

Childhood Facial Palsy –A Case Report

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Citation

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Abstract

Bell's palsy is defined as a neuropathy of the peripheral seventh cranial nerve or the facial nerve. It is usually from an idiopathic cause but it can result from traumatic, compressive, infective, inflammatory or metabolic abnormalities involving the facial nerve (1). Bell's palsy is more common in adults than in children. The incidence of Bell's palsy in children is about 6-7% (2). There are very few studies that describe the magnitude and prognostic features of Bells palsy in children. We present a case of Bell's palsy in an 8-year-old child who completely recovered after treatment with oral antiviral agents and steroids followed by regular facial physiotherapy. The dilemma in management of Bell's palsy in children along with a review of relevant literature is discussed in this report.

INTRODUCTION

Bell's palsy is named after Dr. Charles Bell, who, in 1821, described complete facial paralysis after injury of the stylomastoid foramen.³ It can be defined as acute peripheral facial nerve palsy usually due to an unknown cause. It can result from traumatic, compressive, infective, inflammatory or metabolic abnormalities involving the facial nerve.⁴ It is typically unilateral and can be complete or partial.⁵ Affected patients are usually unable to close their eyes. Facial appearance becomes asymmetric, and saliva dribbles down the angle of the mouth. Depending on the site of the lesion, some patients may complain of noise intolerance or loss of taste sensation.^{4,6}

Bell's palsy is more common in adults than in children. The incidence of Bell's palsy in children is about 6-7%.² The outcome of Bell's palsy in children has been reported to be good with recovery rates ranging from 75% to 93.8%. Although there is agreement on the definition, there is no consensus regarding the etiology, diagnostic approach or management of this enigmatic condition⁶ and role of steroids in treating patients with Bell's palsy is still under trial. We present a case of Bell's palsy in a child with a remarkable recovery following a short course of oral antiviral drugs and steroids. The management options and outcome are discussed with a review of relevant literature.

CASE REPORT

An 8 year-old-male child accompanied by his mother presented to the otorhinolaryngology outpatient department

at a tertiary referral institute in Bangalore, south India with complaints of deviation of angle of the mouth to left side since one day associated with dribbling of food from the right side of mouth and inability to completely close the right eye as noted by the mother since a few hours. A history of right-sided ear infection 6 months ago which subsided with a course of antibiotics was also documented. The mother denies any history of excessive watering from the eyes, fever or recent upper respiratory tract infection. There was no history suggestive of taste disturbances, tingling sensation over face and weakness or numbness of the body. Immunizations were given as per schedule with a normal birth history.

On examination of the face there was obvious deviation of the angle of the mouth to the left when all the facial muscles were at rest. There was incomplete closure of the right eye even after maximum effort and there was absence of creases over the forehead on frowning. (Figure 1)

Figure 1

Figure 1 – Showing the deviation of the angle of mouth to the left along with incomplete closure of the right eye on maximum effort.



Blood investigations included a normal hemoglobin level, total count, differential count and ESR. Titers for EBV were negative. Pure tone audiometry confirmed absence of any hearing abnormality. An initial working diagnosis of Bell's palsy was made and the parents were educated about the disease process, therapeutic interventions and anticipated course of the disease. The conservative line of management with oral Acyclovir 800 mg five times a day for a period of seven days and oral prednisolone 20 mg three times a day for a period of ten days was administered. Adequate eye care was given by taping the eyes in the night during sleep and administration of sterile tear drops to prevent dryness of the eye. Facial physiotherapy was given for a period of 14 days.

Figure 2

Figure 2 – Follow up after 1 month showing a complete recovery.



The patient was reviewed in the outpatient department over the next month and a complete recovery was noted. (Figure 2)

DISCUSSION

Bell's palsy is defined as a neuropathy of the peripheral seventh cranial nerve or the facial nerve. It is usually from

an idiopathic cause but it can result from traumatic, compressive, infective, inflammatory or metabolic abnormalities involving the facial nerve.⁴ The incidence of Bell's palsy in children is about 6-7%. It is generally a unilateral disease affecting both sides of the face equally.⁶ A child with an acute onset of unilateral facial weakness most likely has Bell's palsy. A careful history of the onset and progress of paralysis is important because gradual onset of more than two week's duration is strongly suggestive of a mass lesion. Medical history should include recent rashes, arthralgias, or fevers; history of peripheral nerve palsy; exposure to influenza vaccine or new medications; and exposure to ticks or areas where Lyme disease is endemic.⁷

The physical examination includes careful inspection of the ear canal, tympanic membrane, and oropharynx, as well as evaluation of peripheral nerve functions in the extremities and palpation of the parotid gland. In order to assess forehead involvement, physical examination should also include evaluation of cranial nerve function, including all facial muscles. Laboratory testing is not usually indicated. However, because diabetes mellitus is present in more than 10 percent of patients with Bell's palsy, fasting glucose or A1C testing may be performed in patients with additional risk factors (e.g., family history, obesity, older than 30 years).⁸ Antibiotic therapy may be of benefit; therefore, Lyme antibody titers should be performed if the patient's history suggests possible exposure.⁷

The pathogenesis of Bell's palsy remains controversial. Acute inflammation and edema of the facial nerve are thought to lead to entrapment of the nerve in the bony canal (especially in the labyrinthine segment), which leads to compression and ischemia.^{9,10} An inflammatory process surrounds the nerve fibers. Many viruses, such as HIV, Epstein-Barr virus and hepatitis B virus have been suspected in initiating this inflammation.¹¹ Signs and symptoms atypical for Bell's palsy should prompt further evaluation. Patients with insidious onset or forehead sparing should undergo imaging of the temporal bone. Those with bilateral palsies or those who do not improve within the first two or three weeks after onset of symptoms should be referred to a neurologist.⁷

Patients with Bell's palsy may be unable to close the eye on the affected side, which can lead to irritation and corneal ulceration. The eye should be lubricated with artificial tears until the facial paralysis resolves. Permanent eyelid weakness may require tarsorrhaphy or implantation of gold

weights in the upper lid. Facial asymmetry and muscular contractures may require cosmetic surgical procedures or botulinum toxin (Botox) injections. In these cases, consultation with an ophthalmologist or cosmetic surgeon is needed.^{12, 13}

Increasing evidence is associating HSV with Bell's palsy, and, in time, Bell's palsy may well be reclassified as an HSV mononeuritis of the facial nerve, although designation of this causative agent does not exclude the possibility that other causes may exist or negate the role that entrapment plays in degeneration of the nerve. According to one hypothesis, HSV, dormant in the geniculate ganglion cells, becomes reactivated and replicates, causing inflammation, primarily in the geniculate ganglion and in the labyrinthine segment of the facial nerve. These inflammatory events (evident on magnetic resonance imaging) result in entrapment and ischemia, which lead to neurapraxia or degeneration of the facial nerve distal to the meatal foramen.¹¹

HSV has been identified in the endoneural fluid, posterior auricular muscle and saliva by polymerase chain reaction in patients with Bell's palsy.¹⁴ Increased capillary permeability leads to exudation of fluid, edema and compression of the microcirculation of the nerve, which may be responsible for the vascular ischemia.⁵ Patients generally experience rapid onset of unilateral facial palsy and often describe numbness or stiffness, although no actual sensory loss occurs.⁶

A recent Cochrane study in 2004 on Bell's palsy found results of the 3 best randomized controlled trials in the pediatric population, where steroid therapy provided no benefit over placebo for recovery of motor function.

¹⁵Currently, there is only 1 randomized controlled trial of steroid therapy for Bell's palsy in children. Using the Jadad score of methodological quality, a Cochrane review graded this trial 3 on a scale of 0 (poor) to 5(good). Forty-two children aged 2 to 6 years with onset of Bell's palsy up to 3 days earlier were randomized to receive either oral methylprednisolone (1 mg/kg for 10days), gradually tapering over 3 to 5 days, or placebo. Using a 6-level grading system of facial nerve paralysis, the authors performed unblinded assessments for asymmetry, spasm, tone, and child's ability to move forehead, eyes, and mouth. The study reported no benefit of treatment with methylprednisolone. A large randomized controlled trial is needed to establish whether steroids benefit children with Bell's palsy.¹⁵

Further the differences between childhood and adult Bell's palsy in literature with reference to management and

recovery, do not advocate the use of steroids with antiviral medications in pediatric population which on the other hand worked remarkably well in our patient.

Figure 3

Table 1*- Shows the differences between childhood and adult Bell's palsy:

	Childhood idiopathic Bell's palsy	Idiopathic Adult Bell's palsy
Incidence	66.2 % of the cases of facial palsy	54.9% of the cases of facial palsy
Treatment modalities	Steroid monotherapy (88.5%), acupuncture (11.5%)	Steroid monotherapy (80.1%) Steroid + antiviral (18.5%), Other methods were facial nerve decompression and temporalis muscle transfer (1.4%)
Recovery rate	93.1 %	91.4 %
Prognosis	Better in children	Poorer with advancing age

*Data collected from Chang H, Chai et al. Yonsei Med J. 2008 October 31, an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0/>) which permits unrestricted noncommercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

CONCLUSION

Childhood Bell's palsy is a rare clinical entity that possesses a challenge to the medical team in terms of diagnosing and managing this condition. This case report signifies a rapid and complete recovery in a child with Bell's palsy due to a quick and early diagnosis followed by prompt management with oral steroids and acyclovir. This implies the option to consider a diagnosis of Bell's palsy more strongly in spite of the low incidence in childhood. A later delay in onset of treatment might not result in such a complete recovery. A multidisciplinary team approach involving general practitioners, otolaryngologists, ophthalmologists, plastic surgeons, physiotherapists, and psychologists is essential for management of childhood Bell's palsy effectively.

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