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

BELL PARALİZİSİNDE KLINİK BULGULARIN BKP İNDEKSİYLE KORELASYONU

Yasemin Biçer GÖMCELİ, M.D., Güznilah KUTLU, M.D., G. Semih KURT*, M.D., Alev LEVENTOĞLU, M.D., Reha KURUOĞLU, M.D.

Gazi University, Faculty of Medicine, Department of Neurology, Ankara-Turkey
Gaziosmanpaşa University, Faculty of Medicine, Department of Neurology*, Tokat, Ankara-Turkey
Gazi Medical Journal 2002; 13: 165-169

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-II. Group 2 consisted of severely affected cases assigned to House-Brackmann grades III-IV. Nerve conduction studies of the zygomatic branches of the facial nerve(s) and affected sides were recorded. A compound muscle action potential (CMAP) index was then calculated by dividing the CMAP amplitude of the affected 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 oris muscle remained unchanged (Spearman r=0.14, p=0.111). Compared to group 1, the CMAP amplitudes (n=470, p=0.000) and indices (n=5.26, p=0.000) were significantly reduced in group 2 patients. Although the presence of fibrillatory 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 common 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 genelindekile elektromyografik incelemeye başvurulur. Bu çalışmadı klinik bulguların ve elektrokimyolojik bulguların korelasyonu araştırılmıştır. Yöntem: Ocak 1999-Şubat 2001 tarihleri arasında klinik Nörofizioloji Laboratuvarını başvuran 149 Bell paralizisi olayı'nın retrospective olarak incelendi. Her olayda House-Brackmann sistemine gore değerlendirilmesi olan yaz Kasınlıkları göz keşif kayıtları doğruluğu. Hastalar klinik değerlendirmelerde 2 gruba ayrıldı. House-Brackmann 1. gruba da değerlendirilen hastalar Grup 1, IV-VI olarak değerlendirilenler ise Grup 2 olarak tanımlandı. Sağın ve paralitik taraflarda yapılan X-raysal radyolojik izole etme çalışmalari alınmıştır. Sonuçlar: Grup 1 ve 2’de CMAP indeksi ele alınmıştır. 100 ile çarpılarak ıdade edilmiştir. M. Frontalis ve orbikularis orbiküleride yapılan içme elektromiyografi (EMG) bulguları da değerlendirildi. Kuzey: House-Brackmann derecesi arttıkça CIMP indeksi düşüklüğüne (Spearman r=−0.62, p<0.000). Birkaç yardımcı ordu kalesinde, orbiküleride belirginlemesi (Spearman r=0.14, p=0.111). Grup 1 ve 2’de CMAP amplitüdleri (n=470, p=0.000) ve indeksleri (n=5.26, p=0.000) anlamlı olarak azalması. Gruplar arasında fibrillasyon potansiyelleri ve pozitif kesik dalgaları bakımından değişiklik göstermeyip (x2=0.03, p=0.86), grup 2’dekiinde motor ünite akışyon potansiyeli (MUP) aktivitesinin azalmamasını hazırlıkla daha sik karşılaştırıldığı (x2=33.0, p=0.000). Yorum: House-Brackmann klinik evreleme sistemini BKP indeksine negatif korelasyon göstermektedir. Bell paralizisinin akut dönemden önceki hatları forgiven olunca, altı amacına benzerlik mevcut olmakta anlamlı olarak EMG yapılmasi gereklidir.

This study was presented in part at the 18th National Clinical Neurophysiology EEG-EMG Congress, May 2001, Belek, Antalya, TURKEY

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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 Neuropysiology 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.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal facial movement</td>
</tr>
<tr>
<td>II</td>
<td>Slight asymmetry of facial movement</td>
</tr>
<tr>
<td>III</td>
<td>Obvious asymmetry of facial movement, some forehead movement present</td>
</tr>
<tr>
<td>IV</td>
<td>Obvious asymmetry of facial movement, forehead movement absent</td>
</tr>
<tr>
<td>V</td>
<td>Only slight facial movement</td>
</tr>
<tr>
<td>VI</td>
<td>Absence of any facial movement or tone</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>House-Brackmann</th>
<th>CMAP Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade</td>
<td>Mildly reduced</td>
</tr>
<tr>
<td>I</td>
<td>28</td>
</tr>
<tr>
<td>II</td>
<td>35</td>
</tr>
<tr>
<td>III</td>
<td>24</td>
</tr>
<tr>
<td>IV</td>
<td>10</td>
</tr>
<tr>
<td>V</td>
<td>3</td>
</tr>
<tr>
<td>VI</td>
<td>0</td>
</tr>
<tr>
<td>Total (n=149)</td>
<td>100</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Group 1 (n=106)</th>
<th>Group 2 (n=35)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>40.5±19.1</td>
<td>43.7±21.0</td>
<td>0.416</td>
</tr>
<tr>
<td>TL (ms)</td>
<td>3.4±0.7/2.9±0.4</td>
<td>3.5±1.0/2.8±0.4</td>
<td>0.539/0.431</td>
</tr>
<tr>
<td>Amp (mV)</td>
<td>1.5±1.0/2.8±1.1</td>
<td>0.8±0.7/2.7±1.0</td>
<td>0.000/0.627</td>
</tr>
<tr>
<td>CMAP Index</td>
<td>54.9±24.5</td>
<td>30.8±20.2</td>
<td>0.000</td>
</tr>
</tbody>
</table>

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 71 (59%) 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.

REFERENCES


