RESEARCH ARTICLES

THE CORRELATION OF CLINICAL SIGNS WITH THE CMAP INDEX IN BELL'S PALSY

BELL PARALİZİSİNDE KLİNİK BULGULARIN BKAP İNDEKSİYLE KORELASYONU

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ABSTRACT

Purpose: *Patients with Bell's palsy generally undergo an electromyographic examination during the acute stage of the disease. In this study, we investigated the correlation between the severity of clinical involvement and electrophysiological findings.* ***Methods:*** *We retrospectively evaluated the records of 149 Bell's palsy patients referred to the Laboratory of Clinical Neurophysiology between January 1999 and February 2001. The degree of facial weakness graded according to the House-Brackmann system was noted. Patients were divided into two groups according to the clinical severity of the lesion. Group 1 was composed of mildly affected patients of House-Brackmann grades I-III. Group 2 consisted of severely affected cases assigned to House-Brackmann grades IV-VI. Nerve conduction studies of the zygomatic branches of the facial nerves of the healthy and affected sides were recorded. A compound muscle action potential (CMAP) index was then calculated by dividing the CMAP amplitude of the diseased side by that of the unaffected side. The result was multiplied by 100. Needle electromyography (EMG) findings of the frontalis and orbicularis oris muscles were also evaluated.* ***Results:*** *As the House-Brackmann grading increased, the CMAP index decreased (Spearman r=-0.62, p=0.000), but the terminal latency to the orbicularis oculi muscle remained unchanged (Spearman r=0.14, p=0.111). Compared to group 1, the CMAP amplitudes (t=4.70, p=0.000) and indices (t=5.26, p=0.000) were significantly reduced in group 2 patients. Although the presence of fibrillation potentials and positive sharp waves did not differ between the groups (x2=0.03, p=0.86), voluntary motor unit action potential (MUP) activity was more commonly absent in group 2 patients (x2=33.0, p=0.000).* ***Conclusions:*** *The House-Brackmann grading system shows a negative correlation with the CMAP*

ÖZET

Amaç: *Bell paralizisinin akut döneminde genellikle elektomyografik incelemeye başvurulur. Bu çalışmada klinik tutuluşun ağırlığının elektrofizyolojik bulgularla korelasyonu araştırıldı.* ***Yöntem:*** *Ocak 1999-Şubat 2001 tarihleri arasında Klinik Nörofizyoloji Laboratuarına başvuran,149 Bell paralizisi olgusunu retrospektif olarak inceledik. Her olguda House-Brackmann sistemine gore değerlendirilmiş olan yüz kaslarındaki güç kaybı kaydedildi. Hastalar lezyon şiddetine göre 2 gruba ayrıldı. House-Brackmann evre I-III olarak değerlendirilen hastalar Grup 1,evre IV-VI olarak değerlendirilenler ise Grup 2 olarak tanımlandı. Sağlam ve paralitik taraflarda yapılan N.fasiyalis ramus zigomatikusa ait sinir iletim çalışmaları alındı. Daha sonra hasta tarafın birleşik kas aksiyon potansiyeli(BKAP) sağlam tarafa bölünerek BKAP indeksi elde edildi. Çıkan sonuç 100 ile çarpıldı. M. Frontalis ve orbikülaris okülide yapılan iğne elektromyografi (EMG) bulguları da değerlendirildi.* ***Sonuçlar:*** *House-Brackmann evresi arttıkça BKAP indeksi küçülüyordu (Spearman r=-0.62, p=0.000). Buna karşın orbikülaris okuli kasında ölçülen terminal latans herhangi bir değişkenlik göstermiyordu (Spearman r=0.14, p=0.111). Grup 1 le kıyaslandığında Grup 2 de BKAP amplitüdleri (t=4.70, p=0.000) ve indeksleri (t=5.26, p=0.000) anlamlı olarak küçüktü. Gruplar arasında fibrillasyon potansiyelleri ve pozitif keskin dalgaların bulunması değişkenlik göstermiyorsa da (x2=0.03, p=0.86), grup 2 olgularında istemli motor ünite aksiyon potansiyeli (MUP) aktivitesinin izlenmemesi durumuyla daha sık karşılaşılıyordu (x2=33.0, p=0.000).* ***Yorum:*** *House-Brackmann klinik evreleme sistemi BKAP indeksiyle negatif korelasyon göstermekte olup, Bell paralizisinin akut dönemdeki şiddetinin tayininde iyi bir araçtır. Ancak altta yatan fizyopatolojik sürecin kesin olarak tanımlanabilmesi amacıyla bir EMG yapılması gereklidir.*

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index and is a useful tool in the assessment of the severity of Bell's palsy in the acute stage. However, an EMG examination should still be performed in order to accurately identify the underlying pathophysiology.

Key Words: *Bell's Palsy, Nerve Conduction Studies, Electromyography.*

Anahtar Kelimeler: *Bell Paralizisi, Sinir İletim Çalışmaları, Elektromyografi.*

INTRODUCTION

Bell's palsy is the most common lower motor neuron lesion affecting the seventh cranial nerve (1). The etiology of Bell's palsy is unknown, although vascular (2), hereditary (3) and immunological (4-7) factors are listed among the probable offending causes. Electrophysiological studies may offer a reasonable method of expeditiously predicting an eventual clinical outcome and providing assurance to distraught patients concerned with facial disfigurement. The nerve excitability test is considered unreliable by some (8). While the blink reflex is useful in the determination of demyelinating lesions situated proximally, nerve conduction studies and needle electromyography (EMG) findings are more important in assessing axonal damage. Although some investigators think that compound muscle action potential (CMAP) amplitude (8-10) and terminal latency (11) measurements are more important in estimating the severity of the lesion, others maintain that needle EMG (12, 13) is the most sensitive indicator of axonal injury. In this study we attempted to define the correlation between the clinical severity of the lesion in Bell's palsy with facial nerve conduction studies and needle EMG.

MATERIALS AND METHODS

Patients:

The records of 164 patients referred to the Laboratory of Clinical Neurophysiology for facial nerve conduction studies between January 1999 and February 2001 were retrospectively evaluated. Inclusion criteria required rapidly developing idiopathic unilateral complete or partial facial nerve palsy, without long tract or cerebellar signs. Patients with an identifiable cause were excluded. Cases with recurrent or bilateral facial paralysis were not included in the study because the electrophysiological tests depended on comparing the involved side with the unaffected side. Nine iatrogenic cases due to various surgical procedures, 3 cases with mastoiditis, 2 posttraumatic cases and 1 with Guillain-Barré syndrome were excluded. Therefore, after excluding 15 cases, 149 patients fulfilling the clinical criteria for Bell's palsy remained. Their ages ranged from 6 to 85 years (mean: 41.5 years). There were 70 male and 79 female patients. From the records of the patients, clinical grading of the facial weakness according to the House-Brackmann system (14) (Table 1) was noted. Mild (Grades I-III) patients were classified as group 1 (n=106), while the more severely affected cases (Grades IV-VI) were assigned to group 2 (n=43). No significant difference existed (t=-0.93, p=0.357) between the mean ages (SD) of group 1 and 2 patients, which were 40.5 years (19.1) and 43.7 years (20.2) respectively.

Table 1: House-Brackman grading system of the clinical severity of facial paralysis.

Grade I:	Normal facial movement
Grade II:	Slight asymmetry of facial movement
Grade III:	Obvious asymmetry of facial movement, some forehead movement present
Grade IV:	Obvious asymmetry of facial movement, forehead movement absent
Grade V:	Only slight facial movement
Grade VI:	Absence of any facial movement or tone

Electrophysiological Tests:

Nihon-Kohden Neuropack and Dantec Cantata EMGs were used in all neurophysiological studies. Bandpass filters ranged from 20 Hz to 10 kHz. 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. Silver-silver chloride electrodes were used for recording. Oh's method was used in facial nerve conduction studies (13). The electrophysiological studies were performed 10-34 days after the onset of symptoms. Initially, the normal and then the affected side were investigated. An active surface electrode was placed over the midpoint of the lower portion of the orbicularis oculi muscle and a reference electrode was placed above the eyebrow along the same vertical plane of the active electrode. The zygomatic branch of the facial nerve was stimulated anterior and inferior

to the tragus of the earlobe. Latency was measured from the stimulus onset to the initial deflection of CMAP. CMAP amplitude was measured from peak to peak. The CMAP index was calculated by dividing the CMAP amplitude of the affected side by the CMAP amplitude of the normal side and then by multiplying the result by 100, as shown in the formula below:

$$\text{CMAP Index} = \frac{\text{CMAP of the affected side}}{\text{CMAP of the normal side}} \times 100$$

Needle EMG findings in all patients obtained by using concentric needle electrodes in the frontalis and orbicularis oculi muscles were also assessed. Bandpass filters ranged from 10 Hz to 10 kHz. Sensitivity was adjusted to 100 μV/cm during the investigation of spontaneous activity. Analysis time was set at 100 ms.

Statistical Analysis:

The relationship between the clinical data and CMAP index was analyzed with Spearman correlations. In the analysis of the differences between the mean terminal latencies, CMAP amplitudes and indices between the two groups, patients with CMAP indexes calculated as 0 (no potential on the affected side) were excluded. Therefore, the number of group 2 patients was reduced to 35. Analysis was performed by using Student's t test for independent samples. Chi-square tests were employed to compare the presence of spontaneous electrical and voluntary MUP activity between the two groups. Yates continuity correction was also calculated in the tests, showing significant differences between the groups. An alpha level of <0.05 was considered significant. All analyses were performed by using the Statistical Package for Social Sciences program.

RESULTS

The CMAP index findings, classified as mild, moderate or severe (Fig. 1) according to Olsen (15), are listed in Table 2 in relation to the House-Brackmann clinical grading system. The CMAP index decreased as the House-Brackmann grade increased (Spearman r=-0.62, p=0.000). However, the terminal latency of the zygomatic branch of the facial nerve did not show a significant prolongation in relation to an increase in clinical grading (Spearman r=0.14, p=0.111). Age had no significant effect on the House-Brackmann grading (Spearman r=0.15, p=0.060), CMAP amplitude (Spearman r=-0.13, p=0.123), index (Spearman r=-0.10, p=0.233) or terminal latency (Spearman r=0.16, p=0.053) of the diseased facial nerve. Group 2 patients

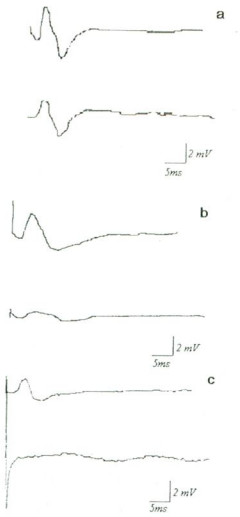

Fig. 1: Nerve conduction studies of the zygomatic branch of the facial nerve, recorded from the orbicularis oculi muscle, in mild, moderate and severe Bell's palsy. Top and bottom tracings in each recording are from the normal and the affected sides respectively. (a). A mild case of Bell's palsy. CMAP amplitude of the healthy side is 4.3 mV, compared to 3.3 mV in the affected side, yielding an index of 76. (b). Moderate involvement in Bell's palsy. Healthy side has a CMAP amplitude of 3.6 mV, compared to an amplitude of 0.9 mV on the affected side. The CMAP index is 25. (c) Severe Bell's palsy. A CMAP amplitude of 2.3 mV on the normal side is in contrast to an absent CMAP on the affected side. The CMAP index is 0. All terminal latencies are within normal range.

Table 2: Patient distribution according to CMAP index classified as mild (>30%), moderate (10-30%) and severely reduced (<10%), as defined by Olsen (15) and the House-Brackmann clinical grading system.

House-Brackmann Grade	CMAP Index		
	Mildly reduced	Moderately reduced	Severely reduced
I	28	0	0
II	35	4	0
III	24	12	3
IV	10	12	10
V	3	3	5
VI	0	0	0
Total (n=149)	100	31	18

Table 3: Mean±standard deviations of the electrophysiologic measurements in both groups.

Measurements	Group 1 (n=106)	Group 2 (n=35)	p
Age	40.5±19.1	43.7±21.0	0.416
TL (ms)	3.4±0.7/2.9±0.4	3.5±1.0/2.8±0.4	0.539/0.431
Amp (mV)	1.5±1.0/2.8±1.1	0.8±0.7/2.7±1.0	0.000/0.627
CMAP Index	54.9±24 5	30.8±20.2	0.000

TL: Terminal latency (affected side/healthy side), Amp: Amplitude (affected side/healthy side), CMAP: Compound muscle action potential.

demonstrated a significant reduction of CMAP amplitudes (t=4.70, p=0.000) and indices (t=5.26, p=0.000) compared to group 1 patients (Table 3), but no significant differences in terminal latencies existed between the two groups (t=-0.62, p=0.539). Fibrillation potentials and positive sharp waves were observed in at least one of the two examined muscles in 90 (85%) group 1 and 36 (83.5%) group 2 patients (x2=0.03, p=0.86). On the other hand, absent voluntary motor unit action potential (MUP) activity in at least one examined muscle was encountered in only 7 (6.5%) group 1 patients, in contrast to 21 (49%) patients in group 2 (x2=33.0, p=0.000).

DISCUSSION

Our findings revealed that as the clinical grading of patients with Bell's palsy deteriorates, the CMAP index becomes progressively smaller. CMAP index and amplitude are also significantly reduced in the group 2 patients with more severe clinical involvement. Previous studies have shown that as the CMAP amplitude is reduced on the affected side, the prognosis becomes increasingly dismal (8). A CMAP index above 30-50% has been associated with good prognosis (8, 9, 15). If the index is between 10 and 30%, recovery is expected to last up to 8 months with some degree of functional impairment (15). An index of less than 10% is an invariable indicator of poor prognosis (8, 15). Although May et al. stated that a CMAP index below 25% indicated incomplete recovery, their patients were followed for only 6 months (9). It is known that recovery may be prolonged for up to a year in Bell's palsy (8). Most investigators recorded the CMAP from nasal ala muscles, in contrast to the orbicularis oculi we employed (8, 9). Olsen used coaxial electrodes for recording from facial muscles (15). We do not think that the employment of different techniques affects the interpretation of results, although a surface recording technique is more valuable than an intramuscular recording, as it picks up the electrical activity of a greater number of motor units (13). Therefore, CMAP amplitude and particularly index measurements in facial nerve conduction studies are of prime importance in assessing the severity and prognosis of the lesion. We found that older patients were not necessarily more severely affected by the disease, which is in contrast to recent studies reporting the poor outcome of Bell's palsy in elderly individuals (16-18). Our findings show that severity of involvement correlates better with nerve conduction studies of the facial nerve than the age of the patient.

Terminal latency measurements do not reflect the clinical severity of the disease. A previous report indicated that a slow nerve conduction velocity of the facial nerve was associated with delayed or incomplete recovery (19). We did not perform nerve conduction velocity measurements, as they present considerable discomfort to the patient. Moreover, the short distance between the proximal and distal stimulating points may preclude an accurate calculation of the conduction velocity. It had previously been stated that recording from the orbicularis oris, a prolonged terminal latency or absent CMAP, would predict an incomplete or delayed functional recovery, as evaluated by the same clinical grading system we employed (11).

Abnormal fibrillation potentials and positive sharp waves were seen in both groups. More severely diseased patients did not necessarily show increased abnormal spontaneous activity on needle EMG. Some authors claim that the presence of spontaneous activity is a sign of poor prognosis (12). However, our findings indicate that spontaneous activity in the form of fibrillation potentials and positive sharp waves can also occur in less severe involvement. Lack of voluntary MUP activity is regarded as a sign of poor prognosis, although

there are reports to the contrary (13). Our study confirms that absent voluntary MUP activity is an ominous sign that merits greater emphasis in the interpretation of the results of needle EMG, rather than the presence or absence of fibrillation potentials and positive sharp waves.

In conclusion, we have verified that the House-Brackmann grading system shows a negative correlation with the CMAP index in Bell's palsy. Therefore, if clinical grading is performed in the acute stage of Bell's palsy, the severity of the lesion can be estimated. However, in individual cases, especially with more severe clinical involvement as demonstrated in Table 2, some show severe reductions in their CMAP indices, while others are mildly affected electrophysiologically. This indicates that it may be impossible to distinguish clinically between conduction block and axonal degeneration as the underlying cause of facial weakness. Therefore, nerve conduction studies and needle EMG should still be conducted to accurately define the pathophysiology of Bell's palsy in patients.

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