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Compression Neuropathies Coexisting with Motor Neuron Disease

Motor Nöron Hastalıkları ile Birlikte Görülen Sıkışma Nöropatileri

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ÖZET

Progresif musküler atrofi, üst motor nöron bulguları olmaksızın izole alt motor nöron tutulumu ile karakterize atipik bir motor nöron hastalığıdır. Motor nöron hastalıklarında duyu semptomları ve duysal sinir ileti çalışmalarında anormallik beklenmez; bu nedenle duysal ileti bozuklukları tanısal belirsizliğe yol açabilir. Bununla birlikte, özellikle ileri yaşta veya immobilize hastalarda kompresyon nöropatileri gibi sekonder durumlar motor nöron hastalığına eşlik edebilir. Bu gibi olgularda duysal sinir aksiyon potansiyellerindeki anormallikler motor nöron hastalığı tanısının gecikmesine neden olabilir. Bu olgu sunumunda, PMA tanısı alan ve eşlik eden kompresyon nöropatilerine bağlı duysal sinir ileti anormallikleri saptanan ileri yaşta bir hasta sunulmuştur. Bu olgu, klinik bulgular ve iğne elektromiyografi anterior boynuz hücresi tutulumunu güçlü biçimde desteklediğinde, duysal ileti anormalliklerinin dikkatle değerlendirilmesi ve sekonder nedenlerin mutlaka göz önünde bulundurulması gerektiğini vurgulamaktadır.

Anahtar Kelimeler: Motor nöron hastalığı, progresif musküler atrofi, kompresyon nöropatisi, elektromiyografi

ABSTRACT

Progressive muscular atrophy (PMA) is an atypical motor neuron disease characterized by isolated lower motor neuron (LMN) involvement without upper motor neuron signs. Sensory symptoms and abnormalities in sensory nerve conduction studies are generally not expected in motor neuron diseases and may lead to diagnostic uncertainty. However, secondary conditions such as compression neuropathies may coexist, particularly in elderly or immobilized patients. In such cases, abnormal sensory nerve action potentials (SNAPs) may delay the diagnosis of motor neuron disease. We report an elderly patient diagnosed with PMA in whom concomitant compression neuropathies resulted in abnormal sensory nerve conduction findings. This case emphasizes that sensory abnormalities should be interpreted cautiously and that secondary causes should be considered when clinical and needle Electromyography findings strongly support anterior horn involvement.

Key words: Motor neuron disease, Progressive muscular atrophy, Compression neuropathy, Electromyography

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INTRODUCTION

Motor neuron diseases (MNDs) are a heterogeneous group of neurodegenerative disorders characterized by progressive degeneration of upper and/or lower motor neurons (1). Amyotrophic lateral sclerosis (ALS) is the most common form and involves both upper and lower motor neurons, whereas atypical variants such as progressive muscular atrophy (PMA) present with isolated lower motor neuron involvement (2). Sensory signs and symptoms are not expected in MNDs, and sensory nerve conduction studies are typically normal (3). Therefore, abnormalities in sensory nerve action potentials (SNAPs) are often considered exclusionary findings and may lead clinicians to pursue alternative diagnoses.

Nevertheless, secondary conditions such as compression neuropathies, age-related sensory axonal loss, or coincidental polyneuropathies may coexist with MND, particularly in elderly or immobilized patients (4-8). These conditions may confound electrophysiological evaluation and delay diagnosis. The present case highlights this diagnostic pitfall by demonstrating PMA accompanied by compression neuropathies causing abnormal sensory nerve conduction findings.

CASE REPORT

An 81-year-old woman presented with progressive weakness of both upper extremities that began approximately nine months prior to admission and gradually worsened. Weakness in the lower extremities developed during the last three to four months. Initially, she experienced easy fatigability and difficulty lifting objects; however, in the weeks preceding admission, she became unable to perform basic activities of daily living, including feeding herself. Dysphagia developed one month before presentation. She denied sensory symptoms, neck or low back pain, and urinary incontinence. A urinary catheter was placed due to severe mobility limitation. Her medical history included hypertension, coronary artery disease, and deep vein thrombosis. There was no history of diabetes mellitus, chronic alcohol use, exposure to neurotoxic agents, or prior peripheral neuropathy.

Neurological examination revealed reduced gag reflex, tongue atrophy with fasciculations, and generalized hypotonia. Muscle strength was 0/5 in proximal upper extremities, 1/5 in distal upper extremities, and 3/5 in both proximal and distal lower extremities. Deep tendon reflexes were absent in all extremities. Sensory examination and sphincter function were normal. Speech was preserved. Weakness was more pronounced in the distal muscles of the upper extremities bilaterally, particularly affecting hand grip and fine motor movements. The patient had marked difficulty holding utensils and performing buttoning tasks. Despite severe motor impairment, no objective sensory deficit was detected on examination. Muscle bulk was visibly reduced in the intrinsic hand muscles, and fasciculations were intermittently observed in the forearm muscles. Cervical magnetic resonance imaging was unremarkable. Electroneuromyography demonstrated reduced compound muscle action potential (CMAP) amplitudes in bilateral median, ulnar, peroneal, and posterior tibial nerves. Sensory nerve conduction studies showed reduced SNAP amplitudes in bilateral median and left ulnar nerves, with absent right ulnar SNAP (Table 1). Needle EMG of bulbar, thoracic paraspinal, and limb muscles innervated by different roots and nerves revealed chronic neurogenic motor unit changes and subacute denervation (Table 2). Based on clinical and electrophysiological findings, PMA was considered the most appropriate diagnosis.

DISCUSSION

Motor neuron diseases cause progressive, painless muscle weakness due to degeneration of motor neurons in the motor cortex, brainstem, and spinal anterior horn. PMA represents a rare phenotype characterized by isolated lower motor neuron involvement (1). The diagnosis of PMA relies primarily on clinical presentation and needle EMG findings demonstrating widespread chronic and active denervation (2). Sensory nerve conduction studies are generally normal in motor neuron diseases and are mainly used to exclude mimicking peripheral neuropathies (3). Preserved SNAPs despite severe muscle atrophy are considered a hallmark of motor neuronopathies. Consequently, abnormal sensory findings may raise diagnostic concern (4,5). Although rare, previous studies have reported mild sensory abnormalities in ALS (6-8), particularly in elderly patients, while pathological involvement of dorsal root ganglia or posterior columns remains uncommon (9,10). Several case reports and small series have described reduced SNAP amplitudes or focal sensory conduction abnormalities in patients with motor neuron diseases, a pattern similarly observed in the present case (7). These findings are often attributed to coexisting conditions such as entrapment neuropathies or age-related axonal degeneration rather than primary involvement of sensory pathways. Additionally, sensory involvement may also be observed in Kennedy disease, a motor neuron disorder, and may be related either to concomitant diabetes or degeneration of the dorsal root ganglia (11,12). Similar to previously reported cases, our patient demonstrated reduced SNAP amplitudes without accompanying sensory complaints, suggesting a secondary rather than primary sensory pathology.

In the present case, abnormal SNAP findings initially complicated the diagnostic process. However, the absence of sensory symptoms, the presence of diffuse LMN signs, and characteristic needle EMG findings strongly supported a diagnosis of PMA. The sensory abnormalities were most

Table 1. Nerve Conduction Study

Nerve	Distance (mm)	Latency (msec)	Velocity (m/sec)	Amplitude (u-mv)	Response
Right peronealis					
ankle		4,04		1,5 mv	F:58,6ms
below the head of fibula	270	9,72	47,5	1,3 mv	
above the head of fibula	100	11,72	50	1 mv	
Right tibialis posterior	370	5,52	51	4 mv	F:53,8ms
Left peronealis	325	4,62	58	0,6 mv	F:48,8ms
Left tibialis posterior	380	3,84	50,7	3,60 mv	F:52,8ms
Right suralis	130	2,88	47,3	10 µv	
Left suralis	120	2,0	60	10,7 µv	
Right median motor					
wrist		2,36		0,35 mv	F:25ms
elbow	240	7,2	49,6	0,30 mv	
axilla	250	11,9	52,5	0,25 mv	
Right ulnar motor					
wrist		2,64		0,39 mv	
below the elbow	180	6,04	52,6	0,35 mv	
above the elbow	100	8,12	48,1	0,33 mv	
axilla	180	11,8	48,4	0,30 mv	
Right median sensory					
3.finger-wrist	130	2,53	50,8	6 µv	
2.finger-wrist	130	3,36	38,7	5 µv	
1.finger-wrist	110	1,84	59,9	6 µv	
Mid palm-wrist	90	2,36	38,1	8 µv	
Right ulnar sensory					
4.finger-wrist		Not obtained			
		Not obtained			
Right median sensory(4.finger-wrist)	115	2,0	57,5	5,33 µv	
Right median sensory (wrist-elbow)	250	3,52	71	30 µv	
Right ulnar sensory (wrist-elbow)		Not obtained			
Left median sensory					
3.finger-wrist	140	3,48	40,2	5 µv	
2.finger-wrist	120	3,24	37	5 µv	
1.finger-wrist	100	2,08	48,1	6 µv	
Mid palm-wrist	80	2,27	35,1	8 µv	
Left ulnar sensory	120	2,36	50,5	5 µv	
Left median sensory (wrist-elbow)	250	3,60	69,4	30 µv	
Left ulnar sensory (wrist-elbow)	220	3,24	67,9	10 µv	
Left median motor					
wrist		2,60		0,5 µv	
elbow	240	7,60	48	0,48 µv	
Left ulnar motor					
wrist		2,56		0,72 µv	
below the elbow	180	6,2	49,5	0,6 µv	
above the elbow	100	8,27	48,1	0,5 µv	
axilla	170	11,7	49,4	0,48 µv	

plausibly explained by compression neuropathies (4), likely related to prolonged immobilization and the use of supportive devices (13). Compression neuropathies are a recognized source of diagnostic confusion in motor neuron diseases. Patients may be misdiagnosed with entrapment neuropathies in early disease stages or may develop secondary compression neuropathies following reduced mobility. This case emphasizes that abnormal SNAP findings should not automatically exclude motor neuron disease when the overall clinical and electrophysiological picture is consistent with anterior horn involvement.

This case contributes to the existing literature by highlighting that bilateral distal upper extremity weakness accompanied by abnormal sensory nerve conduction findings

Table 2. Needle Electromyography Findings

Muscle	Spontaneous Activity	Motor Unit
Right tibialis anterior	Fasciculation +	Long duration (17 ms), polyphasicity MUAP. Decreased interference
Vastuslateralis		Long duration (20 ms), polyphasicity MUAP. Decreased interference
Left tibialis anterior		Large amplitude (6,5 mv), long duration (20 ms) polyphasicity MUAP. Decreased interference
Vastuslateralis	Fasciculation +	Positive sharp waves. Long duration (18 ms) MUAP
Right deltoid		voluntary MUAP could not be obtained
Left deltoid		voluntary MUAP could not be obtained
Biceps	Fasciculation +	Large amplitude (8mv), long duration (22 ms) MUAP. Decreased interference
Right genioglossus		Large amplitude (3mv), long duration (12 ms) MUAP. Decreased interference
Left frontalis		Normal MUAP. interference pattern
Cervical paraspinal		long duration (22 ms), polyphasicity MUAP
Paraspinal Th1		long duration (18 ms), Polyphasicity MUAP
Right sternocleidomastoid		long duration, polyphasicity MUAP.

may still represent a motor neuron disorder when needle EMG and clinical features are strongly suggestive. Awareness of this overlap may prevent misdiagnosis and unnecessary delays in establishing the correct diagnosis.

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