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Original Research Article

Prognostication of Bell's palsy: a new perspective

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ABSTRACT

Background: Bell's palsy is considered as the most frequent cranial neuropathy. Early and adequate risk stratification may help both the patients and the treating physicians in taking informed decisions regarding treatment and understanding their outcomes. We aimed to formulate accessible and sensitive methods of risk stratification in Bell's palsy by utilizing electrophysiological and hematological parameters.
Methods: We prospectively followed up 101 patients with Bell's palsy over a period of 18 months. Electrophysiological parameters were measured thrice i.e., on the first evaluation and after the first week and first month. The N/L, P/L ratio, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were documented in steroid naïve cases. Patients were graded in severity based on the Sunnybrook and House Brackmann systems.
Results: The mean SB and HB grades at admission were 53.89±24.725 and 3.92±1.04 indicatingmoderate severity. The mean N/L, P/L ratios and ESR on the first day was 3.46±3.45, 145.42±162.84 and 22.51±21.105 respectively. There was no statistical correlation with severity at anytimepoint. The mean CMAP indices on the 1st day, 1st week and 1st month were 0.585±0.31,0.43±0.26 and 0.45±0.23 respectively. The CMAP index at 1 month was correlating with severity. Blink amplitude ratios were correlating with the HB scores at 1 week and 1 month ($p<0.0001$ both) and the SB score at 1 month ($p<0.0001$).
Conclusions: Hematological parameters were not correlated to disease severity. However, electrophysiological parameters are correlated to disease severity at one week and one month. Blink amplitude ratio may be a useful indicator for risk stratification of Bell's palsy patients.

Keywords: P/L ratio, N/L ratio, CMAP index, Blink reflex amplitudes

INTRODUCTION

"The facial expressions of human beings fascinate me because they convey both the lowest, most bestial pleasures and the strongest and gentlest emotions of the spirit", with these words, Sir Charles Bell described the facial paralysis for which he became well known for.[1]

Bell's palsy is the commonest cause of 7th cranial nerve paralysis. Almost 85% of Bell's palsy spontaneously recovers normal facial function in 3 weeks.[2] In spite of this fact, Bell's palsy causes considerable distress because of the associated deformity. Most people relate facial deviation with a stroke. It is imperative for neurologists to be able to inform the patient regarding diagnosis, recovery and delineate those who will recover partially. Early and accurate risk stratification tools may help both physicians and patients understand the path to recovery.

Nerve conduction tests are available in most centers and are relatively cheap. Parameters including the compound muscle action potential (CMAP) latency, amplitude, CMAP index and the blink reflex have been used to predict recovery of the facial nerve.[3]

The neutrophil to lymphocyte (N/L) ratio, platelet to lymphocyte (P/L) ratio and C-reactive protein (CRP) have recently come into use as markers of sub clinical

inflammation.[4] Bell's palsy being a disease with inflammation playing a significant role, we considered the use of inflammatory markers in risk stratification. Scoring methods like the House Brackmann (HB) and Sunnybrook (SB) scales which have been utilized to assess severity were correlated to the above variables at various stages of disease.[5] Being minimally invasive and accessible, the above tests are usually well accepted among patients.

The primary aim of our study was to formulate cost effective, sensitive and reproducible methods in the risk stratification of Bell's palsy. We assessed whether nerve conduction studies with special reference to the CMAP values, CMAP index and blink reflex were useful in identifying patients who recover and do not recover from Bell's palsy. Haematological parameters including the total and platelet counts, N/L ratio, P/L ratio, CRP and ESR were also included in the analysis.

Aim

The aims of the study were: to assess the clinical profile of idiopathic Bell's palsy in a subset of South Indian population; and to formulate cost effective, sensitive and reproducible methods in the prognostication of Bell's palsy.

Objectives

Primary

Primary objectives of the study were: to assess the role of nerve conduction studies with special reference to the CMAP values, CMAP index and blink reflex parameters in prognostication of Bell's palsy; and to assess whether the neutrophil to lymphocyte ratio, platelet to lymphocyte ratio, CRP would be effective markers of the degree of damage and inflammation of the facial nerve and prediction of recovery.

Secondary

Secondary objectives of the study were: to find out risk factors if any and their implications in Bell's palsy; and to find the correlation of clinical signs and severity of the disease with the above-mentioned parameters.

METHODS

A total of 101 consecutive patients of Bell's palsy encountered in Jubilee Mission Medical College, Thrissur were assessed.

Study design, population, and sampling

We conducted a prospective study in the outpatient clinics and wards of Jubilee Mission Medical College, Thrissur over a period of 18 months from February 2017 to August 2018. All consecutive patients with idiopathic Bell's palsy were included in the study. Total enumeration method was used for sampling. Sample size calculated was 40.

Inclusion criteria

Patients who conformed to the diagnostic criteria of Bell's palsy were included.[6]

Exclusion criteria

Patients with secondary causes of LMN 7th nerve palsy-tumours, brainstem stroke, sarcoidosis, GBS, autoimmune disorders, otogenic causes, trauma; sequelae of old Bell's palsy; and previous history of Bell's palsy on the same side were excluded.

Data collection

Demographic details, history and risk factors were recorded. Detailed clinical examination was performed. Cases were defined based on the diagnostic criteria for Bell's palsyi.e., acute or subacute onset; impaired eyelid closure, disappearance of forehead wrinkles, lost frown; nasolabial groove becomes shallow or even, angle of mouth is low and veers toward the healthy side; and absent or decreased sense of taste in the anterior 2/3rds of the tongue, auditory handicap, pain in the mastoid region, sensory disability in the external auditory meatus or auricle.[6]

Secondary causes of seventh nerve palsy were ruled out by adequate means. Patients were graded in severity on the basis of the Sunnybrook and House Brackmann scores. The SB score ranges from 0 to 100; 100 indicating complete recovery. The HB grades range from 1 to 6,with higher grades indicating higher severity.

Electrophysiological parameters

Octopus clarity machine was used for evaluation of the cases. Facial nerve conduction studies were carried out on three occasions: on the day the patient was first evaluated, after one week, and after the first month of ictus.

CMAPs and blink reflex parameters were recorded. For the CMAP examination, stimulation was started at 15 m Amp and then increased till maximum increment in CMAP amplitude was obtained. We applied supramaximal single pulse of duration 100 µs over the trunk of the facial nerve, using the bipolar stimulating electrode with the anode between the ramus mandibulae and the mastoid and the cathode in front of the tragus of the ear. Low and high frequency filters were kept at 2 Hz and 5 kHz respectively. The normal CMAP is above 1 mV.[7]

Compound muscle action potentials, velocities and latencies were measured from the orbicularis oculi bilaterally using disc electrodes. The CMAP index was calculated as the ratio of the amplitude of CMAP on the

affected side to that of the healthy side and expressed as a percentage.[8]

The facial nerve degeneration was assessed as follows.

$$Facial\ nerve\ degeneration = [l - n] \times 100\%$$

Where $n = \frac{\text{Affected facial nerve CMAP amplitude}}{\text{Normal facial nerve CMAP amplitude}}$

The blink reflex was also assessed. The supraorbital nerve was stimulated at the supraorbital foramen with an impulse of 15-20 mA of 100 ms duration. The signals were amplified, band-pass filtered (band-width 20 Hz-10 kHz) and the evoked response from both orbicularis oculi muscles recorded. On the side of stimulation two responses R1 and R2 and on the contralateral R2 latencies were noted: R1 being the early phasic and R2 the late tonic component. Minimal latency was assessed from among 5 superimposed responses. Subsequently, we repeated the recording on the opposite side.

Response amplitudes were also documented. Blink amplitude ratios comparing the affected with the normal side were documented as the Ra/Rn ratio. The ipsilateral R1, R2 from the side of palsy and contralateral R2 recorded from the normal side were considered in the estimation of the abnormal latencies in Bell's palsy based on the normal values given below.[9]

Normal values considered were as follows: ipsilateral R1 latency ≤13 msec, ipsilateral R2 latency ≤41 msec, and contralateral R2 latency ≤44 msec.

Relevant blood tests were done. The N/L ratio and P/L ratio were calculated from the hemogram. Erythrocyte sedimentation rate (ESR), CRP and other relevant results were documented in steroid naive patients. The tests were documented only on the first day.

Plan of analysis

Data was analysed using standard techniques by the IBM statistical package for the social sciences (SPSS) 20 software. Numerical variables were expressed as means and standard deviations and categorical variables as frequencies. The correlation of study variables was obtained by the Pearson's correlation coefficient for parametric and Spearman's rank correlation for non-parametric variables. Kruskal Wallis test was applied for comparing the means of the study variables with the severitygrades.

RESULTS

A total of 101 consecutive cases of Bell's palsy were evaluated. 53 females and 48 males were evaluated. There was an almost equal gender distribution with females being 52.47% and males being 47.5% of the population. Age among patients in this study ranged from 15 to 85, with a mean of 43.79 (standard deviation 17.04). Majority of the patients (41.6%) belonged to the 41 to 60 age group (Figure 1) 34 (33.7%) cases had DM. 22 (21.7%) cases had hypertension. There were 3 cases of peripartum Bell's palsy. All 3 achieved complete recovery by 1 month.

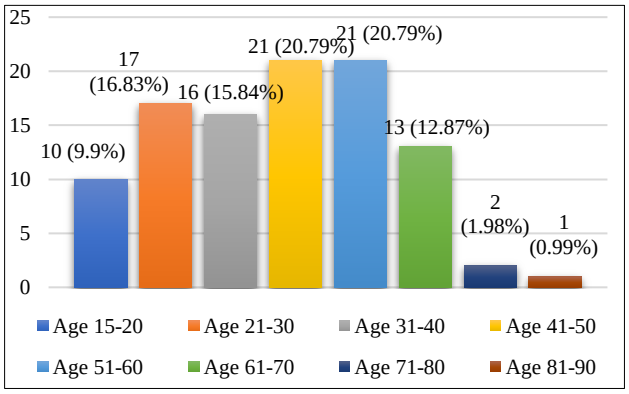

Figure 1: Age distribution of cases (n=101).

The Sunnybrook and House Brackmann scales were used in our study to quantify the severity of Bell's palsy. The mean SB score and HB grade at admission was 53.89±24.725 and 3.92±1.04 respectively which indicates a moderate degree of severity at onset. The HB grades and SB scores were as shown (Table 1).

Table 1: House Brackmann grades and Sunnybrook scores at follow up.

Grades/scores	Day 1	1st week follow up	1st month follow up
House Brackmann grade			
1	1	3	26
2	9	12	15
3	23	17	13
4	35	18	7
5	30	17	7
6	3	1	0
Sunnybrook score			
0-20	10	0	0
20-40	23	17	1
40-60	22	19	7
60-80	27	19	17
80-100	19	13	43
Total	101	68	68

A total of 14 (13.86%) patients had moderate severity with the HB grade being 4-5. Only one case had very severe residual palsy at follow up i.e., a HB grade of 6. Both the HB and SB scales are subjective scales and can be subject to interobserver bias which was minimized in our study as all cases were evaluated by a single person. 26 cases had improved fully by the first month of follow up. We lost 33 cases to follow up at the first month. Whether this occurred due to complete improvement or some other cause could not be ascertained.

Hemogram and inflammatory markers in determining outcome

The hemogram and other inflammatory markers were checked only in those who were not initiated on steroids in order to avoid confounding the results i.e., in 91 patients.

A total of 76 out of 91 evaluated (82.6%) had a normal total count. Only 16 cases had elevated counts out of which 4 had reports in the range of 16,000 to 20,000/microlitre. Similar to the total counts, most had normal platelet counts as well. Thrombocytopenia was not documented in any of our cases.

Similar to the total counts, most of the patients had normal platelet counts as well. Only two patients had elevated platelet counts more than 4,50,000. Thrombocytopenia was not documented in any of our cases.

The neutrophil to lymphocyte and platelet to lymphocyte ratios

The N/L and P/L ratios have come up as novel markers of inflammation. They have been evaluated in the field of oncology, myocardial infarction etc. and have been found to be significant in several studies.[11,12]

The normal N/L ratio which has been determined from various studies is 1.65±1.96 (14). In our study the mean N/L ratio on the first day was 3.46±3.45. Maximum value obtained was 23.25 (Figure 2). The normal P/L ratio is 137± 102. The mean P/L ratio obtained from our study was 145.42±162.84. 71/91 (78%) cases had normal P/L ratios. A few patients with severe disease had elevated P/L ratios. Maximum value obtained from our study was 1150 (Figure 3). Mean ESR on day of admission was 22.51±21.105 with 51% having normal values.

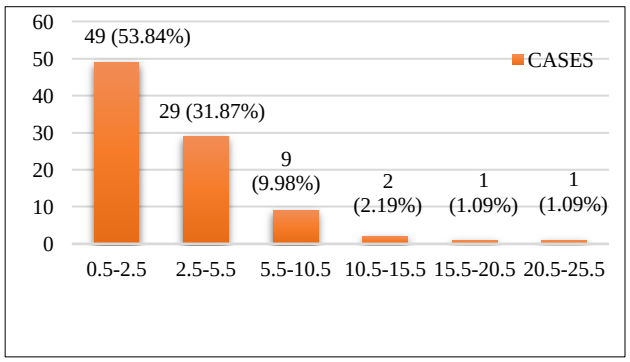

Figure 2: Range of N/L ratios.

CRP although considered an excellent marker of inflammation in several studies proved not useful in our study. 73 cases in our series had a normal CRP i.e. 80.22%. 18 cases who had an elevated CRP though, had a worse outcome compared to others (out of this 10 were diabetic). The above patients probably need evaluation for some secondary cause of CRP elevation.

The distribution of N/L, P/L and ESR was the same across all categories of the Sunnybrook scores and House Brackmann grades according to the independent samples Kruskal Wallis test (p values 0.922, 0.959 and 0.768 respectively). We were not able to find any statistical correlation among these parameters with the Sunnybrook or House Brackmann scales at any time period (Tables 2-4).

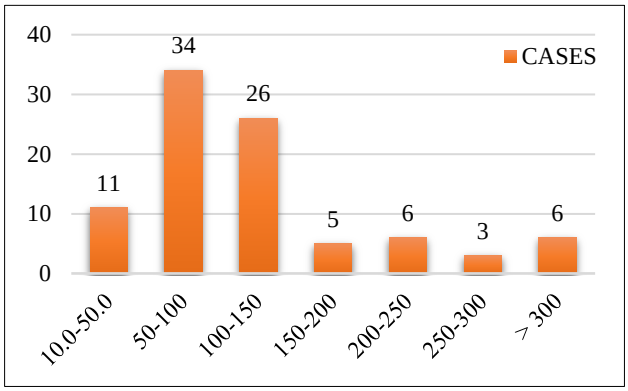

Figure 3: Range of P/L ratios.

Table 2: Correlation between N/L ratio and the Sunnybrook scores and House Brackmann grades.

Grades/scores	N/L ratio	
	Correlation coefficient (r)	P value
SB score		
1st day	0.147	0.163
1st week	0.039	0.762
1st month	0.065	0.612
HB grade		
1st day	0.186	0.077
1st week	0.079	0.539
1st month	0.047	0.715

Table 3: Correlation between P/L ratio and the Sunnybrook scores and House Brackmann grades.

Grades/scores	P/L ratio	
	Correlation coefficient (r)	P value
SB score		
1st day	0.147	0.164
1st week	0.032	0.804
1st month	0.065	0.612
HB grade		
1st day	0.018	0.867
1st week	0.050	0.695
1st month	0.009	0.943

The N/L, P/L ratios and ESR were also compared with the CMAP indices at various stages of follow up. However, there was no statistical significance (Table 5).

Table 4: Correlation between ESR and the Sunnybrook scores and House Brackmann grades.

Grades/scores	ESR	
	Correlation coefficient (r)	P value
SB score		
1st day	0.123	0.255
1st week	0.017	0.896
1st month	0.095	0.468
HB grade		
1st day	0.161	0.134
1st week	0.103	0.428
1st month	0.077	0.552

Table 5: Correlation between N/L ratio, P/L ratio, ESR and CMAP indices.

Variables	N/L ratio	
	Correlation coefficient (r)	P value
CMAP indices		
1st day	0.008	0.941
1st week	0.047	0.706
1st month	0.058	0.649
P/L ratio		
1st day	0.033	0.753
1st week	0.121	0.334
1st month	0.077	0.545
ESR		
1st day	0.096	0.375
1st week	0.176	0.167
1st month	0.059	0.650

Patients with higher P/L ratios and ESR were found to have more severe disease. The ultimate finding was that the inflammatory markers were not good prognostic indicators of the outcome of Bell's palsy and therefore not necessary during initial evaluation of the same.

Electrophysiological parameters

The mean CMAP on the normal side on the first day was 2.452±0.883. The mean CMAP of the abnormal side was 1.354±0.763. Thus, the CMAP values on the initial days of disease remain normal.

The mean CMAP index on the first day of evaluation was 0.585±0.31, indicating a degeneration of 10-73%. The mean CMAP index at 1 week and 1 month were 0.43±0.26 and 0.45±0.23 respectively (Figure 4). This shows varying levels of degeneration in different time frames and probably depends on the day of first evaluation after symptom onset.

A total of 10 cases did not show any degeneration on the first day of evaluation. 86.1% cases (87/101) had CMAP indices between 0.1 and 1.0. Severe degeneration on the first day itself was noted in 4 patients. Majority of cases, both on 1st week and 1st month follow up had CMAP indices in the range of 0.1 to 1.0 i.e., 60.39% and 92.6% respectively.

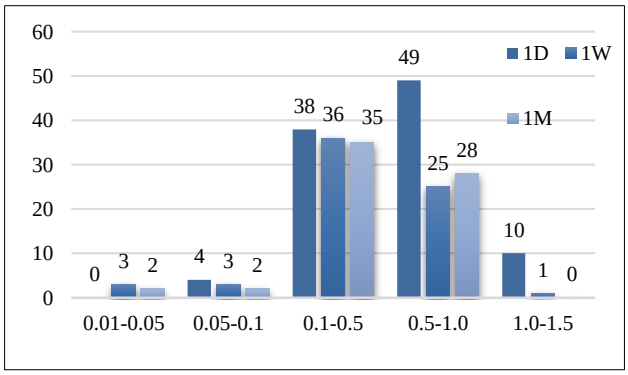

Figure 4: Range of CMAP indices on follow up.

The majority of cases, both on 1st week and 1st month follow up had CMAP indices in the range of 0.1 to 1.0 i.e. 60.39% and 92.6% respectively (Figure 4).

The number of cases without degeneration was only one at the first week as compared to ten on the first day. 6cases showed significant degeneration at the first week and 4 at the end of the first month.

CMAP index showed a negative correlation with lower values showing a greater degree of severity in relation to both scales. CMAP index at 1 month was correlating with the HB grade as well as the Sunnybrook score at 1 month using the Spearman's Rho non parametric correlation test (0.394, 0.409 respectively, p value <0.001). The CMAP index on the first day and first week was not showing any correlation with the scores at any point of time. The was no correlation between CMAP index and the HB score at the first week: 0.337 (p value=0.05) or the SB grade: 0.139 (p value=0.262).

Blink reflex assessment showed mean ipsilateral R1 latencies on the 1st day, 1st week and 1st month as 13.358±4.18, 12.87±2.99 and 12.656±2.57 respectively. The mean ipsilateral R2latencies on the 1st day, 1st week and 1st month were 40.05±7.68, 41.59±7.75 and 38.57±6.11. The mean contralateral R2 latencies on the 1st day, 1st week and 1st month were 33.485±6.39, 34.767±6.43 and 35.314±6.29. These values were not very deviant from the normative values.

Blink reflex latencies were not showing any significant relation to outcome. Significance was shown by the ipsilateral R1 latency and the SB score at the 1st month (0.359, p<0.003) and the contralateral R2 latency with the SB score at the 1st week (0.349, p<0.004).

Blink amplitude ratios were found to have definite significance indicating axonal degeneration, reflecting the CMAP indices. On the first day 38 (37.62%) cases had

ratios between 0.1 and 0.4. By the first week and month the ratios shifted towards worsening degeneration (Figure 5).

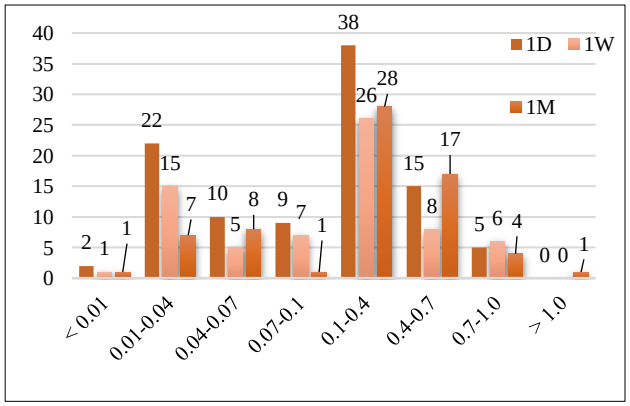

Figure 5: Range of Ra/Rn ratios on follow up.

The ratios at the first week and first month was correlating with the HB scores at 1 week as well as 1 month, using Spearman's Rho test for correlation (0.491, 0.579, p value <0.0001 both). They also correlated with the SB score at 1 month (0.477, p value <0.0001). There was no correlation with the SB score at 1 week (0.276, p value=0.026).

A total of 40.6% patients had improved clinically at the end of one month although the CMAPs and blink amplitudes remained abnormal.

Patients with severe Bell's palsy had elevated N/L and P/L ratios, CRP and ESR; however, they did not have a statistically significant correlation to outcome. The CMAP Index and Ra/Rn ratios estimated after the first week were better outcome predictors compared to others.

In conclusion nerve conduction studies should be done after the first week of ictus and after one month if the patient fails to recover.

DISCUSSION

A total of 101 consecutive cases of idiopathic Bell's palsy were evaluated. The mean SB score and HB scores at admission indicated a moderate degree of severity in most cases. This largely depended on the day disease evaluation was started from our side.

In De Seta's study, at the first examination, the moderate to severe palsies (HB grades IV–VI) represented 54.3% of patients.[10] At ten days from onset, they represented 39.9%, while a complete recovery was observed in 10.2%. Marsk et al evaluated the SB score and showed that at 1 month it accurately predicted non recovery at 12 months.[11]

Going on to the haematological parameters, most of our patients had normal total and platelet counts. The mean N/L and P/L ratios calculated on the first day were not very deviant from normative values. A few patients with severe disease had elevated P/L ratios.

Bucak et al in their study of 54 patients, found that the mean N/L ratio was 2.69±1.48, which was significantly higher than controls. N/L ratios were seen to increase in disease.[12] There were no statistically significant changes of the P/L ratios. They also did not detect any association between the N/L ratio and HB grade.

In the study by Atan et al the mean N/L ratio of 99 patients was 4.37±3.87 and that of controls was 1.89±0.72.[13] The mean P/L ratio was 137.5±81.04 and 113.75±35.83 respectively which also showed a statistical significance. No statistically significant relation was detected between the degree of facial paralysis and N/L and P/L ratios.

Ulusoy et al investigated the relationship in 24 patients.[14] The mean N/L ratio was 2.86±2.01 in the patient group and 1.68±0.34 in the control group, and N/L ratio was found to be significantly higher in the patient group compared with the control group (p=0.016). The P/L ratio was 121.90±52.87 in the patient group and 99.98±16.93 in the control group, but the difference was not statistically significant (p>0.05).

Our study also demonstrated an increase of these parameters in disease. However, the distribution of N/L, P/L ratios and ESR was the same across all categories of the HB grades. This indicated non-utility of the above parameters in prognosticating outcome.

In a letter to the editor of the Balkan medical journal as a pointer to the study by Atan et al and Ümit stated that the changes observed might be attributable to the effects of treatment (glucocorticoids) rather than the disease itself.[15] In our study we evaluated all the haematological parameters and inflammatory markers only in those who had not received steroids prior to inclusion, in contrast to other studies. This would probably be the reason why we did not obtain any significant correlation.

Most patients had a normal CRP (73/91). CRP although considered an excellent marker of inflammation was proven ineffective.[16] 19 cases with elevated CRP though, had a bad outcome. Out of these 10 were diabetics. Majority of our patients had normal ESR and correlation of ESR with various outcomes was not statistically significant.

The electrophysiological parameters are valuable in determining prognosis in the subacute phase because the severity of neuropraxia and axonal damage due to Wallerian degeneration can be quantified. In the subacute phase CMAP amplitude differences between sides ≥75% indicate a poor prognosis.[17,18]

The mean CMAP on the initial days of disease remained almost normal. Our study showed varying levels of degeneration and probably depended on the day of first

evaluation. The majority of cases both in the 1st week and 1st month in our study had CMAP indices in the range of 0.1 and 1.0. The number of cases without degeneration was only one as opposed to ten on the first day. Six cases showed significant degeneration in the first week and four at the end of the first month.

Djordjević et al demonstrated that progressive decrease of the CMAP amplitude was registered from the seventh to the fourteenth day of the illness. The more the amplitude decreased, the slower the recovery was. Most of the patients in whom the amplitude values were 81 to 100% during the above period, recovered completely during the first two months, which suggested mild damage of the nerve (neuropraxia). Patients whose amplitude values were between 41 to 80% recovered in 3 to 4 months. The amplitude values from 11% to 40% indicated a slightly longer recovery, by 5 to 6 months, which corresponded to the second typeof the nerve damage (axonotmesis). The amplitude values from 0 to 10% pointed to heavy nerve damage (neurotmesis) and an incomplete clinical recovery.[19]

Ozgur et al in a study in 65 patients evaluated electrophysiological outcomes and showed that 58 patients having good prognosis at the third month had a CMAP difference of less than 75% in the frontalis, orbicularis oculi and orbicularis oris muscles during the first week of illness. They concluded that any CMAP amplitude difference between sides >75% in the subacute phase indicated a poor prognosis.[20] Chow et al reported that ≥73% difference in CMAP amplitudes indicated a poor prognosis at six months. Amplitude differences of >90% were reported to indicate a poor prognosis when evaluated in the third week of the onset of paralysis.[21]

CMAP index at 1 month was correlating with the HB grade as well as the Sunnybrook score at 1 month in our study. CMAP index was found to be a good outcome measure but only if done after 2 weeks.[22]

In a study by Prakash et al there was strong positive correlation between the severity of facial nerve degeneration as indicated by the CMAP index and the clinical outcome of Bell's palsy at 1 month and 2 months after onset. The correlation coefficients were 0.794 ($p<0.0005$) and 0.732 ($p<0.0005$), respectively. However, there was no statistically significant correlation between the facial paralysis grading at the first visit and the severity of facial nerve regeneration ($p=0.068$).[23]

Blink reflex reflects the conduction through the intracranial segment of the facial nerve and therefore it can be an important prognostic indicator.[24] It is especially useful during early stages of facial nerve regeneration, where the persistence or early return of an initially absent ipsilateral R1 may suggest a satisfactory recovery of facial nerve function. In turn, if this response is absent during the first 2 weeks after onset, an unfavourable prognosis is given.[25] Various studies have described the prolongation of blink reflex latencies as most effective in prognosticating Bell's palsy. However, we had to take a slightly different view after the completion of our study.[26]

We noted that the blink reflex latencies were not very deviant from normative values. Minimal correlation if any was shown by the ipsilateral R1 latency prolongation and the Sunnybrook score at 1 month ($p=0.003$) and the contralateral R2 latency with the Sunnybrook score at the 1st week ($p=0.004$). Therefore, blink reflex latencies proved to be an inconclusive outcome measure.

In a study by Nacimiento et al in 50 patients with Bell's palsy, the ipsilateral R1 and R2 responses on stimulation of the normal side and the contralateral R2 response on stimulation of the paretic side showed no significant difference compared to controls except for a slight delay of the ipsilateral R2 latency ($p<0.05$).[27] The results of Djordjevic's study showed a change in R1 response during the first few days of the illness, either as extended latency or a complete lack of R1 response, which points to intratemporal conduction block; a primary process in Bell's palsy.[19]

Ozgur et al analysed electrophysiological results and compared them with the House–Brackmann scale in 65 patients on the 5th, 20th, and 90th days. In the study, when R1 and R2 responses could not be obtained in the first 3 weeks, poor prognoses were predicted on the 90th day.[20] Hence, the absence of R1 and R2 responses in blink reflex studies within the first 3 weeks was associated with a poor prognosis.

Kimura et al noted that there was a progressive loss of the ipsilateral R1 response in the first week after symptom onset; thereafter, the reflex returned in a majority of patients.[18] The presence of the R1 after the first week was suggested to be a good prognostic sign. Therefore, if blink reflex responses could not be obtained in the first week, clinical recovery was prolonged and generally incomplete.[28]

The Ra/Rn ratios were indirect reflectors of axonal degeneration reflecting the CMAP indices and were found to have significance in our study. The ratios at the first week and first month follow up of the affected side was correlating with the HB scores at 1 week as well as 1 month and the Sunny brook score at 1 month.

In other words, in predicting prognosis, evaluation of CMAP in the subacute phase and blink reflex studies in the acute and subacute phase provides statistically significant results. Blink reflex studies were most useful, showing better sensitivity and specificity than other parameters.[29] We would recommend doing the facial nerve conduction studies and blink reflexes after ten days of ictus. Repeat may be done after one month if the patient fails to recover by that time.

The limitations of the study were that patients were followed up only till the first month. Parameters would probably give a better outcome measure if followed up for a longer period of time. The HB and SB scales are subjective scales and can be subject to interobserver bias which was minimized in our study as all cases were evaluated by a single person.

CONCLUSION

Patients with severe Bell's palsy had elevated neutrophil and platelet to lymphocyte ratios; however, there was no statistically significant correlation to outcome. CRP and ESR were not effective prognostic measures and not needed unless suspecting an underlying disease process. Our study showed that the CMAP index and the Ra/Rn ratios estimated from the blink reflex amplitudes after the first week were the better outcome predictors. The electrophysiological parameters obtained after the first week would be the most effective prognostic indicators. In conclusion nerve conduction studies should be done after the first week of ictus and after 1 month if the patient fails to recover. If applied together, clinical and electrophysiological examinations have great value in the assessment of Bell's palsy. This is very important for the patient in a psychological sense, but also primarily for the purpose of deciding upon the proper form of treatment. Prognoses can be made and excessive tests can be eliminated by proper timing and use of electro-physiological testing and other parameters.

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