

CONFERENCE ARTICLE**RESULTS OF VEGF-A CYTOKINES IN ACUTE SENSORINEURAL HEARING LOSS OF VARIOUS GENESIS****U.S. Khasanov¹, U.P. Abdullaev²**^{1,2} Tashkent Medical Academy, Uzbekistan**ABSTRACT**

The examination of cytokine regulation is crucial for predicting the development of irreversible changes in the auditory analyzer that may arise as a consequence of acute sensorineural hearing loss (ASNHL) and for evaluating the efficacy of therapeutic interventions. The baseline level of vascular endothelial growth factor (VEGF-A) in the blood of healthy individuals was found to be 0.89 (0.6; 2.7) ng/ml, with a minimum value of 0.30 ng/ml and a maximum of 32.8 ng/ml. In the course of our study, it was observed that the level of the studied indicator in patients with ASNHL were almost 10 times higher than those in healthy individuals. In Group 1, consisting of patients with ASNHL who received a combination of complex therapy and hyperbaric oxygenation (HBO), the VEGF-A level at the time of hospitalization was 310.1 (127.4; 320.5) ng/ml, with minimum and maximum values of 120.5 and 335.8 ng/ml, respectively. In Group 2, comprising patients with ASNHL who underwent complex treatment and intratympanic steroid therapy, the VEGF-A level was 193.2 (152.2; 169.4) ng/ml, with individual variations ranging from 50.8 to 188.2 ng/ml.

Keywords: Cytokine, sensorineural hearing loss, steroid therapy.

INTRODUCTION

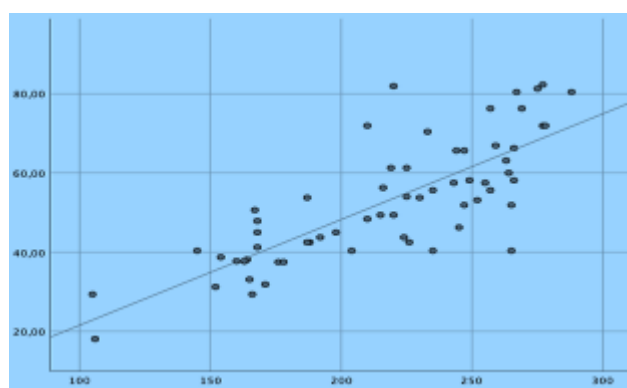
Acute sensorineural hearing loss is a medical emergency that can lead to irreversible and severe complications if not promptly addressed [1]. The condition may present suddenly or develop over several hours. Affected patients commonly report sensations of ear occlusion, tinnitus, and, frequently, dizziness [4]. Hearing loss is typically unilateral with bilateral involvement occurring in 2% of cases.

In acute sensorineural hearing loss, both the outer and inner hair cells of the cochlea, which primarily transduce sound vibrations into electrical impulses, are compromised [2-6]. The cochlea is an organ with a very high demand for oxygen.

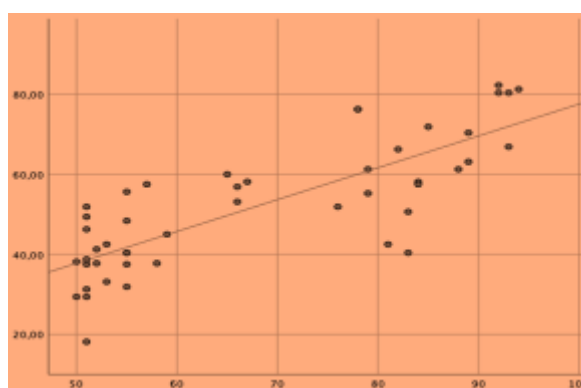
The pathophysiology of acute cochleitis and labyrinthitis is primarily attributed to viral-induced damage to small vessels and subsequent pathological changes in the inner ear tissues. These changes include increased permeability of vascular walls, the formation of extravasations and edema which contributes to elevated intralabyrinthine pressure, impaired intracranial permeability, and significant hypoxia of the sensory receptors [2]. Clinically, ASNHL is characterized by variable (and, in some cases, transient) hearing which may be perceptual or mixed (if endolymphatic mobility is compromised), usually of moderate severity (up to 20-30 dB on an ascending curve with a bone-air gap of 10-20 dB), a sensation of fullness in the ear, low frequency tinnitus, and mild dizziness. Depending on the location of the hypertension (in the cochlea or vestibular portion), symptoms may vary, with hearing loss or dizziness predominating on the affected side [4].

RESULTS

To more accurately study the characteristics of the humoral immune response in the patients under investigation, we conducted a comparative assessment of the level of VEGF-A cytokines in the blood. In the control group of practically healthy individuals, the VEGF-A level was 0.89 (0.6; 2.7) ng/ml, with a minimum value of 0.30 ng/ml and a maximum of 32.8 ng/ml. Our findings revealed that the VEGF-A levels in patients with ASNHL were nearly 10 times higher compared to healthy individuals. In Group 1 comprising patients with ASNHL who received complex therapy and HBO, the VEGF-A level at the time of hospital admission was 310.1 (127.4; 320.5) ng/ml, with a minimum and maximum values of 120.5 and 335.8 ng/ml, respectively. In group consisting of patients with ASNHL who underwent complex treatment and intratympanic steroid therapy, the VEGF-A level was 193.2 (152.2; 169.4) ng/ml, with individual variations ranging from 50.8 to 188.2 ng/ml. These results indicate that on the day of admission to hospital, the concentration of VEGF-A cytokine in group 1 patients was significantly higher than that in group 2 patients (Figure 1) ($p < 0.001$).



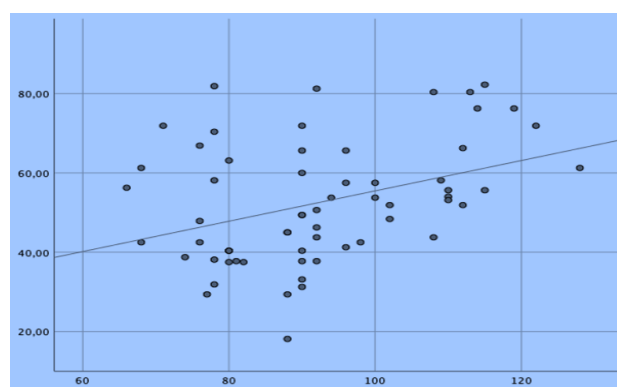
Group 1



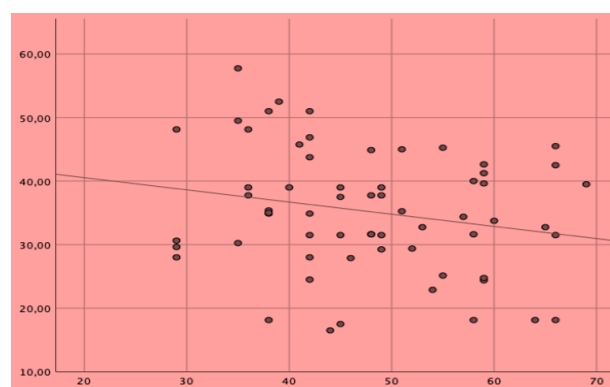
Group 2

Figure 1. Results of VEGF-A cytokine levels on day 1 of hospitalization across groups.

By day 10 of treatment, a reduction in VEGF-A concentrations was observed in both patient groups. Specifically, a two-fold decrease in the VEGF-A index was noted in Group 1 patients, with levels reaching 100.5 (87.4; 171.3) ng/ml, and individual values ranging from 32.0 to 198.3 ng/ml. This value remained significantly higher than the corresponding cytokine levels in Group 2 ($p < 0.001$). In Group 2, the VEGF-A levels were 58.3 (40.7; 72.8) ng/ml, with individual variations spanning from 24.8 to 87.3 ng/ml (Fig. 2).



Group 1



Group 2

Figure 2. Results of VEGF-A cytokine in the dynamics of treatment regimens on the 10th day of hospitalization across groups.

CONCLUSION

In summary, the pro-inflammatory cytokine VEGF-A was found to be significantly elevated in patients of group 2 compared to those in Group 1. A notable reduction in VEGF-A levels was observed following the administration of intratympanic steroid therapy in conjunction with comprehensive treatment protocols.

The alterations in the immunological markers among patients with ASNHL, their patterns over time, and their correlation with the final treatment outcomes, provide valuable insights. These immunopathogenetic indicators may serve as a tool for analyzing data in future studies and contribute to a deeper understanding of the disease's progression and response to treatment.

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