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Research Article

Comparative Analysis of Structural Changes in The Neurons of The Parietal Lobe of The Brain in Rats During Anoxia of Respiratory and Ischemic Genesis

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Abstract

Introduction: Glaucoma, a leading cause of irreversible blindness, presents significant management challenges, especially in patients intolerant to standard treatments. Recent findings from the Laser in Glaucoma and Ocular Hypertension (LiGHT) Trial offer new insights into alternative treatment strategies.

Case Presentation: This report discusses the case of a 79-year-old woman with a longstanding history of open-angle glaucoma (AOG), who was intolerant to conventional glaucoma medications. Her treatment included multiple surgeries: trabeculectomies in her left eye and various laser therapies in her right eye. The LiGHT Trial findings, demonstrating the efficacy of Selective Laser Trabeculoplasty (SLT) in providing long-term disease control with a favorable safety profile, offer relevant insights into similar cases.

Outcomes: As of the last examination, the patient's vision was 20/80 OD and 20/500 OS, with stable intraocular pressures and visual field loss. The case resonates with the LiGHT Trial's advocacy for SLT as a primary treatment option for managing glaucoma effectively.

Conclusion: This case underscores the complexities of managing advanced glaucoma in patients intolerant to traditional medications and highlights the importance of personalized, evolving treatment strategies. The insights from the LiGHT Trial reinforce the potential of SLT as a viable primary treatment, aligning with the need for innovative approaches in glaucoma management.

Key words: advanced open angle; glaucoma treatment

Introduction

Anoxia, as an extreme degree of acute oxygen starvation of the brain, can be caused by various internal and external factors. In particular, complete oxygen deficiency in the brain can occur as a result of hemodynamic disturbances (cardiac arrest) or the influence of an external mechanical factor (closing the airways with a foreign body, drowning, aspiration of gastric contents, etc.) [7].

Total acute oxygen deficiency leads to rapidly developing and often irreversible functional and structural changes in the brain [4]. First of all, the cortex suffers, as a phylogenetically younger structure of the brain. Damage to the parietal lobe cortex leads to disturbances in the response to sensory stimuli and spatial orientation [3, 5].

A feature of anoxia of respiratory origin, in contrast to anoxia of ischemic origin, is that the heartbeat persists for a short time. However, this does not

oxygenate the blood. In this regard, it is advisable to carry out a comparative analysis of structural changes in brain neurons during anoxia of respiratory and ischemic origin.

The goal is to compare structural changes in neurons of the parietal lobe cortex of rats during anoxia of respiratory and ischemic origin.

Materials and methods of research

The study was conducted on outbred white rats (18 males, weight 240±20 g), divided into 3 groups (n=6) in compliance with the requirements of Directive of the European Parliament and Council No. 2010/63/EU dated 22.09.2010 on the protection of animals used for scientific purposes.

The experiments were performed on 2 models of cerebral anoxia: mechanical asphyxia and total ischemia. The control group consisted of sham-operated rats (group 1). Modeling of mechanical asphyxia was carried out by ligating the trachea of rats below the cricoid cartilage of the larynx with a ligature for 24 hours (group 2). Modeling of total cerebral ischemia was carried out by decapitation of rats with material collection after 24 hours (group 3).

The studies were carried out under intravenous anesthesia (sodium thiopental, 40 mg/kg). The brain was removed in the cold and fixed in Carnoy's fluid, after which frontal paraffin sections of the parietal cortex, 7 μm thick, were prepared and stained using the Nissl method [6]. The location of the parietal lobe cortex was determined using a stereotaxic atlas [9].

In each animal, 30 neurons of the fifth layer of the parietal lobe cortex were studied and their size and shape were determined [8]. Changes in the area and shape (form factor, elongation factor) of neurons were assessed using the ImageWarp image analysis program (Bitflow, USA). In histological preparations, different types of neurons were determined by the degree of staining of their cytoplasm (chromatophilia) [2].

The obtained quantitative continuous data were processed using nonparametric statistics methods, licensed computer program Statistica 10.0 for Windows (StatSoft, Inc., USA). Data are presented as Me (LQ; UQ), where Me is the median, LQ is the lower quartile value; UQ – upper quartile value. Differences between the indicators of the control and experimental groups were considered significant at $p < 0.05$ (Mann-Whitney U-test) [1].

Research results

In the rats of the experimental groups, structural changes occurred in the neurons of the parietal lobe of the brain, which manifested themselves in changes in the size and shape of neurons, and the degree of staining of their cytoplasm.

After 24 hours of mechanical asphyxia, the area of neurons in the parietal lobe cortex decreased by 62% ($p < 0.05$) compared to the values in the control group. At the same time, the form factor decreased by 33% ($p<0.05$), and the elongation factor increased by 92% ($p<0.05$), which reflects the loss of sphericity and an increase in the elongation of neurons.

In rats with total cerebral ischemia, by 24 hours of the ischemic period, a more significant decrease in the area of neurons was observed (the area of neurons decreased by 85%, $p < 0.05$) compared with the indicators in the control group. At the same time, the form factor decreased by 33% ($p<0.05$), and the elongation factor increased 2 times ($p<0.05$), which reflects the presence of changes in the shape of neurons that do not differ from those that occurred at 24 hourly mechanical asphyxia ($p>0.05$).

In the control group, up to 95% of the population of neurons in the parietal lobe of the brain consisted of normochromic cells, and the remaining neurons were hypochromic (4%) and hyperchromic wrinkled (1%) cells.

In the animals of the experimental groups, there were no normochromic neurons in the parietal lobe of the brain ($p < 0.05$). During 24-hour mechanical asphyxia, hyperchromic wrinkled neurons were observed in the cortex (up to 64%, $p<0.05$), as well as shadow cells (up to 25%, $p<0.05$) and cells with pericellular edema (11%, $p< 0.05$). In rats with total ischemia, after 24 hours, cells with pericellular edema were observed in the cortex (up to 88%, $p < 0.05$), as well as hyperchromic wrinkled neurons (up to 7%, $p < 0.05$) and shadow cells (up to 5 %, $p<0.05$). So, with total ischemia and mechanical asphyxia, unidirectional changes in chromatophilia were noted ($p < 0.05$), which is manifested in an increase in hyperchromatic wrinkled neurons mainly with mechanical asphyxia, while at the same time, with total ischemia, cells with pericellular edema predominated.

So, a study of the consequences of anoxia for neurons in the parietal lobe cortex of rats under conditions of total ischemia and mechanical asphyxia over a 24-hour period revealed the presence of structural changes: a decrease in area and a change in shape (loss of sphericity and increase in elongation) of cells, as well as a change in the degree chromatophilia, which was manifested by the disappearance of normochromic neurons with a simultaneous significant increase in the number of hyperchromic wrinkled neurons during mechanical asphyxia and cells with pericellular edema during total ischemia. At the same time, total cerebral ischemia led to a more significant decrease in the area of neurons in the cortex of the parietal lobe of the brain compared to mechanical asphyxia and an increase in edematous cells and shadow cells. These differences may be due to the preservation of cardiac activity during mechanical asphyxia for a short period of time.

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