

The diagnosis and treatment of Bell's palsy

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Abstract

Bell's palsy is an idiopathic peripheral nerve disorder involving the facial nerve. It is of rapid onset, and almost always unilateral. It is the most common diagnosis associated with facial nerve palsy. It has been described in patients of all ages, but the incidence is slightly higher after age 40.

Methods: Using internet search, a comprehensive literature review was done and words such as facial nerve palsy, Bell's palsy were searched.

Results: In confirmed Bell's palsy, unless contraindicated, corticosteroids should be given to all patients with Bell's palsy as early as possible, (ideally within 72 hours). Combination therapy with steroids and antiviral agents are recommended for patients with severe to complete paresis. Patients with incomplete eye closure should be given eye protection, with lubricating drops and ointments, to prevent corneal damage.

Conclusion: Establishing the correct diagnosis is imperative to avoid missing another treatable condition. Determining whether the facial nerve paralysis is central or peripheral is important. The history of a Bell's palsy case should include discomfort or sensory symptoms in the distribution of the facial nerve in the hours or days preceding facial palsy, and it is very important to reveal whether the symptoms were progressive in nature.

Although many patients with Bell palsy will experience improvement in their facial nerve function without treatment, persistent facial weakness can have implications for quality of life. Choosing the correct treatment options for suitable patients can optimize the likelihood of recovery. Oral steroids should be prescribed within 72 hours of symptoms onset for Bell's palsy patients. Combined steroid and antiviral treatment are recommended for patients with severe to complete paresis. Physiotherapy may be suggested in severe Bell's palsy. Surgical decompression is not recommended and may be considered in severe facial nerve degeneration on electroneuronography if the patient is willing to accept the surgical risks. Eye protection remains crucial in preventing long-term eye complications. Clinical and ophthalmological follow-up, and referral to a specialist for patients with no improvement or progressive weakness are recommended.

Key words: Facial nerve palsy, Bell's palsy, Glucocorticoids, Antiviral.

Introduction

Bell's palsy is an idiopathic peripheral nerve disorder involving the facial nerve. It is named after Sir Charles Bell, who in 1821 first described the anatomy of the facial nerve (1). It is the most common diagnosis associated with facial nerve weakness/paralysis (2). The annual incidence of Bell's palsy ranges from 11 to 53.3 per 100,000 persons (3,4), with no predilection for sex or ethnicity. It has been described in patients of all ages, but the incidence is slightly higher after age 40 (4,5). Risk factors include diabetes, pregnancy, severe preeclampsia, obesity, and hypertension (6-9). Patients who have had one episode of Bell's palsy have an 8 percent risk of recurrence (5).

Anatomy

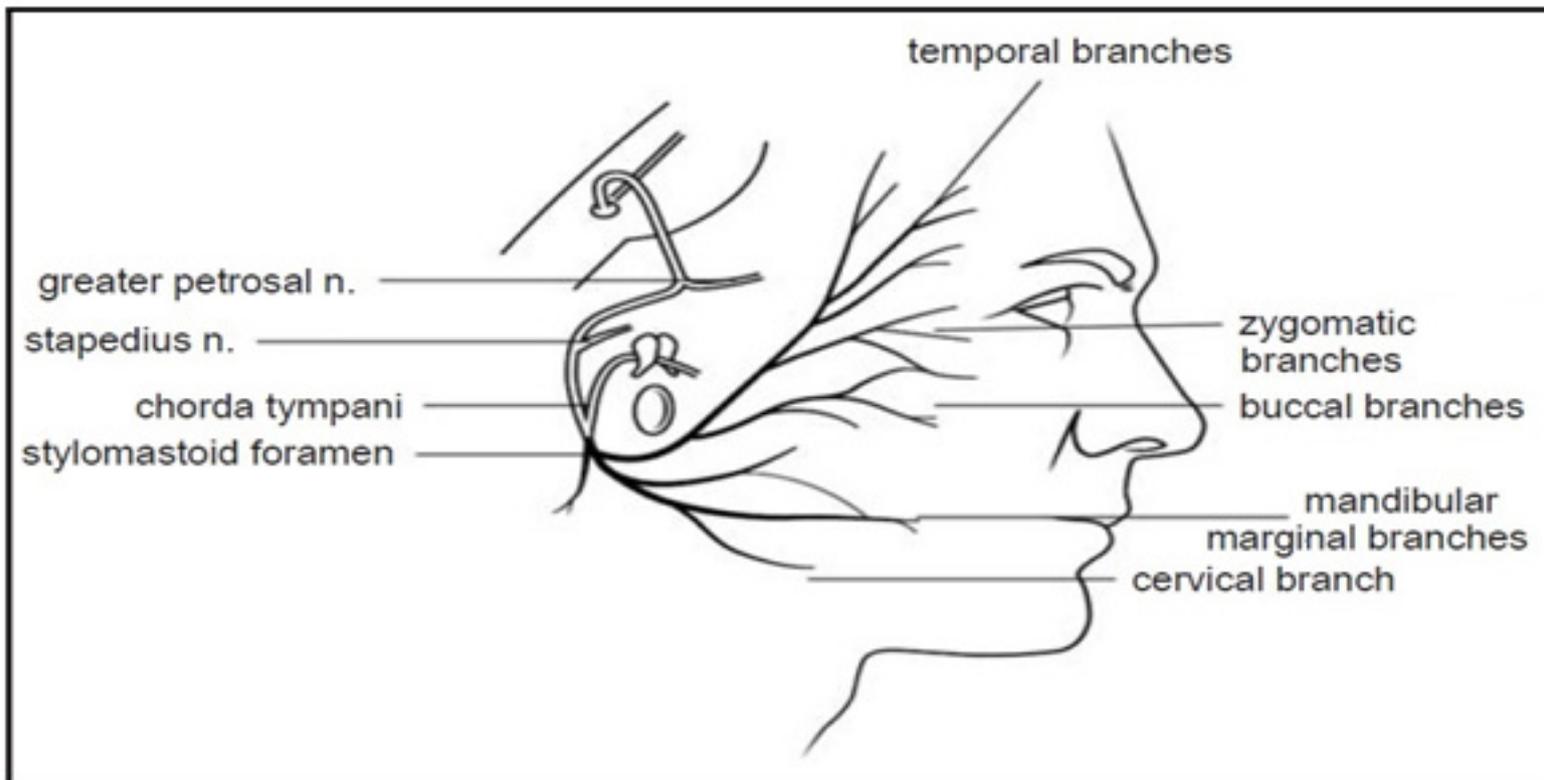
The facial nerve is composed of motor fibers (innervating the muscles of facial expression, posterior belly of digastric, stylohyoid and stapedius), parasympathetic fibers (innervating the lacrimal, submandibular, and sublingual salivary glands), somatic afferents (from the external ear) and afferent taste fibers from the anterior two thirds of the tongue. The facial nerve (motor root) exits the brainstem at the lower aspect of the pons in the cerebellopontine angle and the nervus intermedius (sensory and parasympathetic secretomotor fibers) and emerges between the pons and

the inferior cerebellar peduncle. Both parts of the nerve pass into the internal auditory meatus in the petrous temporal bone, travelling with the VIII nerve until they enter the facial canal. The genu of the facial nerve describes a sharp bend over the promontory of the middle ear where the secretomotor fibers for the lacrimal gland leave via the greater petrosal nerve, and the facial nerve travels inferiorly, in the medial wall of the middle ear cavity. Within the facial canal, the nerve to stapedius and chorda tympani are also given off before the facial nerve enters the stylomastoid foramen. The chorda tympani nerve runs between incus and malleus in the middle ear, entering the infratemporal fossa to join the lingual nerve, carrying taste fibers from the anterior tongue and secretomotor fibers to the submandibular ganglion, supplying the submandibular and sublingual salivary glands (10,11).

The facial nerve has five terminal branches that innervate the muscles of facial expression:

- The temporal branch (muscles of the forehead and superior part of the orbicularis oculi)
- The zygomatic branch (muscles of the nasolabial fold and cheek, eg, nasalis and zygomaticus).
- The buccal branch (the buccinators and inferior part of the orbicularis oculi)
- The marginal mandibular branch (the depressors of the mouth, eg, depressor anguli and mentalis)
- The cervical branch (the platysma muscle). Figure 1

Figure 1: Courses and branches of the facial nerve. The intracranial course is shown as transparent (12).



Clinical characteristics

Clinical features of Bell's palsy include ipsilateral weakness or paralysis of the upper and lower facial muscles of the affected side, drooping of eyelids of the same side, incomplete closure of eye that causes drying of eye, the eye rolls upward (Bell's phenomenon) on attempted closure, epiphora, drooping of the corner of the mouth, altered gustatory sensation on the same side, food and saliva can pool in the affected side of the mouth and may spill out from the corner, altered sensation on the affected side of the face, hyperacusis (sensitivity to sound increases). About 70% of patients have associated ipsilateral pain around the ear. Patients with Bell's palsy usually progress from onset of symptoms to maximal weakness within three days and almost always within one week (2,13). (Figure 2).

The severity of facial nerve palsy is usually rated on the six-point House and Brackmann scale, with grade I corresponding to normal facial nerve function and grade VI corresponding to total paralysis (Table 1) (14). Further rating scales (the Sunnybrook Facial Grading System (FGS) scale) (15) and video documentation are used mainly for follow up of residual weakness and after facial nerve reanimation surgery (16).

Figure 2: Bell's palsy on the left side of the face (17)

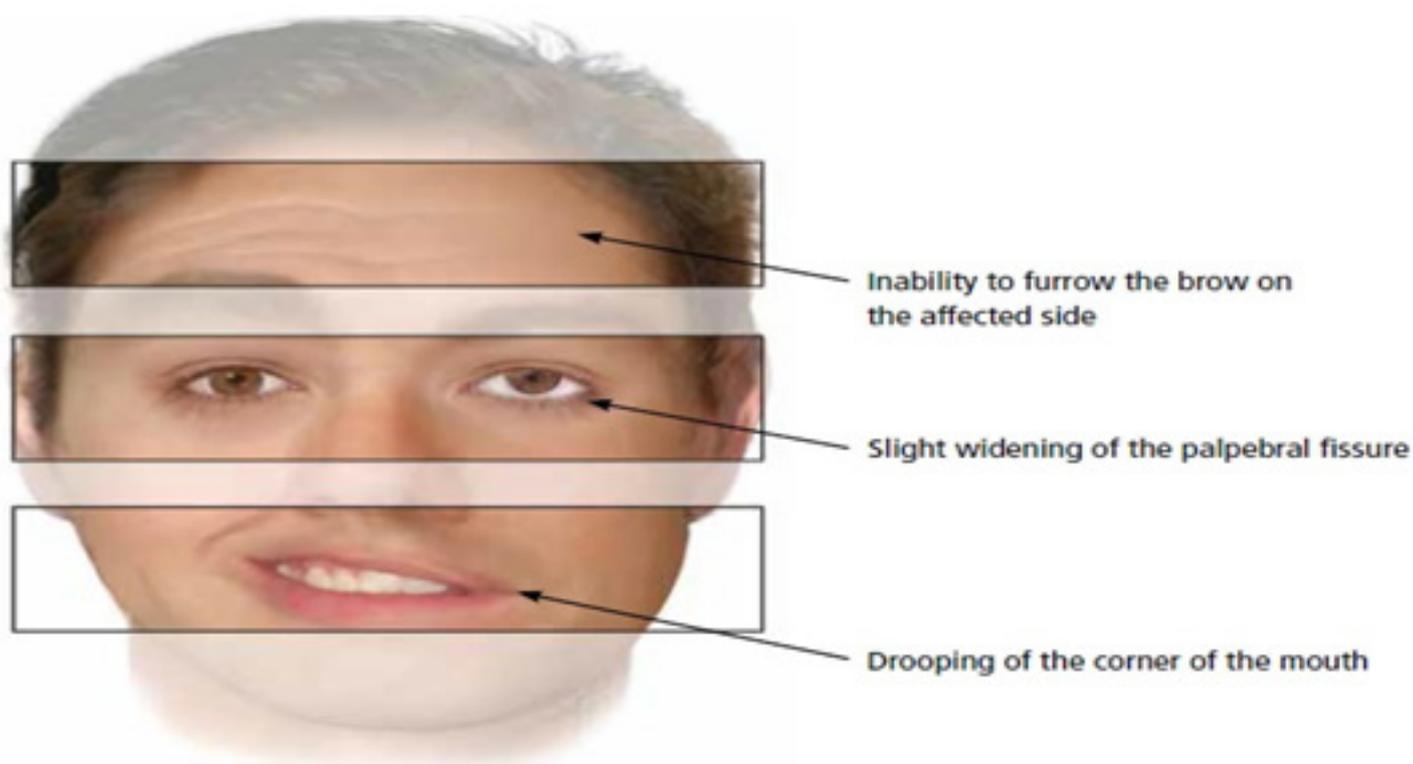


Table 1: House-Brackmann facial nerve grading system

Grade	Description	Findings
I	Normal	Normal facial function in all areas.
II	Mild dysfunction	Slight weakness noticeable only on close inspection. At rest: normal symmetry of forehead, ability to close eye with minimal effort and slight asymmetry, ability to move corners of mouth with maximal effort and slight asymmetry. No synkinesis, contracture, or hemifacial spasm.
III	Moderate dysfunction	Obvious, but not disfiguring difference between two sides, no functional impairment. noticeable but not severe synkinesis, contracture, and/or hemifacial spasm. At rest: normal symmetry and tone. Motion: slight to no movement of forehead, ability to close eye with maximal effort and obvious asymmetry, ability to move corners of mouth with maximal effort and obvious asymmetry. Patients who have obvious but no disfiguring synkinesis, contracture, and/or hemifacial spasm are grade III regardless of degree of motor activity.
IV	Moderately severe dysfunction	Obvious weakness and/or disfiguring asymmetry. At rest: normal symmetry and tone. Motion: no movement of forehead; inability to close eye completely with maximal effort. Patients with synkinesis, mass action, and/or hemifacial spasm severe enough to interfere with function are grade IV regardless of motor activity.
V	Severe dysfunction	Only barely perceptible motion. At rest: possible asymmetry with droop of corner of mouth and decreased or absence of nasal labial fold. Motion: no movement of forehead, incomplete closure of eye and only slight movement of lid with maximal effort, slight movement of corner of mouth. Synkinesis, contracture, and hemifacial spasm usually absent.
VI	Total paralysis	Loss of tone; asymmetry; no motion; no synkinesis, contracture, or hemifacial spasm.

Etiology

Despite its idiopathic status, several theories on the underlying etiology of Bell's palsy have been proposed, including viral reactivation, such as the varicella zoster virus (VZV) (18), herpes simplex virus type 1 (HSV-1) (19), and human herpes virus 6 (20). Isolated facial paralysis after vaccination has been reported with almost all viral vaccines, and it is thought to be immune mediated or induced by viral reactivations (eg, reactivation of a herpes virus infection) (21). However, studies have failed to identify a higher risk of facial paralysis after vaccination (21,22).

Other theories suggested that Bell's palsy results from ischemia (23,24), and inflammation of the facial nerve (A high neutrophil-to-lymphocyte ratio (NLR) (25,26) and decreased percentages of total T cells (CD3) and T helper/inducing cells (CD4) have

been found in patients with Bell's palsy (27)). And several studies found a correlation between the cold season and the number of Bell's palsy cases (28-30).

Recently, there is ongoing public concern regarding the possible adverse effects of SARS-CoV-2 immunization. Two clinical trials of these vaccines reported seven cases of Bell's palsy in the vaccinated group. The US Food and Drug Administration (FDA) did not consider there to be a clear basis on which to conclude a causal relationship. Therefore, the FDA recommended further surveillance of these vaccines as they have been authorized for widespread emergency use (31,32). One letter reported that the observed incidence of Bell's palsy in the mRNA vaccine groups was 1.5 - 3 times higher than would be expected in the general population (33). Other research

Table 2: Etiologies and clinical features of facial paralysis. (2, 40-42)

Differential diagnosis	Cause	Distinguishing characteristics
Idiopathic	Unknown	Classic Bell's palsy with other etiologies excluded
Central nervous system lesion		
Stroke	Ischemia, hemorrhage	Forehead sparing, headache, limb weakness, multiple neurologic signs
Tumor	Metastases, primary brain	Gradual onset; mental status changes. history of cancer
Autoimmune diseases	Guillain-Barré syndrome Multiple sclerosis	Ascending weakness, absent reflexes Upper motor neuron signs, abnormal cerebrospinal fluid
Metabolic diseases	Diabetes (Microvascular disease)	Elevated blood glucose
Infectious diseases		
Meningitis, encephalitis	Viral, bacterial, fungal pathogen	Headache, fever, meningeal signs, abnormal cerebrospinal fluid
Herpes simplex	Reactivation of herpes simplex virus type 1 from geniculate ganglion	Fever, malaise
Lyme disease	Borrelia burgdorferi	Rash, arthralgia, malaise, bilateral facial palsy
Ramsay Hunt syndrome	Varicella zoster	Pain, vesicular eruption
Otitis media	Bacterial pathogens	Gradual onset; ear pain, fever, and conductive hearing loss
Granulomatous disease	Sarcoidosis	Bilateral facial palsy, elevated angiotensin- converting enzyme
Neoplasm	Parotid tumor, facial nerve tumor, metastasis	Insidious onset, palpable mass, partial involvement of facial nerve branches
Trauma	Surgery, basal skull fracture, facial trauma	assess for scars, bruising, blood

reported no association between facial paralysis and mRNA COVID-19 vaccines when compared with other viral vaccines in a disproportionality analysis (34). Another study reported an overall increased risk of Bell's palsy after CoronaVac vaccination but not after BNT162b2 vaccination (35). And one study concluded that patients with COVID-19 have a greater risk of acquiring Bell's palsy than those who were vaccinated against the disease (36). Further long-term analysis is necessary to determine the relationship between COVID-19 and the COVID-19 vaccine on Bell's palsy (35).

Diagnosis

The diagnosis of Bell palsy is made by excluding other causes of unilateral facial paralysis (Table 2). It is the responsibility of the evaluating clinician to conduct an appropriate patient history and to examine the patient with the specific intent of finding an underlying cause. Determining whether the facial nerve paralysis is central or peripheral is therefore key to diagnosis. Central lesions will cause paralysis of the lower face alone, sparing the forehead; however, clinicians must ensure they ask about the duration and nature of symptoms including the presence of associated symptoms such as hyperacusis, posterior auricular pain, taste, and lacrimal changes in their history (37,38). The timing and progression of Bell's palsy helps distinguish it from an acute cause such as stroke (Table 3). A key feature is the progressive nature of Bell's palsy, which can be elucidated by detailed history taking (39). A full cranial nerve examination as well as ocular, otologic and oral examinations must be carried out in all patients presenting with a facial palsy.

Laboratory testing & electrodiagnostic testing

The AAO-HNSF (American Academy of Otolaryngology—Head and Neck Surgery Foundation) guidelines recommend against the use of routine laboratory testing for new-onset Bell's palsy. This is due to low detection rates for the herpes simplex virus, or varicella zoster virus, even with the use of polymerase chain reaction, enzyme-linked immunosorbent assay (ELISA), western blot, and cerebrospinal fluid tapping (39). In Lyme endemic regions, patients should undergo an enzyme-linked immunosorbent assay or an indirect fluorescent antibody test to screen for the disease. If positive, the diagnosis of Lyme disease should be confirmed by Western blot (17). Because diabetes mellitus is present in more than 10 percent of patients with Bell's palsy, fasting glucose or A1C testing may be performed in patients with additional risk factors (e.g., family history, obesity, older than 40 years) (44). Cerebrospinal fluid analysis is generally not helpful in diagnosing Bell's palsy but can differentiate it from Guillain-Barré syndrome, leptomeningeal carcinomatosis, and infection involving the central nervous system (17).

Diagnostic imaging

The AAO-HNSF guidelines recommend against routine diagnostic imaging for new-onset Bell's palsy (39). Also, the Spanish Society of Otolaryngology (SEORL) guidelines recommend against imaging tests when the clinical symptoms indicate Bell's palsy or herpes zoster

paralysis (48). The Canadian guidelines (Bell Palsy Working Group, Canadian Society of Otolaryngology – Head and Neck Surgery and Canadian Neurological Sciences Federation) advocate imaging to rule out neoplasm in patients with no improvement or progressive weakness after treatment (49). French Society of ENT (SFORL) guidelines recommended that in suspected Bell's palsy, contrast-enhanced MRI should be used to study the course of the facial nerve, including the parotid portion, within 1 month if possible, to contribute to positive diagnosis and rule out tumoral etiology. In the absence of the usual favorable progression, imaging should be repeated at 6 months (50).

Treatment

The treatment of Bell palsy focuses on maximizing recovery and minimizing associated complications (17).

Glucocorticoids

The current guidelines of the American Academy of Neurology, updated in 2012, state, "For patients with new-onset Bell palsy, steroids are highly likely to be effective and should be offered to increase the probability of recovery of facial nerve function (51). The AAO-HNSF guidelines strongly recommend the prescription of oral steroids within 72 hours of symptom onset for Bell's palsy cases aged 16 years or older. Two treatment regimens have been endorsed: either (prednisolone 50 mg for 10 days or prednisone 60 mg for 5 days with a 5-day taper) initiated within 72 hours of symptom onset. The benefit of treatment after 72 hours is less clear (39). The Canadian guidelines recommend the use of corticosteroids for all patients with Bell palsy (49). French Society of ENT (SFORL) guidelines recommend the prescription of corticosteroids at 1 mg/kg/day for 7–10 days. And in severe forms (HB grade V or VI), 10 days of high dose corticosteroids (2 mg/kg/day) seem justified unless contraindicated (50). Corticosteroids initiated within 3 days of facial palsy onset in adults increase the likelihood of recovery, shorten the time to recovery, and reduce synkinesis (involuntary movements) (52-55).

Treatment of Bell's palsy with oral corticosteroids is not without risk. Known side effects of oral corticosteroid use include gastrointestinal disturbances, loss of control of glucose levels, reactivation of peptic ulcer disease, elevated blood pressure, peripheral edema, and mood swings or episodes of acute psychosis (39).

Antiviral therapy & combined antiviral-steroid treatment

The AAO-HNSF, SFORL and the Canadian guidelines strongly recommend against antiviral treatment alone for new-onset Bell's palsy (39,49,50).

According to the AAO-HNSF guidelines, the clinician may use a combined treatment within 72 hours of symptom onset for Bell's palsy (39). The AAN guidelines also state that combined treatment may be offered to increase the probability of the recovery of facial function (51). However, the latter also mentions the necessity of counselling

Table 3 - summarizes the common differences between Bell's palsy and acute stroke (43).

	Bell's palsy	Acute stroke
Age, years	30–50	Usually >60
Symptom time course	Progressive; over hours or days	Sudden; over seconds
Unilateral facial paralysis	Yes	Yes
Upper face	Always affected	Usually not affected
Lower face	Always affected	Affected
Ability to close eye on symptomatic side	Not likely	Likely
Ear or TMJ area pain	Likely	Not likely
Hyperacusis	Likely	Not likely
Decreased lacrimation, salivation or change in taste	Likely	Not likely
Pupils affected	Not likely	Sometimes
Arm or leg weakness	Not likely	Likely
Speech or vision affected	Not likely	Likely
TMJ = temporomandibular joint.		

regarding the modest effects of potential benefits of additional antivirals and steroids (51). In the Canadian guidelines, combined treatment is recommended only for patients with severe to complete paresis (49). And the French guidelines, in severe Bell's palsy treated early (within 72 hours), antiviral treatment should be associated to corticosteroids (50). Recommended antiviral treatment dosage is Valcyclovir 1 g three times daily for 7 days or Acyclovir 400 mg five times daily for 7 days (17, 56). Combination therapy with steroids and antiviral agents resulted in significantly higher favorable recovery rates than steroids alone in severe Bell's palsy patients. Combination therapy was particularly more effective than steroids alone in patients aged ≥ 40 years and in patients without hypertension and diabetes (57,58).

The most observed side effects of antiviral therapy are gastrointestinal related and include nausea, vomiting, and diarrhea, with rare severe reactions, including hives, bronchospasm, angioedema, and hepatic or renal failure (39).

Acupuncture and physical therapy

The AAO-HNSF state that no recommendation can be made regarding the effect of acupuncture in Bell's palsy patients (39). Also, the French Society of ENT (SFORL) guidelines recommend against the use of acupuncture in Bell's palsy patients (50). In addition, a recent meta-analysis on acupuncture reported insufficient evidence to support the efficacy and safety of acupuncture due to the poor quality and the heterogeneity of relevant research (59).

In the AAO-HNSF guidelines, no recommendations are made in relation to physical therapy other than that physiotherapy for acute illness is specifically not recommended (39). The Canadian guidelines make no recommendation regarding the use of exercise physiotherapy for acute Bell palsy of any severity. However, they suggest exercise physiotherapy for patients with persistent weakness (49). The French Society of ENT (SFORL) guidelines recommend the treatment by a rehabilitation specialist (speech therapist or physiotherapist, with relevant qualifications) in severe Bell's palsy or in case of factors for poor recovery (50). Also, the Spanish Society of Otolaryngology (SEORL) guidelines recommend rehabilitative physical therapy in severe Bell's palsy to improve sequelae that have developed and help ensure they are less severe if the physical therapy is provided at the right time (48).

The French Society of ENT (SFORL) guidelines and the Canadian guidelines recommended against the use of electrotherapy in Bell's palsy patients (49,50).

Surgical decompression

The AAO-HNSF recommend against surgical decompression for patients with Bell's palsy (39).

On the other hand, the French guidelines state that further well-conducted studies should be performed before declaring such treatment ineffective, and make recommendation that in severe forms, decompression should be performed early to avoid irreversible nerve injury, and if ENMG shows $> 90\%$ degeneration, decompression should be performed rapidly (50). If surgical facial nerve decompression is implemented, it must include the meatal

foramen, the labyrinthine segment, the beginning of the second part of the nerve, and the geniculate ganglion. A transmastoid or a supratemporal approach may be used, but the latter is the gold standard (50).

The Canadian guidelines recommend against the routine use of surgical decompression. They state that patients should consider this option only if they have severe facial nerve degeneration on electroneuronography, if they are willing to accept the surgical risks and if the surgery is to be performed in an advanced treatment facility (49). Surgical decompression has potentially serious risks, including hearing loss (3%–10% of patients), further damage to the facial nerve and leaks of cerebrospinal fluid (4%) (60,61).

Eye care

The main functional complications in Bell's palsy are ophthalmologic: inadequate lubrication or hydration of the cornea can lead to exposure keratitis, corneal ulceration and eventually loss of vision (39, 48-50).

The AAO-HNSF, the Spanish Society of Otolaryngology (SEORL), French Society of ENT (SFORL) and the Canadian guidelines strongly recommend that eye care should be implemented for the treatment of Bell's palsy (39,48-50). It is critical to recommend supportive eye care for all Bell's palsy patients with incomplete eye closure. Initially, eye drops, protective gel or artificial tears several times daily and especially at night should be used in patients with incomplete eye closure (39,48, 50, 56). Patients should be educated about strategies for eye closure (ie, taping), moisturization (ie, eye ointment, artificial tears, humidified eye chambers) and wearing sunglasses with side protection to avoid irritation from the sun's rays and dust (39, 48, 56). The presence of ocular symptoms such as pain, irritation, or itching should prompt an expeditious referral to an ophthalmologist to prevent corneal damage (39, 50). A detailed ophthalmologic evaluation should be done for patients who fail supportive eye care or patients with severe and persistent lagophthalmos. Recommended eye treatments in those patients may include the use of botulinum toxin injections, or temporary or permanent tarsorrhaphy or lid loading with gold-weight, autogenous temporalis fascia and platinum chains (62-65).

Treatment of Bell's palsy in challenging cases

Diabetes mellitus

There is only limited available evidence on the treatment of facial palsy in diabetic patients because such patients were excluded from most of the relevant clinical trials. In one clinical trial, diabetic patients with Bell's palsy had a higher rate of complete recovery of facial weakness if they were treated with glucocorticoids (97% vs. 58%) and many patients under diabetic therapy kept their diabetes under good control (66). In another study, diabetic patients were found to have more severe facial weakness at first, but similar outcomes at six months (6).

The AAO-HNSF state that diabetic patients with Bell's palsy should be treated with steroids on an individualized basis (39).

Children

Several studies indicate that the prognosis of untreated Bell's palsy in children is better and that children show higher rates of spontaneous recovery than do adults; therefore, the potential benefit of corticosteroid treatment is inconclusive (67).

Despite the absence of quality trials supporting steroid use in children, given the presumed similar disease process of Bell's palsy in adults and children, as well as the generally favorable benefit-harm ratio of steroid therapy, the AAO-HNSF state that oral steroids may be considered in pediatric patients with a large role for caregiver involvement in the decision-making process (39). For children, the recommended dose of prednisone or prednisolone is 2 mg/kg for 10 days (56).

Pediatric Bell's palsy patients were not included in the antiviral trials, and therefore there is no evidence supporting the use of antiviral therapy alone in pediatric patients with Bell's palsy. And the AAO-HNSF made no comment regarding whether a steroid and antiviral combination might be recommended (39). Several studies indicate that the combination therapy of acyclovir plus prednisolone is not superior to prednisolone alone except in severe cases (68,69).

Pregnancy

Most cases of Bell's palsy in pregnancy occur during the third trimester or the first week after childbirth and, in general, prognosis is worse than in non-pregnant women (70,71).

The AAO-HNSF state that pregnant women should be treated on an individualized basis with oral steroids (39). The Spanish Society of Otolaryngology (SEORL) recommended that prednisolone or methylprednisolone be used, since they cross the placental barrier to a lesser extent (48). The use of antivirals is controversial, and the risk-benefit ratio should be assessed considering the possibility of herpes zoster infection (48). The AAO-HNSF state that pregnant women should be treated on an individualized basis with combination antiviral therapy (39). The treatment must be coordinated with the patient's obstetrician (48).

Treatment of Bell's palsy during the COVID-19 pandemic

The AAO-HNSF published a statement on Bell's palsy treatment during the COVID pandemic. The recommendations are:

In patients who develop Bell's palsy shortly after vaccination: Corticosteroid therapy with or without anti-herpes viral therapy (acyclovir, valacyclovir) is recommended. Patients should be counseled that the effect of corticosteroids on the safety and efficacy of Pfizer-BioNTech or Moderna vaccines is currently unknown. However, immunocompromising conditions and the use of immunocompromising medications are not contraindications to vaccination with either of these vaccines.

• In unvaccinated patients who develop Bell's palsy: Corticosteroid therapy with or without anti-herpes viral therapy (acyclovir, valacyclovir) is recommended. Patients may proceed with vaccination while being treated for Bell's palsy with corticosteroids versus delaying vaccination until after completion of corticosteroid course, in discussion with their healthcare provider (72).

Patient follow-ups

The AAO-HNSF guidelines recommend that clinicians should reassess or refer to a facial nerve specialist those Bell's palsy patients with (1) new or worsening neurologic findings at any point, (2) ocular symptoms developing at any point, or (3) incomplete facial recovery 3 months after initial symptom onset (39). Similarly, the Canadian guidelines recommend referral to a specialist for patients with no improvement or progressive weakness (49).

The French Society of ENT (SFORL) recommend clinical and ophthalmological follow-up for several months after recovery begins, to ensure against complications, and ophthalmic complications in particular (50).

Prognosis

About 71% of patients with Bell's palsy have motor function recovery completely within 6 months without treatment (2).

Poor prognostic factors include elderly patients, severe palsy at presentation, degeneration on ENoG, hypertension, diabetes mellitus, and impairment of taste (73-77).

13% of patients may have a mild residual paresis (facial asymmetry) that is not distressing, while 4% have severe residual paresis (78). Facial synkinesis is due to aberrant nerve regeneration and occurs in 15% to 20% of patients after recovery from Bell's palsy with 6.6% of patients developing moderate-to-severe synkinesis (79,80). Patients may describe tearing while chewing ("crocodile tears"), which occurs in 3.3% of patients with Bell's palsy after approximately 6 to 9 months (81,82). Facial synkinesis and crocodile tears can be treated with botulinum toxin injection (83,84).

Conclusion

The symptoms of Bell's palsy vary from mild to severe. The etiology of Bell's palsy is still unclear.

Establishing the correct diagnosis is imperative to avoid missing another treatable condition. Determining whether the facial nerve paralysis is central or peripheral is important. The history of a Bell's palsy case should include discomfort or sensory symptoms in the distribution of the facial nerve in the hours or days preceding facial palsy, and it is very important to reveal whether the symptoms were progressive in nature.

Although many patients with Bell palsy will experience improvement in their facial nerve function without treatment, persistent facial weakness can have implications for quality of life. Choosing the correct treatment options for suitable patients can optimize the likelihood of recovery.

Oral steroids should be prescribed within 72 hours of symptoms onset for Bell's palsy patients (39,49,50). Combined steroid and antiviral treatment are recommended for patients with severe to complete paresis (39,49,50). Physiotherapy may be suggested in severe Bell's palsy (49,50). Surgical decompression is not recommended and may be considered in severe facial nerve degeneration on electroneuronography and if the patient is willing to accept the surgical risks (49,50). Eye protection remains crucial in preventing long-term eye complications (39, 48-50). Clinical and ophthalmological follow-up, and referral to a specialist for patients with no improvement or progressive weakness are recommended.

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