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Peroneal nerve palsy secondary to prolonged squatting in seasonal farmworkers

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Objective: The aim of this study was to evaluate the follow-up and treatment results of peroneal nerve palsy secondary to prolonged squatting for working and to determine an approach for its treatment and prevention.

Methods: The study retrospectively evaluated 16 patients (7 males, 9 females; mean age: 23.6 years) diagnosed with peroneal nerve palsy due to squatting for work. Clinical and neurological evaluations were performed and weight and height were measured. Lesion site was determined using electrophysiological testing. After diagnosis, medical and orthotic treatment was initiated and rest was advised. Patients were followed until motor symptoms were resolved.

Results: Clinical and neurophysiological evaluations were consistent with isolated peroneal nerve palsy. The left side was affected in seven patients, the right side in seven and both sides in two. Average onset of the symptoms was 3.3 (range: 1 to 6) weeks and average daily squatting period was 6.8 (range: 6 to 8) hours. Average healing time was 7.4 (range: 3 to 16) weeks. None of the patients was obese or overweight. All patients healed with conservative treatment and no surgical treatment was necessary.

Conclusion: Working conditions and duties should be considered in the evaluation of peroneal nerve palsy. In peroneal nerve palsy secondary to squatting, healing should be expected with conservative treatment, resting and close follow-up.

Key words: Compression neuropathy; entrapment neuropathy; nerve compression syndrome; peroneal nerve palsy.

The peroneal nerve is susceptible to trauma due to its anatomical path. Extending superficially around the neck of the fibula, the nerve is covered only by skin and a thin subcutaneous fat layer, and may be compressed and damaged between the bones in this region due to external forces. The nerve enters the leg in a fibrous arch surrounded by the peroneus longus muscle and intermuscular septum and nerve compression can occur when this arch gets thicker.[1] This fibrous arch has been reported to cause dynamic compression during running and postural compression in movements that require knee flexion such as squatting or leg crossing.[2-5] Nerve palsy is thought to develop due to compression and tension of the peroneal nerve between the biceps and gastrocnemius lateral head

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at the top and between the fibrous arch and the head of the fibula at the bottom in a squatting position.[6,7]

Peroneal neuropathy is the most common neuropathy in the lower extremities and the third most common neuropathy following median and ulnar neuropathies in the entire body.[8] External factors, such as trauma, fractures, sprained ankle, surgical or non-surgical injuries are the most common causes of acute nerve palsy.[1,9] Growing masses, such as tumors, intraneural ganglions, hematoma or cysts can cause compression due to mass effect and lead to a more slowly progressing table.[10,11] Bilateral lesions are quite rare and are reported to constitute approximately 10% of all lesions.[12]

Peroneal neuropathy has not been previously defined in seasonal farmworkers in Turkey, despite being a common entity, especially during harvesting time in regions intensively inhabited by farmworkers.

Therefore, the aim of this study was to evaluate peroneal nerve palsy due to prolonged squatting in this understudied population using clinical manifestations and electrophysiological studies. In addition, we presented follow-up and treatment results and attempted to determine an approach for its treatment and prevention.

Patients and Methods

The study retrospectively evaluated 18 extremities of 16 seasonal farmworkers (7 males, 9 females; mean age: 23.6±5.3 years, range: 18 to 37 years) who were admitted to our polyclinic due to drop foot and sensory symptoms in the peroneal nerve region and who had a history of working in a squatting position between the years 2007 and 2011. Palsies developing due to lumbar discopathy, trauma, fracture, surgery or other reasons such as peripheral neuropathy, motor neuron disease, polyneuropathy, diabetic neuropathy and toxic neuropathy were excluded from the study. All patients had a history of working in a prolonged squatting position as seasonal farmworkers. Informed consent forms were received from all patients.

Symptoms and time of onset, mode and duration of work, weight loss, drug use and metabolic and toxic diseases were questioned in the clinical examination. Tibiofibular muscles atrophy evaluation and muscle strength grading was recorded in the clinical and neurologic examination. Foot eversion (peroneal muscles), foot dorsiflexion (tibialis anterior muscle) and thumb dorsiflexion (extensor hallucis longus muscle) evaluations were performed in the muscle strength examination. The Medical Research Council (MRC) scale graded between 0 and 5 (5: full contraction; 0: no contraction) was used in the evaluation.[13] All sensory areas were checked with palpation or using a needle to rule out polyneuropathy. The Tinel's sign test was performed at the level of the fibular neck. Weight loss and height were measured to evaluate the relationship between peroneal nerve palsy and weakness and patients' weight loss history was questioned. Body mass index values were calculated. All patients underwent electrophysiological analysis following clinical evaluation and the location of the lesion was determined.

Peripheral nerve conduction velocities were measured using standard methods. The tibialis anterior, extensor digitorum brevis, peroneus longus and gastrocnemius muscles were evaluated using electromyography (EMG). Nerve conduction study was made between the ankle, fibular head and popliteal fossa and transmission speeds were determined. Criteria defined by Fabre et al.[1] were used to diagnose sensory and motor deficits. Deceleration in nerve conductive velocity was considered to be the minimum criteria for motor deficit for the determination of sensory deficit. Other electrophysiological findings related with motor neuron such as reduced compound muscle action potential (CMAP), presence of fibrillation and positive sharp wave (PSW) were recorded.

All patients were prescribed vitamin B following diagnosis and the use of an ankle foot orthosis (AFO) device was recommended. Patients were advised to cease working in a squatting position and to rest. Follow-up was performed weekly during the first three weeks and then once every three weeks until all motor complaints were eliminated. EMG was performed again at the 12th week in patients that showed no increase in motor grading according to the MRC scale or improvement in sensory examination. Ankle dorsiflexion exercises were encouraged during the follow-up period. AFO use was discontinued in patients whose complaints improved.

Results

Clinical and neurophysiologic evaluation results of all patients were consistent with isolated peroneal nerve palsy at the level of the fibular head. None of the patients had a pathology or polyneuropathy suggesting diagnosis such as lumbosacral radiculopathy, plexopathy or sciatic neuropathy related to other peripheral nerves apart from peroneal nerve.

Demographic and clinical data is provided in Table 1. Peroneal nerve palsy developed in the left extremity of seven patients, in the right extremity of seven and in both extremities in two. All patients had motor symptoms and only two patients did not have sensory symptoms. The Tinel's sign test was positive in patients with sen-

Table 1. Demographic and clinical data of the patients.

No	Age/Sex	Side	Time from onset of symptoms (weeks)	Muscle strength (MRC)			BMI healing (weeks)	Time to symptoms	Sensory	EMG/NCS
				FE	FD	TD				
1	18/F	R	3	3	4	3	22.6	3	+	□CMAP
2	23/M	R	2	2	2	2	20.2	10	+	Fibrillation, PSW
3	29/F	L	5	2	3	4	21.3	6	+	□NCV
4	30/F	R	4	1	0	0	23.2	16	+	Fibrillation, PSW
		L		1	2	3		8		CMAP
5	19/M	L	3	2	3	3	23.2	6	+	□CMAP
6	18/M	R	4	3	3	2	25.3	5	+	□CMAP
7	25/F	L	1	4	3	4	23.3	7	+	□NCV
8	24/M	L	3	2	4	2	21.4	5	+	Fibrillation, PSW
9	29/F	R	2	2	2	2	22.8	6	+	Fibrillation
		L		2	3	4		6		Fibrillation, PSW
10	22/F	R	3	4	4	4	22.9	4	-	□NCV
11	23/M	R	3	2	2	3	24.3	9	+	□CMAP
12	18/F	L	2	2	1	2	24.0	14	+	Fibrillation, PSW
13	19/M	R	3	2	1	1	23.5	10	+	□CMAP
14	22/F	L	4	3	3	4	24.8	8	+	□CMAP
15	37/M	L	5	4	3	3	20.7	6	-	□NCV
16	23/F	R	6	2	2	2	22.6	4	+	□CMAP

BMI: Body mass index; CMAP: Compound muscle action potential; EMG: Electromyography; FD: Foot dorsiflexion; FE: Foot eversion; MRC: Medical Research Council; NCS: Nerve conduction study; NCV: Nerve conduction velocity; PSW: Positive sharp wave; TD: Thumb dorsiflexion.

sory symptoms. Complaint onset time varied between 1 and 6 (mean: 3.3) weeks and mean daily working time in a squatting position was 6.8 (range: 6 to 8) hours. Patients were observed to work as seasonal farmworkers at different times, picking cotton, lentils, strawberries and tomatoes. All complaints began in the period between May and October, which is working and harvesting period and were therefore evaluated as seasonal and referred to as seasonal farmworker palsy. Mean recovery period was 7.4±3.4 (range: 3 to 16) weeks. Mean body mass index was 22.9±1.4. None of the patients were overweight or obese according to World Health Organization classification.

Electrophysiological studies showed a decrease in peroneal CMAP in eight patients. Evaluation was made according to the presence of an increase of more than 20% according to the measurement performed on the other side. Only proximal CMAP was reduced and distal CMAP was normal in these patients and was evaluated as conduction block due to severe demyelization at the level of the fibular head. These eight patients showed full recovery in follow-up.

Nerve conduction velocity (NCV) decelerated only in four patients. A deceleration of more than 10 m/sec when compared to the distal segment was taken as the deceleration criteria. This result was interpreted as the presence of mild local demyelization at the level of the fibular head and the four patients showed full recovery during follow-up.

Five patients had fibrillation and PSW findings in addition to peroneal CMAP decrease in all (proximal and distal) stimulation areas. These findings were evaluated as axonal damage. Polyclinic follow-up of two patients who were believed to have axonal damage showed no improvement. Surgical treatment was made as nerve conduction studies requested at the 12th week showed no decrease in fibrillation potential and no motor unit potentials to suggest regeneration. The nerve was fully loosened at the level of the fibular head in the surgical treatment. In the postoperative first year follow-up, both patients were observed to improve clinically. Clinical follow-up of the other four patients with axonal damage showed improvement and patients were observed to fully recover by the 10th week follow-up.

Discussion

The peroneal nerve can be acutely damaged from the femur to the fibular head region due to various traumas

such as direct laceration, femur fracture, firearm injury or a high force directly on the nerve. Nerve palsy can develop in a slower fashion, termed entrapment or compression neuropathy, due to prolonged exposure of the nerve to external or internal compression. Nerve compression can occur due to external factors such as plaster compression, compartment syndrome, prolonged sitting in a cross-legged position and bed compression, as well as internal factors such as bone spurs, fracture callus, synovia, tumors, ganglia and compression between muscle plans.[10,11,14,15] Peroneal paralyses are mostly unilateral.[12]

Nerve injury is known to occur mostly around the fibular head.[3] The nerve is quite superficial at this level and is only covered by the skin and subcutaneous tissue.[16] Mobility of the fibular head in this region is also believed to cause nerve irritation.[3] We found that the nerve was compressed in the fibular head region in all patients.

Møller and Kadin reported that nerve compression around the fibular head frequently occurs due to the contact of the nerve area with a hard surface, such as the edge of a bed as a result of being bedridden or prolonged immobility under anesthesia.[3] Postural peroneal nerve palsy can develop due to prolonged squatting, crossing one's leg and sitting cross-legged.[1,4,5,14,17] Squatting is thought to paralyze the nerve by compressing it between the biceps and gastrocnemius lateral head at the top and between the fibrous arch formed by the peroneus longus and fibular head at the bottom.[6,7] It has been reported that the peroneal nerve can be compressed during long-term running or exercise that requires frequent knee flexion and this compression has been termed dynamic compression.[2,18] Peroneal nerve palsy due to prolonged squatting was defined in a case study in the literature.[4,14] Toğrol reported bilateral nerve palsy in three patients following more than three hours of squatting on a daily basis.[14] Peroneal nerve palsy was reported to develop due to natural childbearing by squatting, which is still common in underdeveloped countries.[7,17] In a case of bilateral peroneal nerve palsy, Sahai-Srivastava and Amezcua recommended showing more attention to patients under epidural anesthesia versus peroneal nerve palsy due to childbearing in squatting position.[6] Peroneal nerve palsy was presented in workers employed in duties requiring work in a squatting position. Peroneal nerve palsy was defined in two workers who worked in strawberry picking and turnip harvesting following a minimum of six hours of working a day, and recovered after the harvesting period.[19,20] Apart from these patients, Sangwan et al.[21] defined peroneal nerve palsy due to prolonged squatting in 30 seasonal farmworkers and reported that all patients improved between three and nine weeks. Therefore, Sangwan et al. recommended conservative treatment for these patients. In the present study, 14 out of 16 farmworkers, two of whom had bilateral nerve palsy, were observed to heal by conservative treatment following an average of 7.4 weeks.

Increased risk of peroneal nerve palsy has been attributed to low body weight and excessive weight loss.[22] It has been reported that the nerve becomes more sensitive to minor traumas, mechanical irritations and direct compression due to malnutrition, metabolic factors or decreases in the protective subcutaneous tissue that encircle the nerve.[14,21,23] In addition to postural tension of the nerve due to squatting, placing the knee on the floor during working can also contribute to nerve palsy.[21] Watemberg et al. reported the effect of weight loss on peroneal nerve palsy due to prolonged surfing in the squatting position.[16] In our study, none of the patients were classified as overweight or obese according to body mass index values. The patients reported that they occasionally placed their knees on the ground during working. Therefore, being overweight can be considered as a protective factor for this patient group. Although peroneal nerve palsy was reported to be more common in males in some series, another group of researchers claim that this condition is not correlated with gender.[12,21] In the present study, we found no tendency in distribution of our patients according to gender. Sangwan et al. observed that his patients, consisting of Indian farmers, mostly worked by placing the left knee on the ground and therefore reported that paralysis was more common on the left side.[21] However, in our patients, involvement of the right and left side were equal. Katirji observed no dominant side in a drop foot series that developed for various reasons.[12]

Conservative treatment for a duration of three or four months is recommended in case of compression due to a certain activity or position. Neurolysis or operative loosening of the fascial arch between the two heads of the peroneus longus should be performed in patients with acute axonal lesion showing no improvement in this period.[1,14,24] Thoma et al. reported that a one degree improvement in the muscle force during follow-up is a sign of the positive prognosis of conservative treatment.[24] Surgical treatment is recommended for patients experiencing drop foot for more than one year.[24] Neurolysis should be considered for patients who show no sign of improvement in electrophysiological tests.[1] Sangwan et al. reported that peroneal nerve palsy developing due to working in a squatting position is temporary and, thus, conservative treatment should be applied.[21] Similarly,

we treated our patients mostly by conservative methods. Two of our patients who showed no recovery at the end of the 12th week and showed no sign of improvement in electrophysiological studies underwent surgery. These two patients fully recovered by the first year follow-up. Vastamäki reported that spontaneous recovery can occur between 18 and 24 months.[25] Rapid recovery in a great majority of patients suggests that the conduction block in this compressive neuropathy is not a true demyelization. This can be attributed to the fact that the ischemic-metabolic causes of the conductive block resolved without morphologic changes or displacement and penetration to the next segment of the Ranvier node due to mechanical reasons.[26,27]

In conclusion, peroneal nerve palsy developing due to working in a squatting position is a preventable condition. Job descriptions and working conditions should be taken into account in such cases. Recovery should be expected by resting, conservative treatment and close follow-up. Surgery should be considered if electrophysiological studies show no sign of improvement in a 12-week follow-up, especially in patients with axonal damage. Patient awareness, especially of those who work in regions where agriculture largely depends on manpower, should be increased regarding the relationship between working in prolonged squatting position and peroneal nerve palsy. Warnings provided to workers, such as frequently bringing the extremities to extension while working can prevent nerve palsy or reduce the severity of palsy. More comprehensive epidemiologic studies are necessary to determine its incidence and precisely identify its relationship with seasonal work.

Conflics of Interest: No conflicts declared.

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