

CASE REPORT

Bilateral Simultaneous Facial Palsy in a Pediatric Patient

Leyla Kansu, Nalan Yazıcı, Tarkan Ergün

Departments of Otolaryngology-
Head and Neck Surgery (Leyla
Kansu) Department of Pediatrics
(Nalan Yazıcı), and Radiology
(Tarkan Ergun), Baskent
University, Ankara, Turkey

Correspondent Author:

Leyla Kansu, MD

Department of Otolaryngology-
Head and Neck Surgery Alanya
Medical and Research Center
Baskent University Alanya,
Antalya, Turkey

Tel: +902425112522

Fax: +902425112350

E-mail: leylakansu@hotmail.com

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Bell's palsy (spontaneous idiopathic facial paralysis), which is the most common facial nerve disease, has a sudden onset. Although it is common in adults, Bell's palsy is rare in children. And its recurrence is uncommon in adults and children. Bilateral simultaneous Bell's palsy in children is rarely reported. We describe the diagnosis and treatment of a 4-year-old boy with left-sided Bell's palsy that recurred in the right facial nerve 3 weeks after the conclusion of corticosteroid therapy for the first episode of paralysis.

Bilateral simultaneous Bell's palsy in children is rarely reported. Although viral or bacterial infections, neoplasms, congenital syndromes, trauma, and metabolic or genetic factors have been associated with bilateral facial nerve palsy, the cause of that disorder remains unclear in many cases.^[1] Very few pediatric patients with bilateral simultaneous facial nerve palsy of unknown origin have been described.^[1,2] We report a 4-year-old boy with left-sided Bell's palsy that recurred in the right facial nerve 3 weeks after the conclusion of corticosteroid therapy for the initial episode of paralysis.

CASE REPORT

A 4-year-old boy with immobilization of the left side of his mouth and the inability to close his left eye was admitted to our center. His medical history was unremarkable. The results of initial physical and neurological examinations revealed no abnormalities except for left-sided facial nerve paralysis and a café-au-lait spot (2x2 cm) on his left knee. Otologic examination showed no signs of upper airway infection, and the middle ear impedances were within normal limits and did not suggest any kind of middle ear effusion. Iron-deficiency anemia has been detected with low serum iron and ferritin levels, and a high iron-binding capacity. The patient's white blood cell and platelet counts were within the reference range. The initial hemoglobin level was 8.8 mg/dL, and the differential count was normal except for hypochromia, microcytosis, anisocytosis, and poikilocytosis. Serum biochemistry analyses showed levels of LDH and uric acid within normal limits.

Cranial MRI was performed to detect if any intracranial lesion was evident mainly involving the facial nerve. Mild contrast enhancement was present on the distal intracanalicular part of the left facial nerve and this was evident on both axial and coronal T1-weighted images with gadolinium. The nerve was not swollen or distorted (Figure 1).

Corticosteroid treatment initiated at a dosage of 1 mg/kg/d and was gradually decreased over 18 days. Complete recovery occurred in 10 days. Two days after the cessation of the treatment, the patient was readmitted with facial nerve palsy on the opposite side of his face. Right-sided facial paralysis was the only abnormal finding detected on physical examination. The results of a complete blood count with differential showed mild anemia with a hemoglobin level of 10.7 mg/dL after iron supplementation at a dosage of 6 mg/kg for 3 weeks. The tympanogram was again within normal limits. The second MRI of the internal acoustic canal has been received and it showed mild contrast enhancement of the distal intracanalicular segment of the right facial nerve. In this instance the contrast enhancement of the contralateral facial nerve was disappeared (Figure 2). The levels of serum immunoglobulin E, LDH, CRP, and uric acid were within the normal range. The results of testing to reveal antibodies for herpes simplex, EBV, rubella, Lyme disease, and mumps were negative. Abdominal ultrasound studies and a chest x-rays showed no lymph node hypertrophy or infection. Corticosteroids were administered again at a dosage of 1 mg/kg/d, and the patient's paralysis resolved in 15 days. He was monitored for the next 6 months, during which he remained well and his iron-deficiency anemia resolved completely.



Figure-1: T1-weighted images with gadolinium display mild contrast enhancement of the distal intracanalicular segment of the left facial nerve on axial and coronal images.



Figure-2: Magnetic resonance imaging of the internal acoustic canal with gadolinium revealed mild contrast enhancement of the distal intracanalicular segment of the right facial nerve. The contrast enhancement of the contralateral facial nerve has disappeared.

DISCUSSION

Bell's palsy (spontaneous idiopathic facial paralysis) is the most common facial nerve disease with a sudden onset. The estimated annual incidence of Bell's palsy is 20 to 30 patients per 100,000 adult individuals and 2.7 per 100,000 children younger than 10 years.^[3,4] Recurrent Bell's palsy is uncommon in all age groups, and 6 % of all recurrences occur in children.^[2] Few pediatric patients with bilateral facial nerve paralysis of an identified or unknown cause have been reported.^[1,2,5,7]

Although the cause of Bell's palsy has not been established, microcirculatory failure of the vasorum, viral infection, ischemic neuropathy, and autoimmune reactions are among the proposed etiologies. A viral cause has been widely accepted, but no virus has been consistently isolated in patients with Bell's palsy. The evidence for the viral hypothesis has been based primarily on clinical observation and changes in viral antibody titers. The pathogenesis of the paralysis may be a viral neuropathy alone or ischemic neuropathy caused by a viral infection.^[3,8]

Although acute facial paralysis can occur during many viral illnesses such as mumps, rubella, herpes simplex, and Epstein-Barr virus infection or as a result of the reactivation of the human herpes virus in the geniculate ganglia, no clinical or laboratory evidence of any of those diseases was present in our patient.^[3,8]

Bell's palsy has been classified into the following 5 categories according to the clinical course of disease: unilateral non-recurrent, unilateral recurrent, simultaneous bilateral, alternating bilateral or recurrent bilateral.^[9] Simultaneous bilateral Bell's palsy is diagnosed when both sides of the face are affected in quick succession and without the resolution of paralysis on either side. Such patients exhibit a flat affect. We initially diagnosed simultaneous bilateral Bell's palsy in our patient, but according to the REF-T-S-B-O classification, a single episode occurs when the interval between the 2 attacks of palsy of the

contralateral nerves is 2 weeks.^[10] In our patient, that interval was 3 weeks, and a loss of affect was not evident. We suggested that corticosteroid treatment caused the rapid resolution of the first episode of palsy, so we altered the course of the disease. We concluded that our patient did not have alternating bilateral Bell's palsy because the median interval between the 2 attacks of paralysis in patients with that condition is 1 year.^[9]

Although Bell's palsy is the most common cause of simultaneous bilateral facial palsy, the most common infectious cause of it is Lyme disease, caused by *Borrelia burgdorferi*.^[11] The other causes are Guillain Barre syndrome, leukaemia, sarcoidosis, bacterial meningitis, brainstem encephalitis, syphilis, leprosy, Moebius syndrome, infectious mononucleosis, and cranial fracture.^[12,13] Fukuda et al. reported a case that has simultaneous bilateral facial palsy caused by bilateral masked mastoiditis.^[14] These etiological causes were researched in our case but it was not established.

In addition to cases with Bell's palsy, minimal facial nerve thickening with contrast attenuation are possible to be observed also in cases of Ramsey-Hunt Syndrome, Guillain Barre Syndrome, postoperatively, Lyme's disease, traumatic facial paralysis and following radiotherapy. In cases of facial nerve neurinomas and haemangiomas, on the other hand, the nerve is typically thickened and the neighboring bony structures are eroded. In neurosarcoidosis cases the facial nerve demonstrates severe enhancement in addition to contrast attenuation.

Corticosteroids are the most commonly used agents for reducing inflammation and edema in the nerve sheath.^[15] The prognosis of our patient was favorable with a late resolution of the second palsy compared with the first one with steroid therapy. The duration of the palsy in our patient was 25 days. Eidlitz-Markus and colleagues reported that up to 8 weeks was required for complete recovery from idiopathic facial nerve palsy in pediatric patients.^[12] According to Chen

and colleagues, complete recovery from bilateral recurrent Bell's palsy required 2 weeks to 4 months.^[6] However, the details of treatment were not clear in those reports. Some recent reports do not reveal whether steroid treatment resulted in a statistically significant difference in the recovery from Bell's palsy in pediatric patients.^[15,16] Large randomized controlled trials should be conducted to address that issue.

The cause of recurrent facial nerve palsy in children should be investigated. In pediatric patients with Bell's palsy of unknown origin, treatment with a steroid should be considered. Although the duration of corticosteroid treatment for the first episode of palsy was shorter than second episode in our patient, it did not change the course of the disease, which recurred on the opposite side of the patient's face. The period of recovery from recurrent paralysis was longer than that required for the resolution of the initial attack of palsy.

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