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Developmental Dysplasia of the Hip: Diagnosis, Management, and Clinical Implications in Pediatric Orthopedics

Shianita Limena[1], Seva Ajisma[1], Made Agus Maharjana[2]

[1]Resident, Dept. Orthopaedic & Traumatology, Prof IGNG Ngoerah General Hospital, Udayana University, Bali, Indonesia
[2]Pediatric Orthopaedic Surgeon, Dept. Orthopaedic & Traumatology, Prof IGNG Ngoerah General Hospital, Udayana University, Bali, Indonesia

Corresponding Author: Shianita Limena

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ABSTRACT

Developmental dysplasia of the hip (DDH) represents a spectrum of abnormalities affecting the anatomical structure and stability of the hip joint in infants and children. It ranges from subtle acetabular dysplasia to complete hip dislocation. DDH is one of the most common orthopaedic conditions in newborns and, if left undetected or untreated, leading to long-term disability, including early-onset osteoarthritis and significant functional impairment. Early detection through physical examination and imaging techniques—such as ultrasonography and radiographs—plays a critical role for early management and better outcomes. This review summarizes the etiology, pathophysiology, diagnostic modalities, classification systems, and treatment strategies for DDH, with emphasis on age-specific management approaches. A comprehensive understanding of DDH is essential to provide timely interventions that ensure optimal outcomes and minimize future disability. The integration of multidisciplinary care involving pediatricians, radiologists, and orthopedic surgeons is also key in achieving holistic patient-centered care.

Keywords: Developmental Dysplasia of the Hip; DDH; hip dislocation; Pavlik harness; pediatric orthopedics.

INTRODUCTION

Developmental Dysplasia of the Hip (DDH) encompasses a range of developmental abnormalities affecting the morphology, alignment, and stability of the hip joint, including acetabular dysplasia, subluxation, and dislocation. This condition is most prevalent during infancy, with reported incidences ranging from 1.7% to 20% depending on geographic region, population, and diagnostic methods. The pathogenesis of DDH involves a complex interplay between genetic, mechanical, and environmental factors, including breech presentation, family history, ligamentous laxity, and postnatal positioning.[1]

Clinically, DDH may present as hip instability in the neonatal period, and if undiagnosed or improperly managed, it may lead to progressive structural deformity. These deformities result in gait abnormalities, leg length discrepancy, restricted range of motion, and are a leading cause of early-onset hip osteoarthritis before the age of 60. Timely recognition and management are therefore crucial in preventing long-term disability and ensuring favorable outcomes.[2]

The diagnostic approach to DDH includes a combination of clinical examination techniques—such as the Barlow and Ortolani maneuvers—and imaging modalities like ultrasonography for infants and radiography for older children. The integration of screening strategies and classification systems (e.g., Graf, IHDI) enhances diagnostic accuracy and guides age-appropriate treatment planning.[3]

The primary goal of DDH management is to achieve and maintain concentric hip reduction, promote normal acetabular and femoral head development, and prevent complications such as avascular necrosis. Therapeutic strategies range from non-operative approaches like the Pavlik harness in early infancy, to surgical interventions such as open reduction and osteotomies in older children. A multidisciplinary and individualized approach to care is essential for optimizing functional and anatomical outcomes.[4]

This article aims to provide an up-to-date, comprehensive overview of DDH in Pediatric Orthopaedic, focusing on its pathophysiology, diagnosis, management strategies, and long-term clinical implications.

LITERATURE REVIEW

Definition and Terminology

Developmental Dysplasia of the Hip (DDH) refers to a spectrum of structural abnormalities of the hip joint that may affect the size, shape, orientation, or stability of the acetabulum, femoral head, or both. Historically known as Congenital Dislocation of the Hip (CDH), the terminology evolved to "developmental" to reflect the dynamic nature of the condition, as hip joints that appear anatomically normal at birth may later develop instability, subluxation, or dislocation—particularly during the first 6 to 10 months of life. Klisic (1989) introduced the term DDH to capture this developmental aspect more accurately.[5]

DDH encompasses several overlapping conditions:

- Dysplasia: an underdeveloped acetabulum that predisposes to instability.
- Subluxation: partial contact between the femoral head and acetabulum, with the head partially displaced.
- Dislocation: complete separation of the femoral head from the acetabular socket.
- Unstable hip: a joint that appears reduced but is prone to subluxation or dislocation under stress.
- Teratologic dislocation: a fixed, in utero dislocation associated with syndromic or neuromuscular conditions such as arthrogryposis or myelodysplasia. This type of dislocation is often irreducible and presents early in infancy.

Epidemiology

The reported incidence of DDH varies considerably across studies and populations due to differences in diagnostic criteria, screening protocols, and genetic predispositions. Globally, DDH affects approximately 1 to 10 per 1,000 live births, though subclinical or radiographic signs of dysplasia may be observed in up to 20 per 1,000 newborns. In a prospective study by Kokavec et al., up to 69.5 per 1,000 hips showed dysplastic features on sonography during the neonatal period;[6] however, most of these resolved spontaneously within 6–8 weeks, leaving only about 4.8 per 1,000 requiring interventions

Several risk factors have been identified:[4,7]

- Gender: Female infants are 4–6 times more likely to develop DDH than males.
- Birth order: First-born children are at higher risk due to tighter uterine constraint.
- Presentation: Breech presentation significantly increases DDH risk.
- Family history: A positive family history strongly predicts DDH in offspring.
- Ethnicity: Caucasians and Native Americans have higher rates of DDH, while the condition is rare in populations of African descent.

- Environmental and cultural factors: Tight swaddling or straight-leg wrapping practices (such as traditional bundling) are associated with increased DDH prevalence due to prolonged hip extension and adduction.

Laterality patterns also show asymmetry: unilateral involvement is more common than bilateral, with the left hip affected in approximately 60% of cases. The increased pressure of the fetal spine against the left hip in the typical left occiput anterior fetal position is believed to contribute to this trend. Understanding the epidemiological variability of DDH is crucial for establishing effective regional screening strategies and identifying high-risk populations.

Pathophysiology

The pathophysiology of DDH centers on the disruption of normal biomechanical and anatomical development of the hip joint, particularly the concentric relationship between the femoral head and the acetabulum. This reciprocal alignment is essential during infancy for proper joint modeling and maturation.[7]

In a normally developing hip, the femoral head remains securely seated within the acetabulum, which provides the mechanical stimulus required for the acetabulum to deepen and ossify appropriately. In DDH, this alignment is altered due to a range of contributing factors, including ligamentous laxity, shallow acetabular development, capsular insufficiency, and abnormal fetal positioning. When the femoral head fails to maintain proper contact within the acetabular socket, a pathological cycle is initiated:[8,9]

- The acetabulum becomes shallow and flattened, failing to provide adequate containment.
- The femoral head becomes deformed, often with delayed ossification and a misshapen or aspherical contour.
- Soft tissue structures within the joint, including the pulvinar (fibrofatty tissue), hypertrophied ligamentum teres, and the labrum, may become interposed and obstruct anatomical reduction.[8,9]

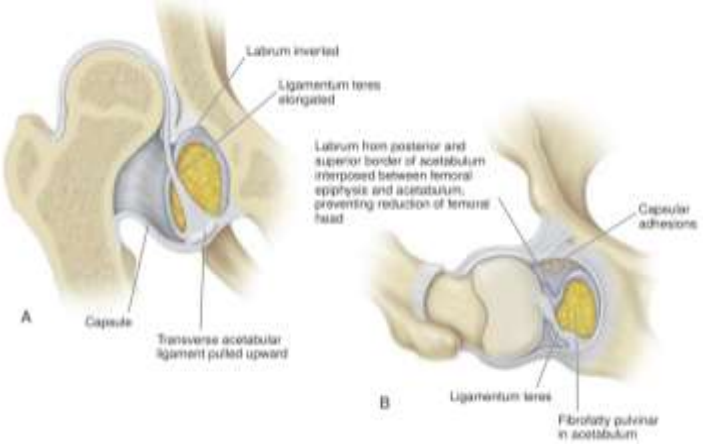
The labrum, which normally contributes to joint stability, may become everted or inverted—leading to further mechanical resistance to reduction. A hypertrophied everted labrum is termed a limbus, while a neolimbus refers to the hypertrophic cartilage ridge formed in subluxated hips, dividing the joint cavity into primary and secondary acetabular zones.[8,9]

As dislocation becomes chronic, the hip capsule contracts and narrows, particularly at the zone traversed by the iliopsoas tendon, creating an “hourglass” constriction that mechanically prevents closed reduction. Additionally, femoral anteversion and neck-shaft angle (coxa valga) may increase, further destabilizing the joint. The longer the femoral head remains displaced, the more profound the morphological changes in both osseous and cartilaginous structures.[9]

In severe or untreated cases, the femoral head migrates superiorly and posteriorly, and a pseudoacetabulum may develop—a false socket formed by remodeling of adjacent bone and soft tissues. Although this may allow for some articulation, it predisposes the joint to early degenerative changes and functional impairment.[9]

It is important to note that these pathological changes are often reversible in early infancy, particularly if a concentric and stable reduction is achieved. However, delayed intervention beyond critical developmental windows may result in irreversible damage and poor long-term prognosis.[9]

Figure 1. Shows an irreducible hip dislocation caused by intra-articular obstacles, including an inverted limbus, ligamentum teres, and fibrofatty pulvinar within the acetabulum.[10,11]



Diagnosis

Early diagnosis of Developmental Dysplasia of the Hip (DDH) is critical to achieving optimal outcomes, as the plasticity of the infant hip allows for spontaneous correction or effective intervention during the early months of life. Delayed recognition often leads to more invasive treatment, reduced success rates, and higher risk of complications such as avascular necrosis (AVN), gait abnormalities, and early-onset osteoarthritis.[12,13]

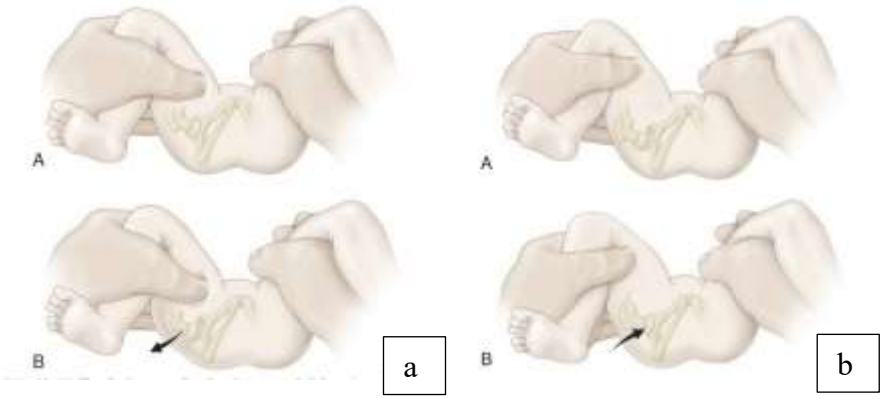
Age 0-6 months

The cornerstone of DDH diagnosis during the neonatal and early infancy period is physical examination, particularly in the first 6 weeks of life. Two classic maneuvers are widely employed:

- Barlow Test: This assesses whether a stable hip can be dislocated. With the infant supine and hips flexed to 90°, gentle posterior pressure is applied along the femur. A positive Barlow test indicates that the femoral head can be dislocated posteriorly out of the acetabulum.[11,14]
- Ortolani Test: This evaluates whether a dislocated hip can be reduced. Gentle abduction and anterior lifting of the femur are performed. A "clunk" suggests reduction of the femoral head into the acetabulum—indicative of a positive Ortolani sign.[11,14]

Other signs include asymmetrical thigh or gluteal folds, apparent limb length discrepancy (Galeazzi sign), limited hip abduction, and leg shortening. However, as the infant grows, these signs become less specific, highlighting the need for imaging.

Figure 2. (a) Barlow Test, (b) Ortolani Test[11]



Ultrasonography is he preferred modality for infants under 6 months, as the cartilaginous structures of the hip are not yet ossified and therefore poorly visualized by radiographs. The Graf method, which uses coronal views through the hip, classifies hips into types based on alpha and beta angles that reflect acetabular development and femoral head coverage. Ultrasonography also facilitates dynamic assessments and is valuable for monitoring response to treatment (e.g., Pavlik harness).[10,16,17]

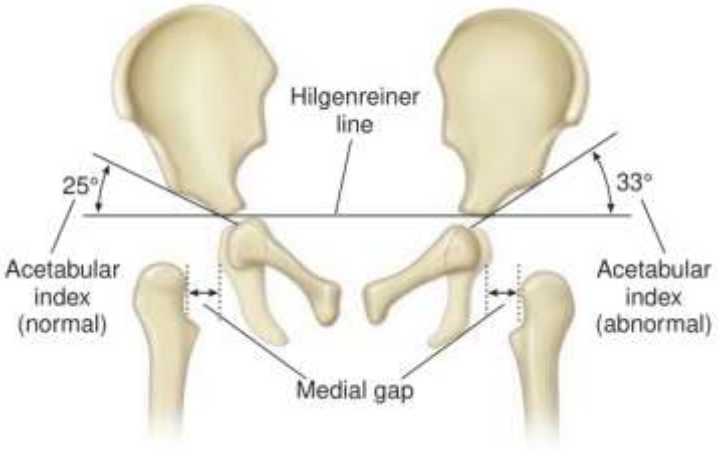
Age 6-18 months

At two to three months of age, DDH continues to progress from instability to irreducible dislocation, which can occur within a few weeks or be delayed until five to six months of age. Unilateral permanent dislocation is characterized by limited hip abduction, thigh shortening—positive on the Galeazzi sign when both hips are flexed to 90°—as well as asymmetric thigh creases and a more proximal position of the greater trochanter on the affected side. In bilateral dislocation, asymmetry of abduction and the Galeazzi sign are often not apparent, so detection requires the Klisic test; by placing the third finger above the greater trochanter and the index finger on the anterior superior iliac spine, the imaginary line that should point to the umbilicus will shift to a point between the umbilicus and the pubis.[11,15]

Radiography is recommended for children older than 4–6 months, when ossification of the femoral head has begun. Key radiographic features assessed on anteroposterior pelvic X-rays include:

- Shenton's line: A smooth arc formed by the inferior margin of the superior pubic ramus and the medial aspect of the femoral neck; disruption suggests subluxation or dislocation.[11,18]
- Hilgenreiner's line and Perkin's line: Used to create quadrants; the normal femoral head should lie in the inferomedial quadrant.[11,18]
- Acetabular index (AI): The angle between the horizontal Hilgenreiner's line and a line through the acetabular roof; values above age-specific norms indicate dysplasia.[11]
- Wilberg Center-edge Angle (CEA) : the angle formed at the intersection of the Perkin line and the line connecting the lateral margin of the acetabulum to the center of the femoral head.[11]

Figure 3. Acetabular index and medial gap: The acetabular index is the angle between the acetabular margin line and Hilgenreiner's line, averaging 27.5° in normal newborns and decreasing with age.[11]



Age 18-24 months

Unilateral hip dislocation in children who are already able to walk presents with the affected limb appearing shorter, so that when stepping on the pathological side, the hip drops, the adductor muscles weaken, and the body weight shifts to the dislocated hip, resulting in a Trendelenburg gait pattern where, when standing on one leg, the body leans toward the affected side

(Trendelenburg sign). Conversely, bilateral dislocation is more difficult to detect because both sides may be symmetrical or only appear as excessive hip drop during the double-support phase. Compensation with excessive lumbar lordosis often occurs due to hip flexion contractures, while knee alignment, symmetrical but limited abduction, and increased internal and external rotation in both hips are also clinical findings.[11,12]

Age >24 months

In children older than 24 months or 2 years, the physical examination that needs to be performed is the same as for children aged 18-24 months, which is the Trendelenburg Sign and Gait Leg Length Discrepancy tests.[11]

Advanced Imaging (CT/MRI): These are reserved for pre-operative planning or post-reduction assessment in complex or teratologic cases, particularly to confirm concentric reduction and identify obstacles to reduction (e.g., interposed tissue or inverted labrum).[19,20]

Table 1. Magnetic resonance imaging findings of anatomical structures involved in developmental hip dysplasia.[19]

Anatomic Structure	Imaging findings
Acetabular morphology	Shallow, dysmorphic acetabulum with abnormal cartilage anlage
Femoral morphology	Delayed ossification of non-spherical femoral head
Labrum	Labral hypertrophy (limbus) with mucoid degeneration
Pulvinar	Pulvinar hypertrophy with fibrofatty proliferation
Ligamentum teres	Hypertrophy with contraction of the transverse acetabular ligament
Hip capsule	Patulous and redundant joint capsule

Screening

Screening for DDH is as important as the management itself. Two primary screening approaches includes:

- Selective Screening: Performed only on infants with clinical instability or risk factors (e.g., breech, family history). This method balances cost and detection efficacy.
- Universal Screening: All neonates undergo ultrasonography, often used in high-prevalence countries (e.g., Austria, Germany). While more sensitive, it may lead to overdiagnosis and overtreatment of transient immaturity.[11]

Management

The management of Developmental Dysplasia of the Hip (DDH) is guided by the age at diagnosis, severity of dysplasia, hip stability, and presence of teratologic features. The primary objective is to achieve and maintain concentric reduction of the femoral head within the acetabulum to allow normal joint development, while minimizing complications such as avascular necrosis (AVN).[4,13,21] Non-operative treatment includes: Proper Swaddling Practice, Pavlic Harness, Hip Abduction Brace, Closed Reduction and Immobilization with Hip Spica. Operative treatment includes: Open Reduction, Pelvic Osteotomy, and Femoral Osteotomy.

Age 0–6 months

The Pavlik harness is the first-line treatment for most infants under 6 months with reducible dislocation or hip instability. It maintains the hips in flexion (90–100°) and abduction (45–60°), allowing dynamic stabilization while preserving motion.

Success rates range from 80–95% in cases with reducible hips. Regular ultrasound monitoring is crucial to confirm proper hip positioning and avoid complications, particularly femoral nerve palsy and iatrogenic AVN due to forced abduction or hyperflexion. The harness is typically worn for 6–12 weeks.[19] Contraindications include: Fixed dislocation, Teratologic dislocation, Failure after 3–4 weeks of appropriate use. When Pavlik harness is unsuccessful or

contraindicated, rigid abduction braces (e.g., Von Rosen splint, Denis Browne bar) may be used. These devices also maintain hip abduction, particularly in cases of residual acetabular dysplasia post-reduction.[13]

Figure 4. Pavlik harness: Transverse chest straps are placed just below the nipple line. The hips are flexed to 120°, and posterior straps should avoid forced abduction.[11]

Age 6-18 months

When conservative treatment fails or diagnosis is delayed (typically after 6 months), closed reduction under general anesthesia is often indicated. The procedure involves: Gentle manipulation of the femoral head into the acetabulum, Arthrography to confirm reduction and identify obstructing structures and application of a hip spica cast in the "human position" (flexion and abduction). Reduction is maintained for 12 weeks, with subsequent bracing if needed. The success rate is reduced in older infants, and failure to achieve reduction or poor containment may require surgical intervention.[11]

Age older than 18 months

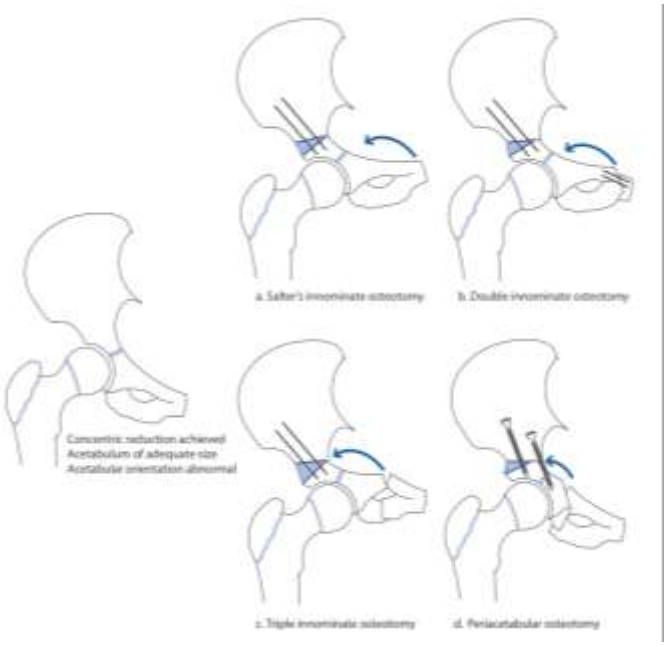
When closed reduction fails, open reduction is performed. Other indication for open reduction includes: Irreducible hips, Late diagnosis (>18 months), and Teratologic dislocation. Anterior or medial approach is used, intra-articular obstacles are removed (e.g., pulvinar, inverted labrum, hypertrophied ligamentum teres), and the femoral head is positioned concentrically. Hip spica casting follows for 6–12 weeks.[11]

Age older than 24 months

In older children, or presenting with residual hip dysplasia, open reduction is performed with extensive measures. Osteotomies and Salvage Procedures is considered, including:

a. Pelvic Osteotomies
   Salter innominate osteotomy, Pemberton, or Dega osteotomy are used to redirect the acetabulum for better femoral head coverage, especially in residual dysplasia with a concentrically reduced hip.[22]
b. Femoral Osteotomies
   Varus derotation osteotomy (VDRO) corrects increased femoral anteversion or neck-shaft angle. It may be combined with pelvic procedures.
c. Salvage procedures[23]
   In older children with dislocated hips and poor acetabular development, salvage options such as Chiari osteotomy or Shelf procedure provide containment but do not restore normal anatomy.[24,25]

Figure 5. Examples of redirectional osteotomies of the pelvis for acetabular dysplasia. (A) Salter innominate osteotomy. (B) Double innominate osteotomy. (C) Triple innominate osteotomy. (D) Periacetabular osteotomy.[22]



Complications

Avascular necrosis (AVN) of the femoral head is one of the most feared complications in the management of developmental dysplasia of the hip (DDH). It can result from excessive abduction, high hip flexion angles, pressure during the reduction maneuver, or compromised blood supply after surgery. The reported incidence varies widely, ranging from 5% to 60% of treated hips, depending on treatment modality and patient age. AVN may cause growth disturbances, premature physeal closure, femoral head deformity, and secondary osteoarthritis.[26] Residual acetabular dysplasia (RAD) may also occur even after successful reduction, characterized by persistent shallowness of the acetabulum that can lead to hip instability, subluxation, and early degenerative changes. Continuous surveillance until skeletal maturity is essential, and corrective osteotomy is indicated if remodeling fails.[11] Redislocation or subluxation is another complication, which can arise after closed or open reduction, particularly when reduction is unstable or obstructed by soft tissue interposition, as well as due to inadequate postoperative immobilization or improper spica casting techniques.[16] Iatrogenic femoral nerve or sciatic nerve palsy may develop from excessive traction or positioning during reduction or casting; while usually transient, it may require brace removal or cast modification.[16] Additionally, joint stiffness and limited range of motion can result from prolonged immobilization or surgical scarring, making physical therapy an essential part of post-treatment rehabilitation.[16]

Prognosis

The overall prognosis of DDH is highly dependent on age at diagnosis, type and timing of treatment, and adequacy of femoral head reduction. Key prognostic considerations include:[11]

- Early diagnosis (<6 months) and successful treatment with Pavlik harness are associated with the highest success rates (>90%) and lowest complication risk.
- Closed reduction before walking age has favorable outcomes if concentric reduction is achieved and maintained.
- Open reduction and osteotomies yield good to satisfactory results in many cases, but increase the risk of AVN and

residual dysplasia, especially in children >18 months.

- Late-diagnosed DDH (after 2–3 years) has significantly worse outcomes, often requiring complex reconstructive procedures and long-term follow-up.
- Untreated or inadequately treated DDH may lead to early-onset osteoarthritis, pain, gait abnormalities, and functional disability, frequently requiring total hip arthroplasty before age 50.

Long-term follow-up until skeletal maturity is recommended, as some complications such as residual dysplasia or AVN may manifest years after initial treatment.

DISCUSSION

Developmental Dysplasia of the Hip (DDH) remains a significant cause of preventable disability in children worldwide. The reviewed literature highlights that early detection and appropriate age-based management are essential in minimizing long-term morbidity. Most guidelines agree on the utility of early clinical screening through Barlow and Ortolani maneuvers, followed by risk-based ultrasonographic evaluation using the Graf method. However, in many regions with limited resources or delayed presentation, these strategies are often underutilized, contributing to late diagnoses and suboptimal outcomes.[1,2]

Table 1. Physical Examination, Imaging, and Management of Hip Dysplasia by Age

Age	Physical Examination	Supporting Examination	Management
0–6 months	Barlow Test Ortolani Test	Ultrasound (USG)	Pavlik Harness Observation
6–18 months	Galeazzi Sign Klisic Sign	Plain Pelvic X-ray	Closed Reduction: - Stable → Hip Spica for 3 months - Unstable → Open Reduction
18–24 months	Trendelenburg Sign & Gait Leg Length Discrepancy	Plain Pelvic X-ray	Closed Reduction + Double Set-Up for Open Reduction (if closed reduction failed) ± Pelvic Osteotomy (if needed)
> 24 months	Trendelenburg Sign & Gait Leg Length Discrepancy	Plain Pelvic X-ray	Open Reduction + Femoral Shortening ± Pelvic Osteotomy (if needed)

In infants aged 0–6 months, the hip joint remains largely cartilaginous and pliable, providing an optimal window for conservative intervention. Clinical maneuvers such as Barlow and Ortolani tests are effective in detecting instability, and ultrasonography using the Graf method allows dynamic and structural evaluation of the developing hip. Management with Pavlik harness during this period has shown success rates exceeding 90% when initiated early, with minimal complications. However, missed diagnoses or delayed initiation significantly reduce the efficacy of bracing and increase the risk for irreversible joint damage.[3,4]

In the 6–18 month age group, the ossification of the femoral head and acetabulum reduces the success of harness therapy, necessitating more invasive strategies. Clinical signs such as the Galeazzi sign and Klisic test become more evident as limb length discrepancy develops. Radiographic imaging becomes the modality of choice due to increasing bone mineralization. At this stage, open reduction becomes the primary treatment modality. The distinction between stable and unstable hips post-reduction is critical; stable hips may be managed with hip spica casting, whereas unstable hips require repeat open reduction or further surgical intervention.[5]

By 18–24 months, clinical signs such as Trendelenburg gait and leg length discrepancy are typically evident. Management becomes more complex, often requiring closed reduction with double set-up

open reduction, and frequently accompanied by Salter or Pemberton osteotomy to correct acetabular dysplasia. The surgical burden increases, and the risk for complications such as avascular necrosis (AVN), redislocation, and limited range of motion becomes more pronounced.[4,7]

In children older than 24 months, the hip joint has often developed in a maladaptive manner, necessitating more extensive surgical correction. In addition to open reduction, procedures such as femoral shortening osteotomy may be required to reduce tension on the neurovascular structures and allow proper seating of the femoral head. Acetabular reconstruction remains essential in many cases to ensure long-term joint stability and prevent early-onset osteoarthritis.[12,13]

Globally, the disparity in early detection rates reflects a lack of uniformity in screening protocols. While countries such as Austria and Germany have implemented universal ultrasound screening, many low- and middle-income countries continue to rely on selective screening or clinical detection, leading to delayed diagnosis often beyond the optimal therapeutic window. Moreover, in resource-limited settings, barriers such as inadequate training of primary healthcare providers, lack of access to pediatric orthopedic expertise, and absence of routine well-baby follow-up contribute to poor outcomes.

There is a growing call for the implementation of regionally-adapted screening algorithms that incorporate local healthcare infrastructure, cultural practices, and population-specific risk factors. Additionally, increasing public and professional awareness, and ensuring access to appropriate imaging modalities, will be vital in reducing the global burden of DDH. Special emphasis should be placed on early identification in the 0–6 month window, where outcomes are most favorable, and ensuring that treatment pathways are clearly delineated by age group, as each age category presents distinct diagnostic and therapeutic challenges.[21]

The success of non-surgical interventions such as Pavlik harness therapy is notably high in infants under six months, underscoring the importance of early identification. Conversely, late-presenting DDH cases require more invasive interventions, including open reduction and osteotomies, which are associated with higher complication rates, particularly avascular necrosis (AVN) and residual dysplasia. Moreover, the literature indicates variation in screening practices worldwide, with countries like Austria and Germany implementing universal ultrasonography, while others rely on selective screening based on clinical signs or risk factors.[3,4]

This review also reveals persistent challenges in follow-up care, especially in ensuring long-term monitoring for residual dysplasia, which may not become radiographically apparent until later childhood. A significant barrier in many healthcare settings is the lack of awareness among primary care providers and limited access to pediatric orthopedic services. There is a growing consensus that establishing national screening protocols, investing in training for early hip examinations, and improving parental education could collectively improve early detection and reduce the burden of late-stage DDH. Age-appropriate diagnosis and treatment management is very resourceful for daily practice (Table 1).

Furthermore, this literature synthesis reflects a need for updated, region-specific guidelines that consider cultural, socioeconomic, and infrastructural differences in healthcare delivery. Studies from Asia and Africa highlight delays in diagnosis beyond one year of age, often due to lack of routine well-baby checkups and poor access to imaging. Addressing these disparities will require a multidisciplinary effort, combining clinical expertise, health policy reform, and community-based awareness programs.

CONCLUSION

Developmental Dysplasia of the Hip (DDH) is a condition with favorable outcomes when diagnosed and treated early, but it poses serious functional and orthopedic challenges when missed or mismanaged. This review emphasizes the critical role of timely screening, accurate imaging, and age-specific interventions in improving prognosis and minimizing complications such as avascular necrosis and residual dysplasia. While universal screening offers theoretical advantages, a well-implemented selective screening model with trained examiners remains practical and cost-effective in many settings. The findings also underscore the urgent need for public health initiatives and clinician education to bridge gaps in early diagnosis, especially in low-resource environments. Long-term follow-up into skeletal maturity remains essential to ensure joint development and prevent early-onset osteoarthritis. A coordinated effort from pediatricians, orthopedic surgeons, and policymakers is vital to reduce the global burden of DDH.

Declaration by Authors

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REFERENCES

1. Nandhagopal T, Tiwari V, Cicco FL De. Developmental Dysplasia of the Hip. *StatPearls*. Published online May 4, 2024. Accessed August 4, 2025. https://www.ncbi.nlm.nih.gov/books/NBK563157/
2. Vaquero-Picado A, González-Morán G, Garay EG, Moraleda L. Developmental dysplasia of the hip: update of management. *EFORT Open Rev*. 2019;4(9):548-556. doi:10.1302/2058-5241.4.180019
3. Raba AA, Gasibat Q, Raba AA, Gasibat Q. Developmental Dysplasia of the Hip: Management of Graf IIa Dysplastic Hip. *Open J Pediatr*. 2020;10(2):247-254. doi:10.4236/OJPED.2020.102025
4. Alrashdi N, Alotaibi M, Alharthi M, et al. Incidence, Prevalence, Risk Factors, and Clinical Treatment for Children with Developmental Dysplasia of the Hip in Saudi Arabia. A Systematic Review. *J Epidemiol Glob Health*. 2024;14(3):549-560. doi:10.1007/S44197-024-00217-5/TABLES/4
5. Krysta W, Dudek P, Pulik Ł, Łęgosz P. Screening of Developmental Dysplasia of the Hip in Europe: A Systematic Review. *Children 2024, Vol 11, Page 97*. 2024;11(1):97. doi:10.3390/CHILDREN11010097
6. Kokavec M, Bialik V. Developmental dysplasia of the hip. Prevention and real incidence. 2007;108(6):251-254. Accessed August 4, 2025. https://pubmed.ncbi.nlm.nih.gov/17972535/
7. Kraus T, De Pellegrin M, Dubs B. DDH: Definition, Epidemiology, Pathogenesis, and Risk Factors. *Developmental Dysplasia of the Hip: From Early Sonographic Diagnosis to Effective Treatment*. Published online January 1, 2022:11-15. doi:10.1007/978-3-030-94956-3_3
8. Harsanyi S, Zamborsky R, Krajciova L, Kokavec M, Danisovic L. Developmental Dysplasia of the Hip: A Review of Etiopathogenesis, Risk Factors, and Genetic Aspects. *Medicina (B Aires)*. 2020;56(4):153. doi:10.3390/medicina56040153
9. Chen J, Zhang WB, He JZ, Zhang R, Cao YQ, Liu X. Developmental dysplasia of the hip: A special pathology. *Chinese Journal of Traumatology*. 2018;21(4):238-242. doi:10.1016/j.cjtee.2018.02.001
10. Wenger DR. Tachdjian's Pediatric Orthopaedics, 4th Edition. *Journal of Pediatric Orthopaedics*. 2008;28(8):891. doi:10.1097/bpo.0b013e31818ee3ad
11. Pediatric Orthopedics. *JAMA*. 2002;288(6):771. doi:10.1001/jama.288.6.771-jbk0814-5-1
12. Agostiniani R, Atti G, Bonforte S, et al. Recommendations for early diagnosis of Developmental Dysplasia of the Hip (DDH): Working group intersociety consensus document. *Ital J Pediatr*. 2020;46(1):1-7. doi:10.1186/S13052-020-00908-2/METRICS
13. O'Beirne J, Chlapoutakis K. Developmental Dysplasia of the Hip: From Early Sonographic Diagnosis to Effective Treatment. *Developmental Dysplasia of the*

Hip: From Early Sonographic Diagnosis to Effective Treatment. Published online January 1, 2022:1-155. doi:10.1007/978-3-030-94956-3/COVER
14. Sulaiman AR, Yusof Z, Munajat I, Lee NAA, Nik Z. Developmental Dysplasia of Hip Screening Using Ortolani and Barlow Testing on Breech Delivered Neonates. *Malays Orthop J*. 2011;5(3):13-16. doi:10.5704/moj.1111.008
15. Anderton MJ, Hastie GR, Paton RW. The positive predictive value of asymmetrical skin creases in the diagnosis of pathological developmental dysplasia of the hip. *The Bone & Joint Journal*. 2018;100-B(5):675-679. doi: 10.1302/0301-620x.100b5.bjj-2017-0994.r2
16. Lovell WW, Winter RB, Wenger DR. Pediatric Orthopedics—(2nd Ed.). *Journal of Pediatric Orthopaedics*. 1987;7(3):344. doi:10.1097/01241398-198705000-00023
17. Jacobino B de CP, Galvão MD, Silva AF da, Castro CC de. Using the Graf method of ultrasound examination to classify hip dysplasia in neonates. *Autops Case Rep*. 2012;2(2):5-10. doi:10.4322/acr.2012.018
18. Barrera CA, Cohen SA, Sankar WN, Ho-Fung VM, Sze RW, Nguyen JC. Imaging of developmental dysplasia of the hip: ultrasound, radiography and magnetic resonance imaging. *Pediatr Radiol*. 2019; 49(12):1652-1668. doi:10.1007/s00247-019-04504-3
19. Loh B, Woollett E. Update on the management of infant and toddler developmental dysplasia of the hip. *Aust J Gen Pract*. 2021;50(4):207-212. doi:10.31128/ajgp-07-20-5543
20. Elsharkawi KM, Barakat MS, Farahat AAK, Ahmed AARY, Bastawi RA. Role of magnetic resonance imaging in assessment of acetabular and femoral version in developmental dysplasia of the hip. *Radiol Bras*. Published online 2022. doi:10.1590/0100-3984.2021.0133
21. Placzek S, Bornemann R, Skoulikaris N. Development of the Hip Joint: Embryology and Anatomy of the Neonatal Hip Joint. *Developmental Dysplasia of the Hip: From Early Sonographic Diagnosis to Effective Treatment*. Published online January 1, 2022:3-9. doi:10.1007/978-3-030-94956-3_2
22. Clarke N. Paediatric Orthopaedics: A System of Decision-Making. *The Annals of The Royal College of Surgeons of England*. 2011;93(1):92-92. doi:10.1308/rcsann.2011.93.1.92b
23. Jamil K, Saharuddin R, Abd Rasid AF, Abd Rashid AH, Ibrahim S. Outcome of Open Reduction Alone or with Concomitant Bony Procedures for Developmental Dysplasia of the Hip (DDH). *Children*. 2022;9(8):1213. doi:10.3390/children9081213
24. Fu M, Xiang S, Zhang Z, et al. The biomechanical differences of rotational acetabular osteotomy, Chiari osteotomy and shelf procedure in developmental dysplasia of hip. *BMC Musculoskelet Disord*. 2014;15(1). doi:10.1186/1471-2474-15-47
25. Staheli LT, Chew DE. Slotted Acetabular Augmentation in Childhood and Adolescence. *Journal of Pediatric Orthopaedics*. 1992;12(5):569-580. doi:10.1097/01241398-199212050-00002
26. van de Sande MAJ, Melisie F. Successful Pavlik treatment in late-diagnosed developmental dysplasia of the hip. *Int Orthop*. 2012;36(8):1661-1668. doi:10.1007/s00264-012-1587-5

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