PERIPHERAL NEUROPATHY ASSOCIATED WITH PSORIASIS

PSORİAZİS İLE İLİŞKİLİ PERİFERİK NÖROPATİ

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ABSTRACT

Peripheral neuropathy is an uncommon complication of the autoimmune dermatoses. We present a patient who developed a right-sided foot muscle weakness and a left-sided foot drop following bilateral peroneal nerve lesions and polyneuropathy occurring 14 years after the onset of psoriasis. No clinical involvement of the central nervous system or any other causative factor for peripheral neuropathy was determined. Although physiotherapy and rehabilitation protocol was applied, clinical and electrodiagnostic findings remained unchanged throughout the one-year follow-up. We describe herein a patient presenting with peripheral neuropathy as an unusual manifestation of psoriasis. Physical Therapy and Rehabilitation specialists' are advised to be alert to the possible association between psoriasis and peripheral neuropathy.

Key words: Psoriasis, neuropathy, foot drop

ÖZET

Periferik nöropati otoimmün dermatozların nadir görülen bir komplikasyonudur. Psoriazis gelişmesinden 14 yıl sonra bilateral peroneal sinir lezyonu ve polinöropatiyi takiben, sağ ayakta kuvvet kaybı ve solda düşük ayak gelişen bir olgu sunulmuştur. Periferik nöropatiye neden olabilen herhangi bir faktör veya santral sinir sistemine ait herhangi bir lezyon belirlenmemiştir. Fizik tedavi ve rehabilitasyon uygulamasına rağmen bir yıllık takip sonunda klinik ve elektrodiagnostik bulgularda değişiklik olmamıştır. Burada psoriazisin olağan dışı bir belirtisi şeklinde periferik nöropati gelişen bir olgu tanımlanmıştır. Fizik Tedavi ve Rehabilitasyon uzmanlarının psoriazis ve periferik nöropati arasındaki olası ilişki bakımından dikkatli olmaları önerilmiştir.

Anahtar kelimeler: Psoriazis, nöropati, düşük ayak

INTRODUCTION

Psoriasis is a common, chronic inflammatory disease of the skin. Like a number of autoimmune and inflammatory diseases, psoriasis has a genetic component, although the inheritance pattern is still unclear. Psoriasis can also be triggered by an environmental stimulus such as psychological stress, physical trauma, sunburn, smoking, and alcohol (1-3). Approximately 70-80% of all patients with psoriasis can be treated adequately with use of topical therapy. Since immunosuppressive agents such as methotrexate, cyclosporin and retinoids have significant drawbacks, such as toxicity and relapse of the disease, patients on these agents must be monitored closely. In recent years, new biologic agents - alefacept, efalizumab, and etanercept - that specifically target key mechanisms of the pathogenesis of psoriasis have had a major impact on the treatment (2,4,5).

Peripheral neuropathy most frequently occurs as a result of mechanical trauma, metabolic disease,

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autoimmune or connective tissue disease (6). Although it has been documented in rheumatoid arthritis, psoriathic arthritis, and Sjögren's syndrome (7-9), the concomitant occurence of peripheral neuropathy in patients with psoriasis has been rarely reported in the literature (10).

Here we present a patient who developed a right-sided foot weakness and a left-sided foot drop following mild axonal sensorimotor polyneuropathy and denervated peroneal nerve, total on the left and severe on the right side occurring 14 years after the onset of psoriasis.

CASE REPORT

A 26-year-old female with psoriasis presented to our outpatient clinic with a right-sided foot weakness and a left-sided foot drop, which developed over a one-month period. Three weeks earlier, she experienced painful paresthesia, bilateral numbness in her calf area and feet, and difficulty lifting both distal lower extremities, especially on the left side, when walking.

She had a 14-year history of histologically confirmed chronic plaque psoriasis. Her joints were not affected. No family history of psoriasis or other prior clinical manifestations were identified. She had previously been treated at various times with topical steroids and methotrexate 15 mg/week p.o. (for a two-month period only two years before) according to her initial visit. She had no evidence or history of trauma, neurologic disorder, hereditary polyneuropathy, systemic illness, exposure to toxic agents, diabetes mellitus, or tobacco or alcohol abuse.

On neurological examination, cranial nerves and cerebellar tests were intact. Physical examination of the muscles of the hip and knee joints on both sides was normal, while the muscle strengths in the ankle dorsiflexors and toes were grade 0/5 on the left and 3/5 on the right. Straight leg raising test was negative. Deep tendon reflexes were normal and plantar responses were flexor. Light touch, vibration, two-point discrimination, and position sense were mildly diminished on both sides (especially on the left side). There was no atrophy or fasciculation in the upper and lower limbs. She had a steppage gait due to left foot drop and right ankle dorsiflexor muscle weakness.

The possibility of a parasagittal cerebral lesion was ruled out by computed tomographic scan. To exclude polyradiculopathy and lumbosacral plexopathy, lumbar magnetic resonance imaging was performed, which revealed diffuse annular bulging at the L5-S1 level, but there was no sign of nerve root compression or neural foraminal stenosis. Two-sided bilateral radiographs of the fibula did not demonstrate any bone cyst or tumor.

Routine laboratory investigations including complete blood cell count, erythrocyte sedimentation rate, liver and renal chemistries, electrolytes, thyroid function, vitamin B12 level, antinuclear antibody, complement levels, rheumatoid factor, and serum protein electrophoresis revealed no abnormalities.

Serial electrophysiological examinations were performed at baseline and at the 12-month follow-up since her admission (Table 1). Nerve conduction studies confirmed the presence of mild axonal sensorimotor polyneuropathy and denervated peroneal nerve, total on the left and severe on the right side. Needle electromyography of the left tibialis anterior revealed fibrillation potentials, and positive sharp waves with no recruitment or activation. Repeat nerve conduction study demonstrated similar results.

Table-I

Results of the nerve conduction studies at baseline (I) and 12-months (II) of follow-up.

	Latency (ms)			Amplitude (mv)			Conduction Velocity (m/s)		
	I	II	Normal	I	II	Normal	I	II	Normal
Right Sural NCS	3.2	3.4	≤4.4	9.4	8.2	≥6.0	40.6	41.2	≥40.0
Left Sural NCS	2.8	3.4	≤4.4	12.0	6.2	≥6.0	42.9	41.2	≥40.0
Right Peroneal NCS									
Ankle-Fibular neck	20.7	0	≤4.1	0.4	0	≥8.0	28.6	0	≥51.0
Fibular neck-Knee	23.5	0		0.3	0		32.1	0	
Left Peroneal NCS									
Ankle-Fibular neck	0	0	≤4.1	0	0	≥8.0	0	0	≥51.0
Fibular neck-Knee	0	0		0	0		0	0	

Skin temperature was above 30°C for all extremities evaluated.
NCS, nerve conduction study

After this evaluation, a physiotherapy and rehabilitation protocol was applied for four weeks. The protocol included superficial heat for 20 minutes followed by neuromuscular electrical stimulation (galvanic current, for a total of 20 minutes, with a rest of 5 minutes between two 10-minute periods), exercises (ambulatory exercises to modify her gait, passive, active-assistive and active range-of-motion and strengthening exercises), and orthotic support (bilateral ankle foot orthoses to prevent foot drop while walking). Approximately one year after her initial presentation, she was able to walk with bilateral ankle foot orthoses without apparent improvement; her clinical and electrodiagnostic findings also remained unchanged throughout the follow-up period.

DISCUSSION

Psoriasis is a chronic condition that presently is not curable. Current treatments may be effective in controlling psoriasis to a certain degree but have the potential for adverse effects and toxicity (2). Drug-induced peripheral neuropathies have been observed in these groups of patients. Kern et al. reported a case of sensorimotor peripheral neuropathy following 40 years' use of an ammoniated mercury ointment in a patient treated for psoriasis (11). Chroni et al. presented a 39-year-old male with chronic plaque psoriasis who developed clinical and electrophysiologic features of polyneuropathy affecting motor and sensory fibers in the upper and lower extremities after three months of treatment with oral acitretin (12). A case of demyelinating disease in a patient treated with etanercept for psoriasis was also reported (13). Our patient did not describe previous use of topical germicidal, oral acitretin or etanercept. Her history revealed only treatment with topical steroids and methotrexate for two months. However, at the time of the investigation, the patient had not received methotrexate for several months, minimizing the probability of the etiology of methotrexate-induced polyneuropathy. Peroneal neuropathies present with foot drop resulting from significant muscle weakness of ankle and foot dorsiflexion (2). Although injury to the peroneal nerve is usually the major cause, in our view, extensive investigations must be done in all patients who are referred with foot drop.

The role of stress as a triggering factor and symmetric distribution of psoriasis have suggested a possible role of neuropeptides, particularly substance P, in the etiopathogenesis of psoriasis. Previous studies have suggested a connection between peripheral nerves and psoriasis. It has been reported that all cutaneous neural elements in patients with psoriasis are altered, and that substance P may be an important mediator in the inflammatory processes of a psoriatic lesion (14,15). However, Chroni et al, in a clinical and electrophysiologic study of 32 patients with psoriasis, did not find evidence of large-fiber neuropathy (16). Concomitant psoriasis and peripheral polyneuropathy had been reported in dermatologic literature describing only three cases by Sindrup et al. (10). They presented the possibility that coincidence of psoriasis with symptoms of polyneuropathy revealed varying degrees of both sensory and motor nerve affection. The results of the neurophysiological examination of their cases presented a wide range of abnormalities. Our patient's neurophysiological findings were similar to their findings. In the one-year follow-up of our case, no improvement was recorded according to clinical and electrophysiological examinations.

Our case serve to strengthen the concept of a relationship between peripheral nerves and psoriasis. Moreover, although there are scarce reports in the literature of such an association, Physical Therapy and Rehabilitation specialists' are advised to be alert to the possible association between psoriasis and peripheral neuropathy.

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