

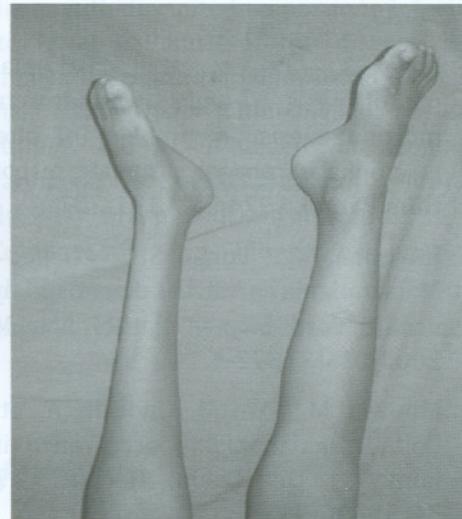
## Resolving Clubfoot Deformity Misleading and Delaying the Diagnosis of Tethered Cord Syndrome

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Neural tube defects comprise of a group of congenital malformations and failed closure at the cranial end is known as anencephaly, is a lethal condition, whereas failed closure at the caudal end usually results in a myelomeningocele.<sup>[1,2]</sup> Tethered cord syndrome (TCS) usually is a stretch induced functional disorder of the spinal cord. The mechanical cause is an inelastic structure anchoring the caudal end of the spinal cord that prevents cephalad movement of the lumbosacral cord.<sup>[3]</sup>

Five years old male child presented with slowly progressive weakness of both lower limbs (left more than right) since birth, pain, paresthesia with shortening of left leg. There was no history of urinary or fecal disturbance. Patient had club foot deformity since birth for that he was evaluated and diagnosed as spastic type of weakness and was on regular physiotherapy at a local hospital. As he grew older, parents noticed shortening of left lower limb. His physical examination showed wasting of muscles in left lower limb in all groups and dissociated sensory loss below L3 with apparent shortening of left lower limb by 2 cm (Fig. 1). Mid thigh circumference of two thighs differed by 5.5 cm. Tone was decreased in left leg with power of 4/5 with absence of ankle and knee reflexes in both legs. On spinal examination, 2 cm defect was palpable over



**Fig 1-** Shortening of left leg and club foot deformity

L3, 4, 5 vertebrae with tuft of hair on the overlying skin. Spinal X-ray showed scoliosis of lumbar spine with convexity to right, transitional vertebrae L5 and S1 (Fig. 2). MRI revealed spina bifida S1 and 2 with evidence of small herniation of dura with herniation of nerve roots with tethered spinal cord at S1 and 2 with small spinal lipoma on right side at S1 level & focally dilated syrinx L1 and L2 level (Fig.3). USG study of abdomen was normal. Patient was subjected to detethering of spinal cord and excision of terminal lipoma. On follow up, patient is doing well with relief in pain and minimal improvement in power. In present case club foot was treated in an isolated manner and other stigmas of tethered cord were not looked for.

Child showed apparent improvement in spastic clubfoot deformity secondary to the development of lower motor neuron type of deficits. It is suggested that tethered cord syndrome should be clinically suspected and investigated and detethering should be done early to avoid the sequelae of the disease as in this case.<sup>[4]</sup> These defects are a complex disorder involving many genetic and environ-

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**Fig 2-** X ray spine shows lumbar scoliosis with convexity to right & transitional vertebrae L5 and S1

mental factors.<sup>[2]</sup> Folate metabolism pathway genes have been examined for association with these congenital defects.<sup>[1,2]</sup>

Animal models demonstrate that periconceptional folate supplementation protects against congenital defects in the face, neural tube, and conotruncal region of the heart. Low folate could directly limit its availability to cells or indirectly disrupt methionine metabolism, thereby increasing homocysteine in the maternal serum.<sup>[5]</sup> Future studies aimed at identifying these risk factors must approach the problem with a wide perspective including several genes and collecting as much environmental data as possible.<sup>[2]</sup> In a study from North-East area of Iran, which has a high prevalence of neural tube defects the role of zinc deficiency has been investigated and it has been shown that there is an association between neural tube defect and low serum zinc level in the newborns.<sup>[1]</sup> This finding focuses attention once again to the importance of nutritional factor in the etiology of neural tube defects. Further studies should be carried out to verify or prove the cause - effect relationship of zinc deficiency with NTD.<sup>[1]</sup> This case illustrates that we should make aware all the medical persons involved in care of these patients and help to investigate all patients with congenital deformities of lower



**Fig 3-** MRI spine shows spinal cord upto S3 level and terminal syrinx (left-T1, right-T2 image)

limbs particularly when it is associated with features of spina bifida occulta and also there is need to take preventive measures to avoid neural tube defects.

## References

1. Golalipour MJ, Mansourian AR, Keshtkar A. Serum zinc levels in newborns with neural tube defects. Indian Pediatr. 2006; 43(9):809-12.
2. Boyles AL, Billups AV, Deak KL, et al. Collaborative Group. Neural tube defects and folate pathway genes: family-based association tests of gene-gene and gene-environment interactions. Environ Health Perspect. 2006 ;114(10):1547-52.
3. Byrne RW, Hayes EA, George TM, McLone DG. Operative resection of 100 spinal lipomas in infants less than 1 year of age. Pediatr Neuros urg. 1995;23:182-6.
4. O'Neill P, Stack JP. Magnetic resonance imaging in the preoperative assessment of closed spinal dysraphism in children. Pediatr Neurosurg. 1990;16:240-6.
5. Rosenquist TH, Finnell RH. Genes, folate and homocysteine in embryonic development. Proc Nutr Soc. 2001 ;60(1):53-61.