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ANNOTATSIYA

O'tgan 8 oy davomida o'rta quloqning kombinatsiyalangan patologiyasi bilan tashqi eshitish yo'lining tug'ma atreziyasi bo'lgan 34 nafar bemorga oldingi yondashuv bilan 34 ta birlamchi operatsiya o'tkazildi. Kuzatuv davri 8 oygacha tashkil etdi. Eshitish natijalari suyak-havo intervali (SHI) 35 dB bo'lgan konduktiv tipdagi eshitish pastigi 32 (94,1%) bemorlarda aniqlangan, 2 bemorda aralash tipdagi eshitish pastligi va SHI 30 dB (5,9%). Yuz nervining shikastlanishi yoki sensorinevral eshitish pastligi holatlari aniqlanmadi. Yuz nervi monitoringi qo'llanildi. Bemorlarning suyaklari buzilmagan holda yoki protezlarni qo’llash eshitish qobiliyatini yaxshilash natijalariga erishildi. Ehtiyotkorlik bilan yumshoq to'qimalar texnikasi, barcha ochiq suyaklarni qoplaydigan split laxtalar stenozning oldini olishning asosi hisoblanadi. To'g'ri yo'naltirish va yumshoq to'qimalar texnikasi tug'ma quloq kanali aterezini muvaffaqiyatli tuzatishning kalitidir.

Kalit so'zlar: tashqi eshitish yo'lining tug’ma atreziyasi, meatoplastika, ossikuloplastika, timpanoplastika.

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НАШ ОПЫТ ХИРУРГИЧЕСКОГО ЛЕЧЕНИЯ ПРИ ВРОЖДЕННОЙ АТРЕЗИИ НАРУЖНОГО СЛУХОВОГО ПРОХОДА С СОЧЕТАННЫМИ ПАТОЛОГИЯМИ СРЕДНЕГО УХА

АННОТАЦИЯ

За последние 8 месяцев 34 пациентам с врожденной атрезией наружного слухового прохода с сочетанной патологии среднего уха было выполнено 34 первичные операции передним доступом. Срок наблюдения варьировал до 8 месяцев. Результаты слуха были у 32 (94,1%) пациентов было обнаружено кондуктивная тугоухость с КВИ 35 дБ, у 2-х пациентов было выявлено снижение слуха по смешанному типу и КВИ составило 30 дБ (5,9%). Случаев повреждения лицевого нерва или нейросенсорной тугоухости не было. Использовался мониторинг лицевого нерва. Результаты слухаулучшения были достигнуты с помощью интактных косточек пациентов или протезов. Тщательная техника мягких тканей с расщепленными трансплантатами, покрывающими всю обнаженную кость. Является ключом к предотвращению стеноза. Правильная ориентация и техника мягких тканей являются ключом к успешной коррекции врожденной атерезии слухового прохода.

Ключевые слова: врожденная атрезия наружного слухового прохода, меатопластика, оссикулопластика, тимпанопластика.

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OUR EXPERIENCE IN SURGICAL TREATMENT IN CONGENITAL ATRESIA OF THE EXTERNAL AUDIO CANAL WITH COMBINED PATHOLOGIES OF THE MIDDLE EAR

ANNOTATION

Over the past 8 months, 34 patients with congenital atresia of the external auditory canal with combined pathology of the middle ear underwent 34 primary operations with an anterior approach. The follow-up period varied up to 8 months. Hearing results were found in 32 (94.1%) patients with conductive hearing loss with a KVI of 35 dB, in 2 patients a hearing loss of a mixed type was detected and the KVI was 30 dB (5.9%). There were no cases of facial nerve injury or sensorineural hearing loss. Facial nerve monitoring was used. Hearing improvement results have been achieved with patients' intact bones or prostheses. Careful soft tissue technique with split grafts covering all exposed bone. Is the key to preventing stenosis. Proper orientation and soft tissue technique are key to successful correction of congenital ear canal ateresis.

Keywords: congenital atresia of the external auditory canal, meatoplasty, ossiculoplasty, tympanoplasty.

Introduction. In contrast to anatomical variants, congenital malformations-synonymously referred to as anomalies or dysplasias-are characterised by deviation not only from normal anatomical development but also from regular function. They may result from developmental delay, abnormal embryogenesis or both due to spontaneous genetic mutations - this occurs in most congenital malformations of the outer and middle ear - genetic transmission and exogenous factors - in about 10% of cases. Anomalies of the outer ear are common and occur in approximately 5% of the total population [1]. The most common malformations include a combined malformation of the outer and middle ear called congenital aural atresia [2]. According to N. Weerda [3], 50% of ENT malformations are ear malformations. In malformations of the external ear, the right side is most commonly affected (58-61%) and most cases (about 70-90%) are unilateral [3,4,5]. The reported prevalence varies by world region, ranging from 0.83 to 17.4 per 10,000 births, and the prevalence is thought to be higher in Latin Americans, Asians, and Native Americans [6].

The overall incidence of ear malformations is about 1: 3800 newborns [5]. Cases of malformations of the external ear have been reported from 1: 6000 newborns [7] to 1:6830 newborns [8]. Severe malformations occur in 1: 10,000-1: 20,000 newborns [3], and gross malformations or aplasias in 1: 17,500 newborns**.**

The prevalence of microtia is higher than 3: 10,000 according to M. Schloss [9].

Congenital atresia of the external auditory canal occurs in 80% of patients with microtia [10].

Ear malformations may be genetic or acquired. Among congenital malformations, 30% are associated with syndromes accompanied by additional malformations and/or functional loss of organs and organ systems. Examples are otofacial dysostosis (e.g., Treacher-Collins syndrome, Goldenhar syndrome), craniofacial dysostosis (e.g., Crouzon syndrome, Aper syndrome), otocervical dysostosis (e.g., Klippel-Feil syndrome, Wilderwank syndrome), otoskeletal dysostosis (e.g. van der Huwe-de Klein syndrome, Albers-Schönberg syndrome) and chromosomal syndromes such as trisomy 13 (Paetau syndrome), trisomy 18 (Edwards syndrome), trisomy 21 (Down syndrome) and 18q syndrome. Non-syndromal ear malformations show only ear anomalies without any other malformations [11] published a detailed list of syndromes and conditions associated with congenital ear malformations. In all genetically determined malformations (syndromal and non-syndromal), a high frequency of spontaneous genetic mutations can be assumed [12]. In numerous studies, especially studies of inner ear development, various genes, transcription factors, secretion factors, growth factors, receptors, cell adhesion proteins and other molecules have been identified as responsible for ear malformations [13].

Congenital ear malformations with an obvious family history show autosomal dominant inheritance in about 9% of cases, autosomal recessive inheritance in about 90%, and X-linked inheritance in about 1% [5]. Non-syndromal congenital hearing impairment has a completely different distribution: autosomal dominant inheritance in approx. 30% of cases, autosomal recessive in about 70%, X-linked in about 2-3%, and sometimes mitochondrial-linked inheritance [14]. On the other hand, in patients with familial non-syndromal high grade microtia Katzbach et al. [15] reported a predominantly autosomal dominant inheritance with variable penetrance. Acquired ear malformations result from exogenous damage during pregnancy.

Many classifications of ear malformations have been proposed. These classifications should facilitate a standardised clinical description of findings and should serve as a prognostic basis for treatment interventions and their comparison. Over time, classification systems have become more detailed, especially due to modern imaging techniques such as CT and MRI.

The closely interrelated development of the external auditory canal and middle ear led to the classification of a combined malformation called atresia auris congenita according to Altmann [16]. Three degrees of severity have been described:

Type I: mild deformity of the external auditory canal, normal or slightly hypoplastic tympanic cavity, deformed auditory ossicles and well-ventilated mastoid process are noted;

II-type: these include blind termination or absence of the external auditory canal, narrow tympanic cavity, deformed and fixed ossicles, decreased pneumatisation of the mastoid cells;

III-type: absence of external auditory canal, middle ear is hypoplastic, ossicles are strongly deformed, besides, there is inhibition of pneumatisation of mastoid cells.

Classification R. Jahrsdoerfer (system J), which was proposed in 1992. [17]. The J-system consists of nine anatomical structures: the presence of the stapes, the anteromedial window, the cochlear window, the ventilated space of the middle ear, the presence of the malleus-anvil joint, the pneumatisation of the

mastoid process, the anvil-anvil joint, the location of the tympanal segment of the facial nerve and the normal appearance of the auricle. The stapes is awarded 2 points because it is considered the most important factor. The other eight anatomical components are scored 1 point each. A total score of J ≥6 indicates that the patient may be a candidate for canaloplasty [18].

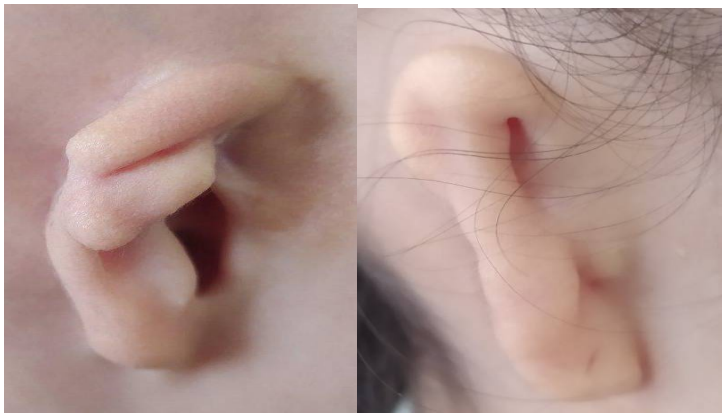
Congenital anomalies of the external and middle ear are rare causes of conductive hearing loss in children. Hearing loss in patients with malformations of the external auditory canal is conductive in nature, although a small proportion of patients will also have a neurosensory component. Conductive loss is usually in the 40-60 dB threshold, depending on ossicular deformity, ossicular mobility and degree of temporal bone pneumatisation. Hearing loss associated with these minor malformations, including congenital ankylosis of the striae, persistent strenal artery, malleolar fixation and absence of an oval window, can range from mild to severe, can be missed in newborn hearing screening and cannot be diagnosed until the child is able to undergo behavioural testing.

Objective: patients with malformation of the external and middle ear in children by optimising the diagnostic algorithm, developing and introducing new methods of surgical treatment of this pathology.

Materials and methods. In the Children's National Medical Centre 34 patients with congenital atresia of the external auditory canal with combined middle ear pathology were performed primary surgeries by anterior access.

The patients were classified according to the Altman morphological classification, and the prognosis of hearing improvement before surgery was predicted according to the Jahrsdoerfe classification.

Patients (34 patients) ranged in age from 6 to 17 years. Boys- 20, girls- 14. In 14 cases atresia of the external auditory canal with combined middle ear pathology was unilateral, in the rest there was bilateral ear pathology. In 25 patients, in addition to the absence of the external auditory canal, there was microtia of the 3rd degree (Fig.1, b), in 4 patients microtia of the 2nd degree (Fig.1, a), in 4 patients the auricle was developed with normal size, in 1 patient the auricle was completely absent.



a b

Figure 1. Types of microtia: a) Grade 2 microtia; grade 3 microtia

Hearing tests in 32 (94.1%) patients revealed conductive hearing loss with a CVI of 35 dB, 2 patients had mixed type hearing loss with a CVI of 30 dB (5.9%). There were no cases of facial nerve damage or sensorineural hearing loss. Facial nerve monitoring was used. In both groups, hearing results were achieved with intact patient ossicles or prostheses.

A postauricular incision was made. The subcutaneous tissue is elevated anterior to the temporomandibular joint. Caution is necessary, as the anomalous facial nerve may exit the temporal bone in this area. T-shaped periosteal incisions are made and the mastoid cortex is exposed. If there is a remnant of the tympanic bone, a new ear canal is started above it, at the level of the middle fossa of the dura mater. If there is no such remnant, drilling is started at the level of the temporal line, immediately behind the articular fossa. Then the mastoid process site was marked and, taking into account the data obtained by computed tomography, the bony part of the external auditory canal and tympanic cavity were formed with a cutting burr. The middle fossa of the dura mater is identified and examined. In all cases, the facial nerve is continuously monitored using the Medtronic NIM-2.

The posterior epitympanum is opened and a fused anvil/hammer mass is identified. The anvil and malleus are carefully separated from the atresia plate. The horizontal portion of the facial nerve is always located medial to these structures and is therefore relatively protected from the bony canal of the facial nerve.

The plate of bony atresia is removed with diamond microbores and curettes to completely expose the ossicles. Care should be taken when dissecting the inferior and posterior sides, as the aberrant facial nerve often passes laterally through the atresia plate in this area. We found an anomaly in the structure of the ossicular apparatus in all cases. The ossicular chain is preserved if it is normally developed. Identification of the stapes may be difficult due to overhang or abnormal facial nerve anatomy. Rudimentary auditory ossicles were removed and, depending on the preservation of the stapes, ossicular reconstruction was performed with an autochondral or titanium implant (TORP or PORP).

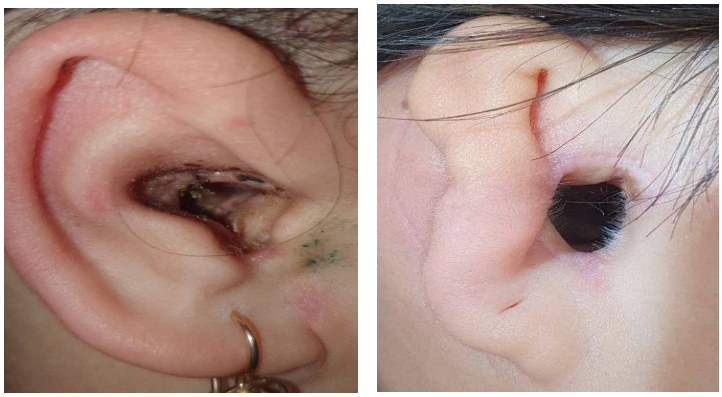
Exposure of the mastoid air cells is avoided as much as possible. A new ear canal approximately 1 ½ times larger than the normal one is created. As a neotympanal flap, a fragment of the temporalis muscle fascia is used; a 20 x 15 mm oval is cut out. Small "tongues" measuring 3 x 6 mm are cut in the anterior and upper parts of the graft. The neotympanal membrane is placed together with the fascia to either the lateral surface of the intact auditory ossicle chain, a partial ossicle prosthesis (PORP), or a total ossicle prosthesis (TORP) covered with an autochartilage overlay. "Overlays" are placed medially in the protimpanum and epitympanum. A 0.5 mm thick split skin graft.

6 x 6 cm, taken from the abdomen or thigh. One edge of the graft is zig-zag cut approximately 1.5 cm deep. The new ear canal is lined circumferentially with split skin with zigzags completely overlapping the reconstructed tympanic membrane. The entire bony portion of the new external auditory canal should be closed. There should be 2-3 cm of excess skin on the side to even out the soft tissue meatusplasty. One layer of antibiotic-impregnated Gelfoam is used to hold the skin and fascia of the new tympanic membrane in place. A 1mm diameter disc of silicone strip is placed on top of this. A myrocele swab soaked in petroleum jelly is then placed in the canal lateral to the silicone strip. The centre is filled with small pieces of gauze soaked in petroleum jelly. Meatoplasty is then performed and the skin, subcutaneous cartilage, and tissue are removed in an area 2 cm in diameter over the new external auditory canal. The ear is inverted and the canal skin is withdrawn through the meatoplasty. The skin graft is attached circumferentially to the skin of the ear canal with non-absorbable sutures (Prolene-6). The soft tissues of the meatus should be completely lined. After that, the behind-the-ear incision is closed.

In the postoperative period, the sutures were removed on the 9th-10th day. Tampons from the external auditory canal were removed on 20-21 days.

Results. Anomalies of the structure of the ossicular apparatus were found in all cases during surgical intervention. Only in 4 cases a free malleus and anvil, a mobile stirrup were found. In these cases ossiculoplasty was not performed. In 9 cases there was a synastosis of the malleus and incus, 14 cases there was a synastosis of all auditory ossicles into a single block, all patients underwent ossiculoplasty with a partial titanium prosthesis, in 4 cases an anomaly of the stapes (congenital ankylosis) was detected and stapedoplasty of autochryasis on autonadchryasis was performed.

As a result of using the described technique, stable results of the external auditory canal tube formation in the long-term follow-up were obtained. Thus, no stenosis of the external auditory canal was observed in all patients 8 months after the operation. In addition, the value of the bone-air interval in the postoperative period averaged 10-15 dB, which should be regarded as a good functional result.



а б

Figure 2: Postoperative results: a) after 4 months; b) after 8 months.

Conclusion. Careful soft tissue technique with split grafts covering the entire exposed bone is the key to prevent stenosis. Proper orientation and soft tissue technique are key to successful correction of congenital atreticity of the auditory canal.

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