

Clinical Characteristics of Facial Nerve Palsy in Children

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Background and Objectives: The aim of this study was to investigate the clinical characteristics of facial nerve palsy in children. **Subjects and Methods:** Clinical records of 25 pediatric patients presenting with acute onset facial nerve palsy between January 2005 and May 2010 were reviewed retrospectively. **Results:** The age at presentation at presentation ranged from 0 to 18 years (mean 9.5). The causes of facial nerve palsy were: 19 cases of Bell's palsy (76%), 3 cases of temporal bone trauma (12%), 1 case of otitis media (4%), and 2 cases of congenital facial nerve palsy (8%). Review of 11 Bell's palsy patients with complete medical records showed complete recovery in 9 of 11 patients (81.8%). Serum antibody to the herpes viruses varicella-zoster virus, Epstein-Barr virus, or herpes simplex virus was detected in 9 of 11 patients (81.8%), but did not correlate with functional outcome. **Conclusions:** The prognosis of facial palsy in children is generally acceptable. Bell's palsy was the most common etiology. Presence of serum antibodies to virus did not alter the outcome of facial nerve function in Bell's palsy patients.

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KEY WORDS: Facial palsy · Children · Virus.

Introduction

Unilateral facial nerve palsy occurs in about 10–30 per 100,000 of the general population.¹⁾ Only about 10% of the facial nerve palsy patients are children. The incidence of Bell's palsy, the most common etiology of acute facial nerve palsy, is reported as 6.6 per 100,000 children.^{2,3)} Other etiology includes trauma, infection, congenital, and neoplasms. Management of facial nerve palsy is needs to be tailored according to different causes. Restoration of facial nerve function without sequalae remains the treatment goal and challenge. Major complications include permanent paralysis, pain, and synkinesis on facial motion.⁴⁾ Also, psychological distress due to facial asymmetry can be severe. This study was performed to analyze the clinical features, functional recovery and the frequency of association of viral infection in pediatric patients with facial nerve palsy.

Subjects and Methods

The clinical data of all children presenting with acute onset unilateral facial nerve palsy to the Yonsei University College of Medicine Gangnam Severance Hospital from be-

tween January 2005 and May 2010 was retrospectively reviewed. Children with facial nerve palsy due to central nervous system disorders were excluded. Clinical records of 25 children with facial nerve palsy were reviewed to determine the etiology of facial nerve palsy including trauma, infection, and neoplasm. Bell's palsy was diagnosed when other recognizable causes were ruled out by careful history review, serological and radiological studies. To analyze the functional outcome, the facial nerve function was assessed by House-Brackmann (HB) facial nerve grading system at initial presentation and final visit. Medical records of 9 patients with Bell's palsy were incomplete and excluded. Complete recovery to HB grades I or II were considered satisfactory compared to incomplete recovery to HB grades III or more. Chi-square test was used to compare clinical outcome between positive and negative serological findings. A *p* value of < 0.05 is considered significant.

Results

Etiology and age distribution

The mean and median age of 25 pediatric patients with facial nerve palsy were 9.5 and 11.0 years, respectively (range,

0–18 years). There were 14 boys (56%) and 11 girls (44%). The right side was affected in 15 cases (60%), the left side cases (28%), and the site involved was not recorded in 3 cases (12%). None of the patients had bilateral involvement or presented with recurrent episodes. The most common cause of facial nerve palsy was Bell's palsy (19 cases, 76%). Three cases (12%) of temporal bone trauma, one case (4%) due to otitis media, and two cases (8%) of congenital facial nerve palsy were included (Table 1). Facial nerve palsy due to iatrogenic cause or neoplasm was not encountered. The age distribution of the patients is shown in Fig. 1. Bell's palsy was diagnosed in all age groups, but was most common in patients 15 to 18 years old (6/19 cases, 31.6%). Facial nerve palsy due to otitis media occurred in one patient within 12 months of age. Temporal bone trauma resulted in facial nerve palsy in two patients between 6–9 years of age, and one patient at 15 years of age. Congenital facial nerve palsy associated with ipsilateral microtia was identified in 2 patients shortly after birth.

Treatment modality

Management of facial nerve palsy in children varied according to the etiology (Table 2). All 19 patients diagnosed with Bell's palsy received a 10-day course of oral steroids upon presentation. The dosage was adjusted according to the child's body weight (prednisolone 1 mg/kg) for the first 5 days, and tapered over the next 5 days. Eight patients received combination therapy of steroid and an antiviral medication (Acyclovir®). Three children with delayed onset facial

nerve palsy after temporal bone trauma were treated with systemic steroids and conservative care. One patient with otitis media underwent myringotomy and intravenous antibiotics therapy. Two cases of congenital facial nerve palsy and microtia did not undergo further treatment.

Facial nerve outcome

Initially, 2 patients with facial nerve palsy after temporal bone trauma presented with HB grades V and one patient with HB grade IV. All three patients recovered to HB grade II at last follow up visit, at least after 3 months since onset. One patient with acute otitis media presented with HB grade V, which recovered completely after treatment. Two patients diagnosed with facial nerve palsy and microtia presented with HB grade IV and V, respectively, and no interval change was noted.

The functional outcome in Bell's palsy patients was evaluated using HB facial nerve grading system. In our study, initial functional status of the facial nerve was not recorded using HB grading system in 8 of 19 patients with Bell's palsy, and they were excluded. The remaining 11 patients with complete records of initial and final HB grades were analyzed for facial nerve outcome.

On initial presentation, majority of patients presented with facial nerve palsy of HB grades III or IV: 3/11 (27.3%) and 5/11 (45.5%) cases, respectively, one patient with HB grade VI, and 2 patients with HB grade II (Fig. 2A). After at least 2 months of follow up, facial nerve function of all except 2 patients showed complete recovery to HB grades I or II (Fig. 2B). Remaining 2 patients had facial nerve palsy of HB grades III and IV respectively. Individual data of facial nerve function outcome is depicted in Fig. 2C.

Findings of serological studies including antibodies to varicella-zoster virus, Epstein-Barr virus, and herpes simplex virus were compared to final facial nerve functional outcome. Nine of 11 patients (81.8%) showed complete recovery. There was no significant relationship between positive results to one or more of the serological studies (Table 3).

Discussion

In our study, facial nerve palsy in children was an uncommon

Table 1. Causes of facial nerve palsy in children

Etiology	Number of patients (n=25)
Bell's palsy	19 (76.0%)
Trauma	3 (12.0%)
Infection	1 (4.0%)
Congenital	2 (8.0%)

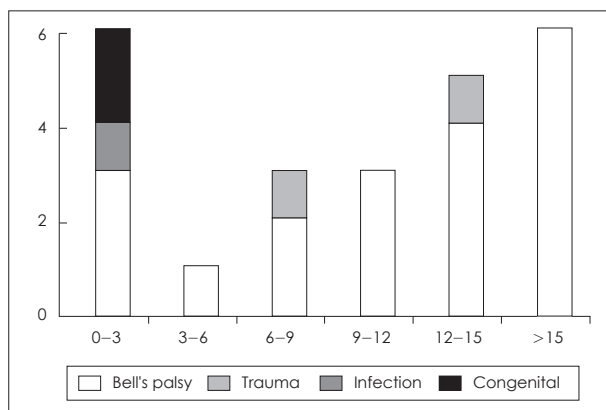


Fig. 1. Age and etiology distribution of facial nerve palsy in children.

Table 2. Treatment modality for facial nerve palsy in children

Etiology	Number of patients	Treatment modality
Bell's palsy	19	Steroid 11
		Steroid+antiviral 8
Trauma	3	Steroid+antibiotics
Infection	1	Antibiotics+myringotomy
Congenital	2	Observation

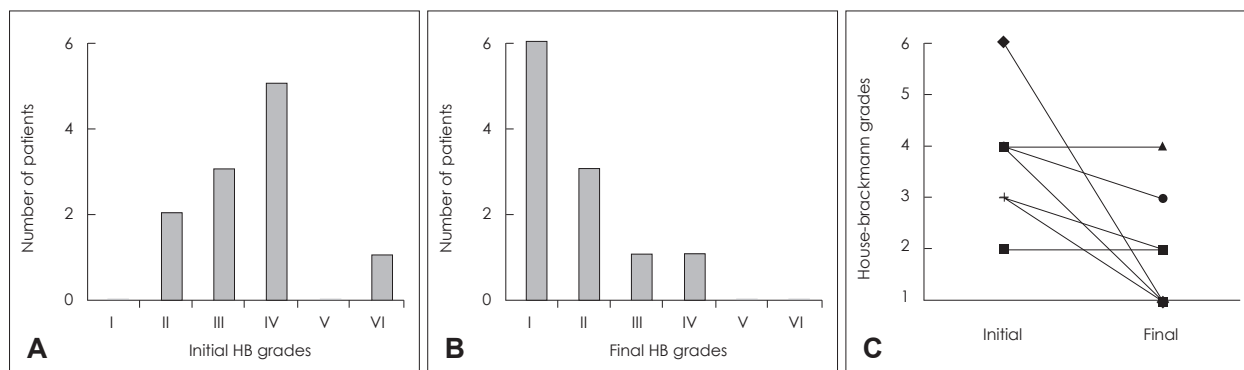


Fig. 2. Facial nerve outcome in 11 Bell's palsy patients with complete records. Facial nerve function at initial presentation (A) and final outcome (B) is compared. Individual data is shown in (C).

Table 3 Relationship between clinical outcome and positive serological findings in 11 Bell's palsy patients

Final outcome	Serological studies*		p (positive vs. negative)
	Positive	Negative	
Complete recovery	4	5	(p < 0.2)
Incomplete recovery	0	2	

Complete recovery: HB grades I or II, Incomplete recovery: HB grades III or worse. *Including varicella-zoster virus, Epstein-Barr virus, herpes simplex virus

mon presentation. The most common etiology was Bell's palsy, and other causes included trauma, infection and congenital. A clinical study of 61 pediatric patients with facial nerve palsy reported Bell's palsy as the most common etiology.⁵⁾ Similarly, an analysis of 85 children presenting with facial nerve palsy at emergency department concluded Bell's palsy as the most common cause.⁶⁾ Two recent studies of Korean pediatric patients have also reported similar etiology. Bell's palsy accounted for 104 of 157 children (66.2%) treated over a 20-year period,⁷⁾ and 16 of 24 children (66.7%) admitted over a 5-year period.⁸⁾ However, some studies have identified infection such as otitis media as the most common cause in pediatric population.^{9,10)} It has been suggested that infectious cause of facial nerve palsy is more common in younger children under the age of 2 years since toddlers are more prone to bouts of acute otitis media.⁹⁾ In our study, cases of facial nerve palsy due to infection and Bell's palsy were identified in children under 3 years of age. Widespread use of antibiotics and timely intervention in children with otitis media may account for the lower incidence of facial nerve palsy due to infection.

Diagnosis of Bell's palsy in children requires exclusion of known causes of facial nerve palsy. Trauma, infective causes such as otitis media, meningitis, Ramsay-Hunt syndrome and Lyme disease, neoplasm, systemic neurological and metabolic disorders must be considered as possible etiology.³⁾ The prognosis of Bell's palsy in children is generally very good,

and recovery is usually expected within 3 months.^{3,9,11)} Although the pathogenesis of Bell's palsy is still undefined, several viruses (including herpes simplex virus, varicella-zoster virus, human immunodeficiency virus, Epstein-Barr virus, and hepatitis B virus) are suspected to be involved in the inflammatory process observed in histopathologic studies of the facial nerve. Damage to the facial nerve is thought to be initiated by virus-induced inflammation, and followed by disruption of the neural components dependent on the host immune response.³⁾ Treatment of Bell's palsy, therefore, aims to reverse inflammatory process and counter viral inflammation. Children with Bell's palsy are usually treated with corticosteroids. However, there are few reports of randomized controlled study to recommend routine use of corticosteroids in pediatric Bell's palsy.^{12,13)} In our study, corticosteroids was prescribed in all, and the antiviral agent in about half of the children. Although the review of evidence does not recommend either medication in children with Bell's palsy, we think lack of evidence does not necessarily correlates with lack of benefit. Prospective studies to evaluate the effect of corticosteroids in children are required.

The notion of viral involvement in the pathogenesis of Bell's palsy is based on evidence of viral association in Bell's palsy. Herpes simplex virus type I DNA was recovered from facial nerve endoneurial fluid in Bell's palsy patients.¹⁴⁾ A more recent study have identified DNA of herpes simplex virus in serum and saliva of Bell's palsy patients.¹⁵⁾ Furuta et al. investigated the presence of viral DNA of varicella-zoster virus in serum and saliva of pediatric patients and suggested that viral reactivation without overt zoster presentation is an important cause of acute facial nerve palsy.¹⁶⁾ However, mere presence of antibodies to a specific virus or viral DNA may not be sufficient to conclude causality between viral infiltration and development of nerve insult. Our findings suggest that the clinical outcome of facial nerve function was not different between patients with confirmed viral infection and

others with negative serological results, as previously reported.¹⁷⁾ Further investigation is needed to substantiate the role of virus involvement in pathogenesis of Bell's palsy.

Conclusion

In conclusion, our data and others emphasize the importance of efforts to identify recognizable and treatable causes of facial nerve palsy in children, so that appropriate and timely treatment is provided. The most common cause of facial nerve palsy was Bell's palsy, and most children recovered completely.

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