Assessment of Corrected Idiopathic Club Feet by Ponseti Technique in Children Older Than Two Years.

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Abstract

The Ponseti technique is rapidly becoming the most widely practiced method for treatment of congenital clubfeet in infants. It is an easy technique to learn and, when applied accurately, yields excellent results.

Good results have been demonstrated at multiple centers and long-term results indicate that foot function is comparable with that of normal feet.

Our study included 50 children (80 feet) older than two years of age, the mean age at beginning of casting was 2.3 week with mean number of serial casting 10.8 casts, they were assessed by pirani scoring system and the results were a mean total score of 0.5, MFS 0.1 and HFS 0.4, the radiographic measurement of talo calcaneal angle on dorso planter view and lateral view and tibio calcaneal angles were respectively as an average of 34^{o}, 35^{o} and 91^{o} degree.

The pirani scoring system is a valid method for assessement of correction and relapsing of club foot and there is a correlation between pirani score and talo calcaneal and tibio calcaneal angles

The Ponseti method was applied with good success and excellent results.

Key Words: club foot, ponseti method, pirani score , talo-calcaneal angle on DP view,talo-calcaneal angle on lat view and tibio-calcaneal angle on lat view.

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LIST OF ABREVIATIONS

CTEV	Conginital talipis eqiuno-varus
FDL	Flexor digitorum longus
FHL	Flexor hallucis longus.
DP View	Dorsoplantar view
CT	Computed tomography
MRI	Magnetic resonance imaginge
PC	Posterior Crease of the ankle
EH	Empty Heel sign
RE	Rigid Equinus sign
MC	Medial Crease of the sole of the foot
LHT	Lateral part of the Head of the Talus
CLB	Curvature of the Lateral Border of the foot
TMS	Total mid foot score
THS	Total hind foot score
TS	Total pirani score

INTRODUCTION

Idiopathic clubfoot, one of the most common problems in pediatric orthopaedics, is characterized by a complex three-dimensional deformity of the foot. In the full-blown equinovarus deformity the heel is in equinus, the entire hind foot in varus and the mid and forefoot adducted and supinated. The exact cause is not known, although the resemblance To other disorders suggests several possible mechanisms.[1]

The aim of treatment is to produce and maintain a plantigrade, supple Foot that will function well.[1]

The treatment of clubfoot is controversial and continues to be one of the biggest challenges in pediatric orthopaedics.[1]

This controversy is due in part to the difficulty in measuring and evaluating the effectiveness of different treatment methods. The heart of the debate is a lack of understanding of the functional anatomy of the deformity, the biological response of young connective tissue to injury and repair, and their combined effect on the long-term treatment outcomes.[2]

The treatment of club foot may be achieved by conservative methods or surgical methods.

The surgical methods include: joint release with tendon lengthening, gradual correction by illizarov frame, tendon transfer and bony procedure in the form of osteotomies or arthrodesis.[3]

Some of the drawback of open surgical release include: joint stiffness, deep scarring, which appears to be particularly severe in infants,wound problems, persistent forefoot supination, loss of reduction and recurrence, overcorrection of the hind foot, dorsal subluxation of the navicular, and loss of normal motion of the ankle and subtalar joints.[4]

Conservative Treatment consists of repeated manipulation that can be achieved with the Kite, the Ponseti and the French physiotherapy techniques.

Recently, Ponseti method has gained popularity in treatment of club foot in early presentation.[5]

All the components of the clubfoot deformity have to be corrected simultaneously with the exception of the equinus which should be corrected last.[5]

The cavus is corrected as the foot is abducted by supinating the forefoot and thereby placing it in proper alignment with the midfoot. While the whole foot is held in supination, it can be gently and gradually abducted under the talus, and secured against rotation in the ankle mortise by applying counter-pressure with the thumb against the lateral aspect of the head of the talus.[6]

The heel varus and foot supination will correct when the entire foot is fully abducted in maximum external rotation under the talus..[7]

After the above is accomplished, the equinus can be corrected by subcutaneous lengthening of tendo-Achilles.[8]

A plaster cast high above knee with knee flexed is applied after each weekly session to retain the degree of correction and soften the ligaments.[9]

After removal of the last cast, foot abduction is prescribed to prevent recurrence of the deformity, to favour remodelling of the joints with the bones in proper alignment.[10]

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THE AIM OF WORK

The aim of this work is to assess *corrected Idiopathic club feet by Ponseti Technique in Children older than two year.*

ETIOLOGY AND PATHOGENESIS

Pathogenesis

Frequency:

The incidence of idiopathic clubfoot is approximately 1 case per 1000 live births in the United States. The incidence differs among ethnicities. For example, it is close to 75 cases per 1000 live births in the Polynesians, especially in places like Tonga.[11]

The male-to-female ratio is 2:1. Bilateral involvement is found in 30-50% of cases. There is a 10% chance of a subsequent child being affected if the parents already have a child with a clubfoot.[12]

Etiology:

The true etiology of idiopathic clubfoot is unknown. Most infants who have clubfoot have no identifiable genetic, syndromal, or extrinsic cause.

Extrinsic associations include teratogenic agents (eg, sodium aminopterin), oligohydramnios, and congenital constriction rings. Genetic associations include Mendelian inheritance (eg, diastrophic dwarfism; autosomal recessive pattern of clubfoot inheritance).[13-15]

Cytogenetic abnormalities can be seen in syndromes involving chromosomal deletion. It has been proposed that idiopathic CTEV in otherwise healthy infants is the result of a multifactorial system of inheritance. Evidence for this is as follows:

- Incidence in the general population is 1 per 1000 live births.

- Incidence in first-degree relations is approximately 2%.
- Incidence in second-degree relations is approximately 0.6%.
- If one monozygotic twin has a CTEV, the second twin has only a 32% chance of having a CTEV.[16]

Pathophysiology:

Theories of the pathogenesis of clubfeet are as follows:

- Arrest of fetal development in the fibular stage
- Defective cartilaginous anlage of the talus
- Neurogenic factors: Histochemical abnormalities have been found in posteromedial and peroneal muscle groups of patients with clubfeet. This is postulated to be due to innervation changes in intrauterine life secondary to a neurological event, such as a stroke leading to mild hemiparesis or paraparesis. This is further supported by a 35% incidence of varus and equinovarus deformity in spina bifida.
- Retracting fibrosis (or myofibrosis) secondary to increased fibrous tissue in muscles and ligaments: In fetal and cadaveric studies, Ponseti also found the collagen in all of the ligamentous and tendinous structures (except the Achilles tendon), and it was very loosely crimped and could be stretched. The Achilles tendon, on the other hand, was made up of tightly crimped collagen and was resistant to stretching. Myoblasts were found in medial fascia on electron microscopy and postulated them to cause medial contracture.