history

A prevalence of hip instability of 1% to 1.5% in newborns has been reported along with an incidence of 5 per 1000 in males and 13 per 1000 in females.[33] However, almost 90% of patients with mild instability at birth are resolved spontaneously within the first eight weeks of life.[34]

A small amount of cases will not correct spontaneously, and instability and/or echographic alterations will persist. These cases represent so-called persistent dysplasia. Persistent dysplasia alters hip biomechanics, overloading the articular cartilage. This overload can wear articular cartilage, leading to early OA. Avoiding OA development is the main goal of the treatment for DDH. It is estimated that DDH represents 2.6% to 9.1% of total cases of total hip replacement (THR) and is the main cause of THR in young people (about 21% to 29%).[35,36] The relative risk for a THR is 2.6 when instability exists at birth in comparison with a normal hip.[36] The costs associated with THR in DDH are higher in comparison with those in THR for primary OA.[37]

The natural history of acetabular dysplasia without dislocation or subluxation is not well-known because it is commonly underdiagnosed.[1] However, its importance on OA development has been well documented.[36] On the other hand, subluxation of the hip invariably leads to hip OA because of the increased contact forces between the femoral head and acetabulum.[38,39] In true dislocation, the presence of a secondary acetabulum is the main risk factor for OA development. OA develops because of the wear of the femoral head against the pelvic bone. Patients without a secondary acetabulum do well, with acceptable range of motion and absence of pain usually until the fourth decade.[1]

In addition to OA development, patients with true dislocation can develop other musculoskeletal and biomechanical changes. In case of unilateral dislocation, lower limb discrepancy, unstable gait, postural scoliosis, flexo-aduction contracture of the hip and valgus deformity of ipsilateral knee occur. Bilateral dislocation provokes lumbar hyperlordosis and an altered gait.

Clinical diagnosis

Diagnosis of instability in the neonatal period can be easily assessed with the Barlow and Ortolani manoeuvres.[34,40,41] While the Barlow manoeuvre tries to dislocate the femoral head with hip adduction and posterior translation,[34] the Ortolani manoeuvre tries to relocate a dislocated femoral head with hip abduction and anterior translation.[40,41] It is important to exam the newborn's hip to rule out the presence of hip instability. Instability manoeuvres should be done universally as a part of the physical examination of the newborn. In a decision analysis model, the lowest probability of developing osteoarthritis of the hip by the age of 60 years was by performing an adequate physical examination of the hip on all newborns and performing ultrasound screening for selective cases.[42] It is important to remark that isolated 'clicks' do not have clinical importance, in comparison with positive manoeuvres of instability.[43]

Although instability is the main sign of DDH in the neonatal period, it rapidly diminishes as muscle strength increases, which occurs after the first week of life.[34] After that, abduction asymmetry is the main clinical sign.[44] Hip abduction should always be assessed. In dislocated hips, there is limited abduction when compared to the healthy side. Hip abduction in a newborn is about 80° to 90°; asymmetrical limitation of abduction must lead to the suspicion of a possible dislocated hip.[45,46] Symmetric limited abduction is not normal and can reflect a possible bilateral dislocation. In case of teratologic dislocations, instability manoeuvres will be negative while a limited abduction of

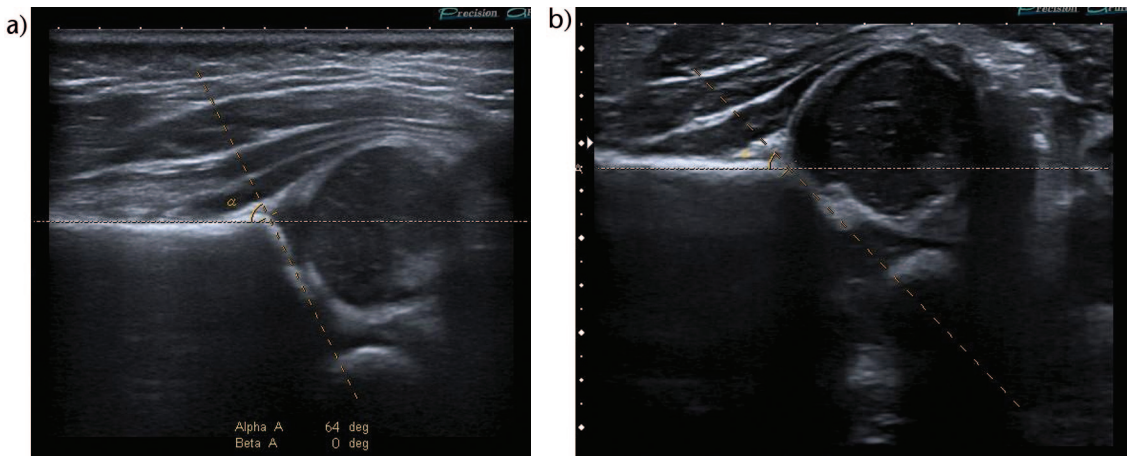

Fig. 1. (a) Normal sonography. (b) In contrast with (a), the α angle is <60° and the hip is subluxated.

the hip will be present. Limited abduction is usually accompanied by limb length discrepancy (Galeazzi sign), with shortening of the affected limb.

Inguinal fold asymmetry, although classically referred, has no true value in the diagnosis of DDH. It is present in up to 30% of cases of normal hips, while it is not present in all patients with DDH.[2] Once the child has started to walk, limping is usual. Hyperlordosis can also be present, especially in bilateral cases. On the other hand, mild dysplasia may not have any symptoms in infants.

However, clinical screening seems to be inadequate to detect all cases of hip dysplasia. It has been reported that up to 92% of patients who sustained a THR for hip dysplasia did not have any neonatal instability.[33] Some authors believe in the existence of a late appearance dysplasia that justifies this mismatch between neonatal dysplasia and THR implanted in mature patients.

Imaging studies

Hip ultrasound

Sonography is valuable in the first months of life.[47–50] Once the ossifying nucleus of the femoral head appears, ultrasound is less valuable and radiograph should be used. Graf et al[51] established a method to evaluate the infant hip according to morphology. Two angles were described: α angle, formed between the ilion and the osseous wall of the acetabulum; and β angle, formed between the ilion and the cartilaginous labrum. The higher the α angle, the more reduced is the hip. (Fig. 1a and 1b) This is a static and morphologic method. In contrast, Terjesen et al[52] proposed another method based on dynamic evaluation of the hip. For them, instability of the hip and percentage of femoral head covered are more important.

It has been recommended to perform an imaging study before the infant is six months old in all cases with high-risk factors (i.e. breech presentation, family history, documented instability, etc.).[32] Controversy remains regarding which risk factors should be considered for performing an ultrasound screening. The European Society of Paediatrics Radiology considers breech presentation and positive family history as the only risk factors that indicate the necessity of performing a hip ultrasound when the neonatal physical examination is normal.

Universal ultrasound screening has been proposed to improve the accuracy of diagnosis in the neonatal period. However, universal ultrasound screening would cause an increased burden for studying a condition that affects 1 per 1000 newborns.[53] Some studies demonstrated a diminished risk for surgical intervention in the past. However, numerous recent well-designed studies and meta-analyses have not demonstrated its utility in diminishing the incidence of late dysplasia.[32,46–49] Furthermore, Laborie et al[54] concluded that a higher rate of overtreatment exists (but not an increased rate of complications) with universal screening while no significant reduction of late dysplasia was observed in comparison with selective screening.

Radiography

Radiographic evaluation is the main method of evaluating the growth and development of the hip after four to six months of life.[43] Adequate ossification and development of the femoral head and acetabulum, as well as avascular necrosis (AVN), can be assessed with radiographs (Fig. 2a and 2b).

Several parameters have been studied to evaluate the growth and development of the hip. The acetabulum's anatomy can be assessed by the acetabular index and

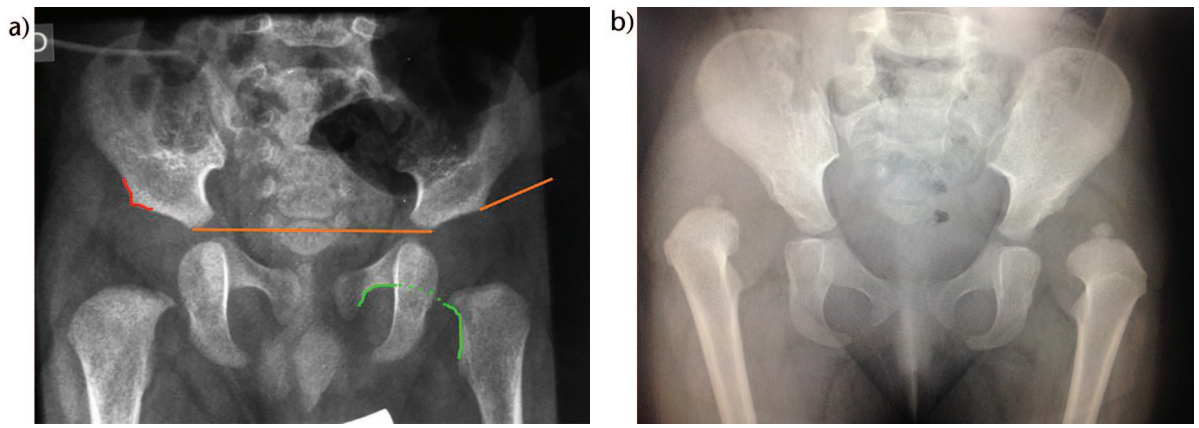

Fig. 2. (a) Radiograph studies are the reference in children older than 4 to 6 months of age. In the image, although the ossification nucleus of the head is absent, indirect signs of concentric reduction as the formed talus (red line) and Shenton line (green lines) are present. The acetabular index (orange line) is the main parameter to control acetabular development during the first years of age. (b) Bilateral dislocation of a two-year-old child.

Sharp's angle (Fig. 2a). Shenton's line evaluates the relationship between the acetabulum and the femoral head. Wiberg's centre edge angle and the percentage of the femoral head that is covered are useful to evaluate the amount of femoral head concentrically reduced.

Severin's classification takes into account acetabular dysplasia, femoral head deformity and subluxation of the hip at maturity. Severin's classification has good correlation with long-term outcomes of the hip.[55]

Treatment

The higher the age at presentation, the worse the outcomes after intervention for DDH. By the age of eight years, it is thought that possible complications of treatment may lead to a poor outcome, no better than if DDH is left untreated.[56,57]

All treatment efforts are based on obtaining a concentrically positioned femoral head into the acetabulum so the latter is stimulated to grow normally. Acetabular potential of correction diminishes dramatically after three to four years of age; therefore, early interventions are paramount to obtain the best results with less surgical aggression.

Hence, the goal of the treatment is to achieve a concentric reduction of the femoral head into the acetabulum and correct development of all structures of the hip. This can be achieved according to three main principles:

1. To achieve a concentric and stable reduction of the hip, avoiding complications such an AVN.
2. To confirm correction of acetabular dysplasia due to the stimulus provoked by the stable and concentric position of the femoral head into the acetabulum. In order to do so, serial radiographs of the hip as the child grows should be necessary. The best parameter to predict acetabular dysplasia at maturity is the evolution of acetabular index.[58]
3. Pelvic or femoral osteotomies are indicated in two situations. First, when there is a residual dysplasia that previous reduction of the hip has not been able to correct. Second, when the potential of correction after hip reduction is expected to be not enough to correct the acetabular dysplasia due to the child's age. Pelvic osteotomies are performed after the age of three to four years, when the acetabular potential of correction diminishes.[59]

A concentric and stable reduction of the hip

It has been reported that up to 96% of pathologic changes observed in echography are resolved spontaneously within the first six weeks of life.[31] Barlow-positive hips could be observed for four to six weeks waiting for spontaneous stabilization. Ortolani-positive and Barlow-positive hips that do not stabilize by themselves in four to six weeks should be treated.

If the femoral head is dislocated, hip reduction is easier within the first months of life. In a child aged under six months, the Pavlik harness is the most used orthosis for reducing a dislocated or subluxated hip and is usually the first line of treatment.[60] Pavlik observed that hip and knee flexion provokes abduction of the hip and this abduction keeps the hip reduced. The Pavlik harness should be worn to allow hip abduction between 30° (less abduction allows hip dislocation) and 60° (higher abduction increases risk of AVN). Its use is totally dependent on the parents' cooperation and proper application.[61] It is not recommended in large children, in patients older than eight months, when the hip needs excessive flexion to keep reduction or when the hip is very unstable with adduction.

Whenever a Pavlik harness is used, the hip should be reduced within the first three to four weeks.[62] It can be confirmed either by physical examination or echography. If the hip is reduced within the harness, it will be kept until the hip stabilizes and the acetabulum normalizes.[62] If not, harness treatment should be suspended because of the risk of AVN. The success rate of a Pavlik harness in reducing an Ortolani-positive hip is in the range of 85% to 92%.[62] Risk factors for failure are: irreducible hip with Ortolani manoeuvre; inverted labrum; high β angle; insufficient coverture of the head; acetabular index >36°; and bilateral dislocation.[61,63,64]

Complications related to the use of the Pavlik harness are rare when it is adequately used.[60] AVN is related to excessive abduction of the hip.[65] AVN is always iatrogenic; it does not occur during the evolution of DDH. Excessive flexion could lead to inferior dislocation or paralysis of the femoral nerve.[65]

When the Pavlik harness has failed to reduce the hip, or in children older than six to eight months, a closed or open reduction in the OR followed by spica cast immobilization is indicated. In order to decide between closed or open reduction, arthrographic evaluation is recommended.[1] Arthrography is useful in the non-ossified skeleton, because it allows for the evaluation of soft tissues and cartilaginous parts of the femoral head and acetabulum.[66] Arthrograms show us if a concentric and stable reduction of the hip is possible by closed means. If not, an open reduction would be necessary in order to release all obstacles (i.e. pulvinar fat, teres ligament, labrum, psoas tendon, anteromedial capsule) or perform a capsulorrhaphy.[67] Open reduction is more often needed as the child gets older.

If a force closed reduction is attempted or a dislocated hip is maintained within the cast, AVN could occur.[66] MRI is useful to assess reduction within the cast. On the other hand, open reduction has also been related to AVN. Some authors believe that AVN after an open reduction is higher when the ossification nucleus of the femoral head is still absent in X-rays. Therefore, delaying open reduction until complete ossification of the femoral head can decrease the risk of AVN. However, the later the hip is reduced, the higher the risk of multiple surgeries.[68]

Once a stable reduction is obtained by closed or open means, it should be stabilized for three months in a spica cast.

Correction of acetabular dysplasia

As stated before, once hip dislocation or subluxation is corrected, acetabular development should be evaluated. During the first months of life, the stimulus provoked by a stable and concentrically positioned femoral head into the acetabulum is usually enough to normalize acetabular development. In the young child, a Pavlik harness can be used to maintain the hip in flexion and abduction, so acetabular growth can be stimulated. In children older than six to eight months, a rigid hip abduction orthosis may be used instead of a Pavlik harness. The older the child, the lower the potential for the normalization of a dysplastic acetabulum. Prevalence of acetabular dysplasia increases with the age of hip reduction.[69] The age when acetabular normalization cannot be achieved after hip reduction is not well-known. Acetabular dysplasia can occur even if the reduction is performed within the first months of life. Up to 19% of patients successfully treated with a Pavlik harness show residual dysplasia; 22% to 33% of patients with closed or open reduction present it.[70,71,72]

In those cases with persistent acetabular dysplasia, acetabular and/or femoral osteotomies are necessary to avoid or minimize the risk of coxarthrosis at maturity. The exact moment to perform them is not well defined, but age and evolution of the acetabular index can orient us to predict the probability of residual dysplasia at maturity.[62]

Femoral osteotomies are designed to reorient the femoral head by derotation and increasing the varus in order to stabilize and stimulate acetabular development.[12,73] These techniques are based on the principle that the proximal femur has increased anteversion and varization, a subject that still remain controversial.[11] Femoral anteversion is the main cause of subluxation recurrence, so derotational osteotomy can be necessary to maintain a stable reduction of the hip. Experimental studies have demonstrated that varization also increases acetabular volume.[12]

Acetabular osteotomies try to increase the coverage of the femoral head acting on the acetabular side. There are two main groups: those that preserve acetabular cartilage; and those that do not preserve the articular cartilage (salvage osteotomies). The first group is composed of reorientation osteotomies (Salter, triple osteotomy, periacetabular osteotomy (PAO)) and acetabuloplasties that alter the morphology (Dega or San Diego, Pemberton). Whether these osteotomies can be used in unstable hips remains controversial. Classically, the majority of authors believed that physiological acetabular osteotomies should be only performed in a reduced and stable hip.

Reorientation osteotomies (Salter, triple osteotomy, PAO) increase lateral and anterior coverage of the femoral head by changing acetabular direction. Acetabuloplasties (Dega or San Diego, Pemberton) offer a higher rate of correction of acetabular dysplasia in comparison with reorientation osteotomies.[74]

In contrast, salvage osteotomies are designed to stabilize the hip and to increase femoral head coverage and acetabular surface of contact. They do not preserve articular cartilage as the contact surface between the femoral head and acetabulum. However, an interposed articular capsule between the femoral head and ilium (in case of Chiari osteotomy) or bone graft (in shelf osteotomy)

develops a cartilage metaplasia that could remember articular cartilage.[75,76]

Pelvic osteotomies do not completely avoid the risk of developing hip arthritis in the young adult. Thomas et al stated that 23.8% of patients treated with a Salter osteotomy had needed a THR 40 years after acetabuloplasty.[77] Furthermore, another 17% presented arthritis grades 3 or 4 (Kellgren and Lawrence classification) although a THR had not been implanted. In a similar way, Steppacher et al found that, after 20 years of follow-up of patients with a PAO, 38% of patients needed a THR.[78]

Conclusions

DDH is the main cause of THR in young people (about 21% to 29%). The higher the age at presentation, the worse the outcomes after intervention for DDH. Therefore, prompt diagnosis is the most important factor related to outcome. Instability manoeuvres and assessment of hip abduction should be done universally as a part of the physical examination of the newborn. Isolated 'clicks' or inguinal fold asymmetry, although classically referred, have no clinical importance. There is a clear consensus about performing a hip ultrasound when the neonatal physical examination is normal if breech presentation or positive family history of DDH is present. Controversy remains around other risk factors that would indicate the necessity of a hip ultrasound. Universal ultrasound screening has not demonstrated its utility to diminish the incidence of late dysplasia and, on the other hand, increases the rate of overtreatment. Radiographic evaluation is the main imaging study to evaluate the growth and development of the hip from four to six months of life until maturity.

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