Ministry of Education and Science of Ukraine
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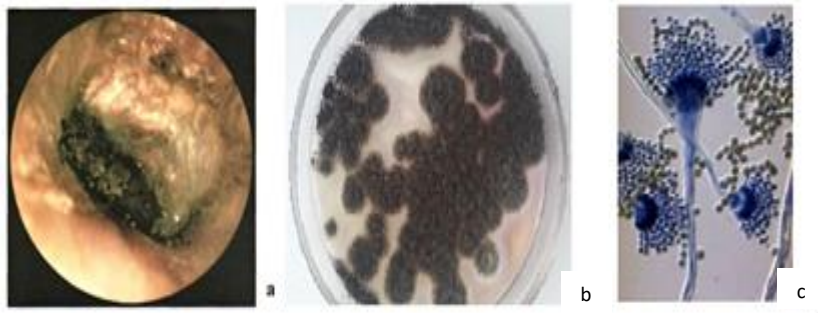
INFLAMMATORY AND NON-INFLAMMATORY DISEASES OF THE OUTER EAR

Study guide

Under the general editorship of MD, professor V. A. Smiyanov



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I-60 **Inflammatory** and non-inflammatory diseases of the outer ear : study guide / V. A. Smiyanov, I. O. Plakhtienko, T. V. Ivakhnyuk, E. V. Smiyanov ; under the general edition V. A. Smiyanov. – Sumy : Sumy State University, 2023. – 120 p.

The study guide is aimed at developing basic knowledge on the prevention, diagnosis and treatment of patients with non-inflammatory and inflammatory diseases of the outer ear. In accordance with the requirements of evidence-based medicine, the treatment of various nosological diseases of the outer ear is summarized, taking into account the microbiological features of the pathogenesis of diseases and personalized results of sensitivity of infectious agents to antibacterial drugs.

Designed for medical students of higher education institutions, interns, otolaryngologists and general practitioners (family medicine).

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CONTENTS

DISEASES OF THE EXTERNAL EAR

Nowadays, the problems of prevention and treatment of patients with inflammatory diseases of the external ear, particularly auricle, external auditory meatus and tympanic membrane epidermal layer, are undoubtedly very actual for practical otolaryngology. Currently, inflammatory diseases of the external ear are very wide-spread among patients of different age groups. Thus, for example, acute external otitis can appear at any age, but the top of the disease arises among children and teens (5-14 y. o.).

According to WHO and some scientists' researches results the frequency of external otitis is 17–30 % of all outer ear inflammatory diseases. It is established that one in ten people have had an inflammatory disease of the external ear at least one time throughout life, and up to 5 % of the population suffers from chronic external otitis. It should be mentioned that the incidence of inflammatory diseases of the external ear is more likely to happen among old age and is due to high-humidity conditions of the environment, heat and swimming.

Referring to statistics in Ukraine, there is over 5 % of inflammatory diseases of the external ear among hospital pathologies of the ear, nose and throat, including acute diffuse otitis externa which amounts 75 %, a furuncle of the external auditory meatus – over 16 %, an abscess of the earlobe – 6 %, external ear eczema is examined among 1.3 % of patients, almost 1.5 % of patients suffer from otomycosis. Taking into account that diseases of the auricle can lead to cosmetic defects and don't have a negative impact on hearing impairment, so in most cases outer ear inflammatory diseases cause a disability of spreading sounds and, as a result, a significantly decreased audibility which leads to the low standards of patients' lives.

The present unfortunate situation in cities, unsustainable abuse of antibiotics, anatomic narrowing of the

external auditory meatus, the use of hearing aids, incorrect technique of the ears hygiene cleaning (including the use of q-tips), leads to the appearing of micro-traumas on the skin of the external auditory meatus, changing in the composition and pH of the earwax to alkaline, stressors, general immunosuppression due to certain diseases (diabetes, HIV, atopic dermatitis, psoriasis, etc.), getting water in the ears – all these and another factors can cause an external otitis – an inflammation of the external auditory meatus caused by different etiology.

It should be mentioned that outer ear inflammatory diseases are characterized by the diversity of clinical symptoms, high probability of recurring according to the predisposing factors, also such diseases require doctors' professionalism in diagnosis and treatment. It is very important to otolaryngologist to use clinical data to make a differential diagnosis of external otitis with another inflammatory diseases of the ear (acute medium otitis, mastoiditis, etc.), parotid gland, inflammatory diseases of the teeth and jaws, because a diagnostic mistake or an inadequate treatment can be a causative factor of different serious complications, which are very dangerous for people's lives.

CLINICAL ANATOMY OF THE EXTERNAL EAR

External ear (auris externa) consists of the auricle (concha auriculae) and the external ear canal (meatus acusticus externus) (Figure.1).

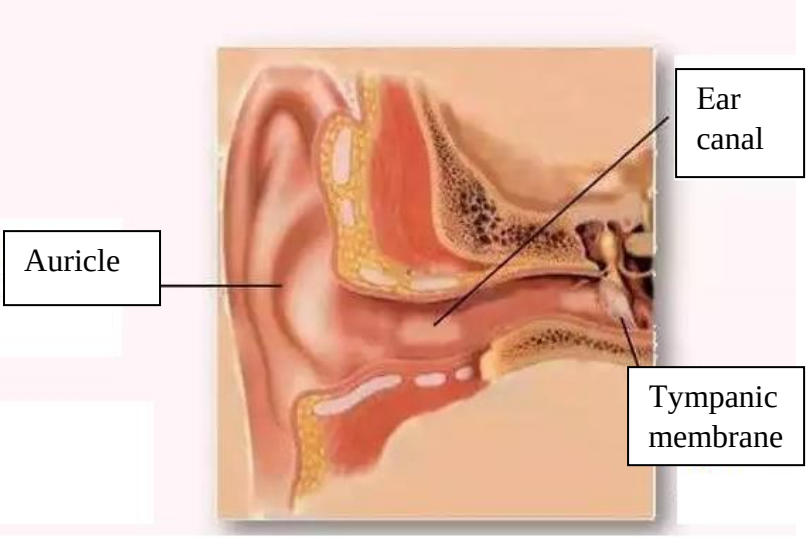

Fig. 1. The structure of the outer ear.

An auricle is located between a temporomandibular joint in front and mastoid process behind and is composed of elastic cartilage 0.5–1 mm thick, perichondrium and is covered with a thin skin layer. There are a hollow external and a convex internal (turned towards a mastoid process) surfaces. An auricle parts are:

1) helix, which encloses an auricle outer edge;

2) antihelix, which is located as a bump to the middle of the helix;

3) scapha – the longitudinal groove between the helix and the antihelix;

4) tragus – a protrusion, anterior to the auditory meatus;

5) antitragus – a protrusion, posterior to the auditory meatus;

6) earlobe (lobulus auriculae), which is composed of adipose tissue covered with skin (Figure 2).

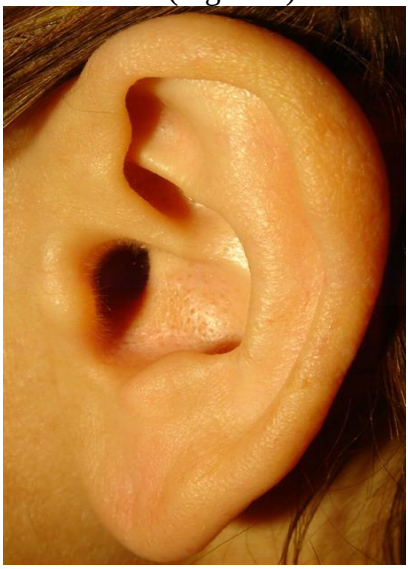

Fig. 2. Lobe of the ear.

Talking about the auricle anatomical peculiarities among children, it should be mentioned that it is very soft, inelastic and with contours that are poorly characterized among babies, helix and earlobe are residually formed to the end of the 4^{th} year. At the time of the birth it was round, with almost the same height and width, the growth is very fast, especially during the 1^{st} year. The growth of the auricle is completely over to 15 years.

These peculiarities should be taken into account while determining the indications for cosmetic operations among children.

The auricle, forming a funnel-shaped narrowing, turns into the external auditory canal.

An external auditory meatus is an S-shaped curved canal up 2.5 sm long and 0.7–0.9 sm in diameter among adults. The canal has got 2 parts: cartilaginous external (2/3 of the canal length) and internal bony part (1/3 of the canal length), which is located in the temporal bone. The cartilaginous and the bony parts of the external auditory canal are connected due to the strong ligament composed of connective tissue. The place of transition of the membranous-cartilaginous part to the bony part – is isthmus – the narrowest part of the auditory canal. The cartilage of the passage forms a groove, supplemented at the top by fibrous connective tissue, so the external auditory canal is able to expand in the cartilaginous part with the introduction of a funnel during otoscopy. Due to the fact that the cartilaginous and bony parts of the external auditory canal form a certain angle, to straighten it during otoscopy in young children it is necessary to pull the auricle back and down, and in older children and adults – back and up.

The external auditory canal is covered with skin with numerous hair follicles, sebaceous and sweat glands that secrete earwax. Sulfur consists of sebaceous matter, pigment and contains cells of the corneal epithelium. The bony part of the skin is thin (up to 0.1 mm) and contains no glands or hair.

The external auditory canal has got 4 walls:

1. The anterior wall of the auditory canal borders the mandibular joint. Therefore, in the presence of inflammation of this wall of the outer ear, chewing movements lead to sharp pain.

2. The lower wall separates the ear canal from the parotid salivary gland, which can cause the spread of infection from the outer ear to this gland and vice versa.

3. The posterior wall of the external auditory canal is the anterior wall of the mastoid process and can often be involved in the inflammatory process in mastoiditis.

4. The upper wall separates the outer ear from the middle cranial fossa, so fractures of the skull can lead to the cerebrospinal fluid or blood showing up from the ear [10].

The anatomy and topography of the structures of the outer and middle ear, as well as the mastoid process of the newborn, infants and young children have their specific features. The external auditory canal of newborns and infants is poorly developed: it is short and narrow, the inner bony part is represented only with the tympanic ring (annulus tympanicus). This should be taken into account while interpreting the results of a tragus symptom – the appearance of pain while pressing on the tragus indicates an inflammation of the middle ear (if a child is less than six months) and the pathology of the outer ear in elder children. The external auditory canal is a slit filled with primary oil (vemix caseosa), which consists of fat with a small admixture of skin epidermis. Taking into account these features, otoscopy in newborns is much more complicated.

The external auditory canal in children, and in adults, has 4 walls. However, some anatomical features of a structure are noted. Thus, the **front** wall is formed of a drum ring, the **lower** – a derivative of the drum ring. Through the slits in the lower wall of the external auditory canal (Santorini's) it can break through abscesses of the parapharyngeal space. In infants, when the mastoid process is not developed yet, the lower wall is attached to the cartilaginous styloid process, which lies almost horizontally and is located near the descending part of the facial nerve, which makes it easy to cause paresis while an inflammation of the external auditory canal occurs. The **posterior** wall is formed partly of the tympanic ring and partly from the scales of the temporal bone. The **upper** wall occurs from the scales of the tympanic bone.

The external auditory canal of a child under 1st year is almost devoid of the bony part, so the pressure on the tragus is

easily transmitted to the walls of the tympanic cavity. It should also be noted that in infants, the joint of the lower jaw is almost close to the external auditory canal. This fact, as well as the softness of the walls explains the change in its width during sucking and chewing. There is the parotid salivary gland near this area, which in some cases leads to the breakthrough of its abscess into the external auditory canal.

The structure of the external auditory canal of a 3–4-year-old child is close to its structure among adults.

The blood supply of the outer ear is from the external carotid artery system: anterior to the superficial temporal artery (a. Temporalis superfacialis), posterior to the posterior auricle (a. Auricularis posterior) and occipital (a. Occipitalis). In the wall of the external auditory canal a deep auricular artery (a. Auricularis profunda) branches from the internal maxillary artery. The same artery is involved in the blood supply of the eardrum.

Venous blood from the outer ear through the same-named veins flows into the venous plexus behind the joint of the mandible, into the external jugular vein. Lymphatic outflow is carried out in the lymph nodes located in front of the tragus, on the mastoid process and under the lower wall of the external auditory canal. Later, the lymph flows into the deep cervical lymph nodes.

An innervation of the external auditory canal is provided by:

– auricular-temporal nerve (n. auriculotemporalis) from the branch of the mandibular nerve (n. mandibularis), which is the 3rd branch of the trigeminal nerve (n. trigemini);

– auricular branch of the vagus nerve (r.auricularis n. vagi);

– large auricular nerve (n. auricularis magnus) branch of the cervical plexus.

All the nerves mentioned above provide a sensitive innervation. It should be noted that the motor innervation for the rudimentary muscles of the auricle is carried out by the posterior auricular nerve (n. Auricularis posterior) from the facial nerve (n. Facialis).

The auditory canal in children and adults ends with a thin translucent plate – the eardrum, which separates the outer and middle ear.

NON-INFLAMMATORY DISORDERS OF THE EXTERNAL EAR

Cerumen impaction of the external auditory meatus.

An excessive sulfur secretion or sulfur plugs occur among 1 in 10 children, 1 in 20 adults, and more than a third part of geriatric patients and people with developmental delays.

A sulfur plug is a cluster of earwax that blocks the external auditory canal. Thus, an ear sulfur is a mixture of secretory secretions of glands and keratinized epithelial particles of the external auditory meatus. Sulfur masses consist of fats, proteins, fatty acids, keratinized epithelium, sweat, hair; antibacterial activity is detected due to the presence of lysozyme and immunoglobulins; may contain dust, foreign bodies, etc. As a rule, the earwax is removed due to the self-cleaning mechanism, moving from the ear canal while the movement of the jaws. Anatomical narrowing of the canal, the use of q-tips, the use of hearing aids, frequent inflammation of the outer or middle ear, periodic ingress of water into the ear canal, comorbidities (psoriasis, diabetes, etc.) – all these factors cause the occurrence of sulfur plugs.

For a long time, the sulfur plug may not cause discomfort to the patient and be asymptomatic until the external auditory canal is not blocked by sulfur masses by more than 70 %, or there is no pressure of the plug on the eardrum. Hearing loss and a feeling of ear congestion are the leading complaints among patients with a sulfur plug. Usually, hearing impairment is caused by water which has entered the ear, as a result, the sulfur plug swells and completely blocks the ear canal. In addition to hearing loss, patients are disturbed by tinnitus, autophony (a patient hears his own voice while his ear is popped); in severe cases, dizziness, migraine, nausea and even heart pain may occur. A sulfur plug can also cause an

earpain. It can occur due to an increase in pressure inside the channel to the site of clot formation.

Usually ***the diagnosis of sulfur plug*** is not difficult. While making a diagnosis, the doctor is guided by the patient's complaints and examination. In the otoscopic picture, the doctor observes the presence of obstruction of the canal with sulfur masses from yellow to brown (Figure 3) of soft, dense or stony consistency. It is necessary to pay attention to a full or partial occupation of the ear canal.

Fig**. 3.** Otoscopic picture of obstruction of the external auditory canal by sulfur masses.

The results of tuning fork examinations with the presence of a sulfur plug in the canal will differ from the normal ones:

– Weber's experiment (W). Normally, the subject hears a sound in the middle of the head or equally in both ears. At unilateral disorder of the sound-spreading structures (a sulfur plug in an auditory canal, a foreign body, an inflammation of a middle ear, perforation of a tympanic membrane) a

lateralization of a sound to a sick ear is observed; with bilateral lesions – towards the ear, which hears worse.

– Rinne's experiment (R) – a comparison of the duration of perception of bone and air conduction. The low-frequency sounding tuning fork (for example, C 128) is installed with a foot on the mastoid process of the examined ear. After the cessation of sound perception on the bone, it is raised by branches to the external auditory canal. Normally, a person hears the tuning fork in the air longer (Rinne's experiment is positive). With impaired sound perception, bone and air conduction deteriorate proportionally, so Rinne's experience remains positive. If the sound conduction suffers, the sound on the bone is perceived longer than in the air (negative Rinne experiment (R-)).

– Schwabach's experiment (Sch) – the measuring of the duration of sound perception through the bone. The sounding tuning fork is placed on the mastoid process of the subject until the patient stops hearing it. Then the researcher (with normal hearing) puts a tuning fork on the top of the mastoid process. If he continues to hear the tuning fork, then the subject's experience of Schwabach is shortened, if he does not hear, the experience of Schwabach is normal. You can compare the duration of sound perception in the subject with the passport data of the tuning fork. Also try it on the other ear. In the presence of a sulfur plug in the external auditory canal, there is an extension of the Schwabach experience. If there are diseases of the sound-spreading structures, on the contrary, its shortening.

– Federici's experiment (F) – a comparison of the duration of perception of bony tissue conduction from the mastoid process and conduction from the tragus. The experiment is similar to the experience of Rinne: after the cessation of the sound of the tuning fork at the top of the mastoid process, it’s put with a foot on the tragus. Normally,

Federici's experience is positive when sound perception is disturbed, so the sound of a tuning fork from the tragus is perceived longer, and when sound conduction is disturbed (including the presence of a sulfur plug in the canal) – it's negative (prolongation of a sound absorption from the top of the mastoid process).

– Experiment Bing (B) – a comparison of the intensity of perception bony tissue conduction from the mastoid process with an open ear canal and closed by pressing the helix to the ear. Normally, with good mobility of the chain of auditory ossicles, turning off the air conduction (with closed auditory canal) prolongs the perception through the bone. If the sound conduction is disturbed, the bone conduction will remain the same while the ear canal will be either opened or closed.

At audiometric research a hearing impairment by type of sound conduction in a sick ear is observed.

Treatment. It is not necessary to try to remove a sulfur plug independently, especially with sharp objects as in this case the ear canal is often injured. In addition, this way you can push the plug deeper into the ear canal, which will complicate its removal.

The most effective treatment is a lavage. If it is impossible to use it, you have to remove the plug from the ear in a dry way.

The rules for washing:

1. The studying of the patient's anamnesis before the procedure. Contraindications to rinsing the sulfur plug are the presence of perforations in the eardrum, chronic otitis media or dizziness of unknown origin. The fluid can get into the middle ear, provoke inflammation or irritate the horizontal semicircular canal, which will increase dizziness.

2. Flushing is indicated in cases when the sulfur plug has not completely blocked the ear canal.

3. Water of a comfortable temperature close to 37°C is used for the procedure.

4. Before removing the solid plugs, they must first be softened. To do this, appoint instillation of special drops (3 % hydrogen peroxide, A-cerumen, devaxyl) into the affected ear three times a day for 2–3 days before the procedure to dissolve the clot. It should be noted that after instillation may be an increase of hearing loss due to swelling of the sulfur plug. This reaction is normal and should not be a reason to worry about.

Washing process consists of the following stages:

1. Pour warm water into the Jeanne syringe or needle-free syringe with a 100–150 ml rubber tip.

2. The stream of water should be directed up and back, along the wall of the ear canal. It is necessary to pull the auricle in the same direction. It should be dragged back and down among children. The patient's head should be tilted to the side opposite the patient's ear. After some time, the patient changes the position of the head. The liquid is poured out, carrying a sulfur plug.

3. Residual moisture must be removed with cotton swabs or a probe with cotton wool wound on its end.

A sulfur plug removal in a dry way. You can clean the ear in a dry way using two main methods: aspiration and curettage. The action of the aspirator is based on creating a pressure difference, as a result of which the plug is "sucked" out of the ear. One of the side effects of the procedure is a violation of the vestibular apparatus. Curettage can be performed under local anesthesia. A probe with a hook is inserted into the patient's ear, if it’s necessary, puncture, and then remove the plug. Control is recommended to perform with otoscopy, otherwise there is a high risk of injury.

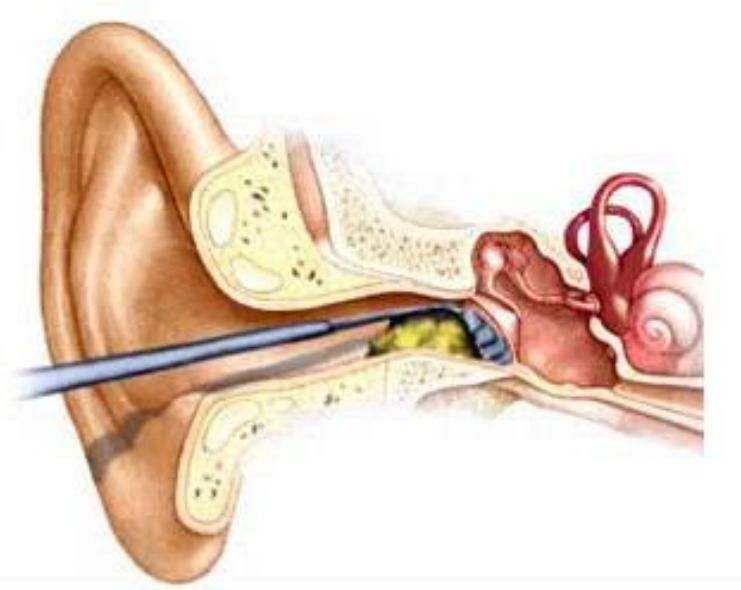

Fig. 4. Instrumental removal of sulfur plug: curettage.

Foreign bodies occlusion of the external auditory meatus

Foreign bodies occlusion of the external auditory meatus occurs in up to 4 % of all urgent visits to the otolaryngologist. Foreign bodies are more often diagnosed among children (buttons, small toys, peas, plasticine, etc.). However, among the adult population it is also a common pathology (insects, headphones, cotton wool, matches, etc.).

Complaints depend on the type of a foreign body, the degree of overlap of the ear canal and the presence or absence of trauma of the skin of the external auditory canal. Depending on this, there may be a hearing loss, a feeling of congestion and distension in the ear. Pain occurs when the skin of the external auditory canal or eardrum is injured, if the foreign body has sharp protrusions, or while joining a further inflammatory process, if the foreign body is not removed in time. Bloody discharges occur for the same reason as pain.

If an alive object enters the ear as a foreign body, it often moves, so it provokes additional unpleasant sensations – such as itching, tickling, tinnitus, dizziness, reflex vomiting when the eardrum is irritated or an insect enters the cavity of the middle ear.

To ***diagnose*** a foreign body in the ear, otoscopy is performed (figure 5). At the same time the foreign body and changes (inflammatory or necrotic) in soft tissues of an auditory canal and a tympanic membrane are visualized. If the patient consults a doctor after the foreign body enters the ear, pathological changes (tissue swelling, inflammation) may develop, which do not allow a simple examination of the external auditory canal.

Fig. 5. Otoscopy – diagnosis of a foreign body in the ear.

At accession of an inflammation and existence of allocations from an ear at a foreign body of an auditory canal it is necessary to appoint ***bacterioscopic*** (under a microscope – for detection and identification of the activator) and ***bacteriological*** (carry out cultivation of allocations from an

ear, expect the growth of the colonies to identify the causative agent of the inflammation while injuring ear tissues by a foreign body) ***research.***

When interpreting the results of the bacteriological or cultural method of examination of the material from the external auditory canal, it is necessary to take into account the species and quantitative spectrum of the selected microbiota. A characteristic feature of the external auditory canal is the presence of various microorganisms (sometimes even pathogenic, in the form of transient microflora). According to clinical studies, 90 % of healthy people in the microflora of the external auditory canal are dominated by gram-positive bacteria that are commensals of this biolocus*: Streptococcus spp., Corynebacterium auris, Turicella otitidis, Staphylococcus spp., Alloiococcus*. Given the fact that these bacteria are pathobionts (opportunistic pathogens), they can activate their pathogenic properties, in case of violation of protective barriers, the presence of sulfur cork or foreign body, pH of the channel, high locus humidity, diabetes and other conditions. associated with immunodeficiencies, which leads to a violation of the barrier function of the main structural components of the external auditory canal, including the quantitative and qualitative composition of the normoflora. Thus, as part of the normal microflora of the external auditory canal, according to studies, can be isolated *Micrococcus spp.,* but with injuries of the skin of the auditory canal, high humidity and temperature, these microorganisms can cause inflammation. According to other studies, the most common representatives of the normoflora of the external auditory canal (up to 63 %) are staphylococci *(e. g. Staphylococcus auricularis and Staphylococcus epidermidis)*, less common *Pseudomonas aeruginosa.*

The clinical and epidemiological significance of Pseudomonas aeruginosa (Pseudomonas aeruginosa) is

determined by the sum of its ecological and pathogenic properties – ubiquity and unpretentiousness, resistance to antibacterial disinfectants, antiseptics and antibacterial drugs, pyogenic substances and products. However, *Pseudomonas aeruginosa*, as a typical opportunist, whose disease is realized only in the presence of immunodeficiency, even at the local level, to implement the invasion and stabilization of the infection requires violation of the skin and mucous membranes, reduced colonization resistance (e. g. foreign body, sulfur crust, trophic disorders); infection with large infectious doses, under conditions of exogenous exposure. It should be noted that the resistance and unpretentiousness of Pseudomonas aeruginosa to food determine the probability of almost universal spread in the hospital environment, creating ample opportunities for the formation of "hospital" strains. Almost the only condition for its survival is sufficient humidity.

Given that the safest method of removing uncomplicated foreign bodies of the external auditory canal is rinsing them with warm water using a Jeanne syringe, it should be noted that *Pseudomonas aeruginosa* can reproduce in a wide range of temperatures, and some strains can reproduce at a temperature of 4 ^{0}C, creating a dangerous contamination of the auditory canal with non-sterile water or instruments "hospital" strains. Pathogens belonging to such ecogroups are formed from out-of-hospital strains under the influence of many factors of the hospital environment and are extremely dangerous due to their high pathogenicity and virulence, multiresistance to antibacterial agents, insensitivity to antiseptics and resistance to non-specific protective conditions. great growth opportunities. One of the leading pathogens of hospital ecovars is *Pseudomonas aeruginosa*.

According to the results of other studies, gram-negative bacteria were more often isolated than gram-positive in the microflora of the external auditory canal, which confirms the

individual qualitative and quantitative nature of the species spectrum of such microflora.

That is why, when joining the inflammatory process in the presence of a foreign body in the ear canal, it is advisable to isolate, identify and determine the degree of contamination with microorganisms from this locus (quantitative and qualitative analysis of the microbiota); obtain susceptibility profiles to antimicrobials to each bacterial or fungal isolate. For this purpose it is necessary to take material from external auditory passages.

Treatment. After clear defining the size, shape and nature of the foreign body, the presence or absence of complications, you should choose the method of its removal. Treatment of this pathology is based on two principles:

1. removal of a foreign body from the ear and

2. elimination of the consequences caused by a foreign object.

Removal of a foreign body can be performed by rinsing, ear hook and in complicated cases – by surgery.

The safest method of removing uncomplicated foreign bodies from the external auditory canal is to rinse them with warm water using a Jeanne syringe, which is performed in the same way as removing a sulfur plug (see above). Flushing is contraindicated in the presence of: batteries, flat or thin foreign objects (for example, pins, needles, etc.) – they can be carried by a stream of water into the external auditory canal and cause perforation of the eardrum.

Extraction of a foreign object with an ear hook is carried out in a following way: the hook is got behind a foreign object and pulled out with push-like movements from an external auditory canal. If foreign bodies with hygroscopic properties should be removed, 96 % ethyl alcohol should be instilled into the ear before the procedure – it has got dehydrating properties, and the foreign body decreases in size,

which leads to its easier removal. Alive insects in the ear canal should be pre-killed by instilling 70 % ethyl alcohol or liquid oil in the ear, then removed by rinsing or by hooking.

If there is pain, then the removal of the foreign body is performed under local anesthesia. After the procedure, an examination of the external auditory canal and eardrum is performed to determine inflammatory changes and tissues defects.

Surgery is performed under local anesthesia if the foreign object cannot be removed from the ear by other means. An incision is made in the soft tissue behind the auricle, exposing the posterior wall of the bony part of the external auditory canal, and removing the foreign body.

A foreign body in the ear can cause ***complications*** such as: otitis externa; perforation of the tympanic membrane by a foreign body; acute otitis media - an inflammatory lesion of the structures of the middle ear, which occurs when a foreign object has damaged the integrity of the eardrum, and if pathogenic microorganisms penetrate the middle ear cavity from the external environment; abscess of the external auditory canal - occurs due to a soft tissue defect caused by a foreign body, purulent bacteria occupy tissues; Phlegmone of the exernal auditory meatus - diffuse purulent soft tissue lesions.

Oto-hematoma

Oto-hematoma – a limited accumulation of blood between the cartilage and the, perichondrium caused by injuries, prolonged compression or spontaneously, without violating the integrity of the auricle.

Otohematoma is a relatively common disease in otolaryngology. Approximately 75–80 % of all cases of this pathological condition are otohematomas of traumatic origin. The disease is caused by sports traumas and occurs in professional wrestlers, boxers, etc. The main cause of

spontaneous hematomas of this localization is hemophilia and various forms of leukemia. In about a quarter of cases, septic complications develop, about 85 % of such complications occur because of the hemorrhage with a statute of limitations of more than 3 days.

Damage to the blood and lymphatic vessels of the auricle is the main etiological factor in the development of hemorrhage. This pathology can occur as a result of a local trauma of the outer ear and current systemic diseases. As a result, it is common to distinguish two forms of otohematoma: post-traumatic and spontaneous.

Pathology occurs in almost all age groups. It is more often diagnosed among young active people in the age group of 30-40 years. The second peak is observed among elder people after 55 years of age, which is associated with the formation and increase of age-related degenerative (destructive) changes in the cartilage of the auricle. It should be noted that men suffer more often than women due to sports activities. Particular attention should be paid to the occurrence of spontaneous otohematoma among infants, as this may indicate the presence of concomitant pathological conditions.

Post-traumatic form of otohematoma is the most common and occurs as a result of a blow to the auricle or its fracture in domestic, sports or industrial injuries. Among old people this disease can occur due to prolonged compression of the auricle (for example, while sleeping).

A slight effect on the auricle leads to a spontaneous form of otohematoma in concomitant pathological conditions. Blood coagulation disorders in hemophilia, antiphospholipid syndrome, oncohematological diseases (hemophilia and various forms of leukemia), hemorrhagic diathesis, thrombocytopenic conditions, avitaminosis, alimentary dystrophy, some infectious diseases (hemorrhagic fevers,

Marine hemorrhoids, Ebola) are the causative factors of this form of otohematoma.

Clinical picture. Otohematoma occurs in the upper part of the outer surface of the auricle. In the initial stages, there is the formation of swelling in the upper part of the auricle between the helix and the scapha. Minor hemorrhages may occur in the area of the triangular fossa. Large otohematomas can be located between the helix and the tragus or the antitragus.

Visually, the otohematoma has got a typical appearance: a round or oval fluctuating protrusion of a roundish bluish hue with redness of the skin over it (Figure 6). With the accumulation of lymph in the cavity of the otogematoma, the color of the skin over it may not be changed. It should be noted that the earlobe in this pathology is intact.

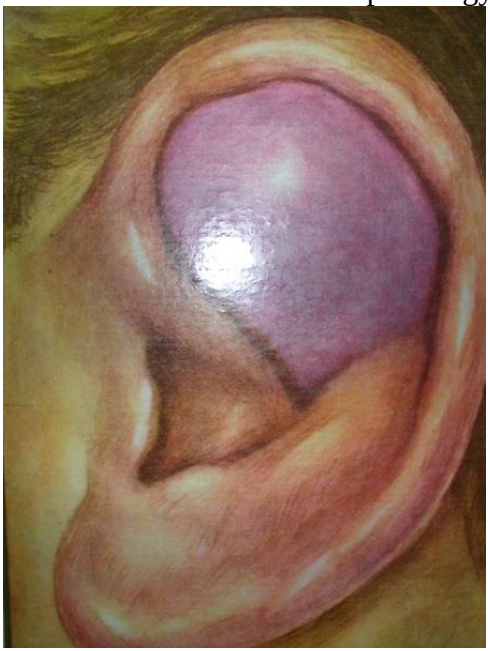

Fig. 6. A typical picture of otohematoma.

Otohematomas of traumatic etiology are not accompanied by pain, feeling of expansion and heaviness that are s not observed in a spontaneous form of this pathology.

In most cases, palpation is painless. With the development of perichondritis of the auricle, there is severe pain.

Small otohematomas can resorb spontaneously, but large ones, if there is the absence of rational treatment, turn into dense scar tissue, which leads to deformation of the auricle. The peculiarity of this pathological condition is that otohematomas often recur due to damage of lymphatic vessels and local coagulation disorders. In these cases, the lymphatic component predominates in the cavity. Also due to the superficial localization such hematomas have a high risk of penetration of bacterial flora and suppuration.

In most cases, the diagnosis of "otohematoma" does not cause difficulties in the examination, collection of complaints and anamnesis. For a complete examination, appoint a general blood test, coagulogram and determine

Treatment. As it was already mentioned, small otohematomas are capable of self-resorption. They should be treated with an alcoholic solution of iodine or diamond green. Topically, you can apply cold to the ear, but heat on the ear in this case is contraindicated!

Large otohematomas in the first 2–3 days of the disease can be punctured with the subsequent application of a pressure bandage during three days. A roller gauze is placed under the auricle, and gauze balls are applied to the area of the otohematoma cavity to increase the pressure on the ear. Then apply a general bandage to the ear.

In addition, large hematomas can be removed by dissection with cleaning of the cavity, which should be drained with rubber strips. Topically prescribe compresses with hypertonic sodium chloride solution, antiseptic or antibacterial

drugs. In this case, local treatment should be supplemented with the appointment of nonsteroidal anti-inflammatory drugs and broad-spectrum antibiotics (beta-lactam antibiotics of the penicillin series, cephalosporins of the II–III generations, fluoroquinolones).

INFLAMMATORY DISEASES OF THE OUTER EAR

Inflammation of the external auditory canal can be bacterial (diffuse otitis externa; limited otitis externa, or furunculosis) and fungal (otomycosis). According to the modern literature, from 60 to 98 % of cases of inflammatory diseases of the outer ear are caused by bacterial agents.

Auricular perichondritis

Auricular perichondritis – a diffuse inflammation of the cartilage of the auricle. The process can spread to the skin of the auricle and the cartilaginous part of the external auditory canal without a damage to the earlobe (Figure 7).

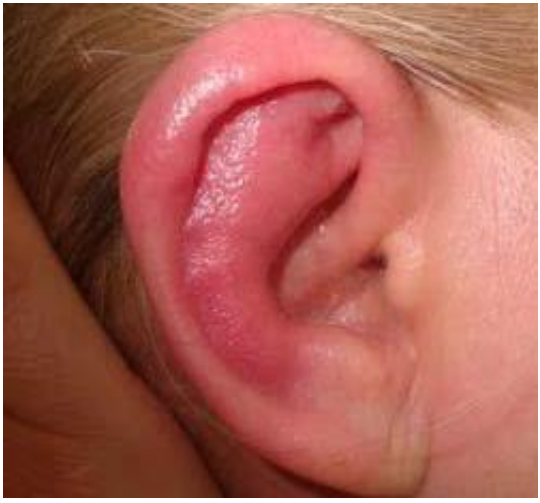

Fig. 7. Diffuse inflammation of the cartilage of the auricle – perichodritis.

The disease begins with serous inflammation, which can lead to the development of purulent inflammation if it was untimely and adequately treated. In some cases, under the condition of a long inflammatory process, the etiological agent

of which is a microorganism in a wide range of pathogenic factors, the inflammatory process can spread to cartilage, with its purulent melting and sequestration.

The etiological factors of perichondritis are mostly *Pseudomonas aeruginosa*, *Staphylococcus aureus*.

The infectious agent can enter the cartilage from trauma to the auricle with damage to the skin and epithelium (for example, an ear piercing with cartilage puncture), insect bites, burns or frostbite; systemic diseases (granulomatosis with recurrent perichondritis). Perichondritis can occur as a complication of an external auditory canal boil or diffuse otitis, influenza or tuberculosis.

There are **serous** and **purulent** types of **perichondritis.** The course of serous perichondritis is with less symptoms and severity than purulent.

Complaints and diagnosis. The first and main complaint in patients with this pathology is pain in the auricle or external auditory canal. And, as a result, a reactive infiltration and swelling of the skin of the entire auricle, smoothing contours, except the earlobe area. Initially, the swelling may be uneven and bumpy. Later, in purulent perichondritis, there is a fluctuation due to the formation of purulent exudate between the cartilage and perichondrium. During palpation the auricle is painful. The pain may radiate to the occipital, parietal, or cervical regions.

Non-specific complaints include signs of general intoxication, such as: fever, chills, sleep disturbances because of a pulsating severe pain in the outer ear, loss of appetite.

Diagnosis of this disease is based on the presence of complaints and objective examination of the patient. In the initial stages of the process, perichondritis should be differentiated from erysipelas and otohematoma.

Given that ear perichondritis more often has a bacterial etiology, there is a high risk of severe infection and cartilage

necrosis is high due to poor blood supply to the ear cartilage. Abscesses characterized by fluctuations in edema are a common complication and should be properly cut and drained, as any pressure on the cartilage can lead to its ischemic necrosis, which will lead to serious cosmetic deformities of the auricle.

If a clear infectious etiology is not detected (e. g. infected piercing), inflammatory diseases should be ruled out.

Differential diagnoses of perichondritis include superficial skin infections, shingles, insect bites, or allergic contact dermatitis, which, at first glance, can be dangerous to mislead the diagnosis of perichondritis. Perichondritis of the auricle should be differentiated from erythema of streptococcal etiology, purulent otogematoma, phlegmon.

Complications: deformities of the auricle, the so-called deformation in the form of "cauliflower" (Figure 8). Posttraumatic perichondritis usually progresses rapidly. Lack of treatment can lead to subperichondrial abscesses with ischemic cartilage necrosis.

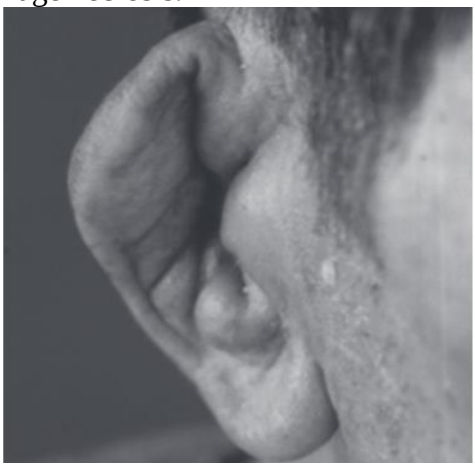

Fig. 8. Cauliflower ear: A cosmetic deformity of the auricle, known as the cauliflower ear, with little chance of satisfactory plastic reconstruction.

Other complications include hematoma or hypertrophic scarring and keloid formation. Toxic shock syndrome has also been described.

Clinical studies of patients with perichondritis show that one of the etiological factors of the bacterial etiology of this disease is ***Pseudomonas aeruginosa***.

That is why, when considering the features of the pathogenesis of perichondritis of this etiology, it is necessary to take into account aspects of clinical microbiology. P. aeruginosa are aerobic non-fermenting catalase and oxidase-positive gram-negative motile psychrophilic-mesophilic bacteria-prototrophs, which have a straight or slightly curved rod-shaped shape (Figure.9).

Pseudomonas aeruginosa is characterized by the universality of metabolic activity, using a wide range of substances as food sources - from simple carbohydrates and tissue substances of the human body to antimicrobial drugs (ethacridine lactate, detergents, furacillin and even formaldehyde), which provides its wide environmental plasticity.

Paradoxically, P. aeruginosa, being an obligate aerobic, can grow and reproduce in oxygen-free conditions, where nitrates can serve as the ultimate electron acceptor. It is the respiratory metabolism, in which Fe-cytochrome oxidase plays a key role, that determines the vital need for Pseudomonas aeruginosa in iron. Therefore, P. aeruginosa has several systems for capturing iron from the environment (siderophores), which include pioverdin, pseudobactin, pyochelin, salicylic acid.

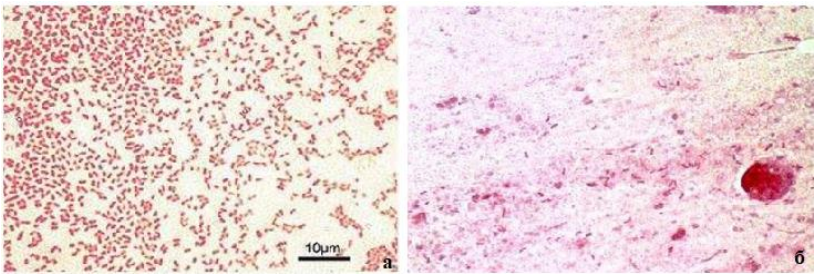

Fig**. 9.** *Pseudomonas aeruginosa*: a - pure culture of bacteria, Gram staining; b – In purulent separation, Gram staining.

Pseudomonas aeruginosa has a large set of so-called pathogenicity or virulence factors that cause tissue damage and ensure the survival of P. aeruginosa in the body. Pathogenic factors in Pseudomonas aeruginosa infection are active at all stages of the infectious process – adhesion, invasion, in the case of dissemination and persistence, as well as cause direct intoxication and provide "slippage" of the immune response.

Against the background of long-term perichondritis without treatment, Pseudomonas aeruginosa actively penetrates through tissue barriers. Mechanisms of invasion include processes aimed at destroying tissue barriers – cells and intercellular substance. The factors of invasion can be directly or indirectly enzymes, toxins of distant and contact types, endotoxin (lipopolysaccharide of the cell wall), apoptosis-inducing proteins, siderophores, secondary toxins of bacterial and tissue origin. The most important proteolytic enzymes of invasion include two variants of elastase – LasA and LasB, alkaline protease (AprA), protease IV (PrpL). All of them are characterized by activity against a wide range of substrates. Elastases destroy elastin, collagen and fibrin, causing destruction of connective tissue and breaking wound barriers; they can cause degradation of class G and A immunoglobulins, interferons. Alkaline protease is active against fibrin, complement system factors and in combination with elastase

destroys γ- and α-interferon molecules. Protease IV causes the destruction of elastin, complement factors, molecules of human immunoglobulin G and Fe-binding proteins – lactoferrin and transferrin.

P. aeruginosa has pathogenetically significant exoenzymes – lipase (LipA, LipB, LipC) and phospholipase C, which alone, and more often in synergism, exhibit hemolytic properties and can destabilize membranes of any cell type, causing severe necrotic changes in tissue.

Pseudomonas aeruginosa produces two different classes of exotoxins. The first class includes exotoxin A (ExoA), which is actively released by bacteria into the environment through the Xcp system by type II secretion. It acts not only locally but also at a distance, being transported through the blood, which enters from the infectious locus. It causes swelling of the skin and soft tissues, affecting vascular cells. The second class of exotoxins differs in the way they are released from the bacterial cell. If most enzymes leave the cytoplasm of Pseudomonas aeruginosa through the I and II secretion systems, the second class of exotoxins can be released using only type III secretion, which is called "macromolecular syringe". The name is due to the fact that the toxin secreted from type III, by means of a surface molecular complex is introduced directly into the cytoplasm of the cell, which is tightly adhered *Pseudomonas aeruginosa*. This damages a specific cell, but prevents the effect of "type III toxins" on other host cells that do not come into contact with the bacterium. Therefore, toxins released in this way are called "contact". In P. aeruginosa, 4 variants of contact toxins were found. ExoS and ExoT toxins are functionally similar: they have the properties of GTP-activating protein and ADP-ribosyltransferase, which synergistically cause rearrangement of actin in the cytoskeleton of the host cell, leading to its immediate death. The most dangerous for human cells is ExoU,

which acts as an intracellular phospholipase that causes rapid cell lysis. The mechanism of contact intoxication allows Pseudomonas aeruginosa to gain a significant advantage in the fight against the immune system, because contact toxins do not enter the extracellular space, and therefore can not be neutralized by antibodies.

Lipopolysaccharide (LPS, endotoxin) P. aeruginosa can have both a generalized effect (pyrogenicity and intoxication) and a direct local toxic effect. The pathogenic properties of LPS depend mainly on lipid A. Interaction with cells occurs due to the reception of LPS elements on molecular patterns specializing in the recognition of pathogens. The endotoxin reception of wild (exogenous) P. aeruginosa strains triggers a cascade of immune responses that suppress the pathogen. However, clinical strains are characterized by a modified structure of lipid A, which "distorts" the protective reactions of the macroorganism, which leads to bacterial survival and local tissue hyper-damage due to autoaggression of immune effectors.

Destruction of host tissues may be due to apoptosis induced by *P. aeruginosa.* A very interesting feature of the "pharmacokinetics" of pathogenic factors of P. aeruginosa is the presence of special systems that improve their transport in human tissues. P. aeruginosa is able to produce fairly stable in the aquatic environment vesicles with a diameter of 50 to 250 Nm, which may include LPS, phospholipase C, lipase, alkaline phosphatase. Vesicles provide a more effective interaction of pathogenic factors with human cells. *Pseudomonas aeruginosa* infection may be accompanied by local tissue self-destruction due to attack by the effectors of the host immune system due to an excessive inflammatory immune response. Tissue breakdown products that appear and are resorbed by the enzymes of P. aeruginosa invasion or by self-destruction also

contribute significantly to further damage, general intoxication, and fever.

Realization of pathogenetic potential of any pathogenic microbe, including pathobionts, in a human body is impossible without its counteraction to immune system of the owner. Pseudomonas aeruginosa, "obeying" this rule, uses numerous mechanisms of "escape" from immune effectors and even direct aggression against the immune system. Antiphagocytic properties are described in capsular polysaccharides, LPS, flagellin (a component of bacterial flagella), proteins of the Omp and Opr families, pigments, alginate. Protection against damage by oxygen radicals is due to pigments, oxidase, alginate. Invasion factors can affect immunocytes to the same extent as other cells. As already mentioned, the enzymes of invasion (elastase, alkaline protease, protease IV) provide the destruction of immunoglobulins, complement factors, γ- and α-interferons. Mucoid (alginate-forming) and biofilm-forming strains are especially protected from the immune system.

However, the most perfect and complex strategy to protect bacteria from immune attack is the formation of ***biofilms***. Given the possibility of a long course of perichondritis caused by *P. aeruginosa* and other pathologies, such as inflammatory processes on the background of a foreign body in the ear canal, it is necessary to understand the microbiological aspects of biofilm formation.

The biofilm contains two mandatory attributes: a cluster of cells and an extracellular (extracellular) matrix that binds them and localizes them to any part of the environment with different physicochemical properties. Depending on the characteristics of the strain and the parameters of the environment, Pseudomonas aeruginosa can form a flat (undifferentiated) or structured (differentiated) biofilm. The flat biofilm is a dense and relatively uniform layer of bonded bacteria. Differentiated biofilm is represented by clusters of

aggregated bacteria separated by water channels. The process of binding of bacteria to each other and to the biofilm matrix is mediated by surface adhesives – sawdust and surface proteins (Omp, Opr, LecA, LecB). In this regard, pharmaceutical adhesion blockade can be a promising way to prevent biofilm formation on the background of this pathology.

Therefore, the treatment of Pseudomonas aeruginosa infection is a rather difficult problem for a number of reasons. Tissue damage caused by P. aeruginosa has a complex mechanism and is caused by the production of cytotoxins, endotoxins, hemolysins and proteases. Preservation of viability and replication of pyocyanosis in human tissues and medical devices is facilitated by the presence of a biofilm that protects the microorganism from antibodies and phagocytes of the host and promotes the development of antibiotic resistance. In addition, Pseudomonas aeruginosa has fimbriae, which facilitate adhesion to the epithelial cells of the airways, which, in turn, causes a high frequency of colonization and is observed in hospitalized patients. Differences between P. aeruginosa strains, namely the presence or absence of a protective mucoid capsule and the ability of the pathogen to acquire antibiotic resistance during treatment, also complicate the choice of antibiotic.

The second most common causative agent of perichondritis is Staphylococcus aureus (Figure 10).

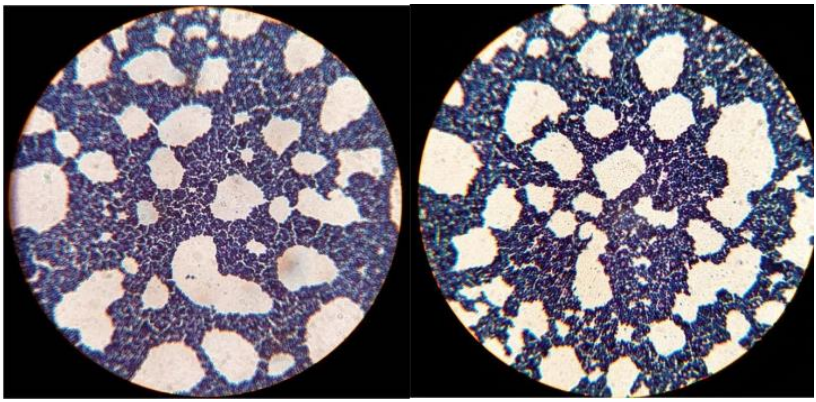

Fig**. 10.** Photomicrograph of S. aureus cells (Gram stain, Approx. × 10, Vol. × 100).

Staphylococci are pathobionts (opportunistic pathogens). These bacteria have a wide range of pathogenic factors and, therefore, under conditions of reduced general biological resistance of the macroorganism or under conditions of contact with sterile tissues, can cause the development of pathological processes. Infectious processes induced by ***opportunistic*** pathogens, including staphylococci, are called opportunistic. Inflammatory processes, which are mediated by staphylococci, are accompanied by the formation of pus, so these bacteria are still classified ***as purulent pyogenic*** cocci (this category also includes streptococci, gono cocci, meningococci). Among all types of staphylococci, S. aureus is characterized by the most pronounced pathogenic potential. Therefore, the identification of this microorganism and the determination of its pathogenic potential is one of the important tasks of laboratory diagnosis of infectious inflammatory processes.

The ability of staphylococci, like Pseudomonas aeruginosa, to mediate disease development depends on the ability of bacteria to "avoid" the immune response, produce surfactants that adhere to host tissues, and induce pathological

changes in host cells and tissues through the production of toxins or enzymes. All these properties are typical for most pathogenic bacteria.

Many surface antigens of protein origin are found in *S. aureus.* These proteins are important factors of pathogenicity, as they can perform functions that ensure the development of the pathological process. For example, the group of *S. aureus* surface proteins that mainly ensure the adhesion of the microorganism to the tissues of the host organism is called MSCRAMM (Microbial Surface Components Recognizing Adhesive Matrix Molecules). These proteins include fibronectin-binding proteins A and B, as well as staphylococcal protein A, which interacts with Fc fragments of IgG1, IgG2 and IgG4 immunoglobulins.

As it was mentioned above**, S. aureus** is a **pantropic microorganism**, so against the background of perichondritis caused by this pathogen, the position that is brilliantly reflected in L. Pasteur's paradox "Microbe – nothing, substrate (ie organism) – everything" is important. With this formula in mind (it is true for all opportunistic infections), we will, however, focus on microbial-dependent factors of staphylococcal invasion and this will be the basis for proper diagnosis and treatment of the inflammatory process of this etiology.

In pyogenic invasions, the central place belongs to neutrophils, although their reactions are only the culmination of events that initiate and develop the inflammatory process. Analysis of the properties of *S. aureus* shows that none of the species of opportunistic bacteria can compete with it in the number of factors with potential pathogenicity. It should be remembered that pathogenicity/virulence is a polyvalent trait that is really revealed only in the dialectic of the infectious process.

S. aureus most often causes abscesses of the skin, penetrating the skin and its appendages. For example, there are two properties that determine the ability of S. aureus to penetrate and persist in hair follicles, sebaceous and sweat glands. First, it has a powerful lipase activity, which allows it to destroy the sebaceous plug in the mouth of the hair follicle, while receiving food. Secondly, it is resistant to high concentrations of sodium chloride and fatty acids, which ensures survival in the secretions of sweat and sebaceous glands. By the way, fatty acids are formed during the breakdown of bacteria of neutral fats, cholesterol and other lipids, and it is possible that they enhance the inflammatory response to microbial invasion. Upon development, the reaction turns into a pathology – the focus of purulent inflammation.

In addition, *S. aureus* is able to synthesize other pathogenic enzymes that play an important role in the development of the infectious process, including the formation of complications of perichondritis: fibrinolysin (staphylokinase), which dissolves fibrin, which limits the local focus of inflammation, which can lead to generalization infections; staphylococcal hyaluronidase is an invading factor because it breaks down an element of the intercellular matrix – hyaluronic acid; Lecithinase disrupts the lecithin of the cell membranes of leukocytes (leading to leukopenia) and other cells of the host organism.

Exfoliative toxins are produced by those strains of staphylococci that induce pathological conditions that are accompanied by a violation of the integrity of the skin. The frequency of detection of such strains that produce exfoliatins is ~ 5 %. There are two types of exfoliative staphylococcal toxins: A (ETA) and B (ETV). ETA is thermostable and encoded by genes embedded in the bacteriophage, while ETV is thermolabile and encoded by plasmid genes. These toxins are

serine proteases that cleave desmoglein-1, a protein from the demosome structure that forms intercellular bridges in the epidermis. The syndrome of "scalded" skin of infants is a clinical manifestation of the activity of this toxin.

An important factor in the pathogenicity of staphylococci (especially in the formation of chronic forms of infection) is the ability to form a biofilm, which facilitates the adhesion and formation of colonies of microorganisms on mucous membranes, skin and wound surface. The biofilm is an exopolysaccharide matrix produced by staphylococci, within which the pathogen is very resistant to environmental factors such as immune protection factors or antibiotics. Diseases that are induced by biofilm-forming microorganisms are difficult to treat.

Given that the main causative agents of perichondritis are opportunistic bacteria (*P. aeruginosa, S. aureus*) that are able to form biofilms and mediate the occurrence of inflammatory infectious processes, and the appearance of biofilms often leads to complications and chronicity of infectious processes, as bacteria in their composition is characterized by increased resistance to environmental factors, and especially to antibiotics, the use of bacteriological diagnostic methods is appropriate and justified. This is primarily due to the fact that this method of diagnosis is a species identification of the pathogen of bacterial etiology and determination of its individual sensitivity to antibacterial drugs (Figure 11) according to the Order of the Ministry of Health of Ukraine № 167 from 05. 04. 2007 "On approval of guidelines for determining sensitivity of microorganisms to antibacterial drugs" (Figure 11). All this will allow for effective therapy of this pathology.

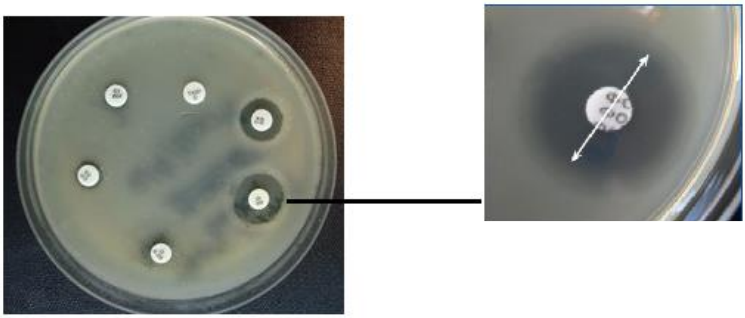

Fig. **11.** The result of the sensitivity of the observed culture of bacteria, seen in the patient, on the disc leaked with an antibiotic.

Differential diagnosis of infectious perichondritis.

Herpetic or vesicular otitis (zosteriform otitis, Hunt's syndrome), herpes zoster oticus (Figure 12) is caused by reactivation of endogenous latent chickenpox virus (VZV) in the knee ganglion with possible neurological and dermatological manifestations. Herpes zoster oticus is rare, but is a serious pathological complication. The virus passes through sensitive nerve fibers into the associated dermatome through the Vriesberg nerve gap, which innervates the apex, external auditory canal, soft palate, and anterior two-thirds of the tongue. The main clinical sign of the disease is considered to be pain in the area of the external auditory canal, eardrum, auricle, which simulates the manifestations of otitis. The process begins with a headache and pain in one ear, as well as herpes rash in the external auditory canal or on the skin of the auricle. These symptoms may be accompanied by facial nerve palsy, tinnitus, hearing loss and dizziness. The association of shingles with paresis of the peripheral facial nerve is defined as Ramsey Hunt syndrome (96 % of cases). Cranial nerves V,

VIII, IX and X can be involved. **Clinic:** tinnitus, hyperacusis and lacrimation.

Treatment: There are conflicting and limited data on the treatment of herpes zoster and Ramsey-Hunt syndrome. Hospitalization and combination intravenous therapy with acyclovir and corticosteroids are usually recommended.

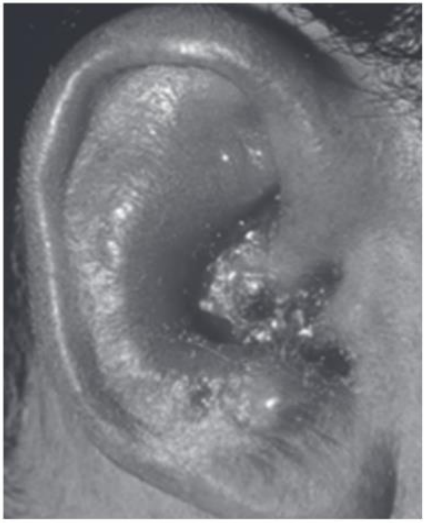

Fig. 12. Shingles of the right ear: reactivation of endogenous latent VZV within the patellar ganglion.

Recurrent polychondritis is a rare immune-mediated connective tissue disease characterized by recurrent inflammation of the cartilage that leads to its destruction. Cartilage type II collagen is thought to be a potential target antigen. Episodes vary in severity and frequency. If it affects the cartilage of the airways of the larynx, trachea and bronchi, it can lead to severe obstructive respiratory diseases with high mortality.

Clinical signs and symptoms of recurrent polychondritis: Ear infections are the most common symptom (up to 90 % of cases): unilateral or bilateral pain, soreness,

diffuse, violet and erythematous inflammation, sparing the earlobes with acute or subacute onset. No history of injuries. Episodes are recurrent and are often associated with ocular (scleritis, conjunctivitis, or uveitis) and nasal manifestations (saddle nose). Treatment of recurrent polychondritis: There is no evidence for the treatment of recurrent polychondritis. The approach is primarily empirical and aims to reduce systemic inflammation.

Treatment of perichondritis in the first days of the disease should begin with local and general antibacterial and anti-inflammatory therapy. Broad-spectrum antibiotics to which Pseudomonas aeruginosa is sensitive are used:

– amoxicillin clavulanate (augmentin, amoxiclav) 1.0 g 2 times a day for 7 days orally;

– macrolides (azithromycin 500 mg 1 time per day 3–5 days, clarithromycin 250 mg 2 times a day 7–10 days) as first-line drugs are considered in cases where there is evidence of the etiological role of atypical flora in the occurrence of perichondritis or there are contraindications for appointment of protected aminopenicillins;

– cephalosporins III – IV generation: cefixime 400 mg 1 time per day (or 200 mg 2 times a day) for 7 days orally, cefpodoxime 200 mg 2 times a day for 7 days orally, ceftriaxone 1.0 g 2 times a day intramuscularly 7 days; cefepime 1.0 g 2 times a day intramuscularly or intravenously for 7–10 days;

– fluoroquinolones (adults and children over 15 years) I – II generation: ciprofloxacin 500 mg 2 times a day for 7–10 days orally; levofloxacin 500 mg once daily for 7–10 days orally.

Topically prescribed compresses with hypertonic 10 % sodium chloride solution, 3–5 % potassium permanganate solution, polymyxin in the form of 1% ointment or emulsion

3–4 times a day. Physiotherapeutic methods of treatment are widely used: UFO, UHF, laser therapy.

On the 3rd – 4th day of the disease with the appearance of fluctuations, in addition to antibacterial and anti-inflammatory drugs, it is necessary to dissect subperichondral abscesses parallel to the contours of the auricle, with the selection of necrotized areas of tissue. The abscess should be drained and bandaged on the ear with chloramphenicol or hyperonic antibiotic solution 2–3 times a day.

With untimely treatment, the process can progress, which in turn will lead to purulent melting of cartilage with the rejection of necrotic tissue. The result is scarring, wrinkling and distortion of the auricle (the so-called "wrestler's ear").

Prevention is important to prevent perichondritis of the auricle. Namely – careful treatment of wounds and injuries of the outer ear, which directly serve as a gateway to infection, antiseptic solutions.

Clinical signs and symptoms of recurrent polychondritis: Injuries to the auricles are the most common sign (up to 90 % of cases): unilateral or bilateral pain, pain, diffuse, purple and erythematous inflammation, sparing the earlobes with acute or subacute onset. No history of injuries. Episodes recur and are often associated with ocular (scleritis, conjunctivitis, or uveitis) and nasal manifestations (saddle nose). ***Treatment of recurrent polychondritis***: There is no evidence for the treatment of recurrent polychondritis. The approach is primarily empirical and aims to reduce systemic inflammation.

External ear eczema

External ear eczema (Figure 13) – a chronic recurrent disease that occurs because of the irritation and subsequent infection of the skin of the auricle and external auditory canal.

This is one of the most common dermatological diseases of this localization. In general, this pathology accounts for about 35 % of all lesions of the skin of the ears,noses and throats. Its prevalence ranges from 2.5 to 6 cases per 1 000 population. The disease occurs in all age groups – from 4 months to 75 years with a peak incidence of 35–45 years. With the exception of seborrheic form, which is more common among men, this disease is diagnosed with equal frequency in both sexes. It should be noted that complications are relatively rare – no more than 15 % of cases

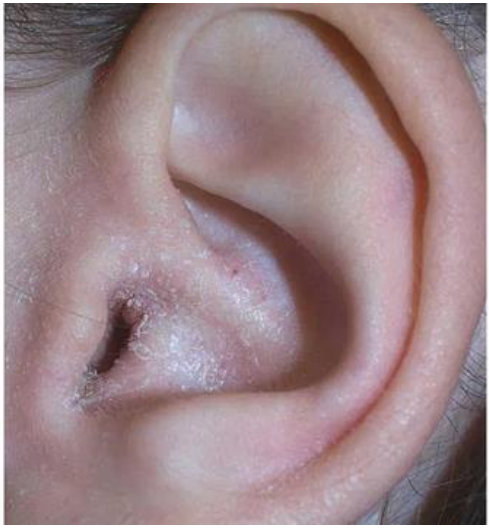

Fig. 13. Outer ear skin eczema (clinical manifestations)

According to the general classification based on clinical and pathogenetic features of the course of the disease, there are true (idiopathic, dyshidrotic, pruriginous, horny), microbial (numerical, paratraumatic, mycotic, intertriginous, varicose, sycosycosmic, zalosolossal, and eczema , seborrheic, pediatric and occupational varieties of eczema. Each of them can have an acute, subacute or chronic course with periodic exacerbations under the influence of trigger factors.

Given the multifactorial nature of the disease, the variability of the clinical course, currently accumulated a large amount of factual material and developed a general pathogenetic classification of eczema, according to which the leading pathogenetic factors are identified:

– hereditary predisposition. Complicated pregnancy, the presence of toxicosis and eating disorders, the presence of concomitant pathology in the mother often leads to the development of eczema in the child.

– dysfunction of the central nervous system. During the exacerbation of the pathological process was determined by a violation of the bioelectrical activity of the brain. Diagnosed conditionally pathological and pathological types of encephalograms.

– disorders of the autonomic nervous system. Changes in the functional state of the higher vegetative centers in eczematous lesions in the acute period lead to autonomic dystonia.

– imbalance of metabolism of neurotransmitters and biologically active substances. The most pronounced violation of neurotransmitters is determined in the elderly in comparison with the young.

– disorders of the cardiovascular system. At eczema the microvascular tone changes: vasoconstriction prevails, capillary perfusion decreases, arteriovenular discharge is activated, arterioles are in a spastic state, capillaries – in spastic-atonic, venules – atonic. In patients with eczema in combination with hypertension, a hyperkinetic type of blood circulation and a decrease in the elasticity of the vascular wall are registered.

– dysfunction of the endocrine glands. With the relatively recent development of acute inflammatory phenomena of the disease, the content of the main anti-inflammatory corticosteroid – hydrocortisone – in the blood

plasma and urine in most patients with eczema is increased. In the long-term eczematous process, which has a torpid course, as a rule, the amount of glucocorticoids in the blood and urine is reduced.

– dysfunction of the excretory organs. In eczema, renal dysfunction is secondary, due to allergic changes caused by eczema, or primary. In both the first and second cases, changes in kidney function and pathological processes in them will have a negative impact on the course of eczema. Pathology of the excretory function of the kidneys can be caused by changes in the organs in which metabolic substances are formed by the kidneys. According to various data, renal dysfunction is present in 46–70 % of patients with eczema.

– immune disorders. In patients with eczematous skin lesions, dysfunction of the humoral and cellular parts of the immune system, imbalance of various cytokines and circulatory immune complexes are determined.

– change in the metabolism of proteins, fats, carbohydrates, macro- and microelements, vitamins. There are deviations in the indicators of macro- and microelements: elevated levels of copper, zinc, sodium, potassium; reduced content of iron, manganese, aluminum, silicon, titanium. Indicators of vitamin status in eczema are dynamic: low content of biotin (vitamin B8), pyridoxine (vitamin B6), nicotinic acid (vitamin PP), ascorbic acid.

– dysfunction of the hepatobiliary system and digestive tract. Almost 100 % of patients suffering from eczema have various disorders of the digestive tract. More than half of the subjects have dyskinesia of the biliary tract, thirds – gastroduodenitis and pancreatitis, almost all patients with eczema during the examination show changes in the intestinal microbiocenosis.

Given all the above, when detecting eczema of the outer ear requires consultation with narrow specialists, determining the pathogenetic cause of the pathology.

It is believed that one of the causes of rashes on the background of eczema is a hypersensitive immune system, so eczema is an allergic reaction to hygiene products (scented soaps, perfumes, lotions), medications, common allergens (food, household, wool, etc.), some metals (nickel, silver), excessive moisture or dust. Sensitization of the body to various allergens contribute to chronic infectious skin lesions (microbial, fungal, viral) and chronic foci of infection of other organs (tonsillitis, etc.). Among the common diseases that lead to the development of eczema of the outer ear are diabetes, gout, and other forms of metabolic disorders. It should be noted that external exposure to the auricle (mechanical friction, hypothermia) or irritation of the external auditory canal by purulent discharge in acute or exacerbation of chronic purulent otitis media can also cause eczema.

The most common examples of misinterpretation of dermatoses of the skin of the outer ear are the clinical symptoms of lesions of the skin of the outer ear in the so-called eczema-like conditions. In fact, when it comes to diseases such as eczema, neurodermatitis, seborrhea, dermatitis, it is difficult to talk about any features of their clinical course on the skin of the outer ear. These diseases often have certain erythema-papulo-lichenification manifestations. On the other hand, the fairly generalized diagnostic concept of "eczema" of the outer ear is extremely free, and importantly, it is misinterpreted by both otolaryngologists and doctors of other specialties. Thus, eczema of the outer ear, in the nosological interpretation, occurs from 3.5 % to 16.6 % of cases per 1 000 patients with eczema [46].

Pathogenesis. In modern clinical immunology, eczematous processes are interpreted as an abnormal immune

reaction of type IV by Jail and Coombs, which leads to inflammatory changes, susceptibility to secondary infection. At the heart of this phenomenon are four main factors: immunodeficiency, hypersensitivity to certain substances, disorders of the central nervous system and hereditary predisposition. The existing insufficiency of the body's defenses is a dysfunction of prostaglandins and cyclic nucleotides, which leads to a decrease in the activity of neutrophils, T-helpers and non-specific protective factors, including phagocytosis. Nervous system disorders are accompanied by increased vascular permeability and hypersensitivity of smooth fibers to inflammation due to hypertension of the parasympathetic part of the peripheral nervous system and dysregulation of the hypothalamus.

Traumatic damage to regional nerve fibers also contributes to the development of this pathology. Against the background of infection in such conditions, the synthesis of autoantibodies that support chronic inflammation in the tissues of the ear [24].

There are main **forms** of eczema:

1. *True* eczema – begins acutely, there is a tendency to frequent recurrence and transition to a chronic form. This form is characterized by a variety of rash elements (vesicles, erosions, crusts, scales) on the hyperemic and swollen affected area of skin. Exfoliation is usually accompanied by severe itching.

2. *Occupational* form of eczema is the result of occupational allergic dermatitis.

3. *Microbial* eczema occurs as a consequence of secondary infection with pathogens (bacterial or fungal).

4. *Seborrheic* eczema – among patients with relevant pathology. Other affected areas of the body should be examined to make this diagnosis.

Eczema of the outer ear can be a primary or secondary process (when moving from the surrounding skin regions).

The most common complications of the eczematous process are the accession of secondary pyococcal *(Staphylococcus spp., Streptococcus spp., Micrococcus spp.)* And fungal *infections (Candida spp., Aspergillus spp., Penicillum spp.),* Which is associated with a decrease in antimicrobial skin surface. Treatment of infectious lesions is complicated due to the growing resistance of the main pathogens of pyoderma – Staphylococcus aureus and Staphylococcus epidermidis – to widely used antibiotics: penicillin and aminoglycosides (up to 75.8 % of strains), fluoroquinolones, 45.5) and tetracyclines (up to 40 %). Uncontrolled use of external antimicrobial drugs, the sensitivity to which is lost, delays the process of remediation of the infection and promotes the subsequent selection of resistant flora. The main principle of therapy of eczematous manifestations, taking into account the polyetiology of the disease, is a comprehensive impact on the body, taking into account the severity, nature, location of the pathological process, duration of the disease, previous treatment and its effectiveness, age and comorbidity.

Clinical picture. Clinically, there are acute and chronic, limited and diffuse, wet and dry forms of eczema.

The acute form of eczema is characterized by lesions mainly of the superficial layers of the skin, while the chronic form – a deeper lesion. In the acute course of the process, redness and infiltration of the skin entail thickening of the auricle and narrowing of the lumen of the external auditory canal. The patient complains of constant itching in the ears, but the pain is mild. There are small bubbles with serous contents, at their opening is an outflow of serous liquid outside. When the bubbles dry on the wetting surface, there is a formation of crusts (Figure 14), which can lead to the accumulation of these

crusts in the form of a plug. At accession of a secondary infection against eczema there is an occurring of limited or diffuse external otitis.

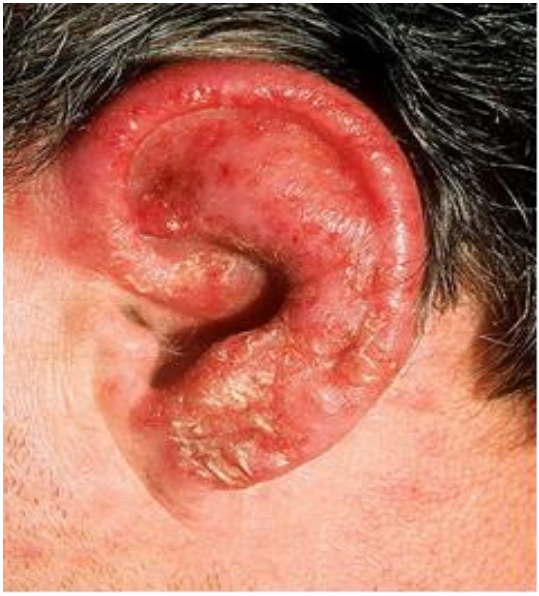

Fig. 14. Clinical picture of acute eczema.

Chronic eczematous process of the auricle develops in the presence of chronic diseases, prolonged and repeated exposure of irritants, untimely and irrational treatment of acute eczema. Itching and clinical manifestations in the chronic process are less pronounced, but there is a thickening of the auricles, cracks in the entrance to the external auditory canal. The course of this pathology is long.

The appropriate diagnosis is made in the presence of an appropriate clinical picture, patient complaints and anamnesis, involving a dermatologist and allergist in the process.

To confirm the allergic nature of the eczematous process there should be:

1) a carefully collected anamnesis (the presence of concomitant allergic reaction to medications, urticaria, burdened heredity);

2) the presence of eosinophilia in cytological swabs from the ear and in the general clinical analysis of blood;

3) skin tests to determine allergens.

It should be noted that among children there are eczema, diathesis, rickets, intestinal intoxication, itching, prolonged use of ointments and lotions, excessive wheezing of the child. In children, the wetting form of eczema predominates over dry form.

Differential diagnosis of the outer ear eczema is performed with erysipelas (Figure 15), limited or diffuse otitis externa, fungal infection. Erysipelas of the outer ear is characterized by painful palpation of the affected area, with a clearly defined limit of inflammation, which is not observed in eczema. The diagnosis of otomycosis is established by the results of bacteriological examination of secretions.

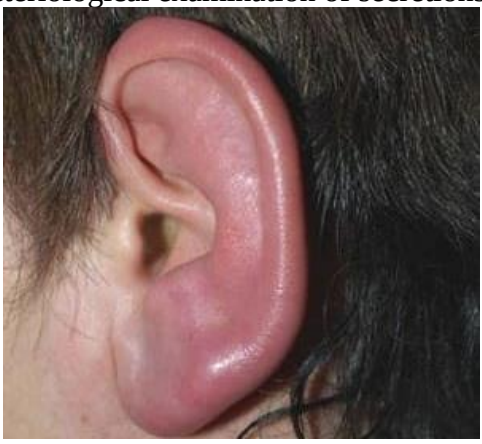

Fig. 15. Clinical picture of erysipelas.

The doctrine of erysipelas is at the junction of three medical specialties: otology, insectology, dermatology. This creates corresponding difficulties in diagnosis, rational

etiological and pathogenetic treatment, development of effective measures for prevention and rehabilitation of the relevant contingent of patients.

Erysipelas of auriculae is a fairly common and until recently life-threatening disease due to its progression and spread to the scalp, mucous membranes of the upper respiratory tract, eyes and others. In children, erysipelas is rare. One auricle is more often affected, less often both.

Rash can occur primarily on the auricle and spread to the face and head, or secondarily when the process passes from the skin of the face and head to the auricle. The primary erythema, spreading to the face and head, sometimes passes through the external auditory canal to the eardrum and causes it to pierce.

Eardrum erythema develops due to penetration into the skin of β-hemolytic streptococcus (*Streptococcus pyogenes*, Figure 16) in case of violation of their integrity, which may be with itching of the ear canal or skin irritation with exudate, when there is purulent otitis media, eczematous lesion of the external ear due to itching, trauma, boils and other diseases.

The most common cause of erysipelas is skin scratching in otitis externa, in which the pathogen can be directly applied with fingers, a match, a cosmetic stick, a hairpin, etc. These scratches are especially dangerous in the presence of pus from the middle ear.

The most common bacterial etiology of erysipelas is β-hemolytic streptococcus group A, although there are clinical observations of cases caused by other groups of β-hemolytic streptococci. Group G streptococcus can cause erysipelas, especially in patients 50 years and older. Erysipelas caused by group B streptococcus is more common in newborns, postpartum and hysteroscopic surgery. Group C and D streptococci rarely cause erysipelas. Non-streptococcal cases are often caused by Staphylococcus aureus. There is also

evidence of erysipelas-like disease in cases of septicemia caused by *Yersinia enterocolitica* and *Campylobacter jejuni*.

Rash occurs as a complication of frostbite of the auricle. An allergic factor (recurrent, habitual erysipelas) is often noted in the genesis of erysipelas.

Due to the fact that the main causative agent of erysipelas is ***Streptococcus pyogenes*** *(Figure 16)*, the pathogenesis of this disease is determined not only by the properties of the human body, but also by the pathogenic properties of this pathogen. Thus, the main factors of pathogenicity include the following:

– protein M – the main factor of pathogenicity of S. pyogenes, is a part of fimbriae. Protein M causes adhesion, inhibits phagocytosis, determines antigen type specificity, has superantigenic properties;

– the capsule suppresses phagocytosis. It contains hyaluronic acid, similar to that contained in the connective tissue of the human body, as a result of which phagocytes do not recognize capsular streptococci as foreign;

– erythrogenin – scarlet fever toxin. It acts as an allergen, causes a rise in body temperature, suppresses the immune system, destroys platelets, so in patients it causes a bright red rash on the skin and mucous membranes;

– streptolysins O and S (hemolysins) destroy erythrocytes, have a cytotoxic effect;

– streptokinase destroys fibrin, which increases the invasive properties of streptococcus. Purified streptokinase is used in medical practice for resorption of blood clots, fibrinous and purulent exudates;

– hyaluronidase – an invading factor that promotes the spread of bacteria through connective tissue;

– proteases destroy various proteins, including associated tissue toxicity;

– DNase causes DNA hydrolysis;

– allergens lead to sensitization of the body.

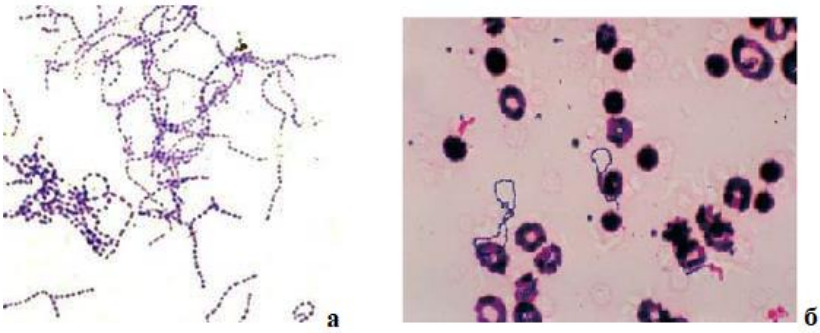

Fig. 16. *Streptococcus pyogenes:* a – pure culture of bacteria, Gram staining; b – *Streptococcus pyogenes* in purulent discharge, Gram stain.

The clinical course of erysipelas depends on the properties of the microorganism, the reactivity of the organism and the intensity of the inflammatory process. If the latter occupies the entire auricle, it increases in volume and becomes red and swollen. Incredibly swollen ears protrude forward. If the inflammatory process affects only a limited area of the auricle, then this place rises above the healthy skin, forming clear boundaries. Rash of the auricle is accompanied by severe pain and inflammation. The skin in the affected area is hot, swollen, hyperemic, shiny (Figure 17, a) and thickened. characteristic lingual protrusions with a sharp separation from healthy areas is almost non-existent. The bullous form of erysipelas is accompanied by the appearance of blisters on the skin with serous content, which is formed as a result of exfoliation of the epidermis by exudate (Figure 17, b). There is a significant violation of the general condition, fever up to 38 – 40^0C, chills. The course of erysipelas depends on the age of the patient. In newborns and infants there is a severe course, in school-age children - easier**.**

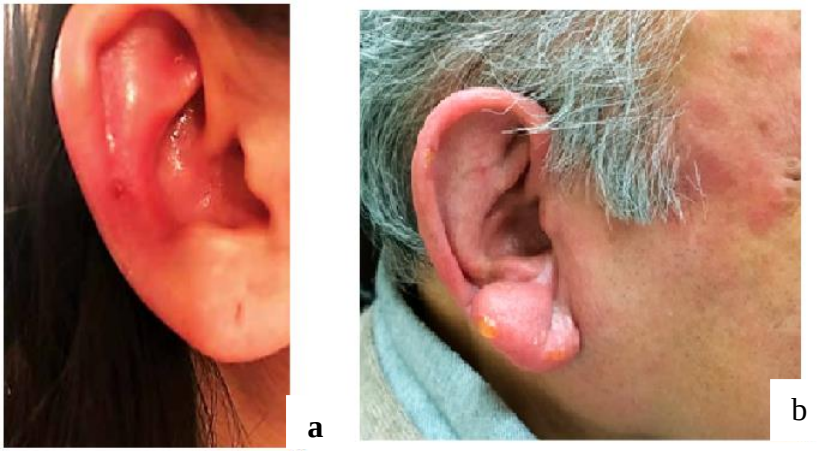

Fig. 17. Clinical symptoms on the background of erysipelas erysipelas: a – the skin in the affected area is swollen, hyperemic, shiny; b – bullous form of erysipelas, accompanied by the appearance of blisters on the skin.

Complications of erysipelas include the development of acute otitis media, so-called erythema otitis media. The described (Betzold, 1960) fatal consequence as a result of development of meningitis at distribution of a primary erythema erythema on a scalp is not observed now.

In typical cases, the ***diagnosis of erysipelas*** is not difficult and is based on the appearance of fever and a sharp rise in body temperature in the presence of severe redness with clear boundaries, swelling, hyperesthesia of the skin. Absence of redness (in sharply weakened patients) or its masking by ointments (ichthyol) considerably complicate diagnosis. In these cases, the presence of severe intoxication, fever and detection on the periphery of the lesion of the raised inflammatory roll, allows you to correctly diagnose erysipelas.

Differential diagnosis of auricle erythema. Reduction of the inflammatory process of the skin of the auricle for 2–3 days when prescribed in the first days of the disease antibiotics

and erythema doh ultraviolet radiation, as well as a history of erythema with other diseases of the auricle – with perichondritis of the shell, erythema, dermatitis.

In the case of perichondritis of the auricle, the lobe is not involved in the process. In contrast to erythema or dermatitis, erysipelas is characterized by: sudden onset with chills and fever, simultaneous development of frostbitten redness and swelling of the skin, a clear roll on the honey of healthy skin, especially on the periphery of the affected area, which does not happen with erythema and dermatitis.

Sharp redness and swelling of the mammary gland can be considered as a sign of mastoiditis. Differential diagnosis between erythema and mastoiditis in the absence of changes in the tympanic membrane, in erysipelas inflammation of the eardrum, is usually intact. In the case of purulent otitis media, the differential diagnosis can be made only after observing the development of the clinical picture of the disease for 2–3 days. Further spread of redness and swelling of the mammary gland indicates the erysipelas. If erysipelas spreads to the middle ear and there is acute purulent inflammation of this ear, it is easy to confuse erythema of the papilla with mastoiditis. Accurate diagnosis is vital. Rash is a bacterial infection that spreads throughout the lymphatic system and is associated with intense and well-defined erythema. An infection after a piercing or injury should raise the suspicion of a pseudomonal infection. In the absence of treatment, streptococcal infection can spread rapidly and lead to deformity of the ear.

Treatment of erysipelas. Prescribe penicillin or cephalosporin antibiotics until complete elimination of the disease. If this is neglected, recurrence is possible. You can use sulfonamide drugs. Topical agents in the form of lubrication of reddened edges with an alcoholic solution of iodine, the use of ichthyol ointment, lotions or compresses with drilling fluid, alcohol, resorcinol, etc. do not work. Levosin-type ointments,

physiotherapeutic methods – UFO in erythemal doses, laser therapy and ultraphonophoresis should be used.

Patients with recurrent erythema and manifestations of lymphostasis on the background of antibiotic therapy are prescribed prednisolone 30 mg per day with a gradual dose reduction or its analogue; nonsteroidal anti-inflammatory drugs, means to reduce the permeability of capillaries. immunostimulants; with severe intoxication – detoxification therapy.

Treatment of eczema of the outer ear should begin with exposure to the underlying disease that led to the eczema. It is necessary to exclude irritating influence of various endogenous and exogenous factors; treatment of purulent otitis media is carried out, chronic foci of infection are sanitized; if necessary, adjustments are made to carbohydrate metabolism. Diet with restriction of table salt, carbohydrates, except for alcohol, chocolate, fried and smoked dishes is important.

Excessive discharge from the ear should be removed by rinsing with warm saline sodium chloride, followed by thorough drying of the skin, as the liquid may increase the itching. In case of eczema of the external auditory canal, it is advisable to prescribe hormonal glucocorticosteroid creams and ointments mono- or multicomponent (prednisolone, hydrocortisone, flucinar, hyoxizon, triacutane, triderm, pimafucite, etc.). For lotions of wet areas of the skin, use 0.25 % solution of silver nitrate.

In the allergic process, hyposensitizing therapy is carried out with first-generation antihistamines, the sedative effect of which helps to reduce clinical manifestations (diphenhydramine, diazoline, tavegil, fencarol). Second-generation antihistamines (loratadine, azelastine, cetirizine) and third (desloratadine, levocetirizine, fexofenadine) are more effective.

Important importance is attached to the daily toilet ears for purulent otitis, swimming in the pool or open water.

At accession of a secondary infection to treatment add antibacterial or antiseptic drugs according to results of sensitivity of microflora to antibiotics.

Therefore, to clarify the sequence of differential diagnosis of eczema and the choice of external drugs, a diagnostic algorithm should be developed, which should be based on clarifying the nosological diagnosis of dermatosis, based on the nature of clinical manifestations, infectious complications, etiological affiliation of infectious agents and its infectious agent.

The basis of these funds should be selected taking into account the stage of the inflammatory process. A differentiated approach to the choice of external therapy should be based on the following features: age of the patient, nature of the disease, location and prevalence of the skin process, the possibility of combination with other drugs, the expected duration of therapy, which will directly affect its effectiveness.

Diffuse otitis externa

Diffuse otitis externa (otitis externa diffusa) is an inflammation of the skin of the external auditory canal. This inflammation occurs as a result of penetration of pathogenic microorganisms due to damage to the skin of the external auditory meatus because of manipulations in the ear. As it was already mentioned, the use of cotton swabs or the presence of a foreign body can cause external diffuse otitis. It should be noted that this pathological condition is often called the "swimmer's ear", as it increases during the bathing season or among people who visit the pools. Concomitant factors for the occurrence of external diffuse otitis are maceration of the skin due to chemical or thermal burns, allergies or metabolic disorders (often bilateral process).

Therefore, the most common ***causes of diffuse otitis externa*** may be:

– moisture in the ear after bathing or swimming, especially in the presence of bone narrowings of the ear canal (exostoses);

– frequent swimming in chlorinated water, as chlorine dries the skin of the ear canal;

– irritation or damage with cotton swabs;

– dry sensitive skin of the ear canal to skin diseases;

– the presence of diabetes or other diseases that cause infections;

– allergies to soap, hairspray, earbuds, hearing aids, etc.;

– eczema.

Pseudomonas aeruginosa, Staphylococcus. epidermidis and S. aureus – 3 most common pathogens isolated in acute external diffuse otitis. In addition, the causative agents of the bacterial etiology of this pathology may be some species of *Microbacterium;* members of the family *Corynebacterium* and *M. otitidis* and *M. alconae*.

The bacteriological method of diagnosing acute diffuse otitis is usually used only in severe cases, when it can help in the choice of antibacterial therapy.

In addition, the cause of diffuse otitis is often the herpes virus, the causative agent of chickenpox. In the case of chickenpox, some viruses survive for many years in the ganglia (nodes of nerve switching). In situations where the immune system is weakened, they become active again and cause inflammation of the skin of the external auditory canal.

Viral infections of the outer ear caused by herpes viruses are rare, but important for the differentiation of acute diffuse otitis externa. Herpes zoster (Ramsay-Hunt syndrome) causes blisters on the external auditory canal and posterior

auricle, severe otalgia, paralysis or paresis of the facial nerve, loss of taste in the front two-thirds of the tongue and reduced tearing on the affected side. Therefore, the differential diagnosis of otitis externa is important, because such pathologies of herpetic etiology in the treatment should include timely systemic antiviral therapy and systemic steroids. Complaints of otalgia in the absence of edema of the ear canal and a clear inflammatory process of the middle ear should raise the suspicion of pathology outside the ear. Perhaps the most common cause of ***otalgia*** is temporomandibular joint syndrome. These patients usually complain of pain not only in the ear, but also in pain radiating to the temple or neck. A history of chewing gum, bruxism, or a recent dental procedure followed by a malocclusion. Elderly patients with a long history of tobacco and ethanol use, and more recently younger patients with papillomavirus infection, may have such symptoms as well. Therefore, in such cases, a complete examination of the head and neck with visualization of the mucous membranes of the head and neck, assessment of any tumors on the neck and palpation of the base of the tongue is recommended. Other potential etiologies include dental pathology (caries, retinal molars), tonsillitis, peritonsillar abscesses, pharyngeal abscesses, styloid elongation, angina pectoris, intrathoracic aneurysms, lingual-pharyngeal neuralgia, and knee neuralgia.

Shingles (Ramsay Hunt syndrome, Figure 18) is an acute independent disease of viral origin and is characterized by the appearance on the anterior surface of the auricle, tragus, parotid, external auditory canal group of vesicles with serous or amber content. Subsequently, this content of bubbles turns into purulent, the bubble bursts, pus pours out and dries up in the form of yellowish crusts. The etiological factor of shingles is alpha-herpesvirus (shingles virus). Activation of the virus is due to insufficient activity of macrophages, T-lymphocytes and

decreased production of immune mediators, including interferons. The interval between the appearance of a skin rash and the development of meningoencephalitis is 5–15 days. After the disease, the virus stays for a long time in the ganglia of sensitive nerves: V, VII, VIII cranial nerves, intervertebral nodes and in the cervical sympathetic ganglia.

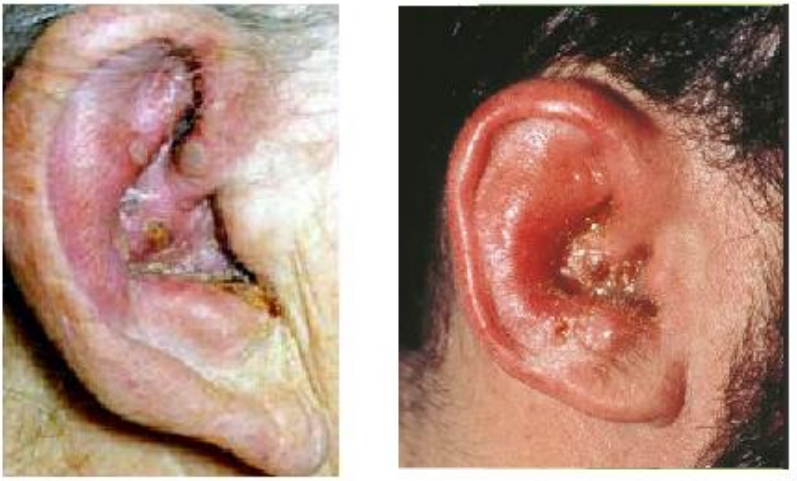

Fig. 18. Clinical picture of Ramsey Hunt syndrome.

The etiology of Ramsey Hunt syndrome is insufficiently studied. Some believe that the disease may be due to polyneuritis of the branches of the trigeminal nerve, according to others - it is serous meningitis, which passes to the nerve pathways. Patients complain of severe pain in the external auditory canal, which radiates in all directions. Fever, chills, and weakness are very common. Rarely, lesions of the V, VII, VIII pairs of cranial nerves, which recover in a few weeks to one year. Paresthesias in the form of itching, pain, heartburn may be felt in the damaged areas of the auricle for some time. Diagnosis of shingles is facilitated with the appearance of a rash in the outer ear, acute onset and

neurological pain make it possible to correctly diagnose. The course of the disease is favorable, but sometimes long.

Treatment of Ramsey Hunt syndrome primarily includes antiviral drugs to prevent the spread of infection due to inhibition of virus replication: acyclovir (valacyclovir, famciclovir, penciclovir), specific gamma and immunoglobulins, interferons and their inducers, human interferon group B, immunomodulators, analgesics, antioxidants. Cephalosporin antibiotics are prescribed when a bacterial infection occurs. Topically – antiseptic and antiviral ointments (3 % eye ointment Zovirax, 2 % tebrofen, 0.5 % florenal, etc.), electrophoresis with novocaine, UHF electric field, UFO. In the case of ineffective treatment of paresis (paralysis) of facial muscles, decompression of the facial nerve is sometimes prescribed.

Fungal infections of the outer ear occur if the skin remains moist in the ear canal for a long period of time. This happens, for example, when wearing a hearing aid or chronic infections of the middle ear. Some dermatitis affects the skin of the ear canal and makes it susceptible to fungal infections. In addition, the side effects of some drugs can contribute to the development of otomycosis. Mold and yeast-like (dimorphic) fungi are often ecological factors of otomycosis.

In the clinical practice of dermatologists, otolaryngologists and family physicians are common ***fungal lesions of the skin of the outer ear***: candidiasis (Figure 19, b), penicilliosis, mucorosis, aspergillosis (Figure 19, a), sometimes the causative agents of fungal lesions of the auricle may be fungi. *(Microsporum canis, Trichophyton rubrum*), the pathogenicity factor of which is a keratolytic enzyme. The etiological role of fungal flora in the occurrence of otitis externa in most cases is not established, although fungi as pathogens are found in 3–18 % of patients with inflammatory processes of the external auditory canal. Among otomycoses

mycotic external otitis prevails, and both at adults, and children. Moreover, mycoses of the outer ear are much more common than they are diagnosed by doctors clinically and mycologically. It is known that in the pathogenesis of otomycotic processes a significant role is played by inflammatory processes of the ear, irrational antibiotic, hormone therapy, trauma to the external auditory canal, mycogenic allergy, general somatic diseases. Patients with diabetes mellitus with fungal infection of the ENT organs are a kind of risk group that requires close attention of family doctors and otolaryngologists.

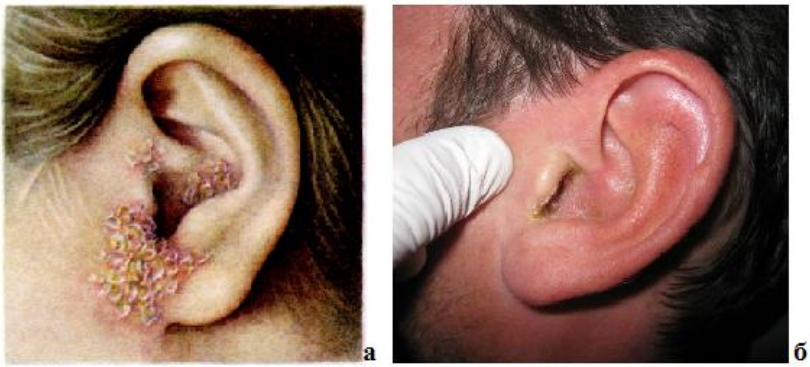

Fig. 19. Clinical symptoms: a – aspergillosis of the external auditory canal and auricle; b – candidiasis of the auricle of the external auditory canal.

Treatment of fungal diseases of the ear is difficult and not always successful, due to the formation of resistance of these pathogens to antifungal drugs and the rare use of mycological examination to determine the antifungal pattern (Figure 20). For the treatment of mycoses of the skin in recent years, topical antifungals of the allylamine series, in particular terbinafine derivatives, have become widely used. They have high antifungal activity, a wide range of action, do not irritate the skin.

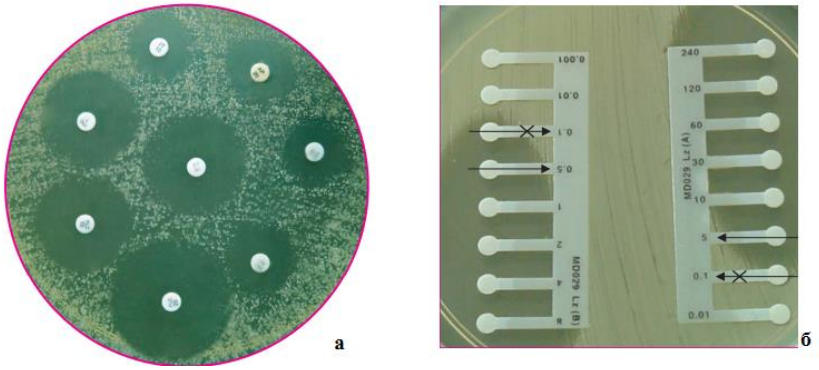

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Fig. 20. Determination of sensitivity of fungi of the genus *Candida* to antifungal drugs: a – the result of the discodiffusion method: the degree of sensitivity or stability of fungi of the genus *Candida* when using discs with antifungals is judged visually by the presence or absence of culture growth around discs with antifungals to which sensitivity is determined; b – result of HighComb MIC test fluconazole: determination of MIC fluconazole against fungi of the genus *Candida.*

Therefore, the emphasis on the manifestations of ***dermatoses on the skin of the outer ear*** is determined by a number of circumstances. First, the skin of the outer ear is often affected in many trivial dermatoses, where it is only one of many simultaneous localizations of skin pathology (dermatitis, eczema, seborrhea, burns, etc.). Second, the localization of some dermatoses on the skin of the auricles is often important and sometimes crucial for diagnosis (disc-shaped lupus and tuberculosis, smallpox). Third, there are diseases of the skin of the outer ear, which have a local-specific nosological significance, such as boils of the outer ear, nodular chondrodermatitis and otomycosis. That is, the diagnosis of ***"otitis externa"*** is a collective concept, which can hardly have a nosological significance, as it unites a large group of diseases

that differ in etiology, pathogenesis, clinical manifestations and methods of treatment.

Complaints on the background of otitis externa. The clinical course of otitis externa is divided into 3 stages: pre-inflammatory, acute inflammatory and chronic inflammatory. The acute inflammatory stage is divided into three stages: mild, moderate and severe. The pre-inflammatory condition is characterized by itching, swelling and a feeling of fullness. The acute stage is accompanied by pain and soreness of the atria. As the infection progresses from mild to severe, the itching, pain, and soreness of the eardrums increase. The canal becomes more swollen, becomes more erythematous. The secretion, initially clean and odorless, will turn into a thick, abundant serous exudate. In the severe stage, the lumen is erased due to increased edema and serous-purulent material. The patient complains of severe pain, especially when chewing or touching. Fever, periauricular edema, and cervical lymphadenopathy may also be present.

Therefore, acute diffuse otitis is characterized by an acute onset of the disease. Undoubtedly, the leading complaint of diffuse otitis externa is severe ear pain, especially at night, which may be accompanied by itching. Pain is aggravated by pressing on the tragus, pulling the auricle, talking and chewing. Patients report hearing loss due to tissue swelling and obstruction of the lumen of the ear canal. Diffuse otitis externa may be accompanied by a rise in body temperature to febrile levels.

In children, the onset of acute otitis media is accompanied by sharp pain in the ear, high body temperature (up to 38–40°C), hearing loss, general intoxication. Infants become restless, cry constantly, shake their heads, press the sick ear to the pillow, rub the ear with his hand. Babies often refuse to eat because sucking and swallowing increase the pain. Periods of anxiety in a child can be replaced by a depressed

state; young children often have diarrhea, belching and vomiting.

Diagnosis (Figure 21) of diffuse otitis externa:

1. Otoscopy reveals swelling and redness of the skin of the external auditory canal, narrowing of its lumen (possibly to complete obstruction). In severe cases, the infiltration may spread to the soft tissues of the mastoid or maxillary area. Maceration of the skin of the auditory canal may be observed. Painful palpation of the tragus is observed as well.

2. The results of tuning fork examinations with external auditory canal obstruction will be the same as in the presence of a sulfur plug in the external auditory canal, and will indicate a violation of sound conduction.

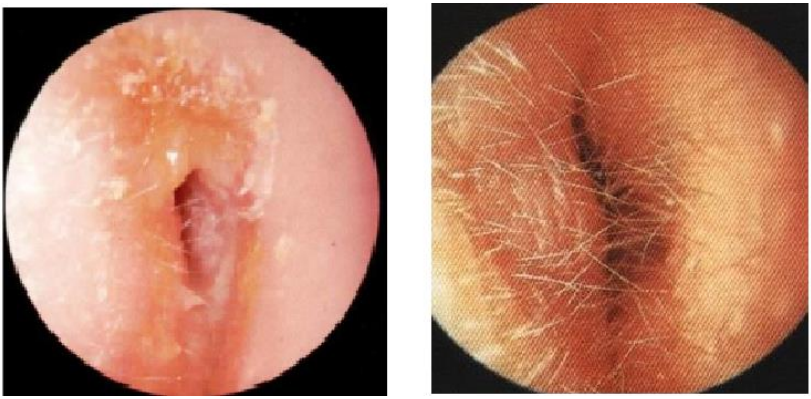

Fig. 21. ***Acutr diffuse otitis externa***.

3. An audiometric research shows a conductive deafness if there is an obturation of an auditory canal.

4. The results of General Blood Count tests may reveal changes in leukoformula (shift of the formula to the left), increase in white blood cells and sed rate. If the patient has diabetes – there is an increased level of sugar in urine and blood.

5. Staphylococcus aureus and *Pseudomonas aeruginosa* are detected in most in bacteriological studies. *Candida, Aspergillus are detected in mycological researches.*

6. In severe cases, for differential diagnosis with inflammatory diseases of the middle ear, CT or MRI of the mastoid processes are performed. To diagnose diffuse otitis externa, the airiness of the cells of the mastoid process and the tympanic cavity must not be disturbed.

7. Palpation of adjacent lymph nodes reveals adenopathy – it's an inflammation.

Treatment of diffuse otitis externa depends on the severity of the pathological process and the results of bacteriological examination of the ear canal swabs to examine the microflora and sensitivity to antibiotics. Since the structure of the pathogens of this pathology is heterogeneous (bacterial, fungal, viral or combined), it is necessary to choose a differentiated approach to the treatment of every patient. Difficulties in treatment could happen due to the growing virulence of pathogens and their resistance to antibiotics and antiseptics, due to unreasonably wide and uncontrolled use of ones.

Treatment can usually be performed in an outpatient setting, however, in severe forms of diffuse otitis externa and violation of the general condition of the patient, it is carried out in a hospital.

If there is a mild or moderate diffuse otitis externa, treatment is based on local therapy. An important role is played by cleaning and rehabilitation of the auditory canal from secretions. ***For local treatment***, multicomponent drops or ointments are used in the external auditory canal (instilled or on the tourniquet). Combination of drugs, in addition to antibacterial or antiseptic agents, may include glucocorticosteroids (hydrocortisone, dexamethasone), which

have anti-edema and anti-inflammatory effects, and anesthetics (lidocaine).

Ear drops: ciprofarm dex (ciprofloxacin, dexamethasone), auridexan (decamethoxine), combinil-duo (ciprofloxacin and dexamethasone), candibiotic (chloramphenicol, polymyxin, lidocaine), dexamethasone, neomycin, polydexa, neomycin , dexamethasone), dioxidine, miramistin. Ointments: triderm, triacutane (betamethasone, clotrimazole, gentamicin), celestoderm (betamethasone), bactroban, bactopic, bacterialis (mupirocin).

An important aspect in the treatment of diffuse otitis externa is the relief of severe pain. For this purpose, prescribe nonsteroidal anti-inflammatory drugs (nimesulide 100 mg 2 times a day, ibuprofen 200 mg 4 times a day, etc.).

If there are ***severe diffuse otitis externa***, general disorders of the body (fever, severe pain), complications (phlegmon that spreads to the parotid area); systemic antibacterial therapy should be added to the current treatment (broad-spectrum or bacteriological examination of the microflora and sensitivity to antibiotics):

– beta-lactam antibiotics of the penicillin series with/without clavulanic acid (amoxicillin, ospamox, augmentin, amoxiclav, flemoclav solutab, etc.);

– macrolides (azithromycin 500 mg 1 time per day for 3–5 days, clarithromycin 250 mg 2 times a day for 7–10 days) as first-line drugs are prescribed in cases where there is a confirmation of the etiological role of atypical flora in the occurrence of diffuse otitis externa or there are contraindications for appointment aminopenicillins;

– cephalosporins of II (cefuroxime (zinacef, zinnat, axef, abicef, etc.) and III (cefixime (loprax, suprax, cefix)), cefpodoxime (cefodox), cefotaxime (loraxime), ceftriaxone (blicef, emsef, emetaf, lorax zacef, ceftum), cefoperazone (hepacef, medocef)) generations;

– fluoroquinolones of I (norfloxacin, ofloxacin) (zanocin, loflox, floxan), ciprofloxacin (flaprox, cifran, cyprinol, ciprolet), lomefloxacin (lomflox)) and II (levofloxacin) (zolev, glevo, abila) generations.

The effectiveness of the prescribed therapy should be evaluated within 48 to 72 hours. Significant regression of pain within patients with diffuse otitis externa is observed in one day, and after 7–10 days of recovery. The ineffectiveness of treatment may be due to insufficient penetration of the drug into the external auditory canal due to its edema, the resistance of the microflora to antibacterial agents. If there is a persistence of symptoms it is necessary to specify the diagnosis, to exclude atopic, contact dermatitis, manifestations of psoriasis or eczema.

Abscess of the external auditory canal

Abscess of the external auditory canal. (boil, carbuncle) **(otitis externa circumscripta)** – is an acute inflammatory disease characterized by suppuration of the hair follicle (or several - with carbuncle), sebaceous glands and surrounding tissues, often in the membranous-cartilaginous part of the external auditory canal, where these formations are located (Figure 22).

The reason for the formation of boils is damage to the skin of the auditory canal and in the presence of purulent pathogens, often staphylococcus, streptococcus, less often - Pseudomonas aeruginosa, Escherichia coli, Proteus, fungi, often due to exogenous and endogenous factors. General and local cooling, infectious and endocrine diseases, eczema, exudative diathesis, etc. are important in its occurrence.

The causative agent of the infection penetrates the hair follicle and sebaceous gland during the violation of the integrity of the skin of the external auditory canal, where there are favorable conditions due to the anatomical and

physiological features of its location and stable microclimate. Boils are much less common in children with direct auditory canals. In patients with high sulfur content, boils are very rare**.**

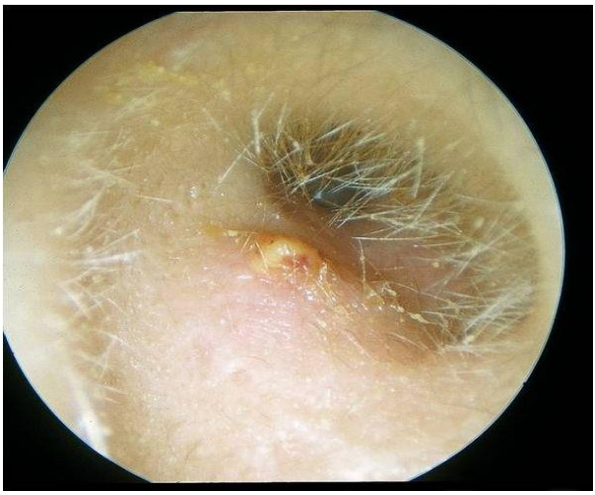

Fig. 22. Boil of the lower wall of the external auditory canal.

Boils of the outer ear can be a manifestation of general furunculosis, for example, in diabetes, hypovitaminosis, immunodeficiency.

Complaints. The symptoms of an external auditory canal boil are similar to diffuse otitis externa. The pathological process begins with itching in the ear canal, which gradually develops into severe pain. At this time, the boil noticeably increases in size, and palpation causes severe pain. Pain can radiate to the jaw, back of the head, mastoid process, neck and the side of the head where the affected ear is located. The pain becomes worse at night, while chewing, swallowing and talking, so very often patients, especially children, with this disease lose their appetite. Severe pain occurs when you press on the tragus or when you pull out the auricle. This

pathological condition is accompanied by febrile temperature and symptoms of general intoxication.

With an increase in the size of the boil there is a narrowing of the lumen of the auditory canal and, thus, hearing acuity becomes significantly reduced. On the 4–6th day of the disease, the boil festers and breaks through at the largest protrusion, accompanied by the release of purulent-necrotic masses. At the same time the intensity of a pain syndrome sharply decreases, and the general condition is estimated by the patient as "satisfactory".

Diagnosis **of an external ear abscess** is based on anamnesis, patient's complaints and examination results.

1. Otoscopically there are two stages of the boil of the auditory canal:

I. *Infiltrative* – is manifested at first by swelling and redness of the skin of the auditory canal in the area of the formation of a boil, narrowing of the lumen of the passage. Infiltration can spread to the soft tissues of the mastoid area, the maxillary area. Painful palpation of the tragus is typical. During the first four days of the disease there is a progress of suppuration and necrotization of soft tissues. At the apex of the conical protrusion (boil) a purulent head with a rod becomes visible.

II. *Abscessing* – on the 4–6th day of the disease the boil breaks in the largest place of protrusion, which is accompanied by the release of purulent-necrotic masses. The introduction of an ear funnel is difficult due to swelling and sharp pain.

2. Audiometric examination: hearing within the age norms with the absence of obstruction of the auditory canal by a boil, and conductive deafness – in the case of its overlap with edema or several boils.

3. In Complete Blood Count tests – reactive changes in leukoformula, an increased sed rate. In the presence of diabetes

- the presence of sugar in the urine and an increased level of it in blood.

4. Staphylococcus aureus et albus are often detected in bacteriological studies.

5. CT or MRI of the head is used in severe cases for the differential diagnosis of inflammatory diseases of the middle ear. Undisturbed airiness of the cells of the mastoid process and the tympanic cavity indicates an abscess of the outer ear.

6. Palpation shows an enlargement and tenderness of adjacent lymph nodes.

Differential diagnosis of boils is performed with:

1. Diffuse otitis externa (look above);

2. Purulent perforated otitis media. To distinguish the disease from suppuration from the cavities of the middle ear, it is necessary to completely clean the external auditory canal from purulent masses and examine the eardrum, which should not have pathological changes if there is a furuncle. In addition, secretions from the middle ear in acute inflammation are mucopurulent, while in abscessing boils – purely purulent.

3. Mastoiditis – infiltration and pain in boils are expressed in the area of attachment of the auricle, in mastoiditis – in the area of the mastoid process. The auditory canal in boils is narrowed in the cartilaginous department, and in mastoiditis – in the bony part because of the overhang of the posterior-upper wall of the passage. The tympanic membrane in boils is gray, while in mastoiditis – hyperemic, infiltrated, and can be perforated.

4. Otomycosis – discharge in otomycosis are "cheesy" or black. To determine the diagnosis, a bacterioscopic examination of the microflora with subsequent sensitivity should be performed. In addition, otomycosis has no acute onset, itching in the ear predominates over pain, also it is not accompanied by a violation of the general condition (fever,

chills), infiltration and redness of the skin of the ear canal have no clear boundaries.

5. Inflammation of the parotid gland. When a boil is placed on the front or lower walls of the auditory canal in the area of the Santorini glands, the infection can spread to the parotid gland. However, in the primary inflammation of this gland there is redness, swelling of the skin directly above the gland and this condition is not accompanied by changes in the ear canal. In addition, sialoadenitis is accompanied by dry mouth, swelling of the neck and face from the lesion.

Treatment of boils of the external auditory canal primarily depends on the stage of the pathological process. In the stage of infiltration in the first days of the disease, the principles of treatment of this pathology are the same as in the treatment of diffuse otitis externa (see above). Broad-spectrum antibacterial drugs are prescribed systemically (intramuscularly or intramuscularly) in dosage according to the patient's age and weight, nonsteroidal anti-inflammatory drugs, and topical drops or ointments in the external auditory canal on the tourniquet.

Indications for surgical treatment are abscessing of the boil on the 4–6th day of the disease, increased pain or intoxication, the appearance of parotid lymphadenitis. In the stage of abscessing, first of all, the boil should be dissected at the site of the largest protrusion under local anesthesia (2 % lidocaine solution, ultranest, septonest, etc.). Removal of purulent contents and a necrotic core should be performed. The cleaned cavity must be treated with antiseptic (decane, dioxidine) or antibacterial (ceftriaxone, levofloxacin) solution. You can install a rubber drain in the postoperative cavity and replace it 1–2 times a day. After that, use turundy 3–4 times a day impregnated with a hypertonic solution of 10 % sodium chloride (to reduce edema) with an antiseptic or antibacterial drug.

In case of recurrent boils of the external auditory canal, courses of autohemotherapy (intramuscular injections of the patient's own venous blood from 4 to 10 ml every 48 hours), courses of antibacterial drugs and antistaphylococcal vaccine are prescribed.

The complications of the boil of the external auditory canal are associated with the development of the infectious process in the regional anatomical structures. Mastoiditis occurs more often (and as a result there is the development of the infectious process in the inner ear, facial nerve), chondritis of the auricle, arthritis of the temporomandibular joint, less often - myringitis and acute otitis media. In the case of severe immunodeficiency, there is a risk of intracranial complications (meningitis, encephalitis), lymphadenitis, thrombosis of the venous sinus, systemic generalization of the infectious process – sepsis.

Otomycosis

Otomycosis – a fungal infection, which can affect the external auditory canal, the eardrum, the middle ear cavity, postoperative cavities (attic and antrum). Otomycosis occupies almost the third part of all the diseases that occur in the external ear. This condition is facilitated by such factors as anatomical features of the structure of the outer ear, eczema or dermatitis of the external auditory canal, a discharge of pus from the middle ear cavity, trauma of the skin of the external auditory canal, irrational antibiotic therapy, metabolic disorders (diabetes) or immunodeficiency, allergies. dysbiosis, etc.

The main causative agents of otomycosis are opportunistic fungi of the genera *Aspergillus* (Figure 23, 2), *Renicillium* (Figure 23, 4), *Mucor* and yeast-like fungi of the genus *Candida* (Figure 23, 3), which are widespread in nature, and only under certain conditions, they can acquire pathogenic properties and cause damage to human organs and systems.

Realization of pathogenic potential is possible due to violation of barrier mechanisms and reduction of protective forces of the macroorganism.

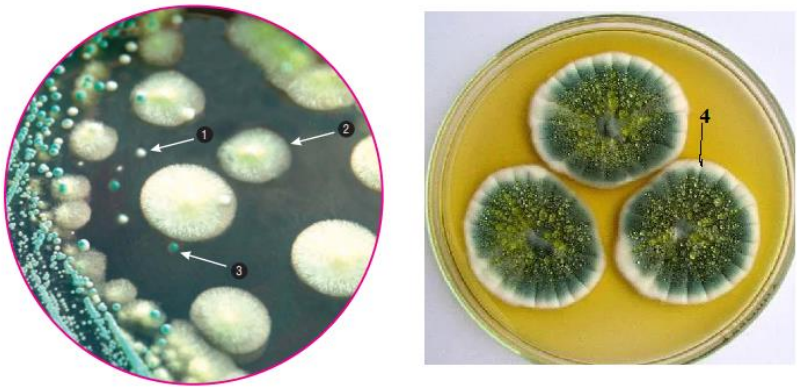

Fig. 22. High Chromium agar with the growth of yeasts and molds: 1. *Saccharomyces cerevisiae;* 2. *Aspergillus brasiliensis; 3. Candida albicans;* 4. growth *of Penicillium* spp. on Saburo's medium.

Aspergillosis in the structure of otomycoses is 61–65 %, penicillitis – 8–10 %, candidiasis – 24–29 %, in some cases the disease is caused by fungi of the genera *Mucor* and *Alternaria*. The leading role of fungi of the genus *Aspergillus* in the etiology of fungal lesions of the ear is due to their constant presence in the environment: air, soil, house dust and causes inevitable contact with skin and mucous membranes, as well as their infection. However, there are some differences in the etiological spectrum of otomycoses in different localizations of the inflammatory process. These features of the etiological factor must be taken into account when choosing drugs for treatment. But in the modern period there is a dominance of ***mixed infection*** in mycoses of different localization.

In the ***pathogenesis of otomycoses*** caused by opportunistic fungi, endogenous and exogenous risk factors for

their development are important, in which fungi go from a saprophytic lifestyle to a parasitic one and cause disease.

Exogenous factors include microtraumas that disrupt the integrity of the skin and mucous membranes, creating a gateway and favorable conditions for adhesion and subsequent invasive growth of fungi, as well as features of the nature of nutrition (protein and vitamin deficiency), working conditions (increased level of humidity, gassiness and dust) of a person, violation of sanitary and hygienic norms, pollution of the environment, increase of radiation background, etc. Prolonged treatment with antibiotics of various chemical groups, independent and uncontrolled use of them, use of corticosteroid hormones, cytostatics, oral contraceptives, the presence of immunodeficiency conditions in patients (especially AIDS) contribute to the emergence and development of fungal lesions of the ear caused by opportunistic fungi. diabetes, pathology of the gastrointestinal tract (dysbacteriosis of various degrees), malignant neoplasms and transplants of organs and tissues.

The main pathogenetic links in the development of fungal disease are the adhesion of fungi to the surface of the mucous membrane or skin, their colonization, which is a consequence of the natural balance between bacteria and fungi, and their invasive growth. Free access of atmospheric air to the external auditory canal and postoperative cavity, the presence in it of oxygen and carbon dioxide necessary for nutrition and growth of fungi, the temperature optimum in the ear cavity (33–36°C), the absence of direct sunlight, anatomical features structures (skin deepening) are favorable factors for the development of otomycosis of these localizations.

Also one of the important factors contributing to the invasion and colonization of fungi is the formation of exudate in prolonged inflammatory processes of the outer and middle ear of bacterial origin, which contains proteins and other chemicals that are nutrients necessary for their growth and

development. A special place in the pathogenesis of otomycosis is an allergic factor.

Conditionally pathogenic yeasts and molds, being on the surface of the skin and mucous membranes for a long time, cause allergic reactions, which in turn stimulate the growth of fungi and increase their pathogenic properties. Integrity of natural barriers – skin and mucous membranes, the process of constant exfoliation of the epithelium, the presence of lactic and fatty acids, enzymes in the secretions of sulfur and sebaceous glands, as well as the presence of (indigenous) obligate microflora that adheres to epitheliocytes, colonizes and secretes substances , which inhibit the reproduction of allochthonous pathogenic microorganisms, forms the colonization resistance of the skin of the external auditory canal and prevents the penetration of fungi into the internal environment of the macroorganism.

Thus, the violation of the integrity of the skin and mucous membranes, their microbiocenoses, resistance of the macroorganism leads to colonization and invasion of fungi and the development of a pathological process.

For a more detailed understanding of the pathogenesis of otomycosis, it is necessary to mention the pathogenic properties of the most common pathogens (80-90% of pathogens are members of the genera *Candida and Aspergillus*), which belong to opportunistic pathogens.

Thus, the factors of pathogenicity of fungi of the genus *Candida* are presented below:

– the ability to adhere to the organs and cells of the host organism;

– toxigenicity due to the synthesis of hemolysin and endotoxins;

– synthesis of hydrolytic enzymes that cause damage to host tissues and facilitates the penetration of pseudomycelium (Figure 23) (secreted aspartylproteinases and phospholipases);

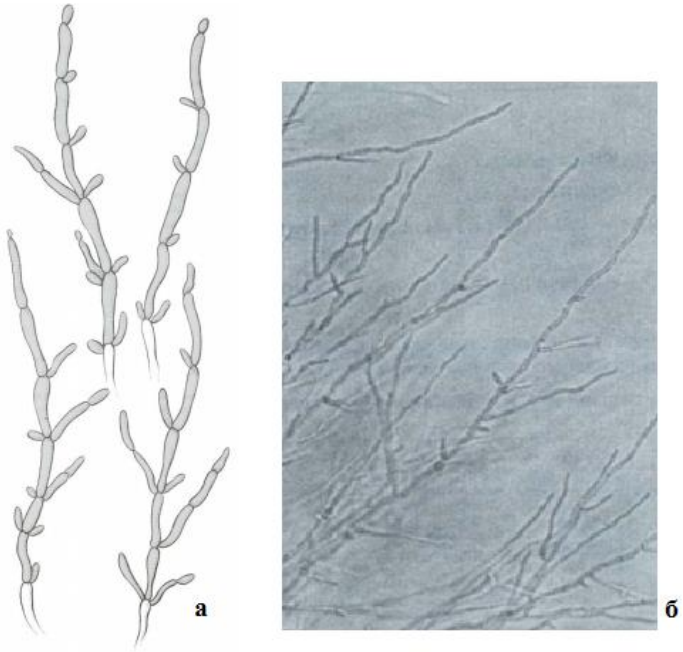

Fig. 23. a - Pseudohyphytes of Candida guilliermondii (10 μm scale); b - repeatedly branched pseudohyphs of Candida parapsilosis with single blastoconidia formed along the hyphae during growth on corn agar in Petri dishes, 460x.

– transformation into pseudomycelium, which invades the tissues of the host;

– the ability to potentiate allergic reactions and immunomodulatory effects that reduce the natural antimicrobial resistance of the macroorganism;

– spontaneous phenotypic variability, which plays an important role in the processes of adaptation of fungi to different anatomical niches of the macroorganism and the acquisition of resistance to antifungals;

– suppression of the obligate microflora of the mucous membranes of the host and the formation of a mixed infection.

The set of pathogenic factors of Candida spp. contributes to the repeated strengthening of their invasive potential in the conditions of violation of the system of antimicrobial resistance of the host and changes in their microenvironment.

It should be noted that fungi of the genus Candida can exist in microbial communities in the macroorganism. Moreover, fungi of the genus Candida not only experience antagonistic pressure from members of the normal microflora, but often form associations with other microorganisms, which, in turn, contribute to the colonization of habitats by fungal flora.

Biological properties of aspergillus: morphologically, aspergillus consists of the same type of mycelium (4–6 μm wide), sometimes "heads" with conidia are found. These fungi have significant biochemical activity, form various enzymes (proteolytic, sucrolytic, lipolytic), and some species contain endotoxin. In addition, aspergillus has an allergenic effect. Aflatoxins, which are produced, for example, by yellow aspergillus, are especially dangerous for humans because they have a carcinogenic effect.

According to various clinical studies, the causative agent of otitis externa increasingly became not only monocultures (Figure 24), but also bacterial-bacterial (Figure 25), fungal-fungal (Figure 26) and **bacterial-fungal** association, which significantly complicates the diagnosis, treatment of this pathology and involves the appointment of drugs with both antibacterial and antifungal activity.

According to various studies *Candida spp*. detected in conjunction with *Rotavirus; Staphylococcus aureus; Staphylococcus epidermidis, Staphylococcus haemolyticus;*

Pseudomonas aeruginosa; Klebsiella pneumonia; Morganella morganii; Proteus vulgaris.

Fig. 24. a – fungal lesion of the ear *Aspergillus niger* – separation of black color (mycelium); b – growth of a monoculture of *Aspergillus niger* from material from a patient on Saburo medium (mycological method of diagnosis); c – the same type of mycelium of aspergillus and conidia.

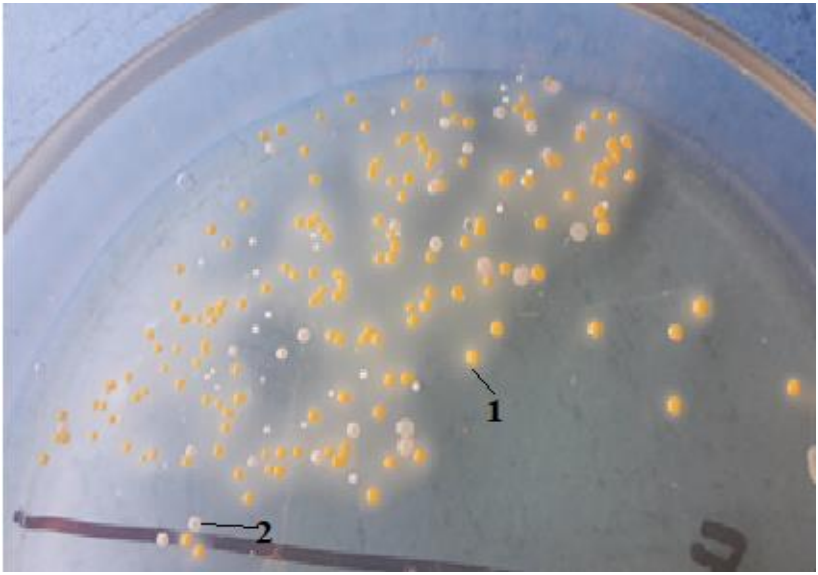

Fig. 25. Growth of *Staphylococcus aureus* (1) and *Staphylococcus epidermidis* (2) at primary inoculation of material from a patient with combined otitis on LSA (bacteriological method of diagnosis).

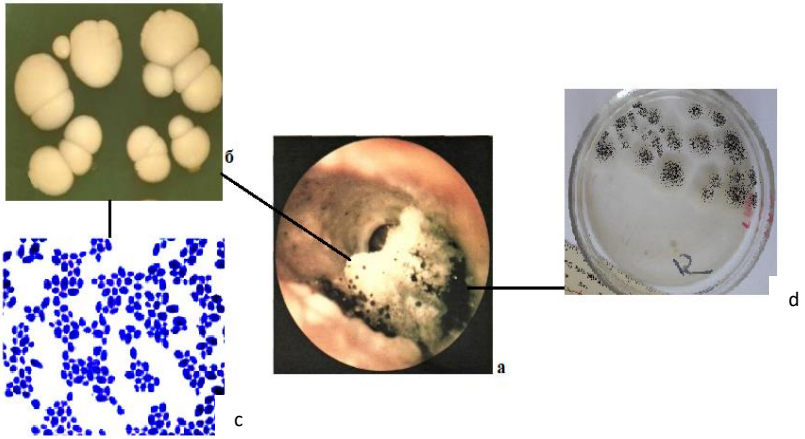

Fig. 26. a – fungal lesions of the ear *Candida albicans* and *Aspergillus niger* – there are typical white discharge along with black inclusions typical white overlays along with black inclusions Aspergillus niger; b – growth of *Candida albicans* from the material from the patient on Saburo medium (mycological method of diagnosis); c – in budding *Candida albicans* cells, Gram stain; d – growth of *Aspergillus niger* from material from a patient on Saburo medium (mycological method of diagnosis).

Clinical manifestations and course of otomycosis are due to a number of factors: the type of pathogen, the localization of the process, the presence of comorbidities, concomitant microflora of bacterial etiology. According to the nature of the course of otomycosis is divided into acute – the duration of the process up to 1 month, subacute – from 1 to 6 months, chronic – more than 6 months.

The main complaints of patients with fungal otitis externa are itching, pain, discomfort, ear congestion, hearing loss, discharge from the ears, hypersensitivity of the external auditory canal and auricle. As mentioned above, fungal

infections of the outer ear are most often caused by fungi of the genus *Aspergillus*. ***At aspergillosis defeat of an external auditory canal*** otoscopically the expressed reactive changes of its skin and a tympanic membrane are observed.

There is infiltration of all walls, resulting in a narrowing of the lumen of the external auditory canal. The tympanic membrane is sharply hyperemic, unevenly infiltrated, its contours are vaguely defined. In the external auditory canal accumulates a large number of pathological secretions, the removal of which visualizes areas of skin and eardrum, which often bleed. The color of secretions depends on the type of pathogen (color of the mycelium of the fungus): pathological masses of black color indicate the defeat of fungi *A. niger*, yellow-green color - characteristic of inflammation caused by fungi *A. flavus*, gray and gray-black discharge are observed when infected with fungi *A. fumigatus*. For penicillin lesions of the outer ear is characterized by the presence in the external auditory canal of pathological secretions of light yellow color. His skin is moderately hyperemic, infiltrated, the lumen of the passage narrows slightly, the process involves the eardrum, which in some cases is erosively altered.

In candidal lesions of the outer ear otoscopic picture has its own characteristics: redness and infiltration of the skin is expressed throughout the external auditory canal, in some areas with the phenomena of maceration. The surface of the skin is covered with a plaque of white or white-yellow color, cheesy consistency, which is easily removed, the reagents of the phenomenon from the eardrum are less pronounced than in lesions of aspergillosis etiology. The process often extends to the auricle and ear area. Hearing loss on tonal audiograms is recorded by air conductivity in the range of 20–30 dB, mainly at low frequencies (100–500 Hz).

As it was mentioned earlier, one of the leading pathogens of mycoses of the auditory canal is fungi of the

genus *Candida*, and the proportion of otitis caused by *Candida albicans* is about 25 %.

Mycotic lesions of the middle ear in most cases are a secondary disease that develops as a result of superinfection of the middle ear cavities on the background of chronic otitis media of bacterial origin. It is also possible to develop otomycosis as a result of surgery, during which there is trauma to the skin and mucous membranes, which creates favorable conditions for colonization, invasion and dissemination of fungal biota [58]. The main clinical manifestations of mycotic lesions of the middle ear are prolonged, profuse discharge from the ear, a feeling of discomfort and severe ear congestion, there is noise and pain in the ear of varying intensity, dizziness. A characteristic feature of the otoscopic picture in fungal otitis media is the accession of signs of otitis externa. Candidal lesions of the middle ear are characterized by the presence of secretions of cheesy or liquid consistency, often white, multiple perforations of the eardrum, severe redness of the skin of the external auditory canal.

Aspergillosis otitis media is characterized by pronounced reactive changes in the skin of the external auditory canal (Figure 27), narrowing of its lumen, eardrum with the phenomena of myringitis, the process spreads to the mucous membranes and periosteum, causing necrotic changes.

As mentioned earlier, one of the leading pathogens of mycoses of the auditory canal is fungi of the genus *Candida*, and the proportion of otitis caused by *Candida albicans* is about 25 %.

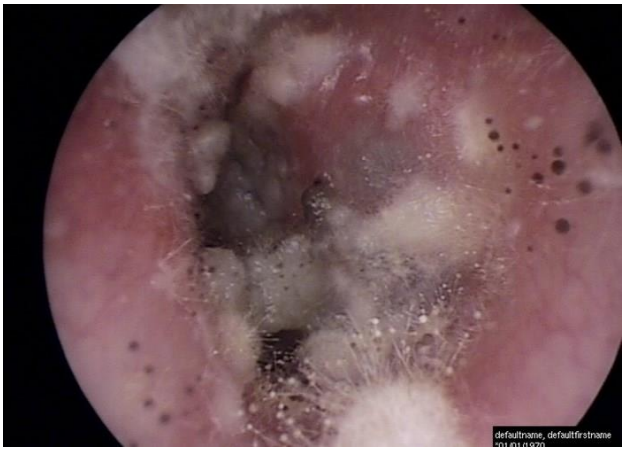

Fig. 27. Otomycosis of aspergillosis etiology.

Peniciliosis of the middle ear has a more favorable course. Otoscopy reveals a small number of pathological secretions of a mucous nature, moderate redness and infiltration of the skin of the external auditory canal.

Otitis media of mucosal etiology is a particularly dangerous inflammation, as the pathogens grow through the walls of blood and lymphatic vessels, which leads to their thrombosis and embolism, causing necrotic tissue changes and causing high mortality. At otoscopy of the postoperative cavity of the middle ear there is a complete or partial filling with pathological secretions, the nature and color of which depends on the type of pathogen, there is no or slow epidermis, the appearance of granulation tissue, redness of the skin of the external auditory canal.

Fungal lesions of the middle ear can lead to the development of intracranial complications (arachnoiditis, meningitis, brain abscess). Hearing loss in mycoses of the middle ear is conductive in nature with an increase in air thresholds from 10 to 25 dB over the entire frequency range.

Summarizing the data on the complaints of otomycosis presented by patients: patients complain of itching (the main symptom), pain, sometimes tinnitus, its congestion, headache on the side of the affected ear. Complaints are sometimes pronounced, despite small changes in the ear canal, which is associated with the toxic effects of fungi. At an otoscopy in an auditory pass the expressed infiltration and hyperemia of its walls, a tympanic membrane and existence of allocations is noted. Its color depends on the type of fungus. At defeat of Aspergillus niger allocations happen gray or black (similar to sulfur masses), at a candidiasis – a cheese kind (Figure 28).

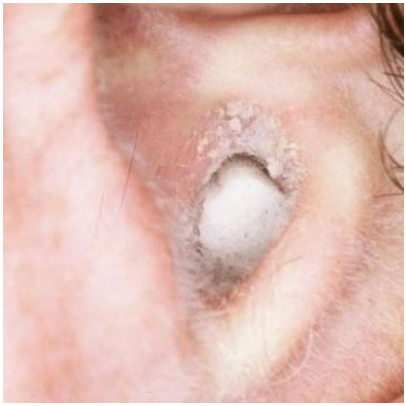

Fig. 28. Otomycosis of candidal etiology.

Fungal otitis media is characterized by viscous light discharge from the perforation of the tympanic membrane, sometimes abundant growth of small granulations around it, a bad effect of conventional conservative therapy. After surgery for a long time there is no epidermization, there is an increase in granulation, specific color separation. The course of otomycosis is long, with periodic exacerbations, which are due to the cycle of development of fungi in the ear and the presence of moisture in the external auditory canal. Some patients have secondary allergic reactions on the skin around the ear. Possible recurrence of the disease.

To diagnose "Otomycosis" should take into account the history of the disease, the patient's complaints, the results of examinations.

1. Fungal infection can be suspected with certainty on the basis of otoscopy and/or otomicroscopy. There is redness and swelling of the skin of the external auditory canal, narrowing of its lumen. The nature of secretions from the ear depends on the type of pathogen. An objective sign of fungal infection is the presence of a specific secretion, the color and consistency of which depend on the type of fungus. When affected by fungi of the genus Aspergillus, the pathological separation in the external auditory canal resembles blotting paper. The color of the discharge becomes black or brown when infected with fungi of the genus Aspergillus niger and yellowish or green – Aspergillus flavus, Aspergillus graneus, gray-black – Aspergillus fumigatus. When affected by fungi of the genus Candida otoscopic picture resembles weeping eczema, the selection has the form of whitish or yellowish crusts, bran-like scales or caseous masses. Fungal lesions may be suspected on the basis of otoscopy and/or otomycoscopy, but microbiological studies are crucial.

2. While obstructing the external auditory canal, the results of tuning fork examinations will be the same as in the presence of a sulfur plug in the external auditory canal, and indicate a violation of sound conduction.

3. At audiometric research at the expressed obturation of an external auditory pass by fungal masses hearing disturbance on type of sound conduction (conductive deafness) is defined.

4. In general blood tests should pay attention to the level of sugar in the urine and blood.

5. CT or MRI of the temporal bones makes it possible to assess the prevalence of the pathological process in the middle ear cavity or postoperative cavity.

Microbiological diagnosis of mycoses is based on microscopic, mycological, histopathological, immunological and molecular genetic studies.

The material for the study of mycotic otitis is pathological discharge from the ears (crusts, scales on the walls and in the lumen of the external auditory canal, the contents of the external auditory canal and postoperative cavity). In order to determine the structural elements of the fungus, the pathological material is examined in native (unstained) and stained preparations. At microscopic research of native drugs it is necessary to carry out their preliminary enlightenment in a solution of 10 % KOH or NaOH. For the preparation of stained preparations, the dried smear is fixed with a mixture of Nikiforov. The fixed smear is stained by the methods of Gram-Welsh, Romanovsky-Gimza and others. The microscopic picture of fungi of the genus Aspergillus is characterized by the presence of septate mycelium, single conidia or their clusters, sometimes - conidial heads (Figure 29).

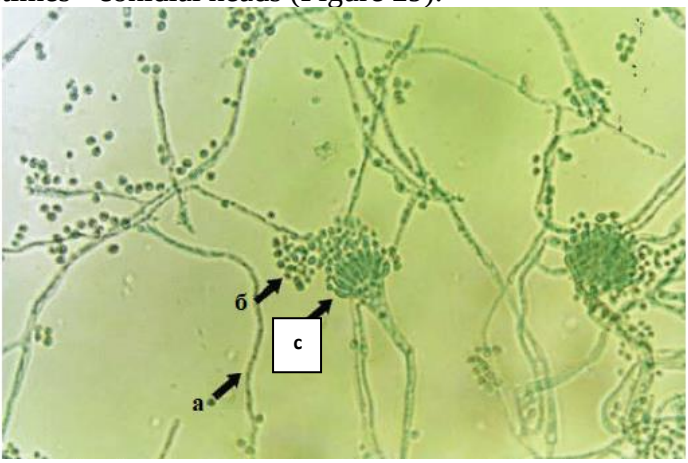

Fig. 29. The microscopic picture of fungi of the genus Aspergillus is characterized by the presence of septate mycelium (a), single conidia (b) or their clusters, sometimes – conidial heads (c).

At microscopsc research of a pathological material mushrooms of Renicillium (Figure 30) come to light in the form of the septated threads of a mycelium, there are conidiophores branched on the end in the form of a brush, separate spores. In the pathological material, the pathogens of mucomycosis under microscopy are determined in the form of branched broad filaments, there are sporangia.

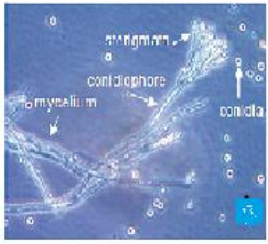

Fig. 30. Microscopic examination of pathological material in fungal lesions of fungi of the genus Renicillium.

The micromorphological features of fungi of the genus Candida are as follows (Figure 30): round or ovoid budding cells, many species form a pseudomycelium, which has no true septa. Mycological diagnosis – determination of the genus and species of the fungus that causes otomycosis – includees microscopy of pathological material with its subsequent inoculation on nutrient media (environment Saburo, Chapek and others, Figure 31) to study their biological (morphological, cultural, antigenic, biochemical and etc.) properties, as well as determination of sensitivity to antifungal agents.

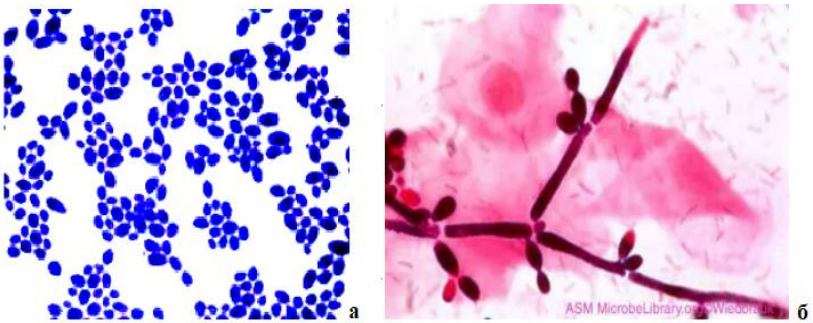

Fig. 30. Micromorphological features of fungi of the genus Candida: round or ovoid budding buds (a), many species form pseudomycelium (b).

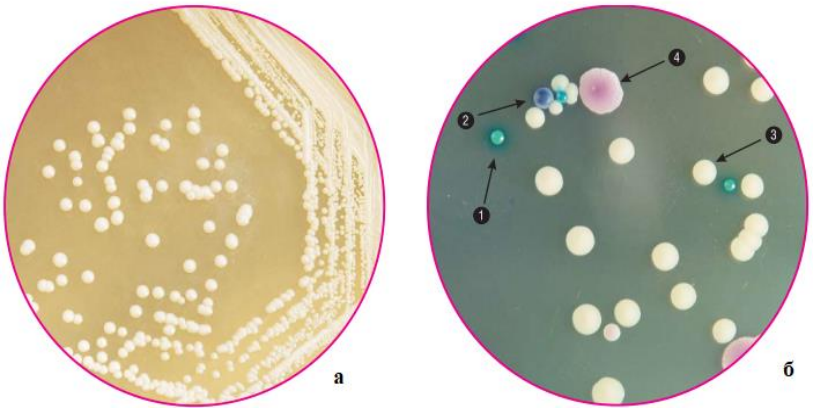

Fig. 31. Growth of fungi of the genus Candida on Saburo medium (a); on HighChrome selective agar for fungi (for differentiation) *Candida (b): 1. Candida albicans; 2. Candida tropicalis; 3. Candida glabrata; 4. Candida krusei.*

Using the culture method, the results can be quantified, which is very important to confirm the diagnosis. The essence of the method: collected with a tampon (Figure 32) or a Folkman spoon, loop the material is transferred into a vial of 5 ml of saline and with glass beads and shaken for five minutes

and inoculated on special media for fungal microflora. The crops are incubated for 48 hours at a temperature of 37°C, after which count the number of grown colonies. This provides data indicating the number of cells in the wash from 1 swab in 1 ml of medium. In the presence of more than 10 CFU / ml we can talk about fungal colonization.

Fig. 32. Transport systems for taking smears.

For convenience of collection and transportation of material from patients commercial transport systems Amies (Figure 32) and Amies with coal can be used; Eimans and others.

Such material collection systems are another modification of the basic transport environment, which maintains the viability of *Escherichia coli, Klebsiella pneumoniae,* and other bacteria of the family *Enterobacteriaceae; Staphylococcus spp.* and other opportunistic and pathogenic microorganisms, including yeasts and molds. This medium is able to maintain microorganisms such as *Neisseria spr., Haemophilus spr., Corynebacteria spp., Streptococcus spp., Enterobacteriaceae,* etc. for more than 3 days, but cultivation within the first 24 hours gives the best results.

The rules for using transport media to take the material are printed on each package. They can be summarized as follows:

1. Open a sterile bag with a transport flask in the place marked "Peel Here".

2. Remove the cap from the test tube.

3. Remove the swab with applicator and take a sample. When sampling, only the applicator head should touch only the sites of infection in order to minimize the risk of potential contamination of other areas. Precautions: When sampling the patient should not use excessive force or strong pressure, as this may lead to deformation of the tampon rod.

4. Place the swab with the applicator in the transport tube with the medium.

5. Write the patient's name and details on a test tube label.

6. Send samples to the laboratory for analysis.

7. Follow the rules of asepsis and antiseptics when using a tampon.

8. It should be noted that any sample may contain infectious microorganisms; in this regard, samples must be handled in accordance with appropriate biosafety precautions. After use, swabs and tubes should be disposed of in accordance with laboratory instructions for infectious waste.

9. Storage of transport test tubes with selected samples should be at a temperature of + 5°C – + 25°C. Do not overcool and overheat (temperature of delivery to the laboratory should be controlled). Transport systems for material collection cannot be used after the expiration date, which is clearly printed on each individual sterile package, as well as on the label of the transport tube with a tampon.

With the help of histological examination of granulation tissue and polyps it is possible to detect the pathogen in body tissues, to study the features of their structure and growth (Figure 33). Sections were stained by the Gomori-Grockot, Gram-Weigert method, hematoxylin with eosin, etc..

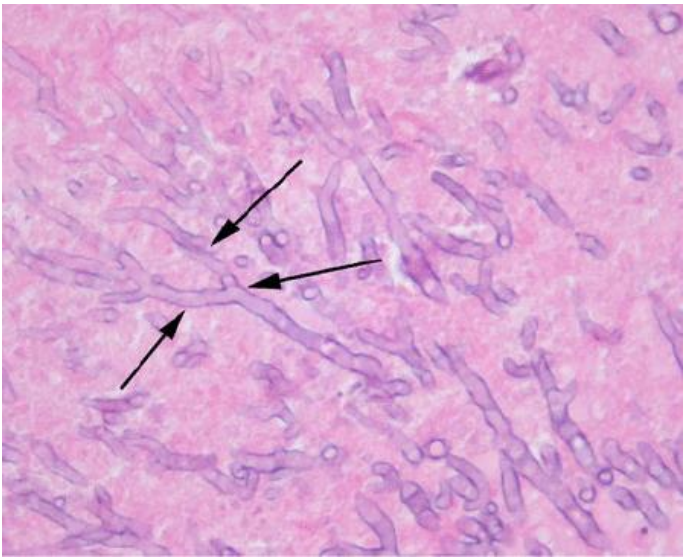

Fig. 33. Fungal hyphae with dichotomous branching in histological examination of fungal lesions of Aspergillus spp. (arrows).

Serological diagnosis allows to determine the presence of antibodies to antigens of the pathogen in the blood serum. Traditional methods of serological diagnosis (agglutination test, precipitation reaction, complement binding reaction, immunofluorescence reaction, etc.) have no independent diagnostic value, their informative value is considered only in combination with microscopic and mycological methods, which remain basic. For serological confirmation of fungal diseases, it is especially important not only to detect specific antibodies, but also to determine their dynamics during the disease. For this purpose, blood is taken from the subject several times: the first time – when the patient is treated, the second time – after 7–10 days, the third time – after 3–4 weeks. Diagnostically significant is the increase in antibody titer in paired sera in serological reactions by 4 times or more. Both

traditional and modern methods of serodiagnosis have their drawbacks: low sensitivity and specificity, due to common antigens for some species of fungi, decreased antibody levels in certain categories of patients (immunodeficiency, diabetes, chronic diseases).

Molecular diagnostic methods include polymerase chain reaction (PCR), which is characterized by speed of execution and high diagnostic sensitivity. However, in the modern period the use of molecular genetic methods in the diagnosis of otomycosis is studied, due to the lack of standardized tests, they do not belong to the generally accepted methods of diagnosing mycoses [59, 60, 65].

According to the information above, the gold standard of microbiological diagnosis of fungal lesions of the external auditory canal is the cultural method (Figure 34), because this method allows to determine the species of the pathogen, the degree of contamination of the locus with the pathogen and determine its individual sensitivity to antifungal drugs.

Treatment of otomycosis depends on the clinical picture, severity of the disease, the general condition of the body, as well as the type of fungi, taking into account the sensitivity of the pathogen to antifungal drugs. An important step in treatment is the toilet of the outer ear before the appointment of local drugs.

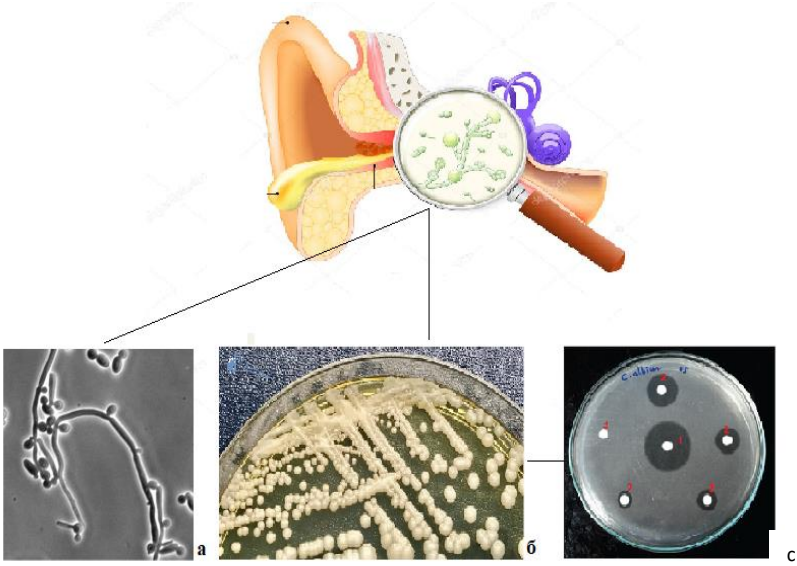

Fig. 33. Algorithm of mycological (cultural) method of diagnosis of fungal lesions of the auditory canal: a – microscopic examination of the material (approximate result); b – sowing of material on special media for fungal microflora with subsequent identification of the pathogen and determination of antimycoticogram (c).

Treatment of otomycosis includes the use of specific antifungal and nonspecific (desensitizing, probiotics, immunomodulators, vitamins) drugs, physiotherapy (UFO), topical drugs, elimination of factors that t developed the disease. The main criterion for rational antifungal treatment is the results of a study of the susceptibility of the pathogen to antifungal drugs. In this period, chemotherapeutic drugs are used for this purpose, which differ in origin (natural and synthetic), chemical structure (polyene antibiotics, azoles, allylamines, echinocandins, etc.), nature of action (fungicidal and fungistatic), method of application (enteral, parenteral, local). The mechanism of action of most antifungal drugs is

related to their effect on the synthesis of ergosterol in the fungal cell membrane. Despite a fairly wide range of modern antifungal drugs, there is no universally effective and completely non-toxic remedy. This explains the significant number of recurrences of the disease, even with positive treatment results. Therefore, the problem of creating new fungicides remains actual.

The main condition for effective local therapy is a careful ear toilet, which is carried out under the supervision of otoscopy by a doctor with a probe and cotton wool soaked in antifungal solution.

During the toilet, the ears remove all pathological contents (fungal masses, epithelial scales), ie. even their remnants support the inflammatory process, which leads to a prolongation of treatment.

At external otitis clear an external auditory pass, in case of fungal defeat of a middle ear remove pathological allocations from a tympanic membrane, washing of a tympanic cavity by antifungal drugs through perforations of a tympanic membrane is possible. In the presence of granulation tissue and polyps, their preliminary removal is performed. For the treatment of otomycosis most often antifungal drugs are used topically.

For local treatment with a gauze turunda 3 times a day appoint nitrofungin, clotrimazole, exoderyl, fluconazole, miconazole, pimafucin, ketoconazole, drops of candibiotics. For the treatment of candidiasis prescribe polyene antibiotics (amphotericin B, levorin, natamycin). The treatment lasts for 2–3 weeks.

In cases of insufficient effectiveness of local therapy, in case of recurrence of mycosis or chronicity of the process, along with local therapy, antifungal drugs should be prescribed systemically: fluconazole 100–150 mg once a day for 7–14

days; itraconazole 100 mg 1–2 times a day for 7–21 days; ketoconazole 200 mg 1–2 times a day for 4 weeks.

In addition to basic treatment, correction of concomitant pathologies that can lead to immunodeficiency states is required. The patient should receive a balanced diet rich in vitamins and exclude from the diet food that causes allergic reactions, sweets.

Systemic antifungal drugs are used in relapses of the disease and the prevalence of the process in the tympanic and postoperative cavities. The most convenient dosage forms for topical therapy of otomycosis are solutions, emulsions, suspensions.

In fungal lesions of the ear caused by fungal-fungal and fungal-bacterial associations, the best efficiency is shown by the naftifine (exoderyl), which has not only antifungal but also antibacterial activity, based on antibioticogram, and a candidate antibiotic with these anti-inflammatory properties, local anesthetic and antiallergic effects. Essential oils are considered to be promising tools in the local treatment of otomycosis. In addition to bactericidal, antiviral, immunomodulatory and anti-inflammatory action, their fungicidal activity has been proven.

In associative bacterial-fungal processes, treatment should be carried out only with antifungal drugs, ie. partial remediation of bacterial infection is carried out due to antibiotics produced by fungi of the genera Aspergillus and Renicillium. In the systemic treatment of otomycosis use antifungal drugs of different chemical groups: nystatin, levorin, Nizoral, fluconazole, orungal, lamisil.

The criteria for the effectiveness of treatment is clinical recovery within 1 month, which is confirmed by otoscopic picture and three times negative results of mycological examination.

Erysipelas of the external ear

Erysipelas of the external ear – an acute infectious-allergic disease of the skin and subcutaneous tissue, affecting the superficial lymphatic system of the skin of the outer ear and adjacent parts, caused by hemolytic streptococcus, with an expressed general reaction of the body.

This inflammation can occur primarily or secondarily during the transition process from the skin of the face and scalp. The causative agent is β-hemolytic streptococcus of the group A. It is proved that the occurring of erysipelas causes the violation of protective immunobiological mechanisms of the body in combination with infection of the auricle and external auditory canal by scratching, cracking, skin scratch with otitis externa. In a severe case of the disease, the process can spread to the eardrum with subsequent perforation and the development of erysipelas of the middle ear.

The main complaint is itching and sharp pain in the ear, which is exacerbated by palpation; deterioration of the general condition of the body with an increase of temperature to 39–40 °C; severe chills and headache.

Diagnosis is based on complaints, objective examination data: hyperemic and infiltrated auricle, including the earlobe with the presence of characteristic clear boundaries (demarcation lines) in the form of "tongues of flame" (Figure 34). In most cases, the inflammation spreads to the area of the mastoid process, which should be differentiated from mastoiditis. There are bubbles with serous contents at a bullous form of erysipelas.

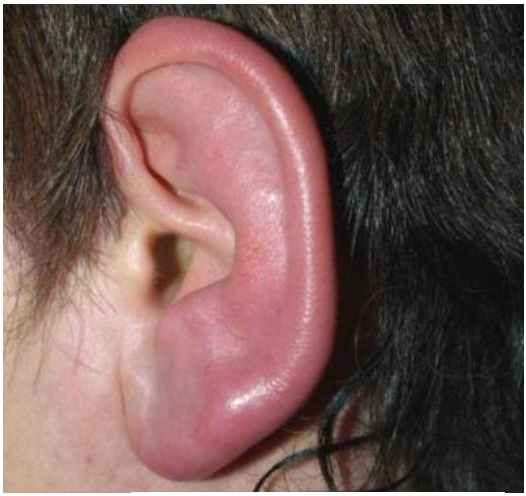

Fig. 34. Erysipelas of the external ear

Differential diagnosis of erysipelas of the outer ear is performed with:

1. Mastoiditis – it has a typical inflammation of the eardrum, overhang of the posterior-inferior wall of the external auditory canal, hearing impairment, which is not observed in erysipelas.

2. Chondroperichondritis of the auricle – in this case the lobe of the auricle is intact.

3. Purulent otitis – the spread of redness and swelling outside the outer ear and on the area of the mammary gland indicates erysipelas.

For the treatment of erysipelas, the drugs of choice are the following:

– penicillin 500 mg 4 times a day orally or 500 000 IU intramuscularly 4 times a day;

– alternatively (if you are allergic to penicillin) prescribe ceftriaxone 1.0 – 2 times a day;

– in infection caused by staphylococcus which is methicillin-resistant – vancomycin 1 g per day intravenously;

– alternative oral therapy – erythromycin 200–500 mg orally 4 times a day; roxithromycin 150 mg 2 times a day. However, it should be noted that macrolide resistance among streptococcus sprecies increases significantly.

The course of antibacterial drugs is prescribed for 10–14 days. Erysipelas can recur and lead to thrombophlebitis, abscess or gangrene.

THE PROBLEM OF RESISTANCE OF CLINICALLY SIGNIFICANT STRAINS OF MICROORGANISMS TO ANTIMICROBIAL DRUGS

As a causative agent of otitis media increasingly began to act not only monocultures, but also bacterial-bacterial and bacterial-fungal associations, which greatly complicates the diagnosis, treatment of this pathology and involves the appointment of drugs with both antibacterial and antifungal activity. According to the Global Antimicrobial Resistance Surveillance System (GLASS), the most common bacterial strains that are resistant to antibacterial drugs, including – multiple, these are *Pseudomonas aeruginosa*, *Escherichia coli*, Klebsiella pneumoniae, *Staphylococcus aureus* and *Streptococcus pneumoniae* and other opportunistic and pathogenic microorganisms.

Among patients with suspected infectious disease, resistance to at least one of the most widely used antibacterial drugs in different countries varies in a fairly wide range – from 0 to 82 %. WHO experts assess the spread of antibiotic resistance as a threatening situation in the health care system, which in case of inaction will lead to a sharp increase in premature mortality in patients with infectious diseases. This strategy assumes that for the treatment of infectious diseases it is necessary to use in drugs from the group "Access" (the goal of the program is to increase their use to 60 % of clinical cases), which have a narrow spectrum of antimicrobial activity,

and therefore their use inhibits the growth drugs from the groups "Watch" and "Reserve", which have a wide range of activity and to which the resistance of microorganisms is formed faster, which makes it impossible to use them as reserve drugs.

Among the yeasts and molds that cause otomycosis, there is also a tendency to develop resistance to antifungal drugs. Resistance of fungi to the action of antifungals can be both natural and acquired, which is formed in the treatment of fungal infections.

The mechanisms of natural resistance include, first of all, the lack of target action of the antifungal drug. The mechanisms of acquired resistance include mutations, which subsequently lead to the emergence of new strains of fungi, the sensitivity of which to antifungals is significantly reduced. This mechanism of implementation of resistance to antifungal drugs is often facilitated by long-term treatment.

The mechanisms of formation of antifungal resistance of fungi largely depend on the group of drugs used. Resistance of fungi to polyene antibiotics develops very slowly and is due to a number of complex genetic mutations, which ultimately lead to changes in the biosynthesis of membrane components.

The mechanisms of resistance of fungi to polyene antibiotics are currently poorly understood, but the available data suggest that resistance is associated with a decrease in the amount of ergosterol in the membrane and an increase in the number of its analogues in resistant strains. Resistance of C. albicans to imidazole and triazoles is associated with the accumulation of mutations in the ERG11 gene encoding sterol-14-demethylase. As a result, the cytochrome gene ceases to bind to azoles, but remains available to the natural substrate, lanosterol. At the same time cross resistance develops to all azoles.

The reasons for the development of resistance to antifungal drugs of yeast include the formation of a biofilm, the formation of which occurs in several stages. Under conditions of reduced protective mechanisms of the body, yeast fungi are first attached and colonized on the surface of the mucous membrane. Then there is their growth and spread, which allows fungi to form microcolonies in the basal layers of mucous membranes. Then there is the formation and growth of pseudohypha and large hyphae, as well as the production of components of the extracellular matrix, and only then is the dissemination of yeast cells from the biofilm and the formation of new sites of the infectious process. Biofilm repeatedly reduces the effectiveness of treatment of candidiasis, depending on the degree of its maturity.

Due to the frequent detection of strains of Candida spp. that are resistant to the most commonly used drugs, there is a need for etiological selection of ones. According to some foreign authors and clinicians, before starting treatment it is necessary to carefully analyze the sensitivity of the selected strains to antifungal drugs. This is especially true of C. albicans, species that often have a resistance to antifungals.

According to the information mentioned above, in order to predict the effectiveness of treatment of bacterial, fungal and fungal-bacterial otitis, it is necessary to determine the sensitivity of each isolated strain to the particular antimicrobial drug using standard methods and the Order of the Ministry of Health of Ukraine № 167 «Pro zatverdzhennya metodychnykh vkazivok «Vyznachennya chutlyvosti mikroorhanizmiv do antybakterial'nykh preparativ»» and unified approaches to determining the sensitivity of clinically relevant microorganisms to antibacterial drugs, based on the recommendations of the European Committee for the Sensitivity of Antimicrobials.

TEST TASKS FOR SELF-CONTROL

Topic "Clinical anatomy of the outer ear"

1. The skin of the auricle is tightly fused with the cartilage:

A) on the outer surface of the auricle;
B) on the inner surface of the auricle;
C) on the upper edge of the auricle;
D) on the lower edge of the auricle.

2. The lobe of the auricle consists of:

A) cartilaginous tissue;
B) bone tissue;
C) duplication of skin and fat;
D) contains a cavity.

3. The length of the external auditory canal in adults age is:

A) 3 cm;
B) 2.5 cm;
C) 1.5 cm;
D) 1.0 cm.

4. The anterior wall of the external auditory canal borders with:

A) temporomandibular joint;
B) the middle cranial fossa;
C) mammary process;
D) parotid salivary gland.

5. The posterior wall of the external auditory canal borders with:

A) temporomandibular joint;
B) the middle cranial fossa;
C) mammary process;
D) parotid salivary gland.

6. The upper wall of the external auditory canal borders with:

A) temporomandibular joint;
B) the middle cranial fossa;
C) papillary process;
D) parotid salivary gland.

7. The lower wall of the external auditory canal borders with:

A) temporomandibular joint;
B) the middle cranial fossa;
C) papillary process;
D) parotid salivary gland.

8. The eardrum separates the external auditory canal from:

A) the tympanic cavity;
B) the ear canal;
C) mammary process;
D) the inner ear.

9. The outer ear consists of:

A) auricle and ear canal;
B) the external auditory canal and eardrum;
C) the auricle and the external auditory canal;
D) ear canal and auricle.

10. The outer ear performs the function of:

A) sound conduction;
B) sound perception;
C) and sound conduction, and sound perception;
D) legibility of language.

11. Do not carry out sensitive innervation of the outer ear:

A) cervical plexus;
B) vagus nerve;
C) facialis;
D) trigeminus.

12. The motor nerve for the muscles of the auricle are:

A) facialis;

B) trigeminus;
C) vagus;
D) occipitalis.

13. Which area of the auricle has no cartilage:

A) helix;
B) anti-helix;
C) lobe;
D) antitragus.

14. On the auricle, the skin is tightly fused with the cartilage in the area of:

A) convex surface;
B) concave surface;
C) lobes;
D) not fused.

15. The lumen of the auditory canal in diameter among adults is:

A) 0.5 – 0.6 cm;
B) 0.6 – 0.7 cm;
C) 0.7 – 0.9 cm;
D) 1.0 – 1.1 cm.

16. The external auditory canal consists only of:

A) the membranous-cartilaginous department;
B) the bone and fibrous departments;
C) fibrous connective tissue;
D) the membranous-cartilage and bone departments.

17. Due to the loose fiber, the ear canal borders with the parotid gland:

A) from below;
B) from above;
C) in front;
D) behind.

18. The narrowest part of the auditory canal, isthmus, is:

A) at the entrance to the external auditory canal;

B) in the middle of the membranous–cartilage;

C) on the border of the membranous-cartilage and bone departments;

D) on the border with the eardrum.

19. The membranous-cartilaginous department of the external auditory canal does not contain:

A) sebaceous glands;

B) sweat glands;

C) sulfur glands;

D) hair.

20. Cough can occur while entering the funnel into the external auditory canal, because it is a:

A) reflex from the trigeminal nerve;

B) vagus nerve reflex;

C) reflex from the lingual-pharyngeal nerve;

D) reflex from the facial nerve.

Topic "Non-inflammatory diseases of the outer ear"

1. Where the sulfur glandscan be located in the ear?

A) near the eardrum;

B) in the bone department;

C) along the entire external auditory canal;

D) in the membranous-cartilaginous department;

E) on the ear shell.

2. "Clogging" of a pea or a metal ball more often occurs:

A) immediately at the entrance to the external auditory canal;

B) in the membranous-cartilaginous department;

C) at the site of transition of the membranous-cartilage to the bone;

D) in the bone before the tympanic membrane.

3. Removing a "clogged" pea in the ear canal can be performed with:

A) crochet;

B) rinsing the ear with water;

C) instill alcohol into the ear canal several times and remove wrinkled peas;

D) forceps, tweezers.

4. It is easier to remove a metal ball "hammered" into the ear cana withl:

A) crochet;

B) tweezers;

C) a magnet;

D) rinse with water.

5. Resting outside, during sleeping the patient felt insect penetration in the ear. At the same time, he noted an unpleasant sensations in the ear, scratching, and after a while the sharp pain – up tovery severe. What is the emergency care for such patients?

A) Fill the ear canal with 96 % ethyl alcohol solution, prescribe painkillers, consulting of an otolaryngologist;

B) Elimination, if possible, of a foreign body by improvised means, prescribe painkillers;

C) Close the external auditory canal tightly with cotton wool soaked in antiseptic solution, prescribe painkillers;

D) Fill the auditory canal with a sterile oil solution, or oil or aqueous solution of antiseptic, prescribe painkillers, consultation with an otolaryngologist;

E) Analgesics, anti-inflammatory therapy, antiseptic ear drops.

6. An alive foreign body is easier to remove with:

A) crochet;

B) immobilize, dripping oil into the ear canal, and remove;

C) tweezers;

D) do not touch, it will work.

7. The most common localization of otohematoma is:

A) the front surface of the upper half of the auricle;

B) earlobe;

C) the inner surface of the auricle;

D) the area of the mammary process.

8. Unilateral deafness, which occurred during taking shower, may be a consequence of:

A) sulfur plug;

B) otosclerosis;

C) Meniere's disease;

D) Wegener's disease.

9. Spontaneous form of otohematoma may occur in concomitant pathological conditions, except:

A) hemophilia;

B) oncohematological diseases;

C) trauma to the auricle;

D) hemorrhagic diathesis.

10. Against the background of a foreign body of the ear may develop the following complications, except:

A) diffuse otitis externa;

B) perichondritis of the auricle;

C) perforation of the eardrum;

D) otitis media.

Topic "Inflammatory diseases of the outer ear"

1. Otomycosis is:

A) acute purulent inflammation of the external auditory canal;

B) chronic purulent inflammation of the external auditory canal;

C) fungal inflammation of the external auditory canal;

D) viral inflammation of the external auditory canal.

2. Erysipeles – a diffuse infectious inflammation of the skin caused by:

A) staphylococcus;

B) streptococcus;

C) pneumococcus;

D) gonococcus.

3. With erysipelas the skin in the affected area is only:

A) sharply hyperemic;

B) significantly infiltrated;

C) limited by the inflammatory shaft from healthy skin;

D) everything is true.

4. Unlike erysipelas perichondritis of the auricle is characterized by:

A) redness and swelling with clear boundaries;

B) the spread of inflammation on the lobe, ear area;

C) the formation of bubbles with serous content on inflamed skin;

D) the lobe of the auricle is not involved in the inflammatory process.

5. *Perichondritis can not be:*

A) bullous with the formation of serous bubbles on the earlobe;

B) serous at the time of an insect bite or burn;

C) purulent when the infection enters the cartilage;

D) the consequence of otohematoma, boil of the ear canal.

6. *Progressive perichondritis of the outer ear is not characterized by:*

A) uniform, bumpy swelling of the skin;

B) fluctuation in the formation of purulent exudate between the cartilage;

C) deformation of the earlobe;

D) purulent melting of the cartilage of the auricle.

7. If there is a fluctuation in perichondritis of the auricle you should not:

A) make a wide incision of tissues;

B) scrape the area of the abscess with a spoon and remove necrotized tissue;

C) drain the open cavity;

D) do not open the abscess, and conduct UHF – therapy.

8. Perichondritis of the auricle is a diffuse inflammation of:

A) cartilage with skin involvement;

B) only the skin;

C) only cartilage;

D) only the cartilage.

9. Perichondritis can be:

A) serous or purulent;

B) hematogenous, lymphogenic;

C) catarrhal, follicular;

D) herpetic, contact.

10. Pain in the ear during chewing indicates the presence of a boil on:

A) the anterior wall of the ear canal;

B) the posterior wall of the ear canal;

C) the lower wall of the ear canal;

D) the upper wall of the ear canal.

11. Boil on the posterior wall of the ear canal is differentiated from:

A) acute otitis externa;

B) sulfur plug;

C) mastoiditis;

D) perichondritis of the auricle.

12. A patient's complaints of severe pain in the right ear, enlargement and redness of the auricle. Ill for 2 days after treatment with an acupuncturist. Objectively: the right auricle

is enlarged, the skin is hyperemic (except for the lobe), its contours are smoothed, palpation is painful, the entrance to the ear canal is sharply narrowed. Hearing: CMM-6m in both ears. What is the presumed diagnosis?

A) right acute diffuse otitis externa;

B) right-sided acute mastoiditis;

C) erysipelas of the right auricle;

D) right acute limited otitis externa;

E) perichondritis of the right auricle.

13.Eczema of the outer ear is not characterized by:

A) redness without a clear border of inflammation;

B) thickening of the skin, itching;

C) soaking, peeling or peeling;

D) a clear line of inflammation.

14. For the chronic form of ear eczema is not typical:

A) involvement in the process of superficial and deep layers of the skin;

B) involvement in the process only of the surface layers of the skin;

C) narrowing of the lumen of the ear canal due to thickening of the skin;

D) the appearance of cracks in the skin at the entrance to the ear canal.

15. Pain in a boil of the auditory canal mainly irradiates:

A) in the hand on the side of defeat;

B) in the eye, teeth, neck;

C) in the upper and lower extremities;

D) diffusely spreads throughout the body.

16. With perichondritis of the auricle palpation is painful:

A) the entire auricle;

B) earlobes;

C) ear area;

D) the entire shell, except the lobe.

17. A boil of the lower wall of an auditory pass can affect:

A) parotid gland;

B) papillary process;

C) facial nerve;

D) the joint of the lower jaw.

18. Chondroperichondritis of the auricle is:

A) diffuse inflammation of the epicartilage;

B) diffuse inflammation of the periosteum;

C) inflammation of the cartilage of the auricle;

D) inflammation of the cartilage and epicartilage of the auricle.

19. Othematoma is an accumulation of blood between:

A) bone and periosteum;

B) cartilage and epicartilage;

C) skin and epicartilage;

D) skin and fiber.

20. Otoscopy for boils of the external auditory canal:

A) rounded elevation of hyperemic skin, narrowing the ear canal is visualized;

B) there is a retracted eardrum;

C) hyperemia and infiltration of the skin of the membranous-cartilage and bone of the ear canal;

D) narrowing of the lumen in the bone, redness of inflamed skin.

21. Tactics of treatment of a boil of an auditory meatus:

A) early autopsy among all patients;

B) opening of the boil and antibacterial therapy;

C) squeezing the boil;

D) hormone therapy.

22. The patient, 27 years old, complains of pain in the right ear, intensifying at night, fever up to 37.6 °C. Objectively: the right auricle is slightly deviated, swelling behind the ear

folds, pain when pressing on the earlobe. The external auditory canal is narrowed in the membranous-cartilaginous department due to infiltration of the posterior wall. The eardrum is pearly gray, hearing – 6 m. Specify the most probable pathology.

A) right labyrinthitis;

B) boil of the right external auditory canal;

C) right mastoiditis;

D) right diffuse otitis externa;

E) right acute otitis media.

23. Contribute to the development of otomycosis:

A) only chronic purulent otitis media;

B) only antibiotic therapy;

C) diabetes mellitus;

D) everything is true.

24. The main symptoms of otomycosis:

A) itching in the ear, congestion, scanty discharge;

B) nausea and vomiting;

C) high fever, frequent dizziness;

D) constant headache, fever, foul-smelling discharge.

25. *For patients with otomycosis yje following therapy is indicated:*

A) antibacterial therapy;

B) antifungal, desensitizing therapy;

C) hormone therapy;

D) antihistamine therapy.

Answers to test questions

Clinical anatomy of the outer ear		*Non-inflammatory diseases of the outer ear*		*Inflammatory diseases of the outer ear*	
1.	A	**1.**	D	**1.**	C
2.	C	**2.**	C	**2.**	B
3.	B	**3.**	C	**3.**	D
4.	A	**4.**	C	**4.**	D
5.	C	**5.**	D	**5.**	A
6.	B	**6.**	B	**6.**	C
7.	D	**7.**	A	**7.**	D
8.	A	**8.**	A	**8.**	A
9.	C	**9.**	C	**9.**	A
10.	A	**10.**	B	**10.**	A
11.	C			**11.**	C
12.	A			**12.**	E
13.	C			**13.**	D
14.	B			**14.**	B
15.	C			**15.**	B
16.	D			**16.**	D
17.	A			**17.**	D
18.	C			**18.**	D
19.	B			**19.**	B
20.	B			**20.**	A
				21.	B
				22.	B
				23.	D
				24.	A
				25.	B

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