

REVIEW ON BELL'S PALSY

¹Kuzhalvaimoazhi P, ²Thenmozhi, ^{*3}Dhanraj Ganapathy

ABSTRACT

Bell's palsy is an acute idiopathic peripheral nerve palsy which affects the seventh cranial nerve-Facial nerve, which supplies all the muscles of facial expression. The seventh cranial nerve carries predominantly motor fibres but also supplies some autonomic innervation, sensation to part of ear, and taste to anterior two thirds of the tongue. It is responsible for lacrimation, tearing. But pathogenesis remains controversial. The paralysis causes disturbances among patients not only in social activities, life style, and psychological aspects but functional activities are also hindered like ability to eat, drink and speech. Many options of treatment exist including medications in the form of steroids, antiviral and neural multivitamins surgery and also the physiotherapy.

Keywords: *Bell's Palsy, nerve, paralysis, neural*

I. INTRODUCTION:

Bell's palsy is named after Sir Charles Bell (1774-1842), who first described the syndrome along with anatomy and function of facial nerve. Bell's palsy is a common disease affecting the seventh cranial nerve-Facial nerve which supplies all the muscles of facial expression. Bell's palsy may begin with symptoms of pain in the mastoid region and produce full or partial paralysis of movement of one side of the face. Facial nerve carries motor fibres but also supplies some autonomic innervation, sensation to part of ear, taste to anterior two-thirds of tongue. So, symptoms also include pain around the ear, altered sense of taste, impaired noise tolerance and decreased tearing. The incidence of Bell's palsy is 15-40 per 1,00,000. It occurs more commonly in patients with diabetes and in pregnant women. The pathogenesis of Bell's palsy remains controversial. But, the experimental evidence suggests that "herpes simplex type 1 infection" may play a role in Bell's palsy. Polymerase chain reaction assays have identified herpes simplex virus in endoneurial fluid, posterior auricular muscle and saliva in patients with Bell's palsy. The paralysis causes significant disturbances among the patients not only in social activities, life style and psychological aspects but also functional activities are hindered like the ability to drink, eat and express oneself either verbally or non-verbally.

¹Graduate Student, Department of Prosthodontics, Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Chennai, India.

²Professor, Department of Anatomy, Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Chennai, India.

³Professor and Head Department of Prosthodontics, Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences, Chennai – 600077 Tamil Nadu, India.

ETIOLOGY AND DIFFERENTIAL DIAGNOSIS:

Etiology of Bell's palsy is unknown; viral infection, ischaemia and auto immune disorders have all been postulated as possible mechanisms[1,2]. Many theories have been proposed to explain the origin of Bell's palsy. It is suggested that Ischaemia (due to disturbed circulation in the vasa nervorum) leads to the nerve injury in Bell's palsy. Vascular spasm causes a swelling of nerve in Fallopian canal, and secondary compressive edema ensues.[3]. This theory is the background for surgical decompression in the treatment of the disease. At present the most likely cause seems to be Herpes simplex virus (HSV-1), where the specific DNA can be isolated from peri neural fluid of cases with Bell's palsy.[4]. Bell's palsy is also believed to be caused by geniculate ganglion which leads to compression and also ischemia and demyelination. This ganglion lies in the facial canal at the junction of the labyrinths and tympanic segments, where the nerve curves sharply towards the stylomastoid foramen. The studies have failed to isolate viral DNA in biopsy specimens, leaving the causative role of HSV-1 in question.[5,6]. Other studies suggest that VZV plays an important etiological role in Bell's palsy, causing acute peripheral facial palsy without vesicles, so called Zoster sine herpette[7]. Before diagnosing Bell's palsy, physicians should consider other causes of acute facial paralysis because some require specific therapies. Infection, trauma, central nervous system disease, and stroke can all present with similar symptoms[8]. Etiologies such as Otitis media, mastoiditis, Hunt's syndrome (where vesicles seen in ear are caused by Varicella Zoster virus), meningitis, Lyme disease, Guillain -Barre syndrome, Myasthenia gravis, head trauma, neoplasm (eg: lymphoma and leukemia), Hypertension, diabetes mellitus and toxicity should all be considered.

SYMPTOMS:

Patients with Bell's palsy typically complain of weakness or complete paralysis of all the muscles on one side of face. Facial creases and nasolabial folds disappear, forehead un furrows, and the corner of mouth droops. The eyelids will not close and lower lip sags; on attempted closure, the eye rolls upward (Bell's phenomenon). Eye irritation often results from lack of lubrication and constant exposure. Tear production decreases. However, the eye may appear to tear excessively because of loss of lid control, which allows the tears to spill freely from the eye. Food and saliva can pool in affected side of the mouth and may spill out from the corner. Patients often complain of feeling of numbness from paralysis, but facial sensation is preserved. Patients with Bell's palsy usually progress from the onset of symptoms to maximal weakness within 3 days and almost within 1 week. Left untreated, 85 percent of patients will show at least partial recovery within 3 weeks of onset.[9]

PHYSICAL EXAMINATION:

Grading facial function is necessary for evaluating and communicating the spontaneous course of and the results of medical and surgical treatment of facial palsy[10]. There are two main types of facial grading systems: Gross and Regional systems. House facial nerve grading system was proposed by House in 1983. After minor modifications, the system was presented by House and Brackmann in 1985[11]. The House Brackmann

system was adopted as a standard of grading facial function by facial nerve disorders committee of American academy of otolaryngology-Head and Neck surgery,

1. Normal
2. Mild dysfunction; slight weakness noticeable. Only on close inspection
3. Moderate dysfunction; obvious, but not disfiguring, difference between two sides.
4. Moderately severe dysfunction; obvious weakness and /or disfiguring asymmetry.
5. Only barely perceptible motion
6. Loss of tone

Regional yanagihara grading system, presented by yanagihara in 1976[12,13] assesses 10 separate aspects of function in different facial muscles. Each function is scored from 0 to 4, giving maximum score of 40. This grading scale does not include any secondary effects. Yanagihara is the most widely used system in Japanese studies for evaluating facial nerve function in Bell's palsy, Herpes Zoster oticus and following acoustic neuroma surgery.[14].

TREATMENT:

Treatment of Bell's palsy has been a matter of debate for decades and no international consensus has yet been reached. Many different treatments have been suggested, but decompression surgery, corticosteroids, and antiviral treatments dominate the literature. However spontaneous recovery rates in Bell's palsy make evaluation of treatment effect difficult and require a large number of patients to be studied.[15,16,17].

SURGICAL TREATMENT:

In 1932, Balance and Duel[18] advocated decompression surgery for Bell's palsy. Indications surgery was based on theory of swellings and entrapment of facial nerve in the bony Fallopian canal. In the past, surgical decompression within three weeks of onset has been recommended for patients who have persistent loss of function (Greater than 90% loss on electroneurography) at two weeks.[19]. The most common complications of surgery is postoperative hearing loss, which affects 3 to 15 percent of patients. Based on significant potential for harms and the paucity of data supporting benefits, the American academy of Neurology does not currently recommend surgical decompression of Bell's palsy.[20]. May, an ardent proponent of surgical decompression[22] changed his opinion in 1984 and embraced the position that decompression was of no benefits in Bell's palsy.[23]. His conclusions were based on transmastoid decompression of the nerve that included the geniculate ganglion and distal portion of the labyrinthine segment. It remains controversial.

CORTICOSTEROIDS:

Oral corticosteroids have traditionally been prescribed to reduce facial nerve inflammation in patients with Bell's palsy. Prednisone is typically prescribed in a 10-day tapering course starting at 60mg per day. In 1954 controlled trial, Taverner[24] treated 14 patients within 10 days after onset of palsy with oral cortisone acetate (200mg daily for 3 days, 100mg for 3 days and 50 mg for 2 days) and did it find a significant reduction in the incidence of denervation compared with 12 untreated patients. A 2004 Cochrane review and metaanalysis of three randomized controlled trials comparing corticosteroids with placebo found small and statistically non-significant reductions in the percentage of patients with incomplete recovery.

ANTIVIRAL TREATMENT:

There is increasing evidence that the nerve injury in Bell's palsy is caused by deactivation of viruses of Herpes group[25]. The proposed link between Bell's palsy and Herpes viruses has led to use of an antiviral agent (Acyclovir or Valaciclovir) in the treatment of disease. Valaciclovir is a prodrug that is rapidly and nearly completely converted to Aciclovir, and its bioavailability is three fold to five fold that of Aciclovir[26]. Treatment with Aciclovir plus Prednisone was statistically more effective in returning volition all muscle motion, and in preventing nerve degeneration than in patients treated with placebo plus prednisone.[27].

SPONTANEOUS RECOVERY:

It is difficult to establish a statistically significant benefit of treatment in placebo-controlled trials because Bell's palsy has a high rate of spontaneous recovery. The Copenhagen facial nerve palsy evaluated 2,570 patients with untreated facial nerve palsy, including 1,701 with idiopathic (Bell's) palsy and 869 with palsy from other causes; 70% had complete paralysis. Because of these findings, some persons have questioned whether the treatment for Bell's palsy should be indicated; However, patients who have incomplete recovery will have obvious cosmetic sequelae and will often be dissatisfied with their outcome.

II. CONCLUSION:

Bell's palsy due to its sudden onset gives an impression of stroke and creates panic in patients and clinicians. Hence proper history and examination is important to arrive at an accurate diagnosis. An expressionless face causes a lot of social dilemma to the patients. Various treatments are available, but early initiation of treatment is the key to recovery in majority of cases. Although corticosteroids remain the main mode of treatment, future studies will be needed to determine which population will most benefit from antiviral therapy.

REFERENCES:

1. Singhi P, Jain V. Bell's palsy in children, *Semin Pediatr Neurol* 2003;10(4):289-97

2. Adour KK, Byl FM, Hilsinger RL Jr, Kahn ZM, Sheldon MI. The true nature of Bell's palsy: analysis of 1000 consecutive patients. *Laryngoscope* 1978;88(5):787-801
3. Miehleke A, Stennert E, Arold R, et al [Surgery of the nerves of nose, neck, and ear region (except Nn. statoacusticus and olfactorius) (authors' translation)]. *Arch otorhinolaryngol.* 1981;231(1):89-449
4. Murakami S, Mizobuchi M, Nakashiro Y, Doi T, Hato N, Yanagihara N. Bell's palsy and Herpes simplex virus: Identification of viral DNA in endoneural fluid and muscle. *Ann Intern Med* 1996;124:27-30
5. Linder T, Bossart W, Badmer D. Bell's palsy and Herpes simplex virus: fact or mystery? *Otol Neurotol.* 2005;26:108-13
6. Stjernquist-Desatnik A, Skoog E, Aurelius E. Detection of Herpes simplex and varicella-zoster viruses in patients with Bell's palsy by the polymerase chain reaction technique. *Ann Otol laryngol* 2006;115:306-11
7. Furuta Y, Ohtani F, Aizawa H, Fukuda S, Kawabata H, Pergstrom T. Varicella zoster virus reactivation is an important cause of acute peripheral facial paralysis in children. *Pediatr Infect Dis. J.* 2005 Feb;24(2):97.col
8. Schwing AG, Gunter JP. Paralysis of facial nerve in children. *Clin pediatr (phela)* 1970;9(2):105-9.
9. Peiterson F. Bell's palsy: The spontaneous course of 2,500 peripheral facial nerve palsies of different etiologies. *Acta Otolaryngol suppl.* 2002;4-30.
10. House JW. Facial nerve grading system. *Laryngoscope* Aug;93(8):1056-69
11. House JW, Brackmann DE. Facial nerve grading system. *Otolaryngeal Head neck surg.* 1985 apr;93(2):146-7
12. Yanagihara N. Grading of facial palsy. Proceedings third international symposium on facial nerve surgery, Zurich, 1976. Fisch U editor. Facial nerve surgery. Birmingham kuglermedicalpublication, Amstelveen, Netherlands; aesculapius publishing 10; 1977. P.533-5.
13. Yanagihara N. Grading of facial palsy. Proceedings third international symposium on facial nerve surgery, Zurich, 1976. Fisch U editor: Kugler Medical publications. Amstelveen, Netherlands; Aesculapius publishing co, Birmingham; 1977
14. Satoh Y, Kanzaki J, Yoshikara S. A comparison and the conversion table of 'the house Brackmann facial nerve grading system' and 'Yanagihara grading system'. *Auris Nasus larynx.* 2000jul;27(3):207-12
15. Grogan PM, Groseth GS. Practice parameter: steroids, acyclovir and surgery for Bell's palsy (an evidence based review): report of quality standards subcommittee of American academy of Neurology. 2001 apr10;56(7):830-6
16. Allen D, Dunn L. Aciclovir or Valaciclovir for Bell's palsy (Idiopathic facial paralysis). *Cochrane Database Syst Rev* 2004(4):CD001869
17. Salinas RA, Alvarz G, Ferreria J. Corticosteroids for Bell's palsy (idiopathic facial nerve paralysis). *Cochrane Database Syst Rev* 2004(4):CD001942

18. Balance C, Duell AB. The operative treatment of facial palsy. *Arch Otolaryngol.* 1932;15:1-70
19. Gantz EJ, Rubinstein JT, Gidley P, Woodworth GG. Surgical management of Bell's palsy
20. Grogan PM, Groseth GS. Practice parameter: steroids, acyclovir
21. Fisch U, Esslen E. Total intratemporal exposure of the facial nerve. *Arch Otolaryngol* 1972;95:335-341
22. May M, Hawkins CD. Bell's palsy: results of surgery; salivation test versus nerve excitability test as a basis of treatment. *Laryngoscope* 1972;82:1337-1348
23. May M, Blumenthal F, Taylor FH. Bell's palsy: Surgery based upon the prognostic indications and results. *Laryngoscope* 1981;91-2092-2103
24. Taverner D. Cortisone treatment of Bell's palsy. *Lancet* 1954 Nov 20;267(6947):1052-4
25. McCornick DP. Herpes simplex virus as a cause of Bell's palsy. *Lancet.* 1972 apr 29;(9757):937-9
26. Beuther K.R. Valacyclovir; A review of its antiviral activity, Pharmacokinetic properties, and clinical efficacy *Antiviral Res.* 1995 dec;28(4):281-90.
27. Adour KK, Wingerd J, Bell DW, Manning JJ, Hurley SP. Prednisone treatment for idiopathic facial paralysis (Bell's palsy). *N Engl J Med.* 1978 dec 21; 287(25):1260-72
28. Gilman GS, Scatkin BM, Klein SR. Bell's palsy in pregnancy. A study of recovery outcome *Otolaryngol head neck surg* 2002;126:26-30