3-Pediatric hip

I-DDH	II-Perth's disease	III-SCFE	IV-Coxa Vara

I. Developmental Dysplasia of the Hip (DDH)

Syllabus:
1-Definition, Terminology, Incidence & Risk factors for DDH
2- Growth & development of the hip **3**-Screening for DDH
4-Signs in DDH **5**-Treatment policies & outcome

1-Pediatric Hip joint anatomy, Figure-1
***Normal growth of the acetabulum** depends on
1-Normal epiphyseal growth of the triradiate cartilage and on the three ossification centers located within the pubis (os acetabulum), ilium (acetabular epiphysis), and ischium. **Figure-1A**
2-The presence of the spherical femoral head within the acetabulum is critical for stimulating normal development of the acetabulum. The hip is a "ball-and-socket" joint that is held together by ligaments and joint capsule.
3- At birth, the hips are lax, head of femurs are cartilaginous, the acetabulum has more cartilage than bone, and the fibrocartilage labrum widened the acetabulum to accommodate 50% of head cover. **Figure -1B**

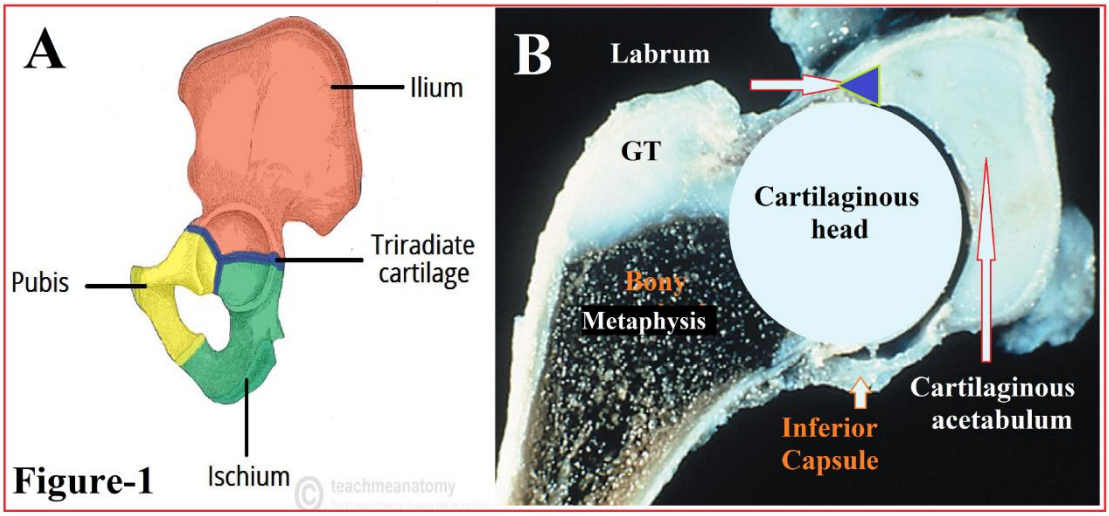

Figure-1

4-Few weeks after delivery, 85-90% of the hips become mature and stable.
5-Babies whose legs are swaddled tightly with the hips and knees straight are at a notably higher risk for developing DDH after birth.
***Definitions of neonatal hip disorders**
A. Developmental dysplasia of the hip (DDH).
The spectrum of abnormal growth of the developing hip, (ranging from acetabular dysplasia, hip subluxation, hip dislocation, or hip instability).

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Acetabular dysplasia: abnormally developed, shallow acetabulum, with an oblique roof and a thickened medial wall.
Subluxation – Incomplete contact between the articular surfaces of the femoral head and acetabulum.
Dislocation – Complete loss of contact between the articular surface of the femoral head and acetabulum.
Instability – Ability to subluxate or dislocate the hip with passive manipulation.

B. **Teratologic (Congenital) dislocation of the hip** (CDH)
Antenatal dislocation in utero and usually stiff irreducible on neonatal examination. (Remember Pavlik harness should not be used in CDH)

***Epidemiology**
1-20:1000 neonatal hip instability, 1-2:1000 treated for dysplasia.
2-80% of affected children are female.
3-The left hip is more commonly involved (60%)., as the commonest head presentation is left occipito posterior, during delivery the left hip lies against the mother's sacrum which forces the hip to be in the adducted position, that favors instability. 4-20% bilateral. 20% right. 5-60% of newborns with hip instability become stable by age 1w, and 90% become stable by age two months, leaving only 10% of them with residual hip instability.

Associated ('packaging') conditions with DDH
1-Torticollis 2-Plagiocephaly 3-Metatarsus adductus
4-Calcaneovalgus feet 5-CTEV

***Etiology and risk factors in DDH.**

1. Etiology:
I-Anatomical factors.
The shallow acetabulum and capsule laxity often coexist at birth, improving the range of hip movement to aid delivery. The femoral head is >50% uncovered at birth, and this predisposes to subluxation/dislocation.
II- 1ry idiopathic hip dysplasia.
III- Multifactorial.
* *Genetic inheritance* → positive family history
* *Racial*: absent in Africa
* *Mechanical*: Breech, Oligohydramnios, 1st born (tight uterus).
A breech presentation may exert its effects using the strong hamstring forces on the hip that result from a knee extension. The increased tension on the hamstrings pulls the femoral head out of the acetabulum.
* *Maternal hormone*: female child. Maternal hormones & fetal estrogen that is produced by the female infant's uterus → Ligament laxity

2- Risk factors 10F
A- **F**rank breech presentation (30 - 50% risk).
B- **F**emale and **F**irstborn, and with

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C- **F**amily history is a strong risk factor.
i. One child has DDH, risk of another child 6%
ii. At least one parent involved: 12% risk
iii. Parent and sibling involved: 36% risk
D-**F**luid abnormality Oligohydramnios)
E- **F**eet Deformity (Metatarsus adductus)
F- **F**etal anomalies
G-**F**aulty Habits (Swaddling) esp. with ligament laxity.
H- **F**acial asymmetry (Torticollis).

Secondary Hip Dysplasia (Pathological)

1-Neurological conditions: Cerebral palsy, Down syndrome.
2-Connective tissue diseases: Ehlers-Danlos syndrome

***Pathology of DDH**

Spectrum: 1-Acetabular dysplasia 2-Subluxation 3-Dislocation

Bony changes Figure-2A

Acetabulum	Femur
= Shallow, dysplastic. = Anteverted deficient anterolaterally. = Delay in teardrop development. = May deficient posteriorly. = New false acetabulum.	= Delayed ossific nucleus = Coxa valgus = Anteversion of the proximal femur = Deformed shape of the head.

Soft tissue Figure-2B

Extra-articular	Intra-articular
Tight psoas tendon Tight adductor muscles Short abductors	1-Wide lax redundant capsule 2-Pulvinar (fibro-fatty tissue) 3-Thick elongated ligamentum teres 4-Infolded thick labrum inside (Inverted limbus) 5-Thick transverse acetabular ligament. 6-Narrow inferior capsule by the Psoas.

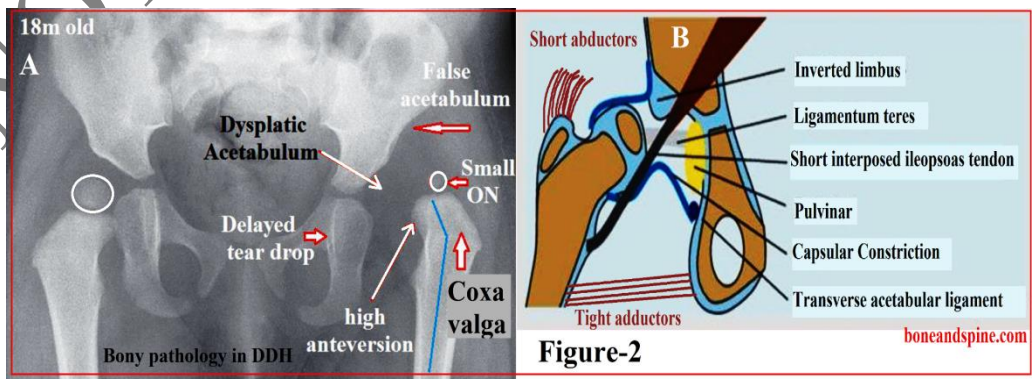

Figure-2

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1- Evaluation

1. Clinical presentation

The clinical presentation varies with age and type of DDH.

1-Incomplete dislocation

= Pre walking: limitation of abduction while changing nappy. **Figure- 3**

= Post walking: limping, tiptoe gait (in unilateral DDH), LLD or waddling gait (in bilateral DDH).

2-In acetabular dysplasia: Asymptomatic.

2. Clinical examination (in frank dislocation)

A. In the neonatal period, (<6 months), e.g., Ortolani test, (Barlow test is not recommended).

B. In infants older than six months –walking,

* Limitation of abduction (>20°), most sensitive test for DDH, **Figure-3**

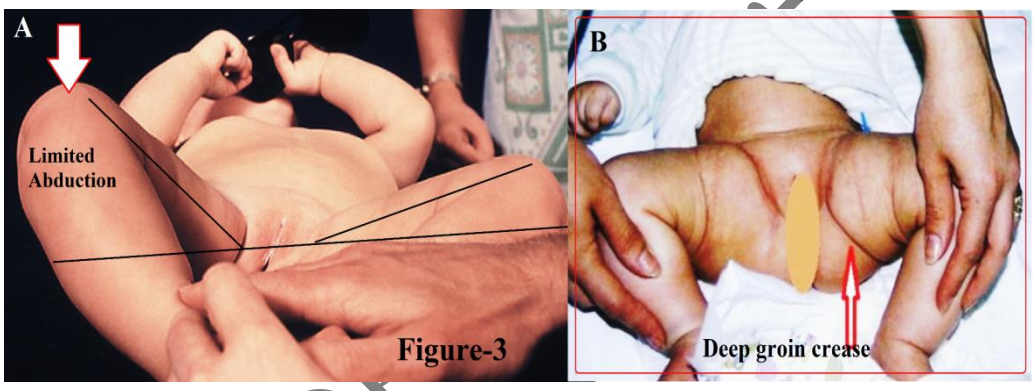

Figure-3

* Apparent limb shortening in unilateral DDH.
* Abnormal deep long groin crease.

C. In toddlers, (+after walking)

* Wide perineum in bilateral DDH
* Lumbar lordosis in bilateral DDH
* Trendelenburg's sign and gait.
* Limping in unilateral DDH
* LLD in unilateral DDH

3. Clinical tests in DDH

Accuracy of the physical examination requires that the child be relaxed.

A- The Ortolani test (reduction test): best tested before 3m of age by holding the knee on one side and fixation of another hip, lifting the proximal femur anteriorly with the middle finger and abducting the hip. **Figure -4**

N.B: Ortolani test is not for dysplastic or subluxated hips**,** only for completely

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dislocated hips 1:500. (poor screening test)

- Thus, the negative Ortolani test does not mean no DDH; it means only you need further evaluation for risky newborns by other methods.
- The test is positive if the dislocated hip is reducible (Clunk), in cases of teratological dislocation, (CDH) the hips are irreducible.

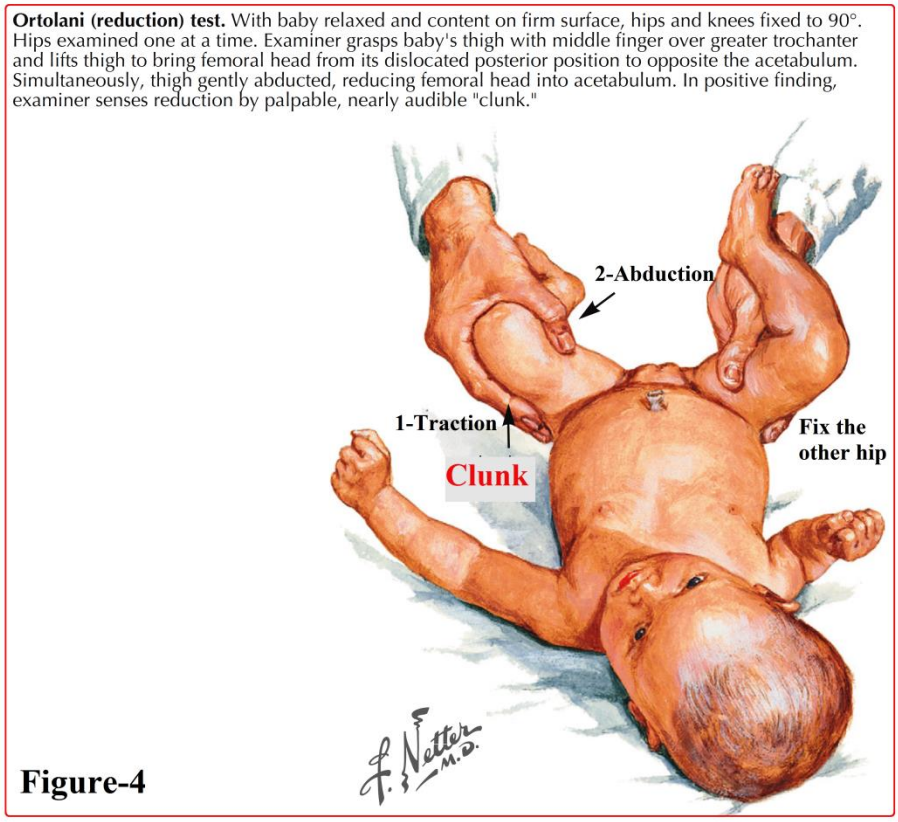

Figure-4

B-The Barlow test: (Dislocation test), harmful test **Figure-5**
Should not be done except during ultrasound examination (by down pressure on the hip in a flexed and adducted position).
The test is positive if the hip subluxate or even dislocates on stress.

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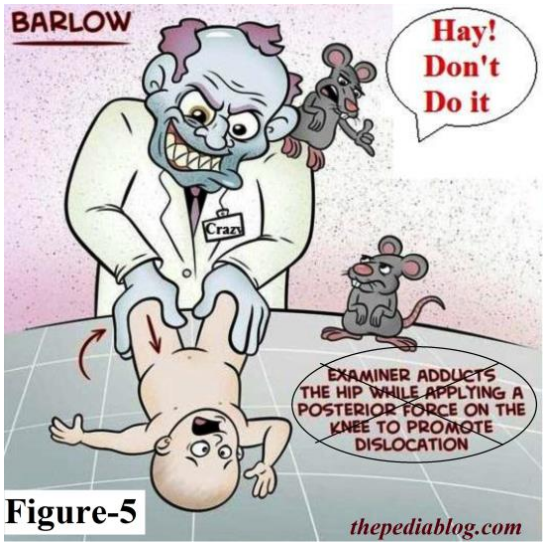
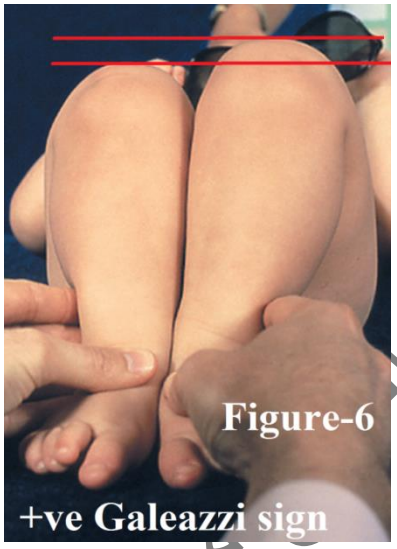


Figure-5



C-The Galeazzi (or Allis) test is positive only in a unilateral dislocation in children above six months of age. **Figure-6**
The hips and knees lexed to 90°; the test is positive if one knee (the involved side) is lower than the other.

4. **Diagnostic tests** (confirmatory)
A-Ultrasonography (USS). (PG). Figure-7
= At six weeks in patients wh considered high risk (female, family history or breech presentation) despite a normal exam.
= Done if available proper ultrasonography service in the first 4- 6 months of life before the appearance of the ossific nucleus.

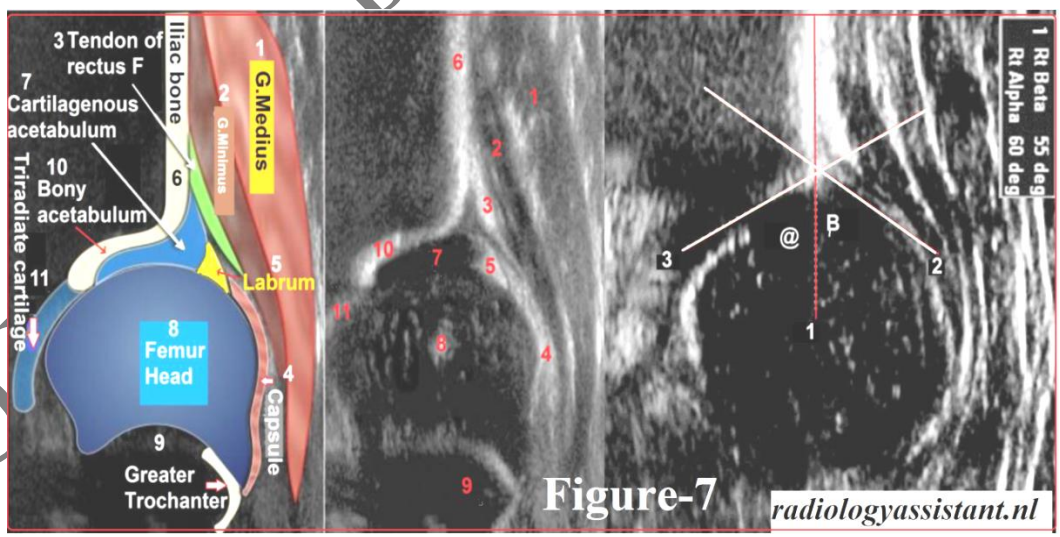


Figure-7

At the age of 6 weeks,
= Normal α angle is >60°. (Acetabular roof angle).
= Normal β angle is <55°. (Labral cartilage roof angle).

= The amount of femoral head coverage should be >50%.

* ***α angle***

The angle between the ossified lateral wall of the ilium and the bony acetabular roof.

* ***β angle***

The angle between the ossified lateral wall of the ilium and the cartilaginous roofline. i.e., it represents the cartilaginous roof of the acetabulum.)

Neonatal screening for DDH

1- Clinical screening (thorough history taking and physical examination) of all newborn infants is necessary, without ultrasonography.

2-Routine ultrasound screening performed for infants with at-risk factors for the condition at the age of 6 weeks, (to give a chance to neonatal unstable hips to be mature and stable (85-90%), otherwise you will over-diagnose and over-treat hips.

3-If no proper ultrasound facility is available, do plain radiograph for the hips at the age of 3 months (make sure the hips to be at 40° abduction & 30° internal rotation).

B. Plain radiographs, Figure 8-9

I. Hilgenreiner line

A line is drawn horizontally through each triradiate cartilage of the pelvis.

II. Perkin line

Perpendicular line to the Hilgenreiner line at the lateral edge of the acetabulum.

This will divide the hip region into four quadrants, (the normally ossific nucleus of the head in the inner lower quadrant, in subluxation it is in the outer lower quadrant, and in a dislocation in the upper outer quadrant).

III. Shenton line

A continuous arch is drawn along the medial border of the femoral neck and the superior border of the obturator foramen.

N.B: False positive in young children with high femoral anteversion

IV. Acetabular index angle (AIA) (Mirror of DDH)

The angle formed by an oblique line (through the outer edge of the acetabulum and triradiate cartilage) and the Hilgenreiner line.

(a) In the infant <3months, a normal value < 30°.

(b) By 6months of age, the acetabular index decreases to 25°.

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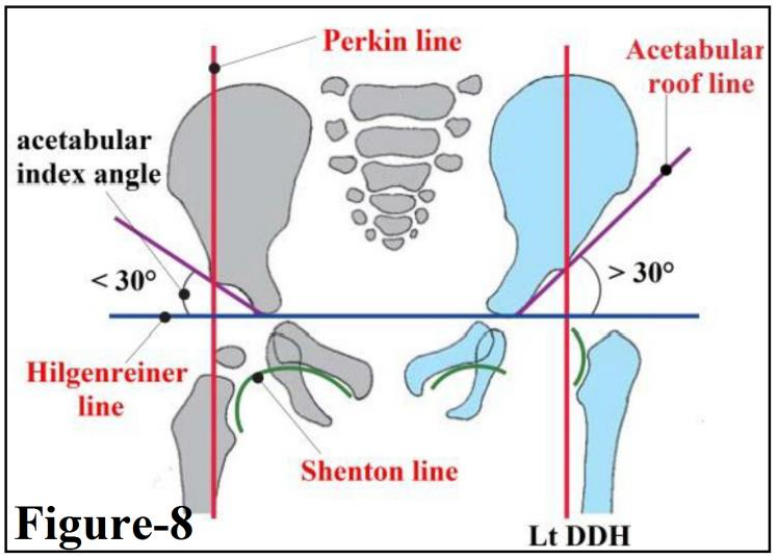

Figure-8

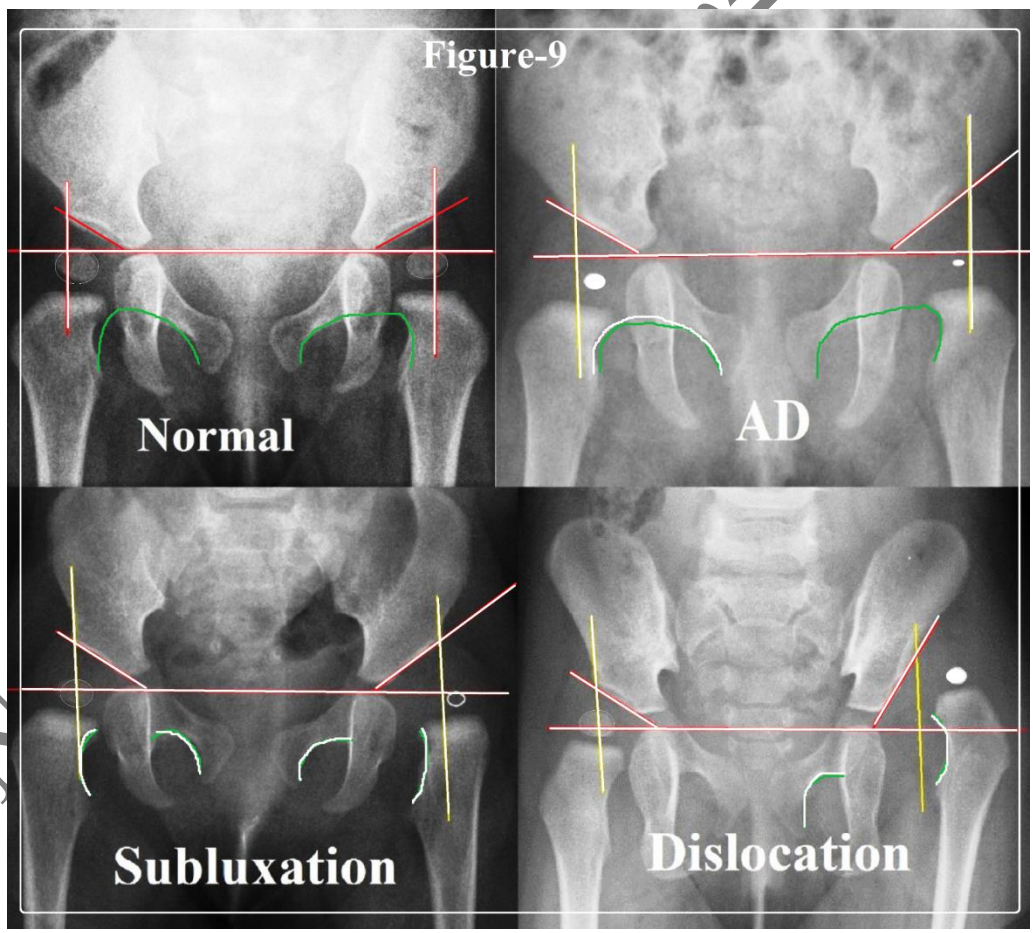

Figure-9

Radiographic finding in acetabular dysplasia

* Increased obliquity of the acetabulum (AIA >30°).
* Loss of acetabular concavity. * Intact Shenton line.

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Natural History of DDH

= 90% of neonatal unstable hips will stabilize by 4-6 weeks of age.
= The maximum remodeling of the acetabulum occurs below the age of 18m.
= Dislocated hips per se do not develop AVN.
= In a dislocated hip the affected leg will become shortened, and this will put pressure on the back, increasing the risk of osteoarthritis to the spine.
The false acetabulum is smaller than a true acetabulum and will develop osteoarthritis between 40-60 years of age if not treated.

5- Management of Developmental Dysplasia of the Hip

The basic principles for treatment are

• Concentric reduction of the dislocation or subluxation.
• Hold the reduction until hip stabilizes.
• Careful follow-up until skeletal maturity.

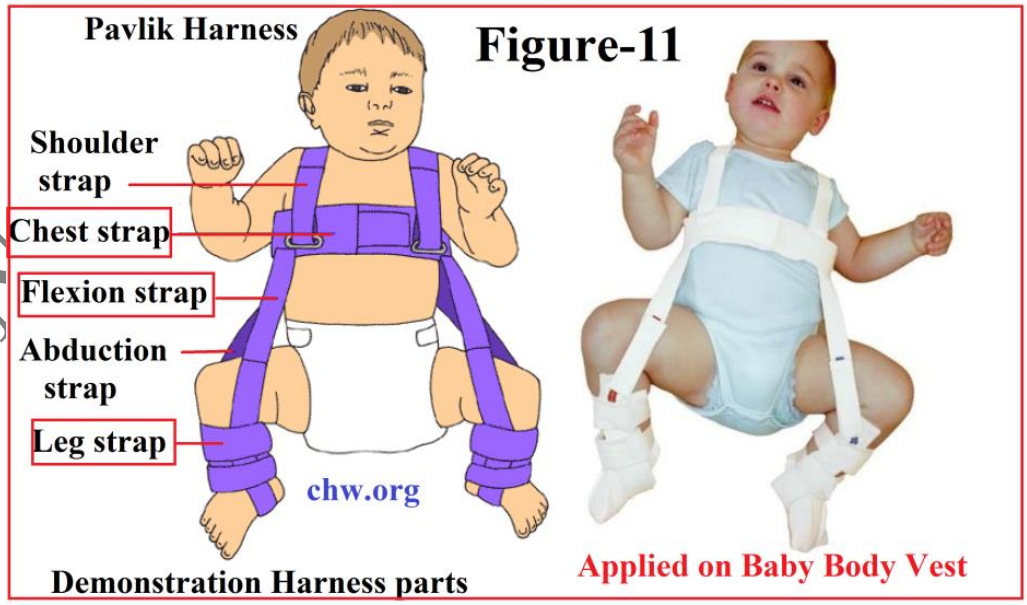
Treatment based on the age of the child, the stability of the hip (unstable versus dislocated hip), and severity of acetabular dysplasia.

1-Dysplastic hips in neonate < 6 months of age

In a child with an abnormal α angle on ultrasound or with an unstable hip (subluxable hip on examination), initial treatment usually includes a Pavlik harness. (Gold standard treatment) **Figure-1.**

A-The hips flexed to 90-100° with 45-60° abduction.
B-Excessive hip flexion → risk of femoral nerve palsy.
C-Excessive hip abduction → increased risk of osteonecrosis
The Pavlik harness is a dynamic flexion/abduction orthosis
Child weight must be <9kg to avoid tearing of the harness by the child.

Duration of the Pavlik Harness: 8-12 weeks**,** until the AIA <30°



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Success rates for Pavlik harness treatment in this setting have been reported at >90% if applied before 2m of age.
The recurrence rate of hip problems in the future is 10-20%; therefore, follow-up evaluation until maturity is necessary.
In a relatively large child or a child older than age six months with a dysplastic hip or with hip subluxation, Spica casting is an option.

N.B: No role for double napkins in the treatment of DDH.

6-Complications of DDH

1-Joint stiffness after open surgery
2-Residual acetabular dysplasia, subluxation, and /or re-dislocation despite adequate treatment. (residual subluxation is ~20%) → FU till maturity.
3-Early osteoarthritis in the hip joint(the 30s)
4-Leg length discrepancy→ back pain, functional scoliosis, and knee pain
5-Genu valgum: Unilateral hip dislocations →fixed adduction deformity in the hip → increased medially directed stress on the knee joint.
6- **Trochanteric overgrowth** → abnormal gluteus function
6-Avascular necrosis (AVN) of the femoral epiphysis, the most devastating. 0-73%. Extreme abduction, especially when combined with extension and internal rotation, results in a higher rate of avascular necrosis.

The potential sequel of avascular necrosis include:-
Femoral head deformity, acetabular dysplasia, lateral subluxation of the femoral head, relative overgrowth of the greater trochanter, and limb length inequalities, osteoarthritis is a common late complication.
Prognosis
Children with DDH who receive early treatment are generally good.

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Quick notes

DDH risk factors. 10F (all they need initial screening)
Remember: normal screening of risk neonate doesn't mean at all normal hips, You need to do ultrasound at 6, 12 weeks or plain radiograph at
3months, Ultrasound can be performed for up to 6 months provided ossific nucleus is nor seen. **α angle.** (acetabular roof) N= > 60°

Clinical signs
-Limited abduction after the age of 3 months
-Ortolani test: (<3months) reduction test for dislocated hip (negative in dysplasia)
-Barlow test: dislocation of dislocatable hip
-Galeazzi test: flex hips and knees to 90° and look from the side for difference in patellar height indicating leg length discrepancy.

Radiological signs
Acetabulum is the mirror of DDH (N. AIA =30°) at the age of 3m.
Hilgenreiner's line: across the triradiate cartilages of the acetabulae;
Perkin's line: perpendicular to Hilgenreiner's passing through the lateral edge of the roof of the acetabulum. The head should lie in the inferomedial quadrant formed by these two lines.
Shenton's line: should not be broken (false positive in high anteversion).

Treatment
Pavlik harness is the best (applied for <6m old for 3 months)
The hip maintained flexed (90-100° and abducted (40-60°)

Obstruction to reduction
Pulvinar (fibrofatty tissue), Thick ligamentum teres, Tight adductors or flexors & Inverted labrum

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II. Legg-Calvé-Perthes Disease.

Syllabus: 1- Definition **2**-Risk Factors **3**-Vascular anatomy of the femoral head **4**- Pathological staging **5**- Lateral pillar classification **6**-Treatment policies & outcome

1. Definition—Legg- Calvé- Perthes disease (LCPD) is Non-inflammatory idiopathic AVN of the femoral head in a growing child.

2. Epidemiology

b. The disease more commonly affects boys than girls (5:1).

c. The hips are involved **bilaterally in 10%** to 12% of cases.

3. Evolution of Blood supply to the femoral head. (PG)

4. Patho-anatomy

1. Etiology

a. Unknown.

c. Disruption of the vascularity of the capital femoral epiphysis.

c. Hydrostatic pressure theory.

Reactive synovitis → capsular distension → compression on retinacular vessels

d. Thrombophilia in 50% of children with LCPD.

e. 75% of patients have coagulopathy, (protein C and S def.)

f. Microtrauma or passive smoking (affects fibrinolysis)

2. Risk factors (Susceptible child)

* Boys (80%). * Poor Social class.

* Short stature with delayed bone age (usually by two years). (90%)

* The child is often thin, very active. * Smaller than his age group.

3. Pathology.

The capital epiphysis and physis are abnormal histologically, with disorganized cartilaginous areas of hypercellularity and fibrillation.

5. Evaluation

1. Clinical presentation

a. Age 4-9 years (range, two years - late teens).

b. Commonly have a limp and pain in the groin, hip, commonly thigh, or knee regions (referred pain).

2. Physical examination

a. Abnormal gait (antalgic). b. Decreased abduction and internal rotation.

c. Late Limb-length inequality, is mild due to femoral head collapse.

3. Diagnostic tests

a. Plain Radiographs. Figure-1

Standard AP and frog-leg lateral views of the pelvis are critical in making the

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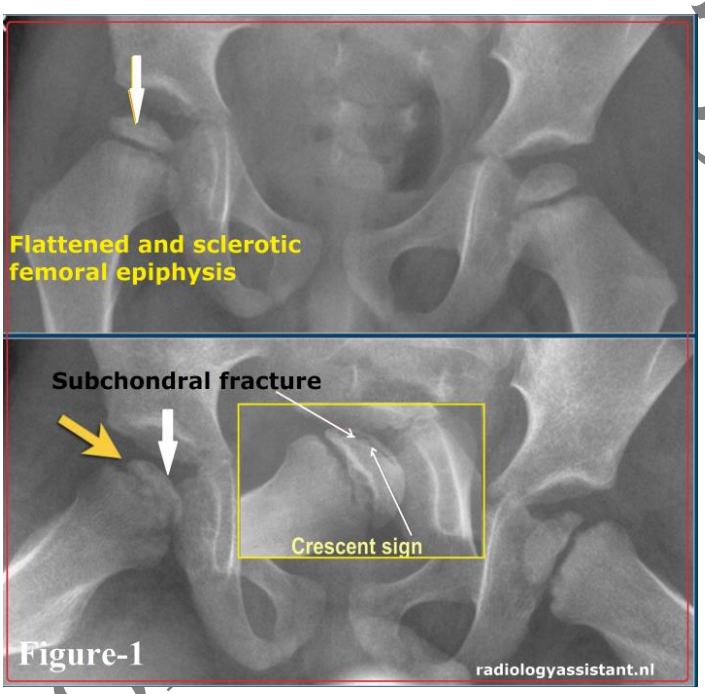
initial diagnosis and assessing the subsequent clinical course. (best)

Radiographic Feature (according to the stage)

• Widening of the joint space and minor subluxation • Sclerosis
• Caffey's sign (Salter), a subchondral # (Crescent sign) on lateral X-ray, (an anterolateral aspect of the femoral capital epiphysis).
• Fragmentation and focal resorption of the epiphysis. • Loss of epiphyseal height.
• Widening of the femoral neck & head (Coxa Magna).
• Lateral uncovering of the femoral head. • Metaphyseal cyst formation.
• Sagging rope sign (Late). • Acetabular remodeling.



LCPD four radiographic stages (**Waldenstrom**).**Figure-2**

a-Necrosis. (Ischemia stage).
= Sclerotic, smaller proximal femoral ossific nucleus
= Widened clear medial space (distance between teardrop and femoral head), due to thickened cartilage.

b-Fragmentation stage (Revascularization)
=Segmental collapse (resorption) of the capital femoral epiphysis
= Increased density of the epiphysis (new bone formation),
= Necrotic resorbed bone replaced by vascular fibrous T → fragmentation shape

c-Re ossification stage (healing)
Necrotic bone continue resorption with subsequent ossification of the capital femoral epiphysis.

d-Final stage (Remodeling).
It begins when the capital femoral epiphysis is completely re ossified.

N.B: In general, necrotic and fragmentation stages last approximately six months each, the re-ossification stage 18 months and the remodeling stage 3 years.

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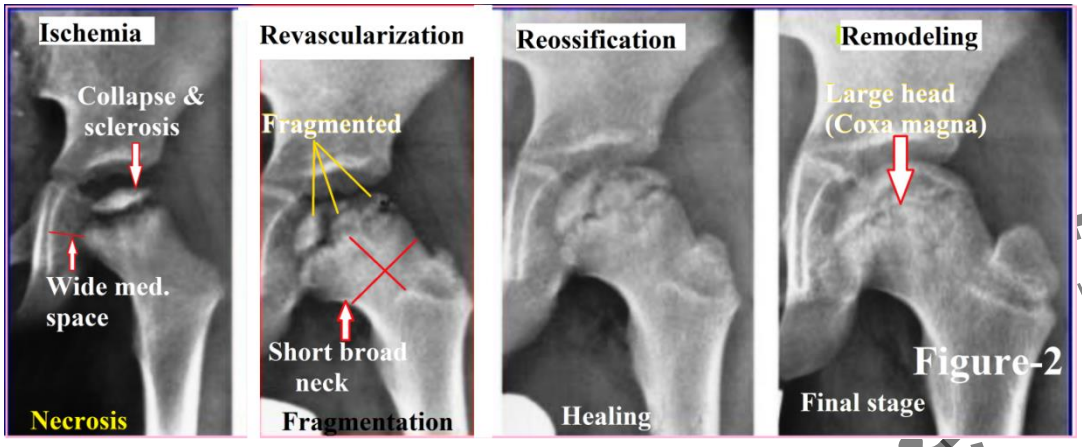


Late Stage

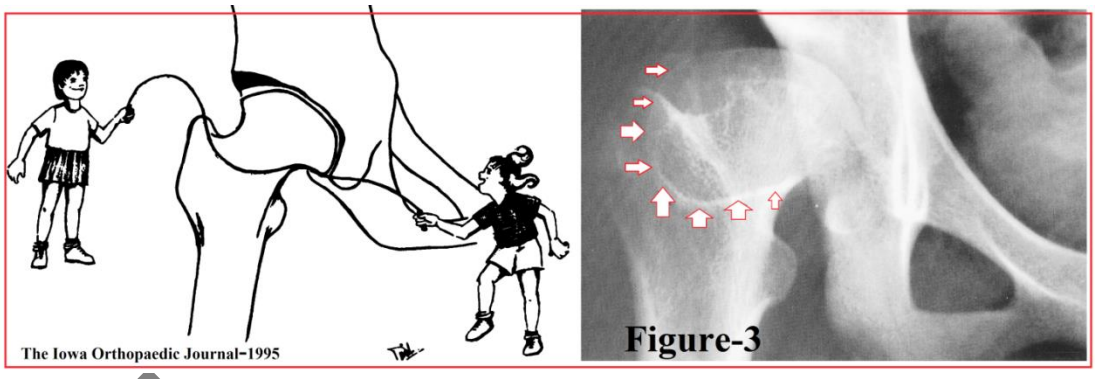
= Coxa magna = High-riding trochanter

=Flattened femoral head =Irregular articular surface

Sagging Rope Sign, Figure-3

• A curvilinear sclerotic line is running horizontally across the femoral neck.

• It is a finding in AP radiograph in a mature hip with Perthes' disease.



b. Other imaging studies:

Bone scans, MRI, and arthrography <u>are not routinely necessary</u>.

i. Bone scan

("Cold" lesion, suggesting decreased blood flow), <u>earliest findings in LCPD</u>.

ii. MRI can also aid in the early diagnosis of LCPD, revealing areas of decreased signal intensity in the capital femoral epiphysis and alterations in the physis.

iii. Arthrography (especially dynamic).

A useful modality to assess coverage and containment of the femoral head.

Arthrography is often used at the time of surgery to confirms the degree of correction needed for femoral and/or pelvic osteotomies.

6. Classification

1. The Herring (lateral pillar) classification <u>(most accurate)</u>

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Based on the height of the lateral pillar of the capital epiphysis on the AP view of the pelvis (**Figure 4**).

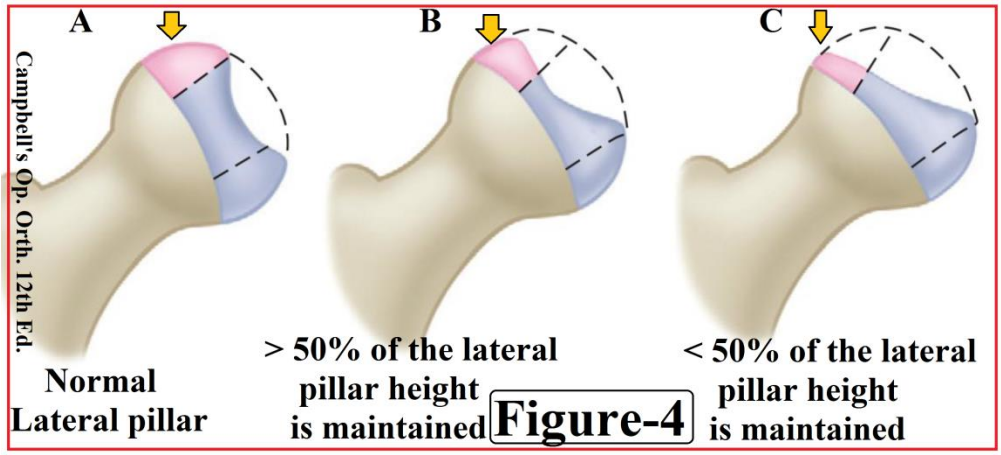

Figure-4

a. Group A: No involvement of the lateral pillar, with no density changes and no loss of height of the lateral pillar.
b. Group B: More than 50% of the lateral pillar height is maintained.
c. Group C: Less than 50% of the lateral pillar height is maintained.
d. B/C border group:
This group has been added more recently to increase the consistency of readings and to increase the prognostic accuracy of the lateral pillar classification.
In this group, the lateral pillar is narrow (2 - 3 mm wide) or poorly ossified, or exactly 50% of the lateral pillar height is maintained.

2-Catterall "head at risk" signs Figure-5
• Clinical signs
1. Progressive loss of hip motion more so abduction.
2. Fixed flexion deformity and adduction deformities of the hip
3. Obese child 4. Age on the higher side

• Radiographic signs
Indicates a more severe disease course.
i. **G**age sign (radiolucency in the shape of a V) in the lateral portion of the epiphysis &/or metaphysis) ii. **C**alcification lateral to the epiphysis iii **D**iffuse metaphyseal rarefaction iv. **L**ateral subluxation of the femoral head
v. **A** horizontal proximal femoral physis.

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Poor prognostic factors in Perthes disease.

1->6 years old **2**-Female: mature earlier with less remodeling potential
3-Catterall's head-at-risk signs (clinical)-2 ≥ "head at risk signs.
4-Obesity **5**-Adduction contracture **6**-Progressive loss of hip motion
7-Flexion with abduction **8**-Advanced stage of disease at diagnosis (B, C)
9-Advanced grade (loss of containment). **10**-Recurrent episodes of stiffness.

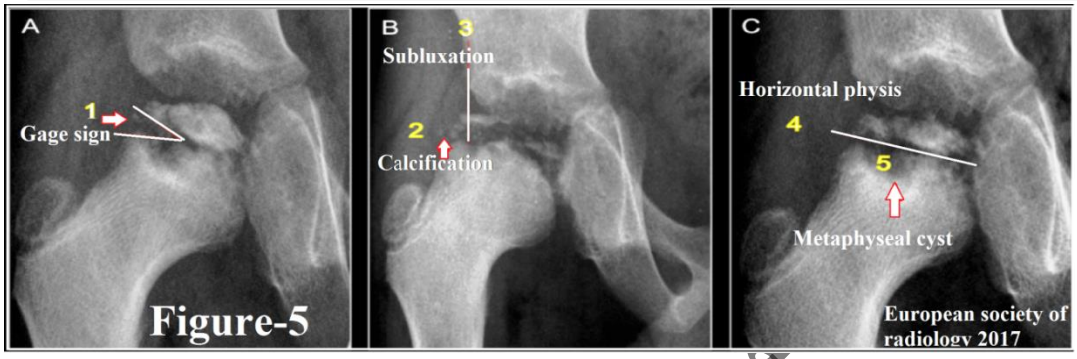

Figure-5

7. Treatment (PG)
Conservative *(this is the mainstay of management).*
NSAIDs, pain killers, physiotherapy.

1. General Principles
2. Containment treatment (Surgical)
3. GT epiphysiodesis: *for high riding GT at the age of >8y*

4. Complications of Perthes disease.
a. Femoral head deformity.
* Premature physeal arrest patterns * Osteochondritis dissecans,
* Labral injury, and * Late osteoarthritis.
b. The most important *prognostic factor*
1- Shape of the femoral head and its congruency at skeletal maturity
2- patient age at onset of disease.
c. Degenerative changes in the hip joint in the fifth or sixth decade of life.

Differential diagnoses

Unilateral Perthes disease	Bilateral Perthes disease
=Transient synovitis = Infection (septic arthritis, T.B. & osteomyelitis) = Blood dyscrasias (lymphoma, leukemia) = Juvenile chronic arthritis = Rheumatic fever. = Sickle cell disease.	= Hypothyroidism = Multiple epiphyseal dysplasias = Spondyloepiphyseal dysplasia = Meyer's dysplasia = Gaucher's disease.

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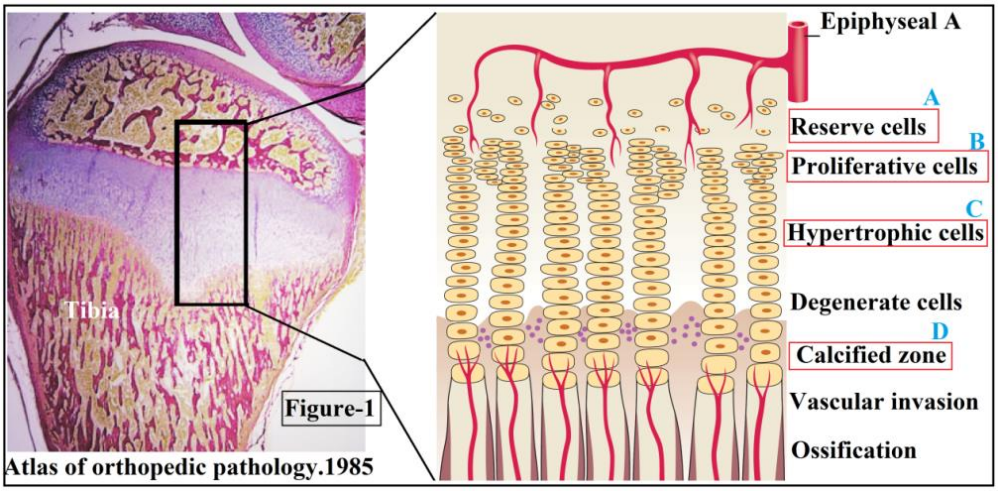
III. Slipped Capital Femoral Epiphysis

Syllabus: **1**- Definition **2**-Risk Factors **3**-Physical signs in SCFE **4**-Classification **5**-Radiological signs of SCFE **6**-Treatment policies & outcome

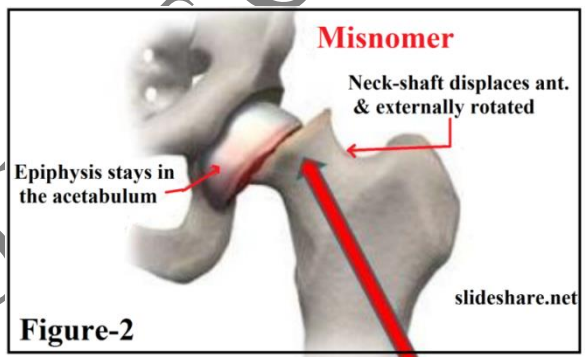
1-Physis layers: Figure-1

A. **Reserve layer**: resting cartilage cells.
B. **Proliferative zone**: actively growing cartilage cells.
C. **Zone of hypertrophy**: chondrocytes become swollen & vacuolated.
D. **Zone of calcification**: chondrocytes become calcified.



2-Definition

A displacement through the growth plate of the immature hip occurs during the rapid growth period in the hypertrophic zone of the physis
The femoral head remains in the acetabulum; the neck displaces anteriorly and rotates externally. **Figure-2**.



3-Epidemiology
a. *Most common* disorder of the hip in adolescents.
b.> *Male* (12–14 years)
c. *Unilateral* (80%),
d. *Obese* hypogonadal male (adiposo genital syndrome), or excessively thin and tall.

4-Etiology and PF: Idiopathic, but in general, weakness of perichondral ring.

Conditions that weaken the physis

* **Endocrinopathies** such as hypothyroidism, GH abnormalities hypogonadism,

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and hyper- or hypoparathyroidism.
If the patient is <9 years or >16 years and has a retarded bone age or short stature, consider endocrinopathy.
* **Systemic diseases** such as chronic RF; radiation therapy to the pelvis.

Explanation of hormonal theory of SCFE (PG)

Mechanical factors increase the load across the physis → SCFE.
(a) *Overweight children* (b) *Increase in femoral retroversion*.
(c) *Decreased femoral anteversion* and femoral neck-shaft angle.
(d) *Vertically oriented physeal plate*. (e) *Thinning of the perichondral ring*.

5-Pathology
The physis abnormally widened with the irregular organization.
The slip occurs through the hypertrophic zone of the physis.

6-Evaluation
1. Clinical presentation
limp and pain in the groin, hip, thigh, or knee region. (Unexplained antalgic limp).
i. Pain in the distal thigh and/or knee region in 30% of cases.
ii. Symptoms are usually present for weeks- months before a diagnosis made.

2. Physical examination, **Figure-3**
a. Abnormal gait (antalgic), waddling gait in bilateral cases and decreased ROM (flexion and internal rotation). b. Obligatory external rotation, i.e., ER of the hip as the hip is brought into flexion. c. Walking with the externally rotated foot.

3. Diagnostic tests
a. Plain radiographs—Standard AP and frog-leg lateral views of the pelvis recommended. **Figure-4**
* Widening and irregularity of physis (appears woolly, earliest sign).
* Decreased epiphysis height (slipped posteriorly).
* **The Klein line**, a line tangential to the superior border of the femoral neck on the AP view intersects the proximal femoral epiphysis in a normal hip. SCFE fail to intersect the proximal femoral epiphysis (**Figure 4**). Frog lateral radiographs are more sensitive in detecting an SCFE.

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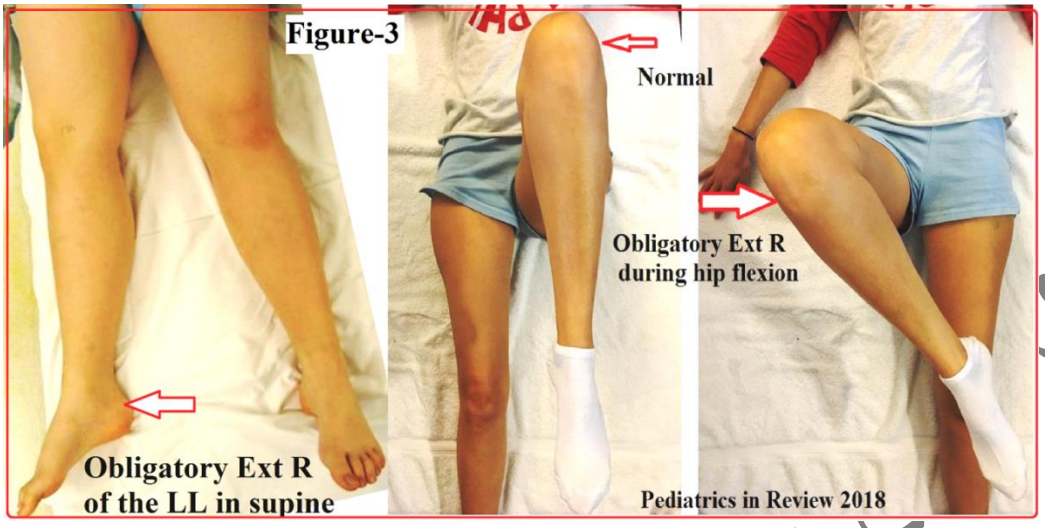
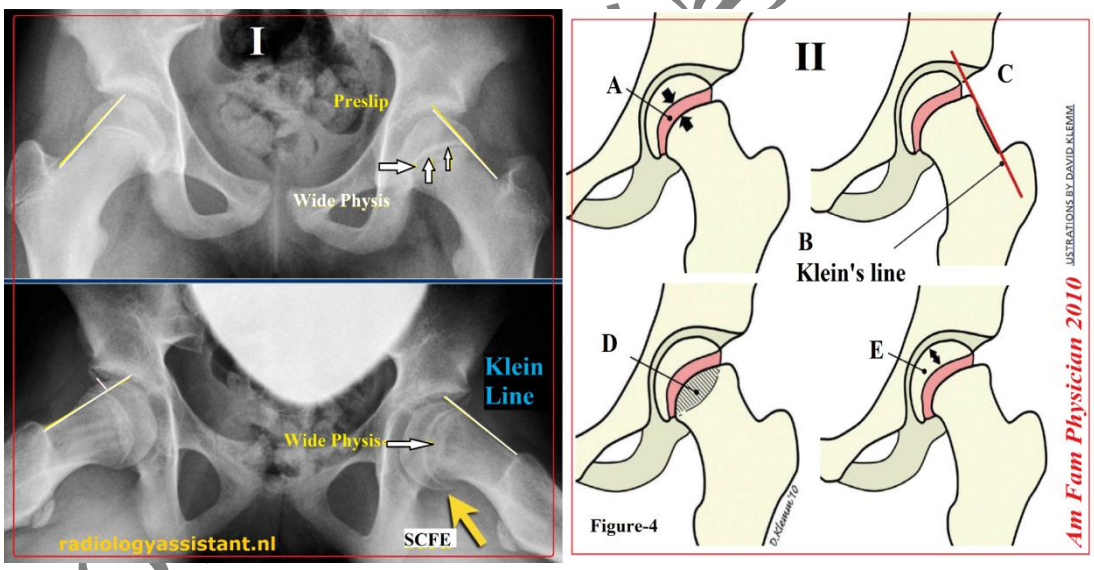


* **Metaphyseal blanch sign** (Steel sign) - Increased crescentic density in the metaphysis due to the overlapping of the metaphysis with the displaced epiphysis. (seen on AP view). **Figure-4**. (I-II)



***C*- Trethowan's sign** is when Klein's line does not intersect the lateral part of the superior femoral epiphysis on an AP radiograph of the pelvis.

***D- Steel sign**:* on anteroposterior radiography, a double density found at the metaphysis (caused by the posterior lip of the epiphysis superimposed on the metaphysis)

b. Other imaging studies

I. MRI is useful in diagnosing "pre-slip" hips. An abnormally widened physis with surrounding edematous changes on MRI are suggestive of pre-slip hips.
II. MRI is helpful in the evaluation of osteonecrosis afterward.
III. CT can be useful in characterizing the proximal femoral deformity, especially

during preoperative planning for reconstructive procedures, although it is generally not needed.

Classification of SCFE

1. The Loder classification (based on SCFE stability)

A. Stable (85%) if the patient can weight bear on the involved extremity (with or without crutches).

B. Unstable if the patient is unable to weight bear on the involved extremity.

- Sudden onset of pain, often after a fall or injury
- Inability to walk or bear weight on the affected leg
- Outward turning (external rotation) of the affected leg
- The discrepancy in leg length—the affected leg may appear shorter than the opposite leg

N.B: AVN in unstable hips 50%; in stable hips, 0%).

2. Radiographic classifications *(PG)*

7-Treatment

The primary goals of management are stabilization of the slip to prevent further progression and promotion of physeal closure.

Surgical

i. In situ screw fixation is the preferred initial treatment of SCFE.

Single cannulated AO screw (for grade I and II slips).

The starting point should be positioned anteriorly because the femoral epiphysis is posterior relative to the femoral neck. **Figure-6**

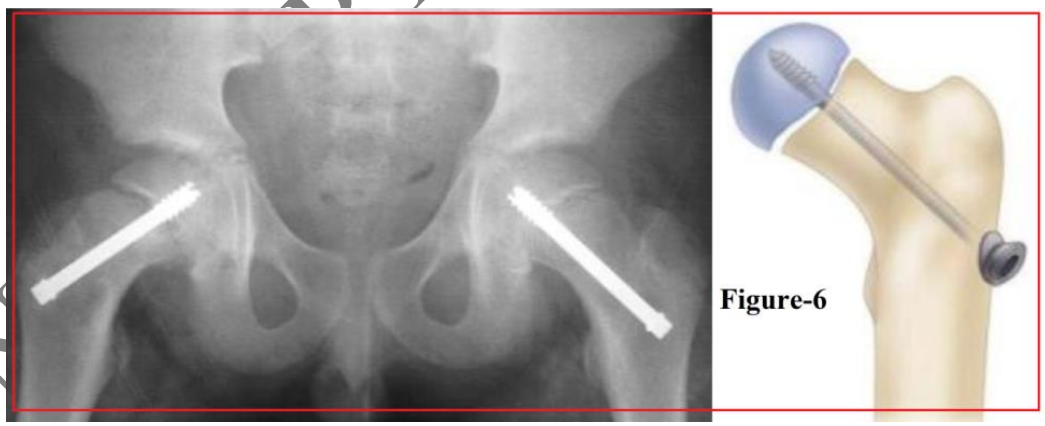


Figure-6

NO manipulation for reduction because it is associated with AVN

For a stable SCFE, a single-screw construct is usually adequate.

8-Complications of SCFE

i. Osteonecrosis (ON):

- 50% risk in unstable slips and 25% in severe slip.
- Screw placement in the posterior and superior femoral neck

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- With open reduction.

ii. Chondrolysis: due to pin penetration of the joint and multiple screw fixation. If penetration is recognized during surgery and corrected, the hip will be safe. Diagnosis indicated by virtually nil range of hip movement, hip pain, and a narrowed joint space. Confirm with MRI.

iii. Slip progression: in 1% to 2%

iv. Subtrochanteric #: with entry sites through the lateral cortex and those at or distal to the lesser trochanter.

v. Degenerative joint disease: 10% of patients with SCFE develop OA.

vi. Residual leg length inequality and rotational deformity
(severe slips that may require late corrective osteotomy)

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Quick notes

Perthes disease

Idiopathic disease >in males, interruptions in the blood supply. 15% bilateral.
Risk factors: small stature, immature bone age and poor urban environment with exposure to passive smoking.

C/P**:** Pain in the hip or groin referred to the knee and antalgic limp. Limited ROM (abduction-IR)

Investigations
AP pelvis and frog-leg lateral X-ray of the hips
1-Initial stage: widened joint space, later sclerosis of the head.
2-Fragmentation dead bone is resorbed resulting in subchondral # (crescent sign).
3- Re-ossification needs 18 months.
4- Remodeling**.**

Prognosis (risk of OA)
1-age > 6 years old at onset have a poor prognosis.
2-The more extent of femoral head involvement → outcome is worse.
3-Loss of Abduction → poor prognosis

Treatment
Contained hip within the acetabulum→ observation
Non contained → containment surgery (femoral osteotomy, pelvic osteotomy or a combination).

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Quick notes

SCFE

due to weakness in the growth plate (zone of hypertrophy) of the femoral head.
Types: Unstable slips → 50% risk AVN, stable → 0% AVN
Risk factors
1-Male > age 12years 2-Obesity 3-+Ve FH 4-Endocrinopathy including hypothyroidism and hypogonadism. 25% bilateral.

C/P: limp and groin pain →knee pain
X-ray: The frog-leg view is the most sensitive (Klein's line)
Treatment
Prevent further slip from by pinning in situ by a single screw
Prophylactic pinning other hip in child <10 years, or with a known endocrinopathy.

IV. Coxa Vara.

1-Definition: abnormally low femoral neck-shaft angle (<120°).
Evolution of the neck-shaft angle in the normal hip. **Figure-1**

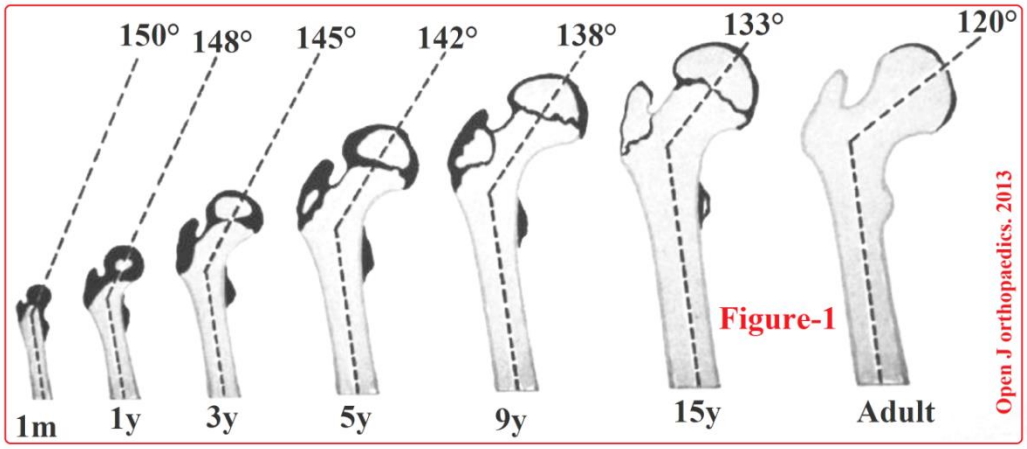

Figure-1

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