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Peroneal Nerve Dysfunction in Patients with Clubfoot Deformity: Evaluation of Clinical Presentation and Treatment

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Background: Complete peroneal nerve dysfunction associated with congenital clubfoot is uncommonly reported. Our retrospective study highlights the recognition of clinical presentation and mid-term outcomes of treatment in these patients.

Methods: Eight out of 658 patients undergoing treatment for clubfoot were identified with unilateral complete peroneal nerve dysfunction associated with congenital clubfoot. Three patients presented primarily to our center; 5 were treated elsewhere initially. All patients were treated with Ponseti casts, Achilles tenotomy, and subsequent foot abduction bracing. Diagnosis of complete peroneal nerve dysfunction was confirmed using nerve conduction velocity studies in all patients. After full-time bracing, an insole polythene molded ankle foot orthosis was given. Three patients underwent tibialis posterior transfer to improve foot dorsiflexor power.

Results: The mean age at presentation was 1.3 years (range, 1 week–5 years). All patients had prominence of lateral 3 metatarsal heads and dimpling of intermetatarsal spaces. At a mean follow-up of 5.1 years, mean shortening of 1.2 cm in tibia (range, 1–2.5 cm) and mean calf wasting of 4.4 cm were observed. There was no relapse of any clubfoot deformity till the final follow-up.

Conclusions: Prominence of lateral metatarsal heads and dimpling of intermetatarsal spaces should raise early suspicion of peroneal nerve dysfunction. Standard Ponseti protocol is useful in treatment of these patients. Tibialis posterior transfer to dorsum partially restores the ankle dorsiflexion.

Keywords: *Nerve, Peroneal, Dysfunction*

Neuromuscular dysfunction in patients with clubfoot is a well-described entity. The reasons cited include anterior horn cell involvement of spinal cord, axonal degeneration, or common peroneal nerve injury at the time of birth.[1] Complete peroneal nerve dysfunction in patients with congenital clubfoot is highly uncommon; there are only isolated case reports in the literature with a short-term outcome.[2-7] Studies are lacking regarding the behavior of these limbs as the child grows older. The purpose of this retrospective study is to describe if these patients can be identified at first clinical presentation and also to describe the early to mid-term follow-up.

METHODS

This study was approved by Institutional Research Committee of Government Medical College and Hospital, Chandigarh (IRB No.17318-24/03780). All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or compa-

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rable ethical standards. All parents of patients consented for this retrospective study and use of their clinical data for purpose of publication.

Records of a total of 658 patients presenting with clubfoot deformity were reviewed retrospectively from July 2014 till July 2019. Out of these, a total of 8 unilateral patients (3 girls and 5 boys presenting without spinal dysraphism, arthrogryposis multiplex congenita, or syndromic associations) were identified to have complete loss of active dorsiflexion. All 8 patients had clubfoot deformity at birth, 3 had left-sided and 5 had right-sided involvement. The mean age at presentation was 1.3 years (range, 1 week–5 years).

The clinical diagnosis of complete absence of dorsiflexion was made at the time of first presentation to us. In older children who were able to follow instructions, this was done by asking the child to actively dorsiflex the toes and in infants, this was done by gently stroking the sole of the foot and the toe dorsiflexor response was observed.[8] Of the 8 patients, 3 patients presented primarily to our center within first month of birth and 5 patients underwent Ponseti casting elsewhere initially, where an average of 9 casts were given (range, 3–20 casts), and were referred to us subsequently. Peroneal nerve dysfunction was suspected in all as there was no syndromic involvement or spinal dysraphism. All the referred patients upon presentation to our center were treated with Ponseti casts for correction of residual deformities with an average of 4 casts (range, 2–7 casts). All patients underwent Achilles tenotomy and 2 patients required a repeat Achilles tenotomy, which was earlier done elsewhere before presenting to us. At the conclusion of casting, passive ankle dorsiflexion was possible in all patients and they were given a foot abduction brace as per the standard Ponseti protocol. These patients were reassessed clinically and were unable to actively dorsiflex the ankle and foot. The diagnosis of complete peroneal nerve dysfunction in clinically suspected patients was confirmed with nerve conduction studies during the period of bracing at the time of follow-up in the outdoor clinic. At 3 months of bracing, all patients were given insole polythene molded ankle foot orthosis for the foot drop gait during awake period.

RESULTS

All 8 patients in our study had no other associated anomaly except 1 who had bilateral inguinal hernia. Nerve conduction studies were done using Schwarzer (Topaz) system for bilateral lower limbs. For sensory evaluation, current stimulation was given at 30–35 milliampere with sensitivity of 20 microvolt and pulse duration of 100 μs. Motor evaluation was done with current stimulation of 35–50 milliamperes with averaged pulse duration of 250 μs and sensitivity of 3,000 μV. Results revealed complete neuropathy of common peroneal nerve of the affected side in all patients.

We observed a limb length discrepancy in 7 patients. Shortening was clinically measured from the anterior superior iliac spine to the medial joint line of the knee for the length of the femur and from the medial joint line of the knee to the tip of the medial malleolus for the length of the tibia, and we found a mean shortening of 1.2 cm in the tibia (range, 1–2.5 cm), whereas no patient had shortening in the femur. All patients had atrophy of the calf muscles of the affected side with a mean atrophy of 4.4 cm (range, of 2–8 cm) as measured at the greatest observed girth of the leg on the affected side and equidistant measurement taken at the contralateral side from the knee joint line (Fig. 1). None of the feet had relapse during bracing period or any residual deformities after treatment at our institute (Table 1).

We found prominence of metatarsal heads of 5th, 4th, and/or 3rd metatarsals on the dorsal aspect in all of our patients. In addition, increased intermetatarsal webbing of lateral 3 or 4 toes without any syndactyly was observed in all the children. These findings were noted at the first presentation itself and prominence increased with growing age (Fig. 2).

The mean duration of follow-up was 5.1 years (range, 1 year 6 months–10 years). The mean age at final

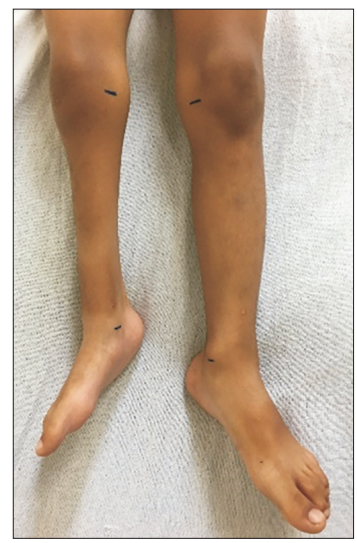

Fig. 1. Photograph showing severe calf atrophy and shortening of the affected side.

Table 1. Summary of Patients

Patient no.	Age (yr) / sex	Side involved	Age at presentation	No. of Ponseti casts received elsewhere	No. of Ponseti casts received at our institution	Any associated disorder	Limb length discrepancy	Calf wasting (cm)	Duration of follow-up (yr)	Passive range of ankle motion at final follow-up	Prominent metatarsal head/ dimpling of web space	Intermetatarsal webbing of lateral toes	Tibialis posterior tendon transfer surgery	Active dorsiflexion
1	9/F	L	1 wk	-	6	No	1 cm shortening in tibia	3	9	5° Dorsiflexion to 20° plantar flexion	5,4,3	Yes	Yes	Till neutral
2	9/F	L	2 yr	5	2	No	No	2.5	7	Neutral dorsiflexion to 10° plantar flexion	5,4,3	Yes	No	Zero
3	3/M	L	1 yr	3	5	No	1 cm shortening in tibia	3	2	Neutral dorsiflexion to 15° plantar flexion	5,4,3	Yes	No	Zero
4	8/M	R	5 yr	20	3	B/L inguinal hernia	1 cm shortening in tibia	5	3	5° Dorsiflexion to 20° plantar flexion	5,4,3	Yes	Yes	Till neutral
5	5/F	R	1 yr	10	5	No	1 cm shortening in tibia	8	4	Neutral dorsiflexion to 10° plantar flexion	5,4,3	Yes	No	Zero
6	10/M	R	1 mo	-	5	No	1 cm shortening in tibia	4	10	Neutral dorsiflexion to 20° plantar flexion	5,4	Yes	Yes	Till neutral
7	3/M	R	1.5 yr	6	2	No	1 cm shortening in tibia	2	1.5	10° Dorsiflexion to 15° plantar flexion	5,4,3	Yes	No	Zero
8	5/M	R	1 mo	-	7	No	2.5 cm shortening in tibia	8	5	Neutral dorsiflexion to 10° plantar flexion	5,4,3	Yes	No	Zero

L: left, R: right, B/L: bilateral.

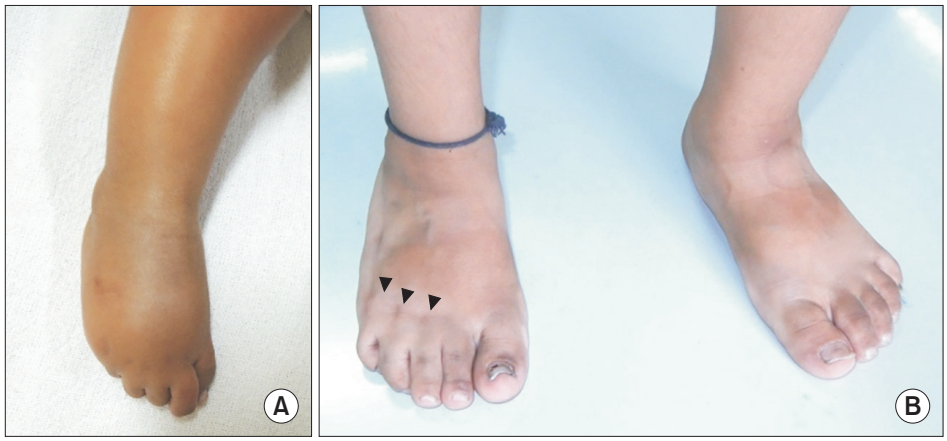

Fig. 2. (A) Photograph showing prominent metatarsal head dimpling in intermetatarsal spaces and increased webbing without syndactyly in a 1-month-old neonate. (B) Photograph showing prominent metatarsal heads (arrowheads), dimpling in intermetatarsal spaces, and increased webbing without syndactyly in a child at follow-up.

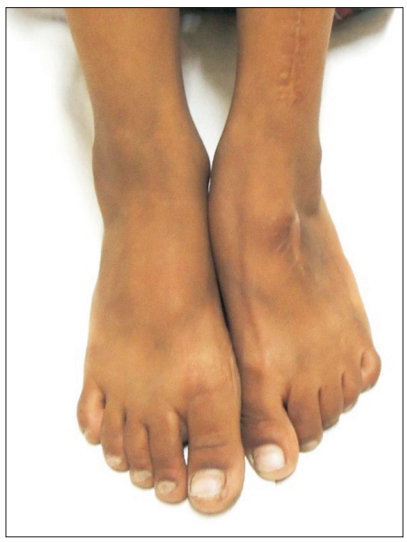

Fig. 3. Photograph of the same patient presented in Fig. 1 after tibialis posterior transfer to dorsum of the foot. Although active dorsiflexion was present, the patient limped as foot drop gait improved only partially.

follow-up was 8 years (range, 3–10 years). At last follow-up, the mean passive ankle range of motion was 5° dorsiflexion (range, neutral–10°) and a mean of 15° plantar flexion (range, 10°–20°). Recovery of dysfunction of the peroneal nerve was not observed in any of our patients till last follow-up. All patients above 5 years of age were offered tibialis posterior transfer to dorsum of the foot to third cuneiform with a pull through suture technique and 3 patients opted for the surgery (Fig. 3). In these 3 patients, active ankle dorsiflexion was possible till neutral (0°) dorsiflexion; however, the foot drop gait pattern was only partly corrected. In these 3 patients, the use of an ankle foot orthosis as well as a foot abduction brace was discontinued. Rest of the patients are being continued on an insole foot drop splint during daytime and a foot abduction brace during the period of sleep. In three patients who are above the age of 5 years, the foot abduction brace has been discontinued but the insole foot drop is continued.

DISCUSSION

Weakness of ankle and foot dorsiflexion in idiopathic congenital club foot can result from a peroneal nerve dysfunction, spinal dysraphism, or anterior horn cell atrophy.[1] Some authors stress on the need for an initial careful, detailed neurologic examination for all clubfoot patients for the drop toe sign; however, this sign seems to be present even in the ones with other forms of neurogenic clubfoot. Recognition of a drop toe sign usually does not alter the management required for correction of the deformity, but does warrant further evaluation.[5,6] The neural involvement may be partial or complete and further management of the patient after correction of deformity depends on that.[1,6]

In a retrospective review of patients with idiopathic clubfeet with relapse following surgical correction, 57% patients were reported to have abnormal electromyography-nerve conduction velocity findings.[9] This indicates that abnormal electrodiagnostic findings may be fairly commonly seen in patients who are diagnosed with idiopathic clubfoot.[9] We confirmed complete peroneal nerve involvement by nerve conduction studies. We feel that nerve conduction studies help to differentiate foot drop resulting from the spinal cord dysfunction from that of peripheral nerve involvement.

The etiology of peroneal nerve dysfunction has been variedly described including peroneal nerve compression at the fibular head to abnormal innervation and dysplastic or absent peroneal nerve branches of sciatic nerve.[10-14] Peroneal nerve palsy in clubfoot as a complication of cast pressure on the peroneal nerve in the area of the fibular

head has been described in one cohort of patients.[10] None of their four cases had normal peroneal nerve function explicitly documented before initiation of cast treatment, so possibly these cases may very well be congenital peroneal nerve dysfunction. Peroneal nerve neuropathy, if due to pressure of cast, is shown to recover fully over a period of time without the need for any specific treatment.[15] None of our patients with peroneal nerve dysfunction recovered, pointing to the fact that the dysfunction was not due to cast pressure.

We observed some findings, which were not previously reported in the literature. These include prominence of 5th, 4th, and/ or 3rd metatarsal heads, which we observed at primary presentation in all of the 8 patients. These findings became more prominent with progression of age. We believe that these could represent morphological changes in the clubfoot associated with peroneal nerve dysfunction. Another observation was increased webbing without any syndactyly of lateral 3–4 toes, which was observed in all of the patients at the initial presentation. Thus, we believe that clinical identifying signs rather than the electrodiagnostic studies as described by us can help to establish the diagnosis of complete peroneal nerve dysfunction at an early stage considering the fact that abnormal electrodiagnostic findings can be present even in a large proportion of idiopathic clubfeet.[9]

The incidence of shortening in unilateral cases of idiopathic clubfoot varies from 1% to 18% and it is hypothesized that in a subset of patients, it could be due to neurological cause and limb dysplasia; however, shortening in the affected extremity in clubfoot patients with concomitant peroneal nerve palsy has not been reported except for an isolated case report.[7,16-18] We observed shortening in 7 out of 8 patients and severe calf atrophy was seen in all of our patients. The importance of this include counselling of the parents regarding tibial shortening and calf atrophy if peroneal nerve dysfunction is suspected. The mean calf atrophy in the reported literature is 1.5 to 3 cm, the mean atrophy in our patients was 4.4 cm; however, we were not able to postulate a possible reason explaining the severe calf atrophy.[19,20]

In patients with clubfoot associated with peroneal nerve dysfunction, correction difficulties have been reported.[4,5,10] Song et al.[4] reported difficulties in correction and recurrence in all 6 patients with concomitant peroneal nerve dysfunction. They reported that 4 of the 6 patients required surgical releases after casting, but eventual results were unsatisfactory. Edmonds et.al reported 9 patients (13 clubfeet) with peroneal nerve dysfunction, and 2 of the 9 patients had complex clubfoot.[5] Four of the nine patients had relapses and required extensive surgical releases after casting and that only one patient had a plantigrade foot position without surgery. There are reports of association of peroneal nerve dysfunction with atypical clubfoot as well.[6] Our findings are contrary to the literature, and none of our patients required soft-tissue corrective surgery. All patients maintained a plantigrade foot. None of our patients developed any atypical foot deformity during casting. We feel that serial manipulation and casting using the standard Ponseti method remains the treatment of choice for these feet with minimal incidence of relapse of the deformity.

Transfer of the tibialis posterior to the dorsum of the foot should be considered in these patients at an appropriate age; however, improvement in foot drop gait may be partial even if the tendon transfer works correctly and the patient is able to actively dorsiflex the ankle after transfer. Reduced ankle range of motion, especially dorsiflexion, and severe atrophy of the calf muscles combined with differences in excursion of tibialis posterior muscle with that of tibialis anterior in these patients may make the transfer less successful.[5] This aspect should be discussed with parents before the procedure.

We conclude that a high index of suspicion of peroneal nerve dysfunction should be kept in patients with unilateral clubfoot presenting with 3 prominent lateral metatarsal heads, intermetatarsal dimpling, and increased webbing of lateral toes. Once discovered, parents need to be forewarned about the possibility of developing shortening of the affected leg along with calf wasting and subsequent gait abnormalities due to foot drop. Ponseti method of management maintains a plantigrade foot without need for open surgical release, and posterior tibial tendon transfer to substitute ankle dorsiflexor power may not give satisfactory improvement in gait.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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