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Bell's palsy: A clinical study of management and outcomes

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Abstract

Facial nerve palsy might be observed for various reasons. The majority of facial paralysis appears as "idiopathic" or "Bell palsy". Of the patients, approximately 80-85% experience spontaneous and complete recovery within the first three months in Bell's palsy. However, it is an accepted fact that these patients should be diagnosed correctly and initiated treatment in the early period. The study aimed to make a retrospective analysis of the treatment modalities and results of patients diagnosed with Bell's Palsy in a tertiary hospital clinic and discuss the subject in the light of current literature.

Keywords: Facial paralysis, bell's palsy, diagnosis, management modalities

Introduction

The vast majority of facial paralysis (FP) is presented in the form of acute idiopathic FP (Bell's palsy) with a relatively good prognosis and is often followed by trauma [1]. Although acute FP, which is mostly seen in adults, is not life-threatening except for its destructive effect on the patient's mood and quality of life, it puts a serious physiological burden on the daily life of the person [2]. Therefore, FP treatment, which includes conventional pharmacological treatment, physical therapy, and surgical options; may require a complicated multidisciplinary approach [3]. Infectious, genetic, vascular, metabolic and autoimmune causes are blamed in the etiology of Bell's Palsy (BP). In recent years, several studies have been reported proving that herpes virus infections play a role in BP etiology [4]. This study aimed to make a retrospective analysis of the treatment modalities and results of patients diagnosed with Bell's palsy in a tertiary clinic and discuss the subject with current literature.

Material and Methods

The study included 108 patients who were admitted to a tertiary otorhinolarngology department with facial paralysis clinic between January 2014 and June 2019 and were diagnosed with BP according to House-Brackmann (HB) scale (Table 1). All patients were examined routinely. Patients with otitis media and central nervous system pathology which were detected by computed tomography (CT) and magnetic resonance imaging (MRI) and with facial paralysis developed due to trauma, were excluded from the study. The files of the patients included in the study were evaluated retrospectively. The patients' age, gender, history of facial paralysis and the side of the paralysis, status of chronic disease (diabetes, high blood pressure) and its accompaniment by pregnancy were taken into consideration and the prognosis of the disease was recorded.

Results

Routine otorhinolaryngologic examination was normal in all patients except facial palsy. Of the 108 BP patients included in our study, 60 (55.5%) were female and 48 (44.5%) were male. The patients were aged between 11 and 75 (mean 46.2 ± 5.28). Peripheral facial paralysis (PFP) was found in 51 patients (47.2%) on the right side and in 57 patients (52.8%) on the left side. The duration of admission to the hospital after the onset of symptoms was determined as 1 day in 65 patients, 2 days in 28 patients, 3 days in 7 patients, 4 days in 4 patients and 5 days in 4 patients. Treatments were started on the day the patients applied to the hospital. When patients were classified according to the House-Brackmann Scoring system in their applications; grade 3 facial paralysis was observed in 52 patients (47%), grade 4 facial paralysis was observed in 32 patients (30%), grade 5 paralysis

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was observed in 16 patients (15%), grade 6 paralysis was observed in 5 patients (5%) and grade 2 paralysis was observed in 3 patients (3%).

Table 1. Hause-Brackmann Scoring system

Stage	Description	Properties
1	Normal	Normal function in all regions
2	Slight loss of function	Slight weakness noticeable in close observation; there may be very mild synkinesia. Normal symmetry and tone at rest, Movement Forehead: Medium good function Eye: Complete closure with minimal effort Mouth: Mild asymmetry
3	Moderate loss of function	Significant but not deformed difference between the two sides, can be seen but not deformed, ckinesia, contracture or hemifacial spasm, normal symmetry and tonus at rest, Movement Forehead: light to medium movement Eye: close with effort Mouth: slight weakness with maximum effort
4	Moderate loss of function	Asymmetry that is distinctive and disfigured between the two sides. Normal symmetry and tone at rest Movement Forehead: none Eye: partial closure Mouth: asymmetry with maximum effort
5	Heavy loss of function Only detectable motion with very strain	Symmetrical at rest Movement Forehead: none Eye: partial closure Mouth: gentle movement
6	Full paralysis	Full paralysis

In 24 (22.2%) of the patients who only had diabetes mellitus (DM), 12 (11.11%) had hypertension (HT), and 16 (14.81%) had both DM and HT as comorbid diseases. Nine patients who had a history of BP were also pregnant (8.33%). Of them, six had BP attacks for the second time and three for the third time. Four patients with recurrent BP had HT and three had both HT and DM as comorbid diseases.

Antiviral agent (valaciclovir 3x1000mg, orally for seven days), proton pump inhibitor (PPI) (pantoprazole 40 mg IV, during steroid therapy) and intravenous (IV) steroid (by decreasing 15 mg every three days) (methylprednisolone 1 mg/kg/day) were used in the treatment.

The pregnant patients were not administered antiviral treatment. The patients were hospitalized for possible complications related to steroids and their daily biochemistry and hemogram values were tracked.

21 of 108 patients (19.44%) made partial improvement of motor functions at the end of the first month (13 patients had HB II, five HB III, two HB V and one HB VI), six patients had hemifacial spasm (5.55%) and 81 patients (75%) recovered fully. Surgical decompression was recommended to five patients with HB V and HB VI who had an over 90% drop in amplitude on electroneurography performed on the 14th day of onset. Surgical decompression was performed in two patients with HB VI who accepted the operation. The surgery was performed in one patient 32 days after the onset of paralysis and in the other patient 40 days after the onset of paralysis. There was no facial dehiscence and the facial nerve was completely freed between the geniculate ganglion and the stylomastoid foramen by the trans-mastoid approach in two patients. Following this, the decompression was performed by opening the fallopian canal. There were edema and congestion in the nerves of two patients. The graft taken from the temporal muscle fascia was laid on the nerve that was freed.

One of the patients was found to have HB III and the other HB IV at the end of the first postoperative month. The first patient had HT as a comorbid disease. CT and MRI were performed on all patients included in the study. Two patients who were found to have cerebellopontine corner tumor were excluded from the study. CT revealed that 26 (24.07%) patients had facial canal dehiscence on the paralyzed side and five (4.62%) patients had bilateral facial canal dehiscence.

Discussion

The facial nerve anatomy and function were first described by Sir Charles Bell in the 1800s. The facial nerve is a mixed nerve containing motor fibers innervating the facial muscles, parasympathetic fibers leading to the lacrimal, submandibular and sublingual salivary glands, afferent fibers for tasting sense of the tongue 2/3 anterior part, the outer ear canal and somatic afferent fibers that take the sense of touch [5].

Facial nerve palsy (FNP) may be caused by a variety of reasons. The controversy regarding FNP continues and genetic factors, vascular ischemia, inflammation due to viral infection, autoimmune diseases, temporal bone fractures, head and neck tumors and central nervous system lesions are among the factors that lead to this disease. Approximately two-thirds of the reasons that cause FNPs remain unidentified and are called “idiopathic” [6,7].

Idiopathic facial paralysis or Bell’s palsy (BP) is the most common type of peripheral facial paralysis. Typically, it is described as self-limiting peripheral lower motor neuron paralysis with acute onset, unknown cause and affecting all muscle groups on only one side of the face. The most common symptom is facial motor dysfunction, which may range from mild paresis to complete paralysis, depending on the amount of neural damage. Clinical findings generally vary according to the localization of the lesion in the facial nerve [8].

Valença et al. found DM to be 11.1% and HT 11.7% and Yanagihara et al. DM to be 11.2% and HT 23% in BP patients [9,10] in their studies on BP, DM and HT coexistence. Also, steroids should be used with caution in terms of possible complications in patients with DM and HT. Because in DM patients, close follow-up and sometimes hospitalization is required in terms of blood sugar regulation. BP is observed 3.3 times more in pregnancies and is frequently observed in the 3rd trimester or early postpartum period. The main reason for this increase in pregnancy is thought to be hormonal changes [10]. In this study, 24 (22.2%) patients

had only diabetes mellitus (DM), 12 (11.11%) hypertension (HT) and 16 both DM and HT (14.81%) and nine patients were pregnant (8.33%). In a study of pregnant women, of 242,000 deliveries, 0.17% of expectant mothers were diagnosed with Bell's palsy [11].

The facial canal is shaped by enchondral ossification of the otic capsule in fetal life. The congenital fallopian duct dehiscence is the developmental defect of the duct surrounding the facial nerve [12]. In anatomical and clinical studies, 30-65.7% dehiscence was reported in the facial canal [13]. A study by Demirci et al. found the rate of facial canal dehiscence to be 55.6% in 45 patients with Bell palsy [14]. Facial canal opening makes the nerve more prone to inflammatory events. However, more studies are needed to assess the relationship between facial canal dehiscence and Bell paralysis. In the present study, 26 (24.07%) patients had facial canal dehiscence on the side of paralysis and five (4.62%) had bilateral facial canal dehiscence.

Corticosteroids, the most commonly used drug in the treatment of facial paralysis, were reported to reduce post-traumatic capillary permeability, edema around the nerve and compression on the nerve, axonal degeneration; increase axonal regeneration, inhibit lipid peroxidation and suppress capillary dilation, fibrin accumulation, cell migration, phagocytosis and is suggested to prevent the development of fibrosis [15].

In a study investigating the effectiveness of corticosteroids (CS) and antivirals in the treatment of Bell's palsy; a group of patients with grade IV-V paralysis in HBS were treated with valaciclovir 1000mg and with prednisolone (60mg for the first three days, then the dose was reduced) for five days and the other group with prednisolone placebo. Then the recovery rates were compared according to the initial severity of facial paralysis (mild, severe and complete). Complete recovery without sequelae was observed in both treatment protocols for mild paralysis, while for patients with severe or complete paralysis, the recovery rates were reported as 86.6% in the prednisolone / placebo group and 95.7% in the prednisolone / valaciclovir group, respectively. These two studies argued that when the CSs are used together with antiviral agents, the improvement in facial functions significantly increases compared to them being used alone [16,17]. A systematic review also found that treatment with prednisolone to reduce the chances of incomplete recovery but the addition of an antiviral drug to be more beneficial [18]. There are several studies on antiviral drugs with or without prednisolone. A randomized prospective study found that a combination of an antiviral and a steroid was more effective in treating severe to complete Bell's palsy than steroid alone [19]. A guideline development group found that there was low-quality evidence of benefit from adding antivirals. Patients who are offered antivirals along with corticosteroids should be counselled that the rate of increase in recovery is less than 7% [20]. A Cochrane review in 2015 found that antivirals combined with corticosteroids improved rates of incomplete recovery compared with the use of corticosteroids alone, but this was not significant and the evidence was low quality [18]. There was moderate-quality evidence that the combination reduced long-term sequelae such as excessive tear production and synkinesis. The outcome for patients who received corticosteroids alone was significantly better than for those who received antivirals alone. Antiviral drugs alone had no benefit over placebo. None of the treatments had significant differences in adverse effects, but the evidence was again of low quality [21]. All patients except the pregnant ones were treated with antiviral agent (valaciclovir 3x1000mg, orally for seven days), proton pump inhibitor (PPI) (pantoprazole 40 mg IV, during steroid therapy) and intravenous (IV) steroid (by decreasing 15 mg every three days) (methylprednisolone 1 mg/kg/day) in the present study.

The prognosis was generally very good in patients with Bell's palsy and full recovery is observed with a high rate between 80% and 90%. The most important factor affecting the healing outcome is partial or complete paralysis [22,23]. Of the 108 patients participating in the present study, 21 (19.44%) had partial improvement of motor functions (13 patients had HB II, five patients had HB III, two patients had HB V and one patient had HB VI), six (5%) developed hemifacial spasm and 81 (75%) recovered fully at the end of the first month,.

In his study, Fisch made decompression in 14 patients with a rate of more than 90% degeneration in ENoG in the first 3 weeks after the onset of paralysis and compared the long-term healing levels with non-surgical patients. He reported patients who underwent surgery to have better long-term improvements [24]. In their study, Gantz et al. performed surgical decompression in patients with a rate of more than 90% degeneration in ENoG and on the 14th day in EMG without voluntary motor unit potential and found that 90% of these patients recovered from HB stage I and II [25]. They emphasized that the operation should be performed within the first 2 weeks following the development of the paralysis. Five patients who had an over 90% drop in amplitude on electroneurography performed on the 14th day were recommended surgical decompression in the present study. Surgical decompression was performed in two patients who accepted the operation in the clinic where the study was conducted and at the end of the first month, one patient was found out to have HB III and the other HB IV.

Conclusion

Although there are many studies on BP today, a full consensus is not reached on the etiology, treatment, and prognosis of the disease. The main agents in the treatment of the disease are steroids and antiviral therapy which is provided by many clinics. Studies with larger patient series should be conducted to clarify the etiology, prognosis and to determine the treatment protocol.

Conflict of interests
We declare that we have no conflict of interest.

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Ethical approval
Afyonkarahisar Health Sciences University Ethical Board. No: 2011-KAEK-3

References

1. Chan JY, Byrne PJ. Management of facial paralysis in the 21st century. Facial Plastic Surgery. 2011;27:346-57.

2. Ho AL, Scott AM, Klassen AF, et al. Measuring quality of life and patient satisfaction in facial paralysis patients: a systematic review of patient-reported outcome measures. Plastic and reconstructive surgery. 2012;130:91-9.

3. Hadlock TA, Greenfield LJ, Wernick□Robinson M, et al. Multimodality approach to management of the paralyzed face. Th e Laryngoscope. 2006;116:138-95.

4. Kang TS, Vrabec JT, Giddings N, et al. Facial nerve grading systems (1985-2002): beyond the House-Brackmann scale. Otology & neurotology. 2002;23:767-71.

5. Ronthal M, Shefner JM, Dashe JF. Bell's palsy: Pathogenesis, clinical features, and diagnosis. 2009.

6. Adour KK. Current concepts in neurology: diagnosis and management of facial paralysis. N Engl J Med. 1982;307:348-51.

7. Peitersen E. Bell's palsy: the spontaneous course of 2,500 peripheral facial nerve palsies of different etiologies. Acta Otolaryngol Suppl. 2002;549:4-30.

8. Madhok V, Falk G, Fahey T, Sullivan FM. Prescribe prednisolone alone for Bell's palsy diagnosed within 72 hours of symptom onset. BMJ. 2009;6:338:b255.

9. Valença MM, Valença LP, Lima MC. Idiopathic facial paralysis (Bell's palsy): a study of 180 patients. Arq Neuropsiquiatr. 2001;59:733-99.

10. Yanagihara N, Hyodo M. Association of diabetes mellitus and hypertension with Bell's palsy and Ramsay Hunt syndrome. Ann Otol Rhinol Laryngol Suppl. 1988;137:5-7.

11. Katz A, Sergienko R, Dior U, Wiznitzer A, Kaplan DM, Sheiner E. Bell's palsy during pregnancy: is it associated with adverse perinatal outcome? Laryngoscope. 2011;121:1395-8

12. Adour KK, Wingerd J. Idiopathic facial paralysis (Bell's palsy): factors affecting severity and outcome in 446 patients. Neurology. 1974;24:1112-16.

13. Proctor B, Nager GT. The facial canal: normal anatomy, variations and anomalies. I. Normal anatomy of the facial canal. Ann Otol Rhinol Laryngol Suppl. 1982;97:33-44.

14. Demirci S, Kurt A, Tüzüner A, Samim AA, Caylan R. Bell paralizili hastalarda fasiyal kanal dehissans oranları. Tr-ENT. 2015;25: 87-91.

15. Al-Bishri A, Dahlin L, Sunzel B, Rosenquist J. Systemic betamethasone accelerates functional recovery after a crush injury to rat sciatic nerve. J Oral Maxillofac Surg. 2005;63:973-7.

16. Hato N, Yamada H, Kohno H, Matsumoto S. Valacyclovir and prednisolone treatment for Bell's palsy: a multicenter, randomized, placebo-controlled study. Otol Neurotol. 2007; 28:408–13.

17. Hato N, Sawai N, Teraoka M, Wakisaka H, Takahashi H, Hinohira Y, Gyo K. Valacyclovir for the treatment of Bell's palsy. Expert Opin Pharmacother. 2008;9:2531-6.

18. Gagyor I, Madhok VB, Daly F, Somasundara D, Sullivan M, Gammie F, et al. Antiviral treatment of Bell's palsy (idiopathic facial paralysis). Cochrane Database Syst Rev. 2015;11:CD001869.

19. de Almeida JR, Al Khabori M, Guyatt GH, Witterick IJ, Lin VY, Nedzelski JM, et al. Combined corticosteroid and antiviral treatment for Bell palsy: a systematic review and metaanalysis. JAMA. 2009;302:985-93.

20. Gronseth GS, Paduga R. American Academy of Neurology Evidence-based guideline update: steroids and antivirals for Bell palsy: report of the Guideline Development Subcommittee of the American Academy of Neurology. Neurology. 2012;79:2209-13.

21. Lee HY, Byun JY, Park MS, Yeo SG. Steroid-antiviral treatment improves the recovery rate in patients with severe Bell's palsy. Am J Med. 2013;126:336-41.

22. Devriese PP, Schumacher T, Scheide A, et al. Incidence, prognosis and recovery of Bell's palsy. A survey of about 1000 patients (1974-1983) Clin Otolaryngol Allied Sci. 1990;15:15-27.

23. Gilden DH. Clinical practice. Bell's Palsy. New Eng J Med. 2004;351:1323-34.

24. Fisch U. Surgery for Bell s palsy. Arch Otolaryngol .1981;107:177- 88.

25. Gantz BJ, Rubinstein JT, Gidley P, Woodworth GG. Surgical management of Bell's palsy. Laryngoscope. 1999;109:1177-88.