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PAEDIATRIC NEURORADIOLOGY

Magnetic resonance imaging of developmental facial paresis: a spectrum of complex anomalies

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Abstract

Purpose Despite its clinical implications, the MRI features of developmental facial paresis (DFP) were described in a few case reports. This study aims to describe MRI features of DFP in relation to the embryological development with a proposed radiological new grading system.

Methods The clinical records and MRI of the brain and internal auditory canal of 11 children with DFP were retrospectively reviewed. The following sequences were analyzed: axial, oblique sagittal SPACE of the internal auditory canal and brainstem; axial T2, T1WI and coronal T2WI of the brain. The severity of the maldevelopment of the seventh nerve was graded from 0 to 4: 0 = no abnormalities, 1 = unilateral facial nerve hypoplasia, 2 = unilateral facial nerve aplasia, 3 = aplasia or hypoplasia involving facial nerves on both sides, and 4 = facial nerve aplasia or hypoplasia associated with other cranial nerve palsy.

Results Isolated facial nerve palsy was diagnosed in seven patients. It was of grade 1 in five and grade 3 in two. Hypoplasia of the nerve with interrupted course was encountered in two cases. Other associated cranial nerve abnormalities (grade 4) were seen in four patients; two of them were diagnosed previously as Moebius syndrome. In addition to inner ear anomalies, middle and external ear and parotid gland anomalies were described.

Conclusion To our knowledge, this is the largest series of patients with DFP that represents a continuum of isolated and combined malformations. Understanding of embryological basis can give insights into the anomalous development of the facial nerve.

Keywords Congenital facial palsy · Moebius syndrome · Hereditary congenital facial paresis · Abducens nerve · Trigeminal nerve

Abbreviations

DFP Developmental facial palsy
HCFP Hereditary congenital facial palsy

Introduction

Congenital facial paresis is defined as seventh cranial nerve palsy which is present at birth or shortly after. It is relatively rare with estimated incidence 0.8–2.1/1000 live birth. It can result from perinatal insult (most commonly birth trauma) or aberration during fetal development (developmental facial paresis; DFP) [1, 2]. Early differentiation between the two conditions is crucial not only for determining the prognosis, but also for medicolegal purposes [2].

Malformations of the facial nerve can be asymptomatic or present with facial weakness. Dehiscence of the facial nerve canal is the most common developmental anomaly. On the other hand, developmental paralysis can result from facial nerve nucleus abnormality, facial nerve aplasia, or hypoplasia. This can occur in isolation or as part of other syndromes [2, 3]. Hereditary congenital facial paresis (HCFP) is a rare syndrome of isolated dysfunction of the facial nerve. Moebius syndrome, Duane retraction syndrome, HCFP, and other syndromes of cranial nerve palsy constitute the many faces of congenital cranial dysinnervation disorders [4].

Magnetic resonance imaging is the modality of choice for examining cranial nerves by using high-resolution heavily T2WI. The facial nerve itself can be traced from the brainstem to the fundus of the internal auditory canal. The labyrinthine, tympanic, and mastoid segments are not visualized in non-

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contrast MRI sequences [5–7]. However, computerized tomography is well-established imaging modality for studying the facial nerve canal within the petrous part of the temporal bone.

Despite their clinical implications, the MRI features of facial paresis secondary to developmental aberrations (DFP), and its associated anomalies were described in a few case reports [8–12]. The aims of this study are to describe MRI findings encountered in patients presented with DFP and to describe the associated findings with correlation to the embryological implications. A grading system was also suggested in order to describe the severity of the encountered facial nerve anomalies in a more objective way.

Material and methods

After ethical committee approval, MRI of patients who presented with DFP were retrospectively reviewed. The patients with perinatal traumatic insult were not included in the study. The recorded clinical data was analyzed with documentation of other cranial nerve palsy and associated abnormalities (e.g., aural atresia). Patients under the age of 6 years were sedated. Patients were imaged using 3 T MRI imaging system (Siemens Magnetom Skyra Tim, Erlangen, Germany). The isotropic SPACE (Sampling Perfection with Application optimized Contrasts using different flip angle Evolution) sequence was obtained in both axial and coronal planes. Scans were obtained at 20 cm FOV, slice thickness 0.5 mm, matrix of 384 × 384 with no gap, TR 1000 ms, and TE 139 ms. Additional oblique sagittal scans were acquired perpendicular to the internal auditory canal. Axial T1WI was obtained for evaluation of the facial muscles Axial T2WI of the brain was also acquired (Table 1).

Image analysis

Axial FSE T2WI of the brain was examined first searching for any cortical or white matter structural or signal abnormalities. Then, by using the axial SPACE sequence, tracing of the facial nerve and vestibulocochlear nerves was performed throughout their cisternal and intra-canalicular course, followed by examination of the internal auditory canal in the oblique sagittal sections of SPACE sequence. Facial nerve is considered absent (facial nerve aplasia) if it was not visualized throughout its course. We designated the facial nerve hypoplasia when it appeared smaller than the facial nerve in the contralateral internal auditory canal in cases with unilateral facial palsy or when it had interrupted course. Hypoplasia was also confirmed by reduction in size in relation to the other nerves of the internal auditory canal (cochlear and vestibular nerves) especially in cases with bilateral facial palsy [13]. Other cranial nerves (oculomotor, trochlear, trigeminal, and abducens) were also analyzed especially if multiple cranial nerve palsy was clinically documented. Sagittal reconstruction of the axial SPACE sequence was performed to assess the morphology of the brainstem. In addition, the brainstem was examined regarding the presence of areas of abnormal signal intensity and the presence or absence and symmetry of facial colliculi.

The severity of the maldevelopment of the seventh nerve was graded from 0 to 4 on the basis of MRI findings: 0 = no abnormalities, 1 = unilateral facial nerve hypoplasia, 2 = unilateral facial nerve aplasia, 3 = aplasia and/or hypoplasia involving facial nerves on both sides; 4 = unilateral or bilateral facial nerve aplasia or hypoplasia associated with other cranial nerve palsy (Fig. 1).

Results

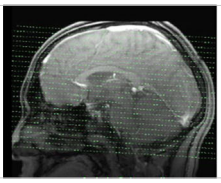
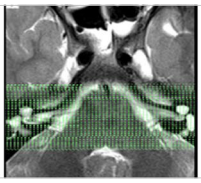
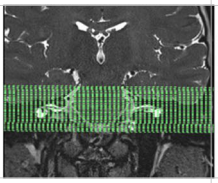
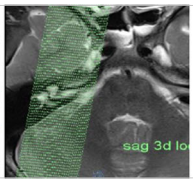
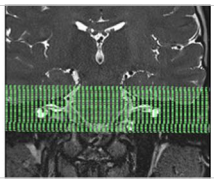
Eleven cases with clinically diagnosed DFP (6 boys and 5 girls) were included in the study, their age ranged 6 months to 17 years (Table 2). DFP was on the right side in five patients (46%), left in two (18%), and bilateral in four (36%) (constituting abnormalities in 15 nerves). CT of the temporal bone was available in two patients (Table 2).

The encountered facial nerve anomalies were ranging from hypoplasia throughout its course (6 nerves, 40%) (Fig. 2), or total aplasia (7 nerves, 47%) (Fig. 3). After proper tracing of the facial nerve, as it exits the brain stem, to exclude the possibility of aberrant course, hypoplasia of its proximal (cisternal) portion with non-visualization of its distal (canalicular) segment (interrupted course) were seen in two nerves (13%) (Figs. 4 and 5); CT of the temporal bone was available in one of them and revealed hypoplastic labyrinthine and tympanic segment of the facial nerve canal (Fig. 4).

Isolated facial nerve abnormality (without affection of other cranial nerves) was present in seven patients constituting grade 1 in five patients (Fig. 2) and grade 3 in two patients. Facial nerve abnormalities associating other cranial nerve palsy (grade 4) was encountered in four patients (associating cochlear nerve aplasia (Fig. 5), abducens nerve aplasia, and trigeminal nerve hypoplasia (Fig. 6). There was one patient with clinically documented oculomotor, trochlear, and abducens nerve palsy; the abducens nerve was aplastic; however, there was no MRI abnormality regarding the other two nerves. Mixed facial and abducens nerve palsy fulfilling the diagnostic criteria of Moebius syndrome was seen in two cases. Isolated unilateral facial aplasia (grade 2) was not encountered in our patients' series.

Regarding other associated findings, aural atresia or hypoplasia was encountered in two patients (Fig. 2). Inner ear anomalies were seen in two patients in the form of dysplastic vestibules and semicircular canals (Fig. 5). Severe atrophy/hypoplasia of the facial muscles was seen at the ipsilateral side of facial palsy in three patients, and on both sides in one case

Table 1 MRI protocol

Sequence	3 plane localiser	T2WI	SPACE	SPACE	SPACE	T1WI
Slice orientation	Axial, coronal, sagittal	Axial sections of the brain	Coronal sections of the IAC and inner ear	Axial sections of the IAC and inner ear	Oblique sagittal sections of the IAC and inner ear	Axial sections of the IAC and inner ear
Localizer						
Slice thickness (mm)	7	5	0.5	0.5	0.5	2.5
Gap (mm)	1.4	1	0	0	0	0
FOV (cm)	25	24	20	20	20	19
TR (ms)	8.6	5300	1000	1000	1000	644
TE (ms)	4	100	139	139	139	10
Matrix	256x233	512x512	384x384	384x384	384x384	179 x256
Flip angle	20	150	120	120	120	120
NEX	2	2	2	2	2	3

FOV field of view, *IAC* internal auditory canal, *NEX* number of excitations

with bilateral facial palsy (Fig. 3). Microcystic lymphatic malformation was seen in the ipsilateral side of hypoplastic facial nerve in one patient (Fig. 2).

Variable brainstem anomalies were encountered as follows: hypoplasia at the pontomedullary junction with absent facial colliculi (in one patient) (Fig. 3); small facial colliculus on the ipsilateral side of facial nerve palsy (in one patient) (Fig. 2) or abnormal signal at dorsal brainstem (in two patients) (Fig. 6).

Discussion

Previous studies have demonstrated perfectly the anatomy of the cranial nerves while traversing the subarachnoid space with high-resolution T2-weighted MR imaging. In addition, oblique sagittal plane obtained perpendicular to the long axis of the internal auditory canal best illustrates the four major nerves passing inside: the facial nerve, cochlear, and superior and inferior vestibular nerves [13–15]. In our study, we encountered different forms of facial nerve developmental aberrations ranging from total aplasia or hypoplasia of the facial nerve, or hypoplasia with interrupted course. As previously published, variable associations of other cranial nerve palsy, brainstem, and ear anomalies were also encountered [8–12]. We proposed a new grading system aiming for objective evaluation of the severity of the encountered anomalies, ranging from unilateral isolated hypoplasia (grade 1) up to abnormality affecting more than one cranial nerve (grade 4).

Among patients with DFP, patients with isolated dysfunction of the facial nerve could represent cases with HCFP. HCFP were described previously in a few case reports documenting facial nerve hypoplasia in only four cases with available MRI; however, MRI was not available in other eight cases [12, 16]. Isolated facial nerve palsy was diagnosed in seven of our cases; facial nerve hypoplasia was seen in five cases (grade 1); one of them had interrupted course, bilateral aplasia (grade 3) in one case, and bilateral abnormality (hypoplasia and aplasia) in another one. To the extent of our knowledge, isolated facial nerve aplasia was rarely described in the literature [8, 14, 15, 17, 18]; and facial nerve hypoplasia with interrupted course was not described previously in cases with isolated facial palsy.

Basic understanding of the hindbrain embryology can give insights into anomalous development of the facial nerve and associated anomalies [19]. This may help the otolaryngologist appreciate and anticipate variation which can be encountered in clinical practice.

During the fourth week of embryogenesis, the notochord promotes the axial ectoderm to form the neural plate, which subsequently forms the neural tube [19]. At its rostral end, the neural tube differentiates into forebrain (prosencephalon), midbrain (mesencephalon), and hindbrain (rhombencephalon). Under the influence of genes, the rhombencephalon is segmented into rhombomeres (r1 to r8) [20]. Facial nerve motor nuclei develop from r4 and migrate caudally through r5 into r6 forming a loop around the abducens nerve nucleus at r5 [19]. This can explain the intimate relation of facial and abducens nerve anomalies in cases of Mobius syndrome as observed in two of our cases as previously described [19, 21, 22]. Absent or small facial colliculi was observed in these two cases associated with abnormal signal at the anatomical location of the intra-axial course of facial and abducens nerves in one of them. Abnormal signal is also seen in another patient with facial nerve palsy without associated abducens nerve dysfunction. Pontine abnormal signal was previously described in cases with DFP in a solitary document and this could represent sequel of prior insult [23].

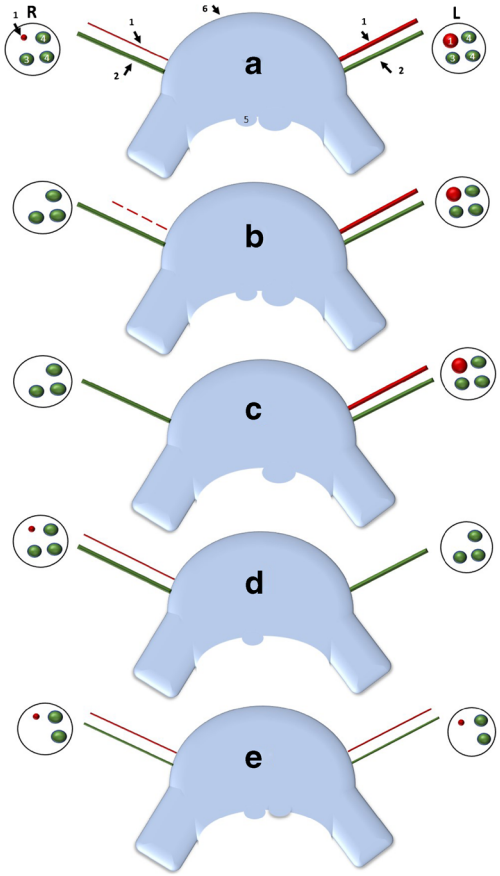


Fig. 1 Diagram illustrates the different grades of facial nerve anomalies a) grade 1: unilateral hypoplasia, the right facial nerve (1) is small in size. Note that the right facial colliculus (5) is smaller in size compared to the left; b) unilateral hypoplasia with interrupted course (dashed line), note that the facial nerve is not visualized in the internal auditory canal; c) grade 2: unilateral aplasia, the right facial nerve is absent on the right side with absent right facial colliculus; d) grade 3: bilateral facial nerve anomalies (in this case: hypoplasia on the right and absent on the left; e) grade 4: unilateral or bilateral facial nerve anomalies associating other cranial nerve palsy (in this case bilateral facial nerve hypoplasia associating bilateral cochlear nerve aplasia). 1: facial nerve; 2: vestibulocochlear nerve; 3: cochlear nerve; 4: superior and inferior divisions of the vestibular nerve; 5: facial colliculus; 6: pons; R: oblique sagittal section of the right internal auditory canal; L: oblique sagittal section of the left internal auditory canal

Despite the similarities between HCFP and Mobius, distinct diagnostic criteria have been proposed for each disorder. The association abducens nerve palsy and either uni- or bilateral facial palsy has been defined for Mobius [24]. Further, heterozygous mutations *PLXND1* or *REV3L* were found in cases with Moebius Syndrome, while *HOXB1* gene mutation was identified in cases with HCFP. It is of note that the majority of patients with either Moebius Syndrome or HCFP were not addressed with these genes. We think that the clinical similarities between Mobius and HCFP should be highlighted within this overlap. Hearing loss and orofacial anomalies can be seen in both Mobius and HCFP [2, 25]. Tomas-Roca et al. have reported a patient diagnosed with bilateral facial paresis and no weakness of the abducens nerves. This patient was found to have heterozygous mutation in *REV3L* that have been identified in individuals with classical Mobius [26]. Moreover, the patients with homozygous *HOXB1* described by Vogel et al. who had bilateral facial palsy and orofacial anomalies was diagnosed initially as Mobius [16]. Additionally, functional studies showed that mutations in the three genes similarly disrupt motor neuron migration to the facial branchiomotor nucleus [26]. Our cases add weight to the suggestion made by Vogel et al. that Mobius and HCFP syndromes are allelic. We suggest that they represent a continuum of phenotypically related dysinnervation disorders.

After induction of motor nuclei, the motor neuron migrates to innervate facial muscles. This process is controlled by other family of genes (*ROBO3* and others) by the effect of chemoattractant and chemorepellent molecules that control axon growth and guidance [27, 28]. We assume that disturbance in this process with failure of these molecules to guide the axons into their destination might cause the nerve to undergo post developmental degeneration after failing to reach its destination and hence, the finding of non-visualized facial nerve in its canalicular course in spite of its visualization in the cisternal course in two of our cases as if the nerve had interrupted course; the degenerated nerve segment might be too small to be detected by the current the resolution of MRI or totally vanished). On the contrary, enlarged facial nerve on the side of facial palsy was documented in a case with familial facial nerve palsy [9]. Other case of proximal thinness with intact distal part was reported. This finding was explained by antegrade destructive changes possibly due to hypoxic episode [8].

The facioacoustic primordium appears during the third week of embryogenesis from neural crest cells. It gives rise to the sensory portion of the facial nerve and geniculate ganglia in addition to the eighth cranial nerve. By the end of the fourth week, the facial and acoustic nerves are better identified. The acoustic nerve terminates on the otocyst (the precursor of the inner ear) which is formed at nearly at the same time [22, 23, 29–31]. This temporally related development of the facial and acoustic nerves can explain the association of facial, cochlear nerve, and inner ear anomalies as observed in two of our cases with sensory neural hearing loss and cochlear nerve and vestibular anomalies. Right facial nerve aplasia associated with bilateral cochlear hypoplasia was previously reported [24, 30]. Bilateral hypoplasia of the facial nerves associated with aplasia of both vestibulocochlear nerves and incomplete partition of both cochleae was also documented [11].

In addition, the external and middle ear development, although independent, is temporally related to facial nerve development which explains the possible coexistence of facial nerve anomalies and deformities of the external and middle ear ranging from mild defects to microtia and atresia [11, 14,

Table 2 MRI features and associated findings in cases with developmental facial palsy

No.	Age	Gender	Facial nerve palsy	Other cranial nerve palsy	MRI findings	Associated anomalies	Grading
1	8 months	f	Right facial palsy	–	• Hypoplasia of the right facial nerve	–	1
2	10 months	f	Left facial palsy	–	• Hypoplasia of the left facial nerve	–	1
3*	1 year	m	Right facial palsy	–	• Hypoplasia of the right facial nerve	• Congenital right microtia • Aural atresia • Hypoplastic middle ear cavity and ossicles	1
4	6 months	m	Left facial palsy	–	• Hypoplasia of the left facial nerve	• Left hemifacial microcystic lymphatic malformation	1
5*	11 years	m	Right facial palsy	–	• Hypoplastic cisternal course of right facial nerve and non-visualized canalicular course • The labyrinthine segment is hypoplastic on CT • Hypoplasia/atrophy of the right facial muscles	–	1
6	7 years	m	Bilateral facial palsy	–	• Hypoplasia of the right facial nerve • Aplasia of the left facial nerve • Dysplastic vestibules semicircular canals	• Bilateral velopharyngeal incompetence (by laryngoscope)	3
7	3 years	m	Bilateral facial palsy	–	• Bilateral aplasia • Normal cochlear nerve, cochlea and semicircular canals • Atrophy/hypoplasia of the facial muscles	• Hypoplasia of the external auditory canal • Bilateral SNHL	3
8	17 years	f	Bilateral facial palsy	Bilateral oculomotor, trochlear, and abducens	• Bilateral facial nerve aplasia • Absent facial colliculi • Bilateral abducens nerve aplasia • Hypoplastic brain stem • Bilateral atrophy/hypoplasia of the facial muscles • Normal MRI of oculomotor, and trochlear nerves	–	4
9	9 years	f	Bilateral facial palsy	Bilateral cochlear	• Hypoplastic cisternal course and almost non-visualized canalicular course on the right side • Hypoplasia on the left side throughout its course. • Aplasia of both cochlear nerves • Dysplastic vestibules and absent semicircular canals	• Bilateral SNHL	4
10	1 year	m	Right facial palsy	Right abducens	• Aplasia of the right facial nerve • Aplasia of the right abducens nerve • Small right facial nerve colliculus • Abnormal signal at the dorsal brain stem	–	4
11	18 months	f	Right facial palsy	Right trigeminal	• Aplasia of the right facial nerve • Normal cochlea and semicircular canals. • Abnormal signal at the dorsal brain stem • Hypoplasia/atrophy of the right facial muscles	• Unilateral SNHL (on the right side) • Neurotrophic keratopathy	4

m male, *f* female, *SNHL* sensory neural hearing loss

*Patients with available CT

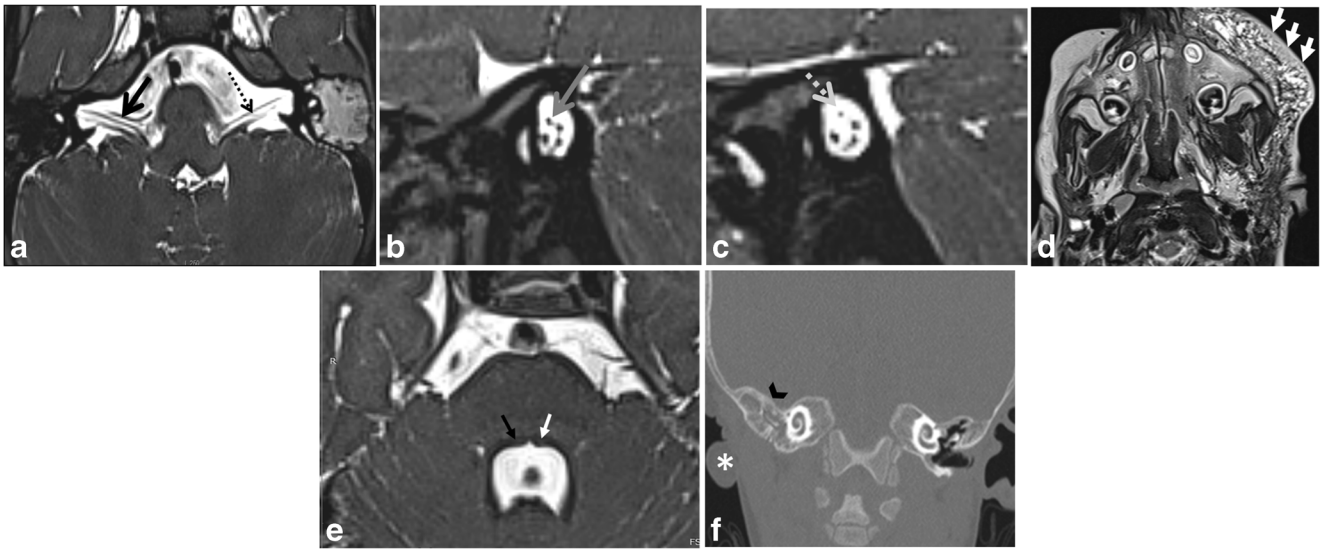


Fig. 2 Two different cases of facial hypoplasia (grade 1) associating microcystic lymphatic malformation (upper raw, patient 4) and middle and external ear anomalies (lower raw, patient 3) a) axial SPACE sequence demonstrates hypoplastic left facial nerve in its cisternal course (dotted arrow) compared to the right one (solid arrow); oblique sagittal SPACE sequence (**b**, **c**). Hypoplastic left facial nerve within the internal auditory canal (dotted arrow), compared to the normal appearing right nerve (solid arrow); **d** Axial T2WI demonstrates left facial microcystic lymphatic malformation (arrows); **e** axial SPACE sequence demonstrates mildly hypoplastic right facial colliculus (black arrow) as compared to normal left side (white arrow); **f** coronal CT of the temporal bone demonstrates bony atresia of the right EAC, hypoplastic ossicles and middle ear cavity (arrow head) and right microtia (asterisk)

32] as observed in two of our cases with isolated facial nerve palsy (grade 1 and grade 3). Auricular malformation was described in previous four cases of HCFP with no evidence of middle ear anomalies. On the contrary, no ear malformations were present at all in the study of Sahin et al. and Uyguner et al. [12, 33].

The parotid gland develops from the parotid primordium early in the eighth week. The extratemporal facial nerve acquires

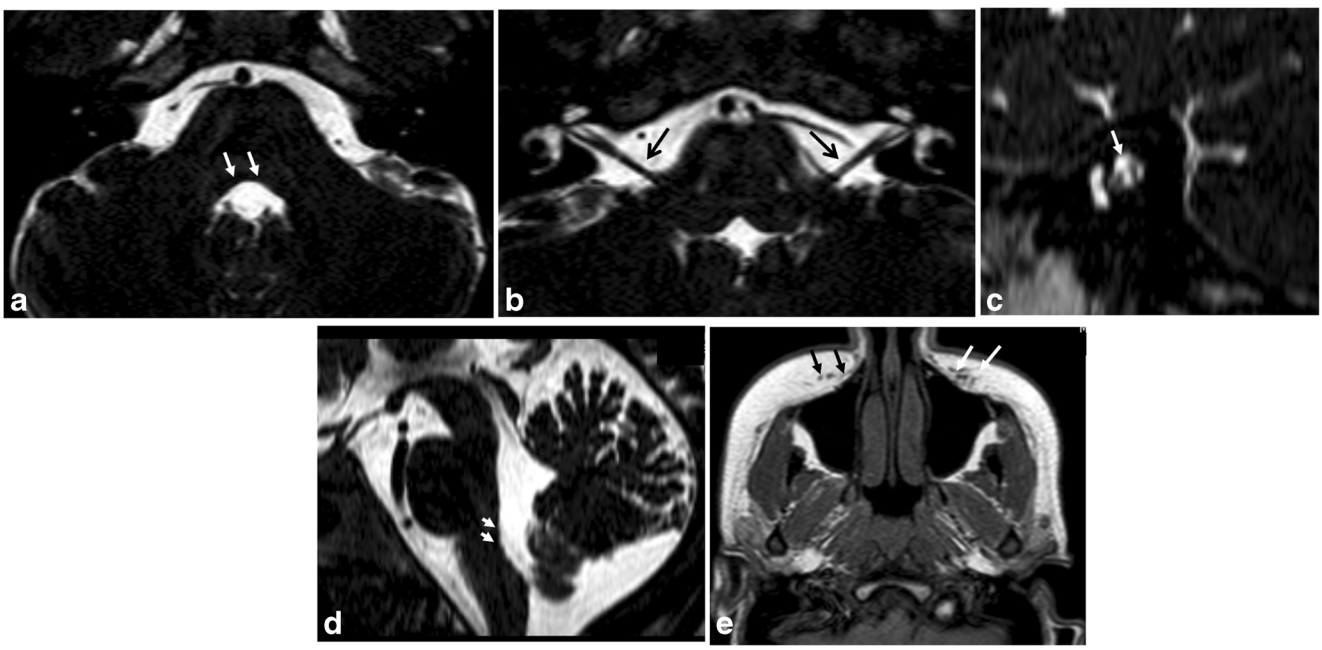


Fig. 3 A 17-year-old girl with multiple cranial nerve palsy (grade 4) (patient 8); **a**, **b** axial SPACE images at the level of the facial colliculi and pontomedullary junction respectively reveal absent facial colliculi (white arrows) and bilateral facial nerve aplasia, only vestibulocochlear nerves are seen (black arrows); **c** oblique sagittal SPACE of the internal auditory canal reveals absent facial nerve (arrow); **d** sagittal reconstruction of the axial SPACE showed hypoplastic brainstem with concave posterior border at pontomedullary junction (arrows). The abducens nerves are not visualized on axial SPACE (not shown); **e** axial T1WI showed marked atrophy/hypoplasia of the facial muscles (arrows)

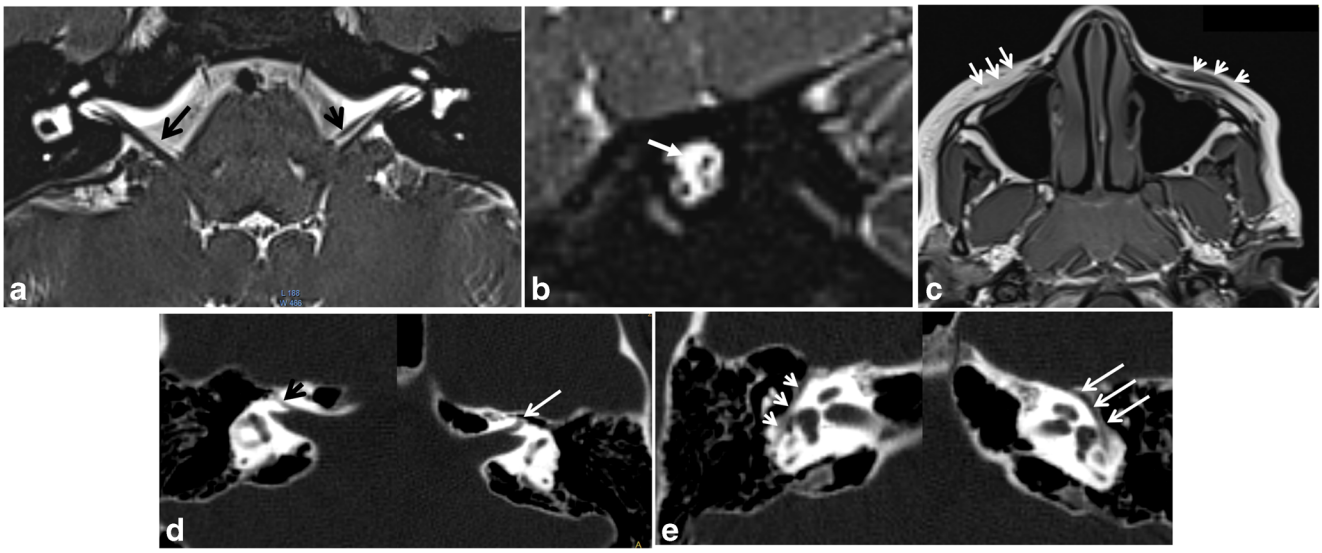


Fig. 4 An 11-year-old boy with right facial nerve hypoplasia with interrupted course (grade 1) (patient 5). **a** Axial SPACE reveals hypoplastic right facial nerve in its cisternal course (long arrow), compared to the normal left side (short arrow); **b** oblique sagittal SPACE through the right internal auditory canal reveals non-visualization of the facial nerve (arrow); **c** Axial T1WI reveals marked atrophy/hypoplasia of the right facial muscle with fat replacement (long arrows) compared to the normal left muscle (short arrows). **d**, **e** CT axial cuts at level of labyrinthine course and at the level of the tympanic course respectively reveals mild hypoplastic right facial nerve bony canal (short arrows) as compared to the left side (long arrows)

a complex relationship with the developing parotid gland as it undergoes progressive branching. By the 12th week, the parotid ductules grow between the branches of the facial nerve to connect the superficial and the deep portions of the gland [29]. Hypoplasia of the facial nerve in association with parotid gland anomalies was described in a solitary case report in which parotid gland agenesis was described [34]. One of our cases with facial nerve hypoplasia was associated with microcystic lymphatic malformation of ipsilateral parotid gland and hemifacial region. We postulate that aberration of the development of the parotid gland, as a result of vascular malformation, might hinder the branching and hence the normal development of the facial nerve. In addition, the presence of microcystic lymphatic malformation might act as a mechanical factor hindering the facial nerve axons to reach its muscular destination which may lead to post developmental degeneration.

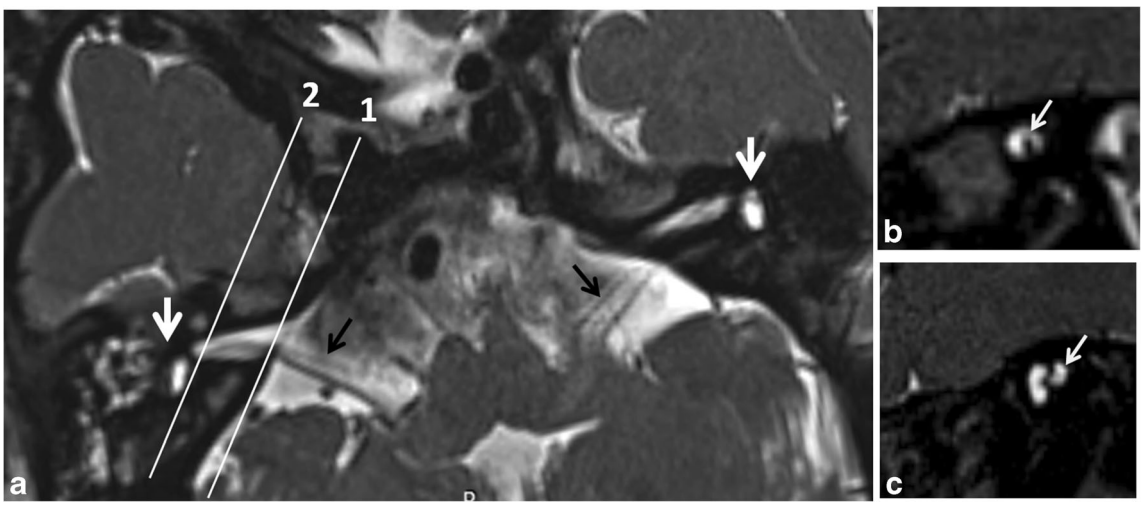


Fig. 5 A 9-year-old girl with bilateral facial nerve paresis and sensory neural hearing loss (patient 9). **a** Axial SPACE reveals bilateral dysplastic vestibules and absent semicircular canals (white arrows), the hypoplastic facial nerves (black arrows) and vestibulocochlear nerves are noted at their cisternal course; **b** sagittal SPACE sequence at the level of medial internal auditory canal (level 1 on image a) reveals single nerve trunk (white arrow); **c** sagittal SPACE at the level of fundus of internal auditory canal (level 2 on image a) reveals no sizable nerves (arrow) in keeping with aplasia of the cochlear nerve and interrupted coarse of the facial nerve

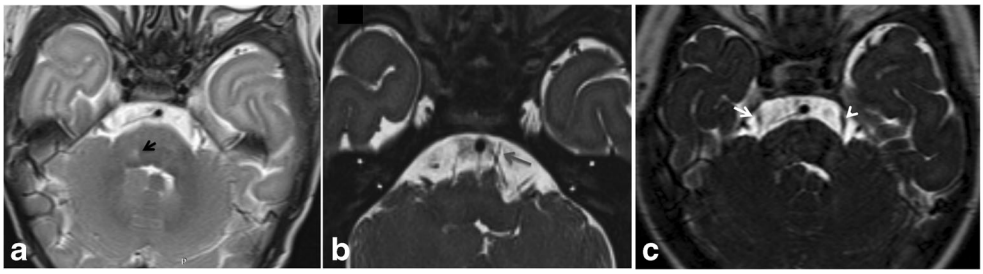


Fig. 6 Two different patients with right facial nerve palsy (grade 4). **a**, **b** A 1-year-old infant with congenital right abducens and facial nerve aplasia diagnosed as Moebius syndrome (patient 10); **a** axial T2WI demonstrates abnormal signal at the dorsal brain stem (black arrow); **b** axial SPACE sequence at the level of pontomedullary junction shows absent right abducent nerve, gray arrow refers to the normal left nerve; **c** An 18-month-old girl with right facial nerve aplasia associated with trigeminal nerve hypoplasia (patient 11). Axial SPACE demonstrates that the right trigeminal nerve (arrow) is relatively small in size compared to the contralateral left side (arrow head) and displays abnormal higher signal

This study is limited by the absence of genetic analysis of the cases of DFP. In addition, apart from Moebius syndrome, other syndromes as Goldenhar or Cayler syndromes and other cases with hereditary myopathies and facial palsy [35–37] were not encountered. However, to the extent of our knowledge, this study constitutes the largest series of cases with DFP describing various congenital facial nerve anomalies that could constitute a continuum of isolated and combined malformations in the spectrum of congenital cranial dysinnervation disorders. Thanks to advent of MRI technology with high-resolution images enabling visualization of tiny nerves passing through the basal subarachnoid space and internal auditory canal.

In conclusion, DFP can be isolated or associated with other cranial nerve palsies. Radiologically, Facial nerve anomalies range from isolated unilateral hypoplasia (grade 1) to abnormality affecting more than one cranial nerve (grade 4). This can be associated with anomalies of the inner, middle, and external ears or parotid gland. We suggest that DFP represent a continuum of phenotypically related dysinnervation disorders.

Compliance with ethical standards

Funding No funding was received for this study.

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval This study was performed in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. For this type of study formal consent is not required.

Informed consent For this type of retrospective study formal consent is not required.

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