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CONGENITAL DEVELOPMENTAL MALFORMATIONS OF THE HEARING ORGAN (REVIEW)

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SUMMARY

The paper provides an overview of scientific sources relating to congenital malformations of the external ear, including changes in the size, position, structure of the auricle, etc.

Purpose of the study *the mechanism of formation of malformations of the external ear, their classification and methods of diagnosis and treatment.*

Material and methods. *The work uses bibliosystematic and analytical methods for searching and analyzing scientific information obtained from available scientific publications.*

Results. *Congenital malformations can be complex and varied , and considerable attention has been paid to their unification. The article describes typical defects of the tubercles, combined defects, their classification by severity and location. A wide range of clinical and instrumental research methods have been developed that are used to diagnose hearing defects, as well as methods for treating ear defects.*

Conclusions. *Malformations of the hearing organ are a complex pathology that requires careful attention and highly qualified doctors. Treatment of patients with this pathology is complex, multi-stage and expensive. Implant prosthetics can provide patients with full-fledged hearing.*

KEY WORDS: *developmental defects; outer ear; middle ear; curls; classification; prosthetics.*

INTRODUCTION

According to WHO, about 15% of children are born with developmental abnormalities. Congenital in development are persistent disorders of structure, function or metabolism that arise in utero as a result of disturbances in the development of the embryo, fetus or, sometimes, after the birth of a child - as a result of a violation of the further formation of organs. Given the complex embryological development.

Since the ear, these defects can affect its individual parts or occur in various combinations. Defects of the external ear include changes in the size, position, structure of the auricle, annotation , anteriorization of the auricle, parotid enlargements , auricular sinuses and fistulas. The external auditory canal (EA) can be aplastic (atretic) or hypoplastic.

The outer ear is formed in the 5th week of embryonic development . According to Helms (1994), defects of the outer ear often appear without defects of the middle or inner ear, and vice versa. This is explained by the different development of embryogenesis [9]. Ishimoto et al . (2005) believed that with certain defects of the auricle, 6–33% have additional defects of the ossicles, 6–15% have defects of the round and oval window, 15% have pneumatization of the mastoid process, 36% have a malformation of the facial nerve and 42% have shortcomings of the sulfur chloride [10]. The inner ear is formed at 4 weeks of intrauterine development. According to Marangos (2002), defects of the oval and, less commonly , round windows may occur [30]. Most often this occurs due to arrest or disruption of embryonic development. Aplasia, hypoplasia and labial defects occur - r int and sensory pathways. The aqueduct of the vestibule may be narrow or widened (Sennaroglu ,Saatci , 2002)[40]. The internal auditory canal may have defects, in particular, arteries and nerves (especially the facial nerve) may be displaced (Swartz , Faerber , 1985) [45].A combined ear defect known as congenital auricular atresia (malformations of the outer and middle ear with atresia of the external ear) occurs in 1:10,000–1:15,000 newborns (Ishimoto et al ., 2005; Klaiber , Weerda , 20; in 15–20% of cases, bilateral defects are observed [10].

Epidemiology

According to Thorn (1994), the frequency of ear defects is 1:3800 among newborn defects [47]. The incidence of malformations of the external ear is 1:6000 (Brent , 1999), severe malformations occur in 1:10,000–1:20,000 newborns (Weerda , 2004), very severe malformations or aplasia - in 1:17,500 newborns . The prevalence of microtia is 3:10,000 (Schloss , 1997) [39].

In 58–61% of cases, defects of the outer and middle ear concern the right side, of which 70–90% are unilateral (Weerda , 2004; Swartz , Faerber , 1985; Thorn , 1994) [52]. Defects can also be bilateral (Marangos , 2002; Sennarogl) [30, 40].

Etiology. Approximately 30% of birth defects are associated with syndromes that include other defects or loss of function of an organ or organ system. In particular, the following genetic syndromes are known, in which congenital ear defects are observed: otofascial dysostosis (Thriller- Collins syndrome , Goldenhar syndrome), craniofacial dysostosis (Crouzon syndrome , Apert syndrome), otopervical dysostosis (Klipp syndrome), otoskeletal dysostosis (Van der Hoewe D e Klein syndrome, Albers -Schoenberg syndrome), chromosomal syndromes (trisomy 13 chromosomes - Patau syndrome ,

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trisomy 18 chromosomes - Edwards syndrome, trisomy ; 21 chromosomes - Down syndrome, 18q syndrome).
There are non-syndromic ear defects. These include only ear anomalies without any defects. Jahrsdoerfer Kim (2004) suggests a high probability of the frequency of spontaneous gene mutations in genetically determined defects (syndromic and non-syndromic) [16]. Autosomal recessive type of inheritance in 90% and an X-linked type of inheritance in 1% (Thorn , 1994) [47].
Acquired defects arise due to the negative influence of exogenous factors during pregnancy: infections (Coxsackie virus , rubella, cytomegalovirus , ECHO virus, etc.), radiation, chemicals (quinines, cytostatics , antibiotics), factor incompatibility , hypoxia (Thorn , 1994)[47]. According to Katzbach et al . (2006), the risk factor is bleeding in the first trimester of pregnancy and metabolic disorders (for example, diabetes mellitus (DM)) [20].

There are several options for classifying ear defects:
– classification according to Weerda (2004) – includes deficiencies of the auricle and the auricle [39];
– classification according to Altmann (1955) – includes congenital malformations of the inner ear [2] ;), Jackler et al . (1987), Jackler , De La Cruz (1989) – include defects of the inner ear [30, 40,13].

Disadvantages of the auricle include, in particular, ear fistulas and ear cysts. Most often, cysts and fistulas are first discovered when inflammation occurs . Upper cervical fistulas and ear fistulas are rootstocks of the CSC due to changes in the first branchial cleft. Fistulas and cysts are lined by squamous epithelium , and their most common location is preauricular and the area around the helix of the auricle (Weerda , 2004) [39].

The following types of hands and fistulas are distinguished:
Type I is a “doubling” of the SSC, lined with skin, running parallel to it and ending blindly above or lateral to the facial nerve. Most often found in the area behind the ear.
Type II are true duplications of the SSC, lined by skin and often containing cartilage. Such cysts and fistulas can :
– blindly end in the transition area between the cartilaginous and bone parts of the SSC;
– opens in the area of the anterior part of the musculus sternocleidomastoideus .
Figure 1 depicts the embryological periods of development of the auricle and the reflection of its tubercles (Weerda , 2004) [39].
As the degree of dysplasia increases, the severity of the defects increases (Weerda , 2004) [39].
There are several classifications of ZSP flaws. By Weerda et al ., 2004 [39] distinguish:
– stenosis of the spinal cord type A – noticeable narrowing of the spinal cord along the entire length;
– stenosis of the spinal cord type B – characterized by the temporary development of the spinal cord with complete atresia in the medial part;
– type C stenosis includes complete bony atresia of the spinal cord .

Due to the close interconnected development of the CSC and the middle ear, combined defects called congenital atresia can occur. Altmann (1955) identified them in a separate classification [2]. Three degrees of severity are described (Fig. 2):

Defects of the 1st degree - mild deformation, small deformities of the spinal cord, normal or somewhat hypoplastic tympanic cavity, deformed auditory ossicles and well- pneumatized soscope and - body process.

Defects of the II degree – moderate deformation; blind ending of the third or absent third, narrow tympanic cavity, deformation and fixation of the auditory ossicles, decreased pneumatization of the mastoid cells.

Defects of the 3rd degree – severe deformations; The SSC is absent, the middle ear is hypoplastic, and the auditory tassels are significantly deformed; complete absence of pneumatization of the mastoid process.
Middle ear defects can be isolated or combined.

Kosling et al . (1997), Müller (1991) distinguish three degrees of severity of isolated middle ear defects:
Light defects – normal configuration of the tympanic cavity + dysplasia of the auditory ossicles. Moderate defects - hypoplasia of the tympanic cavity.

us + vestigial or aplastic auditory tassels .
Severe defects – aplastic or slit-like tympanic cavity [26, 31].

In 10–47% of cases, severe middle ear defects (sometimes with CSC defects) can be combined with inner ear defects, especially in combination with microtia (Swartz , Faerber , 1985; Ishimoto et al ., 2005) [45], [10].

Following the Kösling classification et al ., 1997) isolated defects of the auditory ossicles are classified as a mild group and are described as “minor” defects of the middle ear [31].

To diagnose hearing defects, a wide range of clinical and instrumental research methods are used .

Audi Iometry is the most important functional study for patients with ear defects. Physiological tests for children under 3 years of age include tympanometry , otoacoustic emissions (OAE), and auditory evoked potentials. For older children, traditional total or behavioral audiometry is used. In young children and in patients with multiple defects, a repeat study should be carried out for accuracy (Weerda , 2004; Klaiber , Weerda , 2002; Katzbach et al ., 2006)[52, 24, 20].

Genetic testing is recommended for patients with an autosomal recessive or X-linked recessive disease (heterozygous testing) (Lehnhardt , Koch , 1994; Katzbach et al ., 2006; Aretz et al ., 2006) 3].

Clinical examinations (newborns with ear deformation should undergo examination of craniofacial structures. A thorough examination of the skull , face, neck is performed regarding

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facial proportions, configuration, symmetry, occlusion, masticatory apparatus, swallowing, sensory functions, speech, voice). Perform a thorough examination of the functions of the middle ear. In addition to examining the ears, attention is paid to anatomical features that may jeopardize the successful operation (adenoids, severe curvature of the nasal septum, the presence of a cleft palate, etc.).

According to Schüller , Stenvers and Maier , traditional radiography is of little value in the diagnosis of ear defects. High-resolution computed tomography (HRCT), with its clear imaging of bony structures, is useful in depicting changes in the outer ear, OH, middle ear, and mastoid . anatomical considerations when planning cochlear implantation (CI) (Weerda , 2004; Kösling et al ., 1997; Siegert et al ., 1996; Greess et al ., 2002) [52, 26, 42, 7].

Magnetic resonance imaging (MRI) is suitable for imaging the membranous labyrinth, the neural structures of the internal auditory canal, and the pontine angle. MRI is the only method for demonstrating senohelical non - r- VA simultaneously with the assessment of intracranial segments of the facial nerve.

There are certain signs of ear defects that can be detected on a CT scan . For example, impaired aeration of the tympanic cavity, defects of the ossicles, deformation of the auditory ossicles, caudal-stapedal joints, hypoplasia of the tympanic cavity, stenosis/occlusion of the oval and round window, incomplete development of the helix (normally 2.5 turns) , dysplasia of the vestibule, abnormal course of large vessels base of skull and facial nerve To assess the possibility of performing reconstructive surgery on the middle ear in a patient, a middle ear CT scale was developed (Table 2). Patients with unilateral congenital atresia and a score of 8 or more are considered suitable candidates for surgery; those with bilateral ear involvement and a score of 5 or less are not considered for surgery (Jahrsdoerfer et al ., 1992) [16].

Treatment of ear defects includes combined reconstruction of congenital auricular atresia and severe microtia . For example, for patients with grade III microtia and congenital ear atresia, an operation is performed that includes extraction of autogenous cartilage, production and implantation of an auricle frame. The eardrum and SSC are prepared in advance and stored in a subcutaneous pocket. During the next stage, a new frame is lifted, which is combined with an operation for atresia, using the tympanic membrane and 3D. At the third stage, the cavity of the concha is deepened, the SSC is opened and closed with a skin graft. When auricular plastic surgery is combined with functional middle ear surgery, no additional operations are required . Prefabrication of the TSC and tympanic membrane (creating them from custom-made tissues for use in reconstructive surgery) produces reliable and desirable results.

To restore and improve hearing function, implant prosthetic systems are used: bone conduction implants (BAHA and Bonebridge ; BB) and active middle ear implants (Vibrant Soundbridge ; VSB), stimulating wax cells of the cochlea and CI, stimulating nerve structures. All implantable hearing aid systems provide the desired results for patients with hearing loss.

CONCLUSIONS

1. Malformations of the hearing organ are a complex pathology that requires close attention and highly qualified physicians.
2. Treatment of such patients is complex, unpredictable and expensive.
3. Thanks to the possibility of implantation hearing prosthetics , it is possible to provide patients with full-fledged hearing, which contributes to their integration into society.

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