PERONEAL NERVE DYSFUNCTION IN PATIENTS WITH COMPLEX CLUBFEET

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ABSTRACT

Complex clubfeet represent a subset of clubfeet with unique features. Their correction requires a modification of the Ponseti casting technique and good short term results have been reported. However, these clubfeet are very difficult to treat and there is a higher chance for potential complications. We reviewed the database of patients with clubfeet treated from January 2001 to December 2009. There were 837 patients (1376 feet) with 111 (182 feet) (13%) having complex deformity. Of these, 8 patients (10 complex clubfeet) (0.7%) experienced a peroneal nerve dysfunction. Severity of the dysfunction varied from no active dorsiflexion (2 patients) to weakness for active dorsiflexion or foot eversion (6 patients). Deformity correction required an average of 5 casts (range, 1 to 8). Two patients required an Achilles tenotomy and the average ankle dorsiflexion at last follow up was14 degrees (range: 5 to 25). No surgical releases have been required. Two patients required an ankle foot orthosis to improve gait. There were three relapses (37%) that responded to casting and 1 patient required a tibialis anterior tendon transfer. Only 3 feet have recovered the nerve dysfunction. In conclusion, repeated neurological evaluations and very careful cast placement should be performed during the treatment of complex clubfeet. The modified Ponseti technique, if applied properly, is successful in correcting these feet and avoids extensive surgical releases.

INTRODUCTION

Congenital idiopathic clubfoot is a common foot deformity and most orthopaedic surgeons agree that the initial treatment should be non operative. Treatment with the Ponseti method is very effective in the majority of patients. However, some feet do not respond to the standard casting protocol, and they have been called complex clubfeet.[11] Clinically, complex clubfeet' are defined as having rigid equinus, severe plantar flexion of all metatarsals, a transverse crease in the sole of the foot, a deep crease above the heel, and a short and hyperextended first toe. The Achilles' tendon is exceptionally tight and fibrotic up to the middle of the calf.

Correction of complex clubfeet can be achieved by a modified Ponseti casting technique.[13] However, these feet are very difficult to treat and there is a higher chance for potential complications. In many of these cases, the cast slips very often with the resulting abnormal forces on the foot, heel and possibly the leg that lead to the characteristic deformity. In this report we describe 8 patients (10 feet) with complex clubfeet that had a concomitant peroneal nerve dysfunction. The clinical and practical importance of its early detection before and/or during treatment is discussed as well as treatment recommendations.

PATIENTS AND METHODS

After approval by the Institutional Review Board, we reviewed the records database of 837 patients with clubfoot (1376 clubfeet) consecutively treated at our center from January 2001 to December 2009. Of these, 111 patients (189 feet) (13%) were complex clubfeet.

From the medical records, we evaluated the following variables: age of the patient at first visit to our institution, associated pathology, family history of clubfoot, previous treatment and type of treatment before referral, presence or absence of nerve dysfunction at outside institution, severity of the nerve dysfunction, number of casts required for correction at our institution, physical exam including degree of ankle dorsiflexion and calf muscle atrophy, bracing and compliance, relapses, surgical correction, and recovery from nerve dysfunction.

RESULTS

There were 8 patients (10 clubfeet) that had a peroneal nerve dysfunction associated with the complex clubfoot deformity. Five patients had idiopathic clubfeet, two patients had arthrogryposis and one patient had 47 XYY syndrome without spinal or brain abnormalities.

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TABLE 1. Summary of Patients

Case	Gender	Age initial visit (mos).	Side	Associated Pathology	Severity of peripheral nerve palsy	Nerve palsy was found after casting at our hospital	Previous treatment	Treatment of our hospital	# Ponseti casts	Relapses at our hospital	Age at Follow-up	Dorsiflexion Latest FU (degrees)	Recovery from nerve palsy
1	M	2	L	Complex	No active dorsiflexion of toes and ankle	No	Cast correction	Cast correction, day AFO and FAB	1	0	2 y	15	no
2	F	3	B	Complex Arthrogryposis	Bilateral weak toe extensors	No	Cast correction and TAL	Cast correction, FAB, and day AFO	R: 8 L: 8	1	5 y 11 mo	R:10 L:10	Improved
3	M	4	L	Complex	No active dorsiflexion of toes and ankle	No	Cast correction and tenotomy	Cast correction, tenotomy and FAB	7	0	1 y 2 mo	5	No
4	F	5	L	Complex	Weak dorsiflexion of ankle	Yes	Cast correction and tenotomy	Cast correction, tenotomy and FAB	3	1	2 y 7 mo	20	Improved
5	M	13	B	Complex Arthrogryposis	R: weak extensors 4th and 5th toe L: weak toe extensors and peroneal	No	Cast correction, Tenotomy (x2) and AFO	Cast correction and FAB	R: 3 L: 3	0	4 y 3 mo	R: 20 L: 10	No
6	M	26	L	Complex 47XYY syndrome	Weak toe extensors, tibialis anterior and personeal	Yes	Cast correction and tenotomy	Cast correction, TATT, day AFP and FAB	6	1	5 y 6 mo	25	No
7	M	42	L	Complex	Weak peroneal muscles	No	Cast correction	Cast correction and FAB	7	0	4 y	20	No
8	F	47	L	Complex	Weak toe extensors and tibialis anterior	Yes	Cast correction, TAL, PMR, TATT and day AFO	Cast correction and FAB	8	0	5 y 2 mo	10	No

F: female; M: male; B: bilateral; L: left; R: right; FAB: Foot Abduction Brace; AFO: Ankle foot orthosis; TAL: Achilles lengthening; PMR: Postero-medial release; TATT: Tibialis anterior tendon transfer

Five patients were boys and 3 were girls, and 4 had bilateral clubfeet. One patient had family history of clubfoot. Average age at first visit to our center was 17.7 months (range: 2 to 47 months). The average age at last follow up was 3 years 10 months (range, 1 year 2 months to 5 years and 11 months).

Before their initial visit to our center, all patients had plaster-cast treatment (average: 15 casts). Three patients had an Achilles tenotomy and one patient had two times Achilles tenotomy. One patient had an Achilles lengthening. One patient had a postero-medial release, Achilles lengthening and tibialis anterior tendon transfer. Five of the eight patients with initial treatment elsewhere had not been diagnosed with a neurologic deficit by the initial treating physician. One patient has an electromyography (EMG) that showed no abnormality, but the neurologist concluded based on the clinical exam that is was a peroneal nerve dysfunction.

At presentation, the severity of the peripheral nerve dysfunction varied. Two patients had no active dorsiflexion of toes and ankle. The remaining patients had weakness for active dorsiflexion of toes, ankle or foot eversion. Interestingly, there was no record of a hyperextended first toe which was one of the characteristics of the complex clubfoot.

Two patients were referred to the pediatric neurology department and an MRI of the brain and spine demonstrated no significant abnormalities. Therefore, they were diagnosed as peripheral peroneal nerve dysfunction. Interestingly, three patients treated at our center were found to have a peroneal nerve dysfunction after a series of casting, several months after a tenotomy or a tibialis anterior tendon transfer. However, there was no record of casting problems such as swelling, skin lesions or slippage of cast, or complications of surgery.

Average numbers of casts for full correction were 5 (range: 1 to 8). Two patients required an early Achilles tenotomy followed by casting. No infections, profuse bleeding or skin slough were observed after tenotomy. No surgical releases were required.

At the conclusion of casting, six patients were placed initially in a foot abduction brace full time for 3 months. In one patient, the shoes were "plantar-stop/ AFO" type for more stability. One of these patients was then placed in a hinged AFO that was worn in the daytime and the foot abduction brace was worn at night. Two patients were fit with a solid AFO full time initially. One of these patients was then placed in the foot abduction brace after relapse instead of the AFO. Four patients had formal physical therapy once or twice a week and the others had stretching exercise done by the family. Two patients had transcutaneous electrical nerve stimulation.

Three patients had a relapse (37%) that was treated by additional casting. Two of them were using AFO and the other was using a foot abduction brace with appropriate use. One patient required a tibialis anterior tendon transfer. At the last follow up visit, all feet were well corrected with mean ankle dorsiflexion of 14 degree (range: 5 to 25 degree). All patients had considerable atrophy of the calf muscles. Interestingly, two patients (three clubfeet) recovered a little from the peroneal nerve dysfunction, but the others did not. One of these patients was observed having the nerve dysfunction after treatment so it might be from the pressure of the peroneal nerve (Table 1).

DISCUSSION

Peroneal nerve dysfunction is the most frequently encountered mononeuropathy in the lower limbs in adults. Common causes are due to prolonged posture, surgery, weight loss, trauma, bedridden condition, cast positioning and idiopathic.[1] However, peroneal nerve dysfunction is uncommon in childhood.[3,8,13] Most occur at the level of the fibular head, where the common peroneal nerve winds around the fibular neck. This segment of the nerve is covered only by subcutaneous tissue and is directly apposed to bone, therefore, making the nerve susceptible to compression injury, which causes most pediatric peroneal nerve dysfunction. Jones et al. reported 17 children with peroneal nerve dysfunction. Causes included compression in 10 (59%), trauma in 3 (18%), entrapment in 3 (18%) and indeterminate in 1 (5%).[8] But most compressive peroneal nerve dysfunction resolved within several months.[3,8,13]

Several authors have noted that clubfoot with peroneal nerve dysfunction is very difficult to correct.[5,7,12] Gordon et al. reported peroneal nerve dysfunction as a complication of clubfoot treatment in four cases which were resistant and recurrent.[7] The palsies were not observed until late in the course of treatment so the authors concluded that repeated cast pressure at the fibular head might have been the cause of the dysfunction. Because the result of the peroneal nerve dysfunction (drop foot) resembles that of clubfoot, the authors suggested that it would be easy to miss.

Song et al. reported six patients with congenital clubfoot and concomitant peroneal nerve dysfunction.[12] All of the cases were resistant and recurrent. Four of the six patients underwent corrective surgical releases after casting, but with unsatisfactory results. None of the six patients recovered from the nerve dysfunction, therefore the authors suggested that these peroneal nerve palsies should be differentiated from peroneal nerve palsies which are caused by pressure in the cast that will resolve with time.

Edmonds et al. reported nine patients (13 clubfeet) that had no active dorsiflexion of the toes or ankles and no active eversion on the affected sides.[5] The authors used the drop-toe sign, which is done by resting the toes in plantarflexion and observing for active dorsiflexion by plantar stimulation. All of the patients had a drop toe sign before treatment. Two of the nine patients had complex clubfoot. Four of the nine patients had relapses and underwent extensive surgical releases after casting. One of these patients had exploration of the peroneal nerve bilaterally. The findings were symmetric, revealing a normal-appearing full-diameter peroneal nerve proximal to the proximal leg fascia with only a thin atrophic nerve distally. Only one patient had maintained a plantigrade foot position without intraarticular surgery. The authors suggested that these cases may need the modified Ponseti method, including earlier-than-usual Achilles tendon lengthening.

Because five of our patients were treated elsewhere with casting or corrective surgery before initial visit, it was not clear whether the peroneal nerve palsies were a complication of prolonged casting or whether the palsies were present before as part of the deformity. Three patients were found as having peripheral nerve dysfunction after series of casting or surgery at our institution. Therefore, these patients might have suffered peripheral nerve dysfunction from complication of prolonged treatment. However, only one of these 3 clubfeet made some recovery from the nerve dysfunction. This may indicate the nerve palsies were not from simple pressure by casting because infant peroneal palsies, which caused mainly by pressure on the fibular head, are reported to have good prognosis.[3,8,13]

Feldbrin et al. reported that isolated peroneal nerve damage was seen in 27% of their patients with congenital clubfoot, with 10% having an abnormality of both the peroneal and posterior tibial nerve.[6] Macnicol and Nadeem reported that 9.5 % of their clubfoot patients with previous corrective surgery had abnormal responses to motor electrophysiological tests of the peroneal nerve.[9] On the other hand, Bill and Versfeld failed to find abnormal responses of the peroneal nerve in an electromyographic study of patients with idiopathic clubfoot.[2] Electrodiagnostic studies are helpful to determine diagnosis of nerve palsy.[2,3,6,8-10,12] However, those studies do not change treatment and prognosis. Repeated neurological evaluations are more important.

Regardless of the underlying cause, it is obvious that these cases are very difficult to treat. Most authors have reported failures of treatment for patients with peroneal nerve dysfunction and the requirement for subsequent corrective surgery. However, corrective surgery frequently brings severe scarring leading to joint stiffness and muscle weakness. The long-term result after corrective surgery was very poor.[4] Although three patients had relapses and most patients needed prolonged casting, all of our patients had excellent deformity correction without surgical releases. However, a longer follow up of the patients is needed since late relapses due to the nerve dysfunction may be possible.

In summary, repeated neurological evaluations and very careful cast placement should be performed during the treatment of complex clubfeet. The modified Ponseti technique, if applied properly, is successful and avoids extensive surgical releases.

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