Unilateral Facial Nerve Paralysis in a 13-Year-Old: A Case of Bell's Palsy with Incidental Sinusitis

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ABSTRACT

Bell's palsy is an acute, idiopathic paralysis of the peripheral facial nerve, resulting in sudden unilateral facial weakness. It is often associated with viral infections, particularly herpes simplex, and is more common in adults than children. This case is notable as it involves a 13-year-old male with no prior illness or risk factors. A previously healthy 13-year-old male suddenly developed left-sided facial weakness, including an inability to fully close his left eye and a drooping mouth, without any history of trauma or illness. Neurological examination revealed left-sided facial nerve palsy, lagophthalmos, and Bell's phenomenon, with other cranial nerves unaffected. CT imaging ruled out intracranial abnormalities but showed bilateral maxillary and ethmoid sinusitis. A diagnosis of Bell's palsy was made based on clinical presentation and the exclusion of other conditions. The patient received a treatment regimen of 20 mg oral Prednisolone, 1000 mg Valacyclovir, and methylcobalamin. Supportive care included lubricating eye drops, protective eye taping during sleep, and physiotherapy for facial exercises. The patient was advised to return for reassessment in five days. Pediatric Bell's palsy usually has a positive prognosis, with most cases resolving within weeks to months. Early corticosteroid and antiviral treatment may enhance recovery. This case highlights the urgent need for prompt diagnosis and intervention in pediatric Bell's palsy to prevent complications and ensure optimal recovery. A thorough neurological assessment and appropriate imaging are essential to rule out other causes of facial paralysis.

Keywords: Bell's palsy, Pediatric facial paralysis, Corticosteroid therapy, Valacyclovir, Neurological evaluation.

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Received: 28-01-2025; Revised: 14-03-2025; Accepted: 07-05-2025.

INTRODUCTION

Bell's palsy is a neurological condition characterized by sudden unilateral paralysis of the facial nerve (cranial nerve VII) without a clear cause, distinguishing it from other types of facial paralysis due to trauma or medical conditions. Its exact cause is uncertain but is often linked to viral infections, particularly the herpes simplex virus.¹

Symptoms vary widely, ranging from mild weakness to severe facial muscle impairment. Common signs include drooping on one side of the face, difficulty closing the affected eye, loss of taste in the front two-thirds of the tongue, and altered tear and saliva production. Patients may also experience pain around the jaw or behind the ear before paralysis begins.²



DOI: 10.5530/ijopp.20250318

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Bell's palsy is the leading cause of lower motor neuron facial nerve paralysis, accounting for 60-75% of acute facial paralysis cases.³ Its unilateral nature is a key feature, though rare instances of bilateral paralysis may indicate other underlying conditions, necessitating further investigation.

The precise cause of Bell's palsy remains a topic of ongoing research and is not fully understood. However, it is widely believed that the condition is primarily linked to the reactivation of the Herpes Simplex Virus type 1 (HSV-1). This reactivation occurs within the geniculate ganglion, a cluster of nerve cells located in the facial nerve pathway. When the virus reactivates, it leads to inflammation and swelling of the facial nerve, which can disrupt its normal function and result in the characteristic symptoms of Bell's palsy, such as sudden weakness or paralysis on one side of the face.⁴

In addition to HSV-1, other viral pathogens have also been implicated in the development of Bell's palsy. These include the Varicella-Zoster Virus (VZV), which is responsible for chickenpox and shingles, as well as the Epstein-Barr Virus (EBV), known for causing infectious mononucleosis. Cytomegalovirus (CMV), another member of the herpesvirus family, has also been associated with this condition. The involvement of these viruses suggests that Bell's palsy may have a viral etiology, with the immune response to these infections potentially contributing to the inflammation of the facial nerve.⁵

While viral infections are the most common triggers, there are rarer instances where other factors may contribute to the impairment of facial nerve function. For example, Lyme disease, which is caused by the bacterium Borrelia burgdorferi and transmitted through tick bites, can lead to neurological complications, including facial nerve palsy. Similarly, sarcoidosis, an inflammatory disease that can affect multiple organs, may also involve the facial nerve. Guillain-Barré syndrome, an autoimmune disorder characterized by rapid-onset muscle weakness, has been linked to facial nerve involvement in some cases. Additionally, infections of the ear, such as otitis media, can lead to complications that affect the facial nerve.⁶

Overall, while the exact mechanisms underlying Bell's palsy are not completely elucidated, the interplay of viral reactivation, immune response, and other potential factors highlights the complexity of this condition and the need for further research to better understand its causes and improve treatment options.

Bell's palsy is a neurological condition marked by sudden facial paralysis or weakness, usually on one side of the face. Its global annual incidence is estimated at 15 to 30 cases per 100,000 people,⁷ making it a relatively uncommon but significant health issue.

The condition is more prevalent in adults' aged 15 to 45, likely due to factors like stress and viral infections. While adults account for most cases, children represent about 10% of instances, indicating that Bell's palsy can affect various age groups, though it is less common in children.⁸

Gender studies show no significant preference for males or females, but some research suggests a slight predominance in younger males. Understanding the incidence across age and gender can enhance awareness and improve diagnosis and treatment for those affected by Bell's palsy.⁹

Bell's palsy is characterized by the abrupt emergence of unilateral facial weakness, usually occurring within a 72 hr timeframe. This condition may be evident through various manifestations, such as the inability to fully close the eyelid, known as lagophthalmos, as well as a drooping appearance of the mouth, challenges in expressing emotions, and changes in taste sensation. Furthermore, individuals may experience hyperacusis, which is an increased sensitivity to auditory stimuli, along with either diminished tear production or excessive tearing resulting from compromised lacrimal gland function.^{10,11}

Although Bell's palsy is extensively recognized in adult populations, its manifestation in children is comparatively uncommon, frequently resulting in delayed identification and incorrect diagnoses.¹² This particular case is noteworthy because it presents without any prior viral illness or discernible risk factors, underscoring the necessity for comprehensive diagnostic assessments to rule out secondary etiologies. Additionally, the timely administration of corticosteroids and antiviral medications has been linked to enhanced recovery outcomes, thereby underscoring the critical nature of immediate therapeutic intervention.¹³

This case report seeks to underscore the clinical manifestations, diagnostic evaluation, and treatment strategy employed for a 13-year-old male diagnosed with Bell's palsy. It emphasizes the critical nature of prompt diagnosis, effective management, and diligent follow-up to enhance recovery outcomes. Through the presentation of this case, the intention is to increase awareness among healthcare professionals about pediatric Bell's palsy and to advocate for implementing standardized treatment protocols.

CASE DETAILS

A 13-year-old male presented to the outpatient department of general medicine in Trust Multispeciality Hospitals, Kakinada, on July 30, 2024, with a chief complaint of sudden acute weakness on the left side of his face, which had emerged the previous day. The patient reported an inability to fully close his left eye as shown in the Figure 1, noticeable drooping at the left corner of his mouth, and challenges in performing facial expressions, including smiling or displaying his teeth. He did not experience any accompanying pain, fever, recent injuries, or prior upper respiratory infections. Furthermore, he denied any symptoms indicative of systemic conditions, such as rashes, hearing impairment, or dizziness. Before he visited the outpatient department, the patient sought care at a nearby hospital, where he received prescriptions for Lacryl PF Eye Gel and Carboxymethyl cellulose eye drops to alleviate dryness in his left eye.

The patient presented with an unremarkable medical and surgical history. He received the COVISHIELD vaccine for COVID-19 and reported no known allergies. His family medical history did not reveal any relevant conditions, as no relatives had neurological or autoimmune diseases. As a school-aged adolescent, the patient was considered to be in good health before the emergence of his current symptoms.

Upon assessment, the patient's vital signs were found to be stable, with a pulse rate recorded at 79 beats per minute, a respiratory rate of 18 breaths per minute, and an oxygen saturation level of 97% while breathing ambient air. A thorough neurological assessment indicated a weakness in the left facial nerve, particularly affecting the muscles responsible for facial expressions. This was demonstrated by the patient's inability to fully close the left eye, a condition referred to as lagophthalmos, alongside a noticeable droop at the left corner of the mouth. Additionally, the presence of Bell's phenomenon was observed, which is characterized by an upward movement of the eyeball when the patient attempted to close the eye on the affected side. Importantly, there was no evidence of dysfunction in other cranial nerves, and both motor and sensory functions in the limbs remained intact. Reflexes were assessed as normal, and the physical examination did not reveal any other systemic abnormalities.

A Computed Tomography (CT) scan of the brain was conducted to eliminate the possibility of structural or vascular origins for the observed facial paralysis. The imaging results indicated the presence of bilateral maxillary and ethmoid sinusitis; however, no abnormalities within the cranial cavity were detected, as shown in the Figure 2. The combination of clinical symptoms and imaging findings supported the diagnosis of Bell's palsy, which is characterized as idiopathic peripheral facial nerve palsy frequently linked to viral infections or inflammation affecting the facial nerve.

The patient was initiated on a therapeutic regimen that included Tab. Prednisolone at a dosage of 20 mg, administered as two tablets each morning, is aimed at alleviating inflammation and edema associated with the facial nerve. In addition, Tab. Valacyclovir was prescribed at a dosage of 1000 mg, to be taken twice daily, to address a possible viral cause particularly that related to the herpes simplex virus. Furthermore, Tab. Methylcobalamin was recommended for daily intake in the afternoon to facilitate nerve regeneration. The ongoing use of Lacryl PF Eye Gel and carboxymethylcellulose eye drops was maintained to ensure adequate corneal moisture and prevent dryness. Due to the patient's difficulty in fully closing the left eye, the general physician advised the application of paper plaster to secure the eye during sleep, thereby minimizing the risk of keratitis. Additionally, physiotherapy focusing on facial muscle exercises was proposed to support the recovery process.

The patient was advised to return for a follow-up appointment in five days to re-evaluate his neurological condition and assess the effectiveness of the treatment provided. Although the outlook for children diagnosed with Bell's palsy is typically positive, as most instances tend to resolve on their own within a few weeks to months, timely administration of corticosteroids and antiviral medications can markedly improve recovery results. This situation highlights the critical need for an immediate and comprehensive assessment of sudden facial paralysis to rule out alternative causes and to commence suitable treatment.

DISCUSSION

Bell's palsy represents the predominant etiology of acute, unilateral lower motor neuron facial nerve paralysis; however, its incidence in pediatric populations is notably infrequent.¹ The case of a 13-year-old male exhibiting acute left-sided facial paralysis, lagophthalmos, and facial asymmetry, devoid of any prior viral illness or discernible risk factors, underscores significant clinical and diagnostic implications. The diagnosis was made based on clinical evaluation, which revealed isolated facial nerve impairment without accompanying neurological deficits, and was further corroborated by neuroimaging (CT scan) that excluded any structural or vascular abnormalities.

This case is consistent with the observations made by Peitersen in 2002,¹ who characterized the classic manifestation of Bell's palsy as a rapid onset of unilateral facial weakness that typically reaches its maximum intensity within a 72 hr timeframe. Additionally, the lack of systemic symptoms, including fever or rash, in our patient supports a previous study by Heckmann JG in 2019³ suggesting that Bell's palsy is predominantly an idiopathic condition, distinguishing it from systemic neurological disorders.

The patient received a treatment regimen that included corticosteroids such as Prednisolone at a dosage of 20 mg and antivirals such as Valacyclovir at 1000 mg, supplemented by supportive interventions such as eye care and physiotherapy. This therapeutic strategy is well-supported by research conducted by Hato et al., in 2007,¹⁴ which indicated that the administration of corticosteroids within the first 72 hr can significantly enhance functional recovery. Nevertheless, the added advantage of incorporating antiviral therapy into the treatment plan remains a subject of debate, as evidenced by a meta-analysis conducted by de Almeida et al., in 2011,8 which reported only a slight improvement when corticosteroids were used in conjunction with antivirals. The therapeutic approach involving Prednisolone and Valacyclovir was commenced per the Clinical Practice Guidelines for Bell's Palsy issued by the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS, 2013).13 These guidelines advocate for the prompt initiation of corticosteroid treatment within a 72 hr window to enhance functional recovery outcomes for individuals diagnosed with Bell's palsy.



Figure 1: Showing Unilateral Lower Motor Neuron Facial Nerve Palsy: Incomplete Eye Closure and Brow Droop in Bell's palsy.



Figure 2: Axial CT brain images showing no acute infarction, haemorrhage, mass effect, or midline shift. The ventricular system and sulcal spaces are normal, and there are no abnormalities in the cerebellum, brainstem, or basal ganglia. These findings support a diagnosis of Bell's palsy, with no evidence of structural intracranial pathology.

The chosen regimen of Prednisolone and Valacyclovir was determined under the guidelines established by the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS). These guidelines advocate for the administration of corticosteroids as the principal therapeutic approach, supplemented by antiviral treatment in instances where a viral cause is suspected.¹³

The progression of Bell's palsy in pediatric patients is typically positive, as approximately 90% of instances resolve on their own within six months. Nevertheless, prompt treatment with corticosteroids has demonstrated a significant enhancement in recovery outcomes, especially in instances characterized by moderate to severe paralysis. In this scenario, the timely use of Prednisolone and Valacyclovir likely played a crucial role in shortening the duration of symptoms and facilitating the recovery process.

A significant insight from this case report is the necessity of excluding secondary etiologies of facial paralysis in children. In contrast to adults, where Bell's palsy frequently presents as a clear-cut diagnosis, pediatric facial paralysis can stem from various underlying conditions, including Lyme disease, Guillain-Barré syndrome, otitis media, or neoplastic growths. In the case of our patient, the CT scan revealed incidental findings of sinusitis without any signs of intracranial pathology, underscoring the critical role of imaging in instances where the clinical presentation is atypical.

Furthermore, the management of eye care is an essential aspect of treatment. The patient faced a significant risk of corneal exposure keratitis due to lagophthalmos and inadequate eyelid closure, conditions that can result in lasting visual impairment. To mitigate these risks, the application of lubricating eye drops and the practice of taping the eyes at night were implemented a method that has been strongly endorsed by previous research.¹⁵

This case aligns with previously documented instances involving pediatric patients, while also introducing certain distinctive features. Unlike the observations made by Evans *et al.*, in 2005,⁷ which indicated a greater incidence of Bell's palsy in children after upper respiratory infections, our patient lacked any prior viral illness, thereby rendering the underlying cause ambiguous.

Additionally, research conducted by Baugh *et al.*, in 2013¹³ indicated that pediatric cases of Bell's palsy tend to exhibit a more positive prognosis than those in adults, which may be attributed to the higher levels of neuroplasticity observed in children. This observation is corroborated by our case, where the patient demonstrated a significant improvement following the initiation of early corticosteroid treatment and appropriate symptomatic care.

Another distinguishing feature is the incidental finding of maxillary and ethmoid sinusitis on CT imaging. While sinusitis

is not a direct cause of Bell's palsy, studies by House *et al.*, in 1985⁹ have suggested that adjacent inflammatory processes may contribute to localized facial nerve edema. However, the exact role of sinusitis in this case remains speculative.

Lastly, while the combined use of corticosteroids and antivirals has been debated in the literature, Kasse *et al.*, in 2005¹⁰ found that antiviral therapy may be beneficial in cases with severe initial presentation. In our case, early administration of Valacyclovir was chosen as a precautionary measure, considering the possibility of viral reactivation as a trigger for Bell's palsy.

The findings derived from this single case study cannot be extrapolated to the broader population of pediatric patients with Bell's palsy, as the isolated nature of the case limits its generalizability. To substantiate these observations, a more extensive cohort would be necessary for validation. Despite the observed improvement in our patient, following treatment, the absence of long-term follow-up data at the time of reporting poses challenges in evaluating the overall outcomes and potential complications associated with the condition. Although the clinical diagnosis of Bell's palsy was unequivocal, the lack of Nerve Conduction Studies (NCS) or Electromyography (EMG) means that we missed the opportunity to obtain quantitative data regarding nerve function and prognosis, which could have enriched our understanding of the case.

CONCLUSION

The occurrence of pediatric Bell's palsy in a 13-year-old boy, particularly in the absence of a recognizable viral prodrome, underscores the critical significance of early diagnosis, immediate corticosteroid treatment, and comprehensive supportive care. The lack of identifiable predisposing factors, coupled with the incidental discovery of sinusitis, renders this case particularly remarkable, emphasizing the necessity for meticulous diagnostic assessment in atypical clinical presentations.

Although Bell's palsy in pediatric patients typically presents a positive outlook, this particular case highlights the critical need for prompt intervention to minimize recovery duration and avert complications, especially those related to eye health. Additional research is required to elucidate the efficacy of antiviral treatments in children with Bell's palsy and to explore possible associations between sinus inflammation and the involvement of the facial nerve.

ACKNOWLEDGEMENT

We extend our gratitude to Dr. M. Phani Ramana Bhushan, Consultant Physician in the Department of General Medicine at Trust Multispeciality Hospitals in Kakinada, for his exceptional clinical acumen in the diagnosis and management of this case. We extend our heartfelt appreciation to the medical staff of Trust Multispeciality Hospitals, Kakinada, whose outstanding care played a crucial role in the successful management of the patient's condition. Furthermore, we recognize the important partnership with the patient's representatives throughout the informed consent process, which facilitated our ability to present this case for both educational and research objectives.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHOR'S CONTRIBUTION

Dr. PKY and Dr. MPRB were pivotal in overseeing the case report process and offering mentorship to the students namely ML, ML, SY, and SD during the manuscript drafting phase. Under the supervision of Dr. PKY, ML, and SY actively participated in the writing and preparation of the manuscript. Ultimately, all authors engaged in a thorough review and granted their approval for the final version of the manuscript.

ABBREVIATIONS

CT: Computed Tomography; **HSV-1**: Herpes Simplex Virus Type 1; **VZV**: Varicella-Zoster Virus; **EBV**: Epstein-Barr Virus; **CMV**: Cytomegalovirus; **PF**: Preservative-Free; **EMG**: Electromyography; **NCS**: Nerve Conduction Studies; **AAO-HNS**: American Academy of Otolaryngology-Head and Neck Surgery; **COVID-19**: Coronavirus Disease 2019.

ETHICS APPROVAL AND DECLARATION OF PATIENT CONSENT

The authors affirm that they have secured the necessary consent from the patient's representatives. The patient's representatives have granted permission for the inclusion of his clinical information in the publication. Furthermore, the patient was aware that his name and initials would remain confidential and appropriate measures would be taken to protect his identity, and we assure the same.

Ethics approval was not required for this case report as per the institutional guidelines, as case reports that involve a single patient with anonymized data and without any experimental interventions do not typically mandate formal ethical approval.

INSTITUTIONAL APPROVAL STATEMENT

The authors affirm that the release of this case report received the necessary approval from the Department of Pharmacy Practice at Aditya Pharmacy College (A) in Surampalem, as well as from Trust Multispeciality Hospitals located in Kakinada.

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Cite this article: Yanamadala PK, Bhushan MPR, Lalam M, Lahkar M, Yoganandam S, Danduprolu S. Unilateral Facial Nerve Paralysis in a 13-Year-Old: A Case of Bell's Palsy with Incidental Sinusitis. Indian J Pharmacy Practice. 2025;18(4):445-50.