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REVIEW ARTICLE

Advances in Understanding and Managing Bell's palsy: A Systematic Clinical review

Sakshi R. Chavat[1*], Mansi G. Karnade[1], Vishal B. Rajguru[1], Vaishnavi P. Patil[2], Meena Bhalke[2], Shailesh Kewatkar[3], Shirish Jain[4].

1. Research Scholar, Department of Pharmacology, Rajarshi Shahu College of pharmacy, Buldhana, Maharashtra, India. 443001
2. Assistant Professor, Department of Pharmacology, Rajarshi Shahu College of Pharmacy, Buldhana, Maharashtra, India.443001
3. Associate Professor, Head of Pharmacognosy Department, Rajarshi Shahu College of Pharmacy, Buldhana, Maharashtra, India.443001
4. Principal, Rajarshi Shahu College of Pharmacy, Buldhana, Maharashtra, India.443001

Corresponding Author*: Sakshi R. Chavat
Email – chasakshi9@gmail.com

ABSTRACT

Bell palsy is one of the condition arises due to failure of lower motor neuron. Mainly affecting facial neuron which is present in 7th Cranial nerve. This condition is a frequent form of acute facial paralysis that causes facial muscle weakness and severe social discomfort. Although the precise cause is yet unknown, immunological, infectious, and ischemia processes are implicated; the leading possibility is viral reactivation, namely of herpes viruses. Both sexes are equally affected, with the majority of cases occurring between the ages of 15 and 45. Historically Bell's palsy was first noted in 1683. Facial paralysis can range from moderate to severe, and the symptoms frequently resemble tumours or strokes. Neurophysiological tests, imaging, and laboratory studies are used in the diagnosis process to rule out other disorders. In addition to corticosteroids, antivirals, and eye and oral care, other treatment options include physical therapy and surgery. Age and the timing of therapy are two examples of variables that affect prognosis. Bell's palsy frequently has psychological ramifications in addition to physical ones, such as anxiety and melancholy because of changed facial expressions. In order to restore face motion, new therapeutic approaches emphasize artificial muscles, Neuroprosthetic devices, synthetic conduits, and nerve regeneration. Despite these development, Bell's palsy remain a condition require further research to fully understand its mechanism and improve treatment outcome

Keywords: Bell's palsy, Viral Reactivation, diagnosis and management, Neurophysiological tests, psychosocial impact, nerve regeneration, facial mobility.

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INTRODUCTION

A person's face plays a crucial role in defining his personality and individuality. In addition to physical disabilities, any impairment in facial muscle function causes social and psychological discomfort because facial expressions are essential for social interactions and emotional expression. One of the most common forms of facial paralysis caused by lower motor neurons is Bell's palsy, an acute-onset peripheral facial neuropathy [1]. It indicates that a person with Bell's palsy has facial muscular weakness [2]. Although the exact origin of Bell's palsy is still unknown, immune, infectious, and ischemia mechanisms are all possible causes [3]. Bell's palsy is thought to occur at any age, according to study. But the majority of instances involve people aged 16 to 60 [4].

History

Later Sir Charles Bell described the innervation of the facial muscles and the skin of the face and consequently the trigeminal nerve is called Bell's nerve. Eventually the eponym "Bell's palsy" became

synonymous with idiopathic peripheral facial nerve paralysis. However, Friedreich described the syndrome 23 years before Bell. Bell's palsy was first described by Friedreich in 1797. The clinical entity, now known as Bell's palsy, was first observed in 1683 by Cornelis Stalpart van der Wiel [5]. Bell's palsy, which he dubbed "rheumatic paralysis of the facial muscles" as a result of exposure to cold air, was originally described in a thesis by Nicolaus Anton Friedreich [6]. Sir Charles Bell, who discovered the facial nerve's actual function, similarly thought that rheumatism or cold exposure was the cause of the attack. Despite a plethora of other theories, the actual aetiology of Bell's palsy remains unknown [7].

EPIDEMIOLOGY

Bell's palsy is a prevalent mononeuropathy of the brain. Males and females are equally affected, and while it is slightly more common in midlife and later in life, it happens at all ages. Nearly three-quarters of all acute facial palsies are Bell's palsy, which is most common in those aged 15 to 45 [8]. Most studies fail to illustrate clustering and pandemic occurrences. The recent rise in Bell's palsy cases during an intranasal vaccine delivery study is an exception to this rule [9]. The immunological effects of the detoxified Escherichia coli heat-labile toxin adjuvant utilized in this vaccine administration method may have been the cause of this [10]. Pregnancy, viral upper respiratory tract infections, immunocompromised conditions, diabetes mellitus, and hypertension are associated with increased occurrence. Incidence does not exhibit a clear latitudinal variation or a racial or ethnic bias. According to certain epidemiological statistics, there is a little prevalence of dry regions over non-arid ones, and the frequency is slightly greater in colder months than in warmer ones [8].

Etiopathogenesis:

Certain immunological, ischemia, and genetic variables substantially correlate with the aetiology of Bell's palsy, despite the fact that its exact pathophysiology is unknown and thought to be idiopathic. Recent findings have shown that a key factor in the pathogenesis is the geniculate ganglion's reactivation of the latent herpes virus and its subsequent migration to the facial nerve [11, 3]. Since the HZV virus uses satellite cells to go throughout the nerve, it is thought to be more aggressive. While herpes zoster causes shingles and chickenpox, herpes simplex typically causes cold sores and genital herpes. When there is no active viral replication, the infection is considered latent. However, when antibodies are present or the patient is immune compromised, the facial nerve may become inflamed and damaged, which further compresses the nerve because it is located in a tiny bone canal. Other viruses that have been linked to Bell's palsy include influenza B, CMV, adenovirus, mumps virus, and Epstein Barr virus, which causes infectious mononucleosis [12].Nearly three-quarters of all acute facial palsies are Bell's palsy, with the highest frequency occurring in those aged 15 to 45 (as given in fig.1) [13].

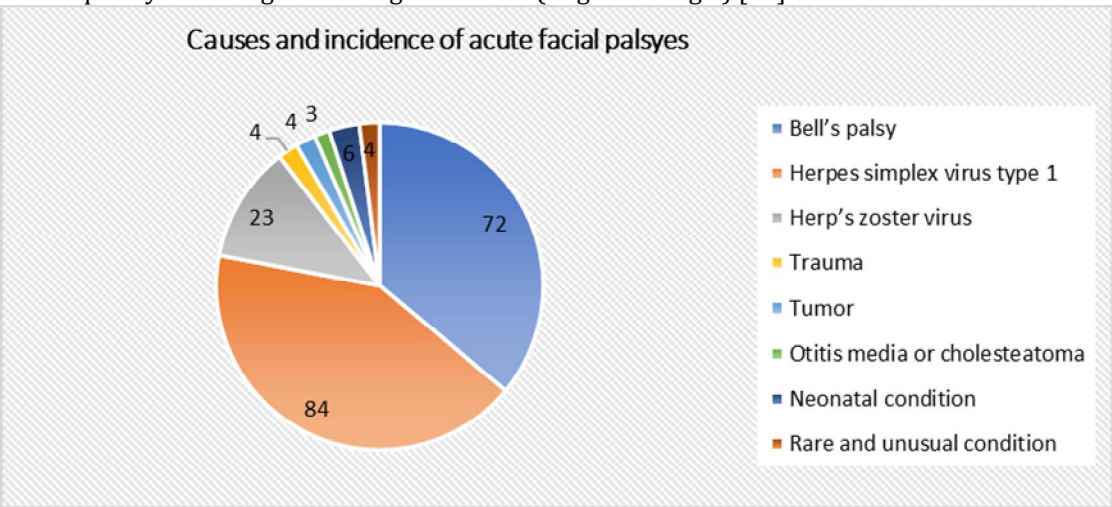

Figure 1: Causes and incidence of acute facial palsy

PATHOPHYSIOLOGY

The pathophysiology of Bell's palsy is multifactorial, involving several mechanism that contribute to facial nerve dysfunction. Detailed explanation of key factor are outlined below (Figure 2).

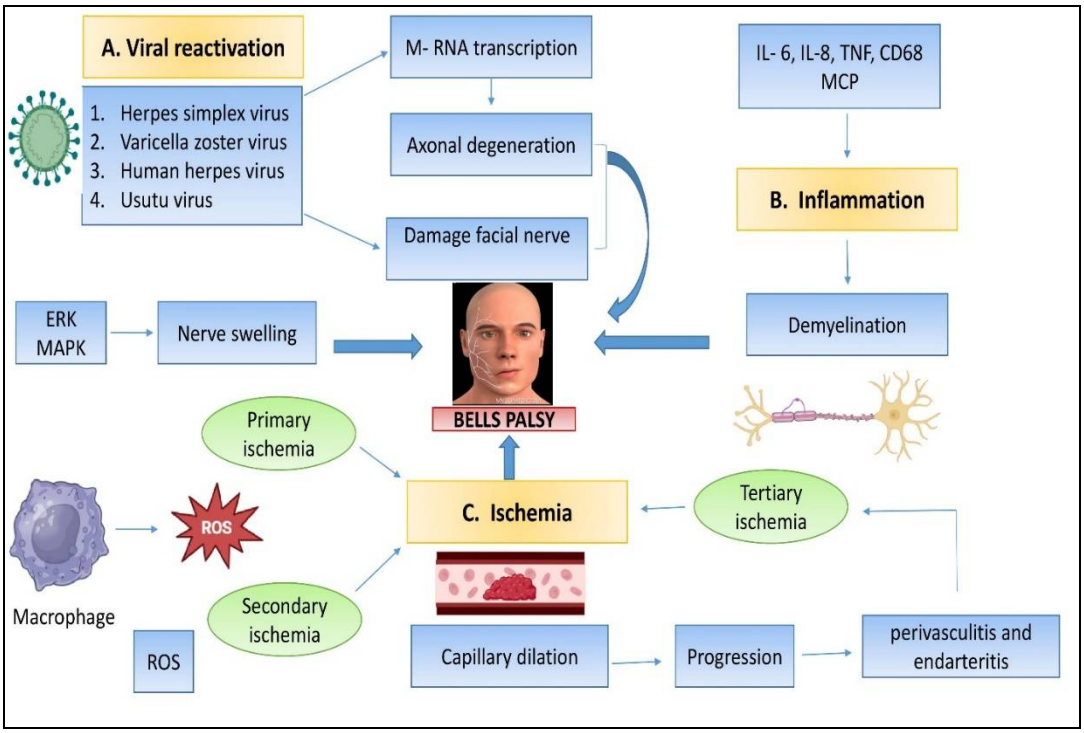

Figure 2. Pathophysiology of Bell's palsy

Viral reactivation

It has been proposed that reactivated viruses, including the Usutu virus, human herpes virus 6 [16], herpes simplex virus type 1 (HSV-1) [15], and varicella zoster virus (VZV) [14], are the cause of BP [17]. Large, enclosed viruses containing double-stranded linear DNA are known as herpes viruses (HV). α-HV infections, such as HSV-1, HSV-2, and VZV, are among the most prevalent viral infections in the world and target peripheral neurons [18].

HSV-1 reactivation and neuronal dysfunction

The geniculate ganglion was the focal point of HSV-1 reactivation, which may have been connected to blood pressure [19]. The existence of HSV-1 DNA in clinical specimens from BP patients, such as intratemporal facial nerve endo-neural fluid [20]. The aberrant expression of the innate immune signalling protein SARM1 and the p53 upregulated modulator of apoptosis (PUMA) may be a bio molecular mechanism of HSV-mediated brain dysfunction [21].

Intra axonal response

α-herpes virus particles stimulate local messenger RNA transcription in peripheral nerve axons [22, 23]. The axons react locally when viruses infiltrate them [24]. Following mitochondrial failure and resulting decreased activity of Na/K-ATPase, sodium channel activity and reversal of the sodium-calcium exchanger might therefore contribute to the axonal degeneration of peripheral axons [25]. The abrupt start of BP is caused by these intra-axonal degeneration mechanisms.

AQP-1 Protein role in nerve oedema

AQP-1 protein levels significantly rose from day 9 to day 16 following HSV-1 inoculation in mice with HSV-1-induced facial palsy. The symptoms of facial palsy in mice are consistent with the overexpression of AQP-1, which is also strongly linked to intratemporal facial nerve oedema in the facial nerve canal. Given that phosphorylated ERK is upregulated in mice with HSV-1-induced facial palsy, it is possible that the ERK MAPK pathway contributes to the rise in AQP-1 in the BP animal model [26].

Ischemia

Acute idiopathic lower motor neuron palsy, or BP, is frequently a unilateral inflammatory disorder that resolves on its own [27]. The periosteal membrane covering the vascular plexus that covers the facial nerve's epineurium makes up the outermost layer of the nerve [28]. Peripherally, the stylomastoid artery supplies blood to this vascular plexus, while the anterior inferior cerebral artery, internal auditory artery, and middle meningeal artery (petrosal branch) supply blood to the central layers [21, 29].

1. Primary ischemia

The facial nerve has a robust epineurium and a large blood supply. Primary ischemic neuropathy, on the other hand, is caused by vasospasms, which reduce blood flow and cause severe inflammation [30]. Inflammation of the nerve starts shortly after acute ischemia when macrophages are recruited and

activated [29]. In animal studies, primary ischemia is also shown, with facial nerve paralysis developing 5–15 minutes after the vascular network is blocked [31].

2. Secondary ischemia

Primary ischemia can result in secondary ischemia, when a transudate is produced as a result of arteriole constriction followed by capillary dilatation. By compressing the lymphatic capillaries, this transudate can increase transudate production and cause ischemia. The resulting histamine-mediated oedema may restrict venous outflow, which would disrupt the vascular plexus's regular arterial circulation [21, 28].

3. Tertiary ischemia

Tertiary ischemia, which is characterized by perivasculitis and endarteritis, can develop from secondary ischemia [32]. At this point, the facial nerve sheath frequently exhibits thickening or fibrosis, which may necessitate surgical decompression to avoid irreversible face paralysis [33].

Inflammation

There is now greater proof that inflammation and blood pressure cause the facial nerve to become demyelinated [21].

The facial nerve's histologic alterations include:

- Round, tiny inflammatory cells infiltrate the nerve connecting the internal auditory meatus to the stylomastoid foramen.
- There is a breakdown of the myelin sheaths surrounding neurons, including macrophages.
- More room is created inside neurons.
- There is no indication of compression of the facial nerve, and the bony fallopian canal is normal.

In inflammatory illnesses, a high neutrophil-to-lymphocyte ratio (NLR) is regarded as a trustworthy etiological indication of disease severity [34, 35]. Both adult and juvenile BP patients had considerably higher mean neutrophil and NLR levels than healthy controls [36, 37]. The part played by cell-mediated immune responses, as BP patients' blood levels of TNF-a, IL-6, and IL-8 are greater than those of controls [38].

In addition to the aforementioned mechanism other element, including infection, impaired immunity, pregnancy, acute cold exposure, diabetes, and blood pressure etc. have been suggested. However, the precise underlying mechanism of these factors remain unclear and required further investigation.

Symptoms:

Bell's palsy symptoms can range from modest weakness to severe weakness, which eventually leads to complete paralysis [39]. About half of patients think they have had a stroke, 25% are afraid of a cerebral tumour, and the other 25% are very nervous but don't know what's wrong. Bell's palsy symptoms can sometimes come on suddenly, and other times they might develop one to two weeks after an eye infection, cold, or ear infection. Bell's palsy patients may have drooping on one side of their face and appear agitated while opening the eye on that side (Figure 3).

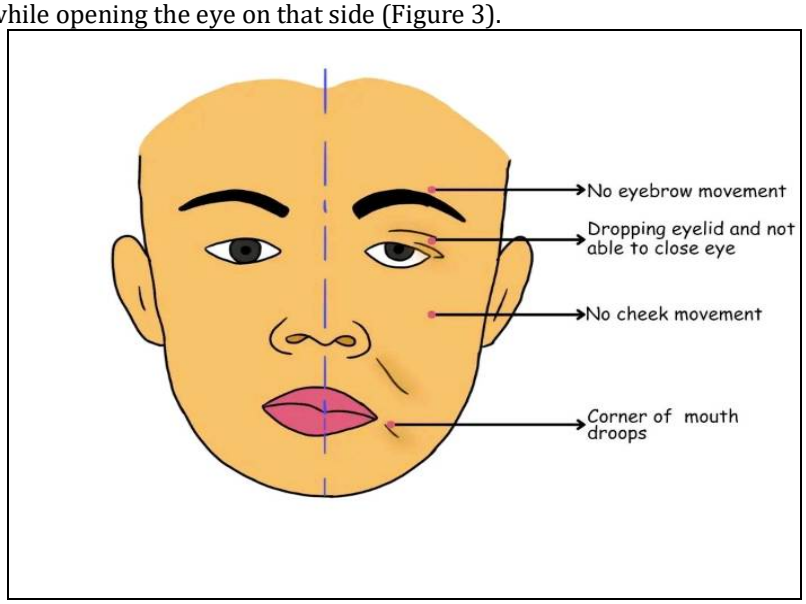

Figure 3: Acute unilateral Bell's palsy symptoms

In rare instances, both sides of the face may be impacted. A drooping mouth, facial weakness, trouble smiling or frowning, trouble pronouncing words, drooling, changed taste, dry mouth and eye, headache,

headache, difficulty eating or drinking, sensitivity to sound, and tinnitus are some other symptoms. Up to 80% of persons with Bell's palsy do not exhibit any symptoms for three to six months. These symptom, however, should never be disregarded because they may resemble a stroke or brain tumour [40, 41].

Clinical examination:

The clinical investigation should involve a comprehensive neurologic and general examination that includes an ophthalmological and ear examination, as well as assessment of the skin and parotid gland [42]. Blisters or scabbing around the ear, a condition known as Ramsay-Hunt syndrome, which can cause hearing loss and facial nerve palsy, may be signs of herpes zoster. Ancillary information and subtle signs of weakness may be revealed by watching the patient during the interview [43].

Bell's palsy patients often go from the beginning of their symptoms to their maximum weakness in three days, and nearly invariably in a week. Rethinking the diagnosis should be prompted by a more subtle beginning or progression that lasts longer than two weeks. Within three weeks of commencement, 85% of patients will exhibit at least a partial recovery if treatment is not received [44, 45]. When evaluating a patient who may have Bell palsy, a methodical approach is advised (as given in fig.4) and described below [46].

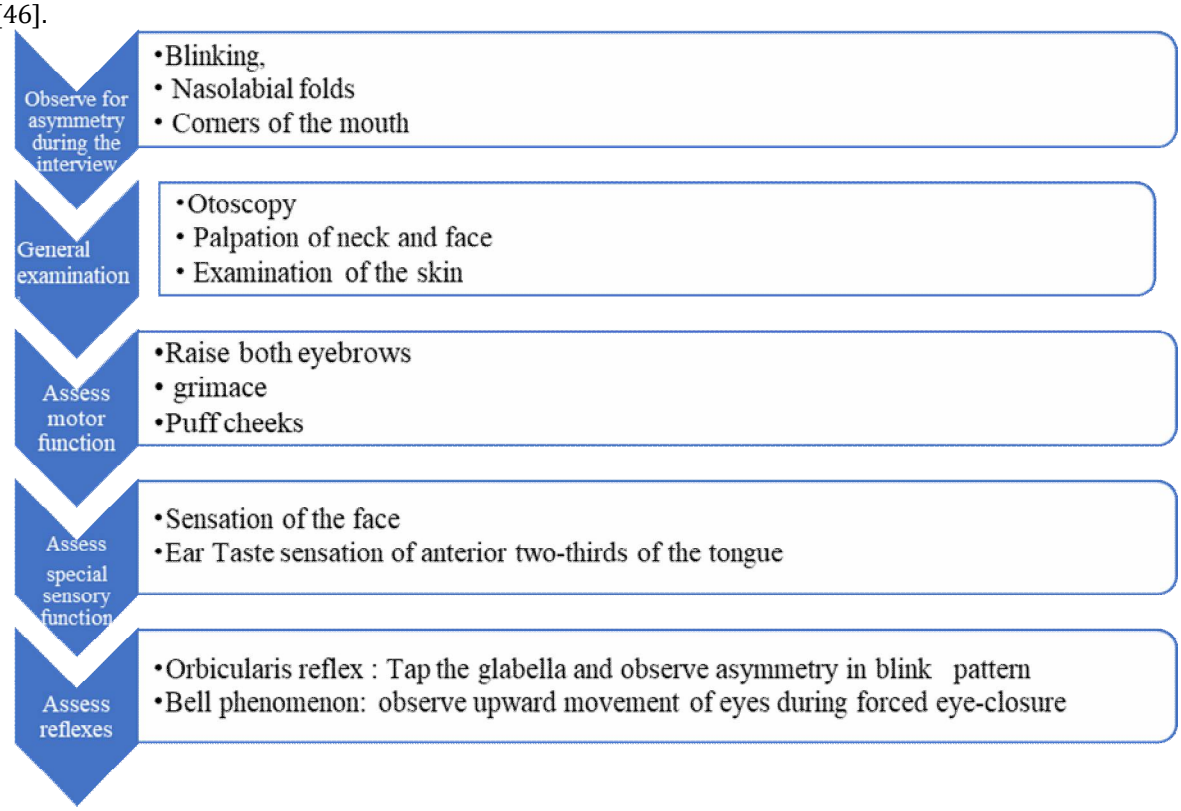

Figure 4: Method for the Bell palsy clinical assessment

Diagnosis:

Neurophysiological ancillary diagnostic studies:

The electro diagnostic test was conducted on the same day as the clinical examination, about two to three weeks following the beginning of facial palsy. However, Tran's cranial canalicular magnetic stimulation may be performed in rare instances where the palsy cannot be accurately categorized as central or peripheral. Blink reflex investigations aid in localizing the lesion in the facial nerve nucleus, pontine fibers, or extra pontine route, whereas canalicular hypo-excitability indicates a peripheral extracerebral origin [47].

The prognosis of facial nerve palsy is predicted using electromyography and electroneurography. As early as 10–14 days following the beginning of symptoms, ENG exhibits axonal damage with a decrease in amplitude, suggesting a favourable prognosis. Potentials produced during attempting muscular contraction are seen on the EMG, indicating nerve recovery and continuity. Axonal injury is indicated by abnormal spontaneous activity on an EMG 10–14 days after the beginning of symptoms, but re-innervation potentials show a favourable prognosis [48].

Imaging studies:
Imaging studies, such as skull x-rays, CT scans, and MRIs, are used to make the diagnosis. By examining the nerves in the face, these tests are carried out to look into the likelihood of a stroke or brain tumor [49]. Although brain MRI is not usually recommended, when it is, the most frequent abnormality observed is contrast enhancement of the facial nerve's distal intracanalicular and labyrinthine segments; the geniculate ganglion, proximal and distal tympanic, and mastoid segments may also be affected. Facial weakness may also result from a central pontine lesion. And is frequently linked to other neurologic symptoms and indicators [50]. It is hypothesized that this imaging result represents vascular congestion of the facial nerve and disturbance of the blood–brain barrier [51].

Laboratory tests:
A differential complete blood cell count might indicate a lymph proliferative disease or an infection. Screening for diabetes mellitus using haemoglobin A1c or fasting blood glucose may be beneficial when needed. Patients in areas where Lyme disease is prevalent should be screened for the illness using an indirect fluorescent antibody test or an enzyme-linked immunosorbent assay. Western blot should be used to confirm the Lyme disease diagnosis if it is positive. Examine serum antibodies for herpes zoster if vesicles are seen. Human immunodeficiency virus, inflammatory markers, and angiotensin-converting enzyme can all be examined in the right clinical situation [46]. Neurologists advise lumbar punctures since they are essential for a precise diagnosis. Treatment may be affected by aberrant CSF findings in 10–40% of patients of idiopathic peripheral facial nerve palsy. Cell count, protein, cytology, lactate, and certain antibodies are important CSF diagnostics. Despite its excellent sensitivity for identifying infections, one should weigh the hazards and other options [52].

Management:
Bell's palsy must be managed in addition to the medicine. The management techniques include.

Eye care
Because of issues with lid closure and the tearing process, Bell's palsy patients' eye treatment focuses on shielding the cornea from drying and abrasion. The patient is trained to report any new symptoms [53].

Oral care:
Oral incontinence and an increased risk of lip and cheek injuries when eating can result from a loss of orbicularis oris sphincter function. Using a straw and selecting soft foods are two examples of strategic eating methods that might be beneficial. Temporary dental spacers on molars can shield the buccal mucosa during chewing, however lower lip problems influence meal choices [8].

Medical Treatment:

Corticosteroid:
Bell's palsy patients have historically been treated with oral corticosteroids to lessen inflammation of the facial nerves. Prednisone and other oral corticosteroids lessen nerve swelling and may hasten the improvement of facial expressions and movements [54]. When this therapy is initiated within 48 hours after symptom onset, it is most effective [55]. For five days, 60 mg of prednisone should be taken orally once day. After that, the dosage should be decreased to 10 mg daily [56]. The percentage of patients with incomplete recovery after six months (relative risk [RR] = 0.86; 95% CI], 0.47 to 1.59) and the percentage of patients with cosmetic complications (RR = 0.86; 95% CI, 0.38 to 1.98) were found to be slightly and statistically not significantly lower in a 2004 Cochrane review and meta-analysis of three randomized controlled trials comparing corticosteroids with placebo. Larger prospective studies are required to prove the benefits of corticosteroids, as these trials only covered 117 individuals [57].

Antiviral:
Given the likely involvement of herpes viruses, antiviral treatment makes sense for Bell's palsy. Acyclovir, a nucleotide analogue, prevents DNA replication by interfering with the herpes virus's DNA polymerase. Due of acyclovir's very low bioavailability (15–30%), 18 novel medications in its family are undergoing clinical trials [58]. In the event of an associated herpes infection, antivirals such as acyclovir can be started at a dose of 400 mg taken orally five times a day and sustained for 10 days [1]. However, the use of these antivirals alone was not supported by enough data, according to a 2004 Cochrane review [59]. Delaying therapy for more than four days after the beginning of symptoms, however, did not result in any advantage (86 percent versus 87 percent) [60].

Surgical treatment:
The facial nerve is decompressed by surgery. Only individuals who have total paralysis and a motor amplitude decrease of more than 90% on a nerve conduction should be evaluated for surgery. However, there are dangers associated with middle fossa craniotomy, such as facial nerve damage, convulsions, deafness, and cerebrospinal fluid leaking. [61].

Physical therapy:
To speed up healing, physical treatments such as massage, electrical stimulation, acupuncture to the afflicted muscles, customized face exercises, and thermotherapy have been employed. Nevertheless, there is no proof of any appreciable advantage [62].

Prognosis:
There are several factors that affect Bell's palsy patients' chances of recovering:

- Recovery rate: improvement within two weeks is indicative of a favourable long-term result [63].
- Complete weakness versus partial weakness: in 94–99% of instances, partial weakness favours complete resolution with full recovery, whereas in 75% of cases, total paralysis is preferred [64].
- Age: the prognosis is often better for younger individuals [65].
- Steroids: starting within 72 hours has been shown to be advantageous [66].

Bell's palsy is diagnosed and treated using a methodical approach in the clinical decision-making process. The distinction between peripheral and central palsy, as well as the proper follow-up procedures, are highlighted in (from Figure 5), which shows a step-by-step flowchart from clinical assessment to several therapy approaches.

Psychosocial and quality of life impact
Many facial activities, such as facial movement, expression, and actions like eating, smiling, and blinking, depend on the operation of the facial nerves.

- In addition to unfavourable self and social evaluations, patients may be more susceptible to anxiety and despair [67].
- People with this illness frequently compare themselves to others, which makes them feel unworthy or unattractive. One's mental health may be significantly impacted by these comparisons, which might engender depressive and loss-related emotions [68].
- Bell's palsy sufferers frequently experience stigma and hurtful remarks, which can worsen the emotional toll and make it more difficult for them to interact with others [69].

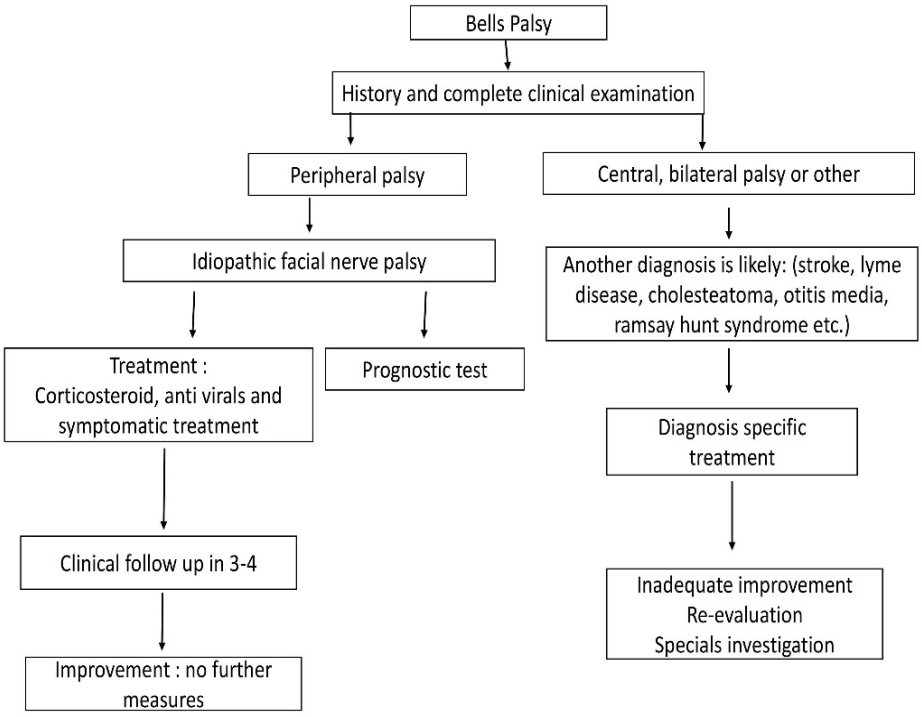

Figure 5: Differentiating Bell's palsy from other neurological condition and decision making from treatment [1]

ADVANCES AND FUTURE DIRECTIONS:

Nerve allografts
Xenografts and cellular or processed allografts are being investigated as possible substitutes for autologous nerve transplants because they can produce an extracellular matrix that is highly ordered and resembles the natural nerve [70]. The FDA has authorized AxoGuard and Avance, two synthetic nerve grafts made by AxoGen. In hand and upper extremity reconstructive surgery, these grafts are becoming more and more common; 70% of hand surgeons in the United States [71] use them to correct nerve

defects (Figure 6.). A prospective clinical investigation to compare the outcomes has not been carried out, and these transplants have not demonstrated better outcomes than autologous tissue [72].

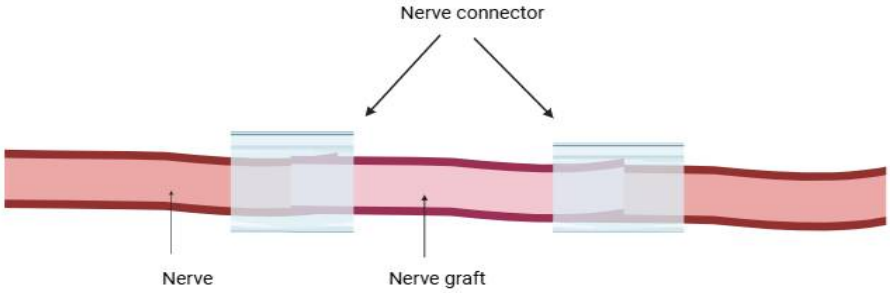

Figure 6. Nerve allograft

Synthetic/Engineered nerve conduits

In cases where facial nerve transections cannot be healed via main procedures, synthetic nerve conduits present an appealing substitute for autologous nerve grafts. These channels improve axonal regeneration by combining extracellular matrix components, support cells, and biological growth and neurotropic agents [73]. Their therapeutic applicability is primarily restricted to digital nerve lesions with a small diameter and short length, and the reported individual outcomes are inconsistently poor yet very diverse [74-76]. Because of their higher cost and worse performance when compared to autografting, synthetic conduits are not frequently utilized in clinical practice [77]. The development of new bioengineered nerve graft substitutes that incorporate methods for promoting nerve regeneration, like Schwann cells and stem cells, controlled release delivery systems of neurotrophic and growth factors, luminal fillers, surface micropatterning and electrospinning techniques, and electroconductive materials will be a key component of nerve conduits in the future [78]. Human olfactory stem cells, stem cells from human exfoliated deciduous teeth, adipose-derived stem cells, bone marrow-derived stem cells, dental pulp cells, and geniciva-derived mesenchymal stem cells are some of the possible adjuncts being used in animal models for investigations into facial nerve regeneration utilizing these experimental designs [79].

OTHER AREAS OF RESEARCH AND THE FUTURE OF TREATMENT

Neuroprosthetic devices

Modern technological advancements in implantable neural and muscular electrodes, signal processing, and integration have produced ground-breaking inventions like cochlear implants for hearing restoration, prosthetic limb design and control, and the hypoglossal nerve stimulator for obstructive sleep apnea. The use of these concepts in facial nerve rehabilitation has led to the development of implanted Neuroprosthetic devices that treat acute facial paralysis by utilizing both functional electric stimulation and closed loop facial pacing. These devices detect movement from the healthy hemiface and stimulate paired muscle contraction on the contralateral side while simultaneously suppressing undesired facial movements through proximal neural blockade [80, 81].

Artificial muscle

A new technology called artificial muscle has been studied as a possible treatment option for people with irreversible facial paralysis in order to restore their face mobility [82]. Subsequent research has examined the biocompatibility and durability of implanted electroactive polymer artificial muscle devices in gerbils [83], as well as eyelid mechanics, including the forces and vectors required to produce optimal eye closure, since Tollefson et al. first proposed the possibility of restoring eye blink through an eyelid sling mechanism using an electroactive polymer [84, 85]. The size of the polymer device required to sufficiently close the eyelids in a human corpse has hindered efforts to further refine Tollefson's prototype artificial eye sling [86].

CONCLUSION

This systematic review focuses on the state of knowledge and treatment for Bell's palsy. Early corticosteroid therapy beginning has been shown to considerably improve recovery results, and antiviral medications may offer additional advantages for certain patient subgroups. Non-pharmacological treatments, such as acupuncture and physical therapy, have potential but need more solid proof from high-caliber studies. The development of targeted therapeutics is constrained by the lack of knowledge regarding the aetiology and pathophysiology of Bell's palsy, despite improvements in treatment. Finding

biomarkers for early diagnosis, improving treatment approaches for severe or recurring cases, and examining the long-term quality of life of those impacted should be the main goals of future study.

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CONFLICT OF INTEREST
All authors declared no conflict of interest.

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