Electromyography Nerve Conduction Velocity Evaluation of Children With Clubfeet

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Abstract

Neurologic deficit has been implicated as a possible etiology for clubfoot and a cause for recurrent deformity in patients who have undergone clubfoot surgery.

In the study reported here, we wanted to determine if clubfoot patients with peroneal weakness had any neurologic deficits on electromyography nerve conduction velocity (EMG-NCV) studies before surgery and if there was any association between neurologic deficit and clubfoot recurrence. We reviewed the EMG-NCV studies of 36 patients involving 57 cases of idiopathic clubfoot and recurrence of the deformity or muscle weakness.

In the clubfoot patients with weak peroneal muscle and no prior surgical history, 45% of the studies were interpreted as normal, 20% as neuropathic, 15% as mixed myopathic and neuropathic, 10% as radicular, and 10% as myopathic. In the clubfoot patients with recurrence after clubfoot repair surgeries, 57% had abnormal EMG-NCV studies. Specifically, peroneal mononeuropathy was the most common disorder (41% of clubfoot patients treated surgically).

Awareness of a significant incidence of neurologic deficit may help in preoperative planning by indicating that ultimately a tendon transfer may be necessary to obtain a plantigrade foot.

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Congenital talipes equinovarus is a common birth deformity, the etiology of which has not been identified conclusively. The various proposed theories have been grouped into 4 primary categories.[1-6] Hippocrates proposed the mechanical theory, which postulated that external mechanical forces force the fetal foot into an equinovarus position. Besides the intrauterine mechanical etiology, there are also theories grouped under the classifications of arrest of fetal development, primary germ plasm defect, and neuromuscular defect.[1,2,6,7]

Clubfoot also has been theorized as having a neuromuscular etiology. According to Tachdjian,[8] White, in 1929, thought a peroneal lesion caused by pressure in the intrauterine stage was the predominant factor; Middleton, in 1934, believed a prenatal lesion of the striated muscle was the cause of the congenital deformity; Flinchum, in 1953, also believed the peroneal muscle was involved, citing a muscle imbalance secondary to dysplasia of the peroneal muscle as the cause; and Isaacs and colleagues, in 1977, found electron-microscopic muscle changes that led them to theorize abnormal muscle innervation and subsequent muscle imbalance were the etiology.

Whatever the cause, the treatment regimen for idiopathic clubfoot includes closed manipulation with casting followed by surgical intervention for recalcitrant cases. Multiple surgical techniques, including those of Turco, Carroll, and McKay,[8,9] have been used to treat talipes equinovarus. At our institution, in the past, we commonly performed the complete subtalar release, as described by Simons.[10,11] Currently, we utilize the Ponseti technique.[12] Each technique has been plagued with recurrences that arise for a variety of reasons. Theories for recurrence include loss of initial reduction secondary to inadequate casting and treatment, persistence of primary deformity, scar tissue formation, dynamic muscle imbalance, and failure to achieve a concentric reduction.

As electromyography is considered an appropriate diagnostic tool in the evaluation of the severity of

the peripheral nerve injury,[13,14] in the present study we wanted to (1) examine clubfoot patients with peroneal weakness before any surgical intervention and determine if there are any neurologic deficits on electromyography nerve conduction velocity (EMG-NCV) studies and (2) determine if there is any association between neurologic deficit and postsurgical clubfoot recurrence.

Methods

Thirty-six patients (mean age, 2 years, 7 months) who were being followed up for apparent idiopathic clubfoot and who demonstrated recurrence of deformity after casting or surgical intervention were evaluated with electromyography studies. Patients were also evaluated with EMG-NCV studies of the peroneal nerve and tibia nerve when they demonstrated muscular weakness of the peroneals on physical examination. The conduction velocity (m/s) for the peroneal and tibial nerves in the normal child is more than 39 m/s.[15] EMG-NCV testing with less than 39 m/s was considered abnormal. From the EMG-NCV, 4 types of abnormalities are classified: neuropathic disorder (mononeuropathic, polyneuropathic), myopathic disorder, radicular disorder, and mixed disorder (neuropathic, myopathic).[16]

Initially, clinic notes were reviewed to determine the treatment that each of the 52 clubfeet had received. Before EMG-NCV evaluation, various procedures were performed, including 20 castings, 24 complete subtalar releases, 5 complete subtalar releases with calcaneocuboid releases, 2 tendo-Achilles lengthening, and 3 posteromedial and posterolateral releases.

Eighteen girls and 18 boys were included in the study. The right foot was affected in 12 patients, the left in 8 patients, and both in 16 patients, and 5 patients (unilateral foot deformity) underwent 2 EMG-NCV studies during their course of treatment, so 57 studies total were included in the study. EMG-NCV studies on 20 feet with peroneal weakness were obtained before any surgical intervention. EMG-NCV studies on 37 clubfeet with recurrence after surgical intervention were performed, with some of these feet undergoing additional operations after electromyographic examination.

The statistical analysis included a descriptive analysis of different types of neuropathy for patients with clubfoot and the odds ratio for the measurement of the association between clubfoot recurrence and deficit (Fisher exact test). $P<.05$ was considered significant.

Results

The subset of clubfoot patients with peroneal weakness that had not been surgically corrected before electromyographic evaluation was studied first. Their 20 clubfeet, however, had been treated with serial casting before electromyographic evaluation. The 20 EMG-NCV interpretations were grouped into several categories: normal (9 feet, 45%), mixed disorder (3 feet, 15%), mononeuropathy (2 feet, 10%), polyneuropathy (2 feet, 10%), primarily myopathic disorder (2 feet, 10%), and primarily radicular disorder (2 feet, 10%).

We reviewed EMG-NCV studies on 37 clubfeet that had undergone some type of operative intervention but experienced recurrence. Of these 37 feet, 16 (43%) were interpreted as normal, 16 (43%) as primarily neuropathic disorder, 3 (8%) as mixed disorder (neuropathic, myopathic), and 2 (6%) primarily radiculopathy. The 16 neuropathic disorders included 15 mononeuropathies involving the peroneal nerve and 1 neuropathy involving the peroneal and tibial nerves.

"Our analysis of the neuropathic disorders defined by EMG-NCV showed that peroneal mononeuropathy was the most common disorder."

The odds ratio for the association between presence of neurologic disorder and clubfoot recurrence was 1.18 (95% confidence interval, 0.37-3.85). The Fisher exact test for association resulted in $P = .79$.

Discussion

Although congenital talipes equinovarus is one of the most common birth deformities, and many theories indicate an etiology of neuromuscular abnormality, relatively few studies have used EMG-NCV to evaluate clubfoot patients, and even fewer have been reported in the English literature. The recurrence rate after operative intervention varies, with Pecak and colleagues[17] reporting 31% recurrence after Turco's operation (requiring reoperation) and 37% recurrence after Achilles tendon lengthening and dorsal capsulotomy. Laaveg and colleagues reported 47% recurrence requiring reoperation and 50% of these patients requiring at least a second reversion.[18] In addition, many such cases are thought to be secondary to a muscle imbalance that could be diagnosed by electromyography.[8] Fifty-five percent of our idiopathic clubfoot patients with weak peroneals were interpreted by electromyography as having an abnormality before any surgical intervention. In comparison, 57% of apparent idiopathic clubfoot patients with surgery and recurrence were determined to have an abnormality.

Our analysis of the neuropathic disorders defined by EMG-NCV showed that peroneal mononeuropathy was the most common disorder: 41% of clubfoot patients who

underwent surgeries. However, no patients were identified in the population with weak peroneals and no prior surgery. Feldbrin and colleagues[2] evaluated 52 children with idiopathic clubfoot and found only 17% completely normal, compared with our 38%. They similarly had pathologic electrophysiologic findings in 66% of their patients treated conservatively, a predominance of neuropathic abnormalities, and a large percentage of isolated peroneal mononeuropathy. In addition, we found by electromyography that 4% of our patients had some form of myopathic deficit, whereas Feldbrin and colleagues reported none.

Investigators have documented myopathic and mixed disorders in their findings. Koczocik-Przedpelska and Marciniak[19] evaluated 47 patients with congenital clubfoot and found myopathic and neurogenic changes in 40% of them. On electromyographic examination, Abbruzzese and colleagues[7] also found a variety of abnormalities, including spontaneous denervation activity in 1 patient. Motor-unit potential duration was increased in 7 patients, decreased in 6, and normal in 8.

Some studies have not revealed a neurogenic or myopathic disorder in patients with clubfeet. Bill and Versfeld[1] evaluated 25 patients with congenital idiopathic clubfoot that had not undergone any casting or operative intervention. They were unable to demonstrate any abnormalities by electromyography, which suggests a neuropathic or myopathic disorder. Tönnis[20] reported no alterations suggesting neural damage or a primary muscular disease in their clubfoot population.

In a recent animal study, however, magnetic resonance imaging (MRI) nerve signal changes occurred as early denervation or reinnervation of EMG-NCV sign changes, indicating the immediate loss of the voluntary activities and compound muscle action potential.[21] These EMG and MRI signal changes corresponded histologically to massive axonal degeneration with a breakdown of cellular and axonal structure.[21]

In summary, patients with recurrent clubfoot deformity in this series had a higher incidence of neurologic deficit as diagnosed by EMG, with a majority of the deficits being a mononeuropathy involving the peroneal nerve. In nonoperative clubfoot patients with clinically apparent peroneal weakness, there is also a high incidence of neurologic deficit as diagnosed by EMG-NCV. The incidence of neurologic deficit in our study was similar in the operated and nonoperated groups. In conclusion, we believe that awareness of these neurologic deficits may help surgeons in discussions regarding treatment and prognosis in this patient population.

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