

Idiopathic Facial Nerve Paralysis (Bell's Palsy) after Surgical Extraction of Lower Third Molar: A 5-month Follow-Up

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Abstract

Background: Facial nerve paralysis is the most common cranial nerve disorder, and it results in a characteristic facial distortion that is determined in part by the nerve branches involved. The initial assessment of a patient presenting with facial weakness distinguishing between a lower motor neuron and an upper motor neuron palsy, as the likely causes and, therefore, treatment for these vary significantly. **Summary:** A 42-year-old female patient reported to the department of oral and maxillofacial surgery presented with complaints of facial asymmetry and muscle weakness of her right side of the face after surgical dis-impaction after 5 day. **Conclusion:** There were few previously documented cases of facial palsy after maxillary and mandibular dental or surgical procedures. In most cases, paralysis began immediately after the injection of the mandibular anesthesia, and the duration of facial weakness was <12 h; a variety of different mechanisms can be associated with facial palsy, including viral reactivation, neural demyelination, edema, vasospasm, and trauma. The purpose of this presentation is to report a rare case of facial paralysis that had occurred on the contralateral side of face after mandibular 3rd molar extraction, to identify the probable cause of facial palsy, clinical evaluation, and examination, and subsequently follow-up with the direct management of the cause that was done with utmost importance of the multidisciplinary approach.

Keywords: Extraction, Molar, Surgery.

INTRODUCTION

Bell's palsy, also called idiopathic facial paralysis, is an isolated, unilateral, lower motor neuron facial weakness. Annual incidence varies in different parts of the world between 11 and 40/100,000 people. The incidence of Bell's palsy is 20–30 cases for 100,000 in India and almost accounts for 70% of all cases of unilateral peripheral facial palsy. However, the name of Sir Charles Bell, who published his findings in 1821, is usually associated with this condition.^[1]

Most Bell's palsy cases are self-limiting. In 71%, it will resolve completely and 84% will have near normal function. The remainder will go on to have seventh nerve dysfunction which includes moderate-to-severe facial weakness, synkinesis, or facial contracture. Management of Bell's palsy therefore aims to achieve complete recovery and reduce negative sequelae in cases that do not resolve spontaneously.^[2]

Etiopathogenesis (Table 1)

Although the precise pathogenesis of Bell's palsy is not known and considered idiopathic, specific immune, ischemic, and hereditary factors strongly correlate with its etiology. Based

on recent reports, the reactivation of dormant herpes virus in the geniculate ganglion and its migration to the facial nerve has been considered to be vital in causation. Herpes zoster virus and herpes simplex virus (HSV) are human neurotropic alpha herpes viruses and are most commonly involved. These may remain latent life-long in the ganglia.^[3]

The infection is said to be latent when there is no active viral replication, but in the presence of antibodies or immunodeficient states, nerve damage and inflammation of the facial nerve may occur, resulting in its further compression due to its fact in a narrow bony canal. Other viruses known to be involved in the causation of Bell's palsy are Epstein-Barr virus causing infectious mononucleosis, cytomegalovirus, adenovirus, mumps virus, influenza B, etc.

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Vascular ischemia may be primary, secondary, or tertiary. Primary ischemic neuropathy, which causes inflammation of the afflicted nerve, is more prone to take place in specific clinical circumstances, such as diabetes mellitus.

It is usually induced by cold or emotional stress. Even though the facial nerve has good vascularity and a tough epineurium, vasospasms can cause a reduction in blood flow and acute inflammation, resulting in primary ischemic neuritis, which is uncommon. It may be followed by secondary ischemia, which further aggravates nerve damage by causing increased capillary permeability leading to fluid accumulation, edema, and thus nerve compression.

In about 4–14% of individuals, hereditary predisposition narrows the fallopian canal. This genetic component is mostly autosomal dominant and puts the nerve at additional risk of early compression with even the lightest edema.^[3]

Clinical diagnosis and examination

A methodical approach to the evaluation of such patients is imperative. The examiner has to observe for facial expressions and test facial movements such as wrinkling of the forehead (temporal branch), ability to close eyes tightly, puffing of face (mandibular), symmetry of smile, screwing up eyes (zygomatic branch), and wrinkling nose (buccal) (Figure 1). The degree and prognosis of facial nerve palsy can be evaluated by the House–Brackmann grading system.

The diagnosis of Bell's palsy is mainly clinical, and it is made by ruling out alternative causes of unilateral facial paralysis. Electrodiagnostic tests performed within 14 days of commencement may provide prognostic information. A careful history, complete head and neck, and otological examination are paramount, along with radiologic studies, blood tests such as HSV, peripheral smear, total count, blood sugar, sedimentation rate, and serology.

In this paper, we report a rare case of contralateral delayed-onset facial palsy following dental procedure which was not fully recovered over 5 months of follow-up.

CASE REPORT

A 42-year-old woman reported to the department with a chief complaint of pain in the lower left back tooth region for 1 week and was diagnosed to have acute apical periodontitis with respect to 38 with mesioangular impaction. She had no pertinent past medical history. Her vitals were in the normal range and appeared in no acute distress with vitals in check; temperature: Afebrile; pulse rate: 77 bpm; respiratory rate: 16 cycles/min; and blood pressure (BP): 134/88 mmHg. Planned for disimpaction of 38 under local anesthesia. Under all aseptic conditions, the patient's face was painted with povidone-iodine and draping was done.

A left inferior alveolar nerve and buccal nerve block anesthesia were administered. A total of 3 mL was administered. The local anesthetic solution used was 2% lignocaine with adrenaline 1:200,000. The injection of the anesthetic solution was uneventful and resulted in adequate anesthesia. Tooth was extracted uneventfully and was recalled for suture removal after 1 week. Tablet amoxicillin 500 mg + clavulanic acid 125 mg (BD), tablet metrogyl 400 mg (TID), and diclofenac sodium 100 mg (BD) prescribed for 3 days. The patient reported to the department, complaining of weakness in facial movements on the right side after 5 days of tooth removal. Surprisingly, wisdom tooth removal was done on the left lower jaw.

The patient also gave a history of facial muscle weakness over the right side, inability to close the right eye and smile properly. On examination, loss of frowning of the forehead over the right side, inability to blow the right cheek, incomplete right eye closure, and asymmetrical smile (Figures 2-6).

According to House–Brackmann facial nerve palsy grading, these clinical features were classified to be Type VI – no movement, loss of tone, no synkinesis, contracture, or spasm. Investigations advised were (1) routine blood investigations, (2) magnetic resonance imaging (MRI), and (3) herpes serology test.

AQ1 Table 1: House-Brackmann scale

HB grading	Describe
I	Normal symmetrical function
II	Slight weakness noticeable only on close inspection Complete eye closure with minimal effort Slight asymmetry of smile with maximal effort Synkinesis barely noticeable, contracture, or spasm absent
III	Obvious weakness, but not disfiguring May not be able to lift eyebrows Complete eye closure and strong but asymmetrical mouth movement Obvious, but not disfiguring synkinesis, mass movement, or spasm
IV	Obvious disfiguring weakness Inability to lift eyebrows Incomplete eye closure and asymmetry of mouth with maximal effort Severe synkinesis, mass movement, or spasm
V	Motion barely perceptible Incomplete eye closure, slight movement at mouth corner Synkinesis, contracture, and spasm usually absent
VI	No movement, loss of tone, no synkinesis, contracture, or spasm

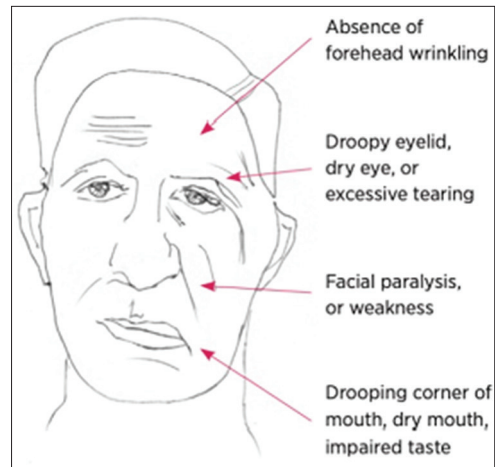


Figure 1: Clinical features of Bell's palsy

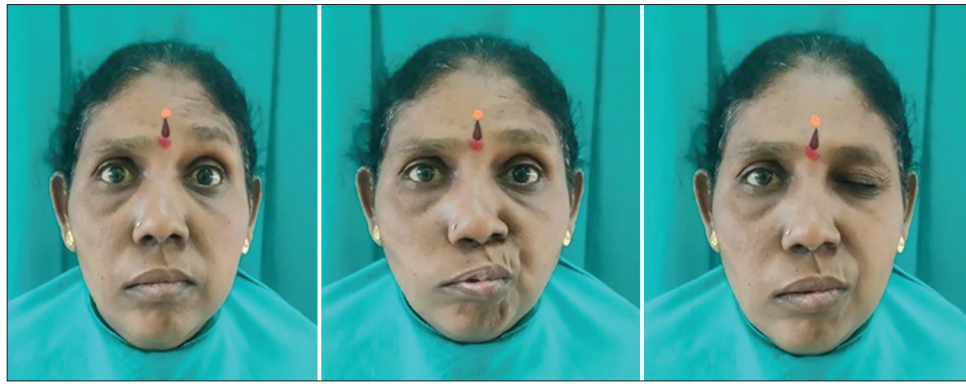


Figure 2: Post OP Day-5



Figure 3: Post OP Day- 30 [1 Month]



Figure 4: Post OP Day - 50 [1 Month 20 Days]

On routine blood investigations, all parameters are in the normal range. MRI report shows no evidence of acute infarct, hemorrhage, or space-occupying mass lesion. On enzyme-linked immunosorbent assay test, an HSV test with a positive immunoglobulin G result means that either has or had an HSV infection at some point of life. However, PCR analysis is an accurate test for the detection of HSV but the patient was not willing. On the basis of clinical and laboratory investigations, diagnosis of idiopathic facial nerve palsy was made.

The patient had persistent facial palsy; tablet methylprednisolone 20 mg TID/PO and tablet acyclovir 400 mg BD/PO 7 days were prescribed and advised physiotherapy for 15 days. After that, the patient was diagnosed as hypertensive and BP was 177/110 mmHg but her preoperatively BP was within normal range, and tablet amlodipine 5 mg OD/PO was prescribed after consulting a physician. Tablet methylprednisolone 20 mg BD/PO was continued and no improvement was noticed. After 1-month slight movement of the right upper eyelid was noticed, tablet methylprednisolone 20 mg tapered and improvement



Figure 5: Post OP Day- 80 [2 Months 20 Days]



Figure 6: Post OP Day-133 [4 Months 13 Days]

was noticed, BP being 152/98 mmHg. After 2 months, tablet methylprednisolone was stopped as the patient complained of facial swelling and BP being 142/94 mmHg. After 3 months, no gross facial asymmetry was detected and improvement in the zygomatic, buccal branches was noticed. The BP was 132/86 mmHg. By 4 months postoperatively, complete closure of the upper eyelid was seen and BP improved to 132/92 mmHg. Right facial paralysis continuously improved during the 5 months of follow-up.

DISCUSSION

In our case, patient reported with right side Bell's palsy after disimpaction of contralateral side which was managed by a conservative approach using tablet methylprednisolone 20 mg TID/PO as the first line of the drug.

Later on with various investigations, we confirmed the etiological factor to be herpes infection and started with the ideal treatment tablet acyclovir 400 mg BD/PO for 7 days along with facial physiotherapy for 15 days.

McCormick^[4] was the first one to find the link between HSV-1 and Bell's palsy which is due to the reactivation of HSV-1, centered around the geniculate ganglion. Moreover, the probable cause was found from clinical trials conducted by Zhang *et al.*;^[5] 2019 which stated that the activated apoptotic

pathways and intra-axonal degradation are the cause for HSV-1-mediated neural dysfunctioning which was observed in specimens of Bell's palsy patients.

In a study by Somasundara and Sullivan,^[6] it was noted that an antiviral agent was not beneficial in improving the outcomes of facial paralysis. In contrast, Hato *et al.*^[7] and Yoo *et al.*^[8] reported that a combination of antiviral therapy and corticosteroids was more effective in treating Bell's palsy.

The American Academy of Otolaryngology suggested that there is a 14% greater recovery rate for patients with Bell's palsy when treated immediately within 72 h from the onset of symptoms with or without the use of concurrent antiviral therapy treated along with oral corticosteroids but in our case, the patient reported to the department on the 5th-day post-onset of palsy symptoms. Corticosteroids were started immediately on the patient's visit, which was beyond 72 h resulting in delayed recovery time of the patient.

On the patient's next visit, that is, 14th-day post-onset of the disease (POOD), the patient had fluctuating BP being 177/110 mmHg and was immediately started with antihypertensives (tablet amlodipine 5 mg OD/PO) as advised by the physician. Hemorrhages into the facial canal are also responsible for facial paralysis with severe hypertension (HTN). Moreover, facial palsy immediately resolved after

better BP control, suggesting an association between the controlled HTN and resolution of facial palsy.^[9]

HTN may increase the risk of Bell's palsy among patients aged older than 40 years.^[9] Coincidentally, our patient did come under the age of 40 and above range which increased the risk of HTN being the major factor for favorable outcomes in patients with Bell's palsy.

Aynaci and Sen^[10] stated that HTN is one of the rare causes for peripheral facial paralysis, this unusual association may delay the diagnosis of HTN and may worsen the patient's condition more. In Savadi-Oskouei *et al.*^[9] study, 37.3% of Bell's palsy patients had HTN and 29% of Bell's palsy patients had diabetes but in this case, the patient was non-diabetic.

Similarly, our case also developed HTN suspecting a combination of high HTN and viral load being the etiological factor for our patient and was treated accordingly.

The patient was again recalled on 4th-week POOD. Moreover, on general examination routine check-up, BP was noted to be 166/106 mmHg cause of which another review was taken from the physician who advised an increase in the anti-hypertensive drug dosage to twice a day in view of uncontrolled BP (tablet amlodipine 5 mg BD/PO).

Previously some studies have also shown that the incidence of Bell's palsy is higher in patients with uncontrolled HTN due to poor compliance with medication. In three adult case studies with known chronic HTN, facial palsy occurred during the exacerbation of the HTN due to non-adherence to medication.

Studies conducted by Yoo *et al.*^[8] state that uncontrolled HTN is associated with facial palsy. Moreover, arterial HTN is diagnosed after a substantial delay. Adequate anti-hypertensive treatment is associated with a favorable outcome, and prognosis is good in patients with HTN controlled by medication.^[9] Moreover, facial palsy immediately resolved after better BP control, suggesting an association between the controlled HTN and the resolution of facial palsy. The initial severity of the facial paralysis and controlled HTN were factors associated with favorable outcomes.

In the present case, appropriate control of BP due to adequate antihypertensive treatment was associated with a good outcome after the development of facial palsy and so considerable outcome had been observed in our case with a conservative approach in treating the disease.

Various treatment options for patients of Bell's palsy are aimed to facilitate functional recovery including corticosteroids and antiviral drugs, alone or in combination.

According to the comparative study of intravenous (IV) methylprednisolone or oral prednisolone conducted by Kumar and Singh,^[11] it was noted that both IV and oral medications completely treated 82% of patients in 3 months of time.

Around 80% of patients recover completely within 4 months without any treatment whereas 20% of patients may not

recover fully. It is noted that about as described by the combined treatment with a corticosteroid and an antiviral agent has been shown to be more effective in treating severe to complete Bell's palsy than corticosteroid treatment alone. It appeared to be effective only in the more severe Bell's palsy, whereas less severe Bell's palsy might result in complete spontaneous recovery, regardless of physical therapy.

It is shown that the grade of Bell's palsy (severity) is the most important prognostic determinant for patient's recovery. The same had been advised to our patient having Class VI Housemann–Brackenn grading for better outcome.

Our case result was similar to those cases reported by Somasundara and Sullivan^[6] who found that patients with facial palsy of lower grades had a better outcome whereas Grade VI patients infrequently had a complete recovery.

In patients with Bell's palsy, early treatment with prednisolone significantly improved the chances of complete recovery at 3–12 months of time. In our case, it took about 2 months for the patient to clinically show initial recovery symptoms of facial nerve palsy based on the medications prescribed.

Consistent with these findings, our result also suggested that treatment of Bell's palsy should be commenced promptly.^[8]

Moreover, it took about approximately 4 months and above for the partial resolution of nerve palsy in the patient and she is still under our evaluation for a better outcome. Thus, conservative approaches such as corticosteroid and antiviral drugs show promising results with a 9–12-month regimen period. If signs and symptoms still persist after the regimen period, surgical management is advised by some authors like Fisch^[12] who reported 14 patients that underwent surgery had better long-term improvements compared to the long-term healing levels with non-surgical patients by decompressing the facial nerve through retroauricular approach in the 1st 3 weeks after the onset of paralysis and Gantz *et al.*^[13] performed surgical decompression in Bell's palsy patients and found that 90% of these patients recovered from HB Stages I and II by microvascular or canal decompression.

CONCLUSION

Bell's palsy is a unique condition that often resolves spontaneously over weeks to months. Conservative approach with proper diagnosis and etiology-based specific drug therapy will definitely give a good outcome. Oral corticosteroids are well accepted as treatment to decrease the duration of Bell's palsy symptoms. Antiviral agents, while commonly used, have inconsistent results. Other approaches such as surgical treatment, especially of the middle fossa decompression, should encourage early surgery in patients with idiopathic facial palsy who have severe nerve fiber degeneration and unfavorable prognosis. Contrarily, reassurance to the patient has a crucial role in the management of Bell's palsy.

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