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REVIEW ARTICLE

UPDATE ON MANAGEMENT OF BELL'S PALSY: A SCOPING REVIEW

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ABSTRACT

Bell's palsy is the most frequent cause of unilateral peripheral facial nerve palsy with an acute onset. Although the exact cause of Bell's palsy is unknown, there is evidence that it is likely related to viral infections such herpes zoster virus and herpes simplex virus-1 (HSV-1). Bell's palsy symptoms usually peak in the first week and then progressively go away over the course of three weeks to three months. It often occurs in patients with diabetes mellitus and in pregnant women. Diagnosis of Bell's palsy is purely on clinical basis. Management of Bell's palsy aims to achieve complete recovery and reduce negative sequelae of patients that do not resolve spontaneously. Treatment should be started early in the course of the disease and within 72 hours of onset. Both antiviral and corticosteroids are used as treatment strategies. Patients who get corticosteroids alone had better results than those who just receive antivirals. Additionally, antiviral medications are no more effective than a placebo.

KEYWORDS

Bell's palsy, antiviral drugs, corticosteroids, facial nerve palsy.

1. INTRODUCTION

The most prevalent acute mononeuropathy affecting the facial nerve is called Bell's palsy, named after the Scottish anatomist Sir Charles Bell (Peitersen, 2002). Bell's palsy is the commonest etiology for peripheral facial nerve paralysis. Bell's palsy is a peripheral and acute facial nerve paralysis of unknown etiology (Singh et al., 2022). Bell's palsy symptoms are believed to be caused by inflammation and oedema of the facial nerve, even if the exact cause of the condition is unknown. Although Bell's palsy symptoms can range from minor facial weakness to severe paralysis, the prognosis is typically favourable (Swain, S.K., Pati, B.K., Mohanty, J.N., 2020). The treatment of Bell's palsy should be started as soon as possible following onset of facial nerve paralysis. The treatment of Bell's palsy includes corticosteroids for reducing the swelling and antiviral drugs for combating infection by herpes simplex virus, which is possible etiology for this lesion (Rim et al., 2023).This review's objective is to provide a thorough analysis of the current management of Bell's palsy.

2. METHODS OF LITERATURE SEARCH

Research articles on current treatment of Bell's palsy were sought after. This started by looking through online databases including Google Scholar, Scopus, PubMed, and Medline, which covered the research that has been published up to this point. This scoping review assessed the current treatment of Bell's palsy and followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) guidelines. The manuscript includes the specific search terms and Boolean operators used e.g. " current treatment of Bell's palsy ". Once the data have been extracted from all articles, numerical and thematic analysis were conducted. All search results were reviewed based on inclusion and exclusion criteria. The data included all age groups and both genders with current treatment of Bell's palsy. Articles were initially screened by their titles to determine eligibility, followed by a thorough review of each abstract to ensure they met the inclusion criteria. The study include in this review are restricted to English language. The citations were employed manually to locate further research articles after the search strategy turned up published article abstracts. Articles were excluded as per the criteria such as non-peer reviewed articles, non-original research, reviews, case reports, letters, editorials, insufficient information on subtotal petrosectomy. The evaluation assessed whether observational studies, comparative studies, case series, case reports, and randomized controlled trials were appropriate. Forty five of the 68 (15 case reports, 26 case series, and 27 original articles) that were discovered in different databases were included in this PRISMA Flow diagram for study selection (Fig. 1). The synthesis of findings was conducted by use of quantitative and qualitative methods. This review article discusses the prevalence, etiopathogenesis, clinical presentations, diagnosis, treatment of Bell's palsy including its surgical decompression and prognosis.

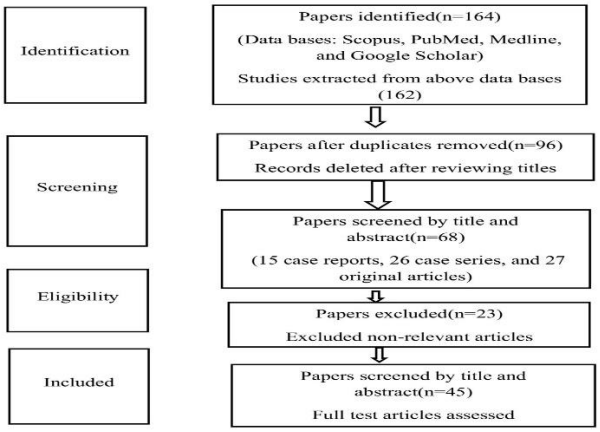

Figure 1: Methods of literature search.

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3. Prevalence

Bell's palsy is a prevalent clinical condition, with an annual incidence of 20 per 100,000. Bell's palsy patients may not fully regain facial function in up to 30% of cases (Varga et al., 2023). Thus, thousands of individuals with Bell's palsy are left with lifelong, possibly disfiguring paralysis of the face every year. The median age is 40, and both sexes are equally affected and can happen at any age. The incidence is lowest in those under the age of ten, rises between the ages of ten and twenty-nine, stays constant between the ages of thirty and sixty-nine, and peaks in those over seventy. Right and left sides are affected equally While some Bell's palsy patients have persistent, deforming facial paralysis, the majority of individuals fully recover (Swain et al., 2017; Katusi et al., 1986).

3.1 Aetiopathogenesis

Bell's palsy postmortem cases show vascular distension, inflammation, and oedema with facial nerve ischemia being the underlying etiopathology (Singh et al., 2022). Although viral reactivation has been suggested, the precise cause of Bell's palsy is still unknown. [8] There is evidence for reactivation of the herpes simplex virus (HSV) within the geniculate ganglion that may promote inflammation and oedema of the facial nerve, resulting in compression of the nerve inside the fallopian canal (Furuta et al., 1998).

The neuronal inflammation associated with Bell's palsy is assumed to be related to viral infection. Bell's palsy patients' endoneurial fluid has been found to contain the herpes simplex virus (Murakami et al., 1996). Viral DNA has been found in the facial nerve and posterior auricular muscle of the affected patients (Ho and Markowsky, 2022). However, HSV DNA has also been detected in person without any history of facial nerve palsy, indicating that presence of virus alone does not fully explain the disease mechanism (Thomas et al., 2005).

Pregnancy, diabetes mellitus, atherosclerosis, and a family history of Bell's palsy are additional risk factors. The risk factors for Bell's palsy include diabetes mellitus, pregnancy, obesity, preeclampsia, and hypertension (Berkowitz, 2014).

3.2 Clinical Presentations

Table 1: Clinical presentations of Bell's palsy

Serial number	**Clinical presentations**
1	Weakness or paralysis of lower and upper facial muscles of the affected side
2	Inability to close the eye completely
3	Dry eye due to incomplete closure of the eye
4	Drooping of ipsilateral eyelids
5	Epiphora due to exposure keratitis
6	Ipsilateral impaired/loss of taste sensation
7	Difficulty with eating due to ipsilateral muscle weakness resulting food to be trapped on the affected side of mouth
8	Dribbling of the saliva at the affected side of mouth
9	Altered sensation on the affected side of the face
10	Pain in or behind the ear
11	Hyperacusis on the affected side if stapedius muscle is affected

Patients with Bell's palsy usually present with acute unexplained unilateral peripheral facial weakness (Swain et al., 2021). Bell's palsy usually manifests abruptly and peaks in a few days (Swain et al., 2021).

Additional symptoms include hyperacusis, impaired taste on the ipsilateral anterior region of the tongue, numbness or tingling in the affected side of the face that is not linked to a neurological examination impairment, and pain in or behind the ear (Table.1) (Dalrymple et al., 2023).Compared to unilateral involvement, bilateral idiopathic facial palsy is less common (Swain et al., 2020).

Although normally Bell's palsy is self-limited, this may produce substantial transient oral incompetence and an inability to close the eyelid, leading to potential eye damage. Rarely does Bell's palsy return. Recurrent or bilateral facial palsy should elicit investigation of myasthenia gravis, Guillain Barre syndrome, and diseases at base of brain, where the facial nerve exits the pons (Swain et al., 2016).

3.3 Diagnosis

Table.2: House-Brackmann facial nerve grading system

Grade	**Description**	**Clinical characteristics**
I	Normal	Normal facial symmetry and functions in all parts of face
II	Mild dysfunction	Slight weakness noticeable on close inspection; normal tone and symmetry at rest; complete eye closure with minimal effort
III	Moderate dysfunction	Obvious bot no disfiguring asymmetry; normal tone at rest; slight to moderate forehead movement; complete eye closure with effort
IV	Moderately severe dysfunction	Marked weakness and asymmetry; absent forehead movement; incomplete eye closure; asymmetric mouth movement
V	Severe dysfunction	Barely perceptible motion; facial asymmetry even at rest; absent forehead and minimal mouth movement
VI	Total paralysis	Complete absence of movement in parts of face

Bell's palsy can be diagnosed with the aid of a clinical examination and history (Ho and Markowsky, 2022). The House-Brackmann facial nerve grading system (Table.2) can be utilized for knowing the degree of facial nerve palsy. Grade I (no weakness) to VI (total weakness) is the range of this grading system. Bell's palsy is also called as idiopathic facial nerve paralysis. It is classified as an acute onset, solitary, unilateral, and lower motor nervon facial weakness. Bell's palsy is considered as a diagnostic of exclusion, which can be identified when there is no other medical reason of facial nerve paralysis and the onset is quick (within 72 hours). There are several diagnostic tests for evaluation of Bell's palsy patients. These tests include laboratory tests, diagnostic imaging studies, and electrodiagnostic tests. Other diagnoses are usually excluded by complete blood tests and other cerebrospinal fluid laboratory parameters, as well as the use of electroneurography (ENoG), electromyography (EMG) and MRI (Rajangam et al., 2024). Within 14 days of commencement, electrodiagnostic testing can be performed to offer prognostic data. [18] The nerve excitability test establishes the excitation threshold by recording minimum electrical stimulus needed to induce observable muscle contraction. In terms of a worse result, a difference of more than 3.5 mA between the affected and unaffected sides is deemed significant. Prognostic significance can be determined by measuring the peak-to-peak amplitude of the elicited compound action potential of the affected side in comparison to the normal side. The prognosis is poor if the affected side's amplitude has decreased by 90% or more (Brackmann and Fetterman, 2007). Currently the trigeminal blink reflex is the sole test to determine intracranial route of the facial nerve and also beneficial to examine diverse post-paralysis sequelae including synkinesis and hemifacial spasms. Bell's palsy is primarily diagnosed clinically, despite advancements in neuroimaging. Determining whether facial palsy is central or peripheral is the primary goal of the diagnosis. Peripheral facial nerve paralysis involves all the facial muscles ipsilateral to the side of the facial nerve involvement whereas central weakening involves lower facial muscles contralateral to the lesion in the brain stem above the pons and cerebral hemisphere. Bells palsy's quick onset over several hours sets it apart from

other causes of facial nerve paralysis, including Ramsay Hunt syndrome, diabetes mellitus, HIV, Lyme disease, sarcoidosis, Sjogren syndrome, parotid-nerve tumors, leprosy, amyloidosis, and polyarteritis nodosa (Tolisano et al., 2019). Secondary causes of facial nerve palsy develop over a period of days to months. Laboratory or radiographic testing is not necessary if the clinical presentation is consistent with Bell's palsy. Patients may be assessed for the primary source of their symptoms if they exhibit unusual characteristics. Likewise, Lyme illness is based on a history of probable tick-born disease. MRI can detect the inflammation of the facial nerve as well as ruling out other disorders including schwannoma, haemangioma or space occupying lesions (Tolisano et al., 2019).

3.4 Treatment

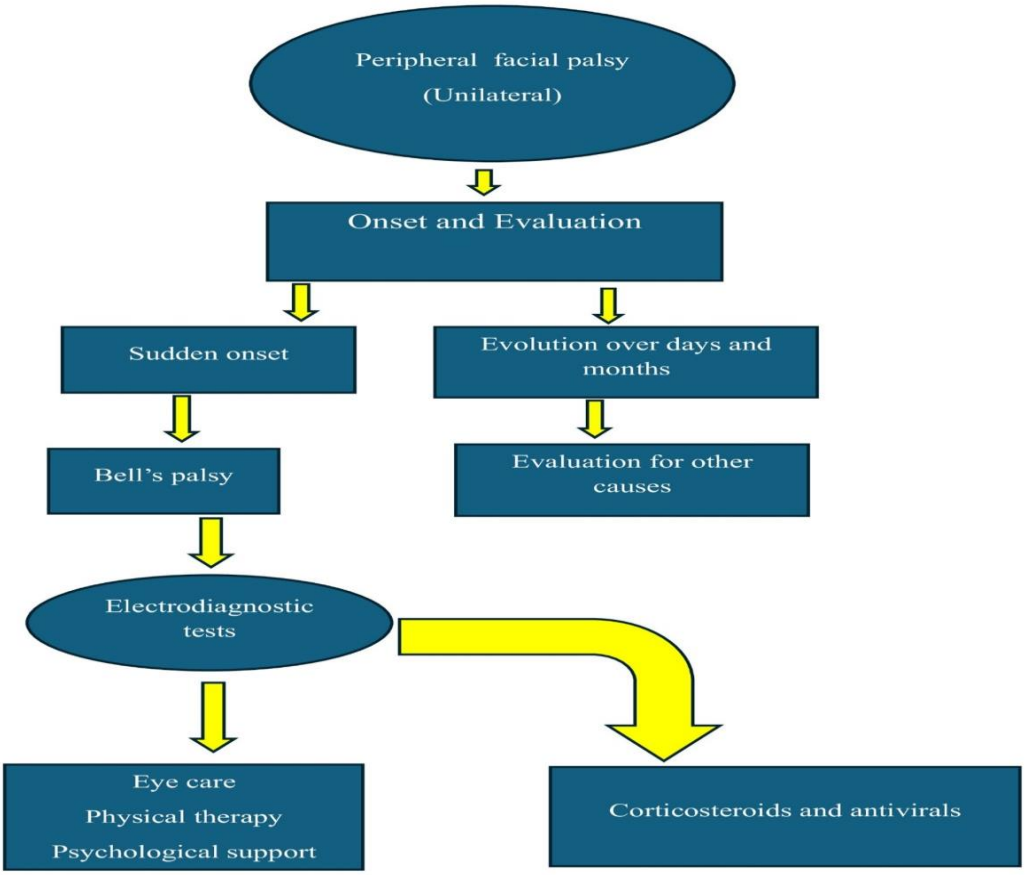

Figure 2: Flow chart for medical treatment.

Medical therapy (corticosteroids, antivirals, alone and in combination), surgical decompression, and complementary and alternative therapies like acupuncture are just a few of the many therapeutic options available for Bell's palsy. Although Bell's palsy is a self-limiting condition, some research indicates that about 70% of patients with mild to moderate conditions would recover on their own without treatment. However, treatment, especially the use of corticosteroids, may improve recovery and lessen long-term complications (Zhang et al., 2020). The goal of Bell's palsy treatment is to hasten recovery and reduce long-term issues. Corneal problems may arise from an inability to close the affected eye. Because eye protection is so important, lubricants and an eye patch are used to keep the cornea from drying out. Topical eye drops, like Hypromellose drops, should be used as an ointment at night and as a lubricant during the day. The eye might need to be partially sutured or taped shut in extreme circumstances. Oral glucocorticoids, glucocorticoids in combination with antivirals, physical therapy, electric stimulation, ocular protection for incomplete eye closure, decompression of the facial nerve, facial remodelling surgery, and acupuncture are common treatments for Bell's palsy (de Almeida et al., 2014). The flow chart for medical treatment is given in Fig.2.

3.5 Role of steroids

Because Bell's palsy is thought to be caused by inflammation and oedema of the facial nerve, corticosteroids are used to treat the condition. Inflammation of the facial nerve is considered as the key to pathogenesis of Bell's palsy. Strong anti-inflammatory properties of corticosteroids should lessen nerve injury and enhance the result. Corticosteroids are the cornerstone of treatment for Bell's palsy, according to the American Academy of Neurology (AAN) and the American Academy of Otolaryngology-Head and Neck Surgery Foundation (AAOHNSF). According to these recommendations, oral steroid therapy-specifically, prednisone 50–60 mg daily followed by tapering should be started within 72 hours after commencement (Stew and Williams, 2013). Prednisolone treatment has been shown to improve Bell's palsy outcomes and shorten recovery times in randomized, double-blind, placebo-controlled trials (Engstrom et al., 2008). The dose of prednisolone in adult was 60 mg per day for five days then decreased by 10 mg per day (for a total treatment time of 10 days) and 50 mg per day (in two divided doses) for 10 days (Nam et al., 2019). Routine antiviral monotherapy is not recommended, although combined steroid-antiviral therapy may be helpful in selected cases (de Almeida et al., 2014). Prednisolone should be used with caution in immunosuppression and sepsis.

3.6 Role of antiviral drugs

The theoretical rationale for antivirals in Bell's palsy lie in the potential role of HSV-1 and zoster sine herpete (herpes zoster reactivation without rash) (Shafshak et al., 1994). In an autopsy study latent HSV type-1 has been isolated from the majority of geniculate ganglion samples (Furuta et al., 1992). HSV-1 genome was detected in 79% of facial nerve endoneurial fluid in patients of Bell's palsy, but not in controls (Murakami et al., 1996). The therapeutic advantage of antivirals may be attributed to their mechanism of action, wherein acyclovir (400mg five times daily for five days) inhibits HSV DNA polymerase, preventing viral replication (Hato et al., 2007). A study showed that valacyclovir (1000 mg/day three times daily for five days) in combination with corticosteroids have reported recovery rates exceeding 96% compared to approximately 89% with steroids (Hato et al., 2002). However, not all studies support the additional use of antivirals for treatment of Bell's palsy. One study showed no significant improvement when acyclovir was combined with prednisolone (Sullivan et al., 2007). Cochrane review indicated that combined treatment of antivirals and corticosteroids reduces long-term sequelae compared with corticosteroids alone (Gagyor et al., 2019). The antivirals used are acyclovir, famciclovir, and valaciclovir (Allen and Dunn, 2004). The most recent guidelines from the American Academy of Neurology suggest that

acyclovir combined with prednisone is possibly effective for treatment of Bell's palsy (Allen and Dunn, 2004). Patients with Bell's palsy often unable to close the eye on the affected side, which can result in irritation and corneal ulceration. The eye should be lubricated by artificial tears until the facial palsy resolves. Permanent eyelid weakness may need tarsorrhaphy or implantation of gold in the upper lid.

3.7 Surgical decompression

Surgical decompression of the facial nerve may be necessary for certain Bell's palsy patients. The geniculate ganglion and labyrinthine segment occupy the entrance to the meatal foramen, which is the narrowest point of the facial nerve. Due to small sample sizes, potential bias in patient selection for surgery, the use of different surgical techniques and systems to evaluate facial nerve function, and the lack of blinding in studies assessing functional outcomes, there is a dearth of data comparing surgical decompression of the facial nerve with medical therapy in Bell's palsy. After surgical decompression of the facial nerve, permanent unilateral hearing loss may occur with range from less than 1% to 15% of patients (Gantz et al., 1999). Decompression should not be carried out 14 days or more after the onset of facial nerve paralysis since significant degeneration of the facial nerve is likely permanent after 2 to 3 weeks (Swain et al., 2021).

3.8 non-medical treatment

The physical therapies such as facial exercises, biofeedback, laser, acupuncture to the affected muscles, massage, thermotherapy and electrical stimulation have been used to hasten recovery of Bell's palsy. However, there is no evidence of any significant benefit. Tailore facial exercises can be helpful to improve facial function, mainly in moderate facial paralysis and chronic cases. Early facial exercise may reduce recovery time, long-term paralysis and number of chronic cases (Teixeira et al., 2011). The results of a Cochrane systematic evaluation evaluating the effectiveness of physical treatments, electrostimulation, and workouts on Bell's palsy outcomes showed that none of these physical therapies significantly improved or worsened the condition. Another systematic review examined the impact of facial exercises combined with mirror or electromyogram biofeedback on the complications of delayed recovery in Bell's palsy. It came to the conclusion that there were not enough randomized controlled trials to analyse the effectiveness of the exercises (Cardoso et al., 2008). Facial exercises, however, shorten recovery times, and their effects require confirmation through high-quality randomized controlled trials (Teixeira et al., 2011). Surgery to release the facial nerve has also been explored. Nevertheless, the quality of the evidence supporting this technique is really poor. [40] Botulinum toxin (Botox) injections or cosmetic surgery may be required to treat the muscle contractures and facial asymmetry (Swain 2025).

3.9 Hyperbaric oxygen

Hyperbaric oxygen (HBO) has greater benefit to patients with Bell's palsy (Racic et al., 1997). HBO therapy is a promising adjunct treatment, but no good quality evidence from large scale-controlled trials to establish it a standard treatment. From case studies and small trials showed recovery and improvement of facial nerve palsy, especially in moderate to severe cases combined with conventional treatments. HBO therapy involves breathing 100% oxygen in a pressurized chamber which reach the blood and finally send oxygen to inflamed facial nerve and surrounding tissues that may be oxygen deprived.

3.10 Nursing care

The eye care should be done by lubricating with artificial tear. To avoid injury to cornea, the patient should wear an eye patch at night. When the patient goes outside, they should use sunglasses. To increase muscle tone, facial muscle workouts must be performed correctly. Patient should be encouraged to eat on the other side of mouth. Patients should be checked regularly for dental check-up.

4. Prognosis

Bell's palsy can cause symptoms ranging from slight weakness to severe paralysis of the facial nerve, but the prognosis is usually favourable. According to the Copenhagen Facial Nerve Study, 71% of Bell's palsy patients regain normal function without treatment (Peitersen, 2002). About 4% have severe weakness that results in significant facial impairment, while about 13% have mild weakness (Peitersen, 2002). Contracture of the facial muscles at the affected side was found in approximately 17% and associated movements were seen in 16%. [43] Approximately 13% of patients with Bell's palsy are left with slight weakness and around 4% with severe weakness leading to major facial nerve dysfunction. Scoring systems like House-Brackmann scale used in randomised controlled trials and systematic reviews may be helpful for monitoring the progress (Swain, 2025). Recurrence is possible in about 7% of persons with a history of Bell's palsy. The average time between the first recurrence and the first episode is 9.8 years. Older age, hypertension, taste impairment, pain in area other than ear, and whole facial weakness are among the poor prognostic factors for Bell's palsy (Swain et al., 2018).

5. Conclusion

Patients with severe or complete Bell's palsy should consider about using antivirals, especially if they have a significant chance of contracting herpes zoster even if they don't have a typical zoster rash. Corticosteroids plus antivirals provide a better degree of facial muscle recovery than does corticosteroids alone. The early protection of the eye with artificial tears and patch are crucial for preventing long-term complications. In certain situations, adding an antiviral medication to prednisolone may be advantageous.

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