INTRODUCTION

Fibular Hemimelia is a congenital disorder characterized by partial or complete absence of the fibula. It is the most common deficiency of long bones which was first described by Gollier 1698 [1].

The term **melia** is derived from the Greek Melos ; meaning Limb.**Amelia** denotes absence of the entire limb.**Hemimelia** refers to loss of half of the limb [2] .

Children with congenital deficiency of the fibula may have Significant shortening of the involved extremity,leading to limb length discrepancy (LLD),which is the main problem of fibular deficiency [3,4] .

Fibular hemimelia syndrome includes a spectrum of problems rather than LLD,including variable degrees of fibular hypoplasia shortening of the tibia and femur,genu valgum and lateral femoral condyle hypoplasia, knee ligament laxity,tibial bowing, ball and socket ankle joint, tarsal coalition,clubfoot(TEV) and missing lateral rays of the foot[5,6,7] .

N.B:

About 15 percent of all patients with congenital absence of the fibula have associated deficiencies of the femur[8] .

Its incidence has been estimated, according to different authors, as being between 7.4 and 20 cases per million births[9]. Males are affected twice as much as females , unilateral more than bilateral and right more than left[10] **.**

The exact cause of fibular hemimelia is unknown in most cases, and the deformity usually occurs sporadically [11] .

Successful management aims to restore normal weight bearing and normal limb length so that the patient can walk with as normal gait as possible [12] .

THE AIM OF THE WORK

The aim of this work is to review the literature of fibular hemimelia regarding the incidence, etiology, types and evaluate the different alternatives of management and current treatment trends.

DEVELOPMENT OF LIMBS

Normal development of the limbs begins at the end of the fourth week after fertilization, with buds forming in mesoderm along the flank of the embryo (Fig. 1). The limbs develop in a proximodistal direction from the limb girdle to the digits. The proximal bones of the limb girdle and the humerus/femur form prior to the differentiation of ridge ectoderm, while development of the remaining bones and digits depends upon the apical ectodermal ridge (AER) [13] (Fig. 2) .

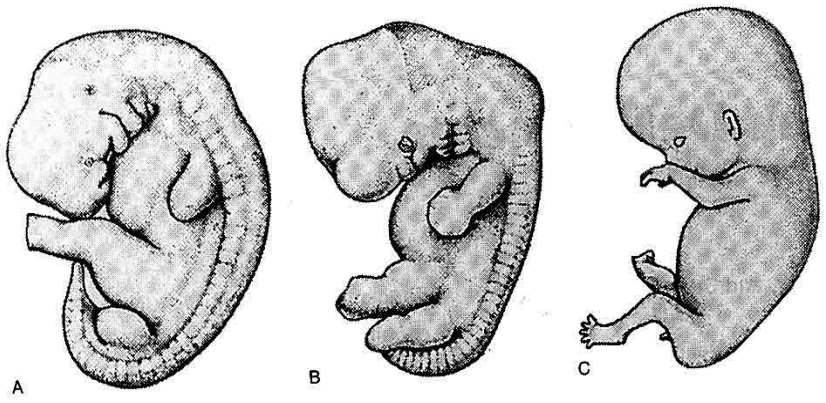


*Fig.No**(1)Development of the limb buds in human embryos. A.At 5 weeks.B.At 6 weeks.C.At 8 weeks. The hindlimb buds are less well developed than those of theforelimbs.(Sadler,2000)***

This ridge exerts an inductive influence on adjacent mesenchyme, causing it to remain as a population of undifferentiated, rapidly proliferating cells, the progress zone. As the limb grows, cells farther from the influence of the AER begin to differentiate into cartilage and muscle. In this manner development of the limb proceeds proximodistally[14].

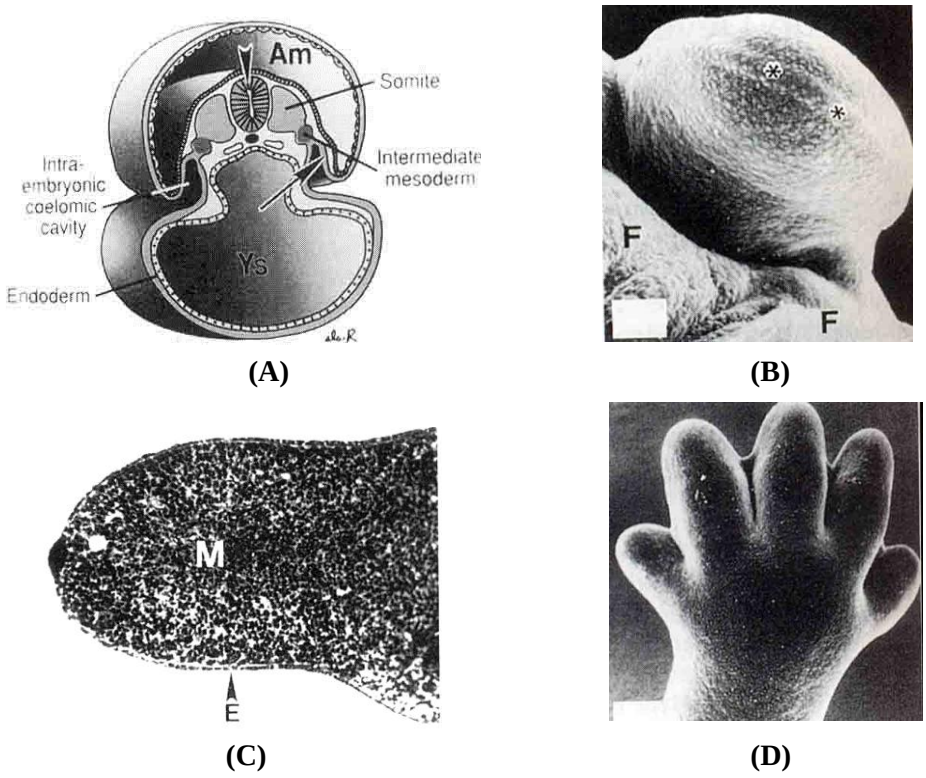


Fig. No (2)

A : Cross section through an embryo. The intermediate mesoderm signals the lateral plate mesoderm to initiate limb development. (Am:amnion; Ys:yolk sac).

B : The early limb bud. Asterisks indicate the location of the apical ectodermal ridge, which regulates proximodistal growth of the limb bud. (F:flank).

C : Longitudinal section of the limb bud showing the ectoderm (E) surrounding a core of undifferentiated mesoderm (M).

D : Digits are forming following programmed cell death in the AER in the spaces between the digits, each digit then continues to grow under the influence of its own AER***. (Herring, 2002)***

Limb development takes place over 4 weeks period from the fifth to the eighth week. The upper limbs develop slightly in advance of the lower limbs, although by the end of the period of limb development the two limbs are nearly synchronized. Development takes place as follows (Fig. 3):

Day 33. In the upper limb, the hand plate, forearm, arm, and shoulder regions can be distinguished. In the lower limb, a somewhat rounded cranial part can be distinguished from a more

tapering caudal part. The distal tip of the tapering caudal part will form the foot.

Day 37. In the hand plate of the upper limb, a central carpal region is surrounded by a thickened crescentic flange, the digital plate, which will form the fingers.In the lower limb, the thigh,leg ,and foot have become distinct.

Day 38. Digital rays are visible as radial thickenings in the digital plate of the upper limb. The tips of the finger rays project slightly,producing a prominent rim on the digital plate.Aprocess of programmed cell death will gradually sculpt the digital rays out of the digital plate to form the fingers and toes. The lower limb bud has increased in length and become demarcated from the trunk, and a clearly defined foot plate is apparent on the caudal side of the distal end of the bud.

Day 44 . In the upper limb, the margin of the digital plate is deeply notched and the grooves between the finger rays are deeper. The elbow is obvious. Toe rays are visible in the digital plate of the foot, but the rim of the plate is not yet prominent.

Day 47. The entire upper limb has undergone horizontal Flexion so that it lies in a parasagital rather than a coronal plane. The lower limb has also begun to flex toward a

parasagittal plane . The toe rays are more prominent , although the margin of the digital plate is still smooth.

Day 52. The upper limbs are slightly bent at the elbows, and the fingers have developed distal swellings called tactile pads. The hands are slightly flexed at the wrists and meet at the midline in front of the cardiac eminence. The legs are longer,and the feet have begun to approach each other at the midline.

Day 56. All regions of the arms and legs are well defined, including the toes .The fingers of the two hands overlap at the midline[15] .

During the seventh week the limbs begin to rotate , with the forelimb turning 90 degrees laterally (positioning the thumb laterally) and the hindlimb turning 90 degrees medially(positioning the big toe medially) digital rays appear in the hand and foot plates . By eighth week the limbs have rotated to their final position and all segments are complete, including the digits. During this time, ossification starts. By 12 weeks, ossification centers are present in all of the long bones [16] .

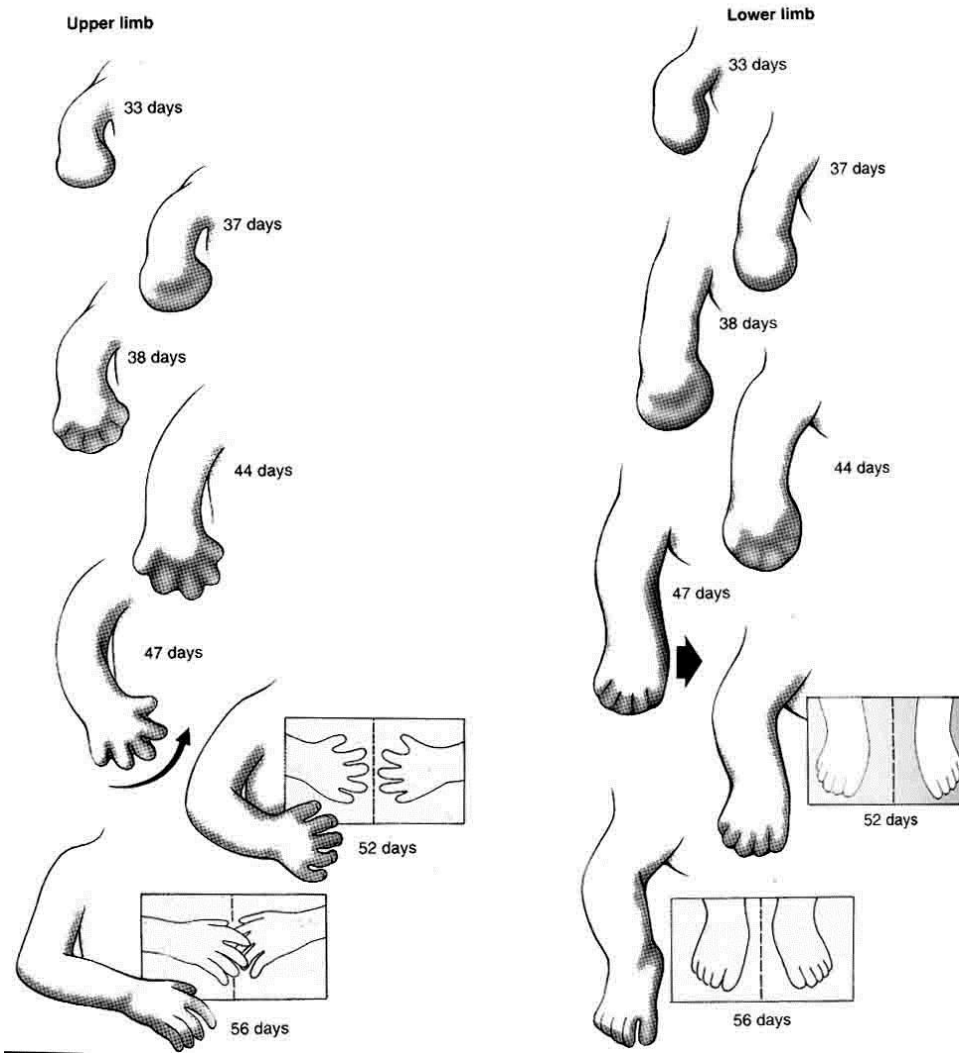


Fig.No (3)The development of the upper and lower limb buds occurs between the fifth and eighth weeks. Nearly every stage in the development of the lower limb bud takes place several days later than in the upper limb bud ***(Larsen,1998)*** ***.***

LOWER LIMB GROWTH

The lower limb grows more than the trunk. The cycle of growth in the lower limb is very predictable. There is a rapid increase in growth during the first 5 years of age, followed by a steady but slower growth from 5 years of age to the onset of the puberty. A slight growth spurt occurs during the accelerated velocity of growth at the beginning of puberty, and finally, early cessation of growth after the velocity peak. The femur grows more than the tibia with a constant relationship between the femur and the tibia throughout growth. Their proportions are set as early as age 5 years. Tibial length is 80% of femoral length. Fibular length is 98% of tibial length [17].

Femoral and Knee growth:

The proximal femoral physis accounts for about one third of the femoral growth whereas the distal physis accounts for about two thirds .As growth around the knee is the largest growth site of all, it accounts for about 65% of growth in the lower limb,about 37% for the distal femoral physis and about 28% for the proximal tibial physis . The distal and proximal femoral growth plates grow approximately 1cm and 0.7cm,respectively each year whereas the tibial growth is approximately 1.3cm each year. During puberty,this rate of growth increases to about

1.2cm for the distal femur , 0.8cm for the proximal femur and 1.6cm for the tibia per year [9,18].

Tibial and Fibular growth:

During the following 12 h of development a region of flattened cells becomes apparent between the diaphysis and distal epiphysis of the fibula and toluidine-blue staining shows that these cells, unlike normal flattened cells in long-bone rudiments, secrete little metachromatic matrix . At about the same time, an asymmetric protrusion develops from the lateral aspect of the distal epiphysis of the tibia which enlarges and joins with the distal epiphysis of the fibula, which separates from the rest of the fibula in the region of the flattened cells. Thus the distal epiphysis of the fibula becomes part of the distal tibia – it becomes the fibulare of the tibiotarsus[19] .

In addition to the loss of the distal epiphysis there is a reduction in the number of cells across the diameter of the fibula whereas the cell number across the tibia remains fairly constant . This leads to an increase in the ratio of tibia/fibula diameters[20] .

The onset of osteogenesis occurs at the same time in both tibia and fibula, and the two subperiosteal osseous collars develop adjacent to one another . However, the bony collar in the fibula

is eccentric since the distal epiphysis is missing. Also, the patterns of ossification differ markedly between the two elements. In the tibia the osteogenic process progresses in the manner where vascular buds penetrate the hypertrophic cartilage in the central diaphysis . These represent the initial sites of cartilage resorption which progress towards both epiphyses. At the same time, the osseous collar develops along the diaphysis, the extent of which invariably reflects the limit of adjacent cartilage hypertrophy. Both of these events appear to allow further sites of resorption to be established nearer the epiphyses, but these seem to play a secondary role to the rapid resorption originating at the centre of the rudiment. There is little or no endochondral ossification occurring at these sites of resorption. The initial bony collar of the tibia is succeeded by a number of transverse trabeculae separating the two, then a third osseous cylinder is evident . Thus the diameter of the element is increased markedly.

In contrast, only a single bony collar is ever laid down in the fibula and cartilage resorption is retarded even though hypertrophy is extensive . However, a number of resorption sites are present along the whole length of the hypertrophic zone . These quickly occupy the diameter of the cartilage and extend longitudinally, eventually fusing to form a marrow cavity . Endochondral ossification is occasionally observed. The failure

of the fibula to form successive subperiosteal bony collars results in very little change in diameter[20] .

Foot growth:

The length of the foot is relatively large during intrauterine life with relative diminution throughout growth. At birth, the foot is approximately 40% of its final size. The foot is the first segment of the musculoskeletal system to show pubertal growth change. The growth spurt of the foot occurs a few months before the start of puberty. The foot is also the first musculoskeletal segment that stops growing at maturity. Foot growth stops about 3 years before the end of skeletal maturation. So arthrodesis of the foot at the beginning of puberty will have no significant impact on the length of the foot.

The foot represents 15% of the standing height in both girls and boys at skeletal maturity, this amount must be taken into consideration during lower limb length assessment, especially in conditions that have foot manifestations,e.g., fibular hemimelia [18] .

INCIDENCE

Congenital longitudinal deficiency of the fibula is the most common congenital defect involving the long bones (followed by aplasia of the radius, femur, tibia, ulna, and humerus). It consists of spectrum of anomalies ranging from mild fibular shortening to bilateral involvement with associated defects of femur, tibia, ankle, and foot [21] .

Its incidence has been estimated, according to different authors, as being between 7.4 and 20 cases per million births[24]. Males are affected twice as much as females, unilateral more than bilateral, and right more than left [9,21] .

ETIOLOGY

By seventh weeks of embryonic life, all parts of the upper and lower limbs are essentially completely formed. Most limb deficiencies occur early in the period of limb morphogenesis, when there is rapid proliferation and differentiation of cells and tissues. This is"sensitive period" of limb formation peaks during the fifth and sixth weeks after fertilization. Thus, major malformations (such as absence of a long bone) appear by seventh weeks of fetal development [16,19] .

The exact cause of limb deficiency is unknown. But there are

at least three ways in which limb deficiencies can be caused: errors in the genetic control of limb development,disruption of the developing arterial supply,and intrauterine amputation from amniotic bands . Modern genetics has shown that the development of the limb is a complex phenomenon that requires the precise interaction of a large number of genes and their effects[11,19] .

Many drugs are known as teratogens ; however the only drug specifically identified with a large number of limb abnormalities is thalidomide [22] . Hereditary appears to play a minor role in congenital absence of the fibula [1] .

Although a number of supposed causes exists , some have Postulated that interference with limb-bud development plays an important role in postaxial hypoplasia of the lower extremity as follows :

The distal tip of the limb bud (apical ectodermal ridge,AER) considers as a source of signals essential for limb development.

When the AER is removed at an early limb bud stage,the most proximal skeletal segment (thigh) forms, but middle (lower leg)

and distal(ankle and foot) segments are absent. When it is removed at a slightly later stage, only the distal segment is missing, Swapping AERs of early and late stage limb buds has no effect on limb skeletal pattern , establishing that AER signals are permissive but not instructive for limb development[11,19] .

These data show that when Fgf4 (fibroplast growth factor 4) and Fgf8 (fibroplast growth factor 8) are never expressed in limb bud ectoderm, there is virtually complete failure of skeletal development; however, when they are transiently active at very early limb bud stages, the most proximal segment develops normally but both the middle and distal segments are severely hypoplastic. Thus , a decrease in limb bud size is observed when Fgf8 is inactivated before it is expressed (in hindlimb buds) . These data provide strong evidence for a previously unknown function of FGF8 in influencing the number of cells present when limb budding first becomes apparent . Thus, our data suggest that hindlimb buds are smaller than normal because they start out smaller, and then decrease further in size as a consequence of proximal cell death. Our finding that embryos lack hindlimbs provides formal genetic evidence that FGF4 and FGF8 together are required to form a limb. In their absence, the other AER-FGFs (FGF9 and FGF17) cannot rescue limb development[19] .

Molecular analysis of mutant hindlimb buds, in which functional Fgf4 and Fgf8 RNAs are never expressed,

demonstrated that these AER-FGFs are not required for AER formation, but are essential for expression of numerous genes in the underlying mesenchyme[11,19] .

PATHOLOGIC ANATOMY

In fibular hemimelia there is a widespread pathology throughout the limb has been noted, even in mild cases of fibular deficiency. During the fetal period, the fibular field of the limb bud controls development of the proximal femur, explaining the frequent association of femoral abnormalities. The remaining associated abnormalities around the knee, leg, ankle, and foot also are related to the fibular field of the lower limb bud. Therefore, postaxial hypoplasia of the lower extremity is a descriptive term for the collection of abnormalities present[23] (Fig. 4).

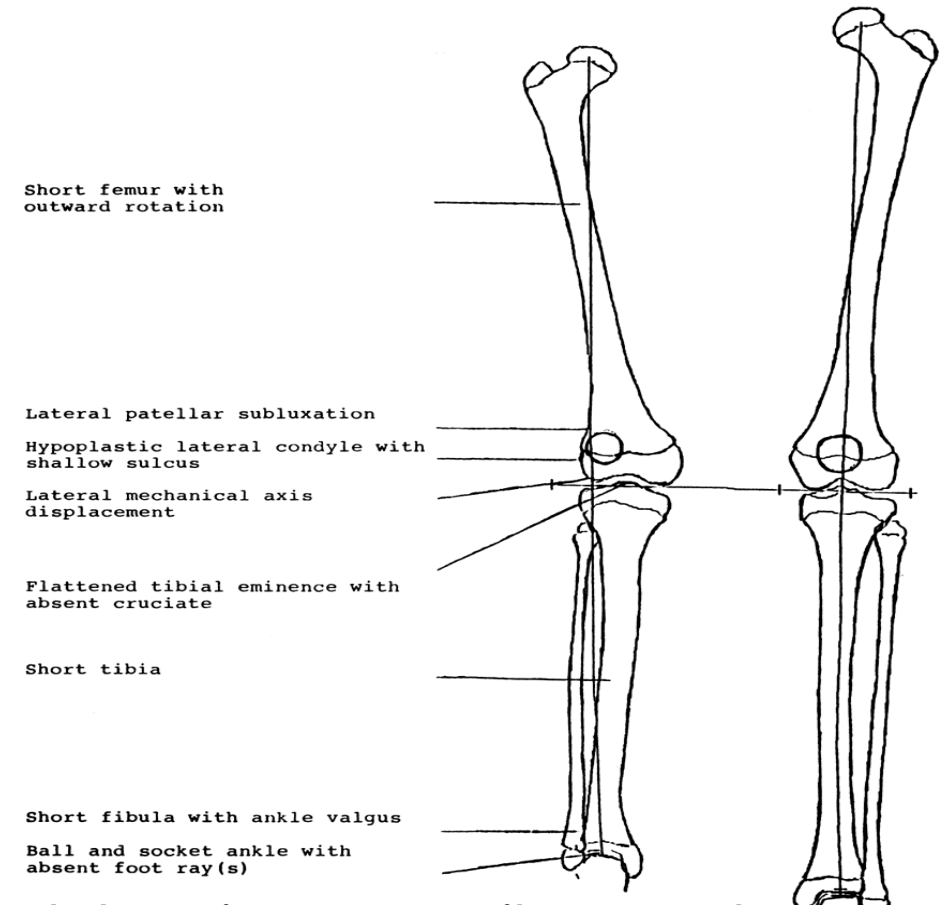


Fig. No (4) Digitalized tracing of anteroposterior view of lower extremities demonstrating features common to postaxial hypoplasia. ***(Stevens and Arms, 2000)***

Leg:

The absence of the fibula may be complete or incomplete. With incomplete absence, the ossification may be considerably delayed, and early roentgenograms may be misleading. The deficient part of the fibula usually replaced by a fibrocartilaginous band (Fig. 5).

Searle et al. 2004, have noted a number of patients with features of fibular hemimelia with radiographically normal fibula. The study was undertaken to further define this group. A review of hospital records and radiographs over a 72-year period identified 149 limbs in 123 patients with features of fibular hemimelia syndrome[(24)] .

Sixteen limbs in fourteen patients had shown the findings of fibular hemimelia with radiographically normal fibula. Thirteen of sixteen had absent lateral rays (a classic feature of lateral limb deficiency) with either ball and socket ankle joint, tarsal coalition or both. Six of the thirteen had limb shortening. Three limbs in three patients did not have absent lateral rays, but had at least two other features of fibular hemimelia syndrome. All of these three limbs had ball and socket ankles and tarsal coalitions and two had shortening. These patients represent a mild subset of fibular hemimelia syndrome and the authors propose that they are classified as type 0 fibular hemimelia. These patients represent 11% of total fibular hemimelia patient population[(24)] .

The peroneal tendons, the Achilles tendon, the posterolateral ligaments, and the capsule of the ankle and subtalar joints are shortened and contribute to the deformity.

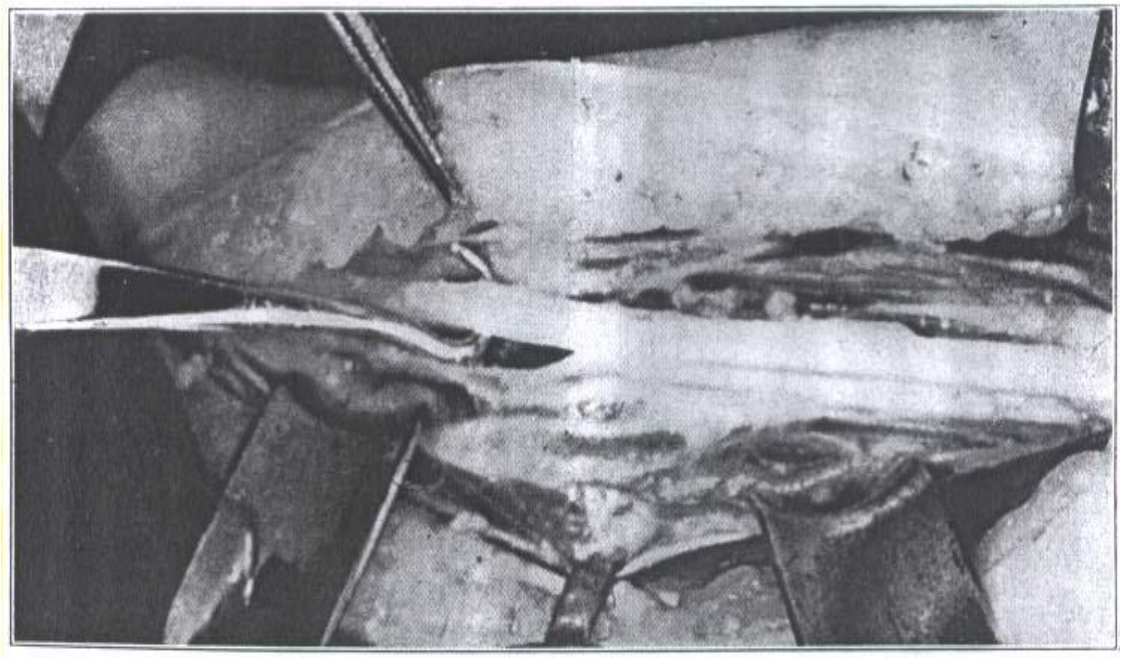
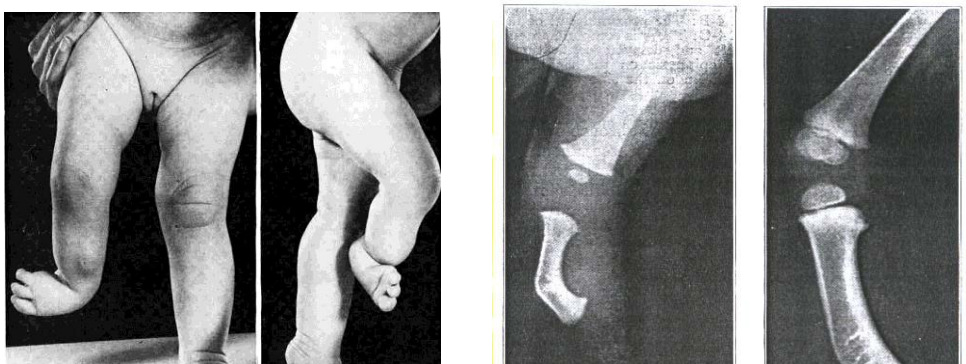


Fig. No (5) Fibular reminant. The fibrocartilaginuous band is continuous with the lower end of the fibula. ***(Farmer and Laurin, 1960)***

Children with fibular hemimelia show anteromedial bowing of the tibia (tibial kyphosis), with thickening of the cortex on the concave side and a dimple in the skin over the apex of the bow (Figs. 6 and 7). The tibia is usually short which is the main cause of lower limb shortening in fibular deficiency cases[(5)] **.**



Ankle and foot:

The ankle may appear normal in some patients with mild deficiencies. The classic appearance is the ball and socket ankle joint (Fig. 8), in which the tip of the fibula extended to the ankle, but did not participate in the articulation. There is disagreement about the origin of this abnormality, some authors suppose that it is congenital in nature, while others suppose that it develops secondary to the tarsal coalitions. If caused by the tarsal coalitions, it is difficult to explain its absence in tarsal coalitions without fibular deficiency[(6)] .

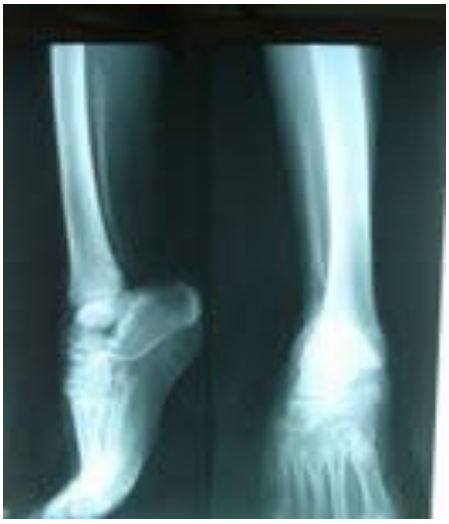


Fig. No (8) A ball and socket ankle joint is a common finding in patients with fibular hemimelia. It is associated with LLD and tarsal coalition. ***(Holmstrom, 2004)***

With total absence of the fibula there is a severe valgus or instability of the ankle joint (Fig. 9) due to deficient lateral malleolus, which may require Syme disarticulation or Gruca reconstruction[25] .

Tarsal coalitions and associated anomalies of hindfoot are also reported in fibular deficiency cases. The most common pattern is talocalcaneal union (Fig. 10), which represents the failure of the embryonic precursors of the talus and the calcaneus to segment completely from one another. These coalitions are most often cartilaginous in young children and thus are visible only after there has been progressive ossification [26] .

Fig. No (9) A significant valgus hindfoot is due to the shortened fibula In an 8-years old boy. *(Holmstorm, 2004)*

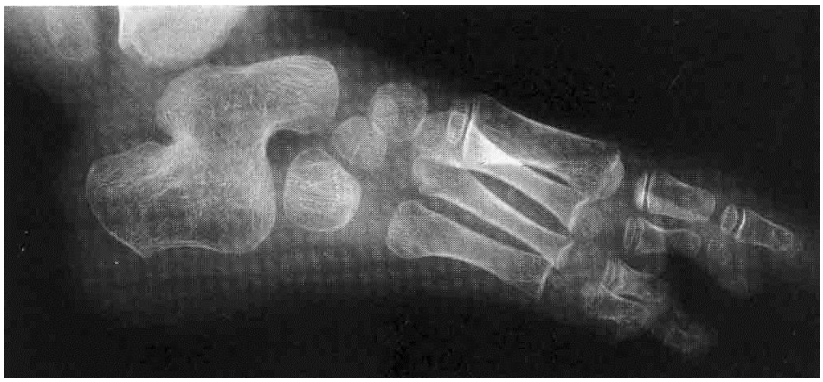


Fig. No (10) Lateral radiograph of the foot of a five years old boy who had type-III fibular hemimelia and four rays. A definite talocalcaneal coalition can be seen***. (Grogan, 1994)***

The presence of a tarsal coalitions contributes to the deformity in the feet of the affected children. For example the valgus deformity is more severe and more rigid in the feet that had a talocalcaneal coalitions compared with those that did not have such coalitions. Other types of tarsal coalitions may be present such as, pantalar coalition, calcaneocuboid coalition, and calcaneonavicular coalition[(27)].

The foot may appear normal, but more frequently is missing one or more lateral rays (Fig. 11). It is a common observation in cases of fibular hemimelia, in more severely involved limbs four or five rays may be absent[28].

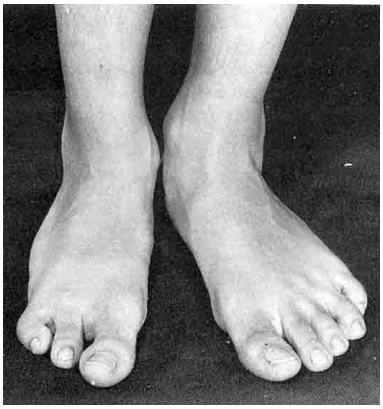


Fig. No (11) A three-ray foot in patient with fibular hemimelia. ***(Maffulli and Fixsen, 1991)***

The association of clubfoot (TEV) and fibular hemimelia has been sporadically reported in the past (Fig. 12 and 13). Traditionally the foot is positioned in equines or equinovalgus. **Caskey and Lester. 2002,** reported that, the association between clubfoot deformity and fibular hemimelia syndrome is more common than previously reported. Vascular anomalies has been described in clubfoot as well as fibular hemimelia syndrome[10,28] .

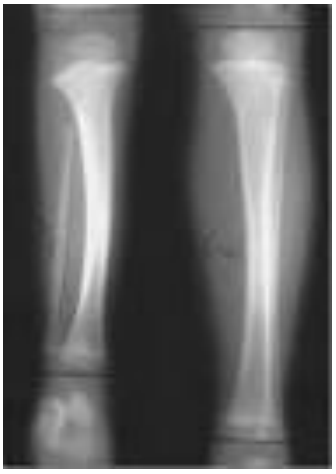


Fig. No (12) A-P radiograph of a patient with FH ,comparing right and left tibia. Note obvious tibial shortening on the right. *(Caskey and Lester, 2002)*

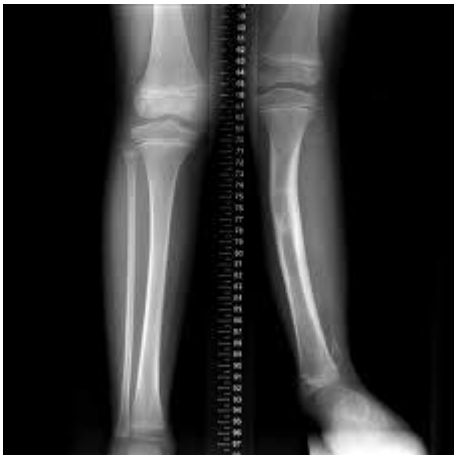


Fig. No (13) A-P radiograph of a patient with fibular hemimelia show club foot deformity on the left. *(Holmstrom, 2004)*

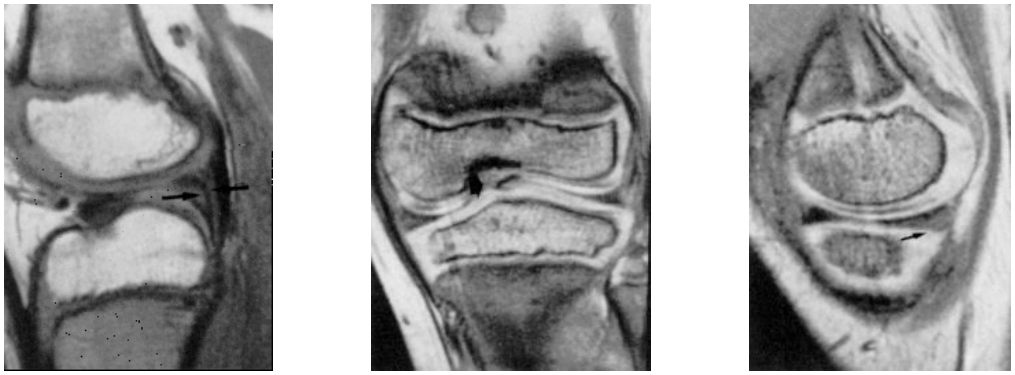
Knee:

The abnormality extends into the knee joint, where the condylar notch of the femur is shallow, the tibial spines small, and there is laxity in the anteroposterior direction.

There is also a valgus deformity of the knee (genu valgum) which is not associated with varus/valgus instability. Although the distal femur has not been considered part of the fibular development field, the lateral femoral condyle may be relatively hypoplastic. This is reflected radiographically by a decreased condylar height ratio and lateral displacement of the mechanical axis. The natural history of this deformity is that it usually worsens with growth. In some cases, the degree of the valgus is more severe than can be explained by the smaller lateral femoral condyle alone[29].

It is widely recognized that these patients may have cruciate deficient knees. Many authors reported anteroposterior laxity of the knee in the majority of children with fibular hemimelia. **Roux and Carlioz. 1999,** has been reported that clinical examination of 69 knees in 66 patients with fibular hemimelia found anteroposterior laxity in the majority of cases (84%) with positive Lachman's test and an anterior glide in (90%) of cases. The jerk test was found to be positive in only (23%) of cases, so lesions in the posterior cruciate ligament are clinically less common than the lesions of the anterior one.

Investigations by MRI and arthoscopy showed absence or hypoplasia of the anterior cruciate ligament (ACL) in 95% of cases and absence or hypoplasia of the posterior cruciate ligament (PCL) in 60% of cases (Fig. 14). The posterior cruciate ligament lesion is never more severe than that of the anterior cruciate ligament.



(A) **(B)** **(C)**

Fig. No (14)

A : The posterior cruciate ligament is present but is hypoplastic and runs an abnormal course that is too vertical.

B : The anterior cruciate ligament is completely aplastic.

C : The posterior horn of the medial meniscus gives an abnormal signal indicating a congrnital or aquried dysplasia. ***(Roux and Carkioz, 1999)***

It is important to take these abnormalities into account in the treatment program, because of the risk of posterior subluxation during femoral elongation in patients with an abnormality of PCL[(30)].

In patients with fibular hemimelia the patella is usually small, and high-riding. Symptomatic,recurrent patellar subluxation may be present with lateral displacement of the patella (Fig. 15). Rarely the patella may be completely absent in some cases[31].

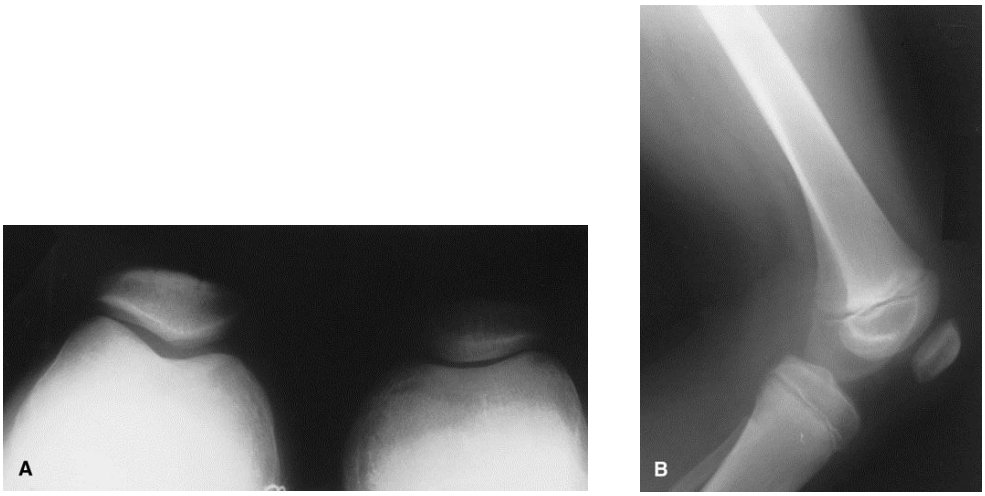


Fig. No (15)
A: Merchant-view radiograph of knees with unilateral hypoplastic lateral femoral condyle, shallow sulcus, and laterally displaced small patella on the left side .
B: Lateral knee radiograph with small,abnormally high patella and hypoplastic lateral femoral condyle. ***(Stevens and Arm, 2000)***

Acetabulum and femur :

The children with congenital fibular deficiency may be present with varying degrees of femoral shortening. Several authors discussed the association of proximal femoral focal deficiency (PFFD) or proximal femoral varus in conjunction with fibular

hypoplasia (Fig. 16). Indeed it has been stated that the proximal femur is part of the "fibular field" that is critical to limb-bud development and growth. Varus deformity may be at the cervical, intertrochanteric, or subtrochanteric level[32] .

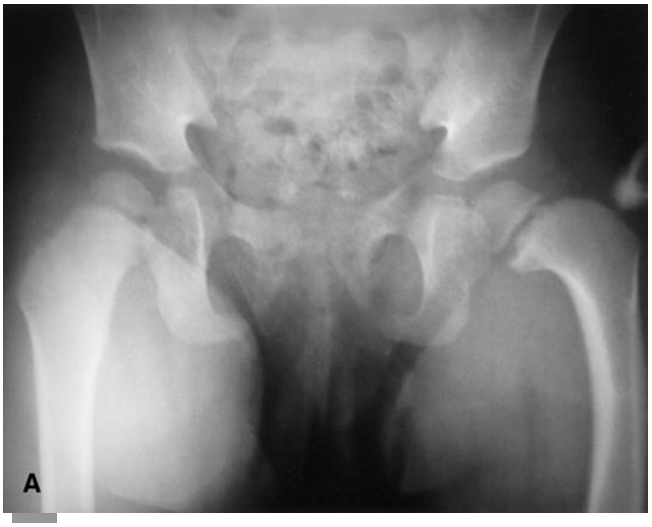


Fig. No (16) AP pelvis radiograph with varus deformity of the proximal left femur. ***(Stevens and Arms, 2000)***

The femoral shortening may be slight to severe, varying from a few centimeters of shortening to a true PFFD. **Amstutz, 1972** reported femoral deficiency in 15% of those with fibular deficiency , whereas **Bohne and Root, 1977** reported femoral deficiencies in almost two-thirds of their patients. **Kalamchi, 1989** noted that 70% of type I and 50% of type II deficiencies were associated with shortening or deformity of the femur[33] .

Dysplasia of the acetabulum may be present (Fig. 17), a procedure to redirect the acetabulum may be indicated before undertaking femoral lengthening[34].

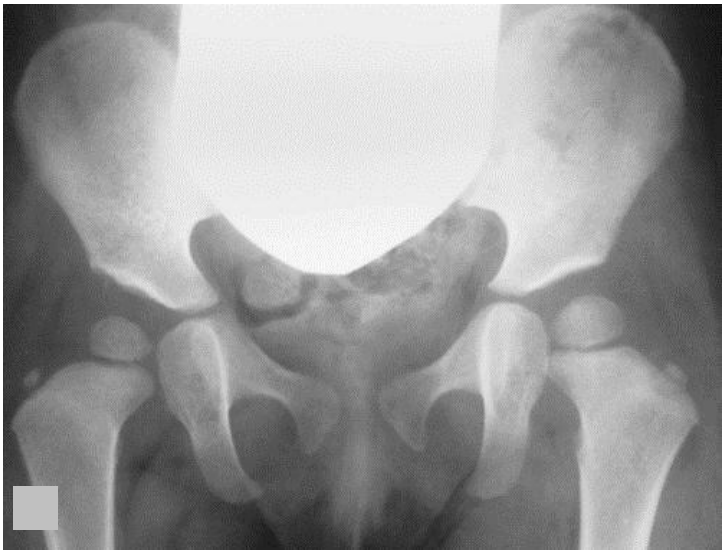


Fig. No (17) AP pelvis radiograph with right hip demonstrating abnormal acetabular index, poorly developed teardrop, and inadequate sourcil.
(Stevens and Arms, 2000)

Other associated anomalies:

Congenital anomalies of the upper limbs have been reported in children with fibular hemimelia. These anomalies may include, defects of the hand, usually the ipsilateral hand. The most common of these defects include absent ulnar rays and syndactyly of the remaining digits, congenital dislocation of the head of the radius, and an entire upper limb may be absent. Systemic involvement is uncommon, but cardiac and renal anomalies may be present[31] .

CLASSIFICATION

It has been described by many authors:

FRANTZ AND O'RAHILLY CLASSIFICATION :

This classification system proposed by **Frantz and O'Rahilly** in **1961** continues to be widely used method of grouping congenital limb deficiencies. Extremity anomalies are categorized as either terminal or intercalary deficiencies. Fibular hemimelia may be an intercalary deficiency with a normal foot or a terminal deficiency with absent rays of the foot. The deficiency is always paraxial (i.e, longitudinal)[(2)].

N.B:

Terminal deficiencies are those in which the entire segment of a limb distal to and in line with the deficit is absent. Intercalary deficiencies are those in which the middle part of a limb is absent but the portions proximal and distal to the affected segment are present. Terminal and intercalary deficiencies may be transverse, in which the entire width of the limb is affected, or paraxial (i.e, longitudinal), in which only the preaxial or postaxial part of the limb is involved[(33)] (Fig 18) .

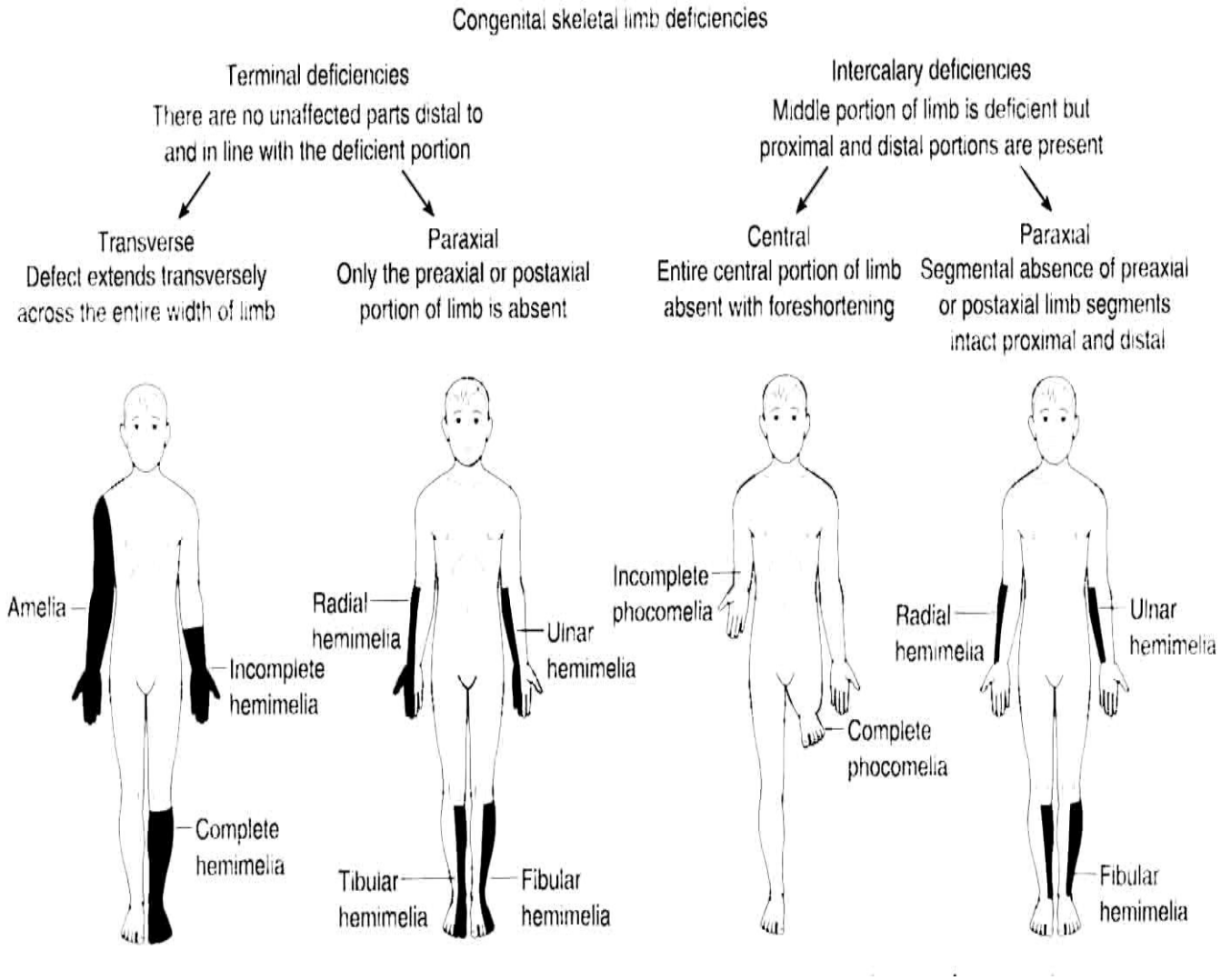


Fig. No (18) Diagramatic representation of the Frantz and O'Rahilly classification of congenital limb deficiencies. ***(Morrissy et al., 2006)***

COVENTRY AND JOHNSON CLASSIFICATION :

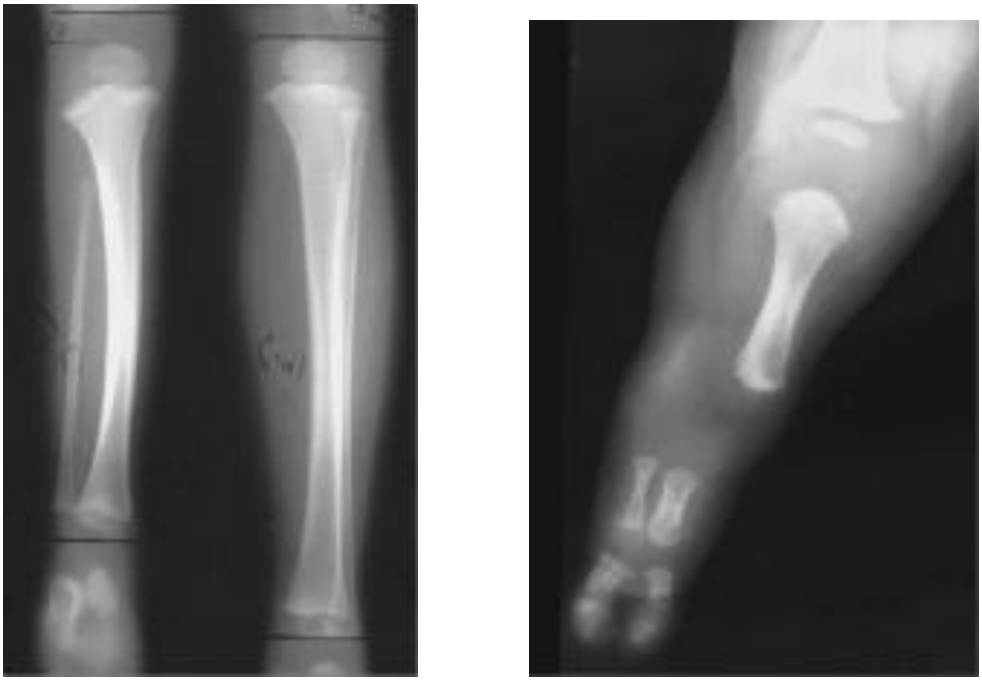
This system classifies patients into 3 groups. Partial absence of the fibula is classified as type I and complete fibular absence as

type II (Figur 19). Type III includes bilateral absence of the fibula and cases of other skeletal abnormalities associated with unilateral fibular absence[1] (Table I) .

Table I : Coventry and Johnson classification

(Coventry and Johnson, 1952)

Type I	Type II	Type III
• Partial unilateral absence of the fibula. • Little or no tibial bowing. • Minimal leg length discrepancy (LLD).	• Unilateral complete absence of the fibula with femoral shortening. • Tibial shortening and anteromedial bowing.	• Bilateral complete absence of the fibula. • Associated with other congenital defects. • The prognosis for functional development is poor than type I and type II.



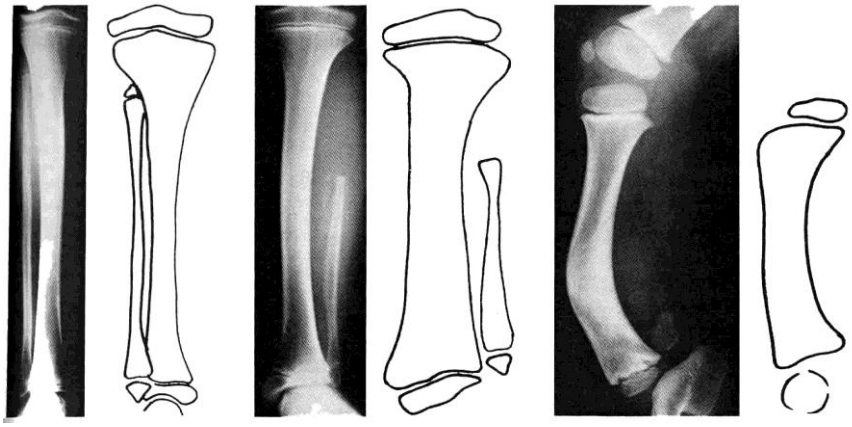
(A) (B)

Fig. No (19) **Coventry and Johnson classification of FH.(Coventry and Joh,1952)**
A:Type I FH;partial absence of fibula.
B:TypeII FH;complete absence of fibula.

ACHTERMAN AND KALAMCHI CLASSIFICATION :

This system classifies fibular hemimelia based on the degree of fibular deficiency present (Fig 20). If any portion of the fibula is present, it is classified as type I, and type I is further subclassified into type IA and type IB. In type IA the epiphysis of the proximal fibula is distal to the level of the tibial growth plate and the physis of the distal fibula is proximal to the dome of the talus. In type IB the fibula is shorter by 30% - 50%, and distally the fibula does not provide any support at the ankle

joint. If the fibula is completely absent, the deformity is classified as type II[31] (Fig 21) (Table II) .

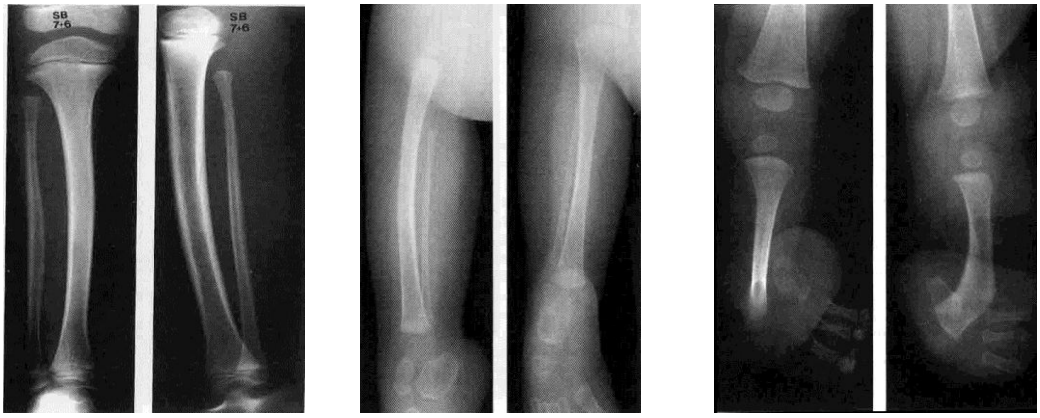


A *B* *C*

Fig. No (20) Achterman and Kalamchi Classification of FH **.**
A: Type IA fibular hypoplasia.
B: Type IB fibular hypoplasia.
C: Type II fibular hypoplasia. ***(Achterman and Kalamchi, 1979)***

Table II : Achterman and Kalamchi classification of fibular hemimelia(Achterman and Kalamchi, 1979)

	Type IA	**Type IB**	**Type II**
	The proximal fibular epiphysis is distal to the level of the tibial growth plate with the distal fibular epiphysis proximal to talar dome.	The proximal fibula is absent for 30-50% of its length, the distal fibula is present but does not adequately support the ankle joint.	The fibula is completely absent.
Tibial discrepancy	6%	17%	25%
Total LLD at maturity	12%	18%	19%



(A) **(B)** **(C)**

Fig. No (21)

A: Anteroposterior and lateral radiographs of 7-years old boy with type IA fibular deficiency of Achterman and Kalamchi. Note the proximal shortening of the fibula and the ball-and-socket ankle joint is easily seen.

B: Type IB fibular deficiency, in which the proximal fibula is missing.This type is often associated with proximal focal deficiency, as it is in this child.

C: Anteroposterior and lateral radiographs of type II fibular deficiency, in which the entire fibula is missing. ***(Morrissy et al., 2006)***

FUNCTIONAL CLASSIFICATION OF FIBULAR DEFICIENCY :

It is also known as **Birch classification, 1998.** The current classification schemes of fibular hemimelia concentrating on the radiographic appearance of the fibula do not adequately aid management of these patients. This classification system for fibular hemimelia could serve as a better guideline for management decisions for this disorder. This classification based on the presence or absence of a functional foot and overall limb shortening relative to the contralateral side (irrespective of the relative contributions of femoral and tibial shortening, or bilateral disease). The classification (with treatment guidelines) is :