Natural Remedies Vol.22, №.1 (2), (2021) pp.117-123 [https: //jnronline. com / ojs / index.php / about / article / view / 908]

11. Azimov S.I. Universal urolog so'rovnomasining sil kasalligi bilan kasallangan bemorlarningurolog patologiyasini tekshirishdagi o'rni // «Tibbiyotda yangi kun» ilmiy referativ, ma'naviy ma'rifiy zhurnal №3 (31), 2020. S.238-241

12. Tilavov, T. B. (2022). Sexual Dysfunction of Men in Bukhara Region. INTERNATIONAL JOURNAL OF HEALTH SYSTEMS AND MEDICAL SCIENCES, 1(5), 19-22.

УДК: 611.068

МОРФОЛОГИЧЕСКИЕ ИЗМЕНЕНИЕ В ГОЛОВНОМ МОЗГЕ И В ЕГО МИКРОСОСУДАХ ПРИ ХРОНИЧЕСКОМ АЛКОГОЛИЗМЕ.

Тухсанова Н.Э. ***https://orcid.org/0000-0002-0475-2539***

Бухарский медицинский институт. (обзорная статья)

Аннотация: Изучены журналы, материалы научных конференций, а также другие информационные источники для собрания достоверной информации об изменениях в головном мозге и его микрососудах при воздействии алкоголя.

Доказано, что при алкогольной интоксикации головного мозга наблюдаются повреждение нейронов во всех отделах головного мозга, а также повреждение не только артерий, но и сосудов микроциркуляторного русла гемато-энцефалического барьера.

Ключевые слова: алкоголь, нейроны, микроциркуляторное русло, гематоэнцефалический барьер, периваскулярный отек.

СУРУНКАЛИ АЛКОГОЛИЗМ ТАЪСИРИДА БОШ МИЯ ВА УНИНГ МИКРОТОМИРЛАРИДА КЕЧАДИГАН МОРФОЛОГИК ЎЗГАРИШЛАРИ

Тухсанова Н.Э.

Бухоро давлат тиббиёт институти. (адабиётлар шарҳи)

Аннотация: Алкоголь таъсирида бош мия ва унинг микротомирларида кечадиган ўзгаришлар тўғрисида журналлар, илмий конференция материаллари шунингдек бошка информацион манбалар ўрганилиб чиқилди.

Алкоголь билан захарланиш натижасида бош миянинг барча нейронлари, кон томирлари, шунингдек гематоэнцефалитик тўсиқ микротомирлари жарохатланиши исботланган.

Калит сўзлар: алкоголь, нейрон, микроциркулятор ўзан, гематоэнцефалик тўсиқ, периваскуляр шиш.

MORPHOLOGICAL CHANGES IN THE BRAIN AND ITS MICROVESSELS DURING CHRONIC ALCOHOLISM

Tukhsanova N.E.

Bukhara State Medical Institute (review article)

Abstract: Journals, materials of scientific conferences, as well as other information sources were studied to collect reliable information about changes in the brain and its microvessels under the influence of alcohol.

It has been proven that alcohol intoxication of the brain causes damage to neurons in all parts of the brain, as well as damage not only to the arteries, but also to the microvasculature of the blood-brain barrier.

Key words: alcohol, neurons, microvasculature, blood-brain barrier, perivascular edema.

RELEVANCE The human brain is an incredibly complex organ that leads to the coordinated functioning of almost all systems in the body, in addition to those responsible for cognitive skills and thought processes. Unfortunately, a fast pace and far from the healthiest lifestyle slowly undermine the body, depleting its reserves and compensatory mechanisms. Today, the world faces an acute problem associated not only with high morbidity, but also with disability and even mortality from alcoholism[3,4,5,13,27].

Currently, the study of the effect of alcohol intoxication on the body of rats continues, the authors touch upon the following topics; intrauterine influence of alcohol on the reactivity of cerebral arterioles of the brain and its susceptibility to ischemic damage in adulthood [17,23].

The medical and demographic consequences of excessive alcohol consumption are manifested in a decrease in general health, an increase in morbidity, and premature mortality of the population, which, in turn, contributes to their early initiation into alcohol. The social consequences of drunkenness and alcoholism are very wide. The problem of alcoholism is a whole complex of social aspects that affect all areas of the normal

functioning of society, which are studied not only by medical workers, but also by specialists in other fields [1,9,10].

In acute alcohol poisoning, swelling of all parts of the brain comes to the fore. In the choroid plexuses of the brain, edema and swelling of the intercellular substance, basal membranes and villous stroma are also observed, which leads to compression and emptying of capillaries, necrosis and desquamation of the epithelium, etc.[7].

In the structures of the brain of rats during acute ethanol intoxication, signs of perivasal edema are detected, often areas of hemorrhages of diapedetic origin, which can be both local and widespread. In some cases, hemorrhagic foci, in addition to white and gray matter, affect the subcortical and stem parts of the brain[24].

With the combined effects of alcohol and carbon monoxide in the brain of animals, a sharp congestion of the vessels of the meninges, choroid plexuses and brain matter was noted. The brain capillaries looked partially collapsed and deserted, while others, on the contrary, were dilated, with signs of stasis. Moderate perivascular and pericellular edema was noted; diapedesis and hemorrhages were sporadic[2].

It has been established that when exposed to ethanol, plethora and stasis occur in the vessels of the pia mater, and plasma permeation of the endothelium occurs; damage to the microvasculature of the blood-brain barrier, which is accompanied by an increase in their permeability; penetration of red blood cells beyond the vascular wall; perivascular swelling of brain tissue, changes in the shape and size of neurons (neuron shrinkage).Thus, in the course of the study, it was found that the administration of ethanol at a dose of 1.5 LD50 causes an increase in the permeability of the microvasculature of the blood-brain barrier, which reflects the initial signs of their damage. Yes, in the group in rats that received only ethanol, during the study, congestion of the capillaries and vessels of the pia mater, stasis, intimal edema, and increased plasma saturation of the capillary endothelium were observed [18].

A study of the histological structure of the brain of persons with chronic alcoholic pathology revealed signs of sclerosis and hyalinosis of its vascular bed. It has been established that they concern not only the arteries, but also the level of microcirculation. It is important to note the presence of infiltrates of mononuclear cells along these vessels. This may be due to the immune system's response to damage to arterial walls and infiltration of plasma proteins during repeated episodes of alcohol consumption.

Impaired blood supply to the brain, as well as the direct effect of ethanol on its tissue, entails the gradual death of neurocytes with their replacement by glial elements[14,15].

In the vessels of the base of the brain, hyalinosis and sclerosis of the intramural arteries are detected, which indicates the toxic effect of ethanol on the vessels. There is 28 damage to the 3rd and 5th layers of brain tissue of the frontal lobes, as well as the molecular and ganglion layers of the cerebellar cortex in the form of an increase in the number of hyperchromic neurons, reduced in volume and a decrease in the number of normochromic cells [24].

Alcohol has significant neurotoxic effects on the developing brain, causing cognitive impairment in young people [10,13].

Other studies [26] revealed cortical atrophy (69%), atrophy of the corpus callosum (61.9%), cerebellar atrophy (30.9%), widening of the cortical sulci, third and lateral ventricles (50%), vasogenic changes (26.1%). In most cases, atrophy of the corpus callosum was combined with atrophy of the cortex and, less often, with vasogenic disorders. More often, pathological changes were localized in the corpus callosum.

In the brain of rats predisposed to alcohol dependence, morphofunctional differences are determined in the anterior part of the hypothalamus, which are expressed in differences in the structural organization at the tissue, cellular and molecular levels, including a significantly lower volumetric density of the perikarya of neurons in the supraoptic nucleus compared to animals not prone to alcohol dependence. to alcoholism[1].

The direct toxic effects of ethanol and its metabolites on neurons are due to the induction of glutamate neurotoxicity as a result of a decrease in neurofilament protein synthesis or impairment of fast axonal transport. Ethanol activates inhibitory GABA receptors and is an antagonist for glutamate (NMDA) receptors.

Chronic alcohol abuse leads to dysfunction of these neurotransmitter systems with a compensatory decrease in GABA activity and an increase in glutamate activity. Due to impaired ethanol metabolism, cytotoxic proteins are formed that reversibly damage cells of the nervous system; There is also a dose-dependent effect of ethanol on the severity of damage to the nervous system. The resulting cytotoxic proteins also affect other tissues[6,8].

Exposure to ethanol in the brain provokes the development of acute (or transient), delayed and chronic reactions of nerve cells. An acute reaction that develops during ethanol poisoning is expressed edema-swelling of neurons and is a consequence of a combination of altering factors: the toxic effects of ethanol and

hyperproduction of catecholamines, the excessive release of which causes hyperpolarization of neurons in the anterior limbic cortex. The acute reaction is more pronounced in the dopaminergic layer III of the anterior limbic checal cortex than in layer V [11].

Alcoholism in humans and chronic alcohol intoxication in animals has a distinct morphological expression in the brain. In this case, alcohol primarily affects blood vessels, especially the microvasculature, which leads to increased permeability of both the vessels themselves and the blood-brain barrier. As a result, ethanol and its metabolites easily penetrate the blood-brain barrier and cause severe damage to neurons, including their death. This is also facilitated by hypoxia associated with hemodynamic disorders[16].

An analysis of the current state of the problem of studying thanatogenesis during intoxication and alcohol poisoning was carried out, highlighting structural changes in the brain. It is emphasized that in the structures of the brain during alcohol intoxication and poisoning, changes in the cerebral vascular system and nervous elements are observed, which depend on the concentration of ethanol in the blood and tissues, the duration of its use, competing conditions (disease, injury, etc.). The severity of pericellular and perivascular edema in different parts of the brain shows the role of vascular and nervous structures in thanatogenesis during blood loss due to alcoholia[7].

R. Emsley et al. found pronounced brain atrophy with predominant damage to subcortical structures in alcoholic patients with Korsakoff syndrome. The authors associate this fact with nutrition-dependent diencephalic pathology, but not with the neurotoxic effect of alcohol on the cerebral cortex[21].

Thus, our analysis of data from domestic and foreign literature shows that the range of effects of ethanol on the central nervous system is quite wide, characterized by an increase in the negative effects of ethanol over time, ranging from minimal changes in the microvasculature to extensive multiorgan pathology with irreversible changes.

References.

1.Гуров Д.Ю., Туманов В.П., и др. Морфологические изменения нейронов супраоптических ядер гипоталамуса крыс, предрасположенных к алкогольной зависимости // современные проблемы науки и образования. – 2019. – № 2. ;url: https://science-education.ru/ru/article/view?id=28750

2.Еникеев Д.А., Ряховский А.Е., Хисамов Э.Н., Куклин Д.С. Структурно -функциональные изменения головного мозга, легких и сердца крыс при отравлении угарным газом на фоне алкогольной интоксикации // Современные проблемы науки и образования. – 2021

3. Заиграев, Г. Г. Алкоголизм и пьянство в России. Пути выхода из кризисной ситуации [Текст] / Г. Г. Заиграев // Социс. – 2009. – № 8.

4.Зиматкин, С.М., Оганесян Н.А., Киселевский Ю.В. Ацетатзависимые механизмы толерантности к этанолу: монография. Гродно: ГрГМУ, 2010, 252с.

5.Зиматкин С. М. Окисление алкоголя в мозге. Гродно: Гродн. гос. мед. ун-т, 2006, 200 с.

6.Зиновьева О. Е., Ващенко Н. В., Мозговая О. Е., Янакаева Т. А., Емельянова А. Ю. Поражение нервной системы при алкогольной болезни // Неврология, нейропсихиатрия, психосоматика. 2019; 1: 84-87. [Zinov'yeva O. Ye., Vashchenko N. V., Mozgovaya O. Ye., Yanakayeva T. A., Yemel'yanova A. Yu. Damage to the nervous system in alcoholic disease // Nevrologiya, neyropsikhiatriya, psikhosomatika. 2019; 1: 84-87.]

7. Индиаминов, С., Якубов, М., & Бойманов, Ф. (2022). Изменения структур головного мозга при алкогольной интоксикации (современное состояние проблемы). Журнал вестник врача, 1(1), 66–70. извлечено от https://inlibrary.uz/index.php/doctors_herald/article/view/5395

8.Лелевич В. В., Лелевич С. В., Алкоголь и мозг (метаболические аспекты) : монография Виницкая. – Гродно : ГрГМУ, 2019. – 244 с

9.Майбогин А.М., Недзьведь М.К., Корнев Н.В. Морфологические изменения головного мозга при циррозе печени алкогольной и вирусной этиологии. Acta biomedica scientifica. 2022; 7(5-2): 122-130. doi: 10.29413/ABS.2022-7.5-2.13

10.Навроцкий, Б. А. Социальные, этические и клинические проблемы современной наркологии [Текст] / Б. А. Навроцкий, С. А. Вешнева, О. В. Поплавская // Биоэтика. – 2015. – № 16. – С. 43–47.

11.Панкрашова Е.Ю., Федоров А.В., Дробленков А.В. Реактивные изменения клеток лимбической коры мозга при отравлении

этанолом, алкогольной абстиненции и хронической алкогольной интоксикации у человека. Журнал анатомии и гистопатологии. 2020; 9(2): 66–75. doi: 10.18499/2225-7357-2020-9-2-66-75

12.Разводовский Ю. Е. Эпидемиология алкоголизма в Беларуси / Ю.Е. Разводовский. Гродно, 2004. 85 с.

13.Сирота, Н. А., Чистова Е. А., Суховерхова З. И., Васильченко О. Ю. Профилактика наркомании и алкоголизма в подростково-молодежной среде (Серия: государственная молодежная политика в Российской Федерации) [Текст] / Н. А. Сирота, Е. А. Чистова, З. И. Суховерхова, О. Ю. Васильченко// М.: МЭГ, 2008. – С. 524.

14.Тухсанова Н.Э. Действие этилового спирта на центральную нервную систему человека и животных/. Вестник ТМА № 5, 2022. Стр43-46

15.Шорманова Н.С. Шорманов С.В. Морфология головного мозга человека при хронической алкогольной болезни Современные тенденции развития науки и технологий 2016 • № 1-2 стр 134

16.Шорманов С.В.. Шорманова Н.С. Гистоморфометрическая характеристика головного мозга человека при острой алкогольной интоксикации // Суд,- мед.эксперт. - 2005. - Т.48, №2. - С. 13-16.

17.Ульянова, Л. И. Нарушение функции иммунной системы при острой алкогольной интоксикации и алкоголизме [Текст]: автореф. дис….. д-ра биол. наук. – М., – 2013. – С. 214 направлений [Заиграев Г. Г., 2009;Позднякова М. Е. и др., 2011; Навроцкий Б. А. и др., 2015]

18. Халютин Д.А., Соловьёва Т.С. и др.Морфологическая оценка повреждений головного мозга при остром отравлении этанолом и их коррекции пептидными препаратами в эксперименте//Токсикологический вестник №3. 2016;(3):15-21. https://doi.org/10.36946/0869-7922-2016-3-15-21

19.Cananzi S., Mayhan W.Constrictor responses of cerebral resistance arterioles in male and female rats exposed to prenatal alcohol//2019.Physiological Reports, 9, e15079. https://doi.org/10.14814/phy2.15079

20.Fillmore, M.T. Response inhibition under alcohol: effects of cognitive and motivational control / M.T. Fillmore, M. Vogel-Sprott // J Stud Alcohol. – 2000. – Vol.61, №2. – P. 239–246.

21.Emsley R., Smith R., Robert M. et al. Magnetic resonance imaging in alcoholics Kosakoff's syndrome: evidence for an association with alcoholic dementia // Alcohol and Alcohol. 1997. Vol. 32. № 5. P. 479–486.

22.Gill, J.S. Reported levels of alcohol consumption and binge drinking within the UK undergraduate student population over the last 25 years / J.S. Gill // Alcohol and Alcoholism. – 2002. – Vol. 37, № 2. – P. 109–120.

23. Haorah, J. Reduction of brain mitochondrial βoxidation impairs complex I and V in chronic alcohol intake: the underlying mechanism for neurodegeneration / J. Haorah, T. J. Rump, H. Xiong. // PLoS One. – 2013. – Vol. 8 (8). – P. 8–85.

24.Igit T, Colcimen N. Stereological examination of effects of ethanol on optic nerve in xperimental alcohol model//,2019 May;38(5):610-615. doi: 10.1177/0960327119828123. Epub 2019 Feb 11.

25.Keith L., Crabbe J., Robertson L., Young E. Ethanol dependence and the pituitaryad renal axis in mice. II. Temporal analysis of dependence and withdrawal / // Life Sci.– 2014. – Vol. 33, № 19. – P. 1889-1897.

26.Laas R. Neuropathologyc of chronic alcoholism / R. Laas, C. Hagel // Clin. Neuropathol . – 2009. –Vol.19. – P. 252-253.

27.Tuksanova N. E. The Effect of Alcohol on the Structures and Vessels of the Brain. International Journal of Health Systems and Medical Science ISSN: 2833-7433Volume 1 | No 5 | Nov-2022.

УДК 616.127

THE ROLE OF HYPOXIA IN THE FORMATION OF FUNCTIONAL MYOCARDIAL DISORDERS IN NEONATAL CEREBROCARDIAL SYNDROME

[1]Tyagusheva E.N. ***https://orcid.org/0000-0002-1193-3178***
[1]Naumenko E.I. ***https://orcid.org/0000-0002-5332-8240***
[1]Vlasova T.I. ***https://orcid.org/0000-0002-2624-6450***

[1]Federal State Budgetary Educational Institution of Higher Education «National Research Ogarev Mordovia State University», Medical Institute, Russia

Resume Cerebrocardial syndrome is a disease characterized by a combination of CNS lesions and cardiac abnormalities, which is based on a violation of the central autonomic regulation of cardiac activity in perinatal hypoxic-ischemic CNS lesion. In this article, the main goal was to determine the effect of hypoxia in the formation of functional disorders of the myocardium in cerebrocardial syndrome of newborn children. To achieve the goal, 90 medical histories of premature newborns were analyzed. According to the results of laboratory and instrumental studies,