Acute onset of facial nerve palsy associated with Lyme disease in a 6-year-old child

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Abstract

Pediatric facial nerve palsy (FNP) can result from a variety of etiologies including Lyme disease, varicella, primary gingivostomatitis, herpes zoster oticus (Ramsay Hunt syndrome), coxsackievirus, trauma, otitis media, HIV, diseases causing tumors or demyelinations, compressions, and possibly Epstein Barr virus. Lyme disease has been implicated as the cause of over 50% of the FNPs in children. The paralysis of the facial nerve disturbs motor function to the muscles of facial expression and results in a flaccid appearance of the face (unilateral or bilateral). This case report details undiagnosed Lyme disease presenting as a facial palsy in a 6 year, 5 month-old white female. The palsy was recognized and consultation with the child's physician prompted definitive diagnosis and treatment. A review of the literature and the implications of facial nerve palsy are discussed. (*Pediatr Dent*. 2002;24;572-574)

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Paralysis of the facial nerve (commonly referred to as Bell's palsy) is a well-recognized phenomenon. However, the etiology and presentation of facial nerve palsy is not commonly discussed in the pediatric dental literature even though it occurs most commonly in children and young adults.[1]

Although sometimes idiopathic in nature, facial nerve palsy (FNP) often results from an infectious or traumatic cause. In the past, middle ear infections were a common bacterial etiology of FNP. However, a more recent study by Cook et al indicated that Lyme disease has surpassed otitis media as the most likely cause of FNP in children.[2]

The gender distribution in FNP is approximately equal and 85% of patients recover within 2 months.[3,4] Longer-term sequelae have been shown to occur in 4% to 16% of patients afflicted with the disease.[5] The long-term emotional and physical effects of this palsy can be disfiguring and harmful. Self-esteem, childhood and adolescent socialization skills, speech and ocular health can all be affected.

Signs and symptoms of a FNP include the loss of ability to use the muscles of facial expression. The ability to smile, frown, blink, raise eyebrows and talk normally is diminished. Inflammation or infection of the facial nerve as it enters the auditory meatus and/or parotid gland can affect the ability of the nerve to perform its normal functions.[1]

Case report

A 6-year, 5-month-old Caucasian female presented with an abnormal facial appearance. Upon visual examination, it was noted that she spoke by pulling her left lips predominately off to one side. According to her mother, she had bitten her right cheek several days prior and complained that it hurt to move her cheek.

Her medical history was positive for severe asthma caused by mold and pollen allergies for which she takes Claritin, Pulmicort, and Preventil daily. She receives allergy injections on a regular basis. She has no known drug allergies or hospitalizations.

An intraoral examination revealed evidence of a healing bite mark on her right cheek. The child was seen 3 days later and there was no resolution of the facial anomaly. The inner cheek appeared to be healed but the child claimed she still couldn't "talk on that side." She reportedly was also falling asleep with a "funny look" on her face.

Suspecting Bell's palsy, a limited cranial nerve examination was performed. The child was asked to close her eyes. Her left eye closed fully but her right eye exhibited a weak partial closure. Further results demonstrated an inability to move her eyebrow, wrinkle her nose, smile or frown on the ipsilateral side. Masticatory function was basically intact. Eye movement in the horizontal and vertical planes was normal.

All other motor functions appeared to be within normal limits.

Her parents were informed of the diagnosis, and a telephone consultation with her pediatrician prompted the concern of Lyme disease, otitis media or viral infections such as HSV-1 or Epstein Barr (EB) virus. Since an infection of bacterial origin is the only easily treatable cause of FNP, her pediatrician empirically prescribed Amoxicillin 50 mg/kg tid for 14 days and suggested the child receive a full diagnostic work-up including physical exam, CBC and serological screens for Lyme disease and EB virus.

The next day the patient developed constitutional symptoms of fever and overall malaise. In hindsight, her parents did report a small, red rash on her arm a couple of weeks prior to the onset of the FNP that likely represented the initial tick bite. At the time, her parents had believed it was a localized reaction to her allergy injections.

Her serologic tests came back positive for both Lyme disease and the EB virus. Her physician felt that Lyme disease was the primary cause of the FNP. Since she lacked several signs of acute mononucleosis (ie, hepatosplenomegaly and sore throat), he felt the serological findings of EB were likely more incidental than clinically significant. She continued on the same Amoxicillin regimen for a total of 4 weeks.

Her constitutional symptoms resolved after 2 weeks of antibiotic therapy and she reportedly was "acting like her normal self." One month after diagnosis the patient regained partial movement of her right eyebrow, but her eyelids, nose and lips showed no improvement. Complete resolution of the facial nerve palsy occurred 3 months after the initial onset of symptoms.

Discussion

To fully understand FNP, a review of the functions of facial nerve VII is indicated. Facial nerve VII exits the skull from the internal acoustic meatus. Motor components enter the stylomastoid foramen, penetrate through the parotid gland and supply the muscles of facial expression.[6] Fibers also join with the lingual nerve to form the chorda tympani while another portion of the nerve gives sympathetic fibers to the lacrimal gland and other mucous membranes.[6]

The facial nerve also innervates smaller accessory muscles. Portions of the facial nerve join with the trigeminal and supply taste to the anterior two-thirds of the tongue. Taste fibers also travel to the palate. The submandibular, sublingual, lacrimal, and minor salivary glands are partially innervated by VII as well.[6] A summary of the major motor functions of the facial nerve is seen in Table 1.

At the entrance to the narrowest portion of the facial canal, the nerve is most likely to be damaged and produce symptoms of FNP.[7] Depending on the location of the inflammation or demyelination, lacrimal function can also be disturbed. If this occurs, the risk of corneal abrasions increases since the eye is unable to close completely or tear.[1]

A study by Cook et al suggests that Lyme disease may account for up to 50% of all FNP in children, thus making it the single most common cause.[2] Other common etiologies include primary herpetic gingivostomatits, herpes zoster, varicella, mumps, rubella, acute otitis media, HIV (indirectly), meningitis, Guillain-Barre syndrome, coxsackievirus, sarcoidosis, Melkersson-Rosenthal syndrome, tumors or other compressions, trauma and/or postsurgical complications, and diseases that cause nerve demyelination.[1,8] Idiopathic Bell's palsy is different from FNP and is hypothesized to occur due to genetic, vascular, metabolic and/or autoimmune reactions and unknown infectious agents.[8,9]

As stated previously, the most likely cause of FNP is inflammation of the nerve as it exits the interal acoustic meatus. Although the patient had reportedly bitten her cheek, the trauma was not located in an area where the facial nerve would have been affected by this incident. The trauma was minor and superficial and likely occurred secondary to the Bell's palsy while chewing.

Lyme disease is caused by a spirochete known as *Borellia burgdorferi*. The disease is not contagious between humans and is spread via an Ixodes genus deer tick bite.[2,10,11] Lyme disease has been noted in 49 of the 50 states with the overall incidence ranging between 20-100/100,000 people in more endemic areas.[12] Children between the ages of 5-10 years old are most often affected by Lyme.[12] The exact percentage of children with Lyme disease who present with a Bell's palsy in the first or second stages was not found in the literature review.

Lyme disease characteristically progresses through 3 stages of infection if left untreated. The chief complaints in the initial stage are usually fever, arthralgias and myalgias, headache, malaise and neck stiffness. Most notably, a classic rash known as erythema migrans usually develops around the site of the tick bite.[2,10,13] In children, the rash is commonly found in the head and neck region.[2,10,11] However, over half of all patients may report no history of a rash or it may be confused with other dermatologic conditions.[2,10,14] FNP is a very common neurologic sign of Lyme disease and occurs bilaterally more frequently in children than in adults who have been infected.[2,11,13,14] FNP often manifests in the first or second stages of this disease.[2,15]

Table 1. Major Functions of Cranial Nerve VII

Muscles innervated	Function
Occipitofrontalis muscle	Raises eyebrow
Obicularis oculi	Blink reflex
Levator labii superioris	Raises/everts lip
Levator anguli oris	Raises/protruded/compresses lip
Depressor labii inferioris	Depresses lower lip
Buccinator	Presses cheek on teeth
Obicularis oris	Compresses/protrudes lip

The secondary stages of Lyme disease often present with lymphocytic meningitis, neuritis, mild encephalitis and cardiac conduction defects. If left untreated in stage II, the disease can progress to the third stage with dehabilitating arthritis and chronic CNS disease.[2,10,16]

After making a diagnosis of FNP, it is important to first rule out the treatable, infectious causes such as Lyme disease and otitis media. The standard treatment for Lyme disease is Amoxiciliin 50 mg/kg 24 hours tid for 14 to 21 days. In children over the age of 8, Doxycycline 100 mg tid for 14 to 21 days can also be used effectively. In cases of FNP, the antibiotic course is extended for 21 to 28 days. Steroids are usually not recommended.[12] The treatment of FNP resulting from viral infections or trauma can be palliative or surgical instead of chemotherapeutic. In some instances, acyclovir is used in cases caused by the herpes simplex virus.[1]

It is important to recognize that FNP can occur in the pediatric population and usually results from an infection, trauma, or compression. The most likely cause of FNP in children is Lyme disease, as it accounts for more than 50% of reported incidents. Although most cases resolve completely, a small percentage of them may have long-term consequences. The dental implications of FNP may include the need to pay particular attention to shaded eye protection while performing treatment and the need to recognize the potential for increased postanesthesia trauma and/or trauma to the soft tissues during normal mastication.

Although there is no documented reference implicating prolonged FNP and occlusal changes, it has been suggested that changes in the equilibrium of facial muscles and those of mastication can lead to occlusal changes.[17,18] The movement and shifting of teeth may occur with a lack of tone from muscles such as the buccinator, mentalis and/or obicularis oris. Recording the occlusion of patients with FNP and monitoring those with prolonged FNP is prudent.

References

1. Roob G, Fazekas F, Hartung H. Peripheral facial palsy: Etiology, diagnosis and treatment. *Eur Neruol.* 1999;41:3-9.
2. Cook S, Macartney K., Rose C, et al. Lyme disease and seventh nerve paralysis in children. *Amer J Otolaryngology*. 1997;18;5:320-323.
3. Katusic SK, Beard CM, Wiederholt WC, et al. Incidence, clinical features and prognosis in Bell's palsy, Rochester Minnesota, 1968-1982. *Ann Neruol*. 1986; 20:222.
4. Kukimoto N, Ikeda M, Yamada K, et al. Viral infections in acute peripheral facial paralysis nationwide analysis centering on CF. *Acta Otolaryngol Suppl Stokh*. 1988;446:17-22.
5. Peitersen E. The natural history of Bell's palsy. *Am J Otol*. 1982;4:107-111.
6. Moore K. *Clinically Oriented Anatomy.* 3rd ed. Philidelphia, Pa: Williams & Williams; 1992.
7. Karnes WE. Diseases of the seventh cranial nerve. In: Dyck PJ, Thomas PK, Griffin JW, Low PA, eds. *Peripheral Neuropathy*. 3rd ed. Philadelphia, Pa: Saunders; 1993:818-836.
8. Bauer C, Coker N. Update on facial nerve disorders. *Otolaryngologic Clinics of North America*. 1996; 29:445-454.
9. Williamson IG, Whelan TR. The clinical problem of Bell's palsy: Is treatment with steroids effective? *Br J Gen Pract*. 1996;46:743-747.
10. Belani K, Regelmann W. Lyme disease in children. *Rheum Dis Clin North Am.* 1989;15:679-690.
11. Christen HJ, Bartlau N, Hanefeld F, et al. Peripheral facial palsy in childhood–Lyme Boreliosis to be suspected unless proven otherwise. *Acta Paediatr Scand*. 1992;79:1219-1224.
12. Shapiro E. Lyme disease. In: Behrman, et al, eds. *Nelson Textbook of Pediatrics* 16th ed. Philadelphia, Pa: WB Saunders Co; 2000:910-914.
13. Belman AL, Iyer M, Coyle PK, et al. Neurologic manifestations in children with North American Lyme disease. *Neurology*. 1993;43:2609-2614.
14. Moscatello AL, Worden DL., Nadelman RB, et al. Otolaryngologic aspects of Lyme disease. *Laryngoscope*. 1991;101:592-595.
15. Gerber MA, Shapiro ED. Diagnosis of lyme disease in children. *J Pediatr.* 1992;121:157-161.
16. Lesser THJ, Dort JC, Simmen DPB. Ear, nose and throat manifestations of Lyme disease. *J Laryngol Otol*. 1990;104:301-304.
17. Graber TM. *Orthodontics Principles and Practice*. 3rd ed. Philadelphia, Pa: WB Saunders Co; 1972:287.
18. Profit WR. *Contemporary Orthodontics*. St. Louis, Mo: CV Mosby Company; 1986:104-109.