

Bell's Palsy: A Case Study

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Abstract

This paper discusses Bell's palsy in a five year old child who was evaluated at a pediatric emergency department. She awakened with the symptoms of left sided facial paralysis. The mother denied any further symptoms. Past medical history was noncontributory, although family history revealed that a sibling had previously experienced an episode of Bell's palsy. Differential diagnosis is discussed as are treatment modalities for idiopathic Bell's palsy, infectious causes of facial paralysis and central nervous system causes (CNS).

The following information was obtained at a pediatric emergency department situated in a medical center. The patient is a five year old hispanic female who was brought in by her mother with complaint of "twisted face".

HISTORY AND PHYSICAL EXAMINATION

History of Present Illness: A five year old hispanic female is accompanied by her mother with complaint of twisted face upon awakening this morning. Mother states that food tends to dribble from the left side of her mouth and she has difficulty with left eye closure. She denies excessive eye tearing, fever, cough, vomiting, diarrhea, cold or recent travel. No gait disturbances, weakness, numbness, hyperacusis, tingling and taste disturbances noted.

Allergies: Denied

Medications: Denied

Past Medical History: Unremarkable except for one episode of right otitis media 6 months prior. No history of varicella or parotitis. Immunizations are up to date, and a PPD done 2/97 is negative. Birth history is non-contributory and growth and development are consistent with anticipated standards.

Family Medical History: Mother is age 40 is well. Father is age 44 and well. The patient has two female siblings - a 4 year old with resolved case of Bell's palsy and a healthy 1 year old. Grandparents are living with history significant for angina in maternal grandmother.

Personal/Social History: Patient lives in an apartment. The neighborhood is noisy, dirty and unsafe in some areas. She

shares a room with her two siblings. She enjoys drawing, playing with other children, chores and trying on new outfits, and demonstrates interest in kindergarten and attends church.

Review of Systems: General: Well nourished child of appropriate height and weight for stated age. Skin/Hair: No lesions, or masses. Nails: Without changes. Head and Face: Denies dizziness and frequent headaches. Eyes: Denies use of corrective or prosthetic devices, diplopia, pain, or photophobia. Last eye exam 8/96 was normal. Ears: Denies prosthetic devices, infections, and tinnitus. Nose and Sinuses: Denies olfactory deficits. Mouth and Throat: Denies hoarseness or lesions. Last dental exam 5/97, without caries. Neck: No masses/node enlargement or pain with movement or palpation. Breasts: Tanner Stage 1. Respiratory: Denies cough, shortness of breath, or wheezing. Heart: Denies murmur, chest pain or swollen extremities. Heme: Denies anemia, bruising, bleeding. GI: Denies abdominal pain, nausea and vomiting. GU: Denies urgency, frequency or bloody urine. Genitalia: Tanner Stage 1. Musculoskeletal: Status post fractured radius 2 years ago. Without current problems. Neuro: Denies seizures, lack of coordination, tingling, difficult speech, tremors, tics or weakened grips. Mental Health: Denies irritability, troubled speech, learning disabilities. Endocrine: Denies weight gain or loss, heat or cold intolerances, thirst or polyuria. Developmental: Denies retardation. Skips/hops/broad jumps/copies shapes. Dresses and undresses with minimal supervision. Displays normal sexual curiosity and identifies coins and four out of five colors.

Physical Examination: Slim, cheerful five year old hispanic

female in no apparent distress. General: Alert and oriented times 3, BP 90/50; p 100; RR 20; T 99po; ht 45 in; wt 48 lbs (95th percentile). Skin: Warm to touch, pink. Brisk capillary refill. HEENT: Unremarkable except for left facial asymmetry. TM's pearly bilaterally.

Pharynx without erythema or exudate. Neck supple without adenopathy. Thyroid borders within normal limits. Without carotid bruits. Chest: Lungs clear, bilateral breath sounds, equal and resonant. Vocal and tactile fremitus equal. Cardiovascular: S1S2 without murmur, rate and rhythm regular. No clicks, rubs or extra heart sounds. Abdomen: Soft and non-tender. Normoactive bowel sounds. Palpation reveals no masses or organomegaly. Extremities: Full range of motion of all extremities. Radial, femoral, dorsalis pedis and posterior tibial pulses equal bilaterally. Strength 5+/5+ in all extremities. Neurologic: Pupils equal and reactive to light. Cranial nerves II-XII grossly intact except for 7th left peripheral cranial nerve palsy. No atrophy, fasciculations, tremors noted. Gait, heel - to - toe, heel and toe walking, knee bends, and hopping all within normal limits. Romberg negative. Grips and arms strong and equal bilaterally. Sensory exam reveals pain, vibration, light touch and stereognosis intact. Reflexes 2+/2+ bilaterally.

Differential Diagnosis: Based on the subjective and objective data a list of differential diagnosis can be developed. The differential diagnosis appropriate for this clinical presentation may include:

1. Bell's palsy
2. Infectious disease, including herpes simplex, Lyme's disease and Epstein- Barr.
3. CNS causation, including invasive tumor or vascular lesion.

PLAN

Diagnostics: CBC with differential, sedimentation rate, EBV/Lymes titer: follow-up on EBV/Lyme titers taken. Infectious diseases such as viral illnesses, EBV/Lyme can be eliminated by evaluation of the diagnostic tests listed above.

Therapeutics: Artificial tears to left eye 2 drops tid in order to offset corneal abrasions secondary to impaired tearing. Eye patch during night time if eye is not closing properly.

Educative: Educate patient and family about disease process, therapeutic interventions and anticipated course of the disease. Health maintenance education should include

information regarding annual PPD, dental care, routine physical at 6 year old, height/weight/hearing/vision/vital signs/UA/lead level/DPT, polio and MMR to be provided by primary care provider.

Anticipatory Guidance: Injury prevention, good parenting practices, sex education, nutrition, discipline as well as information regarding the course of the acute illness.

Follow-up: RTC in 1 week for reevaluation with pediatric neurology or sooner if any other problems occur.

BELL'S PALSY

The diagnosis is predominantly one of exclusion and based on clinical findings (4). Negative history should include trauma, local infection, or disease of the central nervous system.

The facial nerve originates in the pons. Its brachial motor fibers exit the brainstem at the level of cerebellopontine angle to enter the internal auditory meatus on the opposite side. These fibers travel through the geniculate ganglion, where they enter the facial canal, eventually leaving the skull through the stylomastoid foramen. Distal to this foramen, the large nerve fibers branch into small fibers that supply the muscles of facial movement. Lesions in the vicinity of the stylomastoid foramen have been identified during surgical exploration (5).

Complete paralysis of the nerve results in loss of motor, sensory, and parasympathetic function ipsilateral to the lesion. Motor paralysis is characterized by complete loss of voluntary and automatic expression over the affected hemiface. The involved side of the face is immobile during attempts at expression. There is flattening of the nasolabial fold, sagging at the corner of the mouth, and displacement of the lips towards the unaffected side. The forehead flattens and the eyebrows lose their symmetry. The palpebral fissure widens. On attempts at eye closure, Bell's phenomenon is seen. This phenomenon is characterized by rotation of the affected eyeball in an upward position with inability to close the lid. Loss of blinking results in collection of tears in the lower lid. The ipsilateral corneal blink reflex is absent (5).

Symptoms generally present over a 24-48 hour time period with 60% of patients experiencing a viral prodrome, characterized by stuffy nose, sore throat, and generalized achiness (6). Bell's palsy often follows an upper respiratory infection, most often viral, and is believed to be due to postinfectious demyelination of the facial nerve (7). Fifty-

percent of patients will also experience sensory loss of the face, neck or tongue, and 90% will experience hyperacusis which is painful sensitivity to sound (4). Drinking and eating may be affected secondary to paresis, and lacrimation may be decreased (7).

DIAGNOSIS

As stated previously, the diagnosis of Bell’s palsy is predominantly one based on clinical findings and the exclusion of other possible causes of facial paralysis. The practitioner bases her diagnosis on a thorough neurologic examination, history, and physical, history of prodromal viral infection, as well as negative EBV titers/ Lyme titer. Only when all other causes are ruled out can a diagnosis of Bell’s palsy be made.

Diagnostic assessment should include an audiogram in order to evaluate function of the auditory nerve. T1 magnetic resonance imaging (MRI) with contrast may also be ordered, especially in instances of unresolved paresis or aberrant presentation. Bell’s palsy is often associated with segmental enhancement not extending into the auditory canal on MRI (4). Other testing may include the Schirmer test, stapedial reflex, and evaluation of taste and salivation. The Schirmer test measures the amount of tears secreted in 5 minutes in response to irritation. It is measured by a filter paper strip. A normal response is 15 mm of strip moistened. The stapedial reflex is the reflex of the stapes of the inner ear (4). Electroneurography, electromyography, and blink reflex have also been used in evaluation (8).

TREATMENT

Management is predominantly supportive. Eye care is essential. The affected eye should be taped shut during the night if the patient is unable to fully close it. Artificial tears may also be administered 2 drops TID. It is the responsibility of the nurse practitioner to ensure that the patient and family understands the importance of injury prevention and the course of the disease.

In this case, because the patient is a child, it is important that annual PPD, dental care and well child visits be scheduled on a regular basis. Anticipatory guidance by the nurse practitioner should include injury prevention education, good parenting practices, sex education, nutrition and appropriate discipline techniques.

The administration of steroids is controversial. Eighty-four percent of patients recover without any intervention whatsoever (4). Prognosis, of course, is dependent on age,

severity of symptoms, and concomitant disease processes. Recovery occurs within 2-4 weeks following presentation with complete recovery by 6-12 months following paresis (7). In severe cases, oral steroids such as prednisone are often administered in doses of 40-60 mg/day on a tapered dose over 7 to 10 days in order to facilitate resolution of symptoms (5). This intervention is based on the belief that Bell’s palsy results from inflammation and edema of the nerve secondary to a viral causation (7).

Complications include incomplete recovery of facial nerve function, (10-25%) faulty reinnervation, and reoccurrence (7%) (5). Contractures of facial muscles and facial spasms have also been known to occur. Surgery should only be considered when facial paralysis is complete and extensive. In these instances decompression of the facial nerve has been recommended (4). In extreme cases, physical therapy may also be indicated.

INFECTIOUS CAUSES

Common infectious causes of sudden onset facial paralysis include such conditions as Lymes disease, Epstein Barre, Herpes simplex and HIV disease, among others.

Figure 1

Table1: Facial Weakness in Childhood

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| <p>Congenital/structural</p> <ul style="list-style-type: none"> Chari malformation Depressor anguli oris muscle absence (orbifacial syndrome) Inner ear and/or facial nerve malformation Holcus syndrome Spina bifida <p>Ischemic</p> <ul style="list-style-type: none"> Face-oculohumeral dystrophy Facial arterial neuropathy (transient) Face Lock disease Myasthenia gravis (anticholinergic mediated) Myotonic dystrophy Hemifacial microsomia <p>Infectious-inflammatory</p> <ul style="list-style-type: none"> Bacterial meningitis Bell's palsy Casper-Bar infection (infectious mononucleosis) Gullen-Barre syndrome Hiller-Fisher syndrome Mycoplasma pneumoniae infection Lyme disease (borreliosis) Orbita media and mastoiditis Parotitis Poliovirus Ramsay Hunt syndrome (shingles virus) Sarcoidosis Toxoplasma Tuberculosis | <p>Trauma-nerve compression</p> <ul style="list-style-type: none"> Forceps pressure during delivery <p>Cleftocranial dysostosis</p> <ul style="list-style-type: none"> Herpes zoster Hyperostosis frontalis interna Increased intracranial pressure Pituitary bone fracture Pressure from maternal sacrum <p>Metabolic conditions</p> <ul style="list-style-type: none"> Hyperparathyroidism Hypoparathyroidism Congenital infantile hyperparathyroidism Osteopetrosis <p>Neoplasms</p> <ul style="list-style-type: none"> Strabismic glaucoma Parotid gland tumors <p>vascular</p> <ul style="list-style-type: none"> Arterial hypertension Vascular syndromes of the cranial nerves <p>Other</p> <ul style="list-style-type: none"> Idiopathic cranial neuropathy Melkersson-Rosenthal syndrome Multiple sclerosis Myasthenia gravis (immune mediated) Myasthenia gravis (transient neonatal) |
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Source: Adapted with permission from Smith S.A. (1994). Pediatric Neuropathies in Children. In K. F. Swaiman, Pediatric Neurology: Principles and practice (2nd Ed., pp. 1429-1452); Mosby-Year book, Inc. St. Louis, Missouri (7).

Diagnostic evaluation should includes a complete blood count (CBC) and sedimentation rate to evaluate infectious

etiology. Cerebral spinal fluid (CSF) studies add additional information if borrelia (Lyme) infection is considered. Testing for Epstein Barre and HIV should also be considered.

History and physical examination findings provide direction as to possible causation. For instance, a history of previous tick bites or recent history of travel in a Lyme endemic area may lead the practitioner to look for Lyme disease. The presence of a macular rash may be indicative of Lyme's varicella or herpes (1). Generalized adenopathy may direct the practitioner towards infectious mononucleosis (1). Positive risk factors as well as other systemic immunodeficient symptoms may lead the practitioner towards evaluation of cranial nerve neuropathy in a HIV patient (2).

Because of the wide variety of possible infectious agents, treatments may vary. Lyme's disease is generally treated with systemic antibiotics such as doxycycline, while mononucleosis is of viral origin and does not require treatment. Likewise herpes simplex causation may necessitate treatment with acyclovir and steroids, while HIV infection has its own specific treatment protocols.

CENTRAL NERVOUS SYSTEM

Lesions in the deep frontal cerebrum or pons can produce an ipsilateral facial paralysis (6). Of times, this can be volitional, as seen with deep frontal lesions, or associated with paralysis of the abducens in pontine lesions. A cerebellopontine angle lesion can also cause facial paralysis associated with tinnitus and deafness (6).

Generally symptoms associated with CNS lesions do not develop spontaneously, but rather evolve over time. There is no associated prodromal syndrome which can be described, as there is in Bell's palsy or occasionally in infectious causation. Associated symptoms in the pediatric population may include headaches, lethargy, personality changes, and

episodes of vomiting associated with increased intracranial pressure.

Particular care should be taken when evaluating patients under two years of age, as facial palsies are seen more commonly in this age group, secondary to CNS lesions, than in adults (3).

Diagnostic criteria involve evaluation via CT scan or MRI. Treatment is dependent on grading, location, and metastasis associated with the lesion, and may involve surgery, radiation, or chemotherapy.

SUMMARY

The case study reviews the clinical presentation of a pediatric patient with Bell's palsy. While this is a relatively rare event in the pediatric population, Bell's palsy does occur and behooves the practitioner to become familiar with the diagnostic criteria and appropriate interventions for this disease process.

References

1. Bunch B, Avner J. Neurologic Emergencies. In E Crain, J Gershel. *Clinical Manual of Emergency Pediatrics*. 3rd ed. New York: McGraw Hill; 1997;437-74.
2. Geraint J. All that palsies is not Bell's. In *Journal of the Royal Society and Medicine*. 1996;89,184-87.
3. Inamura H, Aoyagi M, Tojima H, Kohsyu H, Koike Y. Facial nerve palsy in children: Clinical aspects of diagnosis and treatment. In *Acta Otolaryngology*, suppl, 511. 1994;150-52.
4. Jabor MA, Gianoli G. Management of Bell's palsy. In *Journal Louisiana State Medical Society*. 1996;148, 279-83.
5. Lynch K. Facial nerve disorders. In E. Barker, editors. *Neuroscience Nursing*. St. Louis, Missouri: Year Book Inc.; 1994;455-58.
6. Rengachary SS. Cranial nerve examination. In RH Wilkins, SS Rengachary, editors. *Neurosurgery*. 2nd ed. New York: McGraw Hill; 1996;67-86.
7. Smith SA. Peripheral neuropathies in children. In KF Swaiman. *Pediatric Neurology: Principles and Practice*. 2nd ed. St. Louis, Missouri: Mosby-Year book, Inc.; 1994;1429-52.
8. Wong V. Outcome of facial nerve palsy in 24 children. *Brain and Development*. 1995;17, 294-96.

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