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대상포진에 의해 발현된 근위축성 신경통

고려대학교 의과대학 신경과학교실

김병조 · 고성범 · 박민규 · 박건우 · 이대희

Neuralgia Amyotrophy Induced by Herpes Zoster

Byung Jo Kim, M.D., Seong Beom Koh, M.D., Min Kyu Park, M.D., Kun Woo Park, M.D., Dae Hie Lee, M.D.

Department of Neurology, Korea University Hospital, Seoul, Korea

- Abstract -

The etiology of neuralgia amyotrophy remains unclear. Herpes zoster induced neuralgia amyotrophy has been reported in extremely rare cases. In this case report, we describe the clinical features and electrophysiologic findings in a 68-year-old patient with neuralgia amyotrophy associated with herpes zoster infection. We suggest that brachial plexus inflammation due to viral infection may be a direct cause of reversible neuralgia amyotrophy.

Key Words : Neuralgia amyotrophy, Herpes zoster, Electrophysiologic findings, Brachial plexus inflammation

INTRODUCTION

Neuralgia amyotrophy is a condition that has been described by a wide range of terms and diagnoses. This multitude of historical synonyms illustrates poor understanding and inconsistent recognition of neuralgia amyotrophy. The etiology also remains unclear. About half the cases have been associated with antecedent illnesses or events such as a vaccination. We describe here a strong correlation with herpes zoster infection.

CASE REPORT

A 68-year old right-hand-dominant man presented with weakness of the left upper limb for approximately four weeks in duration. Six weeks prior to the hospital visit, the patient began to experience severe lancinating pain in the left shoulder and back area. Five weeks prior to the hospital visit, vesicular eruptions developed in the left C5, C6 dermatome. A local clinic physician diagnosed with herpes zoster infection and treated. Four weeks prior to the hospital visit, the left shoulder and back area pain began to subside. However, weakness of the left upper

교신저자 : 이 대 희
서울특별시 성북구 안암동 5가 126 - 1
고려대학교 의과대학 신경과학교실
TEL) 02 - 920 - 5347, FAX) 02 - 929 - 9435, e - mail) nukbj@hananet.net

limb, primarily at the shoulder, began to develop. Nerve conduction studies and needle electromyogram performed at that time were normal.

On hospital visit, remnants of crust were observed on the skin of the left shoulder. Manual muscle testing demonstrated weakness in the left shoulder abduction/external rotation of power 3/5(Medical Research Council scale), and a left elbow flexion of power 4/5. Wrist and finger strength were found to be intact. Strength in the right limbs was normal. There was no muscle atrophy in the left shoulder girdle. The left biceps reflex was less active than the right. The rest of the muscle stretch reflex tests were normoactive. In the left upper limb, decreased sensation to pin prick over the deltoid region and lateral forearm was observed.

An electrodiagnostic study was performed(Table 1). The left lateral antebrachial cutaneous sensory nerve action potential(SNAP) amplitude was reduced by 50% compared to the right. The left axillary and musculocutaneous compound muscle action potential amplitude was reduced by 60% and 70%, respectively, compared to the right. The left musculocutaneous motor latency was more prolonged than the right. The needle electrode examination showed denervation potentials(2+ positive sharp wave & fibrillation) and reduced recruitment of motor unit activation in the supraspinatus, infraspinatus, deltoid, and biceps brachi muscles of the left shoulder and arm. Normal muscle activities were observed in the other whole upper extremity muscles, including the cervical paraspinal muscle, serratus anterior, teres major, and clavicular & sternocostal portion of the pectoralis major. All the described findings suggest consistent involvement of nerves originated from the upper trunk level.

The cervical spine MRI did not show abnormal findings. Lumbar puncture did not indicate any remarkable findings. An extensive search for systemic disease and malignancy was also negative. The patient underwent courses of the physical therapy, and the patient 's condition appeared to be slowly improving. However, no significant improvement was observed at the follow-up examination 3 months later.

Table 1. *Nerve conduction studies*

Nerve	Recording site	Variable	Right	Left	Normal
Motor nerves					
Median	APB	Amplitude, distal(mV)	14.6	13.7	≥6
		Distal latency(ms)	3.1	3.3	≤3.6
		F-wave minimal latency(ms)	26.8	27.1	≤31.0
Ulnar	ADQ	Amplitude, distal(mV)	12.4	10.8	≥7
		Distal latency(ms)	2.8	2.5	≤3.0
		F-wave minimal latency(ms)	26.8	26.4	≤31.0
Radial	EIP	Amplitude, distal(mV)	6.7	7.2	≥5
		Distal latency(ms)	2.0	2.1	≤2.4
Axillary	Deltoid	Amplitude, distal(mV)	6.4	2.5	≥5
		Distal latency(ms)	4.3	4.6	≤5.0
Musculocut	BB	Amplitude, distal(mV)	6.9	2.1	≥2.5
		Distal latency(ms)	4.1	5.3	≤5.0
Sensory nerves					
Median	D2	Amplitude(uV)	18.2	15.4	≥12
		Distal latency(ms)	3.1	2.9	≤3.4
Ulnar	D4	Amplitude(uV)	15.4	16.2	≥12
		Distal latency(ms)	2.4	2.2	≤2.8
Radial	Anatomical	Amplitude(uV)	23.2	20.4	≥10
	snuffbox	Distal latency(ms)	2.1	2.3	≤2.6
Lat Ant Cut	Forearm	Amplitude(uV)	13.4	6.4	≥10
		Distal latency(ms)	2.2	2.5	≤2.6

APB=abductor pollicis brevis; ADQ=abductor digiti quinti; EIP=extensor indicis proprius; Musculocut=musculocutaneous; BB=biceps brachi; D2=digits 2; D4=digits 4; Lat Ant Cut=lateral antebrachial cutaneous nerve. Abnormal values are underlined.

DISCUSSION

This patient 's examination and diagnostic findings described are typical for neuralgia amyotrophy. Acute pain followed by developing weakness is a common characteristic of the disorder. This case describes the multiple mononeuropathy. The normal needle electrode exam in the cervical paraspinal muscles excludes cervical radiculopathy. Moreover, the lateral antebrachial cutaneous sensory amplitude recording is diminished, rendering the possibility of cervical radiculopathy unlikely. Some of the findings from the needle exam suggest either involvement of the upper trunk or the multiple nerves that originate from the upper trunk. However, the upper trunk lesion tends to be excluded by a normal needle exam of the clavicular portion of the pectoralis major and teres major.

Neuralgia amyotrophy has been and continues to be called by many names. Parsonage and Turner described a large series of cases in 1948 and the condition continues to be called Parsonage-Turner Syndrome. Other names include acute brachial neuropathy, acute brachial radiculitis, acute brachial plexitis, acute multiple neuropathy of the shoulder girdle, acute shoulder neuritis, amyotrophic paralysis of the periscapular muscles, and so on. The name neuralgia amyotrophy has been proposed because it does not convey any assumptions about etiology or location of the lesion. Leaving the name " brachial " out of the name is also anatomically correct because there are cases in which there is involvement of nerves that do not pass through the brachial. This condition can also occur in the lower limbs.[1-3]

Although an inflammatory-immune mechanism has been suggested as a cause, the true etiological nature of neuralgia amyotrophy remains unclear. Neuralgia amyotrophy is associated with preceding viral infections, vaccinations, other insults, and the use of immunomodulating agents such as interleukin-2 and interfereon therapy.[4-9] However, neuralgia amyotrophy following herpes zoster infection is extremely rare. In 1861, von Barensprung first documented the nervous system with zoster infection. Despite the preferential involvement of the sensory pathways, motor symptoms may also rarely complicate herpes zoster. Once paresis occurs, maximum weakness appears within hours or days. The most common motor symptom in herpes zoster is facial palsy. Motor symptoms outside the cranial nerve are extremely rare.[10] In 1944 Denny-Brown demonstrated inflammation of the peripheral nerve root postulating that herpes zoster motor involvement results from ventral root involvement by contiguous spreading from the adjacent dorsal root ganglia or inflammation along the length of the mixed nerve.[11] An autopsy study of limb weakness was recently reported. In this report, the histological evidence suggests that brachial plexus neuritis and paresis is due to a direct primary inflammatory process.[12]

We concluded the patient had herpes zoster complicated by neuralgia amyotrophy. In the case of herpes zoster infection, the clinician should consider peripheral neuropathy with motor involvement as well as postherpetic neuralgia with sensory involvement.

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