Irreducible developmental dysplasia of the hip due to acetabular roof cartilage hypertrophy. Diagnostic sonography in 15 hips

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Accepted: 14 March 2011

KEYWORDS
Irreducible developmental dysplasia of the hip; Sonography; Arthrography; MRI; Surgery

Summary
Introduction: Irreducible developmental dysplasia of the hip (DDH) in newborns is a rare entity. The different obstacles preventing reduction have been described in the literature.
Hypothesis: A clinical form of DDH with hypertrophy of the cartilage of the acetabular roof (acetabular bulge) can be reliably identified on ultrasound and should probably be defined as a separate entity.
Materials and methods: For the first time, the authors report their experience, a review of the literature and the radiographic description (ultrasound, arthrography MRI) of irreducible neonatal DDH due to hypertrophy of the cartilage of the acetabular roof (acetabular bulge) in 12 infants (15 hips).
Results: Neonatal sonography seems to be sufficient to identify this specific clinical entity without any additional work-up. This sonographic sign could help determine the therapeutic strategy earlier in this severe and complex form of DDH.
Level of evidence: Level IV. Diagnostic Study.

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Introduction
Sonography of the hip is a remarkable tool for the screening and early diagnosis of developmental dysplasia of the hip (DDH) in newborns. This does not exclude the need for a careful, systematic clinical examination to obtain a complete clinical picture. The term ''DDH'' is debatable.
Many terms are used: instability, pathological dislocation, congenital dislocation of the hip (CDH), or DDH. The irreducible character of this entity is even less well defined. For some, these hips are considered to be irreducible at birth, may sometimes still be called teratological, for others they correspond to dislocated hips that do not respond to appropriate orthopedic treatment.

Irreducible neonatal DDH is a rare entity, corresponding to approximately 1% of all DDH [1]. The obstacles preventing reduction of the dislocated hip have been described in the literature: inversion of the labrum, interposition of the psoas, narrowing of the capsular isthmus, thickening of the round ligament (ligamentum teres) and of the transverse ligament of the acetabulum, thickening of the fibrofatty pulvinar tissue and hypertrophy of the cartilage of the acetabular roof (acetabular bulge) [2–5].

Imaging techniques (sonography, arthrography, MRI) are used to evaluate the mechanisms preventing reduction and play a role in choosing the treatment strategy.

The aim of this study was to describe the sonographic signs of irreducible neonatal DDH from acetabular bulge and to correlate the results with other imaging techniques.

In the presence of suspected DDH at the clinical examination, the goal was to perform early sonography to confirm the diagnosis of this severe form of DDH from acetabular bulge, and to determine the therapeutic strategy, such as first-line surgical reduction, without prior orthopedic treatment.

Materials and methods

This is a retrospective study of 15 pathological hips in 12 patients (9 girls and 3 boys, the bilateral cases only occurred in girls). The study was performed between January 1990 and December 2000, and infants were followed in Pediatric Units in University Hospitals in Rennes and Montpellier, France. Ten patients had no other associated diseases, while one patient presented with a Turner syndrome and another with a convex pes valgus contralateral to the dysplasia.

The clinical examination showed limited abduction in 11 cases, a snapping sensation in five cases and pseudoshortening (positive Galleazzi sign) in three cases.

Early sonography was performed in all children, at between 2 and 40 days after birth (mean age: 21 days). Two types of ultrasound were used: HDI ultramark nine ATL using a 5–10 MHz probe and HDI 5000 ATL using a 5–12 MHz probe. The Couture [6] method was used for the examination. This includes scanning the external frontal plane with the infant in the decubitus dorsal position, with the hip in flexion-adduction. The sonographic references used for this method are the pubic bone which is an essential reference for this scan, the rectilinear coxal bone and the deepest part of the acetabular roof. The normal value for the acetabular fossa is ≤ 6 mm [7] as shown in Fig. 1. A dynamic examination to determine the possibility of realigning the femoral epiphysis completed the morphological examination.

Analysis of each sonography included: the thickness of the acetabular fossa, the bone covering the femoral head, the morphology of the acetabulum, the thickness and the sonographic structure of the acetabular hyaline cartilage, the position of the labrum and the largest diameter of the proximal femoral epiphysis.

A high posterior dislocation of the proximal femoral epiphysis (Fig. 2) was found on sonography in all cases, with an absence of bony coverage by the true acetabulum. The diameter of the proximal femoral epiphysis was small, a mean 12 mm (10–14 mm), for a normal value of 14 mm. In five cases, it was found across from an echogenic neoacetabulum. In 12 cases, a hyperechogenic structure identified as the labrum was found inverted in the joint. In three cases, the labrum was not identified. The sonographic sign that was found in all cases was thickened acetabular roof cartilage (acetabular bulge), with acetabular hyaline cartilage located in the anterosuperior portion of the coxal bone.

**Figure 1** Measurement of the acetabular fossa (white arrow) between the pubic bone and the medial proximal femoral epiphysis.
Irreducible developmental dysplasia of the hip due to acetabular bulge

Figure 2 Irreducible dislocation due to hypertrophy of acetabular roof cartilage. Distance increased by 1.44 cm.

This was hypoechogenic, bulging and convex and between 5 and 7 mm thick while in the healthy controlateral hip the value was 2 mm (Fig. 3). This thickening narrowed the acetabular fossa preventing any attempts at reduction by dynamic manoeuvres (Fig. 4). The mean acetabular fossa was 12.5 mm.

On the 13 arthrographies, the proximal femoral epiphysis was found to be dislocated in a high posterior position, and could not be reduced in the true acetabulum by dynamic manoeuvres. The superior capsular isthmus was narrowed due to cartilage obstruction. This obstacle appeared as a convex form on the scan, and caused a filling defect of the contrast medium in the space between the proximal femoral epiphysis and the acetabular roof. The ligamentum teres was voluminous in six cases (Fig. 5) normal in four and not identified in three cases. The transverse ligament of the acetabulum appeared thick in six cases, normal in two and was not found in five cases. The psoas tendon was found in nine cases. The fibrofatty pulvinar tissue appeared thickened in all cases.

MRI showed hypertrophy of the acetabular cartilage in all the cases identified on sonography: the cartilage appeared as a high intensity signal on T2-weighted and gradient echo sequences and as an intermediate intensity signal on T1-weighted sequences. Coronal slices showed the thick, convex cartilage covering a flat, insufficiently shaped ilium. Axial slices confirmed the thin appearance of the triangular true acetabulum, obstructed by anterior pubic and iliac hypertrophy of the acetabular cartilage. The Y cartilage

Figure 3 Irreducible DDH due to hypertrophy of acetabular roof cartilage (acetabular bulge) (shown in yellow on a reproduction of the same sonographic scan).

Figure 4 Same infant as Fig. 3. Attempted sonographic guided reduction of right DDH, hip in abduction (left image), this hip is irreducible.

Figure 5 Arthrography of the hip showing hypertrophy of the acetabular roof cartilage (arrow). The ligamentum teres is also hypertrophic.
appeared to bulge like the hypertrophic cartilage of the acetabular roof (Fig. 6).

The femoral epiphysis, dislocated high and posteriorly appears flattened in all 10 cases while in four cases a posterosuperior neo-acetabulum was identified as a high intensity signal on T2 sequences.

The ligamentum teres, which produced a low-intensity signal in all sequences, was thickened in six cases, the transverse ligament of the acetabulum was thickened in four cases. The psoas tendon was interposed in all cases. The capsule produced an intermediate intensity signal, which was difficult to differentiate from the muscular structures and appeared thickened in four cases. The soft fibrofatty pulvinar tissue produced a high intensity signal on T-1 sequences and was interposed in the acetabular fossa in all cases.

Discussion

Certain anatomopathological studies in the literature have classified the severity of DDH [8]. The Dunn [2] classification proposes three grades based on the severity of the anatomical disorder: grade I: subluxation with an everted labrum; grade II: intermediate dislocation with a partially inverted labrum; grade III: complete dislocation with an inverted labrum.

Nevertheless this classification does not explain certain observations by Ponseti [3] or Milgram et al. [4] in which hypertrophy of acetabular roof cartilage was observed in cases of irreducible hip dislocation. Ponseti reported the results of autopsies of six cases of infant DDH with hypertrophy of acetabular roof cartilage which the author called "acetabular bulge" [3]. In three cases, this was isolated but in three other cases, it was associated with an inverted labrum. Macroscopic coronal slices showed that the ridge of cartilage divided the articular surface of the acetabulum into two portions: the anteroinferior portion of the true acetabulum (paleoacetabulum) on one hand and the posterosuperior portion associating the inverted labrum attached to the perichondrium and the thick joint capsule (neoacetabulum). In severe forms, the limit between the labrum and the acetabular hyaline cartilage cannot be identified. Cytoarchitectural histiochemical cartilage anomalies raise the question of whether this degenerative process can be reversed. Milgram et Tachdjian [4] reported a similar case in a child presenting with Goldenhar syndrome with the presence of an ineffective true acetabulum and a secondary acetabulum formed by a fibrotic limbus which had fused with the hypertrophic cartilage of the acetabular roof.

We report 15 cases of early diagnosis by sonography of irreducible neonatal DDH due to hypertrophic acetabular roof cartilage. Few authors have evaluated this anatomical element as a major mechanical factor influencing irreducible DDH. In cases of hip dysplasia, Soboleski and Babyn [5] studied the thickness of the hypertrophic acetabular cartilage at the age of 2 months. They report normal values between 2.6 and 3 mm. The mean value in case of dysplasia was 4.6 and a normal concave morphology remained. In our series, this convex thickness was between 5—7 mm. Suzuki [9] reported his experience with anterior axial sonography scans of the hip in the follow-up of 62 cases of DDH treated by a Pavlik harness. In nine cases classified as type C (corresponding to irreducible hip dislocation by the author) the posterior wall of the acetabulum blocked reduction of the proximal femoral epiphysis.

Arthrography plays a role in the diagnosis of the anatomical obstructions to reduction. Astley [10] re-evaluated 82 arthrographies for DDH and found 16 cases of labral obstruction and one case of hypertrophy of the acetabular cartilage preventing reduction. However, the ages of the patients in these arthrographies were not mentioned. Tanaka et al. [11] measured the T thickness of the hypertrophied acetabular cartilage and soft tissue interposition in arthrographs performed during treatment by traction. He identified the dislocated hips in which the T distance did not change during treatment and identified the presence of an obstacle but this was not specifically defined. They suggest: labral interposition, capsular collapse, hypertrophy of the acetabular cartilage. Our series identified 13 cases with evidence of hypertrophy of the acetabular cartilage.

More recently MRI has begun to play a role in understanding the different anatomical forms of DDH in particular thanks to thin-section multiplanar images with T-1 and T-2 weighted sequences making it possible to analyze: the muscular components, the form and the position of the proximal femoral epiphysis, the thickness of the capsule, the morphology of the psoas tendon, the ligamentum teres, the thickness of fibrofatty pulvinar tissue and the labral, triradiate and acetabular cartilage [12].

Aoki et al. [13] attributed signal modifications in the labrum in irreducible DDH to a degenerative process with vascular thrombi. In our series, the labrum was inverted in two cases and appeared as a low intensity signal on T2 sequences and an intermediate intensity signal on T2 sequences in eight cases in which it was impossible to differentiate the capsule from the perichondrium. Aoki et al. [13] performed a comparative study (arthrography, MRI, surgery) in 38 patients presenting with irreducible DDH. MRI results were similar to others for evaluating the anterior labrum and better for evaluating posterior labral deformities. But these results only describe the position and signal of the labrum and do not evaluate hypertrophied acetabular cartilage. Bos et al. [14] described their experience with MRI in 15 cases of DDH, including five which were irreducible. MRI was performed in five children treated by traction then a cast (between 21 and 42 months old) due to suspected...
incongruence of the coxofemoral articulation. In these five children, one case of hypertrophic acetabular roof cartilage was observed. Kashiwagi et al. [15] analysed the acetabular cartilage-labrum complex in 33 cases of DDH treated by Pavlik harness and proposed a three stage classification based on an evaluation of the deformity of the posterior acetabular wall. Stage 1 corresponds to an acute posterior wall (twelve cases), stage 2 to a rounded posterior wall (13 cases) and stage 3 to an inverted acetabular cartilage and labral complex (eight cases). Reduction by Pavlik harness was impossible in stage 3.

Conclusion

We report 15 cases of irreducible DDH with hypertrophy of the acetabular roof cartilage (acetabular bulge) on an external frontal sonographic scan. This hypertrophy, which is convex and bulging prevents any attempts at reduction of the superior femoral epiphysis in the true acetabulum, which is reduced volume. The sonographic structure varies; it is often hypoechoic and sometimes hyperechoic probably because of histological modifications. Sonography is enough to characterize the hip as irreducible. In these cases, an attempt at closed reduction could be avoided and primary treatment could be surgical. This optimal moment for this treatment must still be defined.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References