Otolaryngology Case Reports 20 (2021) 100297

Contents lists available at ScienceDirect

Otolaryngology Case Reports

journal homepage: www.elsevier.com/locate/xocr

Facial nerve palsy in children: A case series and literature review

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ARTICLE INFO

Keywords:
Facial paralysis
Mastoiditis
Bell's palsy
Facial nerve weakness

ABSTRACT

The facial nerve is the most common cranial nerve to have a disorder. In adults, the incidence has been reported to be as high as 40 cases per 100,000 patients annually. In the pediatric population, the frequency of facial nerve palsy is much less. It is estimated that children over the age of 10 have an incidence of 10 per 100,000 annually and those under the age of 10 to be less than 3 per 100,000 annually. Nonetheless, when children are affected, it has a tremendous impact on the child and can cause great distress to the family. As with adults, the most common etiology of facial palsy in children is idiopathic (Bell's Palsy). However, the most frequent identifiable causes of facial palsy in children are different from that in adults. In children, when not idiopathic, infection followed by trauma and congenital conditions are the most commonly etiologies of facial nerve palsy. The diagnosis, evaluation, treatment and outcomes of facial palsy in children will be reviewed. In addition, a series of representative cases of pediatric facial palsy at our children's hospital will be presented.

Introduction

Facial nerve palsy is the most common cranial nerve dysfunction in children and adults. While it occurs less commonly in children, facial nerve paralysis can have a significant effect on the child and cause parents a great amount of distress. The long term effects on vision, eating, drinking, psychological issues, self-esteem, and quality of life are well-established [1]. The most common etiologies of facial paralysis in children are different than what is found for adults. When the etiology can be identified, due to anatomical and immunological factors, simple acute bacterial otitis media is the most common disease to cause facial palsy in a child. In addition, as children are prone to viral exposure, this adds to the frequency of infectious etiologies of facial palsy. Fortunately, due to the natural resilience of children and their inherent advantage in neuroplasticity potential compared to adults, the prognoses of these younger patients overall, is favorable. However, when a child does not show relatively rapid improvement, one must consider uncommon causes such as malignancies and metabolic diseases. Here we will review the diagnosis, etiologies and treatment of facial palsy in children as well as present a series of representative pediatric cases at our institution.

Methods

A Pubmed database search was performed using keywords "facial nerve paralysis in children" and "cranial nerve VII palsy". With this survey of the current literature, we review the etiologies, treatment, and prognosis of facial palsies in children. Through retrospective chart review, we report the clinical courses of 6 cases of patients under 18 years of age who presented to the Children's Medical Center for facial nerve weakness.

Anatomy

The facial nerve (cranial nerve VII) carries both motor, sensory, and parasympathetic functions (Fig. 1). It is particularly susceptible to injury due to its long intraosseous course. The nerve occupies 25–50% of the total diameter of the Fallopian canal. It is believed that the rigid bony canal cannot accommodate the nerve when it becomes inflamed and swollen, leading to vascular compromise of the nerve [2]. The motor nerves, which innervate the facial muscles, originate from the motor nucleus of VII in the pontine tegmentum. After it exits the brainstem, the nerve then joins with nervus intermedius, which carries the preganglionic parasympathetics fibers that innervate the lacrimal, sublingual, and submandibular gland, as well as afferent taste fibers.

There are six segments of the facial nerve in the temporal bone [3].

1. Intracranial: from brainstem to the porus of the internal auditory canal (IAC).

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https://doi.org/10.1016/j.xocr.2021.100297
Received 28 March 2021; Accepted 31 March 2021
Available online 15 April 2021

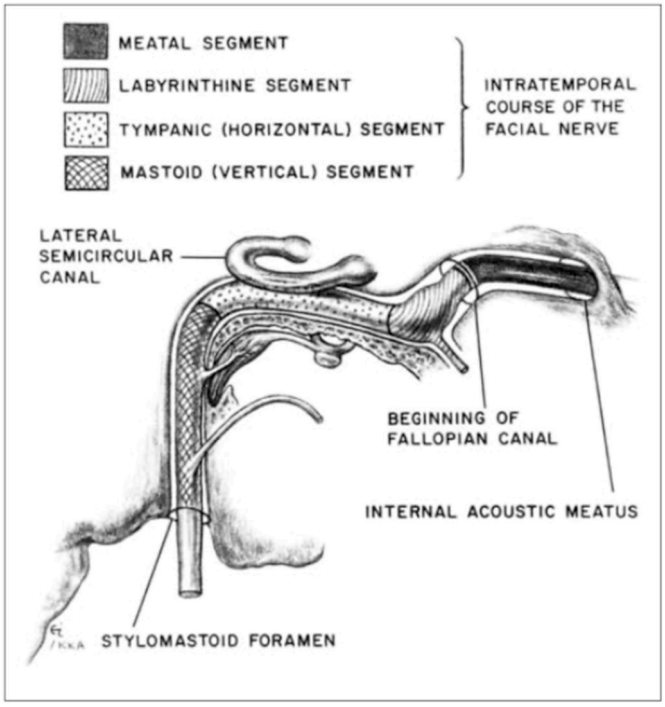

Fig. 1. Diagram of the course of the facial nerve within the temporal bone. Illustration by Kedar K. Adour, MD. Reprinted with previous permission.

2. Meatal: from where the nerve enters the porus of the IAC to the meatal foramen. The facial nerve runs in the anterior superior quadrant of the IAC.
3. Labyrinthine: from the meatal foramen to the geniculate ganglion. This is the narrowest portion of the Fallopian canal. The geniculate ganglion is where the facial nerve gives off its first branch (the greater superficial petrosal nerve).
4. Tympanic: at the geniculate ganglion, the nerve takes a sharp 40–80° turn posteriorly, running horizontally along the medial wall of the tympanic cavity.
5. Vertical: the facial nerve takes another turn inferiorly and runs vertically through the mastoid until it exits the stylomastoid foramen. This is where the chorda tympani and nerve to the stapedius muscle branch off.
6. Extratemporal: after it exits the stylomastoid foramen, the facial nerve courses anteriorly, lateral to the styloid process, and enters the posterior surface of the parotid gland to innervate the facial muscles.

Epidemiology and etiology

Facial nerve paralysis is relatively rare and affects about 15–40 per 100,000 adults annually [4,5]. It occurs 2–4 times less frequently in children than in adults. The incidence in children younger than 10 years old is 2.7 per 100,000 each year and in children older than 10 years of age, it is 10.1 per 100,000 per year [5,6].

There are many possible etiologies for facial nerve weakness, but the most common causes in children are different from those in adults.

Acquired facial nerve paralysis

The majority of cases, in both children and adults, are idiopathic (Bell's Palsy). In children, the incidence of Bell's palsy is estimated to be 6.1 cases per 100,000 per year in children ages 1–15 years old [7]. Reported rates range from 9% to 78.6% of all pediatric facial paralysis cases [8]. Other identifiable acquired causes, including infectious and traumatic, are more common in children than in adults.

Infectious etiologies account for 12–36% of pediatric cases, compared to 0.16–3.1% in adults [8,9]. Acute otitis media (AOM) remains the most common infectious cause of facial nerve paralysis in children [8,10,11]. However, due to widespread use of antibiotics, the rate of complications from AOM, including facial nerve paralysis, has decreased.

An exception to the point above is that in endemic areas, Lyme disease is the most common infectious cause [12], accounting for up to 50% of facial palsy in children in these geographical areas [13]. Facial palsy can be unilateral or bilateral and occurs due to direct invasion of the nerve by the spirochete bacterium Borrelia burgdorferi which is transmitted to the patient via a bite from an infected *Ixodes* species tick.

Reactivation of the varicella zoster virus leads to Ramsay-Hunt syndrome. Also known as zoster oticus, it is characterized by facial palsy, painful eruption of vesicles in the external auditory canal, hearing loss and vestibular dysfunction [14]. Incidence in children under 10 years of age is reported to be 2.7 per 100,000.

Other infections that can cause facial paralysis include human immunodeficiency virus, herpes simplex virus, cytomegalovirus, Epstein-Barr virus, adenovirus, rubella virus, mumps, influenza, echovirus, and coxsackievirus.

Facial paralysis can also result from trauma to the face or temporal bone. Incidence has been shown to have a bimodal distribution with a peak at 2.4 and 8.6 years [11]. Iatrogenic facial nerve paralysis can result from oromaxillofacial, parotid, and otologic surgery. In otologic surgery, unexpected facial paralysis is less common than in other surgeries due to the predictable course of the facial nerve in the temporal bone. However, studies of iatrogenic facial nerve injury in the pediatric population are limited. In a case series of 35 cases of pediatric facial nerve paralysis, only 3 cases were found to be iatrogenic and all fully recovered [11]. A review of 102 adult cases of iatrogenic facial nerve injury reported 17 cases of facial nerve injury due to otologic surgery [15]. In the adult literature, risk of facial nerve injury during primary otologic surgery is cited to be between 0.6 and 3.7% and the risk increases to 4–10% in revision surgery [16].

Neoplasms that cause facial paralysis in children can be benign (cholesteatoma being the most common) or malignant. Other benign neoplasms include facial schwannomas, vestibular schwannomas, and meningiomas. One study of idiopathic pediatric facial nerve paralysis reported 12% of patients had a malignancy, such as leukemia, rhabdomyosarcoma and histiocytosis [17].

A more rare cause of facial nerve paralysis in children is hypertension. In one study, 8% of children with facial palsy was due to hypertension [11]. In addition, other rare systemic causes of facial nerve paralysis include granulomatosis polyangiitis (GPA), sarcoidosis, and systemic lupus erythematosus. These are the least common causes of facial paralysis as data in children is limited to only case reports [18–20]. However, in adults, otologic symptoms can occur in 19–45% of patients with GPA [21]. Yoshida et al. reported that facial palsy was present in 25% of 123 cases of otitis media in antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis [22]. There are also reports of bilateral facial paralysis in patients with vasculitis [23,24].

Congenital facial nerve paralysis

Congenital facial paralysis includes both developmental defects and birth trauma, with birth trauma being more common. Reported incidence ranges from 0.08 to 0.2% of live births [25]. Risk factors for perinatal traumatic facial nerve paralysis include maternal primiparity, birth weight greater than 3500g, prolonged labor, use of forceps, and prematurity [26].

Facial paralysis with multiple cranial nerve abnormalities or other anomalies is more likely due to developmental defects. Moebius syndrome (with an incidence of 1 per 50,000 births) [27] is a rare congenital disorder characterized by bilateral or unilateral 6th and 7th cranial nerve palsies. It is believed to be due to hypoplasia of motor nuclei from hypoxemic-ischemic encephalopathy [28].

Melkersson-Rosenthal syndrome is associated with intermittent

facial paralysis, facial swelling, and a fissured tongue. About 30–50% of these patients will develop permanent facial paralysis [29].

Goldenhar syndrome is characterized by hemifacial microsomia, malformations of structures derived from the first and second branchial arches, and facial paralysis [30]. The incidence is estimated at 1 in 5600 live births with facial weakness present in about 41% of patients with Goldenhar syndrome [31].

Syringobulbia can cause facial paralysis, along with dysphagia and speech difficulties. Arnold Chiari syndrome will have facial paralysis and other cranial nerve palsies due to malformation of the posterior fossa [32].

Genetic disorders, such as myotonic dystrophy and myasthenia can also cause facial nerve paralysis. The genetic loci for isolated hereditary facial paralysis have been identified (chromosome 3q21-22 and 1q21.3–22.1) [33].

Another cause for facial asymmetry that can mimic facial nerve paralysis, is congenital unilateral lower lip palsy, also known as asymmetric crying facies. It has an incidence of 1 per 160 births [34]. This is a result of congenital absence or weakness of the depressor anguli oris muscle, which leads to failure of the lower lip to depress, most notable during crying. However, other branches of the facial nerve are fully functional. This can be associated with congenital heart disease or renal abnormalities [35].

Clinical presentation

Acute peripheral facial nerve paralysis will present with decreased forehead movement, incomplete eye closure, loss of the nasolabial fold, and asymmetric smile. Hyperacusis, decreased tearing, and change in taste can occur as well depending on the location of the facial nerve injury. Sparing of the forehead muscles is indicative of a central lesion rather than a peripheral palsy.

Assessment of facial nerve function includes instructing the child to close their eyes, elevate their brows, frown, smile, and pucker their lips. In patients too young to follow commands, observation of facial movements while crying is used to determine extent of facial paralysis. The House-Brackmann (HB) facial nerve grading scale is used to characterize severity of facial nerve paralysis (Table 1) [36].

Facial nerve paralysis due to otitis media typically will present with middle ear effusions, tympanic membrane erythema, purulent otorrhea and/or mastoid tenderness. These clinical signs typically present 5–8 days prior to onset of facial paralysis [2]. Ramsay-Hunt syndrome is often associated with a vesicular rash (Fig. 2) and vestibulocochlear dysfunction, however 50% of cases in children present with facial paralysis first and delayed-onset of vesicles [37]. Association with other cranial neuropathies or developmental abnormalities would point to a congenital etiology.

Most facial nerve weaknesses have an acute onset. However, recurrent facial nerve weakness would indicate suspicion for malignancy or systemic disease. If no definitive cause of an acute unilateral facial paralysis (with onset < 72 hours) is identified, the diagnosis is then classified as Bell's palsy.

Table 1
House-Brackmann grading scale for facial paralysis.

Grade	Description
I	Normal
II	Mild dysfunction (slight weakness, normal symmetry at rest)
III	Moderate dysfunction (obvious but not disfiguring, synkinesis, symmetry at rest, complete eye closure with maximal effort, good forehead movement)
IV	Moderately severe dysfunction (obvious and disfiguring asymmetry, significant synkinesis), incomplete eye closure, moderate forehead movement
V	Severe dysfunction (almost no motion)
VI	Total paralysis (no movement)

*reprinted from cited reference[36].

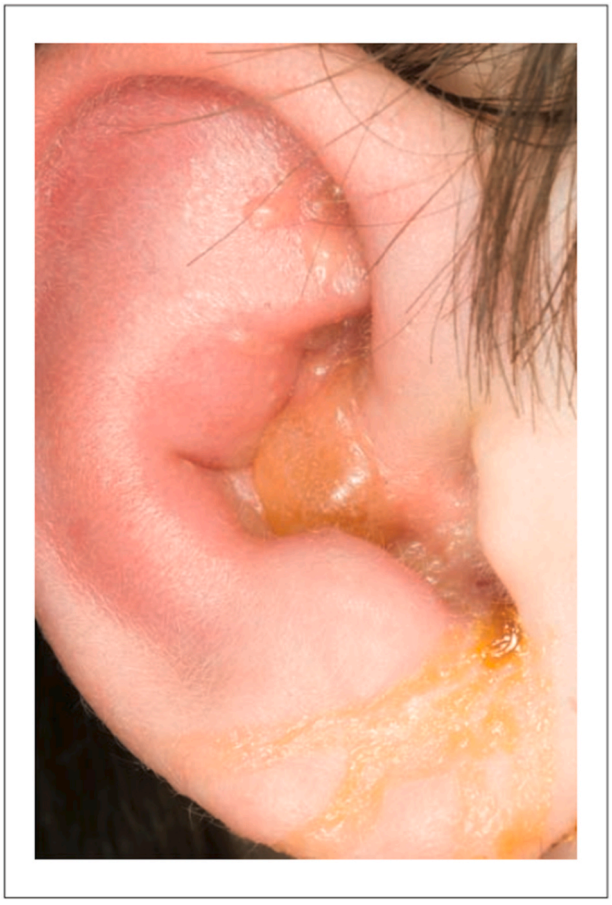

Fig. 2. Typical vesicular rash along the external ear seen in Ramsay Hunt Syndrome. Reprinted from cited reference [2].

Diagnosis and testing

Workup for a child who presents with facial nerve paralysis starts with a thorough history and physical to address time of onset, speed of progression, and associated symptoms (hyperacusis, change in taste). A detailed past medical history should be taken to ask about diabetes or otitis media and possible exposures to tick bites or other infectious causes. On physical exam, special attention should be paid to examining the external auditory canal, auricle and mastoid as well as to accurately characterizing the severity of facial weakness, using the HB grading scale. An audiogram and tympanogram should be conducted in all children with acute facial nerve paralysis.

Serologic testing (immunoglobulin G and M) for Lyme is recommended in patients who present with acute facial paralysis in Lyme-endemic territories during the spring and autumn months. After the first 4–6 weeks of infection, serologies are highly sensitive and specific. If other inflammatory etiologies are being considered, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR) and anti-neutrophil cytoplasmic antibodies (ANCA) studies should be ordered.

Imaging such as computed tomography (CT) or magnetic resonance imaging (MRI) is recommended to evaluate for suspected chronic otitis media, acute mastoiditis, trauma, or neoplasm. Repeat imaging is often suggested if facial nerve paralysis continues to worsen or has not improved after 6 months.

Electroneuronography (ENoG) can be performed after 3–14 days after onset of complete paralysis in patients. Patients with >90% degeneration on ENoG and no motor unit potentials on electromyography are recommended to undergo surgical decompression of the facial nerve [38].

Lumbar puncture is performed when there is concern for Lyme meningitis (symptoms would include nuchal rigidity, fever, headache, and papilledema). Cerebrospinal fluid studies would yield an increased white blood cell count and increased protein in central nervous system infections. Per the recommendation of the Infectious Diseases Society of America and the American Academy of Neurology, it is of limited use in peripheral nervous system involvement and should only be performed if there is concern for central nervous system infection [39,40].

Treatment

The underlying cause of the facial nerve paralysis will dictate the type of treatment. As with adults, eye care is very important to prevent corneal abrasions, exposure keratitis, and vision loss. If there is incomplete eye closure, it is important to use saline eye drops during the day and ophthalmic ointment while sleeping.

Infectious causes should be treated accordingly. Empiric treatment of Lyme disease is recommended in patients with recent exposure to endemic areas during peak season. The recommended treatment for neurologic Lyme disease is 14–21 days of oral doxycycline or intravenous (IV) ceftriaxone, cefotaxime, or penicillin G [41]. The benefits of concurrent treatment with steroids still remains unclear and requires further study [42,43].

Patients with facial palsy due to acute otitis media should be started on broad spectrum IV antibiotics with a second or third generation cephalosporin. If CT imaging does not show an abscess or coalescent mastoiditis, a myringotomy with or without tube placement and subsequent application of topical ciprofloxacin and dexamethasone drops is recommended. Coalescent mastoiditis is characterized by opacification of mastoid air cells along with erosion of bony septae of the sigmoid plate (Fig. 3). If CT imaging demonstrates these findings, a mastoidectomy and tympanostomy tube placement should be performed.

Children with Bell's palsy can be treated with steroids and acyclovir, which have been recommended for treatment in patients 16 years and older [44–46]. Pediatric studies have been inconclusive. However, starting steroids and antivirals within 3 days of onset is recommended. Ramsay Hunt syndrome should be treated with prednisone and acyclovir within 3 days of symptom onset as well.

Prognosis

Children are known to recover well from facial paralysis. In children ≤14 years with Bell's palsy, 90% achieved full recovery of facial function, compared to about one-third of patients >60 years old [47]. In infectious cases, recovery takes 1 month or less [11]. While functional recovery is common, Biebl et al. demonstrated in 175 children with facial palsy that while most functionally fully recovered, up to 50% continued to have a residual facial asymmetry [48].

In general, the prognosis of nerve injury is correlated to the severity of paralysis and younger age at presentation [48]. Congenital facial palsy does not often recover as the etiology is abnormal development of the nerve. However, perinatal traumatic congenital facial palsy will have 100% of patients show some improvement in function within 2–4 months [26]. The median time to recovery for Lyme disease is 26 days [49]. Facial nerve recovery after Ramsay Hunt syndrome is typically poor and patients are often left with permanent sequelae [47]. Also with Ramsay Hunt syndrome, there is a higher incidence of associated symptoms such as hearing loss (73%) and balance disturbances (64%) [47].

Case series

We present 6 cases of facial paralysis in children from our own institution. The first 3 cases are examples of facial palsy secondary to infection and otitis media, all of which resulted in relatively rapid, full recovery of nerve function. The last 3 cases are examples of facial nerve injury secondary to autoimmune or malignancy-related etiologies, all of whom experienced prolonged facial nerve weakness or incomplete recovery for nerve function. Case demographics, etiologies, facial paralysis severity and outcomes are summarized in Table 2.

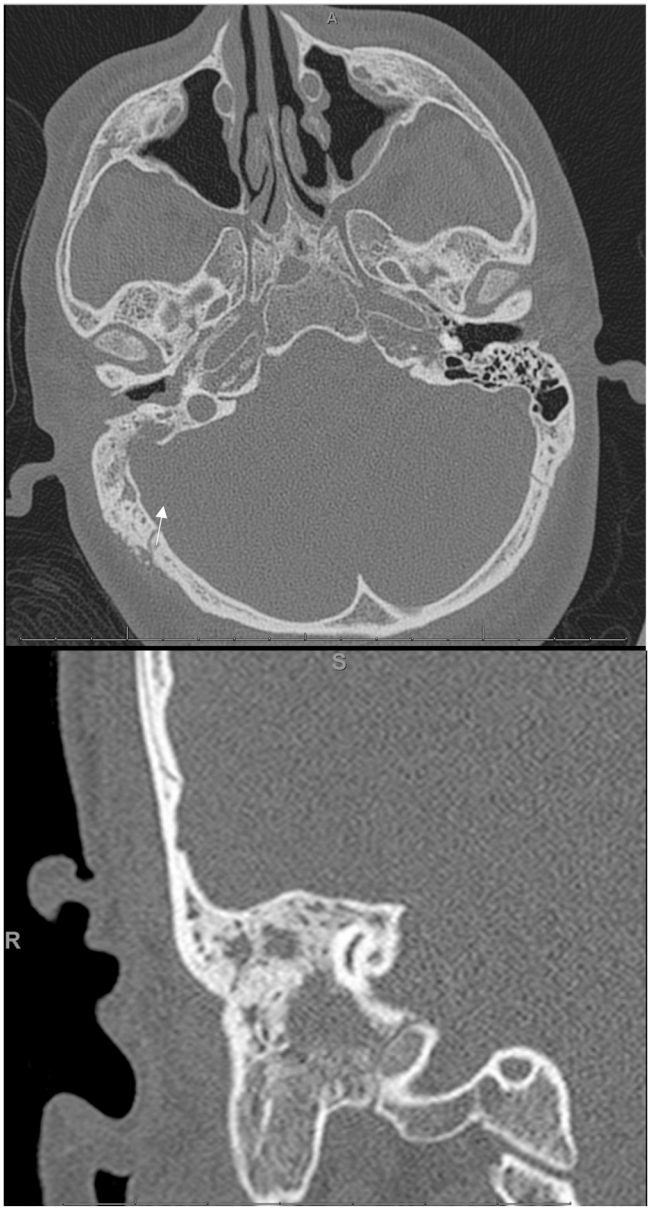

Fig. 3. Axial (a) and coronal (b) computed tomography of the temporal bone showing right coalescent mastoiditis. There is loss of bony septations and erosion of the sigmoid plate (white arrow).

Case 1

A 13-year-old female with no significant past medical history who presented with 10 days of left ear pain. She was started on amoxicillin but then presented to the Emergency Department (ED) 3 days later with worsening left ear pain and otorrhea. Her antibiotics were changed to Cefdinir and she was started on ciprofloxacin/dexamethasone otic drops. However, 5 days later she developed left facial weakness with subjective decreased hearing in her left ear. She was afebrile and on exam, her left ear canal was filled with white debris, she had mild tenderness on palpation over the left mastoid and complete paralysis of her left facial nerve (HB VI). A CT head showed left non-coalescent

Table 2
Facial nerve paralysis outcomes in 6 pediatric cases.

Case	Age (years)	Gender	Etiology	Initial HB grade	Outcome HB grade	Duration
1	13	Female	Infectious	VI	I	3 months
2	16	Male	Infectious	V	I	3 weeks
3	2	Female	Infectious	III	I	2 days
4	2	Male	Neoplasm	NA	Incomplete recovery	3 years
5	16	Male	Autoimmune	VI (bilateral)	III	4 months
6	16	Male	Autoimmune	VI	III	10 months

HB: House-Brackmann.

mastoiditis (Fig. 4). She was taken to the operating room the next day for a left tympanostomy tube placement. She completed a total of 14 days of antibiotics (3 days of IV followed by 11 days of oral (PO) amoxicillin/clavulanic acid and clindamycin) as well as 14 days of Ciprofloxacin/dexamethasone drops and 7 days of prednisone with a 7 day taper. She was noted to have improvement in facial weakness postoperative day (POD) 1, with complete recovery to HB I at her 3-month follow up appointment.

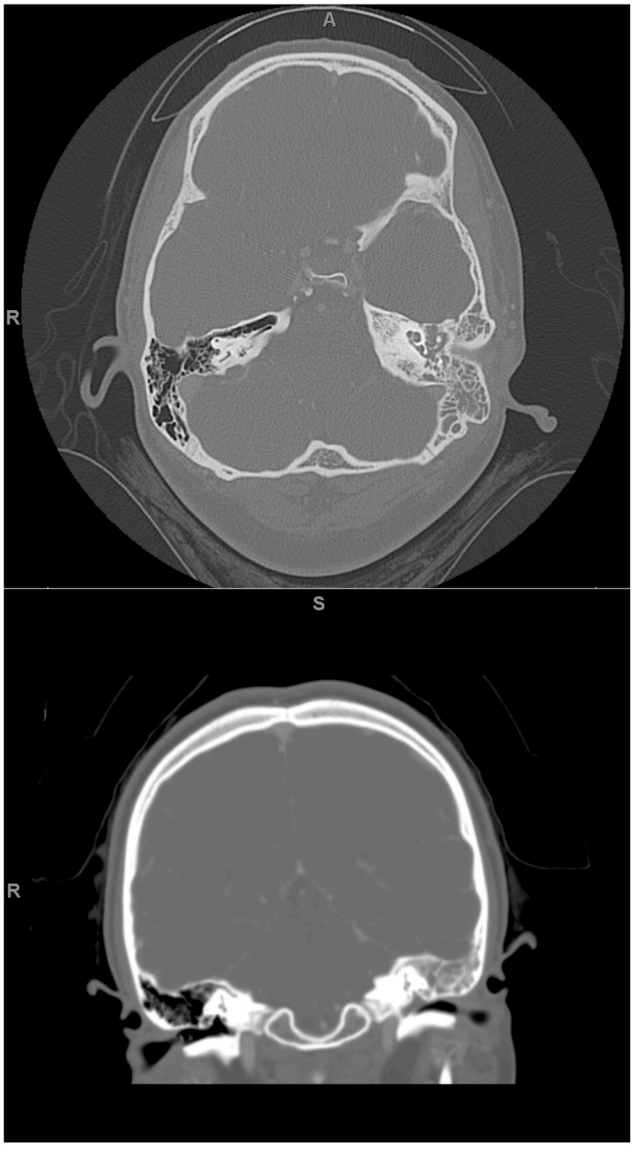

Fig. 4. Axial (a) and coronal (b) cuts of computed tomography showing left non-coalescent mastoiditis in a 13-year-old female with complete facial paralysis secondary to acute otitis media.

Case 2

A 16-year-old male with a history of chronic serous otitis media and multiple tympanostomy tubes as well as bilateral cochlear implants 8 years prior presented with 2 days of pain over the left implant site and 1 day of left facial weakness. On exam, he was noted to have a bulging left tympanic membrane with purulent otorrhea and left facial nerve weakness (HB V). He was admitted for IV clindamycin and steroids. CT scan showed bony erosive changes and concern for left internal jugular vein thrombosis. He was taken to the OR for a left cortical mastoidectomy, left cochlear implant explantation and was noted to have a left anterior inferior tympanic membrane perforation.

His facial nerve paralysis improved POD1 to a HB III and POD2 to a HB II. He was discharged home with 10 days of PO clindamycin and 10 days of ciprofloxacin/dexamethasone drops. His facial nerve paralysis was completely resolved on follow up exam 3 weeks later.

Case 3

A 2 year-old female with severe bilateral sensorineural hearing loss and a history of bilateral cochlear implants and tympanostomy tubes presented with pain and drainage from her implant site 6 months later. She was started on PO clindamycin, but then developed left facial weakness. On exam she had left facial paresis (HB III). Her implant site was erythematous and tender to touch. She had an elevated white blood cell (WBC) of 19.5×10^9/L (normal range: 5.0–14.5×10^9/L). CT scan showed otomastoiditis with near complete opacification of mastoid cavity and middle ear. She was taken to the OR the next day for a left CI explantation. She was treated with ciprofloxacin/dexamethasone drops and 5 doses of IV dexamethasone. She was discharged with 10 days of clindamycin PO and 14 days of ciprofloxacin/dexamethasone drops. Her facial nerve weakness resolved completely by POD 1.

Case 4

A 2 year old male with no past medical history presented with 1 day of left facial weakness, fever and an elevated WBC of 333×10^9/L (normal range: 3.4–10.4×10^9/L). On exam he was noted to have abdominal distension, bruising, and left facial paralysis with incomplete eye closure. Work up and bone marrow biopsy was consistent with T-cell acute lymphoblastic leukemia (ALL). He completed cranial radiation and chemotherapy, with slow improvement of his facial paralysis. His facial nerve function was noted to be almost completely recovered 3 years after symptom onset. HB facial nerve grading was not recorded in his visits.

Case 5

A 16 year old male with Ehler's Danlos syndrome presented with bilateral facial nerve paralysis (HB VI). He reported one month of nasal congestion, sinus pressure, and muffled hearing. Tympanostomy tubes were placed at an outside facility, after which he experienced acute onset right facial nerve weakness. He then underwent a right mastoidectomy for presumed acute mastoiditis. Three weeks later, he presented to our institution for 2 days of left facial weakness and left mastoid pain.

His labs were significant for a WBC of 12.8 × 10^9/L (normal range: 3.4–10.4 × 10^9/L), elevated ESR 95 (normal range: 0–15 mm/hr) and CRP 13.7 (normal range: 0.0–1.0 mg/dL). A CT scan showed chronic left otomastoiditis and acute otomastoiditis on the right with a large abscess. Magnetic resonance imaging (MRI) showed enhancement of the right facial nerve in the internal auditory canal (Fig. 5). He was taken to the OR for bilateral PE tubes and bilateral middle ear biopsies. Intraoperative findings included bilateral serous effusions with granulation tissue in the right middle ear. He was started on a prednisone taper. Further work up revealed that he was positive for proteinase 3 (PR3)-ANCA, suggestive of ANCA-associated vasculitis. The middle ear biopsies were inconclusive. The patient was started on Rituxan and methotrexate. At his 4 month follow up, this patient continued to have facial nerve deficits, with HB III bilaterally.

Case 6

A 16 year-old male with no past medical history who presented with 2.5 weeks of bilateral otalgia, 6 days of bilateral otorrhea, and 2 days of complete left facial paralysis (HB VI). He had no history of fever and no leukocytosis. Lab work was notable for elevated ESR ad CRP, but ANCA was negative. On exam he was noted to have a right middle ear effusion and an erythematous and polypoid left tympanic membrane. CT scan was concerning for mastoiditis. He was taken to the OR for bilateral tympanostomy tube placement with intraoperative findings of bilateral purulent middle ear effusions. Two weeks later, he developed decreased hearing in bilateral ears and continued complete facial nerve paralysis. A CT scan showed bilateral mastoid opacification and an MRI demonstrated enhancement of the geniculate, tympanic, and vertical segments of the left facial nerve. He was given 3 doses of dexamethasone and was taken to the operating room for a mastoidectomy given his non-resolving palsy. He was initially lost to close follow up, but then presented after 2 months with no improvement in his facial function. At this point, an EMG was performed, which demonstrated 92% degeneration on the left. Also at this time, he had episodes of recurrent epistaxis and was admitted for new onset anemia (Hgb 7.1). Further work up revealed bilateral lung nodules. A repeat MRI showed persistent bilateral mastoid opacification with left labyrinthitis and neuritis. After a nondiagnostic CT-guided needle biopsy, a video assisted thoracoscopy and lung wedge biopsy revealed lymphomatoid granulomatosis, consistent with ANCA-negative granulomatosis polyangiitis. 10 months after his initial presentation, he was noted to have some improvement of his facial nerve palsy to a HB III with treatment of his underlying disease.

Conclusion

Pediatric facial nerve palsy will most likely be idiopathic, similar to in the adult population. However, if a cause can be identified, the differential diagnosis is different in children when compared to adults. If facial nerve function does not quickly recover, uncommon etiologies must be considered.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethical statement

This study is submitted with IRB exempt status with our institution.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

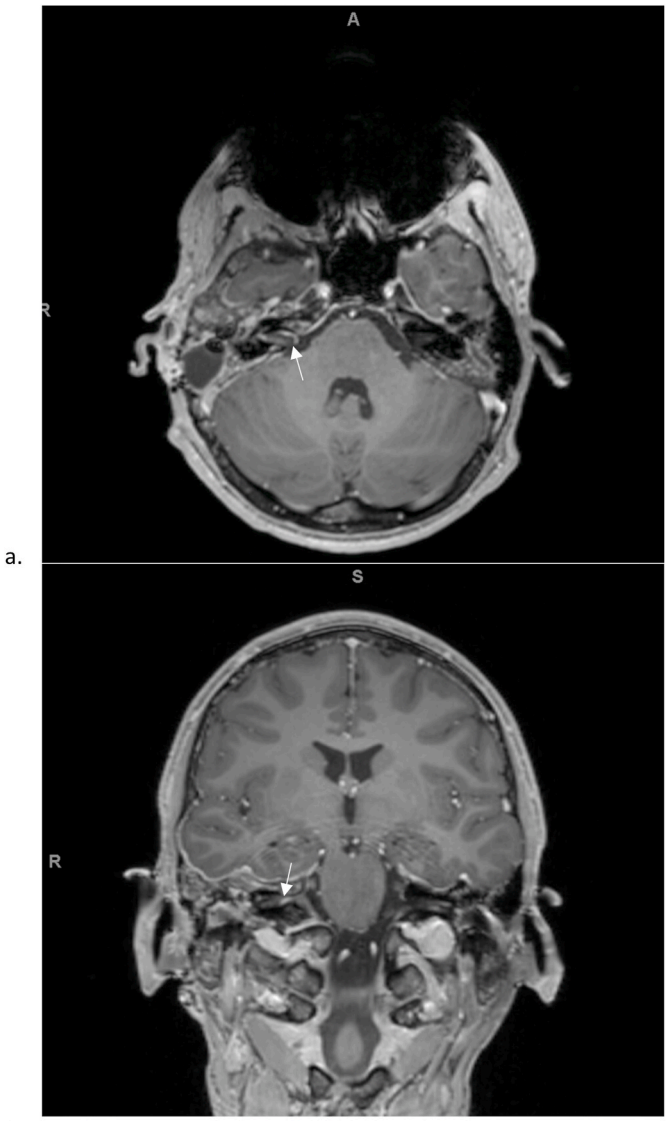

Fig. 5. Axial (a) and coronal (b) cuts of a T1-weighted post contrast brain magnetic resonance imaging showing right facial nerve enhancement in the internal auditory canal (white arrow).

Kenneth Lee MD, PhD reports a relationship with Med El Corporation that includes: board membership and travel reimbursement. Kenneth Lee MD, PhD reports a relationship with Advanced Bionics that includes: consulting or advisory and travel reimbursement. Kenneth Lee MD, PhD reports a relationship with Oticon Medical that includes: consulting or advisory, speaking and lecture fees, and travel reimbursement. Kenneth Lee MD, PhD reports a relationship with Qualia Oto that includes: equity or stocks.

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