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CLINICAL AND ELECTROPHYSIOLOGICAL EVALUATION OF BELL'S PALSY CASES IN RESPONSE TO TREATMENT

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ABSTRACT

Background: Bell's palsy is an abrupt, isolated, unilateral, peripheral facial paralysis without detectable cause. It is the most common cause of unilateral facial weakness. **Patients and methods:** The study was carried out at the unit of Neurology in Al-Kadhymia Teaching Hospital, during the period from the 1st of October 2004 – 1st of October 2005. Patients graded clinically according to the House-Brackmann Grading System into (Grades I-VI) and classified according to the electrophysiological data into: group I (demyelinating) and group II (axonal). All Patients received the same course of treatment consisting of steroids (prednisolone) and antiviral (acyclovir). **Results:** The follow up within the first, fourth and sixth weeks showed highly significant improvement regarding group I, while no significant improvement regarding group II. On the other hand, clinical evaluation of those patients showed no response to treatment, a group of factors probably were contributing to the delay of recovery that include: severe post-auricular pain, hyperacusis, loss of taste and delayed administration of treatment. **Conclusion:** Most of Bell's palsy cases are of the Demyelinating type, which has a better prognosis than the axonal type. Hyperacusis, loss of taste, and sever post auricular pain may indicate poor outcome, and early initiation of combined treatment with acyclovir and corticosteroid within three days of onset will improve outcome.

KEYWORDS: Bell's palsy, House-Brackmann, hyperacusis.

INTRODUCTION

Bell's palsy is an abrupt, isolated, unilateral, peripheral facial paralysis without detectable cause. It is the most common cause of unilateral facial weakness.^[1]

The incidence of Bell's palsy is 20-30 cases per 100, 000 people per year. It accounts for 60-75% of all cases of unilateral facial paralysis. [2]

This syndrome of idiopathic facial paralysis was first described more than a century ago by Sir Charles Bell, yet much controversy still surrounds its etiology and management. [3]

Some cases of Bell's palsy have been attributed to ischemia from diabetes and atherosclerosis which helps to explain the increased incidence of Bell's palsy in elderly patients. However, HSV type I (HSV-1) is probably the cause of most cases of Bell's palsy.^[1]

Actual pathophysiology is unknown. A popular theory champion inflammation of the facial nerve, during the

process the nerve increased in diameter and become compressed as it coarses through the temporal bone. [2]

The onset of Bell's palsy is fairly abrupt and maximal weakness being attained by 48 hours and usually there is no further deterioration after the fifth day. Pain behind the ear may precede the paralysis for a day or two and can be quite severe^[1,3]

There is facial muscle paralysis equally affecting the upper and lower parts of the face. [1,4]

Depending upon the site of the lesion, there may be associated impairment of taste, lacrimation or hyperacuesis in the ipsilateral side of the lesion.^[1]

The first step in evaluating any patient who present with facial nerve paralysis involves taking a careful and thorough history. It is important to determine the onset of the paralysis, the duration and the rate of progression.

The Facial Nerve Disorders Committee adopted a universal standard for grading facial nerve recovery The

system involves a six – point scale with I being normal and VI total facial paralysis table (1). [3]

To assist in placing patients in the proper group, an easy method of measuring facial movement has been developed. This system involves making measurement of the movement of the eyebrow and corner of the mouth and comparing the results with those on the unaffected side. A scale with 0.25 cm division is used for the measurements There is a total possible score of 8 (4 or 1 cm for the mouth and 4 or 1 cm for the eyebrow). These results can be easily converted to the six-point scale. Table (1). [6]

Table 1: House Brackmann's Grading System.

Grade	Degree	Description	Measurement
I	Normal	Normal facial movement, no synkinesis	8/8
II	Slight	Mild deformity, mild synkinesis, good forehead function, slight asymmetry	7/8
III	Moderate	Obvious facial weakness, forehead motion present, good eye closure, asymmetry, Bell's phenomenon present	5/8-6/8
IV	Moderately severe	Obvious weakness, increasing synkinesis, no forehead motion	3/8-4/8
V	Severe	Very obvious facial paralysis, some tone present, cannot close eye	1/8-2/8
VI	Total	Complete facial paralysis, absent tone	0/8

Electrophysiological Studies are useful to distinguish between axonal and demyelinating types. In the former, electromyography typically reveals little or no evidence of denervation but there is conduction block or marked slowing of maximal conducting velocity. In the axonal type, electromyography shows that denervation has occurred but maximal nerve conduction velocity is normal or slowed only slightly. [4]

The most important electrophysiological studies of the facial nerve include. [9]

a) Stimulation of the facial nerve

This test consists of applying shocks of increasing intensity and recording the resulting muscle action potentials. The principle of this test is to compare the activation threshold on the damaged and the healthy sides. [9]

A complete section of the nerve result in loss of distal excitability by the end of the first week Hence, a normal distal response at the end of the first week after damage suggests good prognosis.^[9]

b) Electromyography

Electromyographic signs in Bell's palsy are analytically similar to those classically seen in lesions of the peripheral nerves .There are fibrillations and positive sharp waves. [4] The fibrillations sometimes appears from the seventh day of paralysis (generally from the tenth day). [3]

Persisting motor activity and absence of fibrillations have been regarded as predictive of complete recovery while the presence of early fibrillations would reveal poor recovery. [3]

c) The blink reflex

The blink reflex is analogous to the clinically elicited corneal reflex. The afferent limb of the reflex is the trigeminal nerve and the efferent limb is the facial nerve .The electrical stimulation of the trigeminal nerve results in contraction of the orbicularis oculi muscles bilaterally. However, EMG recordings of the blink reflex shows two separate components. [9]

- 1. An early R1 component is evoked only on the side of the stimulation.
- 2. A late R2 component recorded bilaterally.

In the facial paralysis, the analysis of blink reflex is particularly useful to assess mild cases and to follow the prognosis of the disease, in such cases, there is a protracted latency of R1 on the damaged side and reduced amplitude, also R2 has an increased latency and reduced amplitude on the affected side, while R2 remains normal in the contralateral side.

Protection of the eye during sleep and message of the weakened muscles are the measures generally employed in the management of such cases. [1,3]

The administration of prednisolone (40-60 mg/day) during the first week to 10 days after onset has been beneficial in most trials. These medications are thought to decrease the probability of permanent paralysis from swelling of the nerve in the tight facial canal.

It has been found that patients treated by prednisolone and acyclovir (400 mg 5 times daily for 10 days) had a better outcome than patients treated with prednisolone alone. [1,4,7,8]

Approximately 50% of Bell's palsy patients will have essentially complete recoveries in a short time, another 35% will have good recoveries in less than a year, 10%

are bothered by some asymmetry of facial muscles, while 5% experience severe deformities. [2,3,5]

Clinically, Bell's palsy have a bad prognosis in those over 60 years of age, severe initial symptoms and those with complete paralysis.

The electrophysiological data are definitively useful, but they should not considered alone. In facial nerve stimulation, a normal response at the end of the first week after damage suggests a good prognosis and in the electromyography, persisting motor activity and absence of fibrillations indicate good prognosis. [1,3]

AIM OF THE STUDY

To recognize the clinical and electrophysiological factors that affecting the prognosis of Bell's palsy.

PATIENTS AND METHODS

This study was carried out at the Department of Neurology in Al-kadhemia teaching hospital during the period extending from the 1st of October 2004 to the 1st of October 2005.

The cohort of the study included sixty five patients with Bell's palsy, they were examined during the first week of the illness, and they were instructed to come for a second visit (after 4 weeks) and a third visit (after 6 weeks) for follow up after giving treatment (prednisolone 40-60 mg/day orally and acyclovir 400 mg 5 times daily for 10 days orally).

Patient's ages ranged from 15-72 years, 60% were females (39 patients) and 40% were males (26 patients).

We were able to follow 45 patients from the total for second and third visits by clinical and neurophysiological examination.

At presentation, history was taken from all the patients emphasizing on onset, pain association, progression of the facial paralysis, impaired lacrimation and salivary secretion, and taste loss. All patient were examined clinically as mentioned previously and graded according to the House-Brackmann Grading System.

The following electrophysiological studies were done for each patient

- 1. Needle EMG of right and left orbicularis oculi muscles.
- Direct motor nerve conduction study of both right and left facial nerves.

Parameters of EMG include

- Spontaneous activity.
- Motor unit action potential (duration, amplitude, and polyphasia).
- Recruitment or interference pattern.

- Parameters of direct motor nerve conduction study include:
- Distal motor latency.
- Compound muscle action potential amplitude.
- Conduction velocity.

Note: temperature was kept within limits to minimize effect of temperature on the figures of nerve study.

To distinguish the axonal and demyelinating types of Bell's palsy, we depend on the following features:

- 1. In axonal type
- Normal or near normal distal motor latency.
- Normal or near normal conduction velocity.
- Reduced compound muscle action potential amplitude (CMAP).
- EMG shows reduced recruitment pattern in acute stage and signs of denervation (large motor unit and increased polyphasia) in chronic stage.
- 2. In demyelinating type
- Prolonged distal motor latency.
- Conduction block or marked slowing of maximal conduction velocity.
- EMG shows no volitional activity or reduced recruitment pattern in normal motor pattern.

Machine: Micromed System + full option EMG System "Myoquick."

Electrodes: Standard Concentric Needle Electrodes.

Statistical analysis was carried out using Chi Square test.

RESULTS

The total number of patients in our study were 65 patients, Males were 26 (40%), whereas female were 39 (60%), and as shown in figure (1).

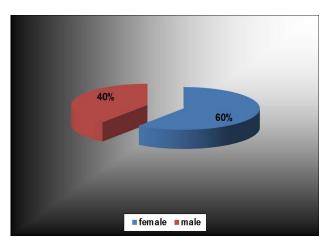


Figure 1: The percentage of patients according to their sex.

The right side was affected in 41 patients (63.1%), while the left side was affected in 24 patients (36.9%), and as shown in figure (2).

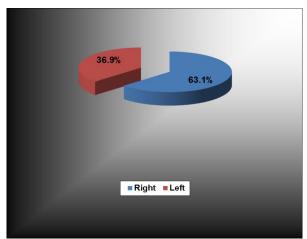


Figure 2: Distribution of the cases according to the side of involvement.

The (65) patients were grouped according to their age, which showed predominantly more within two age groups (10-19) and (30-39) which show (29.20 % and 27.70%) respectively, and as shown in figure (3).

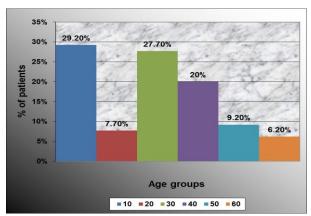


Figure 3: Distribution of cases according to their age.

Clinical evaluation at time of presentation showed that all patients have paralysis of facial muscles(100%) and the majority (about 80%) has postauricular pain, and as shown in figure (4).

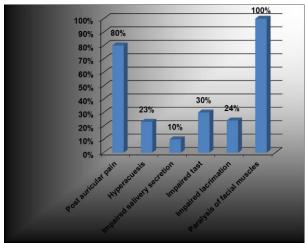


Figure 4: Signs and symptoms.

Fourty five patients classified according to the electrophysiological studies into group I: demyelinating (33 patients 73.3%) and group II: axonal (12 patients 26.7%), and as shown in figure (5).

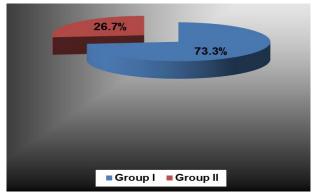


Figure 5: Distribution of patients according to the electrophysiological studies.

Grading of patients in group I according to House-Brackmann Grading System in the 1st visit showed that the majority of patients distributed within grade IV (42.40%), and as shown in figure (6).

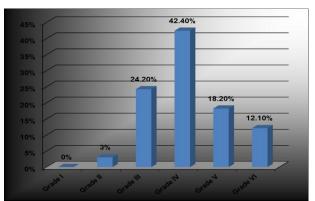


Figure 6: Grading of patients in group I according to House-Brackmann Grading System.

While grading of the patients in group II according to the House-Brackmann grading system show that 50% of the patients lie in grade V, and as shown in figure (7).

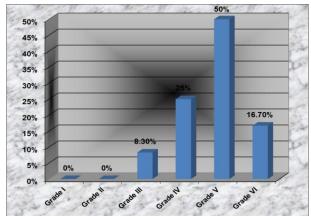


Figure 7: Grading of patients in group II according to House-Brackmann Grading System.

Most of group I cases during the first visit lie within grade IV, while in the last visit most of these cases lie within grade I, and as shown in table (2).

Table 2: Follow up of patients according to House-Brackmann Grading System (Group I).

Viait	Grade						
Visit	I	II	III	IV	V	VI	
1 st visit	0	1	8	14	6	4	
2 nd visit	7	4	6	9	3	4	
3 rd visit	20	3	2	1	3	4	

Most of the cases in group II lie in grade V during the 1^{st} , 2^{nd} and 3^{rd} visit, and as shown in table (3).

Table 3: Follow up of patients according to House-Brackmann Grading System (Group II).

Visit	Grade						
VISIT	I	II	III	IV	V	VI	
1 st visit	0	0	1	3	6	2	
2 nd visit	0	0	3	1	6	2	
3 rd visit	0	1	2	1	6	2	

The most common factors that affect the recovery in group I are: postauricular pain (62.5%), Hyperacusis (53.8%), Delayed treatment (46%), and loss of taste (20%) And as shown in figure (8).

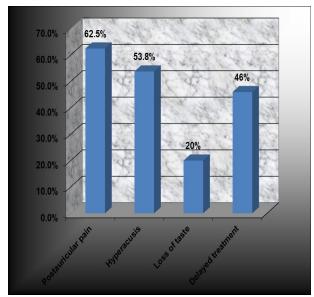


Figure 8: Factors affecting recovery of the patients on group I.

DISCUSSION

In this study clinical evaluation revealed, paralysis of facial expression's muscles (100%), hyperacusis (23%), impaired salivary secretion(10%) and loss of taste in the first two-thirds of the tongue (30%) ipsilateral to the facial nerve lesion, indicating facial nerve lesion distal to the geniculate ganglion within the facial canal. [12,13] About (20%) of patients had only facial muscles paralysis, this consent with facial nerve lesion within the

stylomastoid foramen, [12,14] while impaired lacrimation seen in (24%) of the patients localizing the lesion proximal to the geniculate ganglion. [12,15]

Clinical classification of patients according to House-Brackmann grading system ,showed that grade IV,V (moderate to severe dysfunction) was the most frequent presentation of patients with Bell's palsy, while grade II and VI(mild dysfunction-and total paralysis respectively) were to less extent the features of facial paralysis. These findings were agree with most studies whom Bell's palsy was graded according to this system. [6,10]

In this study patients were divided according to the electrophysiological data into: group I (Demyelinating) and group II (Axonal), and it showed that the majority of patients (73.3%) were demyelinating and the other (26.7%) were axonal. These findings were in harmony with previous study done by Kimura et al, where only 27 patients out of 127 showed features indicating axonal degeneration. [9]

Follow up of patients according to House-Brackmann grading system within the 1st ,4th and 6th weeks showed that for group I (Demyelinating) there was a highly significant improvement and transformation to other grade or grades reaching grade I, while for group II (Axonal) there was no significant improvement in spite of receiving the same treatment. These data was broadly agreed with previous studies done by Axelsson et al and Hato et al. [16,17]

On the other hand thirteen patients from the demyelinating type showed either poor or no response to treatment, and it showed that they share some factors that probably responsible for this poor outcome.

The majority of those patients had at presentation one or more of these features:- hyperacusis (53%), loss of taste (20%), and sever post auricular pain (62%) which had been proved to be a poor prognostic factors in a previous studies. [8,19]

Forty-six percent of those patients found to be received their treatment after more than four days of onset, and this may be responsible for their poor outcome, which agreed with the study of Hato et al, whose found that the recovery rate in patients whose begun the combined treatment within three days of onset of the palsy was (100%), while in those who started their treatment four days or more after onset was (86%).^[17,20]

CONCLUSION

Most of Bell's palsy cases are of the Demyelinating type, which has a better prognosis than the axonal type. Hyperacusis, loss of taste, and sever post auricular pain may indicate poor outcome, and early initiation of combined treatment with acyclovir and corticosteroid within three days of onset will improve outcome.

REFERENCES

- Donald H. Gilden, M. D.: Bell's palsy. N. Eng. J Med, 2004; 1323.
- Micheal Lambert, MD: Bell's Palsy E Medicine, 2005; 1-6.
- 3. Francis B. Quinn Jr., M.D.: facial nerve paralysis. Dept of otolaryngology, UTMB, Grand Rounds, 1996; 3-6.
- Dennis L. Kasper, Stephen L. Hauser, J. Larry Jameson. Bell's palsy. In: Internal medicine, 2005; 2436.
- Gary Jackson, MD, FACS, and Peter G. Von Doersten, MD. The facial nerve. Otolaryngology for internist, 2000; 184-186.
- 6. House J W, Brackmann DE: facial nerve grading system. Otolaryngology- head and neck surgery, 1985; 93(2): 146-148.
- 7. Adams RD, Nctor M, Ropper: principles of neurology. McGraw-Hill, 2005; 1181-1182.
- 8. Roger P. Simon, Michael J. Aminoff, Devid A. Greenberg. Motor difecit In: Clinical Neurology 6th edition, Lange, 2005; 5: 182.
- 9. Kimura, J. Electro diagnosis in disease of nerve and muscle: Principles and practice. F.A Davis Company. Philadelphia. 5th printing, 1986.
- 10. Khalil I, Atta G. Bell's palsy, Clinical and Blink reflex evaluation in response treatment, 2000; 42-5.
- 11. C. David Marsden, Timothy J. Fowler. Cranial nerves syndromes. In: clinical Neurology 2nd edition, Arnold 1998; 8: 165-7.
- 12. Afifi AK, Bergmn RA: Basic Neuroscience. Urban and Schwavzenberg, 1986.
- Banniter SR:Brain's clinical Neurology. London, Oxford. University press, 1985: 63-70.
- 14. Parisi L, Valente., Mariorenz R et al., Bell's palsy. Longitudinal study of 120 cases. Riv. Neurol, 1986; 56(4): 225-35.
- 15. Gbolade BA: Recurrent lower motor neuron facial paralysis in four successive pregnancies. J. Laryngol. Otol, 1994; 108(7): 587-8.
- 16. Axelsson S, Lindberg S, Stjernquist –Desatnik A. Outcome of treatment with valacyclovir and predinson in patients with Bell's palsy. Ann Otol Rhinol Laryngol, 2003; 112(3): 197-201.
- 17. Hato N, Matsumoto S,Kisaki H, et al: Efficacy of early treatment of Bell's palsy with oral acyclovir and predinsolon. Otol Neurotol.2003; 24(6): 948-51.
- 18. Adour KK, Wingerd J. Idiopathic facial paralysis (BELL'S PALSY): Neurology 1974; 24: 1112-6.
- 19. Diamant H, E kstrand T, Wiberg A. Prognosis of idiopathic Bell; s palsy. Arch Otolaryngol, 1972; 95: 431-3.
- Grogan PM, Gronseth GS. Practice parameter: steroids, acyclovir, and surgery for Bell's palsy: report of the Quality Standars Subcommitee of the American Academy Of Neurology. Neurology, 2001; 56: 830-6.