

**Republic of Iraq
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Club foot treatment in children less than one year

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يَعْلَمُونَ إِنَّمَا يَتَذَكَّرُ أُولُو الْأَلْبَابِ}

صدق الله العلي العظيم

سورة الزمر : الآية 9

الاهداء

الاهداء اولاً الى وجه الله تعالى

في جميع مراحل الحياة اناس يستحقون منا الشكر والتقدير واولى الناس
بالشكر والدي العزيز الذي جرع الكأس فارغاً ليسقيني قطرة حب
والدي العزيزة التي وضعتني على طريق الحياة وكان لها الفضل الكبير لنجاحي
والى جميع من وقفوا بجاني وساعدوني وبالخصوص الى اصدقائي والى جميع
اساتذتي الكرام بوجه عام والى المشرف على البحث بوجه خاص الذي كان
له دور كبير في اعطائي المعلومات القيمة اهدي لكم بحث تخرجي المتواضع
واتمنى ان تحوز على رضاكم

الشكر والتقدير

الحمد لله الذي هدانا واعدنا وامدنا والهمنا الصبر على المشاق ووفقنا لما نحن
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فله الحمد والشكر ابتداءً وانتهاءً

وارفع كلمة الشكر الى الدكتور عمار نجم

وفقه الله فقد كان سنداً لي على طول الطريق

والى كل من مد يد العون لي من قريب او بعيد

وقبل ان امضي اقدم اسمى ايات الشكر والامتنان والتقدير والمحبة الى الدين

مهدوا لي طريق العلم والمعرفة

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Abstract:-

clubfoot is a complex deformity that affects the muscles, ligaments, bones, and joints of the developing foot and ankle. The ankle is turned inward, and the toes are pointed inward toward the opposite knee. Clubfoot is a visible deformity that is easily identified. It is classified into four types: equinus, midfoot cavus, forefoot adduction, and hind foot varus. Several hypotheses have been proposed to clarify its etiology. Mechanical Factors in Utero , Neuromuscular Defect , Primary plasma Defect , Arrested Fetal Development. There are many classifications of clubfoot in the literature. Dimeglio defined four types of club feet based on joint motion and ability to correct deformities . Club foot assessment criteria include: ankle motion, bimalleolar angle, triceps surae weight, heel, forefoot, flexor hallucis longus, painful gait, subtalar pain, shoe wear, and sports. Treating successfully clubfoot and understanding the pathomechanics has always been an unsolved point for modern medicine. Clubfoot is usually diagnosed immediately after birth simply by looking at the foot. Most Orthopaedic surgeons have agreed that the initial treatment of a clubfoot should be nonsurgical and start as soon as possible after birth. Care preferences have shifted to a non-surgical approach through the Ponseti procedure. The Ponseti technique's correction method is split into two phases: The Treatment Phase , The Maintenance Phase . When the final cast is removed, the newborn is put in an orthosis, or harness, which keeps the foot in the right position. After the casting process in the Ponseti technique, the object of this splinting is to keep the foot in the correct position, with the forefeet set apart and pointing upward. This is done with a belt made up of shoes attached to a bar.

The aim of this study: is to identify clubfoot, its causes, how to diagnose it, and the most important techniques used in its treatment

1. Introduction

Hippocrates described clubfoot, also known as Congenital talipes equinovarus (CTEV), around 300 B.C. He distinguished between two types of clubfeet: congenital and acquired in childhood. The term talipes equinovarus derives from the Latin words talus (ankle) and pes (foot); equinus (the heel in plantar flexion) and varus (inverted and adducted).(1)

congenital clubfoot, also known as talipes equinovarus, is a complex deformity that affects the muscles, ligaments, bones, and joints of the developing foot and ankle. The ankle is turned inward, and the toes are pointed inward toward the opposite knee.(2)

Many of the foot bones are normally present, but they are out of place. Many of the foot and leg muscles are present, but some are smaller and weaker than they should be. Tendons and ligaments are tense, especially behind the ankle and along the instep.(2)club foot affects about 1-2 out of every thousand live births. It is one of the most common birth defects that affects the musculoskeletal system (Figure 1).

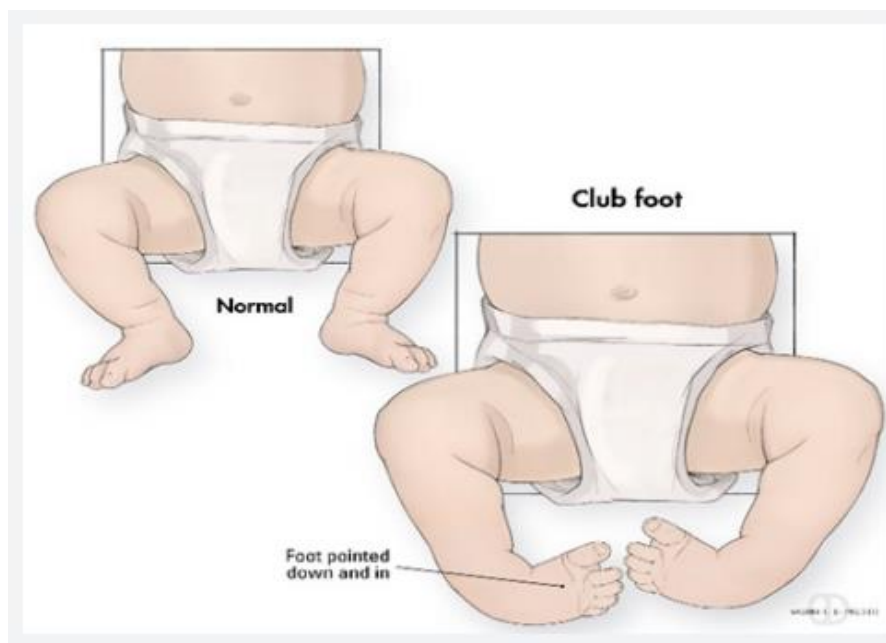


Figure 1: comparison between normal and club foot

In about half of the cases, the involvement is bilateral, and in unilateral cases, the right side is more frequently affected than the left . Males are twice as likely as females to have idiopathic clubfoot.(1)

Clubfoot is a visible deformity that is easily identified. It is classified into four types: equinus, midfoot cavus, forefoot adduction, and hind foot varus. If left untreated, children with clubfoot walk on the sides and/or tops of their feet, resulting in callus formation, skin and bone infections, significant mobility limitations, limited work opportunities, and inability to wear regular shoes.(3)

It is remarkable that, despite widespread interest, there is still a need to concentrate on its etiology and identify methods of assessment and treatment. . Idiopathic club foot is a cosmetic and functional deformity characterized by hypoplasia of the skin, muscles, bones, tendons, ligaments, and neurovascular bundle on the medial side, as well as a smaller-than-average foot .(4)

Initial trials of manipulation and serial casting are currently used to treat clubfoot deformity. 30-50 percent of feet handled in this manner need surgical correction at some point .(5)

It is well understood that clubfeet are vulnerable to rapid deterioration after birth. According to research, this is due to the rapid exchange and production of collagen in tendons and ligaments throughout the first few weeks of existence. This effect becomes substantially less pronounced after the age of 5, indicating that collagen accretion in ligaments is already significantly delayed .(6)

Looking for variations in myosin fibers, researchers discovered that defects in their structure are the most common cause of congenital contracture syndromes in general. The authors proposed that congenital clubfoot is an isolated congenital contracture caused by a defect in the embryonic myosin of the foot's flexor muscles, especially the inn. tibialis posterior (7)

Increased fibrous tissue density was found in the muscles, fasciae, ligaments, and tendon sheaths, primarily in the clubfoot's posterior and medial aspects. Their examination of an electronicmicroscopic preparation revealed the presence of three cell types in fascia from the medial side of the curved foot: normal fibroblasts, myofibroblast-like cells, and mast cells. (8)discovered myofibroblast-like cells in the calcaneonavicular ligament of the curved foot and hypothesized that clubfoot is caused by fibromatosis in the medial tarsal ligaments. (8)

Etiology

Club foot may be caused by myelodysplasia, arthrogryposis, or a variety of other congenital defects, but it is most generally an isolated idiopathic birth defect. Several hypotheses have been proposed to clarify its etiology. (9)

Mechanical Factors in Utero

According to Hippocrates' theory, the foot became equinovarus due to uterine compression. Parker and Browne, on the other hand, assumed that oligohydramnios restricted fetal movement (5).

Neuromuscular Defect

Some researchers believe that equinovarus foot is often the product of a neuromuscular defect. On the contrary, some histological studies indicate no anomalies or deformities .(10)

Primary plasma Defect

Irani and Sherman dissected 11 equinovarus feet and 14 healthy feet and found no primary anomalies of the nerves, vessels, tendon, and muscle insertions. The anomalies were always located in the anterior part of the talus. The talus was

undersized and had a medially rotated anterior section. They hypothesized that the deformity was caused by a primary germ plasma defect .(11)

Arrested Fetal Development

- Intrauterine environment: In 1863, Heuter and Von Volkman suggested that fetal development arrest early in embryonic life was a cause of congenital clubfoot.(3)
- Teratogenic agents' adverse effects on fetal environment and development are well demonstrated by the effects of rubella and thalidomide. Many authors conclude that club foot and transient growth retardation are caused by a variety of environmental factors . (12)

Honein et al. indicated that cigarette smoke exposure and family history are associated with the causative factors for club feet, especially during the antenatal period .(4)

Polygenic theory of Hereditary Pattern

In a large number of instances, club foot is hereditary. Wynne Davis advocated the polygenic hypothesis by demonstrating a rapid decrease in clubfoot incidence from first to second to third degree relatives. This deformity affected about 2.9 percent of siblings in the first degree relatives, compared to 1-2 per thousand in the general population, and the chances of being affected in siblings are more than 25 times higher (13)

Insley discovered a connection between clubfoot and a lack of a portion of chromosome eighteen's long arm. (3)

Since the talus anlagen are fully formed at 6 weeks and the tarsal joints are well-developed at 7 weeks, it is difficult to imagine an exogenous trauma at this point that could affect only the anterior part of the talus and, all too often, only one foot.(14)

Environmental factors have been implicated as well. External strain in the uterus (whether due to hydroamnios or oligoamnios), infectious disease during pregnancy and maternal nutrition defects, vitamin deficiency, toxic agents such as azaserine, d-tubocurarine, aminopterin, and others, and maternal metabolic disorders are some of these.(15)

2. Classification of clubfoot

There are many classifications of clubfoot in the literature. Dimeglio defined four types of club feet based on joint motion and ability to correct deformities .(15)

1. Soft foot, also known as postural foot, can be handled with physiotherapy and regular casting.
2. Soft foot > stiff foot occurs in 33% of cases. It is normally a long foot that is more than 50% reducible and can be treated with casting to achieve complete foot correction in 7-8 months; if not, surgery is needed.
3. Stiff > Soft foot is present in 61% of cases. After physiotherapy and casting, it is less than 50% reducible. Surgery may be done after the procedure if particular conditions are met.
4. Stiff foot is isteratologic and difficult to treat. It has extreme bilateral equinus deformity and needs extensive surgical correction.

3. Clinical Features

Initially the assessment was purely subjective and based on the severity of the deformity and flexibility of foot. Mac Even assessed the clubfeet by the degree of dorsiflexion possible, heel varus, forefoot adduction, calf atrophy and graded the result as excellent, good, fair and poor .(16)

Congenital clubfoot is differentiated by structural, postural and secondary type. The postural clubfoot can occur by abnormal position during birth and manipulative control. The patient should be thoroughly examined to assess the features of paralytic clubfoot. Club foot assessment criteria include: ankle motion, bimalleolar angle, triceps surae weight, heel, forefoot, flexor hallucis longus, painful gait, subtalar pain, shoe wear, and sports.(3)

4. Treatment

A normal developing foot turns into a clubfoot during the second trimester of pregnancy. Clubfoot is not an embryonic abnormality. Treating successfully clubfoot and understanding the pathomechanics has always been unsolved point for modern medicine. Clubfoot is usually diagnosed immediately after birth simply by looking at the foot. Most Orthopaedic surgeons have agreed that the initial treatment of a clubfoot should be nonsurgical and start as soon as possible after birth .(17)

Extensive research has been conducted over the last few decades to determine the best cure for clubfoot. Historically, the mainstay treatment choice was surgical correction, specifically a comprehensive postero-medial soft tissue release. This intervention, on the other hand, has been linked to extreme scarring, joint stiffness, muscle fatigue, gait irregularities, and relapses. Complications such as wound infections, skin necrosis, and neurovascular injuries have also been recorded. Furthermore, the deformity may be over- or under-corrected, causing the talus to flatten or even necrosis.(13)

Given the potential for catastrophic complications and discouraging long-term outcomes, care preferences have shifted to a non-surgical approach through the Ponseti procedure. If used correctly, the procedure has become the standard of treatment and fully removes the need for comprehensive operative correction in over 98 percent of patients.(2)

1. Ponseti technique

This technique is based upon Ponseti's experiences with the wide variety of treatments being applied at that time and his observations in the clinic and operating room, as well as his anatomic dissections and analysis by using a movie camera to produce radiographic images. Utilizing these principles and his understanding of clubfoot anatomy, Dr. Ponseti began employing this technique in 1948 at the University of Iowa. Recently, his observations have been confirmed using modern techniques, including Magnetic Resonance Imaging (MRI).(18)

The Ponseti technique has been the most commonly used initial treatment procedure for babies born with clubfeet. It is a simple technique to master, and when used correctly, it produces excellent results. The Ponseti method is a specific method of casting, serial manipulation and surgery of cutting down the achillestendon i.e tenotomy (8)

The Ponseti technique's correction method is split into two phase:

1. The Treatment Phase - During this time, the deformity is absolutely corrected.
2. The Maintenance Phase - During this time, a belt is worn to avoid recurrence.

The Treatment Phase

Treatment should begin as soon as possible, ideally within the first week of life. Gentle bribery and casting are done once a week. Each cast keeps the foot in the correct spot, allowing it to reshape gradually. In most cases, five to six casts are needed to completely correct the foot and ankle alignment. The majority of babies (70 percent or higher) would need a percutaneous surgical operation

(with a minor incision into the skin) to achieve enough duration of their Achilles tendon at the time of the final cast.(19)

The Maintenance Phase

Treatment should start as soon as possible, preferably within the first week of birth. Once a week, gentle bribery and casting are performed. Each cast holds the foot in the correct position, allowing it to eventually reshape. In most cases, five to six casts are needed to correct the foot and ankle alignment fully. The majority of babies (70 percent or higher) will need a percutaneous surgical procedure (with a small incision into the skin) to reach sufficient Achilles tendon length at the time of the final cast.(19)

Manipulation and Casting - Distinct Elements of the Ponseti Method

The Ponseti technique's unusual modulation and casting maneuvers are only two of several features that set it apart from other casting methods. (20)

First Cast:

The orientation of the forefoot (front of the foot) in relation to the heel causes cavus (abnormally elevated arch) of the foot prior to casting. The first cast application corrects the foot deformity by bringing the forefoot into alignment with the hindfoot (back of the foot). As a result, the cavus (Figure 2) is corrected (Figure 3), typically after just one cast. (Figure 4). (20)



Figure 2: Before treatment. The marked curvature of the foot, called a cavus deformity, is characterized by a visible crease in the midsection of the foot. The foot is tilted down due to tightness of the Achilles tendon.



Figure 3: The initial Ponseti cast. Note the positioning of the forefoot to align with the heel, with the outer edge of the foot tilted even farther downward due to Achilles tendon tightness.



Figure 4: After the first cast, the foot is straight and the cavus and crease are no longer evident

It is normally safest to add the cast in two stages: first, a short-leg cast to just below the knee, which is then stretched above the knee up to the groin until the plaster has hardened. This is preferable in older children (over the age of 2 or 3 months), who are tougher and less readily consoled during the casting. Ponseti stresses the role of long-leg casts in maintaining sufficient tendons and ligament stretching.(20)

1. Second Cast:

One week after, the first cast is removed, and the second toe-to-groin plaster cast is added following a brief period of manipulation. (See Figure 4)



Figure 5: The second cast is applied with the outer edge of the foot still tilted downward and the forefoot moved slightly outward.

This stage of the manipulation and casting process focuses on straightening the foot and aligning the forefoot and heel. Care is required to preserve the foot's downward tilt; reversal of this downward tilt - due to ankle tightness - will result in future casts. Before applying the plaster cast, the surgeon manipulates the forefoot using Ponseti's carefully defined procedure to stretch the foot and determine the amount of correction that should be maintained until the plaster cast is applied.(21)

Another important difference between the Ponseti technique and other methods is that the heel is never specifically influenced. The incremental correction of the hindfoot and midfoot causes the heel to automatically step into the proper position.(21)

2. Further Casting:

Manipulation and casting was repeated weekly for the next two to three weeks to eventually straighten the forefoot, causing it to move in line with the heel.(Figure 5)



Figure 6: The third cast. The Achilles tendon is stretched, bringing the outer edge of the foot into a more normal position as the forefoot is turned further outward.

Standard foot location can be found after four to five casts have been added.

a. The Achilles tendon:

The Achilles tendon is a rope that runs behind the foot that causes it to go up and down. This tendon is shortened in children with clubfoot, preventing the ankle from properly bending up. The tendon in the majority of these children must be lengthened in order to allow adequate ankle motion. The Ponseti procedure accomplishes this by performing a percutaneous surgical release of the tendon, allowing the ankle to be aligned at a right angle with the knee. Percutaneous release is a simple, sterile procedure that is usually performed under local anesthesia via a small puncture.(22)

The final cast:

After that, the foot and ankle are casted in their final, corrected position. (Figure 6)



Figure 7: The final cast is applied, and the Achilles tendon is stretched farther with the forefeet pointed upward. This cast is typically applied in 2 stages, with the short leg component extended up to the groin once the lower component has hardened.

Usually, five or six casts are used to correct the foot and ankle. More research is needed in the most serious cases of clubfoot.

Maintenance and Recurrence Prevention

When the final cast is removed, the newborn is put in an orthosis, or harness, which keeps the foot in the right position. After the casting process in the Ponseti technique, the object of this splinting is to keep the foot in the correct position, with the forefeet set apart and pointing upward. This is done with a belt made up of shoes attached to a bar. (Figure 7) (7)



Figure 8: Image of the foot orthotic

The brace is worn 23 hours a day for the first three months after casting and then while sleeping for many years, normally before the child is five years old.

Many tests have shown that if the brace is not worn according to these recommendations, there is a high chance of recurrence. The causes for recurrence of feet that seem to be entirely corrected have not yet been unequivocally confirmed, but recurrence tends to be close to zero when the bracing regimen is followed precisely.(7)

b. Management of Recurrence

The chance of recurrence lasts for several years after the casting is removed. Ponseti registered a recurrence rate of approximately 50% in his early episodes,

but noticed a decline as more focus was put on the use of the foot orthotic. Early recurrences are better handled with a series of long-leg plaster casts applied at two-week intervals. The first cast could be required to correct chronic foot deformity, with subsequent casts to correct ankle tightness. If there is inadequate ankle correction, an Achilles tendon lengthening may be needed, and a tendon transfer (of the tibialis anterior tendon) may be done in older children to help sustain the correction.(21)

Conclusion :

club foot is a cosmetic and functional deformity characterized by hypoplasia of the skin, muscles, bones, tendons, ligaments, and neurovascular bundle on the medial side, as well as a smaller-than-average foot. Increased fibrous tissue density was found in the muscles, fasciae, ligaments, and tendon sheaths, primarily in the clubfoot's posterior and medial aspects. Mechanical Factors in Utero , Neuromuscular Defect , Primary plasma Defect , Arrested Fetal Development all these is etiology of club foot .

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