INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA):IJPS00]
Journal Homepage: https://www.ijpsjournal.com

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Review Article

Bell's Palsy: New Emerging Disease

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ARTICLE INFO

Received: 08 Feb 2024
Accepted: 12 Feb 2024
Published: 21 Feb 2024
Keywords:
electromyography, nerve excitability test, acyclovir, corticosteroids, lower motor neuron palsy, facial nerve
DOI:
10.5281/zenodo.10688837

ABSTRACT

Bell's palsy, also known as "unexplained facial paralysis", is a medical condition that causes paralysis or complete paralysis of the facial muscles, which is characteristic on the one hand and occurs immediately and can occur within 48 hours on the other. It is caused by facial nerve insufficiency due to injury or inflammation of the seventh cranial nerve or facial nerve or its branch (only in the bone canal). Both sexes are equally affected and although there is no age- related immunity, the incidence increases with age. It is more likely to occur in diabetic patients, people with high blood pressure, pregnant women, obese people and people with upper respiratory tract diseases. It is generally considered idiopathic and is diagnosed by exclusion of other causes. Bell's Palsy can cause physical and mental problems and harm the patient and their relatives. Therefore, early diagnosis and rapid determination of the cause are the main roles of correct treatment. However, the exact cause of Bell's palsy is unknown, which prevents its treatment. However, determining the effective and relevant factors is important for the clinical approach and requires a comprehensive examination and a complete history. Although most patients recover in less than three weeks without treatment. However, there is always a risk of residual hemiparesis, which may require medical intervention after treatment or rehabilitation. The aim of this review is to provide a better understanding of Bell's palsy by focusing on anatomy, etiology, clinical features, diagnosis, treatment evaluation results and treatment preferences.

INTRODUCTION

Bell's palsy is an idiopathic condition caused by dysfunction of the seventh cranial nerve (CN VII), also known as the facial nerve [1]. CN Vil contains motor, sensory, and parasympathetic components, CN Vil innervates the muscles of facial expression, the taste muscles of the anterior [2,3] of the tongue, and the parasympathetic innervation of most of the lacrimal and salivary glands Risk factors for Bell's palsy include diabetes, high blood pressure, toxins, infections (herpes simplex virus I (HSV 1), human immunodeficiency virus (HIV),

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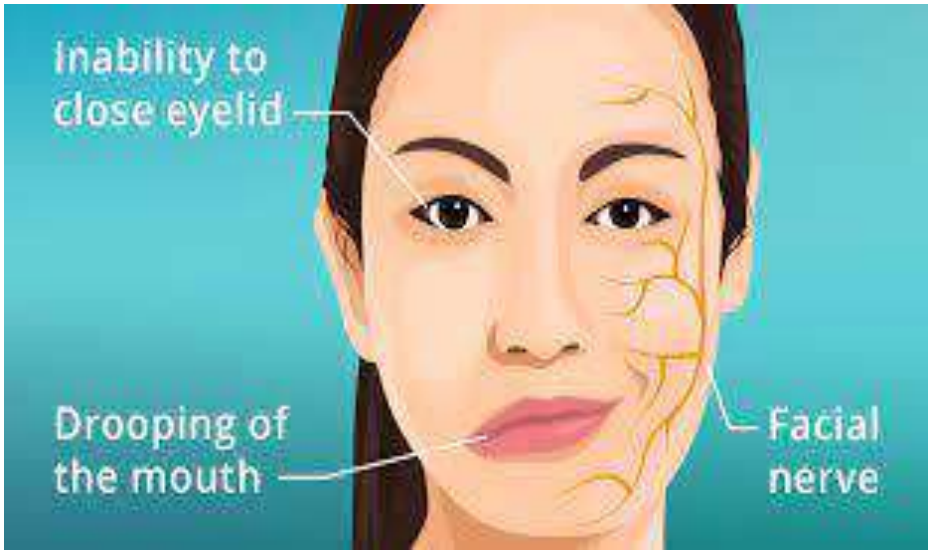
Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

shingles/chickenpox, Lyme disease, Epstein-Barr virus, and ischemia). Bell's palsy can occur at any age, but most occur between the ages of 15-60 and is believed to be a response to infection, causing inflammation and swelling of CN VII [4]. Many studies support the use of corticosteroids and eye treatments to improve Bell's palsy symptoms [5]. However, there is insufficient evidence to support PT treatment. There is some evidence that face masks may increase recovery time, but good evidence is lacking. This study describes a patient with Bell's palsy who presented with mild to severe facial paralysis and weakness on the right side, making it difficult to drink, speak, and control facial muscles. The purpose of this report is to describe tests and treatments for Bell's palsy and to help physical therapists develop treatment plans in the absence of good evidence [6].



Material And Methodology

This study describes a patient with Bell's palsy who presented with complaints of severe facial pain, weakness on the right side, difficulty drinking, speaking and controlling facial muscles. The purpose of this report is to describe tests and treatments for Bell's palsy and to help physical therapists develop treatment plans in the absence of good evidence.

History

Bell's palsy is named after Sir Charles Bell (1774-1842). Although he was the first to reveal the anatomical basis of Bell's palsy, recent studies indicate that other European physicians also contributed to the early description and description of seventh cranial nerve palsy [7]. The first publication on idiopathic facial paralysis is believed to have been published by Nicolaus Friedrich, a professor in Würzburg in the 18th century. The case report describes three middle-aged men with similar cases of paralysis, which were subacute or severe and gradually improved over several weeks to six months. Later, Charles Bell studied the facial nerve in animals. He encountered many cases of vascular complications during his surgical practice in London. The most famous and most quoted case of facial paralysis was published in 1828; where he described a man who was gored by a bull, causing injuries that resulted in facial paralysis [8].

Anatomical perspective

For a better comprehension of the etiopathogenesis of Bel's palsy, basic knowledge about the course and innervations of the facial nerve is required. The facial nerve has three nuclei motor sensory and parasympathetic nuclei. The course of the facial nerve can be divided into six segments. The first segment is the intracranial segment which comprises of facial nerve's motor nucleus located in the pons from where the motor fibers originate,

hook around the abducens nerve nucleus, and are joned by the intermediate nerve which cames sensory and parasympathetic components. Further, this mixed nerve passes through the posterior cranial fossa and enters the bony facial canal (fallopian canal) through the anterior superior quadrant of the internal acoustic meatus. This is known as the meatal or canalicular segment inside the inner ear, the facial nerve passes in the fallopian canal in between the cochlea and vestibule and then bends posteriorly at the geniculate ganglion (frit genu). This segment is the shortest and narrowest and is most prone to inflammation and ischemia. It is known as the labyrinthine segment. The labyrinthine segment extends and forms the tympanic segment in the middle ear, takes another turn just distal to the pyramidal eminence (second genu), and passes vertically downwards as the mastoid segment. Often, blood vessels in blood vessels can separate in some places, making them more vulnerable to damage. The mastoid segment starts from the second knee, branches and forms the segment by cutting through the stylomastoid foramen. It continues along the face and depth of the parotid gland and finally cuts into five branches at the edge of the throat [8,9]

Pathogenesis

Although the exact pathogenesis of Bell's palsy is unknown and is considered idiopathic, specific immune, ischemic and genetic factors are associated with its etiology According to recent data, reactivation of dormant herpes viruses in the geniculate ganglion and migration to the facial nerve is considered an important cause of association [8,12]. Herpes zoster virus (HZV) and herpes simplex virus (HSV) are the most common human neurotropic alphaherpes viruses [13]. These may remain hidden in the ganglia throughout life [14]. HZV infection is considered more serious because it can spread to the blood vessels of the satellite brain. It is often associated with herpes simplex cold sores and genital herpes, while shingles causes measles and shingles. He said that when there is no infection, the disease is latent, but in the presence of antibodies or immunity, the nerves are damaged and swelling occurs in the facial nerve, which will cause further compression of the facial nerve due to its location. In a narrow channel.. Other viruses known to cause Bell's palsy include the Epstein Barr virus, which causes mononucleosis, cytomegalovirus, adenovirus, mumps virus, influenza B virus, etc. takes place [15]. Vascular ischemia can be primary, secondary or tertiary. Primary ischemic neuropathy causes inflammation of the affected nerves and is more likely to occur with certain medical conditions, such as diabetes. It is usually caused by a cold or stress. Although the facial nerve has cood vascularity and an intact epineurium vasospasm can reduce blood flow and cause severe pain, leading to rare primary ischemic neuritis [16]. Secondary ischemia may then occur, resulting in increased capillary permeability, resulting in fluid accumulation and edema that can damage and further damage blood vessels [17]. Approximately 4-14% of people are genetically predisposed to fallopian tube stenosis. This genetic component is often somatically dominant, placing the vessel at risk of premature compression and even minimal edema [18].

Clinical findings

Symptoms appear suddenly and peak within 48 to 72 hours; It ranges from mild muscle pain to severe paralysis on the same side of the body. Symptoms of Bell's palsy include inability to blink or close the eyes, pursing of the lips or lifting of the corner of the mouth, loss of half of the face, loss of eyelashes on the same side, flattening of the nasolabial fold, pain or loss of vision. Hearing and dry eyes or dry mouth around the ear on the same side [19]. Other symptoms include hyperacusis due to stapedius nerve tear, taste changes, and dry eye syndrome due to parasympathetic neuralgia.

Some patients report facial paresthesia and perceived hearing or hallucinations, which are not usually interpreted as symptoms of changes [20].

Clinical Examination and Diagnosis

In addition to a correct neurological examination, clinical investigations should also include ear examination, eye examination, skin and tumor screening [21]. Shingles can be thought of as blisters or rashes around the ears (called Ramsay Hunt syndrome) that can cause hearing loss and facial paralysis. Observing the patient during the interview can reveal subtle symptoms of the disorder and provide supporting information [7].

A good method should be followed in the evaluation of patients. The examiner should examine the face and measure the face, such as forehead wrinkles (length of the legs), ability to close the eyes tightly, bulging face (chin), symmetry of the smile strabismus (zygomatic branch), wrinkled nose (buccal branch). department)). The degree and prognosis of facial nerve palsy can be evaluated by the House-Brackmann grading system. As shown in Table Tablell, it has six arades with Grade 1 beina no paralysis and Grade 6 meanina complete paralysis [22]. There should be no wrinkles or asymmetry on the same side of the forehead when raising the eyebrows. The ipsilateral eye appears partially closed and remains slightly open when the patient tries to close the eye, and the affected eyelid lags slightly behind when the patient blinks. The examiner may trigger Bell's phenomenon by asking the patient to close his or her eyes tightly while trying to open the patient's eyes. In these cases, the eyes move upward and backward. This technique also measures the strength of the orbicularis oculi muscle. Ears should be examined carefully for cholesteatoma, chronic suppurative otitis media, chronic suppurative otitis media, malignant otitis media, and other signs of middle ear disease. A red chorda tympani (dilated veins in the area tested by tympanometry) is seen in Bell's palsy. Various hearing tests can be performed, such as pure tone audiometry, speech audiometry, brain evoked response audiometry, and special tests to determine cochlear and retrocochlear pathology. To understand which part of the facial nerve is affected, doctors need to perform tests such as tearing, salivation, taste and stapedial reflex, while electrodiagnostic tests will show the depth of the damage. The diagnosis of Bell's palsy is primarily clinical and is made by excluding other causes of stroke [23]. Electrodiagnostic testing within 14 days of onset may provide evidence. Most facial nerve palsies are caused by other conditions similar to Bell's palsy, such as stroke or central nervous system diseases such as demyelinating disease, cholesteatoma, tumor, middle ear cancer disease, Lyme disease, diabetes, granulomatous disease[20,24,25]. When combined with history and diagnosis, MRI can be used to diagnose Bayesian palsy. Tympanic and vertical segment development can be seen in normal individuals, but labyrinthine segment development is especially seen in patients with Bell palsy [26].

TREATMENT

Side effects are generally good for most patients. According to the Copenhagen Facial Nerve Study, most patients recover completely, approximately 13% develop mild paralysis, and 4% to 5% develop facial paralysis [27]. Treatment remains problematic as recovery often occurs, but medication and therapy can help reduce symptoms and speed recovery. Prednisone and other oral corticosteroids can reduce blood vessel inflammation and improve the appearance and expression of the face. These medical Oral corticosteroids, such as prednisone, can reduce blood vessel inflammation and improve the appearance and speed of expression [29]. It is best to start this treatment within 48 hours of the appearance of symptoms [30]. The recommended dose of prednisone is 60 mg per day for five days

and then reduced to 10 mg per day [31]. If herpes is present, antiviral medications such as acyclovir (Zovirax) can be started at 400 mg orally 5 times a day for 10 days. ions are most effective when started within 48 hours of the onset of symptoms [12]. It can be treated quickly when antibiotics (such as acyclovir for herpes) are taken along with corticosteroids. This combination therapy is brief and cost-effective and is therefore generally recommended unless contraindicated [28] Ear infections can be resolved using antibiotics. and surgery by evaluating the importance and function of the nerve . Electrodiagnostic testing helps predict axonal damage by measuring the firing potential of the muscle. Patients with axonal degeneration greater than 90% can be treated with surgical decompression. In contrast, patients with low levels of axonal degeneration do not require surgical intervention and have a good prognosis.

Various non-pharmacological measures, such as facial exercises, neuromuscular reeducation, and physical therapy such as acupuncture, can also treat Bell's palsy and have been reported to provide relief [32]. As with Bell's palsy, the patient's ability to close or blink is impaired and the affected eye suffers from dryness and possible injury. Eye pads, goggles, or small inserts can provide protection, and daytime lubricating eye drops and artificial tears can control dryness [33]. In Bell's Palsy, muscle weakness and associated facial asymmetry can cause difficulty swallowing, slurred speech, and difficulty drinking and eating. Such patients may receive occupational and speech therapy, which can help improve speech clarity, reduce problems with dysphagia, and reduce embarrassment. If the paralysis does not improve by six to eight weeks, facial nerve decompression may be tried by opening the shell or removing the egg bone. In severe cases, for those who fail to recover, facial plastic surgery may be an option to correct facial asymmetry and help the eyes close. Laser acupuncture has also been tried in some parts of Asia to treat patients with Bell's palsy, but its role in Bell's palsy remains controversial [34]. It is a painless, non-invasive treatment for many inflammatory conditions and is an effective treatment for patients who are not recovering from Bell's palsy [35].

CONCLUSION

Bell's palsy is an ipsilateral, idiopathic, chronic lower motor neuron paralysis of the seventh cranial nerve, which causes weakness in the platysma and facial muscles and affects the patient's face, lifestyle and health. The symptoms begin with weakness of the facial muscles without any abnormality in the nervous system, increase in the first week and then gradually decrease over three weeks to three months, although there is no cure, but it can cause many problems and leave patients with different symptoms. Types of muscle paralysis. In the following cases: Failure to make rapid diagnosis and intervention. It can occur at any age, affecting both sexes equally, but its incidence increases in people in their 40s and is often seen in people with diabetes. Diagnosis is a matter of exclusion and requires careful history and careful examination. Testing for Lyme disease and diabetes may be recommended if there is a medical history or risk factors. Inadequate eye closure causes dry eyes, difficulty swallowing, and slurred speech as a short-term problem. A rare long-term problem is contraction and persistent weakness of the facial muscles. Although most patients recover, it is recommended to begin short-term valacyclovir or acyclovir therapy together with prednisone injection within three days of the onset of symptoms, shortening the duration and shortening the path to full recovery.

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HOW TO CITE: Mrunal D. Thakare, M. D. Game, Aditi G. Asole, Bell's Palsy: New Emerging Disease, Int. J. of Pharm. Sci., 2024, Vol 2, Issue 2, 590-596. https://doi.org/10.5281/zenodo.10688837