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REVIEW

Developmental unilateral facial palsy in a newborn: six cases and literature review

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Abstract

Unilateral facial palsy in a newborn is rarely caused by a developmental defect. It occurs either isolated or in the context of a syndrome. This article describes a multidisciplinary approach towards unilateral, isolated congenital facial palsy along with a literature review. We report six patients, three boys and three girls, who presented with a unilateral facial palsy at birth. Clinical assessment was performed by an ear-nose-throat (ENT) surgeon, a pediatric neurologist, and an ophthalmologist. Magnetic resonance imaging (MRI) of the posterior fossa and computerized tomography (CT) of the temporal bone were requested to exclude structural anomalies of the facial nerve. Imaging revealed the underlying cause in five patients out of six (80%), showing an ipsilateral facial nerve aplasia or hypoplasia. These findings point towards an underlying developmental defect and underscore the importance of MRI in the diagnostic work-up. Surgical and non-surgical therapies were discussed with the parents.

Conclusion: Congenital unilateral facial palsy caused by a developmental defect outside the context of a syndrome is rare. A multidisciplinary approach is recommended to differentiate between various causes and to initiate timely treatment.

What is Known:
- *Congenital facial palsy is mostly caused by environmental/external fcators.*
- *However in rare cases it can be developmental defect.*

What is New:
- *This paper describes 6 cases of isolated congenital facial palsy related to a developmental defect and presents the largest case series in the literature caused by aplasia/hypoplasia of the facial nerve.*
- *MRI and CT-imaging allow for an assessment of the facial nerve at the root entry zone of the brainstem and along its course through the middle ear or the face. Moreover, they proved to be helpful in differentiating between several causes of congenital facial palsy.*

Keywords Isolated congenital developmental facial nerve palsy · Facial nerve aplasia · Facial nerve hypoplasia

Abbreviations

3D-CISS	Three-dimensional constructive interference steady state
AICA	Anterior inferior cerebellar artery
CPAP	Continuous positive airway pressure
CT	Computed tomography
Dr.	Doctor
EMG	Electromyography
ENT	Ear-nose-throat
GSPN	Greater superficial petrosal nerve

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HB	House-Brackmann
IAC	Internal acoustic canal
M.	Musculus
MRI	Magnetic resonance imaging
N.	Nervus
UMCG	University Medical Center Groningen

Introduction

Facial palsy present at birth (congenital facial palsy) may be caused by external/environmental factors present before birth or due to a developmental defect. The most common external factor is birth trauma or prenatal compression. In neonates, the facial nerve is located more superficially as it leaves the stylomastoid foramen and is therefore more vulnerable to compression against the sacral prominence of the mother's pelvis or to injury during forceps extraction at birth. Other external factors are prenatal infections, vascular injury, or exposure to teratogens [12, 21, 25, 27]. Developmental congenital unilateral facial palsy is an uncommon pathology with an estimated incidence of 2.1/1000. It may occur as an isolated condition or in the context of a syndrome [7, 22].

A second distinction should be made between "central" and "peripheral" facial palsy. In central facial palsy, the lesion is located along the corticobulbar tract affecting the upper and lower motor neurons. Central facial palsy affects the upper motor neurons and the ventral division of the lower motor neurons, leaving the dorsal division of the lower motor neurons intact. This will result in a contralateral facial muscle weakness of the lower part of the face, while preserving the muscle function of the forehead. A peripheral facial palsy results from a lesion at the root entry zone of the brainstem or more distally along the course of the facial nerve in the middle ear or the face and will result in an ipsilateral muscle weakness involving both the upper and lower part of the face [14, 18]. In addition, the facial nerve also has sensory and visceral functions. Therefore, a deficit may provide a variable clinical picture [5, 18]. Facial asymmetry, lagophthalmos, hyperacusis, change in taste (first two-thirds of the tongue), or increased tear production are symptoms that may occur [5].

According to the literature, congenital facial palsy caused by birth trauma is mostly transient and usually resolves within 2 years. Facial palsy caused by a developmental defect has a less favorable prognosis [12, 13, 19, 24]. Nevertheless, even with a history of a birth trauma, a complete work-up is recommended to exclude other causes and to avoid therapeutic implications [8, 12, 28]. The presence of dysmorphic features such as malformation of the pinna, with or without atresia of the external ear canal, absence of the major pectoral muscle, impaired abduction of the eye, or bilateral congenital facial palsy point towards a syndromic cause. On the other hand, a history of a prolonged labor, the use of forceps, and presence of a hematoma at the ear- and mastoid region, are more suggestive for a birth trauma [14, 20, 22, 24].

In this paper, we discuss the etiology and multidisciplinary approach to six newborns with a peripheral, unilateral congenital facial palsy due to a developmental anomaly outside the context of a syndrome.

Case reports

Six patients with unilateral peripheral facial palsy present at birth were seen at the Department of Pediatric Neurology and Pediatric Otorhinolaryngology of the Antwerp University Hospital. A detailed description of these six cases is presented in Table 1.

All neonates were born at full term after an uneventful pregnancy. One patient (case 4) was born through a secondary cesarean section. A vacuum extraction was used in case 2. Two neonates had some difficulties immediately after birth, which could be treated successfully by supportive care. There were no signs of birth trauma in any of these cases. There was no history of facial palsy in the families.

Physical examination revealed a House-Brackmann grade 4 facial palsy at the right side in three cases and at the left side in the other three patients. All infants passed the neonatal hearing screening. Ophthalmological investigation showed hyperopic anisometropic amblyopia in one case (patient 6), which could be treated with exercises and glasses. Patient 2 presented a mild superficial keratitis and was easily treated with hyaluronic acid. To prevent eye damage, prophylactic use of therapy with artificial tears was recommended in every patient. Neurological examination in patient 3 showed a plagiocephaly with a slight deformity of the auricle and neglect of the left side of the body. Patient 5 had a developmental delay, which was already visible from early childhood. No signs of a syndrome or involvement of other cranial nerves were seen in any case. Additional investigations by karyotyping and micro-array showed no clinically relevant abnormalities.

Magnetic resonance imaging (MRI) was performed in every patient, using a 1.5 T superconducting system. In addition to routine MR-sequences, the 3D-CISS technique was also obtained to make a better differentiation between the cranial nerves and cerebrospinal fluid. Aplasia of the facial nerve was seen in three cases (patient 2, 4, and 5), hypoplasia in two cases (patient 3 and 6), and only one patient (patient 1) showed no abnormalities of the facial

Table 1 Overview of the patients' data. *CPAP*, continuous positive airway pressure; *CT*, computed tomography; *L*, left; *MRI*, magnetic resonance imaging; *M*, musculus; *NL*, Netherlands; *OME*, otitis media with effusion; *R*, right; *UMC*, University Medical Center; *w*, weeks; *y*, years

Patient (initials; age at presentation)	Location of facial paralysis; grade (HB-scale)	Obstetrics	Hearing assessment	EMG facial region	CT/MRI temporal bone	Other remarks/treatments
Patient 1; ♀ (7 w)	Right; Grade 4; Incomplete palsy: involvement of R corner of the mouth and lack of R eye closure.	Full term.	Normal neonatal hearing screening.	-Abundant arbitrary activity. -Response after stimulation: *R orbicular oris region: Borderline normal response *R frontalis region: Absent response -Activity at rest is not measurable.	-MRI: no lesions. -CT: aberrant vertical segment of the R facial nerve (Fibrotic cord).	-Neck ultrasound: normal -Crossed facial nerve graft (two stages): satisfactory result. (UMC Groningen; NL) -Karyotype and micro-array: normal -Speech therapist
Patient 2; ♂ (2 y and 9 m)	Right; Grade 4; Lack of R eye closure.	Birth at 40 weeks' gestation with ventouse (vacuum extraction).	Normal neonatal hearing screening.	Not performed.	-MRI: Aplasia of the R facial nerve and atrophy of the R facial musculature (M. nasalis and M. zygomaticus). -CT: small R facial canal.	-Micro-array: class 3 deletion (14q22.1), also present in the asymptomatic mother. No clinical relevance. -Surgery: awaiting policy.
Patient 3; ♂ (15 w)	Left; Grade 4; Asymmetric crying face.	Birth at 40 weeks' gestation. Respiratory distress treated with CPAP for a short period.	Normal neonatal hearing screening.	-Reduced arbitrary activity. -Response after stimulation: *L orbicularis oris region: Normal response *L frontal region: Disturbed response	-MRI: hypoplasia of the L facial nerve. -CT: normal facial canal on both sides.	-Plagiocephaly and mild dysplasia of auricle at the L side. -Preferred positioning on the right side. Reduced use of the L body side, treated with physiotherapy. -Delayed speech-language development, treated with speech therapy. -Karyotype: normal. -Micro-array: Pathogenic duplication in the 22q11.21 region. -No other cranial nerves involved. -Further awaiting policy.
Patient 4; ♀ (6 w)	Left; Grade 4; Depressed L corner of the mouth. Lack of L eye closure and increased tear production.	Birth at 39 weeks with secondary cesarean section.	Normal neonatal hearing screening.	Not performed.	-MRI: Aplasia of the L facial nerve. -CT: Duplication of left-sided labyrinthine segment. The L tympanic segment is not well aligned. Partial opacification of the mastoid cells.	-Surgery envisaged around the age of three. (UMC Groningen; NL)
Patient 5; ♀ (4 y)	Left; Grade 4; Discovered 6 months after birth.	Normal pregnancy. Induced labor. APGAR-score 4.	Normal neonatal hearing screening.	Not performed.	-MRI: aplasia of the L facial nerve. -CT: normal facial canals. Partial opacification of the L mastoid cells.	-Micro-array: duplication in 6q27 region, without clinical relevance. -Autism spectrum disorder. -Swallow difficulties and development disorder.

Table 1 (continued)

Patient (initials; age at presentation)	Location of facial paralysis; grade (HB-scale)	Obstetrics	Hearing assessment	EMG facial region	CT/MRI temporal bone	Other remarks/treatments
Patient 6; ♂ (1 y and 3 m)	Right; Grade 4; R eye is more photosensitive and produces more tears.	No obstetric difficulties.	Normal neonatal hearing screening.	Not performed.	-MRI: R facial nerve hypoplasia. -CT: bilateral opacification of mastoid cells.	-Amblyopia (R eye). -Placement of tympanostomy tubes, because of recurrent OME. -Further awaiting policy.

nerve on MRI (Appendix). Complementary information by CT-imaging showed an aberrant development of the facial canal in three cases (patient 1, 3, and 4). Facial EMG was performed in only two patients to obtain an estimate on the prognosis (patient 1 and 3).

Discussion

Congenital unilateral facial nerve palsy is an uncommon condition. This pathology is most often acquired but can also occur due to a developmental defect, either isolated or in the context of a syndrome. After careful investigation our patients all presented a peripheral facial palsy due to an isolated developmental defect. In contrast to the acquired conditions, only a few cases of isolated development facial palsy have been reported in the literature (Table 2). We present the largest series of five patients with unilateral congenital facial palsy caused by an aplasia/hypoplasia of the facial nerve. It is noteworthy that despite the hypoplasia or aplasia of the facial nerve, none of them had a total facial palsy. Little is known about the etiology and embryogenesis of this isolated condition [11]. Trezis et al. described aplasia/hypoplasia of the cranial nerve nuclei, nuclear agenesis, peripheral abnormalities of the facial nerve, and primary myopathy as the four most important types of anatomic anomalies of the facial nerve, leading to developmental peripheral facial palsy [18, 24].

The development of the facial nerve starts at 3 weeks of gestation until week 16, and the nuclei are located in the brainstem [18, 23]. The motor fibers start within the reticular formation of the lower third of the pons and go around the nucleus of the abducens nerve. They continue their course up to the cerebellopontine angle, via the temporal bone, and exit the skull through the stylomastoid foramen [18, 24]. The extracranial segment supplies the facial musculature, the scalp, the auricle, the buccinator and platysma, the stylohyoideus, and the posterior part of the digastric muscle [18, 20]. The sensory and visceral fibers branch from the intermediate nerve, which starts to develop at week five until week six. The nervus intermedius leaves the brainstem at the pons and passes through the internal auditory meatus between the motor fibers of N. VII and N. VIII. The nerve can be subdivided into the greater superficial petrosal nerve (GSPN), which branches to the lacrimal gland, and the chorda tympani, which delivers fibers to the submandibular and the sublingual glands. The chorda tympani, whose cell bodies are located in the ganglion geniculi, is also responsible for the sense of taste of the first two-thirds of the tongue [18]. In addition, the intermediate nerve receives somatic sensations

Table 2 Summary of previous studies on isolated developmental facial palsy

Authors	Type of article	*N*	Main conclusions
JK. Terzis, K. Anesti et al. 2011 [24]	Non-systematic review	Not applicable	Review about pathogenesis, and how to approach developmental facial palsy present at birth.
I. Kumar, A. Verma, R. Ojha, P. Aggarwal et al. 2016 [11]	Case report	Two patients with an isolated developmental facial palsy present at birth.	The crucial role of MRI in the diagnosis of a developmental defect in the context of a congenital facial palsy.
B. Jemec, AO. Grobbelaar, DH. Harrison et al. 2000 [10]	Non-systematic review	Fifteen patients with an isolated developmental facial palsy present at birth.	This study investigated the frequency of nuclear aplasia as the cause of an isolated facial nerve palsy at birth. An abnormality of the CNS was found in four patients (27%) by using MRI-scans.
M. Sasaki, Y. Imamura, N. Sato et al. 2008 [22]	Case report	Two patients with a developmental facial palsy present at birth: One patient due to an isolated defect, the other in the context of a syndrome.	The importance of (3D-CISS) MRI to differentiate between a congenital nerve aplasia and a birth trauma.
Y. Elikplim, O. Azdad, M. Lahkim, L. Jroundi, F. Zahrae et al. 2018 [16]	Non-systematic review + Case report	One patient with a congenital developmental facial palsy due to agenesis of the facial nerve.	Facial nerve aplasia is an extremely rare condition, an isolated facial nerve aplasia even rarer. Diagnosis is possible with the use of T2 weighted 3D MRI sequence.

CNS, central nervous system; *N*, number of included patients; *3D-CISS*, three-dimensional constructive interference in steady state

derived from the external auditory canal, the pinna, and the region of the mastoid [9, 18].

A newborn presenting a facial nerve palsy should pass a complete clinical examination, and the parents should be questioned in detail about the pregnancy and delivery [18]. During the history taking, information should be obtained from the obstetric records, the temporal course of the palsy, psychomotor development, and visual problems. We recommend a multidisciplinary approach including an examination of the neonate by a pediatric ENT surgeon, a pediatric neurologist, and a pediatric ophthalmologist. A first step is to document the severity of the facial palsy, to notice improvement or progression at future appointments [14, 18]. The House-Brackmann scale is a commonly used clinical tool for staging, ranging from one to six depending on the severity of the palsy. A score above three indicates an incomplete eye closure. This condition should be treated prophylactically with artificial tears or ophthalmic ointments to avoid keratopathy and subsequent blindness [2]. Redness of the eye is a warning sign and should be treated promptly [14].

Unlike adults it is not possible for a newborn to execute certain assignments on command; consequently it is important to look for spontaneous facial movements and ask for the parental observations [14]. In addition, complementary information can be obtained from photos and videos taken by the parents. Clinical examination should distinguish between a central and a peripheral palsy, and attention should be paid to syndromic signs [14, 18]. The presence of different dysmorphic features or bilateral facial palsy makes a syndrome more likely [14, 20, 22, 24]. Moebius, Poland, Goldenhar, or cardiofacial syndromes are some examples of syndromes associated with peripheral facial palsy. Moebius syndrome is a rare condition and is recognized by an alteration in the development of the facial nerve together with the abducens nerve. In addition, association of other cranial nerves deficits, orofacial malformations, and limb defects are possible as well. Verzijl et al. described this pathology not only as a developmental problem of the cranial nerves or nuclei, but also as a disorder of the rhombencephalic development. Neuropathologic examination has revealed hypoplasia of the entire brainstem in patients with Moebius syndrome [25]. Poland syndrome is characterized by congenital facial palsy and the absence of the pectoralis major muscle. Goldenhar syndrome may present as a congenital facial palsy associated to facial deformities (aural atresia, mandibular hypoplasia) together with developmental problems of the skeleton or the central nervous system. Cardiofacial syndrome is a combination of defect of

the musculus anguli oris together with cardiac malformation. A deletion of 22q11.2 is frequently present.

After careful history taking and clinical investigation, all newborns should undergo a neonatal hearing screening. In case of failure or when in doubt, objective assessment should be performed by "auditory brainstem response testing" (ABR) [6]. Tympanometry is a complementary test which allows to discover the presence of the acoustic stapedius reflex and could help to locate the lesion along the facial nerve [13]. This test is less accurate when used in neonates aged under 6 months and could be replaced by wideband acoustic immittance [1].

In the case of each of our patients, either aplasia/hypoplasia of the facial nerve or an aberrant facial canal was found by imaging and the facial palsy was attributed to this anatomical abnormality. MRI is the examination of choice to evaluate soft-tissue changes and is superior to CT-imaging [9]. In case of congenital facial palsy we highly recommend the use of MR-imaging, more specifically 3D-CISS technique, to make a proper differentiation. 3D-CISS is an MRI-technique which is often used to detect anatomical abnormalities of the cranial nerves, due to the superior contrast resolution between a nerve and the cerebrospinal fluid [11, 15, 22]. This technique provides superior topographic information, which is often missed by routine MRI sequences. It is important to look for abnormalities throughout the whole course of the facial nerve, starting at the brainstem until the parotid gland [9, 13]. When the facial nerve becomes small on MRI while traveling through the bone, it is an indication of hypoplasia. When no lesions are found on MRI, CT-imaging can give some complementary information about the bony facial canal, the presence of middle- or inner ear pathology, and the surrounding anatomic structures. This information is not only useful during the diagnostic process but also required if surgery is necessary (Diagram 1). When looking at the CT-images we suggest to trace carefully for aberrant routes throughout the entire course of the facial nerve, since many cases were described in the literature where the facial nerve

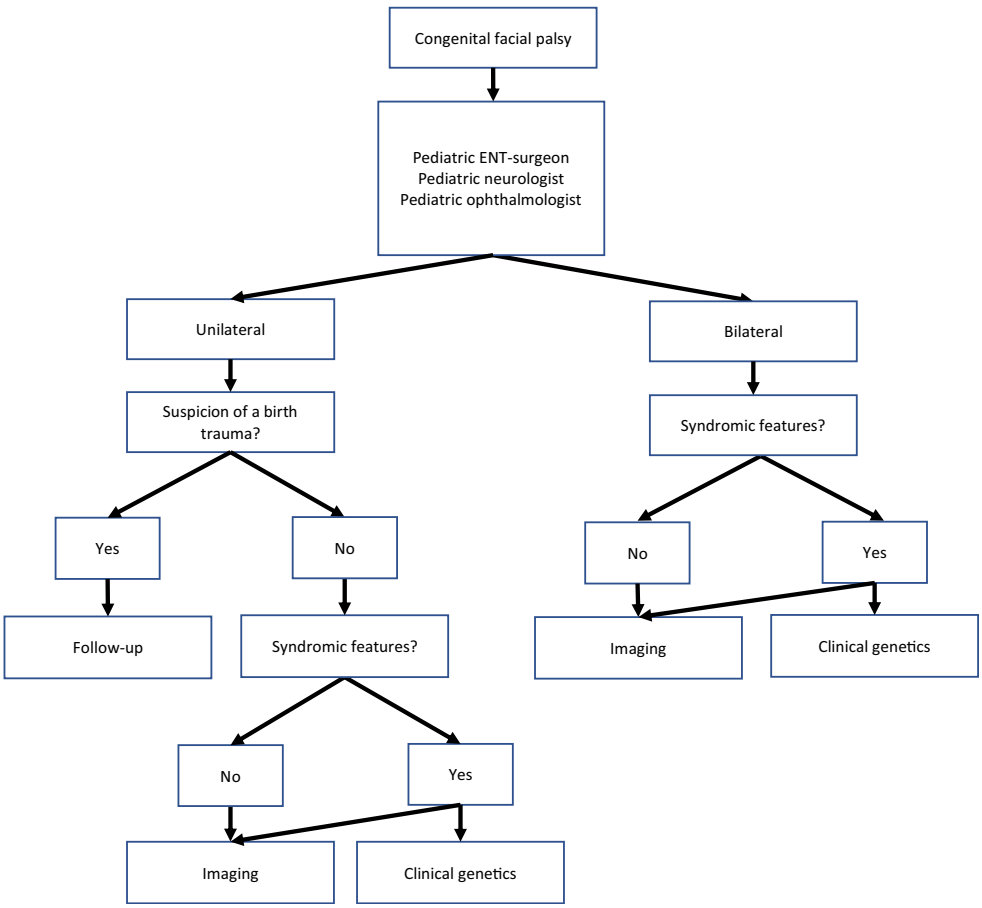

Diagram 1 Diagnostic approach to congenital facial palsy. 3D-CISS, three-dimensional constructive interference in steady state; CT, computed tomography; EMG, electromyography; ENT surgeon, ear-nose-throat surgeon

exits the IAC earlier or where the nerve runs parallel to the IAC [9, 11, 13, 24].

In certain cases EMG was performed to predict the outcome. An electrode was inserted in the M. orbicularis oris, the M. orbicularis oculi, and the M. frontalis to measure both voluntary and spontaneous muscle contractions. The information of the nerve deficit together with the muscle deficit helps to distinguish between a developmental or a traumatic cause [6, 19, 24].

The facial nerve is mainly responsible for motor function, which makes it possible for a child to communicate with others and to show emotions [8, 28]. A properly working facial musculature is necessary for a good development [8]. The palsy of the facial nerve can be very hard bearing for a child. They are confronted with functional problems and often experience a high level of psychological stress. The main goals of treatment are to ensure a proper development and to monitor the well-being of the infant during childhood. Most of the neonates with permanent facial palsy experience few functional problems during the first months of life, justifying a watchful waiting policy. In addition, we recommend mime therapy as a complementary treatment. According to the literature, this therapy could improve facial symmetry and reduces the severity of the facial palsy by inhibiting synkinesis and promoting both emotional expression and functional movements [3, 4, 26]. Patients are encouraged to modulate coordination of viable muscles rather than trying to stimulate paralyzed ones. To achieve the best results, they should carry out these exercises at home. Mime therapy not only is beneficial to improve functionality of the facial musculature but also contributes to the well-being and the self-esteem of the patient [2, 3, 17].

If the facial palsy is a barrier for a proper development or well-being of the patient, then surgery should be considered. There are two categories of surgical techniques, namely the static and the dynamic reconstructions. Static reconstructions will ensure that the face looks normal at resting position, without regaining any function. In contrast, the dynamic techniques will reconstruct the normal appearance of the face and additionally generate the facial movements [6]. There are different types of dynamic techniques such as a temporalis elongation myoplasty, microvascular free flap, sural nerve grafting, or the cross-facial nerve grafting. The crossed facial nerve graft and the free vascularized muscle transfer are rather exceptionally used, but are the most effective surgical techniques for children with congenital unilateral facial palsy [26]. One of our patients (patient 1) received a crossed facial nerve graft, executed in two tempi. Afterwards mime therapy was started to regain facial movements and to minimize the risk of synkinesis. Although it is impossible to obtain the same functional muscle movements as a normal individual, yet optimal results will still be achieved if treated early and if surgery is followed by mime therapy to achieve a better outcome [8, 24, 26].

Conclusion

Isolated developmental unilateral facial palsy is an uncommon type of facial palsy, which is rarely described in the literature. A multidisciplinary approach is recommended to differentiate between an acquired defect and a developmental problem and to allow for timely treatment of the condition when appropriate. Through MRI and CT-imaging, an assessment could be made about the facial nerve and the anatomy of the internal auditory canal. A congenital aplasia or hypoplasia of the facial nerve without the presence of a syndrome points towards an isolated developmental defect. Treatment should focus on the development and well-being of the child. For most children the facial palsy has little functional impact and a wait-and-see policy is advised. Lagophthalmos should be treated promptly to prevent keratitis. Mime therapy is recommended as a complementary treatment, given its beneficial effect on facial muscle control and the inhibition of synkinesis. In exceptional cases surgery may be considered.

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Authors' contribution Authors LD, AB and BC are responsible for content and scripture of manuscript. Author CV is responsible for neuroimaging evaluations. Author AB is responsible for ENT evaluations. Authors BC is repsonsible for clinicals evaluations. All authors have read and agreed to the content of the manuscript.

Compliance with ethical standards

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

Ethical approval All procedures performed in studies involving human participants were 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.

Informed consent Informed consent was obtained from all individual participants included in the study.

Appendix

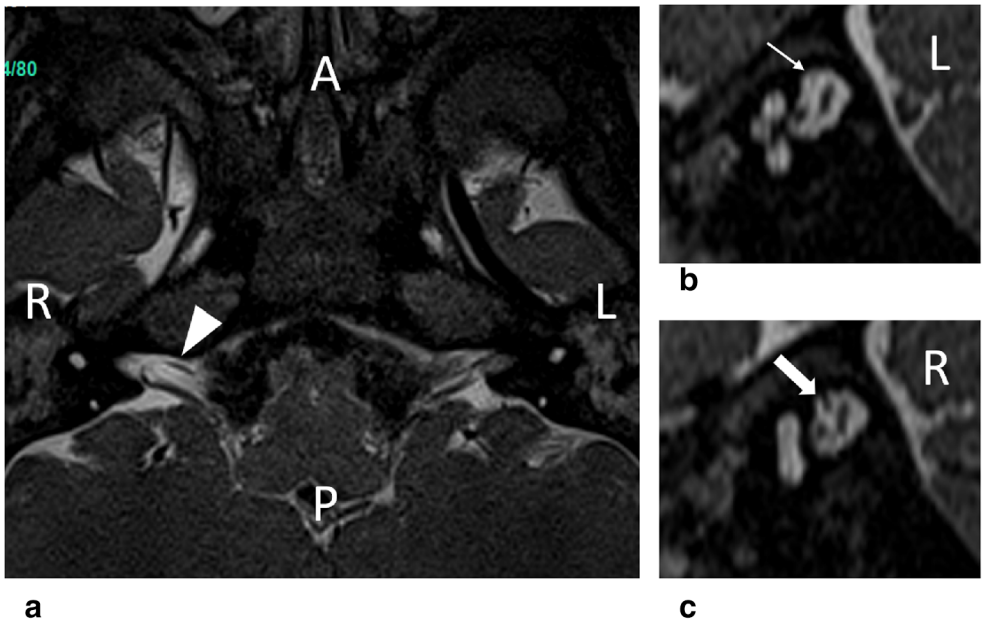

Fig. 1 MRI images of patient 2 performed in 2012. **a** Axial 0.4-mm 3D-constructive interference in steady state (3D-CISS) sequence shows an aplasia of the right facial nerve (white arrowhead). **b** Parasagittal 0.5-mm reconstructions through the right internal auditory canal (IAC) show an aplastic facial nerve (small white arrow). **c** Parasagittal 0.5-mm reconstructions through the left internal auditory canal (IAC) show a normal facial nerve (thick white arrow). A, anterior; L, left; P, posterior; R, right

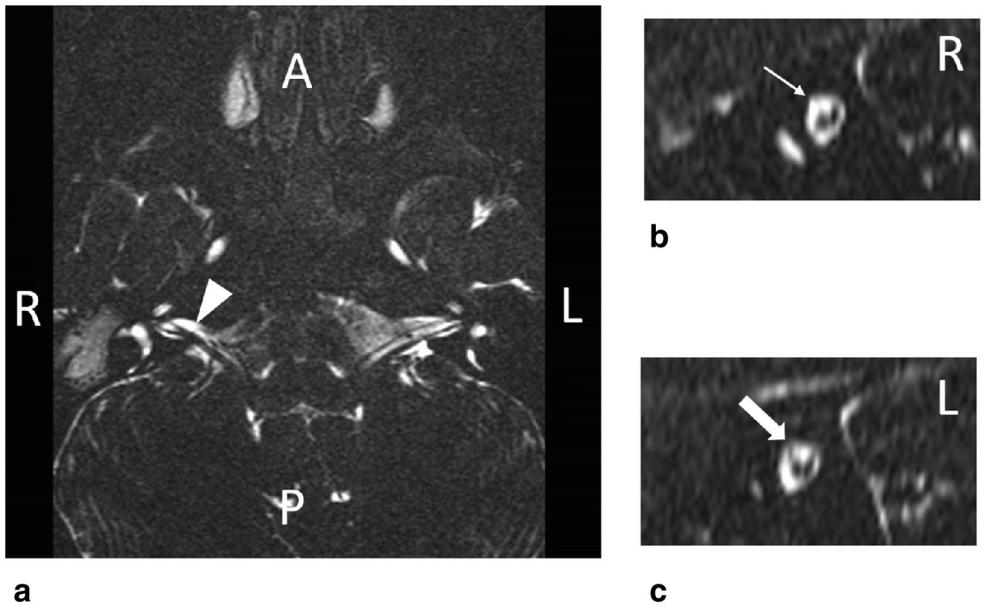

Fig. 2 MRI of patient 6 performed in 2013. **a** Axial 0.4-mm 3D-constructive interference in steady state (3D-CISS) sequence shows a hypoplastic right facial nerve (white arrowhead). It is important not to confuse the anterior inferior cerebellar artery (AICA) loop in the internal auditory canal (IAC) with a nerve. **b** Parasagittal 0.5-mm reconstructions through the right IAC show a hypoplastic facial nerve (small white arrow). **c** Parasagittal 0.5-mm reconstructions through the left IAC show a normal facial nerve (thick white arrow). A, anterior; L, left; P, posterior; R, right

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