

A CASE REPORT ON CHILD DEFORMITY: CLUBFOOT

Abdul Wahid Ishaque* and Nishant Khatiwada

Acharya Dr. Sarvepalli Radhakrishnan Road, Near Hesaraghatta Main Road, Soldevanahalli, Chikkabanavara Post, Bengaluru, Karnataka 560090.

*Corresponding Author: Abdul Wahid Ishaque

Acharya Dr. Sarvepalli Radhakrishnan Road,, Near Hesaraghatta Main Road, Soldevanahalli, Chikkabanavara Post, Bengaluru, Karnataka 560090.

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ABSTRACT

A clubfoot is a birth defect in which the foot is twisted out of shape or position. Clubfoot is also known as congenital talipes equinovarus. It is the commonest congenital anomaly with an incidence of one to two per 1000 live births. This happens because the tendons and muscles in and around the foot are shorter than they should be. There are no known cause of clubfoot but there are certain known risk factors such as gender (two-third of babies with clubfoot are male), lifestyle choices (smoking whiles pregnant), family history. The main treatment forms of clubfoot are casting and surgery. A 1-year-old male child was brought to the hospital with a chief complaint of deformity of right foot since 1 year. The child was diagnosed based on subjective evidence which is the deformity in the right foot and surgery was performed. Conservative treatment was given with weekly follow up and now the legs of the child look straight with no signs of deformities.

KEYWORDS: Clubfoot, Congenital anomaly, congenital talocalcaneonavicular, hydramnios and primiparous uterus.

INTRODUCTION

A clubfoot is a birth defect in which the foot is twisted out of shape or position. Clubfoot is also known as congenital talipes equinovarus. It is the commonest congenital anomaly with an incidence of one to two per 1000 live births. Approximately 80% of these will be in low- and middle-income countries.^[1] The incidence of clubfoot varies around the world. Without treatment, the clubfoot deformity causes a lifetime of disability as the affected individual experiences pain and difficulty in walking. People with untreated clubfoot find it difficult to access education, employment and experience exclusion from society.^[2] This happens because the tendons and muscles in and around the foot are shorter than they should be. There are no known cause of clubfoot but there are certain known risk factors such as gender (two-third of babies with clubfoot are male), lifestyle choices (smoking whiles pregnant), family history.^[3] The symptoms of clubfoot is very easily to be noticed due to the deformity of the foot in the born child. The initial treatment of a clubfoot should be nonsurgical and should be started as soon as possible after birth. Ponseti method is a specific method of casting, serial manipulation and surgery. The foot's ligament and tendon are stretched and manipulated on the weekly basis followed by implementation of the cast of soft fiber glass that helps to bring the ligament in its original position.^[4]

Objective: the main objective of this case report.

- To education the public about their family history especially women and risk of smoking.
- To encourage good dietary intake during pregnancy and the need of regular checkup.
- To prevent congenital deformities of the baby.

CASE REPORT

A 1-year-old male patient came to the hospital with chief complaints of deformity of the right foot since birth. The parents noticed this deformity of right foot since birth. No history of delayed development or difficult in walking. A detailed history regarding the antenatal checkup, socio economic status, dietary intake and supplemental iron calcium, history of medical illness of the mother was taken. The child is able to walk or run on his own and has history of NICU stay for 42 days. There are similar complaints in their family. The child was immediately posted for surgery the next day. The type of surgery procedure performed was mini external fixator application. The type of application performed was Joshi's External Stabilization System (JESS). After the surgery the patient was monitored at the ward for 2 days then discharged.

DISCUSSION

Clubfoot is mainly idiopathic which indicates that the cause is unknown. However, there have been a lot of Authors who have established theories on the etiology of clubfoot. The anatomy was first highlighted by Scarpa in 1800 and has been subsequently verified by other authors

such as Kite and Turco. Scarpa described clubfoot as congenital talocalcaneonavicular (TCN) joint dislocation, which is the current accepted view.^[5] In contrast, author Goldstein believes that the primary abnormality is outward rotation of the talus in the ankle mortise. Hippocrates postulated a theory which described clubfoot as a result from an elevated intrauterine pressure during pregnancy. This theory was later disputed because of the absence of increased incidence in an overcrowded uterus (twining, large babies, hydramnios and primiparous uterus).^[6] A neuromuscular etiology was proposed on the histochemical analysis of the clubfoot. The authors involved observed an increase in Type 1:11 muscle fiber ratio from 1:2 to 7:1, which suggest a possible neural basis.^[7] Ignacio V. Ponseti supported the "arrest of development" theory. The theory explains the harmful influence of teratogenic agents on fetal environment and development are well established by the effect of rubella and thalidomide. Some authors also believed that temporary growth arrest and clubfoot can happen due to environmental factors.^[8] Palmer supported multifactorial system of inheritance, possible with intrauterine factors having some effects. In case of family history, Wynne Davies postulated the polygenic theory and showed a rapid decrease in incidence of clubfoot from first to third degree relatives. About 2.9% of siblings in the first-degree relatives had this deformity as compared to 1-2/1000 in general population that is 25 times more chances in siblings of an affected child.^[9] Genetic factors and some specific genes changes have been associated with it but these theories on genes are not yet understood.^[10] Research has found a link between the incidence of clubfoot and maternal age as well as whether the mother smokes cigarettes or if she has diabetes. Another link has also been established for a higher chance between a clubfoot and amniocentesis before 13 weeks of gestation during pregnancy.^[11] Infectious disease during pregnancy and maternal nutrition defects, vitamin deficiency, toxic agents like azaserine and maternal metabolic disorders which cause arrested intrauterine fetal development. Vitamin B12 is essential for the functioning of the nervous system. Women with low levels of B12 not only may risk health problems of their own, but also may increase the chance that their children may be born with a serious birth defect." Women of childbearing age, women in early pregnancy, and women who hope to become pregnant should take balanced diet includes foods rich in vitamin B12 or take supplements to reduce their risk of vitamin B12 deficiency and birth defects. Pregnant women should go for regular checkup to ensure that they have the required nutritional balance to prevent the risk of giving birth to defected child. There have been a lot of techniques which has been established for the correction of clubfoot.^[12] Recently, International Clubfoot Study Group, established in 2003, has approved Kite's, Ponseti's and Bensahel's techniques as the standardized conservative regimes for the treatment of clubfoot all over the world.

CONCLUSION

Clubfoot is one of the frequent conditions encountered in clinical practice. It mainly due to dietary deficiency during antenatal period. Surgery has shown higher results in children 1 year and above.

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REFERENCES

1. Khas KS, Pandey PM, Ray AR. Design and development of a device to measure the deformities of clubfoot. *Proc Inst Mech Eng H*, 2015 Mar; 229(3): 194-204.
2. Basit S, Khoshhal KI. Genetics of clubfoot; recent progress and future perspectives. *Eur J Med Genet*, 2018 Feb; 61(2): 107-113.
3. Chen C, Kaushal N, Scher DM, Doyle SM, Blanco JS, Dodwell ER. Clubfoot Etiology: A Meta-Analysis and Systematic Review of Observational and Randomized Trials. *J Pediatr Orthop*, 2018 Sep; 38(8): e462-e469.
4. Radler C, Mindler GT. Kindlicher Klumpfuß: Rezidivbehandlung [Pediatric clubfoot: Treatment of recurrence]. *Orthopade*, 2016 Oct; 45(10): 909-24. German.
5. Besselaar AT, Sakkars RJB, Schuppers HA, Witbreuk MMEH, Zeegers EVC, Visser JD, Boekstijn RA, Margés SD, Van der Steen MCM, Burger KNJ. Guideline on the diagnosis and treatment of primary idiopathic clubfoot. *Acta Orthop*, 2017 Jun; 88(3): 305-309.
6. Herceg MB, Weiner DS, Agamanolis DP, Hawk D. Histologic and histochemical analysis of muscle specimens in idiopathic talipes equinovarus. *J Pediatr Orthop*, 2006 Jan-Feb; 26(1): 91-3.
7. Ezeukwu AO, Maduagwu SM. Physiotherapy management of an infant with Bilateral Congenital Talipes Equino varus. *Afr Health Sci*, 2011 Sep; 11(3): 444-8.
8. Patwardhan S, Shyam A, Sancheti P. Percutaneous Needle Tenotomy for Tendo-achillis Release in Clubfoot - Technical Note. *J Orthop Case Rep*, 2012 Jan-Mar; 2(1): 35-6.
9. Dobbs MB, Gurnett CA. Update on clubfoot: etiology and treatment. *Clin Orthop Relat Res*, 2009 May; 467(5): 1146-53. doi: 10.1007/s11999-009-0734-9. Epub 2009 Feb 18.
10. Ishizuka T, Hung YY, Weintraub MR, Kaiser SP, Williams ML. Ponseti Idiopathic and Nonidiopathic Clubfoot Correction With Secondary Surgeries. *J Foot Ankle Surg*, 2021 Mar 6: S1067-2516(21)00071-5.
11. Shipton MJ, Thachil J. Vitamin B12 deficiency - A 21st century perspective. *Clin Med (Lond)*, 2015 Apr; 15(2): 145-50.

12. Garcia LC, de Jesus LR, Trindade MO, Garcia FC, Pinheiro ML, de Sá RJP. Evaluation of kite and ponseti methods in the treatment of idiopathic congenital clubfoot. *Acta Ortop Bras*, 2018; 26(6): 366-369.