Evaluation of Ponseti method as closed management for clubfeet

Essay
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ABSTRACT

Clubfoot is a congenital foot deformity has four components which are cavus, adduction, varus, and equinus. Most of orthopedic surgeons agreed that the initial treatment of clubfoot is non-operative. Ponseti method treat clubfoot by doing multiple casts after special maneuver for correction of the deformities of clubfoot, followed by tendo Achillis subcutaneous tenotomy, then putting the foot in Dennis-Browne splint until the child reach 3 to 4 years old. Tibialis anterior tendon transfere may required in cases of dynamic supination or relapses. His method shows an excellent outcome without needing another complementary surgery.

Keyword:
Ponseti method as closed management for clubfoot comparing to other conservative methods.

INDEX

FIGURES

INTRODUCTION

Club foot is a congenital foot deformity where the foot points downward with toes turned inward and the foot bottom twisted inward. The bones, joints and muscles of the foot are abnormal. In addition to the foot; the muscles in the lower leg are not as large as usual and will not develop correctly. Finally, the joints in the ankle do not move as much as normal. ***[25]***

When a baby is born with club foot, the bones are misshaped and the foot is stuck in its club foot position. Left untreated, a child will begin to walk on the outer edge of their foot and is likely to develop many related health problems. ***[46]***

The aim of treatment is to produce and maintain a plantigrade, supple foot that will function well. There are several methods of treatment; Ponseti method is one of them.

Ponseti method was developed by Ignacio Ponseti, MD, of the University of Lowa. His method delivers excellent correction of club foot without the associated risks and complications of major foot surgery. Moreover, studies show that patients treated with the Ponseti method enjoy a more flexible foot and ankle than those treated surgically. Long term

studies of the Ponseti method have demonstrated that cast correction of club foot not only helps dramatically during childhood, but also in adulthood. ***[45]***

In this essay, the Ponseti method will be discussed regarding the technique, results and its complications.

Incidence and Etiology of Club Foot

The incidence of congenital clubfoot is approximately 1 in every 1000 live births. Although most cases are sporadic occurrences, families have been reported with clubfoot presenting as an autosomal dominant trait with incomplete penetrance. Bilateral deformities occur in 50% of patients. ***[12]***

In 1929, Böhm ***[8]***, wrote at the beginning of his article on ICTEV: 'I feel there is a deficiency in the field of scientific orthopaedic surgery relative to the question of the origin of deformities and their pathological anatomy'. The causes of idiopathic congenital talipes equinovarus (ICTEV) are little better understood today.

Hippocrates was the first to document a hypothesis to explain the aetiology of congenital talipes equinovarus. Today a number of hypotheses remain, and research findings do not clearly support any particular one. Proposed mechanisms are uterine restriction, abnormalities of joint and/or bone formation, connective tissue, distal limb vasculature, neurological development, muscle migration or an underlying developmental abnormality or developmental arrest. The basis and evidence for each of these theories is given below.

Hoffa (1902) ***[27]***, promoted the widely held hypothesis of uterine restriction, believing that restriction of fetal foot

movement by the uterus caused ICTEV. He suggested that ICTEV arise from oligohydramnios sequence, i.e. believing that reduced amniotic fluid volume is in itself a cause.

This theory may be supported by the general conclusions of the early amniocentesis trial. However, oligohydramnios sequence is generally associated with additional developmental anomalies and may itself have a clear neurological cause. Furthermore, in the early amniocentesis trial, amniotic leakage was only documented in some of the cases. Thus, the mechanism underlying ICTEV after early amniocentesis may have a different aetiology. ***[17]***

The bone/joint hypothesis postulates that positional bony abnormalities underlie the anomaly. Hippocrates wrote: The deformity involves the entire combination of bones which make up the skeleton of the foot.

All the changes seen in the soft part are secondary. Later writers have used the association with other bony abnormalities of the foot. ***[52]***

Endochondral ossification of the foot is disturbed, and its co-ordination with perichondral ossification is also disrupted. ***[19]***

The connective tissue hypothesis is suggested that a primary abnormality of the connective tissue is responsible for

ICTEV. This is supported by the association of ICTEV with joint laxity. ***[52]*** Affected children have marked plantar fibrosis at surgery. Fetal studies give conflicting evidence. ***Atlas et al 1980*** [2], performed a systematic pathological study of 12 fetuses with ICTEV. They concluded, 'muscular, tendinous, fascial & other soft tissue elements are not conspicuously abnormal'.

In contrast, ***Ippolito & Ponseti (1980)*** [30], documented the presence of increased fibrous tissue in muscles, fascia, ligaments and tendon sheaths. From this study of five clubfeet and three normal feet, the authors concluded that a retracting fibrosis might be a primary aetiological factor.

Atlas et al 1980 [2], also studied vasculature in clubfoot. They documented vascular abnormalities in 'all deformed feet of 12 foetuses'. At the level of the sinus tarsi there was blocking of one or more branches of the vascular tree of the foot. This was most conspicuous in the early period of foetal life, and reduced to a simple knot of fatty infiltration and fibrous tissue in older specimens and the stillborn. Individuals with idiopathic congenital talipes equinovarus have muscle wasting of the ipsilateral calf, which may be related to reduced perfusion through the anterior tibial artery in development. It is possible that the association of ICTEV with both early amniocentesis and smoking may be mediated, at least in part, by vascular insufficiency. ***[17, 26]***

During late normal human limb development (9 to 38 weeks), chondrification of the foot is completed, ossification commences, joint cavitation and ligament formation is completed and the distal limb rotates medially. This rotation process allows the sole of the foot to be placed flat on the ground, rather than being orientated with the sole facing inwards, as is seen in feet in the late embryonic period. Pronation continues beyond birth and into postnatal development. ***[3]***

Böhm (1929) [31], investigated the developmental arrest hypothesis of Hueter and von Volkmann in a careful anatomical description of the foot during development. He made wax models of the skeleton of the fetal foot at different gestational ages. His observations led him to conclude that 'a severe club-foot resembles an embryonic foot at the beginning of the second month and the deformity is accompanied by underdevelopment of the bones and muscles'.

These studies support the view that clubfoot may arise due to an arrest of the normal medial rotation of the foot in late foetal development. Indeed, it may be that ICTEV occurs as a result of aberrant genetic control of this rotation process or its disruption.

Genetic and environmental factors are important in the development of ICTEV. There is evidence that development of

bone, joint, connective tissue, innervation, vasculature and muscle may each be implicated in the pathophysiology. Disturbance of the overall process of medial rotation of the fetal foot may be the common pathway linked to all these aspects of maldevelopment. ***[12]***

It is likely there is more than one different cause, and at least in some cases the phenotype may occur as a result of a threshold effect of different factors acting together. The hand is never affected in ICTEV, and thus explanation of its pathology is likely to lead to identification of genes whose effects are exclusive to the foot and lower limb. Advances in genetic mapping techniques, development of mouse models, improved understanding of the control of developmental processes and genetic epidemiology studies are all likely to help to elucidate the causes of idiopathic congenital talipes equinovarus, in the not too distant future. ***[18]***

There is also an increased risk for clubfoot associated with certain neurogenic conditions (spina bifida, cerebral palsy, tethered cord, arthrogryposis), connective tissue disorders (Larsen's syndrome, diastrophic dwarfism), and mechanical conditions (oligohydramnios, congenital constriction bands). The foot deformity seen with the above conditions is often more severe and often requires early surgical correction. ***[35]***

Pathoanatomy of Club Foot

The pathological changes caused by congenital clubfoot must be understood if the anomaly is to be treated effectively. The three basic components of clubfoot are equinus, varus, and adduction deformities.

However, the deformity varies in severity; the entire foot may be in an equinus and varus position with the forefoot adducted and a cavus deformity present or the condition may be much less severe, with the foot being in only a mild equinus and varus position (Fig.1). Clubfoot is accompanied by internal tibial torsion. The ankle, midtarsal, and subtalar joints all are involved in the pathological process. ***[4]***

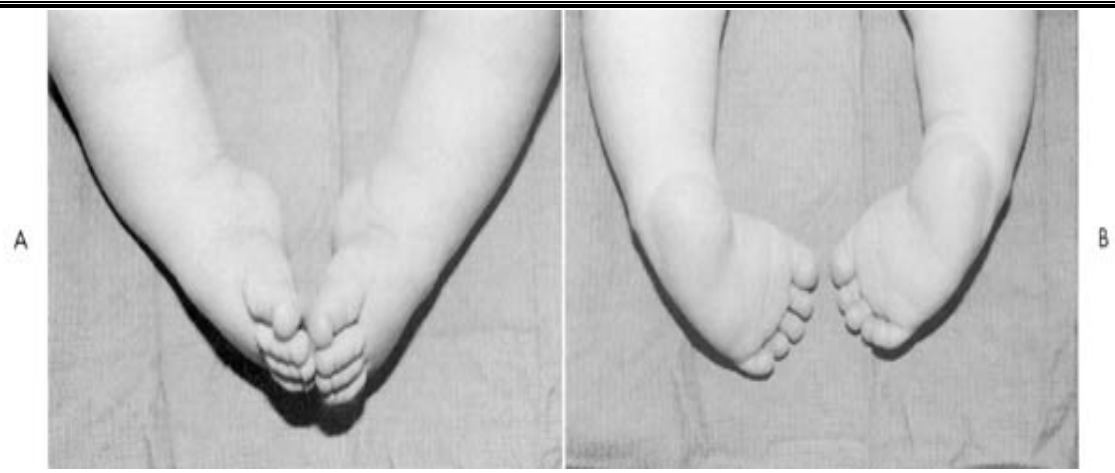

Fig (1): Bilateral congenital clubfoot in newborn. **A**: Anterior view: adduction and supination of forefoot and equinus of hindfoot. **B**: Posterior view: inversion, plantar flexion, and internal rotation of calcaneus, as well as cavus deformity with transverse plantar crease. ***(Barker et al 2001)***