REVIEW ARTICLE

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Management of Facial Palsy in Children: A Narrative Review

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Keywords

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Abstract

Background & Objective

Facial nerve palsy in children arises from diverse aetiologies, including Bell's palsy as well as infectious, neoplastic, and neurologic conditions. Differentiating peripheral from central causes is critical, as each entails distinct management and prognostic implications. This narrative review summarizes the diagnostic criteria and management strategies of paediatric facial palsy by comparing protocols from major reference centres and reviewing the recent literature. We present four clinical cases that underscore the importance of a multidisciplinary approach, provide practical clues for timely diagnosis, and offer key messages for the management of facial palsy. Finally, we propose a management algorithm derived from these findings.

Methods

We reviewed literature (2010–2025), reference centre protocols, and four Hôpital Universitaire de Bruxelles paediatric cases to summarize diagnosis, management, and outcomes of paediatric facial palsy, adapting adult guidelines where paediatric data were lacking.

Key Content and Findings

Paediatric facial palsy requires a systematic diagnostic and multidisciplinary approach, as both aetiology and management vary. This review covers clinical diagnosis, indications for imaging, multidisciplinary follow-up, treatment options and prognosis. Severe cases benefit from corticosteroids; antivirals are reserved for Ramsay Hunt syndrome. Neuroborreliosis should be considered in endemic areas, even with initially negative serological tests. Early ophthalmologic care and multidisciplinary follow-up are essential to prevent complications.

Conclusion

Early, thorough evaluation and a multidisciplinary approach are essential to identify underlying causes of paediatric facial palsy. Corticosteroid use remains debated and varies across centres; based on our review, we propose an algorithm to guide management.

Introduction

Facial nerve palsy is a frequent presentation in the paediatric emergency ward, with an incidence that varies from 5 to 21 children per 100,000 children per year under the age of 18 due to variation in settings from different studies (1-3). Facial nerve palsy can be divided into congenital or acquired facial palsy, peripheral or central facial palsy. We excluded congenital facial palsy in this review. Peripheral facial palsy (PFP) and central facial palsy (CFP) differ both clinically and etiologically. To understand the difference between CFP and PFP, a quick anatomical review is needed. The facial nerve is the 7th cranial nerve, composed of fibres from the corticobulbar tract joining in multiple brainstem nuclei. The forehead muscles are innervated by fibres from both

cerebral hemispheres, while the inferior part of the face is innervated only by fibres from the opposite cerebral hemisphere as a result of the decussation of the fibres. Thus, a CFP resulting from a supranuclear lesion will lead to a palsy concerning the contralateral inferior part of the face, leaving the forehead muscles innervated by the fibres coming from the ipsilateral cerebral hemisphere. A PFP, resulting from a lesion under the nuclei, will lead to a palsy concerning both inferior and superior part of the ipsilateral face. Only a clinical examination will allow differentiation of both types (4). After its intracranial path, the facial nerve then enters the internal auditory canal into the petrous portion of the temporal bone. It exits the skull through the stylomastoid foramen and goes through the parotid gland where it divides into 5 branches which innervate the face muscles. Early differentiation between PFP and CFP is essential, as management pathways diverge significantly and delayed recognition of atypical or central causes remains a major source of morbidity in children (1,5). Most cases of PFP are Bell's palsy, but it is an exclusion diagnosis. Therefore, many aetiologies must be ruled out through further testing. According to several studies, the main causes are infectious diseases (1). In a non-Lyme endemic region, acute otitis media (AOM) is the most common, followed by herpes simplex virus (HSV) reactivation, varicella zoster virus (VZV) reactivation with or without Ramsay Hunt syndrome. Other viruses can be involved such as Epstein-Barr Virus (EBV), cytomegalovirus (CMV), coxsackie, human immunodeficiency virus (HIV), adenovirus (1,6). In an endemic region like central or eastern Europe, Lyme disease (Borrelia burgdorferi) has become one of the main causes (1,2). In this case, bilateral PFP is more frequent (4). Other causes of PFP are Guillain-Barré syndrome, trauma such as basal skull fracture or middle ear surgery, as well as malignant tumours of the temporal bone or parotid gland (1,6). CFP results from a central lesion, including vascular involvement, multiple sclerosis and less frequently, sarcoidosis. Neoplasms such as leukaemia or posterior fossa tumours, which account for 50% to 70% of all paediatric brain tumours, are also associated with cranial nerve impairment, most notably affecting the facial nerve, when located up the VII nerve nucleus (1,7). Posterior fossa tumours can also lead to PFP if affecting the VII nerve nucleus itself or beyond the nucleus. (7). The aim of this narrative review is to discuss the causes of facial palsy (FP) in children, the diagnostic criteria, and management strategies by comparing protocols from various reference centres and reviewing the literature published on the subject. Several clinical cases are presented to highlight the causes of FP that should not be overlooked. This study also proposes a management algorithm.

Methodology

We chose a narrative review because it allows flexible integration of heterogeneous evidence and clinical experience in an area with limited paediatric data, combining research studies, clinical guidelines, institutional protocols, and case reports to emphasize the importance of early diagnosis, multidisciplinary management, and recognition of less frequent aetiologies. We conducted a thorough search across electronic databases including PubMed, UpToDate, and the Cochrane Library using various terms related to "Facial palsy," such as "7th cranial nerve palsy" and "Facial paralysis," alongside phrases like "Management of Facial Palsy," "Treatment of facial palsy," and "Neuroborreliosis in children." We specifically targeted articles published after 2020 and written in English. We utilized the Scopus application to calculate CiteScore metrics, measuring the citation impact of the articles we included. To further enrich our review, we reached out to reputable children's hospitals to obtain their internal protocols for managing facial palsy. Protocols were selected based on their online availability and accessibility, and on inclusion of the authors' institution, the other major paediatric centre in Brussels, and a Flemish reference centre. Additionally, we meticulously reviewed the bibliographies of identified studies, review articles, and guidelines. From this comprehensive search, we identified 27 relevant articles and four guidelines that collectively offer a comprehensive overview of the management of FP in children. The four clinical cases presented are children who have been treated in "Hôpital Universitaire de Bruxelles" including the "Hôpital des enfants Reine Fabiola" and the "Hôpital Erasme", for whose consent has been obtained for the use of their data for this publication. We reviewed the English writing with ChatGPT and https://beta.springernature.com/pre-submission/writing-quality. The keywords were searched with MeSH on https://meshb.nlm.nih.gov/MeSHonDemand.

Diagnosis

A thorough medical history and clinical examination must be performed. The practitioner must review how the facial palsy (FP) started, its onset, progression, and associated symptoms such as fever, headache, ear pain, facial pain, hearing disorder or behavioural disorders. The child's medical history should be thoroughly reviewed, with particular attention to any previous episodes of similar symptoms, which should alert the practitioner. The clinical examination must include an ear, nose and throat (ENT) examination, an evaluation of periauricular vesicles or erythema migrans lesions and a full neurological examination. The facial nerve function is evaluated by assessing the forehead movements, elevating the brow, frowning, the ability to close the eye, open the mouth and puckering the lips (1,8). The FP is graded through the House-Brackmann (HB) scale from grade I characterized by a normal eye closure, normal mobility and without facial asymmetry to grade VI characterized by an incomplete eye closure, 0% of mobility and a facial asymmetry at rest. The HB grade should therefore be clearly recorded in the patient's medical file at each evaluation. We evaluated and compared the recommendations for complementary examinations from various university hospital centres across Europe (Belgium and France), Australia and America (9-13). Recommendations found in the literature led to the conclusion that it is necessary to conduct a lab testing for all children presenting with FP, including a full blood count, C-reactive protein (CRP), glycaemia, serological testing for mycoplasma, HSV, VZV, CMV, EBV and B. burgdorferi in regions endemic to Lyme disease (1). Additionally, a lumbar puncture with analysis of cytology, blood cultures, oligoclonal banding and B. burgdorferi serology should be performed if the clinician suspects meningitis or another central nervous system disease, or if the FP is bilateral, if another cranial nerve is affected or if an infection by B. burgdorferi is suspected (9-13, Table 1). Emergency imaging should not be performed as a routine evaluation, but should be guided by specific clinical indications or (Table 1 and 2):

- For children under 3 years of age without AOM.
- If there is a suspicion of a neurological cause, if the child presents with CFP, bilateral facial palsy, additional abnormal neurological findings such as concomitant paralysis of another cranial nerve, recurrent FP.
- A CT scan with contrast should be considered if the child presents with chronic otitis media, a cervical mass, a suspected mastoiditis, or if there is a history of temporal bone trauma or surgery (1,9-13).

Follow-up

Various specialists should be involved in the diagnosis and follow-up of children presenting with PFP. An ENT evaluation should be conducted promptly for every child presenting with PFP, within

TABLE 1: Diagnostic management of facial palsy recommended by different centres (9-13).

	Hôpital des Enfants Reine Fabiola, Brussels, Belgium	Université Catholique de Louvain, Brussels, Belgium	UZ Gent, Belgium	The royal children's Hospital Melbourne, Australia	Clinical Practice Guideline: Bell's Palsy - Otolaryngology–Head and Neck Surgery
Laboratory Testing	General blood test † + serology Lyme, HSV, HZV, CMV, EBV, Mycoplasma, mumps	General blood test + serology HSV, HZV, EBV, CMV, Mycoplasma, Lyme if evocative context (second test at 1 month if negative ‡)	General blood test + serology HSV, VZV, EBV, CMV, HIV, Syphilis, Lyme. If immunocompromised add adenovirus, parainfluenza, measles, mumps, rubella	If Suspected malignancy or other cause than Bell's Palsy, or commencing steroids: General blood test + serology HSV, HZV if vesicles are present	Routine laboratory testing is not required. Required if there is atypical presentation, suspected neoplasia, suggestive Lyme exposure.
Lumbar punction	If bilateral or balanced FP, other cranial nerve affected, signs of encephalitis/meningitis, suspected Lyme, suspected malignancy	If signs of meningitis/radiculitis, suspected Lyme.			
Imaging	MRI: if CPF, Other neurological abnormality § Cerebral CT scan if cranial bone trauma	CFP, other cranial nerve affected, mastoïditis, chronic otitis, cranial trauma, recurrence, worsening > 3 weeks. MRI or CT scan depending on the situation.	Cerebral CT scan if abnormal otoscopy, suspected cranial bone fracture, parotid or neck tumour. MRI if recurrency of facial palsy, HB grade 4, associated with hearing loss or vestibular syndrome with normal otoscopy, with central neurological symptoms, history of carcinoma, or no improvement after 6weeks/worsening after 2weeks. Ultrasound if suspected parotiditis or parotid tumour.	If other cause than Bell's Palsy is suspected	If cranial trauma, history of tumour, other cranial nerve affected, no sign of recovery after 3 months. Imaging of choice: MRI with and without contrast. If MRI is contraindicated: contrast-enhanced CT can be used.

† Blood test: Blood count, CRP, renal function, liver enzymes, electrolytes. ‡ Lyme serology can be negative if the exposition is less than 6 weeks' time. § Other neurological abnormality: other cranial nerve affected, cerebellar syndrome, meningeal syndrome, bilateral FP, myotatic areflexia.

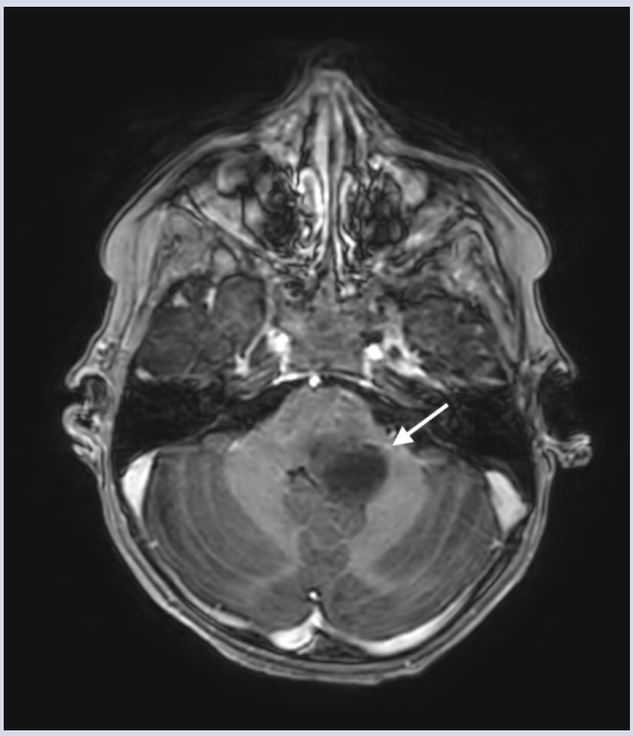
72 hours of onset. Urgent ENT evaluation is needed if the child presents with AOM, signs of mastoiditis, a cervical mass, a vesicular rash in or around the ear, loss of balance, hearing impairment or a history of head trauma. An assessment by a neurologist is urgently necessary for children under 3 years of age, if there is an additional abnormal neurological anomaly, if the child presents with CFP, or if there is a gradual deterioration of the FP. Imaging should not be routinely performed in children with typical peripheral facial palsy who show early and progressive clinical improvement. However, careful clinical follow-up remains essential. In accordance with published recommendations and institutional protocols, neuroimaging should be reconsidered not only in cases of worsening facial weakness or absence of improvement after 4 weeks, but also in cases of delayed or incomplete recovery when the clinical course deviates from the expected pattern (1,4,9–13). Although most children show clear improvement within the first weeks and achieve near-complete recovery within months (1,3), persistent significant weakness beyond 1 month, stagnation of recovery, or residual severe deficit (HB grade IV–VI) without continued improvement should prompt reassessment and discussion of brain MRI, even if the overall evolution appears globally favourable. The decision should be individualized and guided by the clinical trajectory, associated symptoms, and neurological examination findings.

Clinical Cases

Case 1

A 6-year-old male with no significant medical history who presented with a left PFP (HB IV), was started on prednisolone 2 mg/kg/day for a total of 10 days. Subsequently, the child went on a two-month vacation abroad, thus no reassessment was programmed. After two months, he still suffered a left FP and started to have balance disorders, muscular weakness in the left limb and was complaining of diplopia. On examination, he was noted to have an asymmetric smile, a loss of the nasolabial fold, but a complete eye closure and no decreased forehead movements. Thus the FP was assessed as a left CPF. Hemiparesis was noted in the left lower limb, along with a VI cranial nerve palsy. A CT-scan was performed, showing a solid expansive lesion in the brain stem. The MRI showed a 22 mm x 26 mm mass with no sign of hydrocephalus (Figure 1). The diagnosis of a H3 K27M-mutant diffuse midline glioma was established by biopsy. Unfortunately the child died 8 months later. This mutant is a high-grade glioma, grade 4 by the WHO classification, associated with a poor prognosis. The most common symptoms are cranial nerve palsy, with nerves VI and VII being the most commonly affected, along with ataxia (14). This clinical case highlights the importance of a meticulous clinical examination and the necessity of follow-up for

FIGURE 1: T1 weighted post contrast sequence on an axial plane with fat saturation. Left posterolateral portion of the pons. Visualization of a solid tumour in the right posterolateral portion of the pons, at the level of the middle cerebellar peduncles, encompassing the pontine nucleus of the left facial nerve as well as its fascicular segment.



all patients presenting with FP. It also underscores the possibility of a PFP resulting from a central lesion affecting the facial nerve. A neuro imaging must be performed if the PFP worsens over 3 weeks, or with no improvement after 4-weeks of onset, if another cranial nerve is damaged, or if the child presents with CFP (1,9).

Case 2

A 2-year-old toddler with no significant medical history presents at the emergency department for daily pyrexia for two weeks, left otalgia and left temporal headache. The child has recently been treated for AOM with amoxicillin. The child was prostrate, with a stiff neck, without any sign of mastoiditis, the eardrum was slightly erythematous and not bulging. A biology test was performed, which revealed a moderate inflammatory syndrome (CRP: 114 mg/L; white blood cell (WBC) count: 12180/µL) and a lumbar puncture which revealed a slight leukocytosis (WBC count: 21/µL). A broad spectrum antibiotic was started and the child was admitted to the hospital. The following day, the child developed left PFP (HB II). He underwent a brain CT scan (Figure 2) and a brain MRI which identified a collapsed formation at the petrous apex associated with osteitis (Figure 3). The ENT found an AOM that was not obvious in the first otoscopy and a trans tympanic ventilation tube was placed. The child was treated with ceftriaxone and metronidazole for a period of six weeks followed by an oral relay with cefuroxime. During hospitalization, the child developed keratitis of the left eye. The child has recovered all his facial motor skills but still presents a deafness for which the installation of a hearing aid is programmed. The interest of this clinical case is twofold. First, it evokes a rather rare complication of otitis associated with FP: petrous apicitis by extension of otitis. This highlights the importance of imaging and followed by a lumbar puncture if the child has signs of meningeal involvement with otitis. Secondly, it insists on the importance of applying artificial tears very regularly during the day and applying eye ointment and an eye patch during the night until complete recovery, since the consequences on the eye can be disastrous.

Case 3

A 15-year-old child with no significant medical history presented with right otalgia for 4 days, hypoacusis for 2 days, and a progressively worsening right PFP (HB grade III). On exam, he was found to have a right AOM without any associated rash. A lab testing showed no signs of inflammation and serology testing for VZV, HSV, EBV and B. burgdorferi came back negative. The child was discharged with corticosteroid treatment of methylprednisolone at 1 mg/kg/day for a total of 10 days and was scheduled for an appointment with an ENT specialist the following day. Upon examination, a painful vesicular rash on the right external ear was noted. The diagnosis of Ramsay Hunt syndrome was established, and aciclovir at 80 mg/kg/day for a total of 7 days was added to the corticosteroids treatment. Ramsay Hunt syndrome is caused by the reactivation of the VZV. The incidence is reported to be 2.7 per 100.000 children under the age of 10 (1). Herpetic vesicles are present in 80% of cases and usually appear several days after onset of FP, lasting 1-3 days (6). The take home message for this clinical case is the importance of scheduling an ENT evaluation, that eruptions do not appear after the visit to the emergency.

Case 4

An 8-year-old child with no significant history presented with right PFP (HB V) progressing over six days, as well as the emergence of a macular plaque rash on the right cheek progressing for the last 12 hours. Three days prior to presentation, there was paraesthesia noted in the region of the right trapezius muscle. A blood

TABLE 2: Indications for imaging in paediatric facial palsy. Reference: Université libre de Bruxelles (ULB), Hôpital Universitaire de Bruxelles (H.U.B), Hôpital Universitaire des Enfants Reine Fabiola (HUDERF), Service des urgences pédiatriques, Avenue J.J. Crocq 15 1020 Bruxelles – Belgium.

Imaging	When?
Brain + brain stem + temporal bone + parotid MRI T1 Weighted post-contrast sequences with fat saturation	Suspected inflammatory / neoplasia: - Atypical signs: bilateral FP, other cranial nerve affected - Suspected neoplasia; recurrency, CFP - Progression over 3 weeks or no improvement after 1 month of onset
Brain CT Scan with contrast	- Temporal bone trauma or surgery - ENT causes (chronic otitis media, suspected mastoiditis, ENT mass). > to rule out vascular complications such as intracranial venous thrombosis.

FIGURE 2: Non contrast CT scan, bone window. Axial sections. Erosion of the cortex of the left petrous apex (intact appearance on the right).

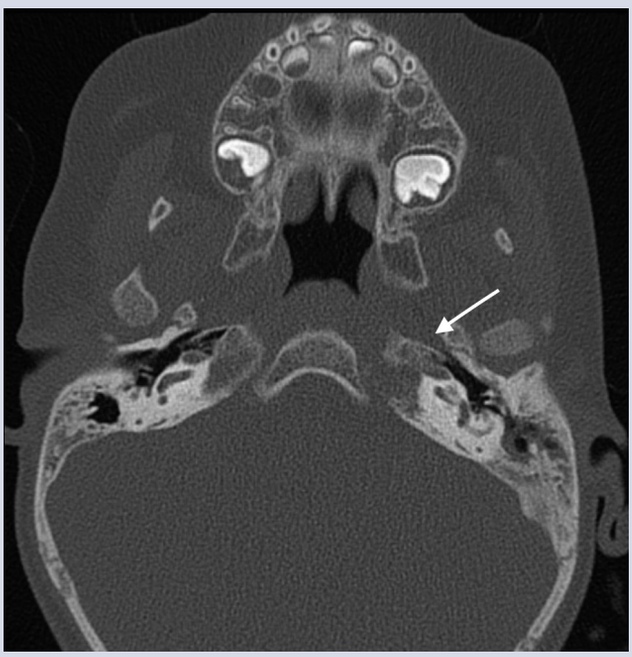
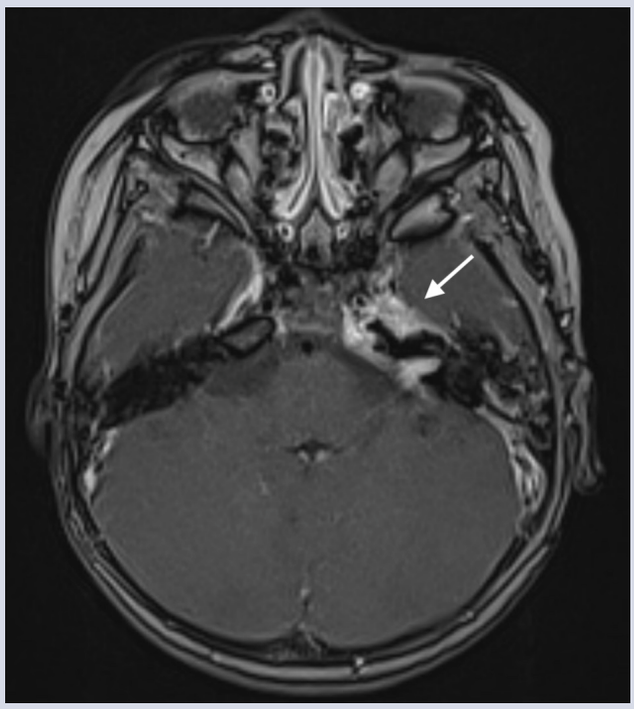


FIGURE 3: Non contrast CT scan, bone window. Axial sections. Erosion of the cortex of the left petrous apex (intact appearance on the right).



Axial T1 DIXON sequence (water-only image). Contrast enhancement of the bone marrow of the left petrous apex and left acousticofacial bundle and Meckel's cavum. Partial pneumatization of the petrous apex. Reactive dural enhancement. Asymmetric calibre of the left cavernous segment of the internal carotid artery, with a permeable appearance (arteritis).

test was performed, showing no signs of inflammation, and the serology testing for VZV, HSV, CMV, EBV and for B. burgdorferi were negative. An audiometry and tympanometry were performed and revealed normal results. The patient was discharged with a diagnosis of Ramsay Hunt syndrome and treated with corticosteroids and acyclovir for a week. Seven days later, the patient started to develop pain in the back and limbs without paraesthesia. No fever was noted. On exam, the patient presents with hyperesthesia in the neck, back and upper limbs. There was no observed weakness, and myotatic reflexes were normal. Right FP showed clear improvement. A brain MRI was performed and came back normal, as well as a second blood test. It was concluded to be neuropathic pain, and an outpatient MRI of the spine was scheduled. The patient was discharged and prescribed gabapentin and tier 1 analgesics. Seven days thereafter, the patient was evaluated by a neurologist, and a left PFP (HBIII) was noted. Neuropathic pain persisted, partially alleviated by gabapentin. The patient was admitted to expedite further evaluation. A series of additional examinations were conducted, including a normal MRI of the spine, a lumbar puncture revealing leukocytosis (111/µL WBC count) with lymphocyte predominance (100%), elevated cerebrospinal fluid (CSF) proteins levels (1.17g/ dL), and the presence of oligoclonal bands, as well as blood tests with serological analysis. Serological testing for both CSF (IgG 168 AU/ml) and blood (IgG 1019 AU/ml and IgM positive) came back positive for B. burgdorferi. The diagnosis of neuroborreliosis with bilateral PFP was established. Joint pain in the back and limbs raised suspicion of Banwarth syndrome. Meningoradiculitis was identified as one the manifestations of neuroborreliosis (17). Treatment with doxycycline for 28 days was initiated, resulting in favourable evolution without any sequelae. The interest of this clinical case is multiple. First, it stresses the importance of not excluding certain diagnoses only from serological testing. CSF serology for B. burgdorferi is sometimes negative at the beginning of the disease. Second, the majority of bilateral PFP has an identifiable cause and the association with Lyme disease is well known and must be always investigated (18,19). However, the history and initial clinical examination for Lyme disease is not specific. PFP may be the first manifestation of the infection, and there is rarely a history of recent tick bites or old migrant erythema (19). It should also be noted that the presence of oligoclonal bands is frequently observed in the CSF of patients with Lyme disease (20).

Treatment

The therapeutic approach to paediatric facial palsy depends on the underlying aetiology and severity, as summarized in Table 3. Management of peripheral facial palsy (PFP) must address two parallel objectives: (1) identification and treatment of the underlying cause and (2) prevention of complications directly related to facial weakness, particularly ocular damage. Due to incomplete eyelid closure, children are at risk of corneal dryness, keratitis and ulceration. Protective measures should therefore be initiated immediately in all patients with impaired eye closure, regardless of aetiology. Artificial tears should be administered regularly during the day, and lubricating ophthalmic ointment combined with eyelid taping or patching should be applied at night (Table 3) (2,9). Ophthalmologic assessment is warranted in cases of HB grade IV–VI or when corneal involvement is suspected. Regarding

TABLE 3: Treatment management of facial palsy recommended by different centres (6-9).

	Hôpital des Enfants Reine Fabiola, Brussels, Belgium	Université Catholique de Louvain, Brussels, Belgium	UZ Gent, Belgium	The royal children's Hospital Melbourne, Australia	Clinical Practice Guideline: Bell's Palsy - Otolaryngology–Head and Neck Surgery
Steroids	Oral prednisolone 1 mg/Kg/day (max 60 mg) for 10 days for HB 5-6 or for children more than 16 years old, within 72h of onset.	Oral prednisolone 2 mg/ Kg/day (max 64 mg) for 5 days, within 72h of onset. Extended to 10 days if painful or Ramsay Hunt Syndrome.	Prednisolone 2 mg/ Kg/day in 3 doses (max 70 mg) for 7 days, within 72h of onset (up to 7 days). Oral steroids if HB1-3. Intravenous steroids if HB ≥ 4 or suspect-ed VZV infection.	No consensus on the use of steroids in children due to lack of evidence.	No strict consensus on the use of oral steroids in children
Antivirals in combination with oral steroids	If Ramsay Hunt Syndrome: Acyclovir 20 mg/Kg/dose 4x/ day max 800 mg/ dose, for 7 days.	If Ramsay Hunt Syndrome: aciclovir 20 mg/Kg/ dose 3x/day (max 800 mg /dose) for 7 days.	If grade ≥ 4 or sus-pected ZVZ infection: Valacyclovir 3 x 1 g/day.	If vesicular rash is present.	If treated within 72 hours of onset of FP, in agreement with the patient as no real benefit has been proven.
Eye Care	For all patients : lubricating drops + ointment + occlusive bandage at night.	If incomplete eye closure: lubricating drops + ointment.	If incomplete eye clo-sure: lubricating drops + ointment + occlusive bandage at night.	If incomplete eye clo-sure: lubricating drops + ointment + occlusive bandage at night.	If incomplete eye closure: Lubricating drops and/or ointment
Surgical Decompression					Not recommended
Physical Therapy			If grade 4.		Not recommended

medications, two compounds are commonly used, although their indications in paediatrics are not clearly established.

1. Corticosteroids:

Corticosteroids have the effect of reducing the oedema of the facial nerve and therefore its compression. Their effectiveness has been repeatedly demonstrated in adults, and the latest Cochrane review mentions that there is no longer a need to prove their effectiveness in adults (21). Regarding pharmacological treatment (Table 3), corticosteroids remain the most debated intervention in children. A systematic review from 2012 identified all studies released between 2000 and 2010. Out of these 2293 studies, the quality and level of evidence, mainly consisting of small retrospective studies, did not provide a definitive answer regarding the efficacy of corticosteroid treatment in children (22). In 2022, an Australian study was published. This multicentre and double-blind study involving 187 randomized patients failed to demonstrate the efficacy of corticosteroids in children. The difference observed was not statistically significant, and in children, recovery is almost always complete (23). Nevertheless, this Australian study suffered from a limited number of participants. A Swedish study is currently underway with plans to include 500 children and adolescents aged 1 to 17. Results are expected at the earliest for 2026 (2). In children, the recovery from PFP is almost always complete, with cases of incomplete recovery being exceptionally rare. Therefore, it is generally unnecessary to overuse corticosteroids. While corticosteroids can be beneficial in certain cases, their effectiveness in children is not clearly demonstrated, and excessive use can lead to undesirable side effects. It is important to weigh the benefits and risks before prescribing them. Based on data from the literature and an intra-hospital consensus (Table 3), we propose treating only patients with a HB grade of IV or higher. We recommend administering corticosteroids (methylprednisolone) at a dose of 1 mg/kg/day (max 64mg) for 10 days if the patient presents within 72 hours of symptoms onset. For children aged 14 and older, we suggest following the adult treatment protocol and using corticosteroids regardless of the HB grade. However, if the child has a relative contraindication for corticosteroids administration (such as diabetes or gastro-intestinal contra-indications), we advise against treatment.

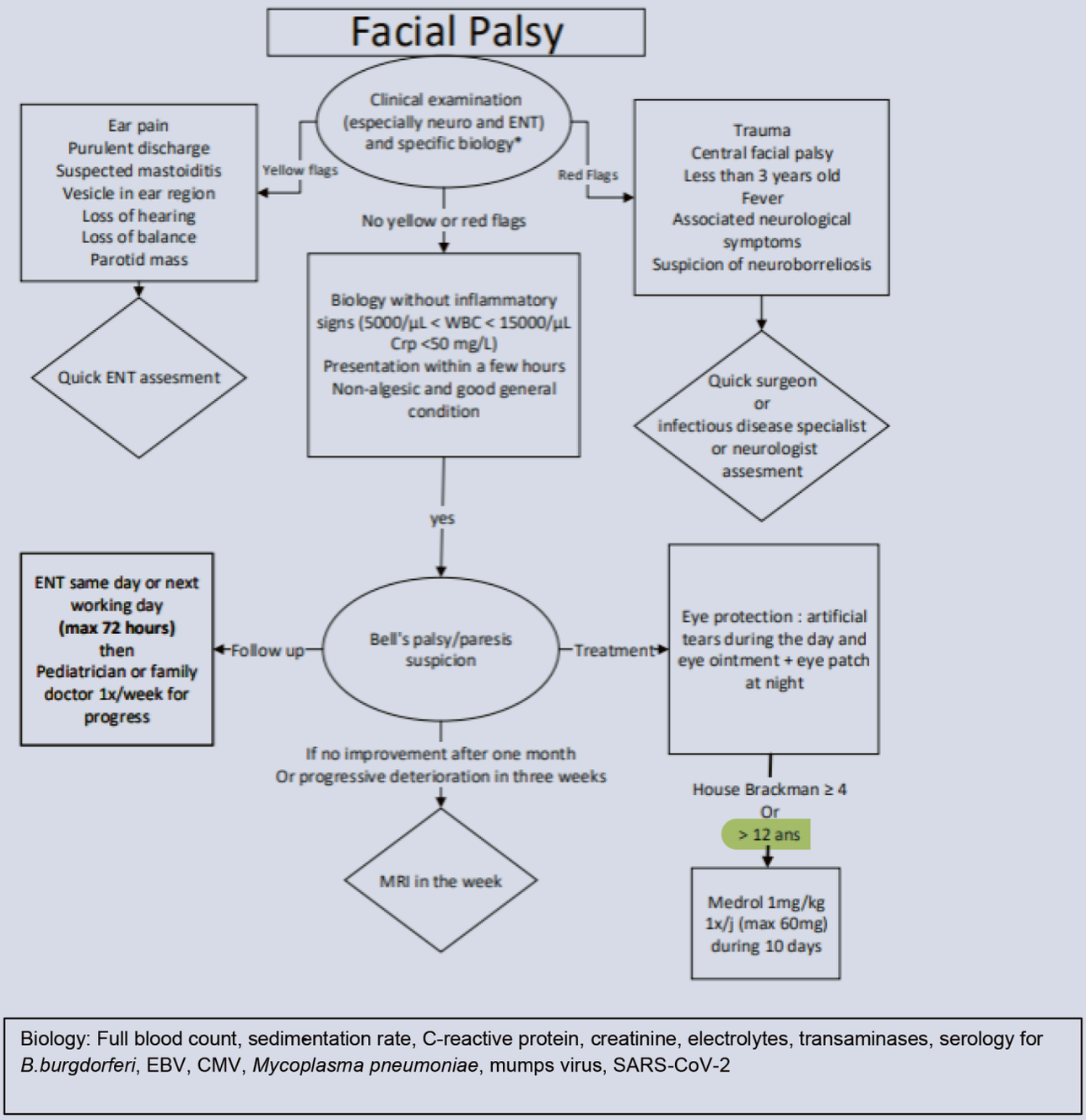
2. Antiviral treatment

We conducted a thorough review of the latest systematic reviews and meta-analysis concerning the use of antivirals (aciclovir, valaciclovir, famciclovir) for treating PF in adults, either as monotherapy or in conjunction with corticosteroids. These antivirals function by impeding HSV infection through the inhibition of DNA polymerase, thus hindering its replication. Upon comparing the recovery of facial function using the HB scale, it was concluded that antivirals alone yielded no benefit compared to placebo, the efficacy of corticosteroids alone is superior over antivirals alone, and there were no significant differences in recovery rates between corticosteroids alone or in combination with antivirals, though a slight benefit cannot be entirely ruled out (9,21-23). No similarly significant studies have been conducted in children. Therefore, and based on the intra-hospital consensus we reviewed (Table 3), we recommend supplementing corticosteroid treatment with an antiviral only in cases suspected to be of viral origin (such as Ramsay Hunt syndrome). Specifically, we recommend acyclovir at a dose of 20 mg/kg/dose 4 times a day with a maximum of 3200 mg/day, administered for a total duration of 7 days (24).

3. Physiotherapy and facial exercise

A 2011 Cochrane review concluded that high-quality evidence supporting physical therapy in Bell's palsy was lacking (25). More recently, an updated systematic review by Khan et al. reported emerging evidence suggesting potential benefits of facial exercise therapy, including when initiated early in recovery (26). However, heterogeneity in study design and methodological limitations prevented firm conclusions. In 2023, Nakano et al.

FIGURE 4: Management algorithm for paediatric facial palsy.



conducted a systematic review and meta-analysis including seven randomized controlled trials in adults and reported that physical therapy may reduce non-recovery rates (RR 0.51, low-certainty evidence) and improve Sunnybrook facial grading scores. Nevertheless, the certainty of evidence was rated as low or very low due to high risk of bias, imprecision, and heterogeneity in timing and therapeutic protocols (27). Importantly, the optimal timing for initiating physiotherapy remains uncertain, even in adults. Included studies-initiated therapy at varying time points, ranging from the acute phase to several months after onset. Concerns have been raised that inappropriate or forceful exercises during the early inflammatory phase may promote maladaptive motor patterns or synkinesis. In children, where spontaneous recovery is generally excellent, evidence remains extremely limited (1,3). Therefore, facial physiotherapy should not be systematically initiated during the acute phase but rather considered on a case-by-case basis, particularly in children with delayed recovery, residual weakness, or emerging synkinesis, and in those able to actively cooperate with therapy. In our institution, physiotherapy is proposed mainly in cases of prolonged or incomplete recovery, with initial sessions focused on education and gentle, supervised exercises to be continued at home (10).

4. Electrostimulation

At present, there is no clinical evidence supporting efficacy (25). Recently, a randomized controlled study was conducted with adults and appears to show some efficacy but their cohort was

small. Additional studies are needed to offer this type of therapy to the patient and even more in children because recovery is almost always complete after one year (28).

Neuroborreliosis

The prevalence of borreliosis varies depending on the country. For instance, countries like Finland or Sweden have a very high prevalence of borreliosis, whereas no cases have been reported in Australia. Consequently, diagnostic procedures differ between countries based on the varying presence of B. burgdorferi (29). In Belgium, 10% of ticks are infected with B. burgdorferi, and there is no apparent increase in incidence (30). In neuroborreliosis, the challenge lies in its diagnosis and treatment. Serological blood and/or CSF tests may yield negative results, which do not rule out early stages of Lyme disease. The antibody response initiates within the first days of infection, often remaining below the laboratory's detection threshold if tested too early. It is recommended to use a combination of ELISA and Western blot techniques for antibodies detection (STTT: standardized 2-tier testing: double testing by ELISA and Western-Blot). The mainstay remains the detection of intrathecal antibodies, although recent studies have shown that the sensitivity ranges between 55 and 80% and is lower in patients with shorter symptom durations. Therefore, it is recommended to repeat serological tests 2-4 weeks later if initial results are negatives but the context is evocatory of Lyme disease. A promising new technique, the C6 ELISA Test, is proposed to replace the STTT and intrathecal antibody detection, though further studies are necessary to validate its efficacy (29). Moreover, most cases of FP related to neuroborreliosis do not present recent tick bites or erythema migrans. A retrospective US study identified certain risk factors neuroborreliosis-related FP, including headache and/or pyrexia, seasonal occurrence (June to October in Belgium), bilateral FP, erythema migrans or recent tick bite (16). For additional testing, blood serology or CSF analysis can be performed. Lumbar puncture may reveal lymphocytic pleocytosis with hyperproteinorrachia. The IgG CSF/IgG index blood may be significantly elevated, and the presence of oligoclonal bands is sometimes observed. However, these results are not always positive, and repeated analysis may be necessary. Regarding treatment, there is ongoing debate regarding the use of intravenous versus oral antibiotic therapy. Clinical trials in Europe suggest that oral doxycycline is comparable to intravenous antibiotics, although it is only used in children over 8 years old, with ceftriaxone being preferred for younger children (29). The efficacy of corticosteroids, however, remains uncertain (16). Thus, we recommend treating patients with suspected neuroborreliosis on a case-by-case basis, consulting with your institution's paediatric infectious disease specialist. A 2008 study shows an excellent recovery rate at 6-month follow-up (31).

Prognosis

The recovery rate in children presenting with PFP is excellent and most children show complete recovery within 6 months, although some studies indicate mild dysfunction at follow-up (2,3,24,30). Possible persisting symptoms include impaired eye closure, affected pronunciation, minor nerve dysfunction, and synkinesis (2,29,31).

Limitations

There are a few limitations to our review. First, we sought expert opinions for subjects still under debate in the literature, such as the utility of corticosteroids in treating FP in children. Second, the proportions of causes differ between regions where Lyme disease is endemic and those where it is not, and the literature does not always account for this variation. Third, the HB scale is not entirely objective, which can affect the consistency in the initiation of treatment.

Conclusion

FP is an uncommon but clinically significant presentation in the paediatric emergency setting, with multiple potential aetiologies that require careful differentiation. Distinguishing central from peripheral facial palsy is essential, as management strategies differ substantially. Bell's palsy remains a diagnosis of exclusion. A structured approach based on thorough medical history, detailed neurological and ENT examination, and systematic grading using the House-Brackmann scale is crucial. Complementary investigations should be guided by clinical findings and risk factors. Neuroimaging and lumbar puncture must be considered in cases of bilateral palsy, central signs, associated cranial nerve involvement, atypical progression, or absence of improvement after 4 weeks. Eye protection measures are mandatory in all patients with incomplete eyelid closure. Regarding pharmacological treatment, we recommend corticosteroids (1 mg/kg/day, max 64 mg for 10 days) in children with severe PFP (HB grade IV–VI) when initiated within 72 hours of symptom onset. For adolescents aged 14 years and older, adult protocols may be followed. Antiviral therapy should be reserved for suspected viral aetiologies such as Ramsay Hunt syndrome. Current evidence does not support the systematic use of physiotherapy or electrostimulation in the acute phase. While emerging adult data suggests a potential benefit of facial exercise therapy, the certainty of evidence remains low and paediatric data are lacking. Physiotherapy may therefore be considered on a case-by-case basis, particularly in children with delayed recovery, residual weakness, or synkinesis. Finally, we propose a practical management algorithm to assist clinicians in the evaluation and treatment of paediatric facial palsy (Figure 4).

Statements

The authors have no conflicts of interest relevant to this article to disclose. AI assistance: ChatGPT (OpenAI) was used for English proofreading.

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