

Facial Palsy: From Diagnosis to Treatment: A Comprehensive Review

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Abstract: Facial palsy is the most common mononeuropathy that causes acute unilateral partial or complete paralysis of face. The condition may be associated with numerous signs and symptoms, including post-auricular pain, drooping of the eyelid, loss of taste sensation and decreased lacrimation. It can be idiopathic, due to inflammation, infections, trauma, and neoplastic. Early diagnosis and identification of the underlying cause play a crucial role in managing this condition effectively; however, treatment reduces cases of incomplete recovery and entails the use of corticosteroids, with a possible role for antivirals if a viral etiology is suspected. For patients with incomplete recovery, long-term complications like esthetic, physiological and psychological implications, which greatly affect the quality of life. This article aims to provide a concise overview of the existing literature regarding the causes, diagnostic approaches, and therapeutic strategies for facial palsy.

Keywords: Bells palsy, Facial paralysis, Facial nerve

1. Introduction

The human face functions as the primary means of conveying emotions and facilitating communication¹. Expressing emotions through facial expressions involves the coordinated contraction of facial muscles. However, when there is damage to the facial nerve, it can lead to substantial alterations in how these expressions are generated². The seventh cranial nerve, known as the facial nerve, encompasses motor, sensory, and parasympathetic (secretomotor) nerve fibers, serving as the source of innervation for various areas in the head and neck region.³ This nerve plays a crucial role in essential functions like speech, smiling, and conveying emotions. Consequently, any impairment or degeneration of the facial nerve can have a profound impact on individuals' ability to engage in social interactions and express themselves in society⁴.

Facial paralysis (FP) is characterized by either a complete or partial impairment of the facial mimic muscles due to injury to the motor fibers of the facial nerve⁵. It is termed central facial paralysis⁶ in the proximal part of the pons nuclei and peripheral facial paralysis⁶ in the distal part⁵.

Approximately 50% of facial palsies are of unknown origin, often referred to as Bell's palsy, and the remaining result from various factors, such as iatrogenic or traumatic injuries, head and neck neoplasms, Infections, and congenital birth defects⁶.

2. Etiology

The causes for facial nerve paralysis can be broadly divided into idiopathic, traumatic, infectious, and neoplastic⁷.

Idiopathic

The idiopathic facial nerve paralysis can be Bell's palsy and Melkersson-Rosenthal syndrome¹. Bell's palsy is the most common cause of acute unilateral facial nerve paralysis⁸. It was first described in the early 19th century by Sir Charles Bell⁹. Even though it is considered to be idiopathic, although

reactivation of latent herpes virus infection in the geniculate ganglion can be the leading suspected cause of Bell's palsy⁸. It is typically sudden in onset, unilateral, and resolves spontaneously within 6 months⁷.

Traumatic

The second most etiological cause for facial nerve paralysis are traumatic injuries which comprises 8–22% of cases⁷. The possible causes can be basal skull fractures, birth traumas, facial injuries and otitic barotrauma¹.

Infectious

It may occur as a result of Ramsay Hunt syndrome, Lyme disease, Tuberculosis, Otitis media, Leprosy, Cat scratch fever, and Dengue fever, as well as viral infections like Human Papilloma Virus (HIV), polio, mumps, cytomegalovirus, and mononucleosis⁷. Infections of the middle ear and external auditory canal and mastoid can also result in facial nerve paralysis⁸.

Neoplastic

The facial palsy can be caused by malignancies [2.2 – 5%] such as parotid malignancies, facial and acoustic neuromas, meningioma, and arachnoid cysts¹⁰. Tumor-related facial paralysis may be classified as: primary and secondary⁸.

Miscellaneous Causes

Congenital

Facial palsy present at birth may be caused by external or environmental factors present before birth or due to a developmental defect¹¹. The most common external factor is birth trauma or prenatal compression¹¹. Traumatic as in cases of high birth weight, forceps delivery, prematurity, or birth by cesarean section¹⁰. The congenital facial palsy caused by birth trauma is mostly transient and usually resolves within 2 years¹¹.

The syndromes associated with facial nerve paralysis are Melkersson-Rosenthal Syndrome, Albers-Schonberg Disease (Osteopetrosis), Mobius Syndrome and Goldenhar Syndrome (Oculoauriculovertebral Dysplasia)¹².

Volume 13 Issue 1, January 2024

Fully Refereed | Open Access | Double Blind Peer Reviewed Journal

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Neurologic causes:

It includes such as, Multiple Sclerosis, Myasthenia Gravis, Guillain-Barre Syndrome, Hereditary Hypertrophic Neuropathy, Melkersson-Rosenthal syndrome, Moebius syndrome and Cerebrovascular accident

Systemic & Metabolic causes:

It includes Diabetes mellitus, Hyperthyroidism, Hypertension, Pregnancy, Acute Porphyria, Autoimmune Syndrome, Sarcoidosis, Amyloidosis, Carbon Monoxide

Toxicity, Tetanus, Diphtheria, Vitamin A Deficiency, Ethylene Glycol Ingestion and Alcoholism

3. Types**Central facial palsy**

Central facial palsy arises due to upper motor neuron damage, frequently occurring as a consequence of acquired brain injuries, particularly in stroke patients¹³. The features of central palsy include as shown in the figure below¹³:

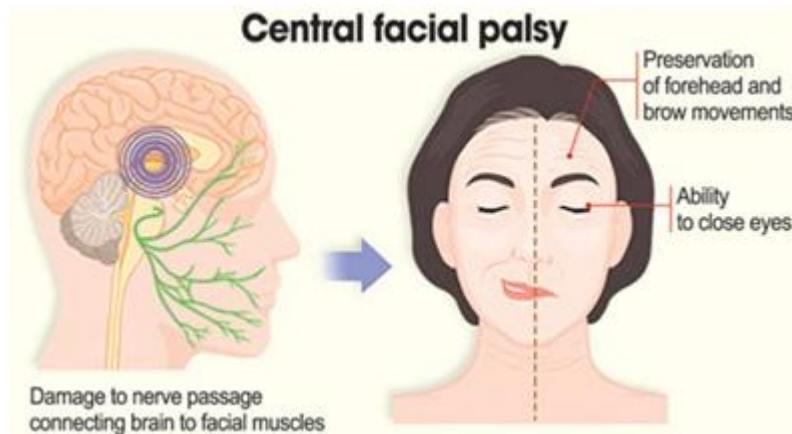


Figure 1: Features of Central Facial Palsy

Peripheral facial palsy

Peripheral facial palsy occurs due to damage to the lower motor neurons, resulting in weakness on the same side of the face in both the upper and lower regions, and it can also lead to synkinesis during voluntary facial movements¹⁴. The

primary cause is often idiopathic, however it can also result from conditions like Guillain-Barré Syndrome or certain infectious diseases, including Varicella Zoster, Lyme disease, and COVID-19¹⁴. The features of peripheral palsy are as shown in figure below¹³:

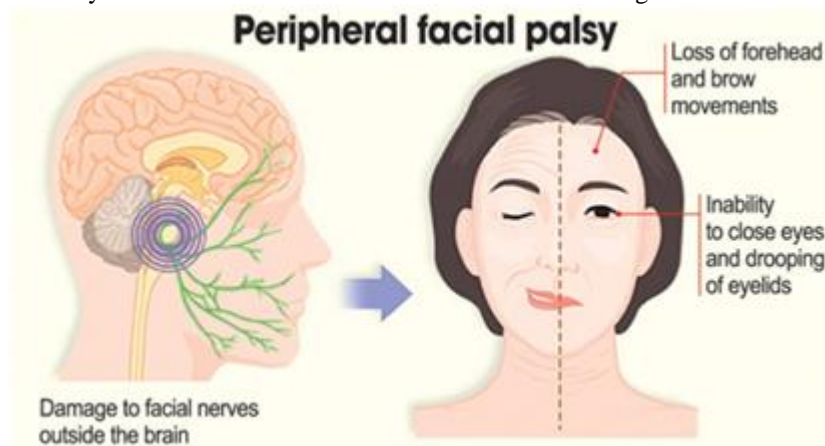


Figure 2: Features of Peripheral Facial Palsy

4. Clinical Presentation

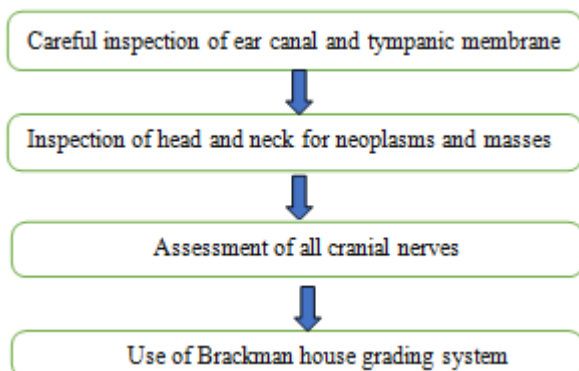
The clinical presentation varies from person to person and range in severity from mild weakness to total paralysis¹⁵. Patients with facial palsy typically complain of weakness or complete paralysis of all the muscles on one side of the face¹⁶. The patient will be unable to raise the eyebrow or tightly close the eyelid on the affected side. The nasolabial fold is typically absent, and the mouth may be drawn toward the unaffected side. Patients may drool from the affected side because of inability to keep the mouth closed¹².

The patients with Herpes simplex virus, and Lyme disease may have associated pain and skin changes with facial

palsy¹⁷. In Ramsay hunt syndrome, there will be a triad of facial nerve palsy, otalgia or vertigo, and vesicles in the ipsilateral external ear, palate or anterior tongue.¹⁸ In Lyme disease it includes erythema migrans, joint pain, fever, fatigue or neck stiffness in association with unilateral or bilateral facial nerve palsy¹⁸. The Patients suffering from a parotid gland tumor or viral otitis may experience facial palsy with sensory and hearing impairment¹⁷. Temporal bone fractures can lead to facial nerve palsy as well as conductive or sensorineural hearing loss, disequilibrium, vertigo and CSF leakage with post-auricular bruising, Battle's sign and hemotympanum¹⁸.

5. Clinical Evaluation

A thorough medical evaluation, including a detailed examination of both the ear, nose, and throat area (otolaryngological) and the nervous system (neurological), should be conducted for all individuals experiencing congenital or acquired facial nerve palsy¹². When facial nerve palsy is observed either at birth or shortly afterward, it's important to inquire about any incidents of prolonged labor, the use of forceps during delivery, or the presence of facial and periauricular ecchymosis around the time of childbirth or shortly². A clinical examination algorithm for the diagnosis of facial palsy is described¹⁹



Ear Examination:

It is essential to conduct a thorough ear examination to rule out any indications of conditions such as otitis externa, otitis media, chronic otitis media, or cholesteatoma.

Parotid Examination:

Examine for the presence of any masses that may suggest a parotid malignancy. Additionally, it is crucial to assess the oral cavity for parapharyngeal swellings and vesicular eruptions.

Eye Examination:

Examine the eye to assess the ability of the lid to close properly. If complete eyelid closure cannot be achieved, it is advisable to urgently refer the patient to an ophthalmologist and provide them with eye protection equipment³

Additional tests which can be carried out are;

- Blink test (corneal reflex) – when tapping on the patient's glabella, a delayed or reduced blinking response may be observed on the affected side.
- Schirmer test (evaluating lacrimation from the lacrimal gland) – Using a folded strip of blotting paper in the lower conjunctival fornix, a reduction of 75% in tear production compared to the unaffected side can be observed.
- Stapedial reflex test – This automatic response to loud sound stimuli involves the contraction of the stapedius muscle, which is controlled by the facial nerve. Assessment of the stapedial reflex can be conducted through tympanometry.
- Salivary Test – The rate of salivation is measured by stimulating the submandibular duct with a 6% citric acid

solution. A positive result is indicated by a 25% reduction in salivation on the affected side, suggesting a lesion located at or near the root of the chorda tympani.

- Taste Sensation Test – This involves applying salt, sweet, sour, and bitter tastes to the lateral regions of the anterior two-thirds of the tongue. A positive outcome on this test suggests a lesion in or near the root of the chorda tympana¹⁰

The degree of facial nerve paralysis is evaluated using the House-Brackman grading system¹⁰.

Grade	Definition
I	Normal symmetrical function throughout
II	Slight weakness on close inspection and slight asymmetry of smile
III	Obvious non-disfiguring weakness, complete eye closure
IV	Obvious disfiguring weakness, cannot lift the brow, incomplete eye closure, severe synkinesis
V	Barely perceptible motion, incomplete eye closure, slight movement of corner of mouth, absent synkinesis
VI	No movement, atonic

Laboratory testing

- Complete blood cell count
- Fasting blood glucose or hemoglobin A1c
- Enzyme-linked immunosorbent assay or an indirect fluorescent antibody test
- Serum antibodies for herpes zoster.
- Angiotensin-converting enzyme, human immunodeficiency virus, and
- inflammatory markers
- Cerebrospinal fluid analysis to differentiate it from Guillain-Barré syndrome, leptomenigeal carcinomatosis, and infection involving the central nervous system¹⁷

Imaging

CT and MRI can be used for the detection of infratemporal lesions¹⁰

6. Complications

The complications of facial palsy include²⁰

- 1) Ocular problems -Impaired vision from excessive corneal dryness and blindness²¹
- 2) Motor synkinesis -abnormal involuntary facial movement such as eye closure during smiling or mouth movement during blinking
- 3) Crocodile tear syndrome [Bogorad syndrome]

It is unilateral lacrimation tearing while eating or drinking due to abnormal nerve recovery²²

- 4) Incomplete recovery
- 5) Contracture of facial muscles
- 6) Reduction or loss of taste sensation
- 7) Dysarthria due to facial muscle weakness

7. Treatment

The primary objective in managing patients with facial paralysis is to achieve an early diagnosis and initiate early⁸Patients afflicted with facial palsy require regular clinical monitoring with frequent reevaluation to ensure that the treatment administered is appropriate, as the palsy can either improve or worsen over¹³

Conservative Management

Antibiotics

Patients with facial palsy having lyme disease should be treated with antibiotics. Doxycycline should be the first-line antibiotic, given its high efficacy and excellent central nervous system penetration¹³. For patients with doxycycline allergy or intolerance, a b-lactam antibiotic (e.g., amoxicillin) should be used²

Corticosteroids

Corticosteroids like prednisolone should be initiated within three days of the onset of peripheral facial palsy. This early initiation can enhance the chances of recovery, shorten the recovery period, and minimize the risk of development of synkinesis²

Antivirals

Acyclovir can be administered as a treatment for peripheral facial palsy when there is an association with herpes simplex virus infection. In cases of severe Bell's palsy, it should be prescribed alongside corticosteroids²

Eye care

Patients should be instructed to apply artificial tears every hour while they are awake in order to maintain corneal moisture and reduce the risk of injury². Utilizing a moisture chamber, eyelid taping, or a protective shield can be beneficial in safeguarding the eye while sleeping¹

Surgical management

Surgical intervention should be considered for patients who are unlikely to achieve a satisfactory spontaneous recovery following a period of observation or medical¹.

Facial reanimation: Encompasses both static and dynamic procedures. Static procedures aim to enhance facial symmetry by supporting the soft tissue structures of the paralyzed face but do not enable active movement. Dynamic procedures, on the other hand, aim to restore facial animation by utilizing innervated muscles. These methods include primary facial nerve repair, nerve grafting, nerve transposition, muscle-tendon unit (MTU) transposition, and free functional neuromuscular unit transfer¹

Facial nerve decompression is an option in cases of virally induced facial nerve palsy and also Bell palsy. A transmastoid approach would be best for cases of tympanic or mastoid segments damage to the facial nerve. If the damage extends to the labyrinthine segment, then a middle fossae approach allows appropriate decompression.¹⁰

Muscle transfer techniques are suitable for those patients with chronic facial nerve palsy (older than two years). Regional muscle transfer most commonly utilizes the

temporalis muscle; however, the digastric (marginal mandibular nerve injury) and masseter (smile reanimation) are also options¹⁰

Nerve grafting options tend to be utilized in intermediate durations of facial paralysis (3 weeks to 2 years) with some studies suggesting the best outcomes if performed within six months of insult¹⁰

Transcutaneous Nerve Stimulation is an additional new treatment option for those with unilateral facial nerve palsy. The technology uses EMG signals from muscles on the intact side of the face to simultaneously stimulate the corresponding muscles on the side of paresis. The ultimate aim of therapy is to achieve facial symmetry¹⁰.

Acupuncture: One of the most unique treatments in the Chinese medicine system and has become a universal medicine advocated by the World Health Organization (WHO) 64. It is a safe therapy with a low risk of adverse events in clinical practice²³.

8. Conclusion

Facial palsy, a condition characterized by the paralysis or weakness of facial muscles, presents a complex and multifaceted clinical challenge. It can arise from a multitude of causes, including viral infections, trauma, and neurological disorders. The diversity in its origins necessitates a tailored approach to diagnosis and management, with a focus on addressing the underlying cause whenever possible.

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