ORIGINAL ARTICLE

Is Bell's palsy a component of polyneuropathy?

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OBJECTIVE: To define whether Bell's palsy is associated with polyneuropathy.

MATERIALS AND METHODS: Thirty patients with unilateral Bell's palsy were investigated in this study. All patients underwent facial and peripheral nerve conduction and needle electromyography studies. Eighteen healthy volunteers comprised the control group. Electrophysiological findings of patients with Bell's palsy were compared with those of normal subjects.

RESULTS: Eight patients had sensorimotor peripheral neuropathy. Twenty-two patients had no prominent abnormalities in their electrophysiological investigations. None of the 18 healthy volunteers had any electrophysiologic abnormalities.

CONCLUSION: Bell's palsy might be a part of subclinical polyneuropathy.

Bell's palsy is an isolated, sudden, peripheral facial paralysis of unknown etiology. In the differential diagnosis of Bell's palsy, conditions such as temporal bone fractures, acoustic neuroma, infection or tumor of the middle ear, and disorders of the parotid gland must be excluded.[1] Bell's palsy is generally thought to be a peripheral neuropathy due to viral infection that causes swelling of the cells of the facial nerve. Recently, some investigations have implicated the herpes simplex virus as a cause of facial nerve inflammation and dysfunction that commonly leads to acute facial paralysis.[2] Abdel-Baki and colleagues concluded that idiopathic facial paralysis is part of a cranial polyneuropathy.[3] Some authors also argue that viral agents may trigger cranial or generalized polyneuro-pathy in Bell's palsy.[1]

In this prospective study, we aimed to investigate the involvement of peripheral nerves in acute Bell's palsy and to determine whether there is any relation between Bell's palsy and peripheral polyneuropathy.

PATIENTS AND METHODS

Forty-five patients with unilateral acute Bell's palsy who were admitted to our otolaryngology department between January 2002 and January 2003 were investigated prospectively in this study. The medical ethics committee of the hospital approved the study protocol. Informed consent was obtained from each participant according to the Declaration of Helsinki.[4] Fifteen of the patients were excluded for middle ear infection (8 patients), diabetes mellitus (5 patients), and thyroid disease (2 patients).

The remaining 30 patients who had no history of diseases that can lead to peripheral neuropathy were included in the study (17 men, 13 women; mean age, 41.1±16.2 years; range, 19-70 years; Table 1). As a control group, 18 healthy volunteers were enrolled in the study (7 men, 11 women; mean age, 39.1±14.3 years; range, 21-66 years).

All patients were examined on the 15th day after the first appearance of symptoms. Patients were examined thoroughly by both an otoneurologist and a general neurologist. Bell's palsy was encountered 14 times on the left side and 16 times on the right. Except for unilateral Bell's palsy, the clinical neurological examination was normal in all patients.

All patients underwent facial and peripheral nerve conduction and needle electromyography (EMG) studies. Facial nerve conduction study was performed as described by Oh.[6] The zygomatic branch of the facial nerve was stimulated anterior and inferior to the tragus. Surface electrodes were used for recording. The active electrode was placed over the midpoint of the lower portion of the orbicularis oculi, and the reference electrode was placed above the midpoint of the eyebrow. The ratio of amplitudes between the paretic side and the normal side is considered to reflect the proportion of surviving neurons. Needle EMG of the

Table 1. Demographics of patients and controls.

Variables	
Patients	30
Sex, m/f	17/13
Mean age of patients	41.10±16.20
Mean age of patients with peripheral neuropathy	57.75±13.21
Mean age of patients without peripheral neuropathy	38.00±13.93
Controls	18
Sex, m/f	7/11
Mean age of controls	39.10±14.30

face was performed as follows. Bipolar needle electrodes were inserted into the orbicularis oculi, orbicularis oris, and frontalis muscles, all on the paretic side. The occurrence of fibrillation and/or sharp positive waves at rest was regarded as pathologic. Facial nerve conduction and needle EMG studies were not performed on healthy volunteers.

To investigate peripheral neuropathy, right upper extremity and both lower extremities were studied in each participant (patients and healthy volunteers) as described by Oh.[5]

Nerve conduction studies investigated sensory and motor involvement in the following nerves: 1 ulnar, 1 median, 1 peroneal, 1 posterior tibial, and both sural. Nerve conduction studies were carried out with the technique described by Oh.5 A Medelec Synergy EMG monitor (Oxford Instruments, Surrey, UK) was used for evaluation. For sensory nerve conduction studies, the orthodromic method was used. Band filters ranged from 20 Hz to 10 kHz, 2 Hz to 10 kHz, and 10 Hz to 10 kHz for motor and sensory nerve studies and needle EMG, respectively. Sensitivity varied between 0.5 and 5 mV/cm, and the sweep speed was set at 5 ms/cm, for an analysis time of 50 ms. The peak-to-peak amplitude was calculated in microvolts. We measured latency of sensory nerves from stimulus onset to the peak of the major negative deflection of the sensory compound nerve action potential. The diagnostic criteria of peripheral neuropathy is abnormal motor or sensory velocity or abnormal motor or sensory amplitudes or abnormal motor distal latency.[5]

Needle EMG was performed in the tibialis anterior, vastus lateralis, biceps, and abductor pollicis brevis muscles, all on the same side. When median nerve pathology was found, the contralateral extremity was examined to exclude carpal tunnel syndrome, and the forearm segment was examined to exclude proximal lesions.

All patients were still receiving steroid therapy when the nerve conduction studies were performed. The electrophysiologic findings of the patients with Bell's palsy were compared with those of matched healthy volunteers. Statistical analysis included Fischer's exact test to evaluate the difference between Bell's patients and healthy individuals. Statistical significance was defined as $P<.05$.

RESULTS

A total of 30 patients with Bell's palsy were evaluated. Their demographic data are summarized in Table 1. Sensorimotor peripheral neuropathy was found electrophysiologically in 8 patients (4 men). Three of these had also denervation potentials during needle EMG (Table 2).

The remaining 22 patients (13 men) had no prominent abnormalities in their electrophysiologic investigations (Table 3). None of the healthy individuals had pathologic findings in EMG results. The difference between patient and control groups was statistically significant ($P<.05$).

Table 2. Electrophysiological findings of 8 patients with Bell's palsy and peripheral neuropathy

no	Age	Sex	Median Motor				Ulnar Motor				Peroneal Motor				Post tibial Motor				2. F-W		5. F-W		Right Sural		Left Sural	
			DL	Vel	Amp	F	DL	Vel	Amp	F	DL	Vel	Amp	F	DL	Vel	Amp	F	Vel	Amp	Vel	Amp	Vel	Amp	Ve	Anp
1¶	70	M	3,6	52,4	5	22	2,3	58	6,3	27	(-)*	(-)*	(-)*	(-)*	5,8	44,8	3,5*	45	(-)*	(-)*	42	31,8	38	2,1*	29*	13,6
2	60	M	3,2	51,7	6,2	27,6	2,2	50,8	7,3	31*	5,1	43,7	4,4	51	5,1	41,6	3,8	49	37*	4,1*	46,7	3,3*	30,6*	4,3*	31,2*	2,1*
3¶	56	F	4,2*	43,7*	5,3	34,9*	2,95	52,3	10,3	34,9*	7,6*	38,4*	2,7*	53*	10,45*	43,1	7,6	56*	(-)*	(-)*	(-)*	(-)*	16,5*	6,9	(-)*	(-)**
4¶	66	M	5,1*	50*	2,2*	31,2*	4,8*	58,4	1,1	28,6	(-)*	(-)*	(-)*	(-)*	(-)*	(-)*	(-)*	(-)*	31,8*	13,3	(-)*	(-)*	(-)*	(-)*	(-)*	(-)*
5	51	F	5,5*	52,1*	8,7	25,7	2,25	60	7,1	25,2	6,25*	48,3	3,1*	46,8	4,05	41,3	6,4	36,5	31,7*	7,3*	41,8	13,7	39,3	1,2*	30,1*	8,2
6	42	F	2,6	59,1*	11,3	23	2,1	50,3	6,8	26	5,6	44,6	5,1	52,3*	5,75	41,2	4,9	53,3*	36,2*	3,1*	51,3	3,2*	25,3*	3,6*	35,1*	3,3*
7	56	F	4,6*	46,2	6,3	28,5	2,8	49,7	7,6	28,8	5,2	38,7*	1*	54,2*	5,1	36,5*	2,6*	52,3*	39,4	11	44,4	9,5	10,6*	9,1	37	3,9*
8	29	M	2,6	54,2*	17	26,5	2,65	48,9*	7,8	30	4,3	52	7,3	46,2	4,4	48,3	7	48,9	56,2	1,2*	52	2,3*	45,2	1,3*	42,1	2,1*

*Abnormal findings

¶Denervation potentials in needle electromyography-F, female; M, male; DL, distal latency; Vel,. velocity; amp, amplitude; F, F latency; F-W, finger-wrist segment; Post, posterior, (-),:no potential

Table 3. Electrophysiological findings of Bell's palsy patients and controls.

	Patient	Control
Median nerve		
Motor		
Distal latency	3.09±0.49	3.11±0.33
Velocity (wrist-elbow)	55.68± 5.27	57.38± 5.17
Amplitude	8.65± 2.83	7.85±2.58
F latency	25.72± 1.29	25.72± 1.81
Sensory		
2nd finger-wrist velocity	49.59± 9.39	50.45± 7.26
Amplitude	11.29± 4.53	11.94± 4.09
Ulnar nerve		
Motor		
Distal latency	2.4± 0.28	2.4± 0.44
Velocity (wrist-elbow)	61.26± 10.29	62.79± 6.54
Amplitude	9.6± 2.29	9.72± 2.16
F latency	25.95± 2.74	26.2± 2.02
Sensory		
5th finger-wrist velocity	47.14± 7.5	48.54± 8.59
Amplitude	12.41± 7.04	11.29± 5.45
Peroneal nerve		
Motor		
Distal latency	4.54± 1.59	4.43± 0.93
Velocity (ankle-fibula head)	50.3± 5.85	51.91± 4.6
Amplitude	5.05± 1.89	5.2± 2.08
F latency	41.42± 5.89	45.36± 5.06
Posterior tibial nerve		
Motor		
Distal latency	4.22± 0.51	4.54± 0.96
Velocity (ankle-popliteal fossa)	46.35± 4.51	45.5± 4.03
Amplitude	7.57± 2.16	9.65± 3.48
F latency	46.27± 6.82	45.83± 7.85
Right sural nerve		
Velocity	41.65± 6.02	40.48± 10.69
Amplitude	21.84± 9.53	25.57± 14.26
Left sural nerve		
Velocity	39.99± 5.75	60.2±104.77
Amplitude	24.17± 19.96	24.56± 14.11

DISCUSSION

The etiology of Bell's palsy is not known. Vascular, hereditary, and immunologic factors have been implicated.[7] Many authors have suggested that the facial nerve can become infected by a virus such as herpes simplex, giving rise to Bell's palsy via an autoimmune response or reactivation of the latent virus. Degeneration of myelin sheaths and axons with an increase of phagocytic cells has been found in 10% to 30% of the facial nerve fibers in patients with Bell's palsy.[8] These findings suggest that the paralysis might be caused by a viral infection.[8]

Aviel and colleagues have argued that Bell's palsy and Guillain-Barré syndrome share many clinical, histopathologic, and immunologic features.[8] The role of viruses and immune responses in the pathogenesis of peripheral and central nervous disease has been studied extensively. Some authors consider Bell's palsy to be a part of cranial polyneuropathy.[9] We suggest that Bell's palsy is not an isolated disease of the facial nerve but is part of a subclinical systemic polyneuropathy. Chaco studied 30 patients with Bell's palsy and found low conduction velocity in median and ulnar nerves in 14 of these patients.[10] Abdel-Baki and colleagues studied peripheral nerves on both sides in the arm and forearm segments and found normal axillary F latencies in all cases and motor conduction delay in 1 or more segments of the ulnar nerve in 7 patients.[3] Neither Chaco and colleagues nor Abdel-Baki and colleagues measured conduction velocities in the lower extremities.[3,10]

In our study, 8 patients (4 men) had peripheral neuropathy as demonstrated by electrophysiological examinations.

The electrophysiological findings of the remaining 22 patients and 18 healthy individuals were normal. The statistically significant difference observed between Bell's palsy patients and healthy controls recommends an important relation between Bell's palsy and polyneuropathy. To support this view, further studies with larger patient and control population is needed.

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