

Hip Growth Disturbances in Childhood

María Gabriela Miranda,^{***} Claudio A. Fernández^{**}

^{*}*Orthopedics and Traumatology Service, Hospital de Niños “Sor María Ludovica”, La Plata, Buenos Aires, Argentina*

^{**}*School of Medical Sciences, Universidad Nacional de La Plata, Buenos Aires, Argentina*

ABSTRACT

Growth disturbances of the proximal femur, described in the medical literature as avascular necrosis, osteochondritis, or post-reduction epiphysitis, are associated almost exclusively with the treatment of developmental dysplasia of the hip. Some of these disturbances can lead to later joint degeneration, with clinical, functional, and social implications. Various treatment-related factors have been identified. However, preexisting idiosyncratic, morphological, and vascular factors also determine specific risk profiles. This study aims to analyze the natural history of these disturbances and identify possible strategies to prevent them or reduce their sequelae. Understanding hip growth, development, and vascular supply is essential to understanding their pathogenesis. Diagnosis and treatment of developmental dysplasia of the hip during the neonatal period remain the gold standard, given the extremely low rates of treatment failure and residual abnormalities.

Keywords: Developmental dysplasia of the hip; femoral growth disturbance; natural history; children.

Level of Evidence: V

Alteraciones del crecimiento de la cadera en la infancia

RESUMEN

Las alteraciones del crecimiento del fémur proximal, referidas en la literatura médica como necrosis avascular, osteocondritis o epifisitis posreduccional, se relacionan casi exclusivamente con la terapéutica empleada en la displasia del desarrollo de la cadera. Algunas de ellas condicionan la ulterior degeneración articular con sus implicancias clínicas, funcionales y sociales. Se han individualizado una diversidad de factores relacionados con el tratamiento. Sin embargo, otras variables preexistentes idiosincrásicas, morfológicas y vasculares configuran perfiles de riesgo específicos. El objetivo de esta investigación es analizar la historia natural de estas alteraciones y establecer posibles formas de prevención o reducir sus secuelas. Una síntesis del crecimiento, el desarrollo y la vascularización de la cadera es imprescindible para la comprensión de la patogenia. El diagnóstico y el tratamiento de la displasia del desarrollo en el período neonatal siguen siendo el método de referencia, dado las ínfimas tasas de fracaso terapéutico y anomalías residuales.

Palabras clave: Displasia del desarrollo de la cadera; alteración del crecimiento femoral; historia natural; niños.

Nivel de Evidencia: V

INTRODUCTION

Proximal femoral growth disturbances (PFGDs) in childhood are morphological abnormalities involving the epiphysis, physis, femoral neck, or a combination of these structures. Although PFGDs are predominantly associated with the treatment of developmental dysplasia of the hip (DDH), they have also been observed in the contralateral, nondysplastic hip after immobilization.¹ The untreated hip may also show signs of dysplasia at skeletal maturity in 13%–40% of cases, consistent with the term “developmental dysplasia.”^{2,3} PFGDs may be transient and reversible or permanent and irreversible, with various deformities and joint degeneration that remain clinically silent for years, reflecting a discrepancy between radiographic and clinical findings.

Received on June 16th, 2026. Accepted after evaluation on July 7th, 2026 • Dr. CLAUDIO A. FERNÁNDEZ • claudioalfredofernandez619@gmail.com

 <https://orcid.org/0000-0003-2350-3885>

How to cite this article: Miranda MG, Fernández CA. Hip Growth Disturbances in Childhood. *Rev Asoc Argent Ortop Traumatol* 2026;91(5):510-518. <https://doi.org/10.15417/issn.1852-7434.2026.91.5.2411>

This study aims to analyze the natural history of PFGDs and identify ways to prevent or reduce their sequelae. In DDH, the primary driver of deformity lies in the acetabulum, whereas in PFGDs, the initial changes occur in the proximal femur and acetabular involvement is secondary. The terms avascular necrosis, post-reduction osteochondritis, and epiphysitis are used synonymously in the scientific literature. Histological uncertainty has led many investigators to favor the euphemism “proximal femoral growth disturbance.”^{1,4-6} Proposed etiopathogenic mechanisms include mechanical factors (excessive joint pressure) and ischemia, hypotheses supported by experimental studies in pigs.⁷ These mechanisms may also interact (Figure 1).⁸

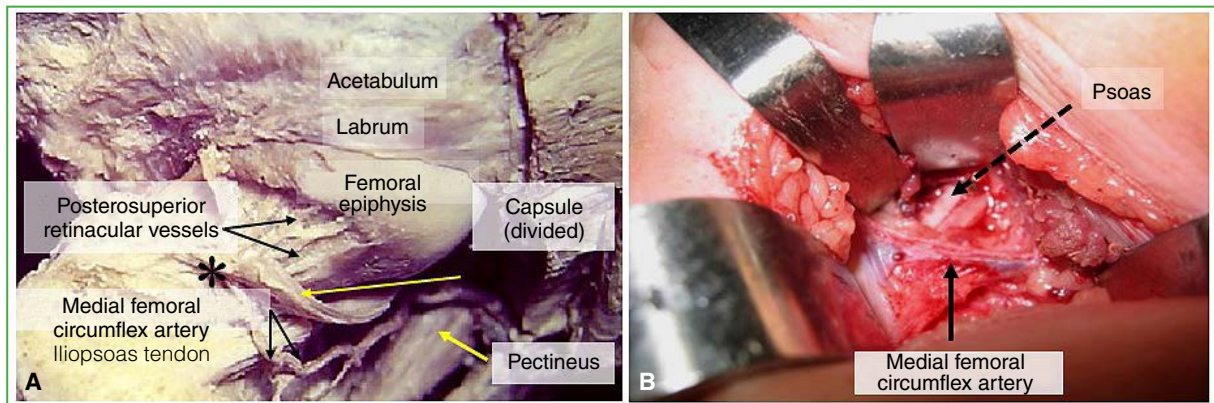


Figure 1. **A.** Dissection of the right hip in the inguinal region (cadaveric specimen). Note the relationship of the pectineus muscle and iliopsoas tendon to the medial circumflex femoral artery and its entry into the retrotrochanteric fossa (black asterisk). (Dissection by Prof. Rodolfo Cosentino, MD.) **B.** Corresponding intraoperative photograph showing the course of the medial circumflex femoral artery (solid arrow) and its relationship to the iliopsoas tendon (dashed arrow). The Farabeuf retractors at the apex are positioned anterior to the femoral epiphysis. Arterial compression may occur between the iliopsoas tendon and the pectineus muscle. In young children, the underdeveloped femoral neck makes the retinacular vessels more susceptible to compression by the labrum during extreme abduction and internal rotation.

The reported prevalence of PFGDs varies widely, from 0% to 73%, possibly reflecting differences in diagnostic rigor and follow-up duration. Numerous predisposing factors have been described: age >6 months at reduction, an unossified femoral head nucleus, greater cartilage volume, high dislocations, an inverted labrum, soft tissue interposition, nonconcentric reduction, preoperative traction, all surgical modalities, forceful reduction, immobilization in $\geq 60^\circ$ of abduction combined with internal rotation, prolonged immobilization, and surgery after failed treatment.^{1,9-11} In a prospective multicenter study of dysplastic hip dislocations, 20% of children already had epiphyseal abnormalities and femoral neck widening before treatment. During follow-up, 42% met the criteria for PFGDs, demonstrating that these abnormalities *are not iatrogenic* but pre-existing.⁶ Similarly, a *pretreatment* MRI study revealed *impaired perfusion and an immature microcirculatory pattern*.¹² Comparable findings have been reported with real-time contrast-enhanced ultrasound (CEUS; Optison®).¹³ The prevalence of PFGDs with devices such as the Pavlik harness, von Rosen splint, and Frejka pillow is *exceptionally low*, at 0%–1%. However, delaying treatment by several weeks or persisting with treatment after failed reduction raises the risk above 25%.^{2,11,12,14} Rates of PFGDs following closed reduction under general anesthesia and various surgical procedures range from 20% to 60%.^{3-6,8,11,14-16} Even without PFGDs, joint quality may deteriorate over time, as dysplasia cannot be regarded solely as a mechanical fetal disorder. Regardless of treatment, the risk of PFGDs is related to the high position of the proximal femur, underscoring the importance of classification systems with excellent intra- and interobserver reliability (Figures 2 and 3).¹⁷⁻²⁰

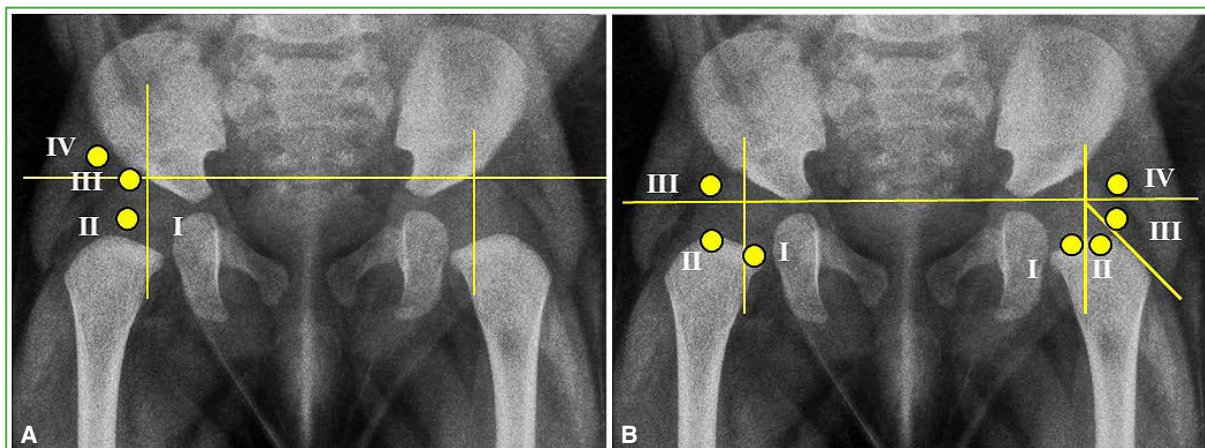


Figure 2. Topographic classifications of the unossified femoral epiphysis based on anteroposterior radiographs of both hips. In all three systems, grade I is normal, indicating that at least one-third of the femoral metaphysis lies medial to Perkins' line. **A.** Tönnis classification: a horizontal line through the acetabular roofs and the vertical Perkins' line. **B.** Right hip: Terjesen parameters, using the vertical Perkins' lines and the horizontal Hilgenreiner's line through the triradiate cartilages. The medial metaphyseal point indicates the epiphyseal position. Left hip: *International Hip Dysplasia Institute* (IHDI) method, using the resultant vector between the lines described in B. A midpoint on the metaphysis provides an objective reference for the epiphyseal position and, consequently, the level of dislocation.

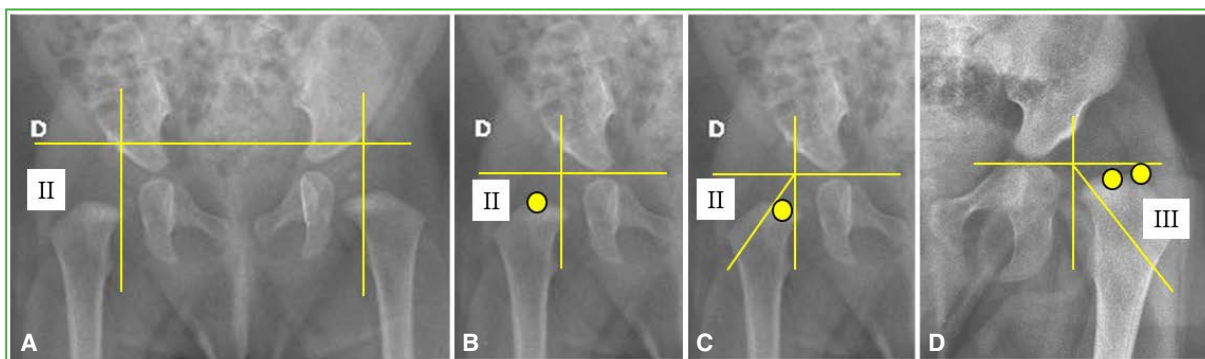


Figure 3. Anteroposterior hip radiographs of a 5-month-old girl showing grade II right hip dislocation according to all three classification systems. **A.** Tönnis. **B.** Terjesen. **C.** International Hip Dysplasia Institute (IHDI). Note the metaphyseal reference point in the latter two systems. **D.** Anteroposterior hip radiograph of a 7-month-old girl showing grade III dislocation according to the Terjesen and IHDI classifications.

HIP GROWTH AND DEVELOPMENT

The proximal femur and acetabulum arise from the same embryonic mesenchyme. During the eleventh week of intrauterine life, the fully formed hip begins to differentiate into cartilage, following the pattern of endochondral ossification, supplemented after birth by periosteal bone apposition. In the newborn, both joint components consist of hyaline cartilage (Figure 4).¹

The femoral epiphysis and greater trochanteric apophysis are *spherical chondroepiphyses*. Their growth is *centripetal*, driven by the metabolic activity of peripheral epiphyseal chondrocytes, whereas ossification is *centrifugal and radial*, proceeding from the center toward the periphery. The proximal femoral growth plate is *linear* and resembles an inverted L with three segments: the physeal or discoid segment, perpendicular to the femoral neck axis; the cervicotrochanteric segment; and the isthmus, which connects the other two and contributes to femoral neck thickness.¹ The calcification front of this configuration may occasionally be visible on radiographs as Harris lines. Interruption or distortion of these lines is considered *predictive* of PFGDs (O'Brien's sign) (Figure 5).¹

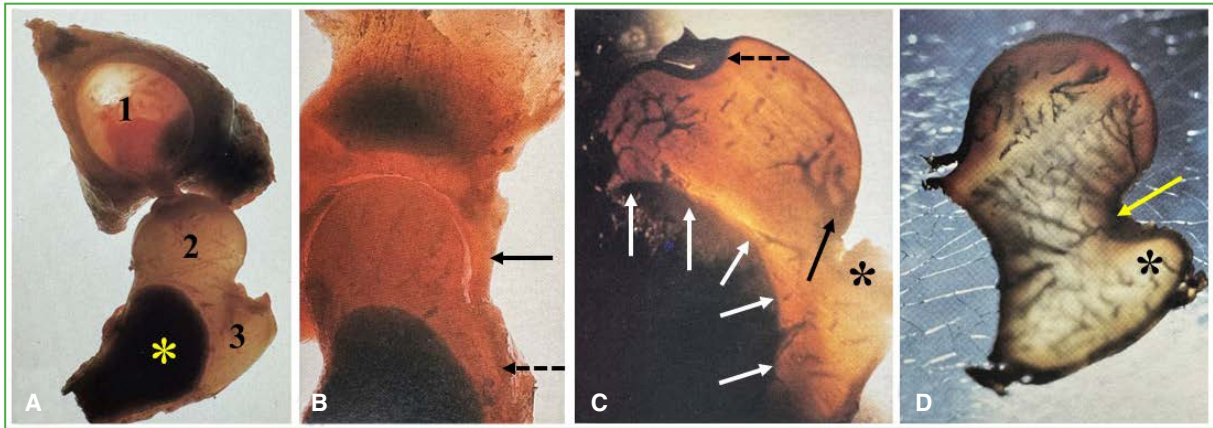


Figure 4. A-C. Transillumination images of the hip of a neonatal cadaveric specimen at 38 weeks' gestation, showing the fibro-hyaline composition of both joint components. **A.** Disarticulated specimen: 1. Acetabulum. 2. Femoral chondroepiphysis. 3. Greater trochanteric chondroepiphysis. 4. Osseous femoral metaphysis (yellow asterisk). **B.** Hip in situ. Capsule and labrum (black arrow), underdeveloped femoral neck, and trochanteric region at the entry site of the posterosuperior retinacular vessels (dotted arrow). **C.** Enlarged view of B. White arrows indicate the physis. From left to right: discoid cartilage, isthmus, and femoral neck. Greater trochanter and retrotrochanteric fossa (black asterisk), entry of the posterosuperior retinacular arteries (black arrow), and artery of the ligamentum teres (dotted arrow). **D.** Apical view of the femoral epiphysis and neck of a 25-week fetus. Greater trochanter (black asterisk) and the previously described retinacular vessels forming a rich branching network (yellow arrow). (Reproduced from: Teot L, Deschamps F. *Atlas d'Échographie de la hanche dans la première année de la vie*, 1987. With permission from Sauramps Medical.)

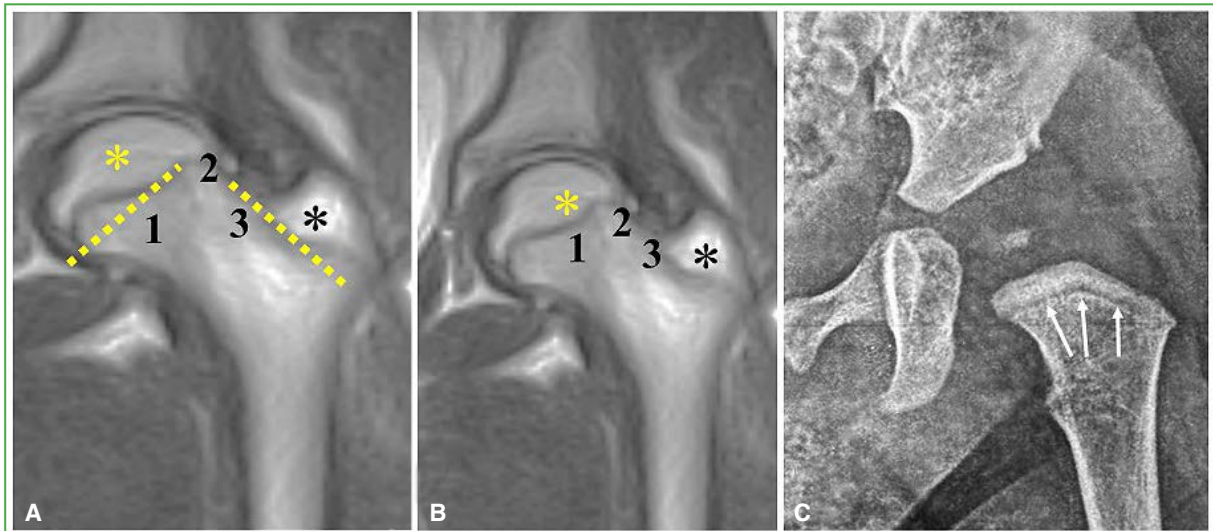


Figure 5. A and B. Coronal T1-weighted MRI of the hip of a 4-year-old child. Yellow asterisk: femoral chondroepiphysis; black asterisk: greater trochanter. The proximal femoral physis forms an inverted L with three continuous segments: 1. Femoral discoid cartilage. 2. Isthmus. 3. Femoral neck. **C.** Anteroposterior radiograph of the left hip of a 4-month-old child in slight internal rotation. Arrows indicate Harris lines that mirror the morphology of the physis.

Physiological physeal closure begins in the *lateral, superior, and slightly posterior* regions of the physis and then progresses in all directions.^{21,22} The blood supply comes from an extracapsular vascular network at the base of the femoral neck, supplied by the circumflex arteries. The medial circumflex artery passes between the iliopsoas muscle, enters the joint through the retrotrochanteric fossa, and gives rise to the retinacular vessels, particularly the posterosuperior vessels, which provide 80% of the blood supply to the epiphysis, isthmus, and much of the femoral metaphysis. The anterior retinacular branches arising from the lateral circumflex artery provide a supplementary

supply. This vascular network forms a *bipartite* system: the retinacular vessels perfuse the epiphyseal ossification center and physis for the synthesis of ground substance and osteoid, while the metaphyseal system contributes to substrate mineralization and bone resorption according to the Hueter–Volkman law. *This vascular network does not follow a fixed pattern; it shows substantial anatomical variation according to sex, ethnicity, the number and diameter of the arteries, and the variable presence of intra-articular vascular rings.*

Classification and characterization of PFGDs

Several authors have proposed classifications of PFGDs. In chronological order, these include: Salter and Kostuk,⁷ Bucholtz and Ogden,² Kalamchi and MacEwen,²³ and Kruczynski²⁴ (Figure 6). All have moderate interobserver reliability.⁶ Given their similarity and widespread use in the specialized literature, the Kalamchi–MacEwen and Bucholtz–Ogden classifications will be considered together in this analysis. Type I is characterized by *delayed ossification and an appearance of epiphyseal fragmentation*. Although reversible, these changes may precede lesions with a poorer prognosis (Figure 7).

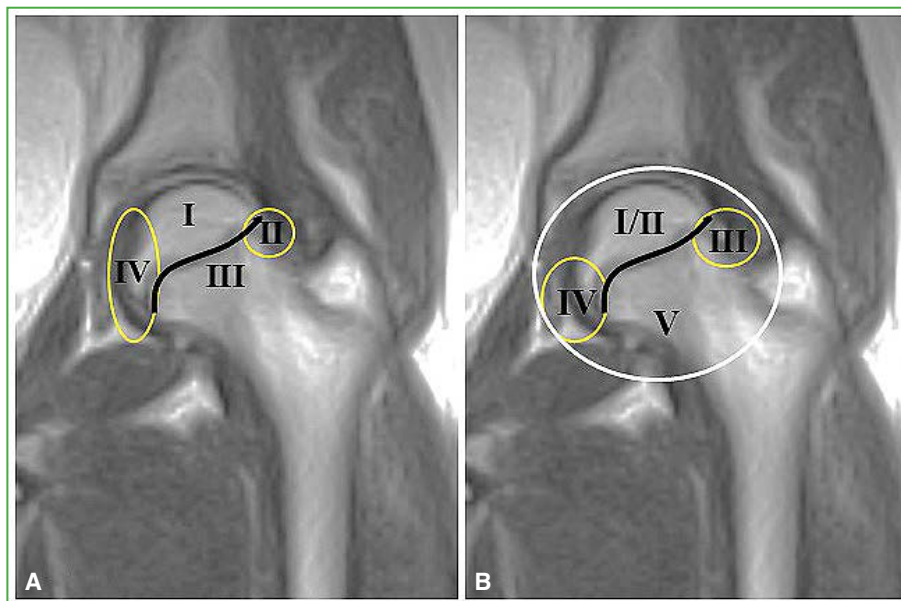


Figure 6. Coronal T1-weighted MRI of the hip of a 4-year-old child illustrating the classifications. **A.** Bucholtz and Ogden (combined with Kalamchi and MacEwen). I. Femoral epiphyseal fragmentation. II. Isthmic epiphysiodesis. III. Severe physeal damage. IV. Medial lesion. **B.** Kruczynski. The difference lies in the numerical assignments. Type V represents global damage, indicated by the white circle.

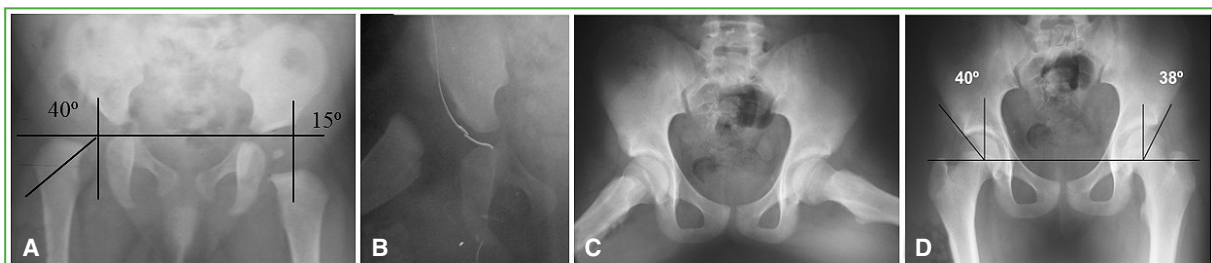


Figure 7. Anteroposterior and lateral hip radiographs. **A and B.** A 9-month-old girl with grade III right hip dislocation according to the Terjesen and International Hip Dysplasia Institute (IHDI) classifications and an uncalcified epiphyseal ossification center. Previous treatment consisted of an unsuccessful trial of a Pavlik harness, followed by unsuccessful closed reduction and hip spica casting under general anesthesia. Hilgenreiner's angle (alpha): 40°. **C and D.** Anteroposterior and lateral hip radiographs at 13 years of age, showing the outcome of open reduction through a medial approach using the Weinstein–Ponseti technique. Severin grade IA. Absence or fragmentation of the femoral epiphysis may be transient and reversible.

The type II pattern has a prevalence of 25% and is defined by *epiphysiodesis or a lateral physeal bar*, which causes valgus tilt of the epiphysis relative to the femoral neck and may lead to secondary subluxation (Figure 8A). One study evaluated 58 hips in 48 patients with type II PFGDs treated by closed reduction ($n = 36$) or surgery through an inguinal approach ($n = 22$), with a follow-up of 21 years (range, 10–55). The abnormality became radiographically apparent at 10 years of age (range, 4–14). Outcomes were classified as Severin groups I/II in 59% of hips; the remainder had unsatisfactory outcomes (Table).^{4,24}

Table. Severin radiographic classification

I. Normal hip	a. CEA $>19^\circ$ at ages 6–13 years
	CEA $>25^\circ$ from age 14 years onward
	b. CEA 15° – 19° at ages 6–13 years
	CEA 20° – 25° from age 14 years onward
II. Moderate deformity	a and b. Same CEA values as in group I
III. Dysplasia without subluxation	CEA $<15^\circ$ at ages 6–13 years and $<20^\circ$ at ages >14 years
IV. Subluxation	Moderate: CEA positive or zero
	Severe: CEA negative
V. Dislocation	Femoral head articulating with the upper part of the original acetabulum
VI. Redislocation	Femoral head in a neoacetabulum

CEA = cervicoepiphyseal angle.²⁵

Another study included 155 dislocated hips treated by closed reduction at a mean age of 15 months. Type II PFGDs were present in 23% and were detected at a mean age of 8.9 years (range, 3–12). Sixty-six percent were classified as Severin grades I/II, while the remainder had suboptimal outcomes (Figures 8A and 9).²⁵

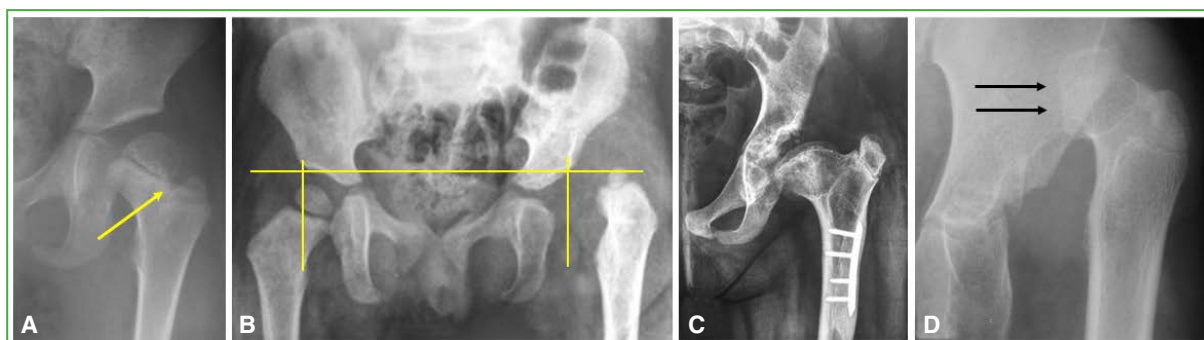


Figure 8. Anteroposterior radiographs. **A.** A 6-year-old boy with a lesion of the physeal isthmus following joint infection (yellow arrow), resulting in valgus tilt of the epiphysis. Type II proximal femoral growth disturbance. **B.** A 20-month-old girl with a previously undiagnosed Tönnis grade III left hip dislocation. Treatment consisted of open reduction through a Smith–Petersen approach, adductor longus tenotomy, Salter osteotomy, and femoral derotation and shortening osteotomies. **C.** Type III proximal femoral growth disturbance with evident dysplasia, coxa vara, and relative elevation of the greater trochanter. **D.** Preoperative radiograph of a previously untreated 7-year-old girl showing a type IV proximal femoral growth disturbance.



Figure 9. A girl aged 10 years and 10 months presenting with a limp, a positive Trendelenburg sign, and thigh pain. Coronal images of both hips. **A.** Radiograph showing valgus tilt of both epiphyses and isthmus epiphysiodesis of both femoral necks, defining a type II proximal femoral growth disturbance according to Bucholz and Ogden/Kalamchi and MacEwen, or type III according to Kruczynski (black arrows). Reimers' migration percentage is 20%. Alsberg's angle, which describes the angular relationship between the physis and femoral shaft, is 90° (reference value, 65°). Note the crossover sign of the acetabular lines, suggesting retroversion (yellow arrows). **B.** Corresponding CT and MRI findings showing the eccentric physal bridge at the isthmus (yellow arrows).

This phenomenon appears to reproduce physiological proximal femoral physal closure *prematurely*.²²

Type III PFGDs have a prevalence of 14%–30% and are the most damaging deformities, causing progressive joint and functional deterioration. They involve *severe epiphyseal and physal damage*, leading to reduced femoral neck growth and varus deformity, lower limb shortening, relative elevation of the greater trochanter with gluteus medius dysfunction, and coxa magna. Severe ischemia is the presumed cause (Figures 8B and C, 10A-C).²

In a study that followed 29 hips in 22 patients over four decades, the abnormality became radiographically visible a mean of 10 months (range, 5–19) after closed reduction or surgery through an inguinal approach. High dislocations (Tönnis grade IV) had an odds ratio of 3.43 for joint deterioration compared with grades II and III. At skeletal maturity, 90% of hips were classified as Severin grades III/IV. By 39 years of age, 32% of patients had undergone total hip replacement (Table).^{5,24}

In light of these findings, some surgeons opted for an anterolateral approach combined with femoral and pelvic osteotomies. The results were also disappointing, however, as 32% developed type II/III PFGDs (Figure 7B and C).¹⁵ Thus, type III PFGD represents the worst treatment outcome, regardless of the method used. Type IV is a medial epiphyseal–metaphyseal defect characteristic of longstanding dislocations or subluxations and is observed *before* any treatment (Figure 8D). Finally, 25% of cases show unclassifiable or mixed patterns (Figure 10D and E).

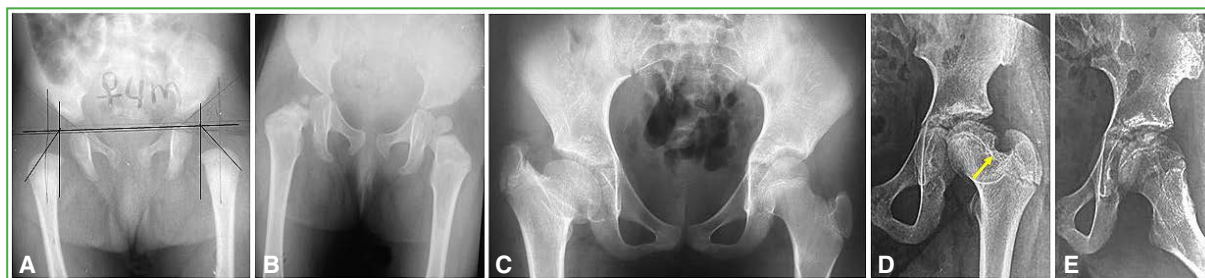


Figure 10. AP and lateral radiographs of both hips. **A.** A 4-month-old girl with bilateral grade II dysplasia according to the Terjesen and *International Hip Dysplasia Institute* (IHDI) classifications, treated with closed reduction and immobilization. **B.** Follow-up at 10 months showing epiphyseal hypoplasia and an apparently underdeveloped femoral neck. **C.** At 30 years of age, coxa vara with relative elevation of the greater trochanter and reversal of its relationship to the joint. These are classic sequelae of a type III proximal femoral growth disturbance according to Bucholz and Ogden/Kalamchi and MacEwen. **D.** A 13-year-old girl with a history of Tönnis grade IV left hip dislocation. After unsuccessful treatment with a Pavlik harness, open reduction was performed at 5 months of age through a Smith–Petersen approach. Mixed proximal femoral growth disturbance. The anteroposterior radiograph clearly shows a type II lesion (yellow arrow indicating epiphysiodesis), while the lateral view shows a shortened, widened femoral neck and evident coxa magna, consistent with type III according to Bucholz and Ogden/Kalamchi and MacEwen.

CONCLUSIONS

The gold standard for preventing PFGDs or mitigating their sequelae is neonatal or perinatal diagnosis and treatment of DDH. Beyond the epidemiological and treatment-related variables associated with DDH, *individual anatomical characteristics* (both morphological and vascular) also predispose patients to PFGDs, as demonstrated by advances in imaging technology and recent clinical research. Following children throughout growth is essential for a definitive and accurate assessment of the outcome.

Conflicts of interest: The authors declare no conflicts of interest.

M. G. Miranda ORCID ID: <https://orcid.org/0000-0003-4949-9407>

REFERENCES

1. Weinstein SL, Dolan LA. Proximal femoral growth disturbance in developmental dysplasia of the hip: what do we know? *J Child Orthop* 2018;12(4):331-41. <https://doi.org/10.1302/1863-2548.12.180070>
2. Bucholz R, Ogden JA (eds.). *Patterns of ischemic necrosis of the proximal femur in nonoperative treated congenital hip disease*. St. Louis: C.V. Mosby; 1978.
3. Kobayashi D, Satsuma S, Kuroda R, Kurosaka M. Acetabular development in the contralateral hip in patients with unilateral developmental dysplasia of the hip. *J Bone Joint Surg Am* 2010;92(6):1390-7. <https://doi.org/10.2106/JBJS.I.00840>
4. Kim HW, Morcuende JA, Dolan LA, Weinstein SL. Acetabular development in developmental dysplasia of the hip complicated by lateral growth disturbance of the capital femoral epiphysis. *J Bone Joint Surg Am* 2000;82(12):1692-700. <https://doi.org/10.2106/00004623-200012000-00002>
5. Fernandez CA, Dolan LA, Weinstein SL, Morcuende JA. Natural history of type III growth disturbance after treatment of developmental dislocation of the hip. *Iowa Orthop J* 2008;28:27-35. PMID: 19223945
6. England P, Schaeffer E, Price C, Mulpuri K, Sankar WN; Global Hip Dysplasia Registry. Proximal femoral growth alterations can be seen prior to treatment of developmental dysplasia of the hip: a multicenter cohort study. *J Pediatr Orthop Soc North Am* 2024;5(3):669. <https://doi.org/10.55275/JPOSNA-2023-669>
7. Salter RB, Kostuik J, Dallas S. Avascular necrosis of the femoral head as a complication of treatment for congenital dislocation of the hip in young children: a clinical and experimental investigation. *Can J Surg* 1969;12(1):44-61. PMID: 5762671
8. Morcuende JA, Meyer MD, Dolan LA, Weinstein SL. Long-term outcome after open reduction through an anteromedial approach for congenital dislocation of the hip. *J Bone Joint Surg Am* 1997;79(6):810-7. <https://doi.org/10.2106/00004623-199706000-00002>
9. Wu J, Yuan Z, Li J, Zhu M, Canavese F, Xun F, et al. Does the size of the femoral head correlate with the incidence of avascular necrosis of the proximal femoral epiphysis in children with developmental dysplasia of the hip treated by closed reduction? *J Child Orthop* 2020;14(3):175-83. <https://doi.org/10.1302/1863-2548.14.190176>
10. Bian Z, Guo Y, Lyu X, Zhu Z, Yang Z, Wang Y. Risk factors for avascular necrosis after closed reduction for developmental dysplasia of the hip. *J Pediatr Orthop* 2022;42(9):467-73. <https://doi.org/10.1097/BPO.0000000000002228>
11. Gahleitner M, Pisecky L, Gotterbarm T, Högl W, Luger M, Klotz MC. Long-term results of developmental hip dysplasia under therapy with Pavlik harness. *J Pediatr Orthop* 2024;44(3):135-40. <https://doi.org/10.1097/BPO.0000000000002575>
12. Wu J, Yuan Z, Li J, Zhu M, Canavese F, Fuxing X, et al. Does the vascular development of the femoral head correlate with the incidence of avascular necrosis of the proximal femoral epiphysis in children with developmental dysplasia of the hip treated by closed reduction? *J Child Orthop* 2021;15(4):395-401. <https://doi.org/10.1302/1863-2548.15.210059>

13. Matheney T, Shore B, Miller P, Barnewolt C, Kim YJ, Paltiel H. Comparison of contrast-enhanced ultrasound and contrast-enhanced magnetic resonance imaging in the assessment of infant hip perfusion and prediction of proximal femoral growth disturbance following closed and open reduction for developmental dysplasia of the hip: a preliminary study. *J Pediatr Orthop* 2024;44(4):e335-43. <https://doi.org/10.1097/BPO.000000000>
14. Tegnander A, Holen KJ, Anda S, Terjesen T. Good results after treatment with the Frejka pillow for hip dysplasia in newborns: a 3-year to 6-year follow-up study. *J Pediatr Orthop B* 2001;10(3):173-9. PMID: 11497357
15. Wu Q, Li Y, Lin Y, Sun X, Ma H, Sun J, et al. Risk factor analysis of femoral avascular necrosis after operation for Tönnis grade IV developmental dysplasia of the hip. *Int Orthop* 2024;48(4):1011-6. <https://doi.org/10.1007/s00264-023-05996-3>
16. Sibiński M, Synder M. Lateral growth disturbances of the capital femoral epiphysis after nonoperative treatment of late developmental dislocation of the hip: thirty-five cases followed to skeletal maturity. *J Pediatr Orthop* 2006;26(3):307-9. <https://doi.org/10.1097/01.bpo.0000217731.46911.e6>
17. Tönnis D (ed.). *Congenital hip dislocation: avascular necrosis*. New York: Thieme-Stratton; 1982.
18. Narayanan U, Mulpuri K, Sankar WN, Clarke NM, Hosalkar H, Price CT, et al. Reliability of a new radiographic classification for developmental dysplasia of the hip. *J Pediatr Orthop* 2015;35(5):478-84. <https://doi.org/10.1097/BPO.0000000000000318>
19. Terjesen T, Horn J. Prognostic value of severity of dislocation in late-detected developmental dysplasia of the hip. *J Child Orthop* 2020;14(4):266-72. <https://doi.org/10.1302/1863-2548.14.200079>
20. Singh KA, Ganjwala D, Gupta P, Vazhayil Kottamttavida I, Varma M, Shah H. Reliability of three radiologic classifications for the severity of developmental dysplasia of the hip in children older than four years. *J Pediatr Orthop* 2022;42(1):23-9. <https://doi.org/10.1097/BPO.0000000000002004>
21. Struijs PA, Oostra RJ, van Rijn RR, Besselaar PP. Abnormal growth of the proximal femur due to apophyseal-epiphyseal coalescence resulting in coxa valga: report of two cases in adolescents. *Acta Orthop* 2011;82(4):507-9. <https://doi.org/10.3109/17453674.2011.584210>
22. Dvonch VM, Bunch WH. Pattern of closure of the proximal femoral and tibial epiphyses in man. *J Pediatr Orthop* 1983;3(4):498-501. <https://doi.org/10.1097/01241398-198309000-00015>
23. Kalamchi A, MacEwen GD. Avascular necrosis following treatment of congenital dislocation of the hip. *J Bone Joint Surg Am* 1980;62(6):876-88. PMID: 7430175
24. Kruczyński J. Avascular necrosis after nonoperative treatment of developmental hip dislocation: prognosis in 36 patients followed 17-26 years. *Acta Orthop Scand* 1995;66(3):239-44. <https://doi.org/10.3109/17453679508995532>
25. Severin E. Contribution to the knowledge of congenital dislocation of the hip joint: late results of closed reduction and arthrographic studies of recent cases. *Acta Chir Scand* 1941;63:1-142.