SECTION XXV. MEDICAL SCIENCES AND PUBLIC HEALTH

ABSTRACT

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DRUG-INDUCED OTOTOXICITY. AMINOGLYCOSIDE OTOTOXICITY

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In modern clinical practice, there are often cases of cochleovestibular disorders induced by the use of drugs. The term "drug-induced ototoxicity" is directly related to the introduction of streptomycin in medical practice and means the ability of drugs or chemicals to cause functional disorders and degeneration of internal ear structures: sensory-epithelial (hair) cells of the organ of Corti, neurons of the eighth cranial nerve (nervus vestibulocochlearis) or auditory centers of the brain. These injuries can be temporary or permanent.

There are 2 types of ototoxicosis: cochlear and vestibular toxicity. Cochleotoxicity usually occurs due to the adverse effects of the drug to the cochlea or auditory nerve. Further, the damaged structure causes symptoms of hearing loss in different ranges or complete deafness and tinnitus, which the patient experiences. If the toxic agent is in the vestibulum, semicircular canals or has a negative effect on the vestibular nerve, the patient experiences a violation of orientation and balance, dizziness, which are symptoms of vestibular toxicity. Also, when the vestibular system is affected, functional damage to the eye can be observed: nystagmus and oscilloscope. Damage to the parietal nerve or brain centers by toxic drugs causes the same symptoms, but the lesion usually remains permanent [1].

Modern pharmaceutical arsenal contains drugs whose ototoxic effect has been confirmed experimentally in laboratory animals: antibiotics (streptomycin, amikacin, gentamicin, neomycin, tobramycin and macrolides), loop diuretics (furosemide, bumetanide, ethacrynic acid), cytostatics (carboplatin, methotrexate, vinblastine), NSAIDs (aspirin, indomethacin), anticonvulsants (carbamazepine), antidepressants (amitriptyline), tranquilizers (meprobamate, chlordiazepoxide), antiarrhythmics (quinidine), and muscle relaxants (cyclobenzaprine) [2].

Most of cases of ototoxicity are associated with the use of antibiotics - aminoglycosides, which are active against aerobic gram-negative bacteria. Their mechanism of action is the binding of the active substance to the ribosomes of the bacterium and disruption of the synthesis of vital proteins, which causes a bactericidal effect [3].
The most ototoxic drugs are the first generation of aminoglycosides: Streptomycin, Neomycin, Kanamycin. Modern clinical protocols recommend avoiding their use or using analogues of new generations of aminoglycosides. Negative effect on the inner ear occurs by the mechanism of creation of active forms of oxygen, destruction of receptor elements of the sense organ - hair cells, direct damage to the spiral ganglion by the active substance of drugs. Amikacin has the same ototoxic effects as Kanamycin because it is a semi-synthetic derivative [4].

The frequency of such an adverse drug reaction is 1-10%. Symptomatically, it can be manifested by tinnitus, a feeling of "stuffiness" of the ears, hearing loss (up to complete deafness). Unlike Neomycin and Kanamycin, Streptomycin and Gentamicin are more vestibulotoxic with minimal exposure to the auditory analyzer.

Risk factors for aminoglycoside-induced ototoxicosis are:
1) Combination of these drugs with other ototoxic drugs - antibiotics, loop diuretics, cytostatics, NSAIDs, tranquilizers, which can lead to irreversible hearing loss;
2) The use of aminoglycosides in elderly patients can cause ototoxicosis on the background of cerebral circulatory disorders and the development of otosclerosis.
3) Genetic predisposition to ototoxicity of aminoglycosides [5].

References: