Pediatric Hip Disorders

Embryological Origins, Classifications, and Comprehensive Clinical Profiles of DDH, Perthes, and SCFE

Prof. Omar Samarah

JUH

2026

Growth and Development of the Hip

- Acetabulum and proximal femur develops from 1 cartilage
- At 11th week of gestation the hip joint is formed
- One growth plate is formed and developed into 3 components :
    1. Physeal plate of femoral head
    2. Growth plate of Greater trochanter
    3. Femoral neck isthmus growth plate

Pediatric Hip Etiology Matrix

Category	Pathology Examples	Primary Mechanical Defect	Diagnostic Age Range
Congenital	PFFD, Teratologic Hip	Intrauterine development arrest	At Birth / Infancy
Developmental	DDH, LCPD, SCFE	Physeal or structural growth defect	Infancy through Adolescence
Syndromic	Down Syndrome, Marfan	Generalized capsule laxity	Early Childhood
neurological	Cerebral Palsy spasticity	Severe muscle force imbalance	Variable with ambulation

Pediatric Hip Etiology Matrix

Developmental	DDH, LCPD, SCFE	Physeal or structural growth defect	Infancy through Adolescence

Developmental Dysplasia (DDH)

A systematic review of the DDH spectrum: Pathoanatomy, clinical maneuvers, imaging, treatment protocols, and complications.

Largest determinant of acetabular development is

Reduced femoral head

DDH: Pathological Definition

Spectrum of Instability
DDH is a spectrum of progressive structural abnormal relationships between the femoral head and the acetabulum.

This ranges from subluxation (partial displacement) to complete dislocation. It is highly associated with intrauterine crowding, breech presentation, and female sex.

Pathoanatomy of Dysplasia
Without stable, concentric femoral head containment, the acetabular roof becomes shallow, vertical, and hypoplastic.

Soft tissue barriers (pulvinar fat hypertrophy, ligamentum teres thickening, inverted limbus) develop, blocking reduction.

Risk Factors

- Ligamentous Laxity
- Prenatal Position
- Postnatal Position
- Female Child
- Family History
- First Born Baby
- Oligohydramnios
- Packing Disorders

Risk Factors

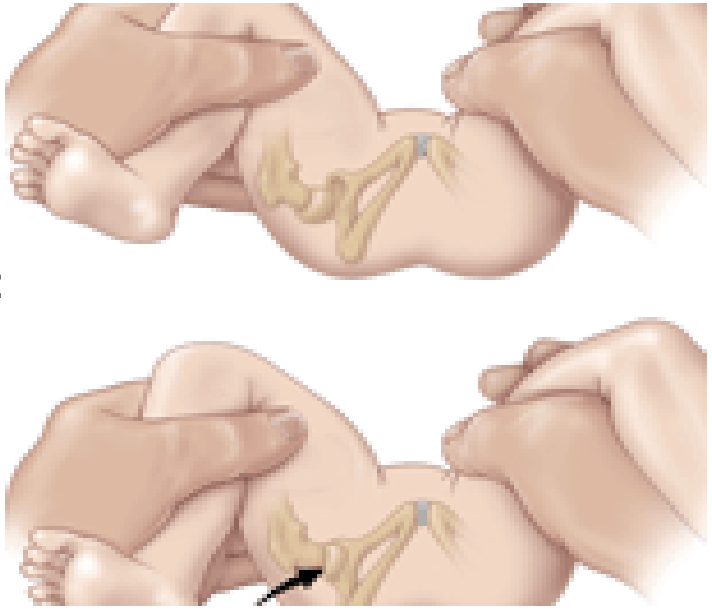
- Female Child
- Family History
- First Born Baby

DDH: Clinical Presentation Signs

Barlow Provocative Test:

1

Ortolani Reductive Test

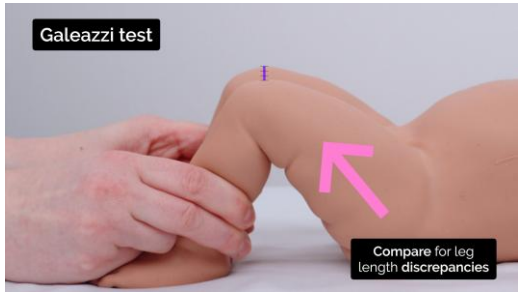


2

3

4

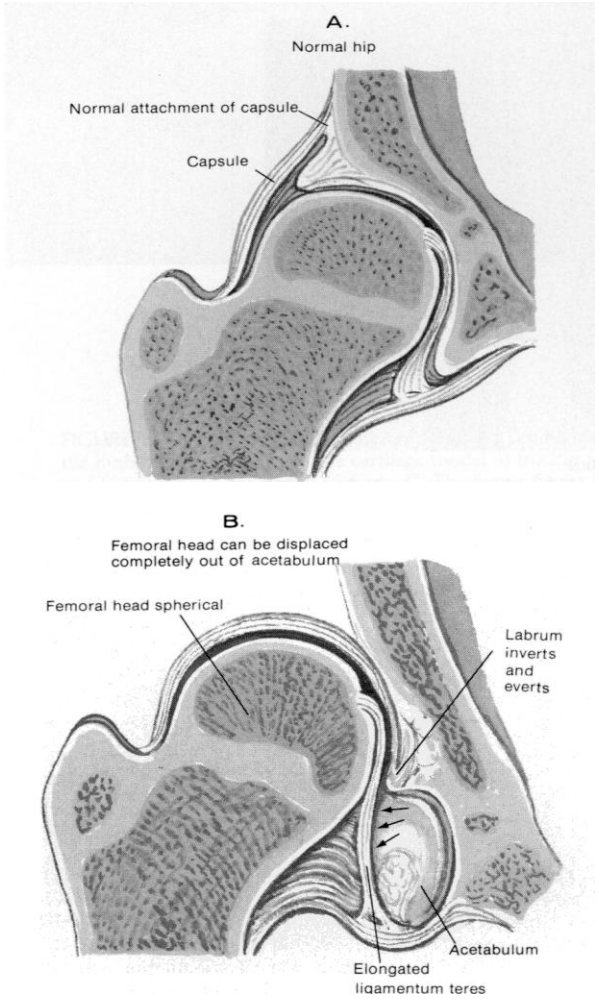
Galeazzi Discrepancy:



Late Walkers (Trendelenburg gait)

Pathological Anatomy

- Lax capsule results in lateral subluxation
- Eventual dislocation
- Pulvinar fills floor of acetabulum
- Acetabulum becomes more shallow
- Ligamentum teres elongates
- Femoral head becomes oval with medial flattening
- Transverse acetabular ligament hypertrophies



DDH: Diagnostic Ultrasonography

Ultrasound (Age ‹ 4 Months)

Ultrasonography is the gold standard before the secondary femoral head ossification center appears.
The Graf classification evaluates the bony and cartilaginous roof coverage of the acetabulum:
Alpha angle (or): Measures bony roof slope.
Normal coverage is:

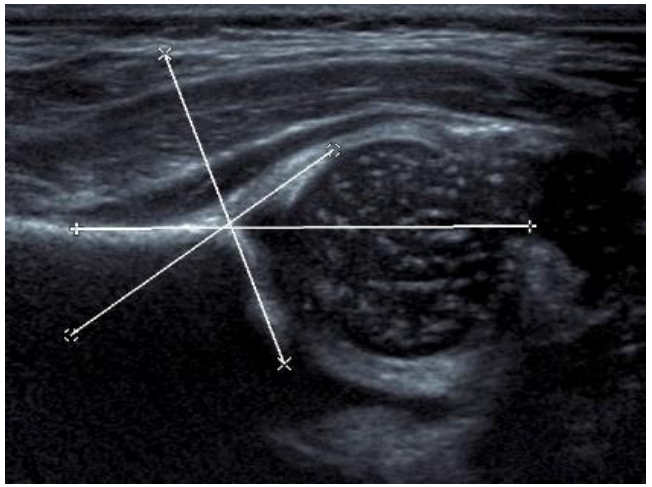
a >60°

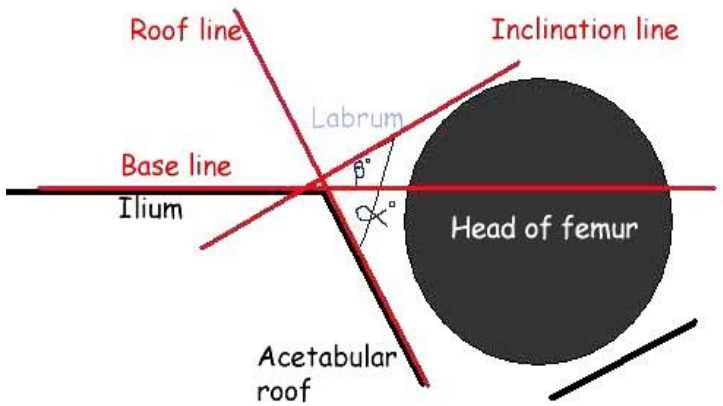
Type	Maturity	Bony roof	Bony angle	Bony rim	Cartilage roof	β-angle	Age
Type I	mature	good	$\alpha \geq 60^{o}$	sharp	good coverage femoral head	Ia = β < 55° Ib = β > 55°	All
Type II a+	immature but appropriate for age	adequate	50-59°	blunt	coverage femoral head		< 3 mo
Type II a-	immature and inappropriate for age	deficient	50-59°	rounded	coverage femoral head		< 3 mo
Type II b	delay in development	deficient	50-59°	rounded	coverage femoral head		> 3 mo
Type II c	stable or unstable	severely deficient	43-49°	rounded / flat	still coverage femoral head	β < 77°	All
Type D	decentring hip	severely deficient	43-49°	rounded / flat	displaced	β > 77°	All
Type III	eccentric hip	poor	< 43°	flat	labrum pressed upwards		All
Type IV	eccentric hip	poor	< 43°	flat	labrum pressed downwards		All

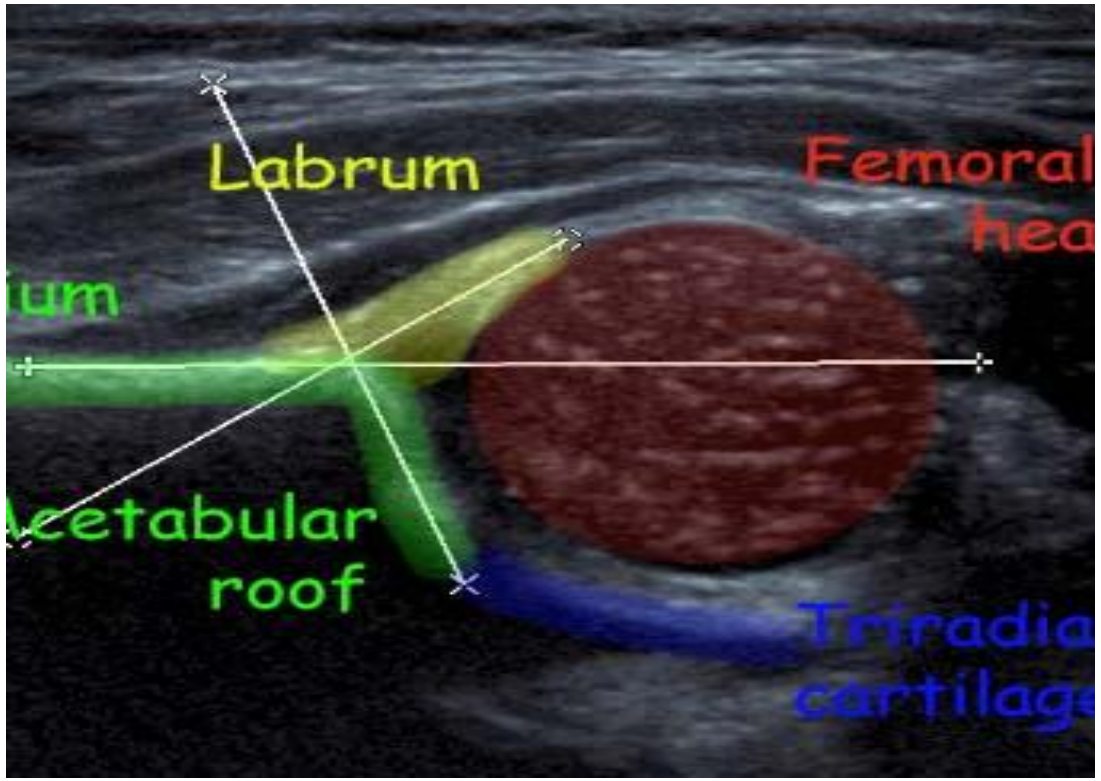
Radiographic findings

Ultrasonography

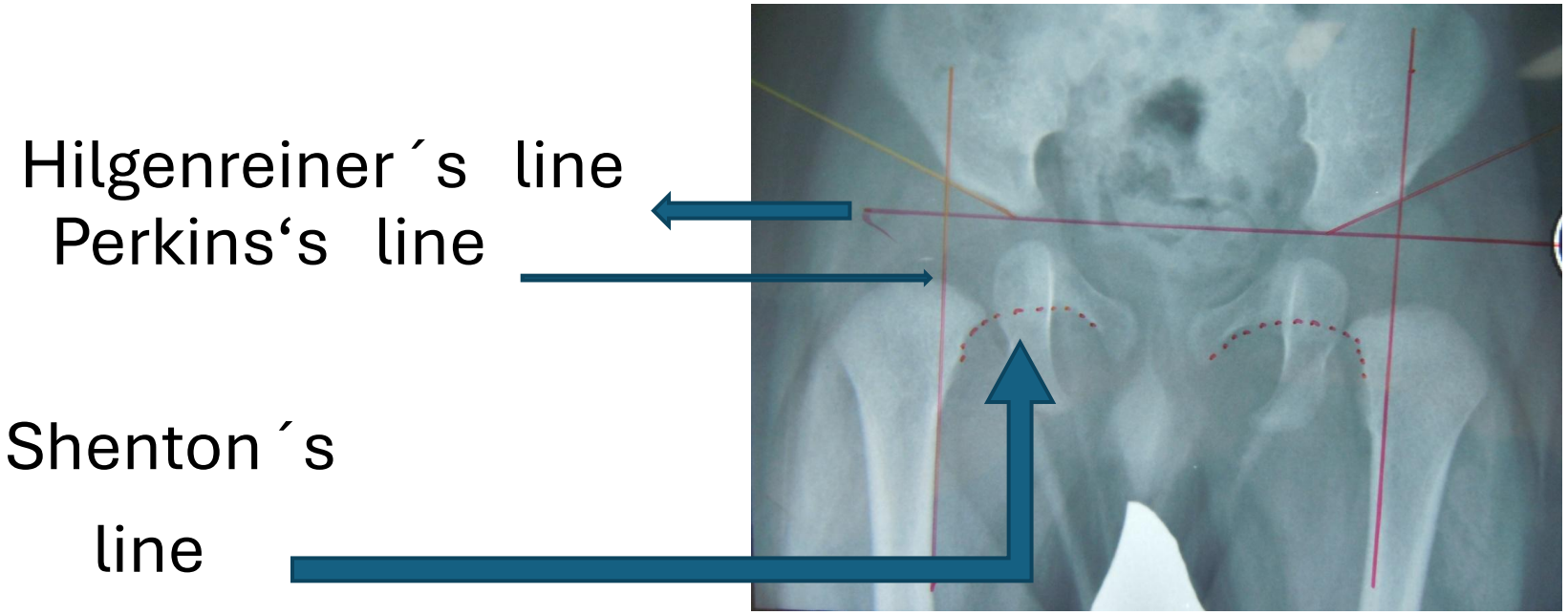
Coronal image



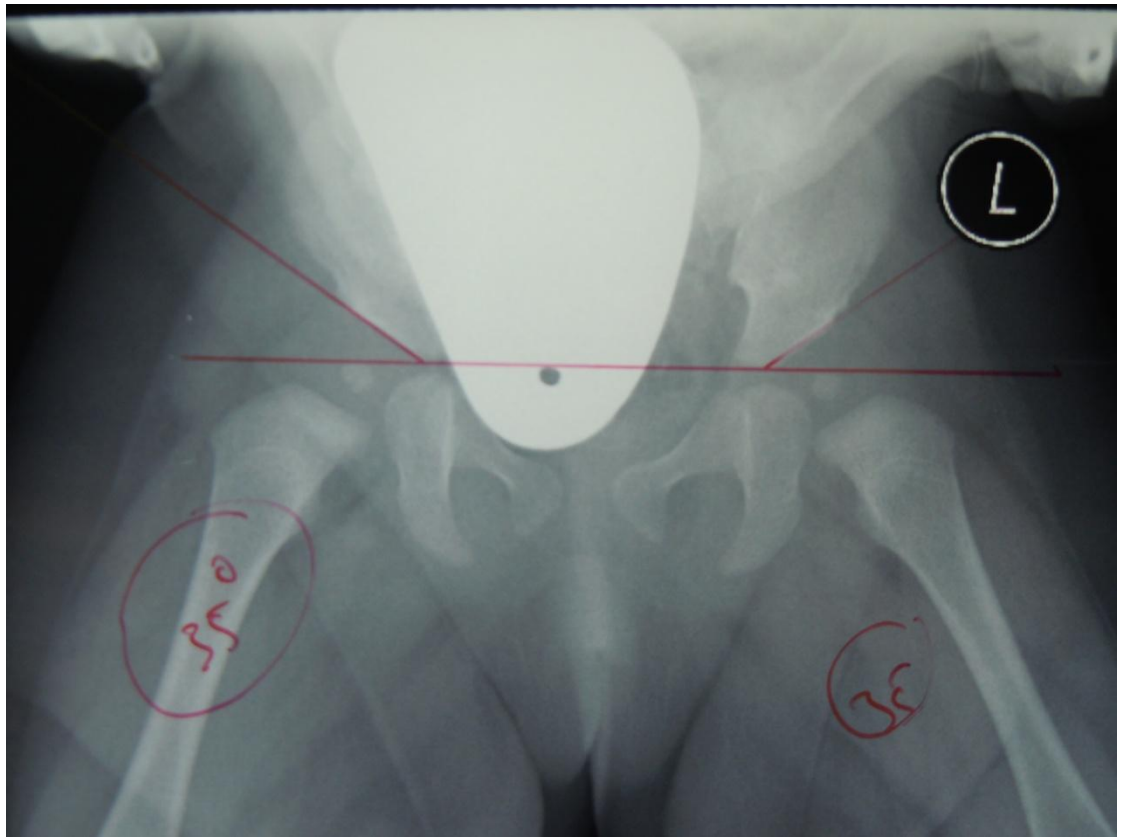




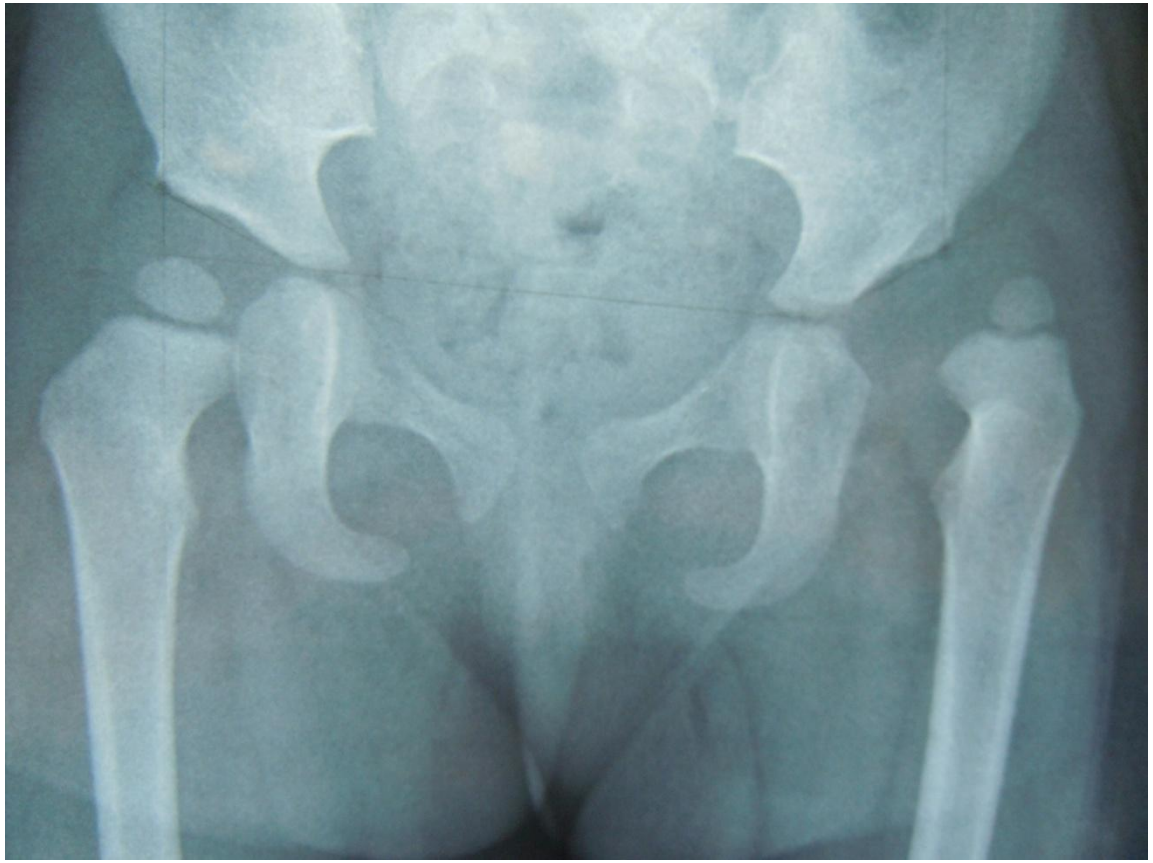
Normal Hip Radiography



Acetabular Dysplasia



Dislocation



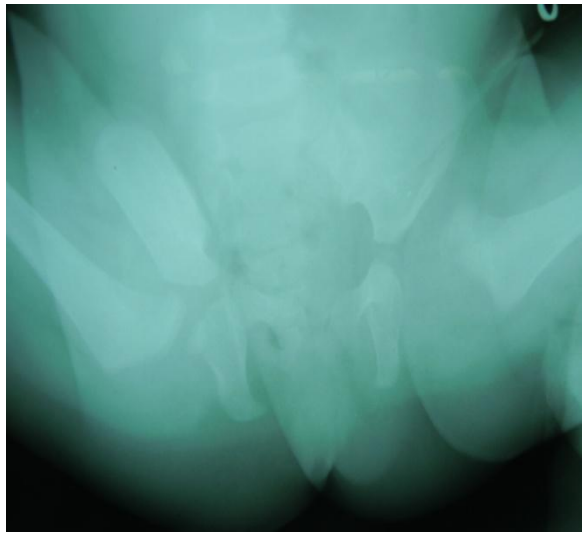
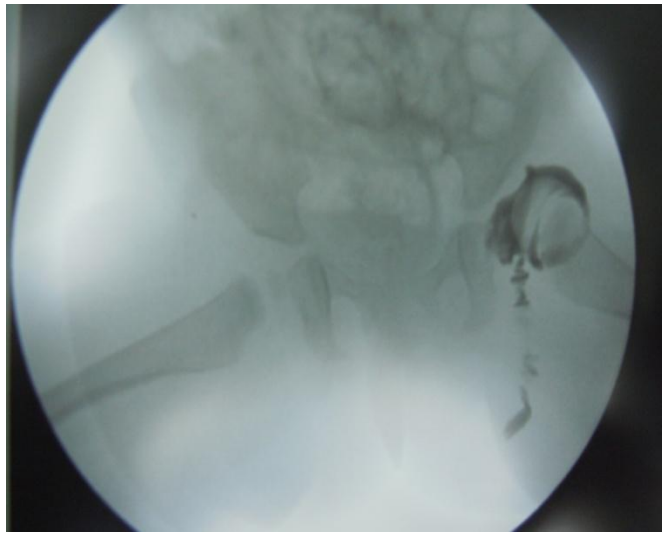
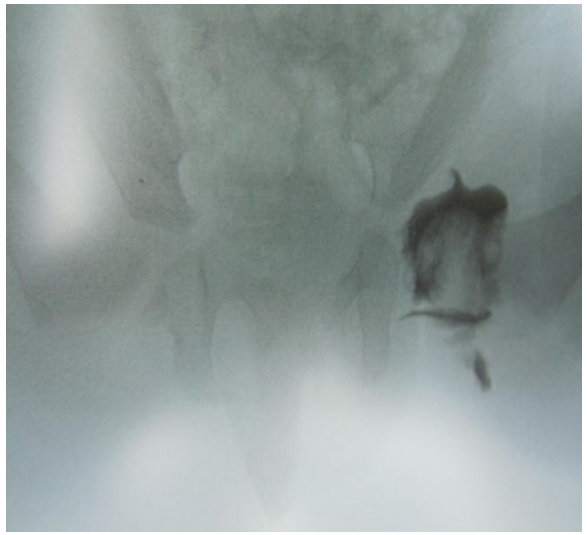
The goals in the treatment of DDH are to obtain a ***concentric reduction*** *and then maintain that reduction to provide an optimal environment for femoral head and acetabular development.*

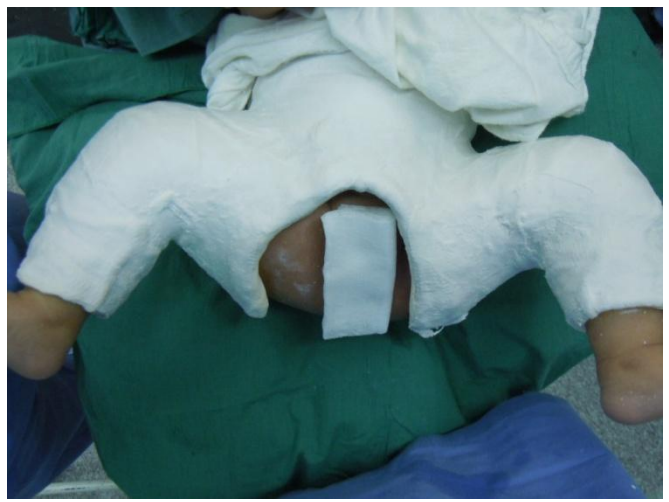
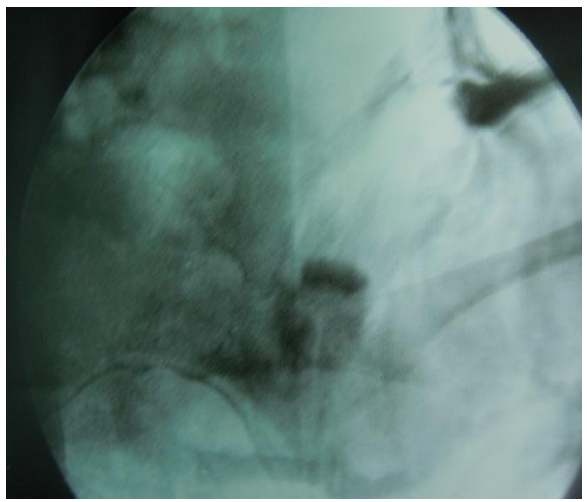
0 – 6 month-age infant

Abduction Brace

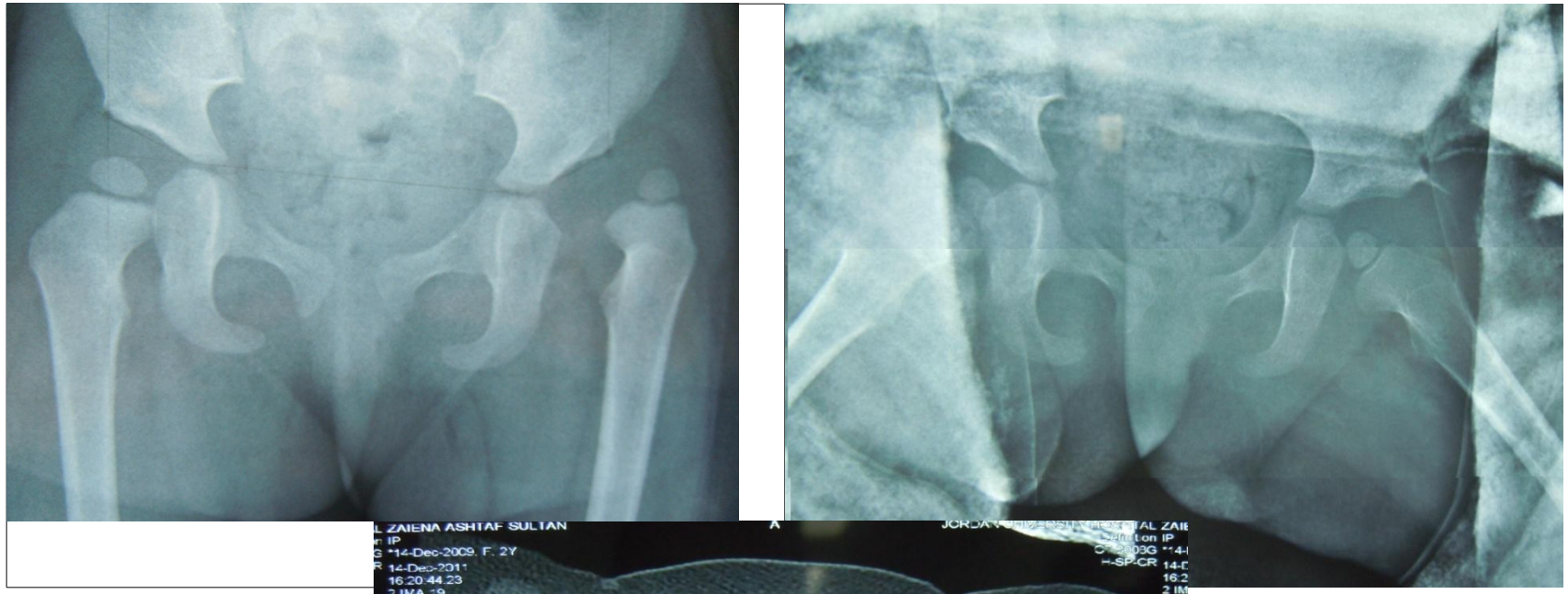
Those who failed with PH & those between 6 – 12 months

Closed Reduction +/- Arthrography & Hip Spica (CAST)

21-04-



Older Children
OR & Acetabular Osteotomy



Complications and pitfalls

i. AVN

ii. INADEQUATE REDUCTION AND REDISLOCATION

iii. RESIDUAL ACETABULAR DYSPLASIA

Legg-Calves'-Perthes Disease overview

- **D**efinition

A common childhood disease resulting from ***avascular necrosis*** of the femoral head epiphysis in children with immature skeleton

Thompson, G.H.; Choi, I.H. Legg-Calve-Perthes Disease Centenary. J. Pediatr. Orthop. **2011**, 31, S129.

Legg-Calves'-Perthes Disease overview

- **E**pidemiology

Children aged 4 to 12 years

More frequent in males than females.

Reported incidence rates in children <15 years vary, ranging from 0.2 - 19 per 100,000

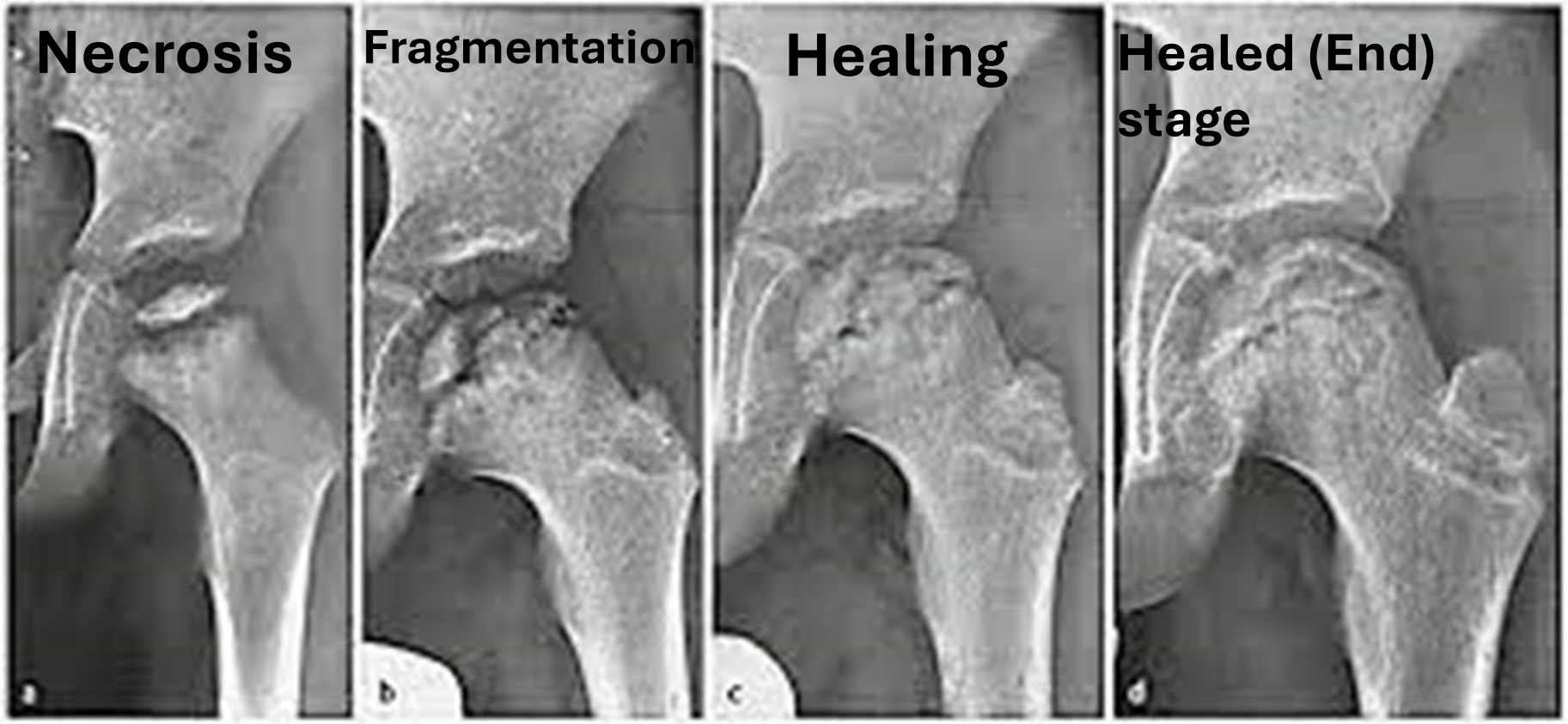
Perry, D.C.; Machin, D.M.G.; Pope, D.; Bruce, C.E.; Dangerfield, P.; Platt, M.J.; Hall, A.J. Racial and Geographic Factors in the Incidence of Legg-Calvé-Perthes' Disease: A Systematic Review. Am. J. Epidemiol. **2012**, 175, 159–166.

Legg-Calves’-Perthes Disease overview

- **Pathogenesis**

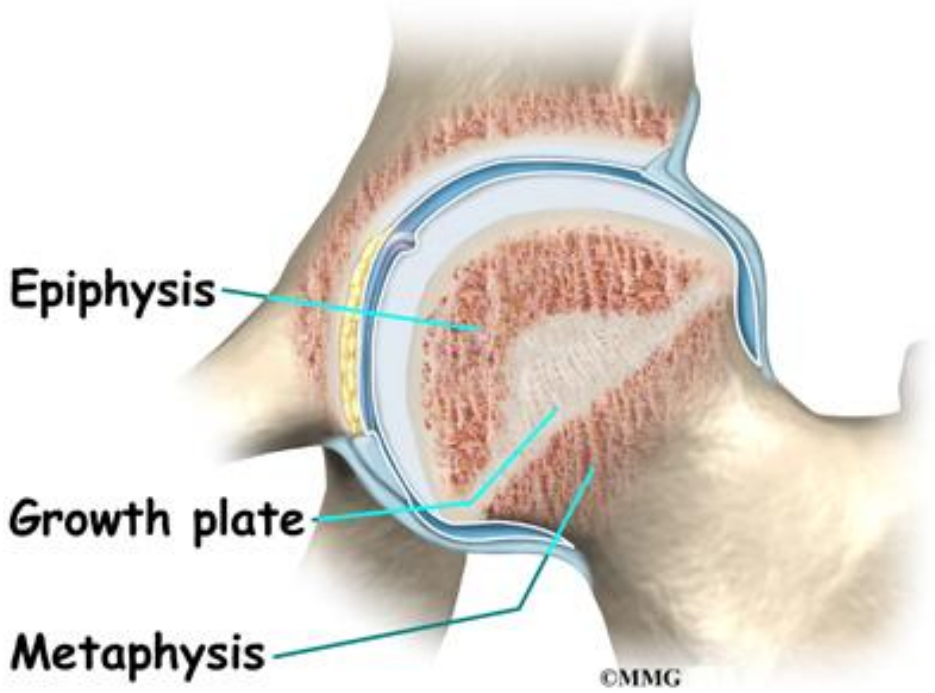
Legg-Calves’-Perthes Disease overview

Pathogenesis



Legg-Calves'-Perthes Disease overview

1. Articular Cartilage Layer
2. Epiphyseal Cartilage Layer
3. Germinal Cell Layer



https://eorthopod.com/perthes-disease/

Legg-Calves'-Perthes Disease overview

Synovial Fluid

Epiphyseal Blood Supply

1. Articular Cartilage Layer
2. Epiphyseal Cartilage Layer
3. Germinal Cell Layer

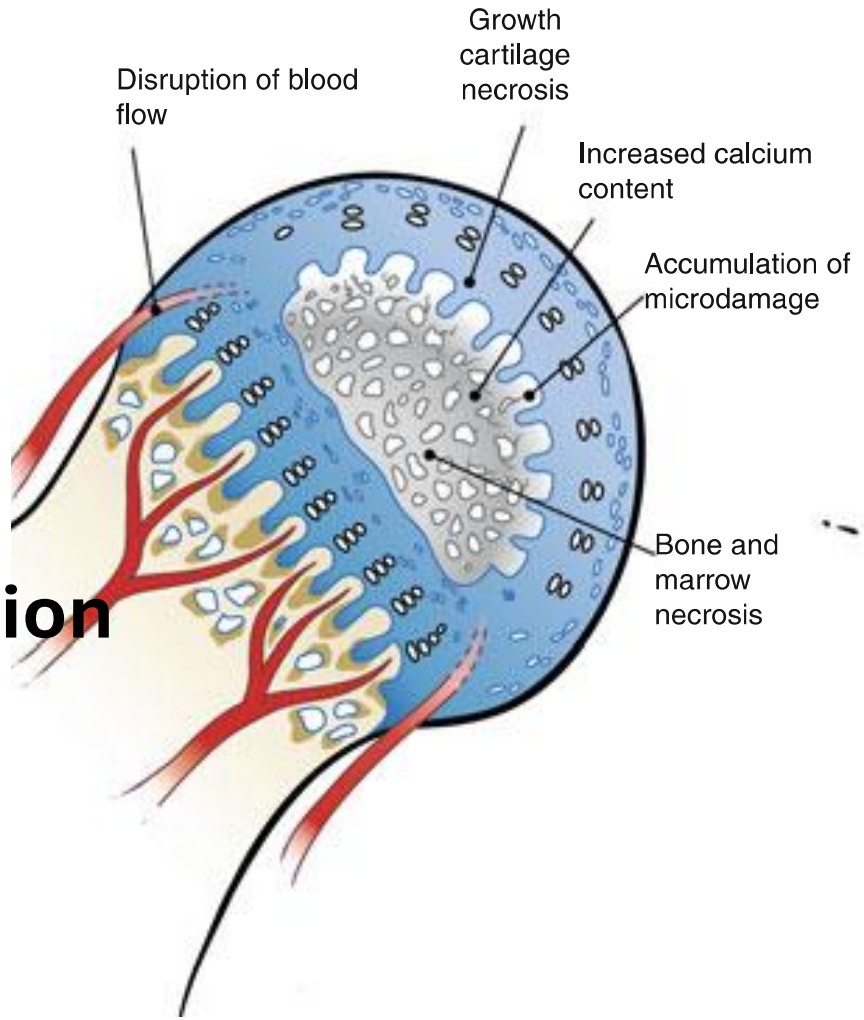
Legg-Calves’-Perthes Disease overview

What Triggers the changes in LCPD?

Legg-Calves'-Perthes Disease overview

Pathogenesis

1st Ischemic attack



a. Cells of germinal layer will die

So, Cessation of endochondral ossification

b. Ossific nucleus remains small

Kim, H. K. W. (2014). *Legg-Calvé-Perthes Disease: Pathology, Pathophysiology, and Pathogenesis of Deformity. Osteonecrosis, 427–436.*doi:10.1007/978-3-642-35767-1_59

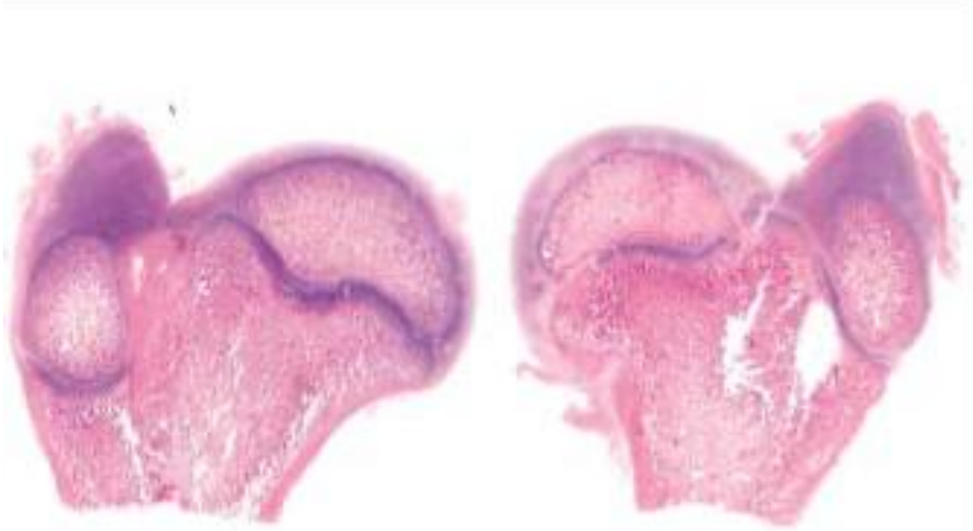
Legg-Calves'-Perthes Disease overview

Pathogenesis

1st Ischemic attack

a. Cessation of endochondral ossifica

b. Ossific nucleus remains small



Late Complications of Legg-Calve-Perthes Disease. Sung Man Rowe, M.D. Department of Orthopedics, Chonnam National University Hospital, Gwangju, Korea *J. of Korean Orthop. Assoc.* 2004; 39: 439-54

Legg-Calves’-Perthes Disease overview

Legg-Calves’-Perthes Disease overview

Pathogenesis

If no new attack, the disease will be silent and will heal perfectly

Legg-Calves'-Perthes Disease overview

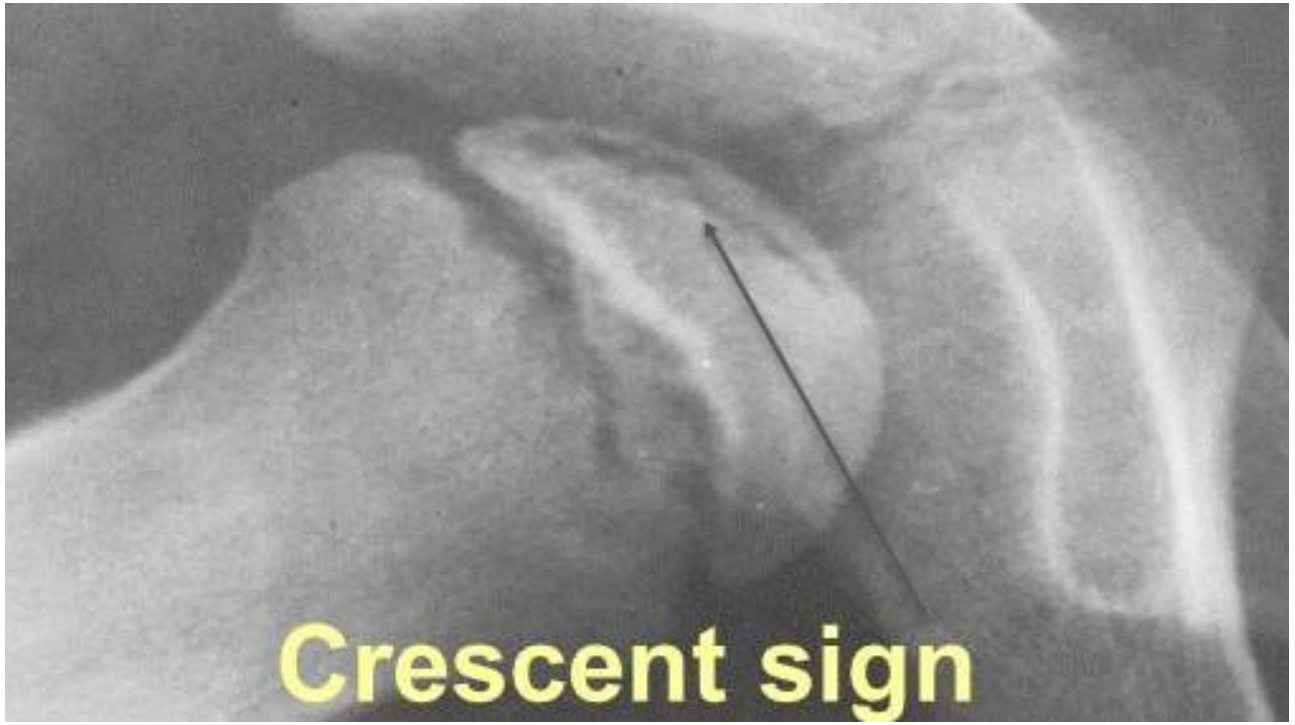
Pathogenesis

2nd Ischemic Attack

Mechanical

Causing Subchondral Fracture

Release of inflammatory mediators intraarticularly



Starting of True LCPD

Legg-Calves’-Perthes Disease overview

Pathogenesis

Fragmentation Stage

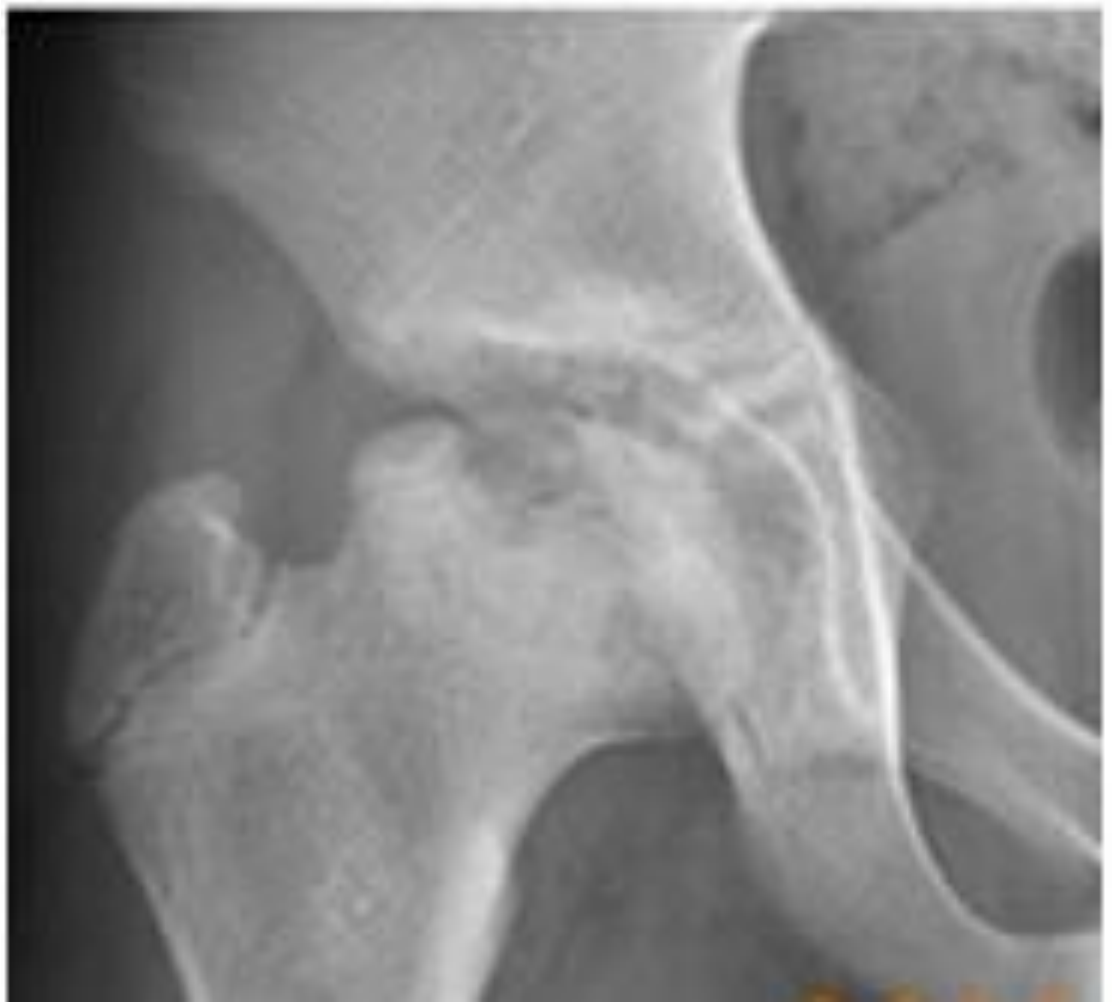
Legg-Calves'-Perthes Disease overview

Pathogenesis

Healing Stage

Head is **NOT** Moldable

Whole Mark is **Subchondral Ossification**



Legg-Calves'-Perthes Disease overview

- **Classification**

The plain radiograph is still the best convenient way for the classification of the LCPD.

Legg-Calves'-Perthes Disease overview

- **Classifications**

Active stage

- Catterall
- Salter & Thompson
- Lateral Pillar

Healed stage

- Stulberg

Legg-Calves'-Perthes Disease overview

Lateral Pillar 1992

Lateral Pillar separated from the central portion by a radiolucent line , If Not present then lateral 25 - 30% of the head

Legg-Calves'-Perthes Disease overview

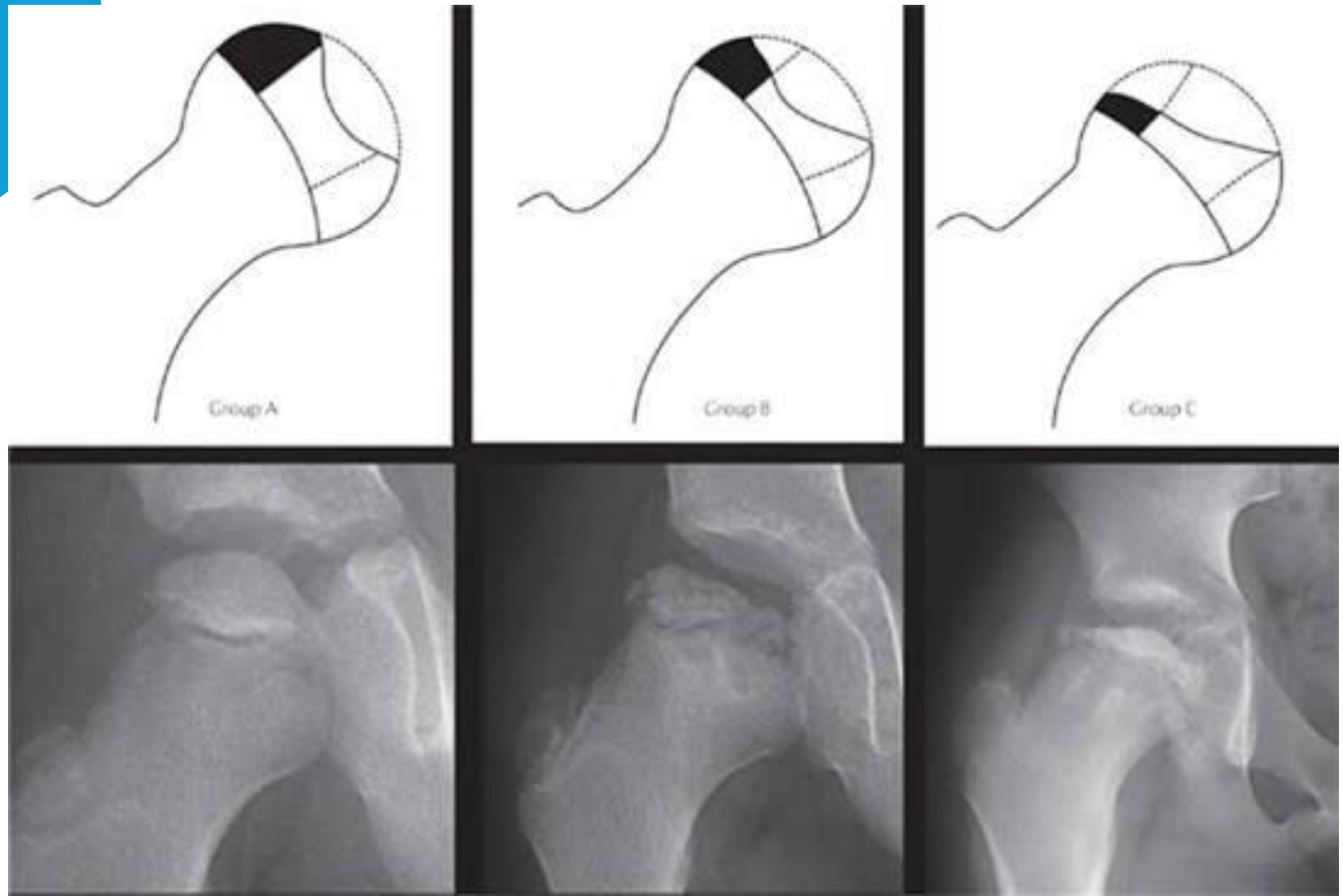
Prognosis and evaluation of classifications in respect of prognosis

Prognostic factor	Prognostic significance
Age	+++
State of lateral pillar[1]	+++
Lateral calcification[2]	++
Subluxation[2]	++
Overall extent of necrosis[3]	++
Mobility	++
Sex	+
Metaphyseal involvement[2]	+
Gage sign[2]	–
Transverse epiphyseal plate[2]	–

[1] Classifikation acc. to Herring
[2] risk factors acc. to Catterall
[3] Classifikation acc. to Catterall or Salter and Thompson or. Herring

Legg-Calvé-Perthes disease (LCPD), Pediatric Orthopedics in Practice. Frizt Huefte 2015

Age & Lateral Pillar type



Legg-Calves'-Perthes Disease overview

Treatment

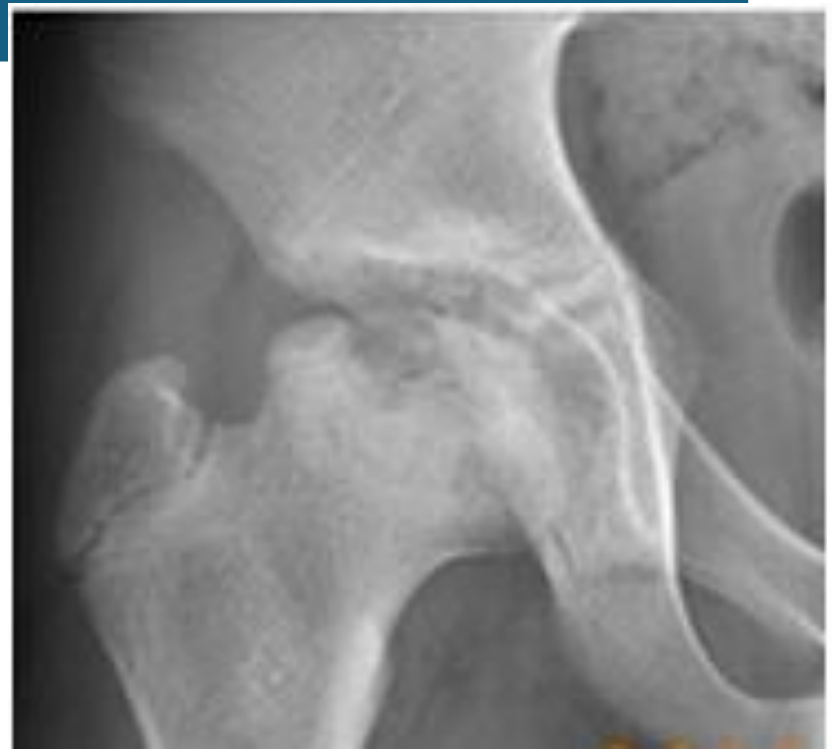
Active stage

To prevent deformity

Legg-Calves'-Perthes Disease overview

Treatment

Cut off Point is Subchondral Ossification



Legg-Calves'-Perthes Disease overview

Treatment

All '**TYPE A**' lateral pillar

Conservative

***Maintain good ROM**
- **PT exercises**
- **Braces vs cast**

***Bisphosphonate**

Legg-Calves'-Perthes Disease overview

Treatment

Type B lateral pillar > 8 years

Operative

***Pelvic Osteotomy**
***Femoral Osteotomy**
*** Hip Arthrodiastasis**

Slipped Capital Femoral Epiphysis

Defining the Slip

SCFE is a common hip disorder in adolescents. a mechanical displacement of the femoral metaphysis anteriorly and superiorly relative to the epiphysis.

This displacement occurs selectively through the weakened hypertrophic zone of the physeal growth plate.

Biomechanical Shear

Under shear stress, the growth plate slips. The structural failure behaves similarly to a Salter-Harris Type I fracture.

Slipped Capital Femoral Epiphysis

Risk Profiles & Etiology

Mechanical Factors

Obesity shifts body weight, increasing shear force across a verticalized physis during the adolescent growth spurt.

Demographic Indicators

More common in rapidly growing adolescent males aged 10 to 14 years. It is often bilateral in up to 50% of cases.

Endocrine Factors

Atypical presentation outside standard age groups points to hypothyroidism, GH excess, or renal osteodystrophy.

Slipped Capital Femoral Epiphysis

Types of SCFE

Stable

Child has pain in the knee or in the groin BUT still **can bear weight** on the same side

Unstable

Child has severe pain and sometimes stiffness that limit his movement where **he cannot bear weight** on the involved side

Slipped Capital Femoral Epiphysis

Clinical Presentation

Knee Pain

May last weeks

Slipped Capital Femoral Epiphysis

Clinical Presentation

Knee Pain
May last weeks

Antalgic Gait

More common in rapidly growing adolescent
males aged 10 to 14 years. It is often bilateral
in up to 50% of cases.

Slipped Capital Femoral Epiphysis

Clinical Presentation

Knee Pain
May last weeks

Antalgic Gait

More common in rapidly growing adolescent males aged 10 to 14 years. It is often bilateral

External Rotation of lower limb

Slipped Capital Femoral Epiphysis

Clinical Presentation

Knee Pain
May last weeks

Antalgic Gait

More common in rapidly growing adolescent males aged 10 to 14 years. It is often bilateral

External Rotation of lower limb

Drehmann sign

Slipped Capital Femoral Epiphysis

Clinical Presentation

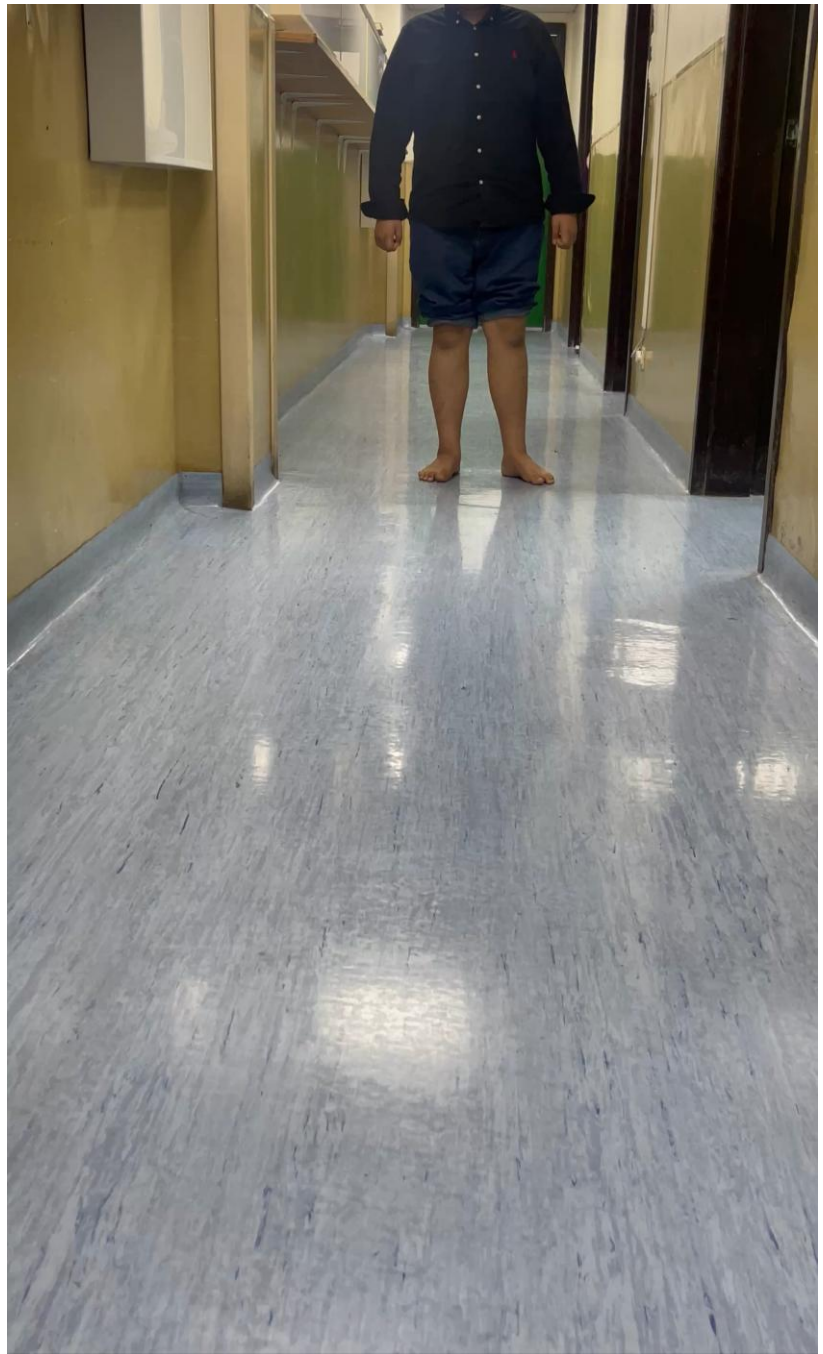
Knee Pain
May last weeks

Drehmann sign

Antalgic Gait

More common in rapidly growing adolescent males aged 10 to 14 years. It is often bilateral

External Rotation of lower limb



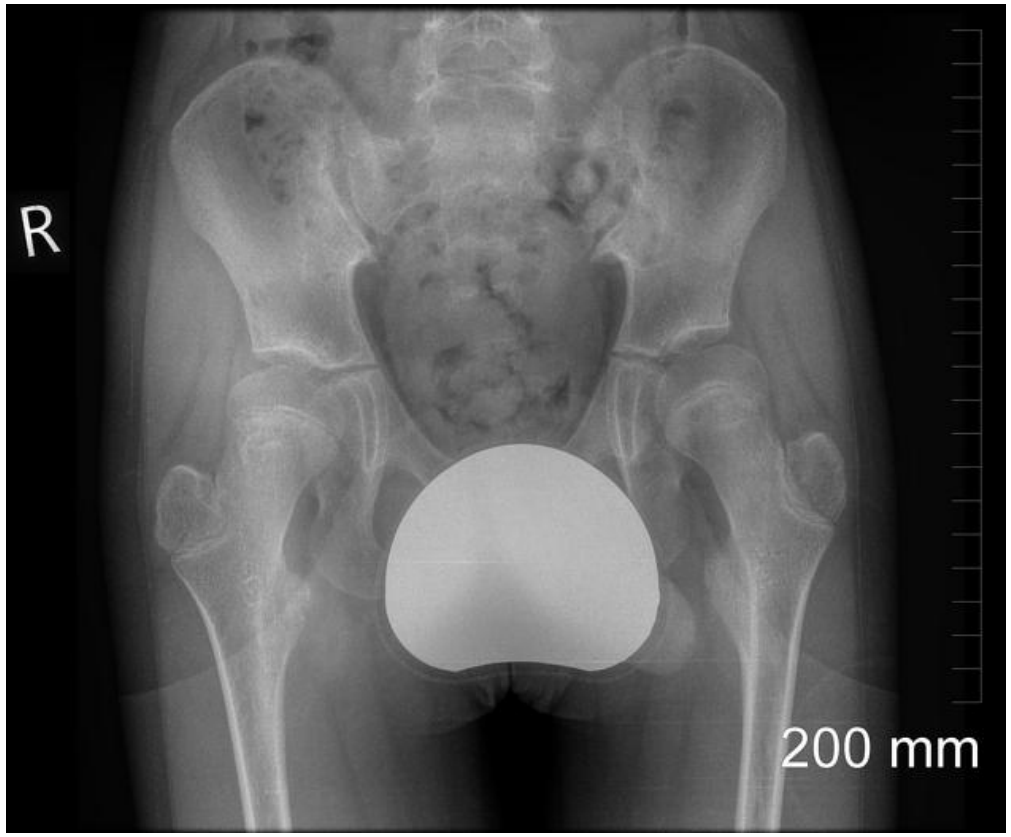
Radiological Diagnosis

Plain radiograph of the pelvis is standard for typical presentation

Slipped Capital Femoral Epiphysis

Radiological Diagnosis

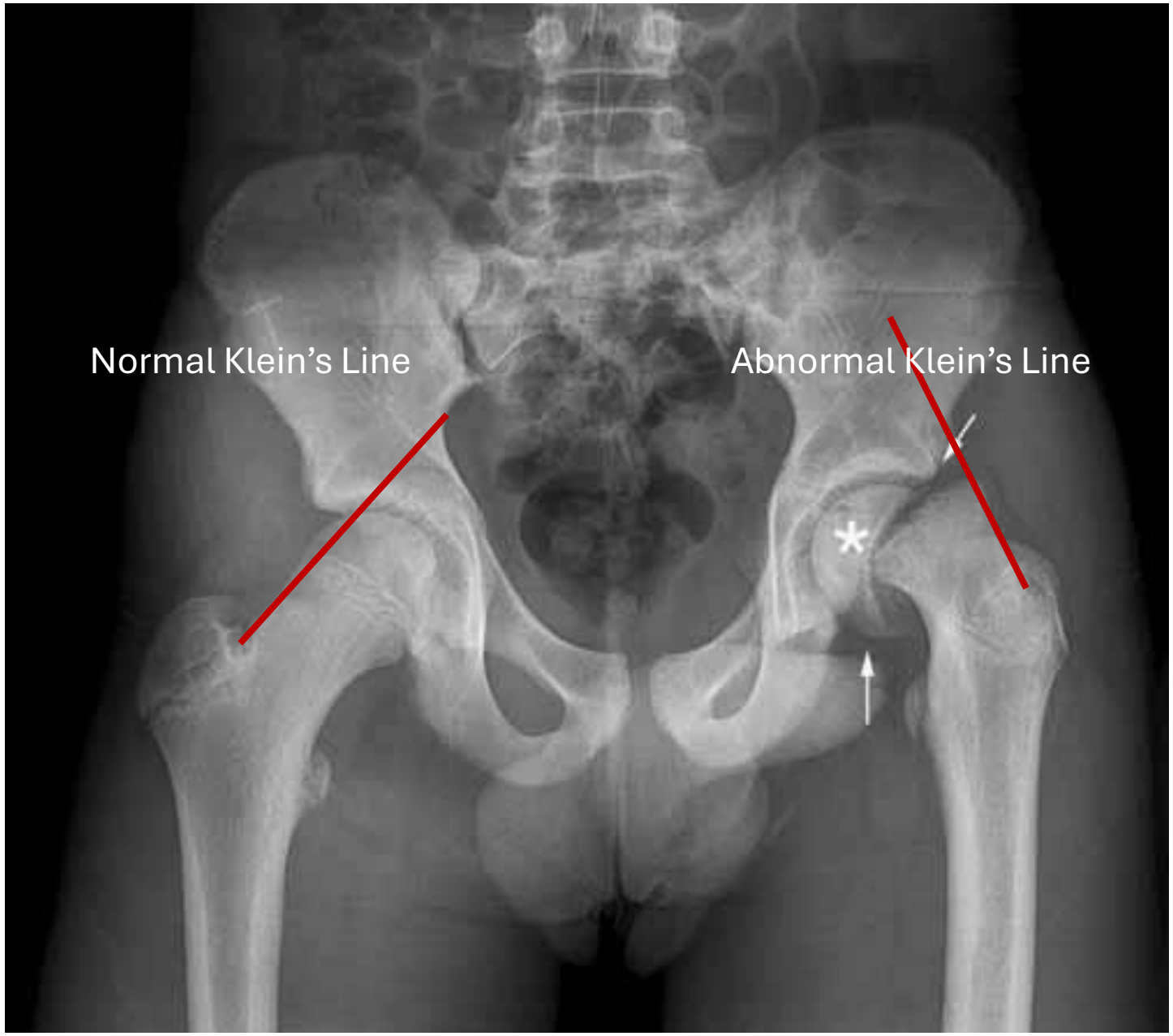
Plain radiograph of the pelvis is standard for typical presentation



Slipped Capital Femoral Epiphysis

Radiological Diagnosis

Plain radiograph of the pelvis is standard for typical presentation



Surgical Treatment

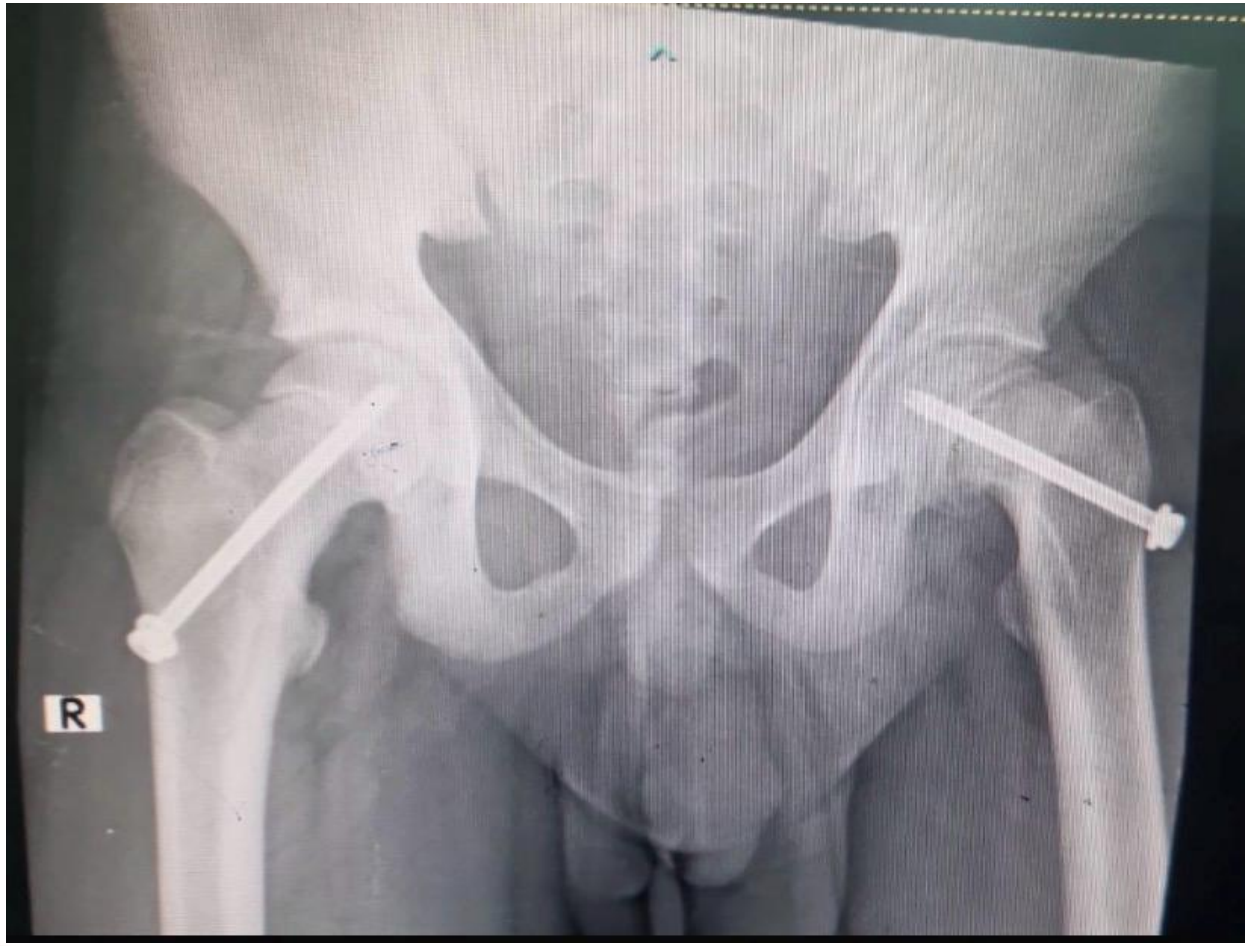
Aims:
Preventing further slippage and encouraging closure.

Slipped Capital Femoral Epiphysis

Surgical Treatment

Aims:
Preventing further slippage and encouraging closure.

Single Screw fixation



Complications

Avascular Necrosis (AVN): Highly associated with unstable slips and aggressive manipulation of acute deformities.

Slipped Capital Femoral Epiphysis

Complications

Avascular Necrosis (AVN): Highly associated with unstable slips and aggressive manipulation deformities.

Chondrolysis: Destruction of articular cartilage, leading to joint stiffness, severe pain, and early fusion of the joint cavity.

Slipped Capital Femoral Epiphysis

Complications

Avascular Necrosis (AVN): Highly associated with unstable slips and aggressive manipulation deformities.

Chondrolysis: Destruction of articular cartilage, leading to joint stiffness, severe pain, and early fusion

Femoroacetabular Impingement: Residual osseous deformity of the anterior metaphysis can damage the acetabular labrum.

Complications

Avascular Necrosis (AVN): Highly associated with unstable slips and aggressive manipulation deformities.

Chondrolysis: Destruction of articular cartilage, leading to joint stiffness, severe pain, and early fusion c

Femoroacetabular Impingement: Residual osseous deformity of the anterior metaphysis can damage the acetabular labrum.

Contralateral Slippage: Up to 50% of patients will slip on the opposite side. Consider prophylactic pinning in high-risk patients.