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Research Article

Bell's Palsy

Ruslan Ramlan Ramli[1*], Stanley Sesar Tjahjadi[2]
[1]Faculty of Medicine, Alkhairaat University
[2]Emergency Departments, RS.Woodward

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ABSTRACT

Bell's Palsy is defined as acute facial nerve paralysis affecting one side, with unknown causes. The cause of Bell's Palsy itself is upper respiratory tract viral infection, inflammation, autoimmune and ischemic. However, if no specific cause can be identified, this condition is known as bell's palsy which is caused by inflammation or nerve damage. Often the eye on the affected side cannot be closed, the nasolabial folds and forehead lines disappear. Corticosteroids have been found to improve Bell's palsy when used early, while anti-viral drugs have not shown many signs of improvement 10 days after onset, even without treatment. This article aims to review Bell's Palsy, especially its pathophysiology. The level of severity determines the healing process for Bell's Palsy.

Corresponding Author:

Ruslan Ramlan Ramli
Faculty of Medicine, Alkhairaat University
Email: ramlanruslan@gmail.com

1. INTRODUCTION

Bell's palsy is an acute nerve disorder of the face that affects one side of the face with lower motor neuron type paralysis which is characterized by unilateral facial weakness and without a definite cause due to acute peripheral facial nerve paralysis (1),(2),(3). Facial weakness can include reduced forehead wrinkles, difficulty closing the eyes, weakness of the corners of the lips, loss of taste sensation, and other symptoms based on the branch of the facial nerve affected (1),(2).

The incidence of Bell's palsy is found in 75% of cases of acute facial nerve paralysis. This disease affects both men and women in relatively equal percentages, with an incidence rate of 20 per 100,000 population with 40,000 new cases every day and 8-12% recurrence can occur. with a slightly higher incidence in the elderly, but can occur at any age. The incidence increases during pregnancy, in upper respiratory tract viral infections, immunocompromise, and in patients with diabetes mellitus and hypertension. Some epidemiological data shows that the incidence is slightly higher in winter than in summer. The incidence of Bell's palsy collected in 4 hospitals in Indonesia showed that the frequency of Bell's palsy was 19.55% of all neuropathy cases, and most occurred at the age of 21-30 years (2),(3).

Classic symptoms of Bell's palsy include disappearing forehead wrinkles, eyelid weakness, dry eyes, epiphora, paralysis or facial weakness, weakness at the corners of the mouth, dry mouth, changes in taste sensation (4),(5).

2. METHODOLOGY

The cause of paralysis in Bell's palsy is still being debated. Some experts state that the cause is continuous exposure to cold wind on one side of the face, there are also those who state that this is caused by the herpes virus which stays in the body and is reactivated due to trauma, environmental factors, stress, etc. Some sufferers can recover without treatment, but they are still advised to undergo physiotherapy and medication. The most common cause is herpes simplex virus type 1, other causes include: other viral infections: mumps and HIV, neoplasms: removal of brain tumors (acoustic neuroma) or other tumors, trauma: basal skull fractures, injuries to the middle ear and diving, neurological: Guillain Barre syndrome, metabolic: pregnancy, diabetes mellitus,

hyperthyroidism and hypertension, and toxic: alcohol, thalidomide, tetanus, and carbon monoxide. The pathogenesis of Bell's palsy is also thought to originate from retrograde epineural compression edema characterized by facial nerve ischemia. Although the etiology remains unclear, an attractive theory is that vasospasm, from several causes, along the facial nerve may also involve the chorda tympani.

Bell's palsy degree classification Facial grading system is a scoring system used to assess facial nerve function. This system is needed to determine the severity of facial dysfunction, follow the progression of facial paralysis, and compare treatment results. The grading system that is often and has been widely used in research, especially in the United States and Europe, is the HB grading system. The House Brackmann grading system has been adopted as a standard by the American Academy of Otolaryngology-Head and Neck Surgery and has been used. This system is based on 6 levels of scores (I-Vl) which provide an evaluation of the motor function of the facial nerve and also an evaluation of the sequelae (6).

Grade	Explanation	characteristic
I	Normal	Facial function is normal in all areas
II	Mild Dysfunction	Gross appearance: mild weakness visible on close inspection, perhaps mild synkinesis
		At rest: symmetrical and normal tone
		When moving: Forehead: moderate to good function Eyes: Close completely with minimal effort Mouth: mild asymmetry
III	Moderate Dysfunction	Gross appearance: obvious but indistinguishable between the two sides, visible but not severe synkinesis, contractures, and/or hemifacial spasm
		At rest: symmetrical and normal tone
		While moving: Forehead: slightly to moderately mobile Eyes: close completely with effort Mouth: mild weakness with maximal effort
IV	Moderate-severe dysfunction	Gross appearance: marked weakness and/or visible asymmetry
		At rest: symmetrical and normal tone While moving:
		While moving: Forehead: no movement Eyes: close incompletely Mouth: asymmetry with maximum effort
V	Severe Dysfunction	Gross appearance: barely perceptible movements
		At rest: symmetrical and normal tone
		While moving: Forehead: no movement Eyes: close incompletely Mouth: slight movement
VI	Total paralysis	No movement

3. CLINICAL SYMPTOMS

Clinical manifestations of Bell's palsy are symptoms on the ipsilateral side of the face in the form of: ipsilateral facial muscle weakness, ipsilateral disappearing forehead wrinkles, looking like a tired person, unable or difficult to blink, stiff nose, difficulty speaking, difficulty eating and drinking, sensitivity to sound in the form of hyperacusis, excessive or reduced salivation, facial swelling, reduced or lost taste, and frequent drooling, pain behind the ears, otalgia, hyperacusis, decreased tear production. Based on the discussion above, the pathophysiology of facial nerve paresis is thought to be due to the facial muscles under the forehead receiving innervation from the contralateral cortex, namely only the corticobulbar fibers that cross. Therefore, a rostral lesion of the facial nucleus originating from a central facial lesion will cause paralysis of the contralateral facial muscles, unless the frontalis and orbicularis oculi muscles receive bilateral cortical innervation, then these muscles will not be paralyzed due to lesions affecting one motor cortex or pathway. corticobulbar. Complete destruction of the facial nucleus itself or its branchial efferent fibers (facial nerve proper) paralyzes all ipsilateral facial muscles, this is equivalent to a peripheral facial lesion (7),(8).

4. PATHOPHYSIOLOGY

Pathogenesis The mechanism of Bell's palsy has been debated for decades, with the cause of the neuropathy remaining elusive with several existing theories. One theory explains that Bell's palsy is an acute demyelinating disease, which may have a pathogenesis mechanism similar to Guillain-Barre syndrome. It is suspected that both are demyelinating inflammatory neuritis, namely Bell's palsy can be considered as a mononeuritis variant of Guillain-Barre. The pathogenesis of Bell's palsy is thought to originate from retrograde epineural compression edema characterized by facial nerve ischemia. Although the etiology remains unclear, an attractive theory is that vasospasm, from several causes, along a branch of the facial nerve may also involve the chorda tympani. Where the labyrinth segment is the narrowest first part of the facial canal. This location is the most common place for compression of the facial nerve (9).

5. SUPPORTING INVESTIGATION

Supporting examinations that can be carried out for Bell's Palsy are CT Scan, MRI and EMG (electromyography) (9),(10).

6. THERAPY

The therapy used is Acyclovir and prednisone with the recommended prednisone dose of 1 mg/kg or 60 mg/day for 6 days where prednisone itself plays a role in reducing inflammation and peripheral nerve degeneration. Meanwhile, acyclovir therapy is at a dose of 400 mg orally 5 times a day for 10 days. Combination therapy of antiviral and prednisone has better results. The patient did not undergo laboratory and radiological examinations which were also considered appropriate because supporting examinations are not routinely carried out in Bell's palsy patients. Apart from that, physiotherapy was carried out in the form of superficial heat therapy, electrotherapy and facial massage accompanied by hot compresses. In general, the prognosis for Bell's palsy is good, around 80-90% of sufferers recover within 6 weeks to three months. without any defects. Patients aged 60 years or more have a 40% chance of complete recovery and are at high risk of leaving residual symptoms. Patients aged 30 years or less only have a 10-15 percent chance of recovering completely and leaving residual symptoms. If it does not heal within 4 months, the sufferer tends to be left with residual symptoms, namely synkinesis, crocodile tears and sometimes hemifacial spasm. Conclusion Bell's palsy is a neurological disorder caused by damage to the facial nerve which causes weakness or paralysis on one side of the face that appears suddenly due to nerve lesions facialist (8),(9),(10).

7. CONCLUSION

Bell's palsy is the most common disorder that affects the facial nerve due to acute edema that occurs in the stylomastoid foramen. Until now, the etiology of Bell's palsy is still unclear. There are several theories proposed, namely anatomical structure, viral infection, ischemia, inflammation and cold stimulation. There are also clinical symptoms that arise due to Bell's Palsy, namely paralysis of the entire face on the affected side, drooping corners of the mouth, disappearing forehead creases and eyes that do not close completely. The diagnosis of Bell's palsy is made on a clinical basis. The recommended therapy is steroid administration. In general, Bell's palsy has a good prognosis because it is a disease that can improve on its own (self-limiting disease).

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