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Modern Approaches to the Problem of Chronic Alcoholism

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ABSTRACT

Objective: *The aim of this study is to investigate the long-term effects of ethanol intoxication on various body organs and systems, particularly focusing on the progression from minimal vascular damage to multi-organ pathology, as well as the development of specific clinical and psychopathological symptoms associated with alcoholism.* ***Methods:*** *This research employs a comprehensive review and analysis of existing studies on alcohol disease, examining its impact on microcirculation, organ morphology, and functional alterations caused by repeated ethanol exposure. Additionally, clinical data on the psychopathological symptoms exhibited by individuals with alcohol-related diseases are analyzed.* ***Results:*** *The findings highlight that chronic ethanol intoxication leads to significant morphological and functional changes in body organs, particularly affecting the vascular system and microcirculation. Over time, these alterations progress into multi-organ pathology, with clinical manifestations that include various psychopathological symptoms.* ***Novelty:*** *This study provides new insights into the specific mechanisms by which long-term ethanol exposure induces multi-organ pathology and psychopathological disorders. It emphasizes the gradual progression of alcohol disease, contributing to a deeper understanding of its pathophysiology and potential clinical interventions.*

INTRODUCTION

According to the latest data from scientific research, there is no dose of alcohol, not to mention harmless, "useful". Regular intake of a minimum dose of alcohol not only destroys the liver, but also contributes to the development of cancer, diabetes, hypertension and other deadly diseases.

In addition to harm to health, excessive alcohol consumption also has negative social consequences. Every third crime under investigation is committed by persons who are in a state of intoxication. Alcohol consumption significantly increases the risk of becoming a victim of crimes such as murder, beatings, sexual assault, theft. Increased mortality rates, reduced life expectancy, decreased labor capacity, decreased labor productivity, treatment costs for alcohol-related illnesses, social payments, damage caused by fires, traffic accidents, prisoner maintenance and crime and the cost of combating homelessness alcohol.

Holding alcohol-abusing employees is also a major problem for enterprise executives, significantly increasing the costs of the enterprise. These are marriage in production, violation of discipline, delays, exits, frequent illness, premature loss of working capacity, damage in production and, as a result, a decrease in the reputation of the entire enterprise.

To understand that a person is chronically abusing alcohol, it allows us to identify transferrin (or CDT)-a blood protein involved in the transport of iron, which is a carbohydrate deficiency in his blood. It typically contains four to eight attached sialic acid residues. This is a "lack", i.e. with little or no residues of sialic acids, it occurs with specific alcoholic damage to the liver, or rather with regular consumption of more than 60 grams of ethanol (2-3 bottles of beer or 0.75 liters of wine per day) for 7-14 days. Two weeks after stopping drinking alcohol, the figure normalizes.

The clinical picture of chronic alcohol poisoning includes damage to the hepatobiliary, cardiovascular and central nervous systems. Alcohol withdrawal syndrome with a symptom complex of somatic, neurological and psychopathological diseases is the main one in patients with alcoholism, which occurs as a result of a sudden cessation of alcohol consumption or a reduction in its dose in the context of complications related to alcohol abuse, while the development of liver and heart pathology continues during the removal period [1], [2].

RESEARCH METHOD

To select the treatment regimen and sequential stages of chronic alcoholism and alcohol withdrawal symptoms, it is necessary to understand the pathogenesis of the disease and the principles of action of the drugs used, as well as the stages of pharmacotherapy [1].

Pathogenesis of chronic disease-alcohol dependence in the limbic structures of the brain-dopamine and catecholamine systems, as well as endogenous opioid, GABAergic, n-methyl-D-aspartate (NMDA) systems are actively involved in the pathogenesis of chronic alcohol dependence. The inhibitory effect of ethanol on the central nervous system can be achieved with gamma-aminobutyric acid (GABA). According to modern scientific research, ionotropic gamma-Amine-oil A (GABA) and gamma-Amine-oil C, as well as metabotropic gamma-Amine-oil B-receptors, have been identified.

Ligand-dependent ionized GABA channel stimulation can induce membrane hyperpolarization by enhancing the flow of ionized chlorine particles, with a general deceleration process of neurotransmission expressed in anticonvulsant, sedative, and ank - siolytic events. The effects under consideration can be caused by many pharmaceutical agents and others that are relatively active to GABA receptors that partially activate benzodiazepines.

The purpose of this study was to determine the decrease in the toxic effect of deprimating agents on the action of antioxidants in the treatment of chronic alcoholism (in an experiment), and its tasks were as follows:

1. Describe the pathogenesis of chronic alcoholism.
2. Consider the effects of chronic alcoholism on the condition of organs and systems.
3. The study of the effect of introducing antioxidants into schemes of application with deprimating agents in the treatment of chronic alcoholism in accordance with the

idea of reducing the toxic effects of deprimating substances when used in combination with a representative of the class of antioxidants.

4. Choose the most important scientific research on the use of antioxidants in chronic alcoholism treatment regimens.
5. Support in the literature the hypothesis of reducing the toxic effects of deprimating substances when used in conjunction with an antioxidant class representative.

Frequent, regular use of alcohol can activate the release of the neurotransmitter GABA and actively increase the expression of sensitive GABA receptors, which can lead to increased inhibitory neurotransmission. In addition, ethanol is able to effectively inhibit glycine Binding of the brain to N-methyl-D-aspartate receptors, which inevitably leads to the Prevention of the effects of glutamate, an effective neurotransmitter with the main trigger, on N-methyl-D-aspartate receptors. Chronic ethanol consumption can lead to inevitable dependence, which manifests itself in increasing its doses to maintain the initial psychotropic effects of ethanol [3].

RESULTS AND DISCUSSION

According to targeted statistical studies, every second Russian over the age of 18 has a higher risk of developing alcohol problems and alcoholic visceropathies [4]. Of the population aged 15-49, about 10% of the world's deaths are related to alcohol-related diseases [5].

A. The Effect of Alcohol on the Cardiovascular System

With alcohol abuse, the cardiovascular system is often affected, alcoholic cardiomyopathy appears. Alcohol abuse contributes to the development of coronary artery disease and arterial hypertension – the main diseases that in 80% of cases lead to the development of chronic heart failure [6], [7]. Alcohol factor contributes significantly negatively to death from diseases of the circulatory system, including cerebrovascular and all forms of coronary artery disease [8]. Direct toxic effects of ethanol and acetaldehyde, impaired conjugation between contraction and excitation, free radical injury, impaired lipid metabolism, catecholamines, and ion balance are the main mechanisms of cardiomyopathogenic action. All of the above leads to the development of heart failure, which is characterized by a violation of the structure of the contractile apparatus of myocardial cells and their functional asymmetry. In microscopy, this is vacuolization of the cytoplasm of cardiomyocytes and small droplet fat dystrophy, accumulation of lipids in the myocardial stroma, around intramural vessels, pigment grains are detected throughout the cytoplasm, etc.with macroscopy-the expansion of all the cavities of the heart, the myocardium acquires a yellowish color. In this case, the coronary arteries often remain intact. The main mechanism of death development is the electrical instability of the myocardium, which leads to ventricular fibrillation [9], [10].

B. The Effect of Alcohol on the Central Nervous System

Direct toxic effects of ethanol and its metabolites on neurons are associated with decreased neurofilament protein synthesis or the appearance of glutamate neurotoxicity

as a result of impaired rapid axonal transport. Ethanol activates inhibitory GABA receptors and is an antagonist for glutamate receptors (NMDA). Chronic alcohol abuse leads to dysfunction of these neurotransmitter systems with a compensatory decrease in GABA activity and an increase in glutamate. Impaired ethanol metabolism results in cytotoxic proteins, which have the opposite effect on the cells of the nervous system; there is also a dose-dependent effect of ethanol on the severity of damage to the nervous system. The cytotoxic proteins obtained also affect other tissues [11].

C. The Effect of Alcohol on the Kidneys

Alcohol consumption affects the kidneys, which can manifest clinically with necronephrosis, glomerulonephritis and pyelonephritis. Alcoholic necronephrosis is manifested by small proteinuria, micro or macro hematuria. The changes are due to the direct toxic effect of high concentrations of ethanol and acetaldehyde on kidney tissue, as well as impaired microcirculation.

The renal tubules are affected, the glomerular apparatus remains unchanged. Dystrophic changes occur in the epithelium of the tubules until the development of necrosis, epithelial cells fall into the lumen of the tubules. Another variant of alcoholic toxic nephropathy, manifested by acute renal failure, is possible.

Alcoholic glomerulonephritis develops with chronic alcoholic intoxication in combination with alcoholic liver damage. In this case, the kidneys are damaged by immune complexes. Their basis is the" antigen – antibody " complex, which accumulates in the glomerulus and disrupts their function. Antigen is an alcoholic hyalin of the liver. Also, ida and/or IDM immunoglobulins and a fraction of the complement C3 are found in the glomeruli in the immune complex (IQ). IQ accumulates in the mesangia in a subendothelial or subepithelial manner. Clinically, it manifests as a hematuric form of glomerulonephritis.

In urinary tract infection, pyelonephritis develops in connection with the inhibition of immune defenses by the action of ethanol [12].

D. The Effect of Alcohol on the Liver

Currently, alcohol abuse and its surrogates are etiological factors for toxic liver damage. 60-100% of alcoholics and almost every person with alcoholism develops alcoholic liver disease (ABP) [13], [14], [15], [16].

ABP can manifest itself with several options – steatosis, alcoholic hepatitis (steatohepatitis), fibrosis and liver cirrhosis. The effect of ethanol on the liver can be direct and indirect.

The direct harmful effects of ethanol are the destruction of mitochondrial membranes and the development of liver fat dystrophy. The action of ethanol on the cell wall of hepatocytes leads to an increase in membrane permeability, disruption of transmembrane transport, the functioning of cellular receptors and membrane-bound enzymes, necrosis of liver cells.

Indirect hepatotoxic effects of ethanol are achieved by acetaldehyde accumulation within hepatocytes and enhanced lipid peroxidation (Pol).

E. Floor Products

1. Stimulate collagen synthesis and the development of liver fibrosis (through the development of hypoxia);
2. Enhance the direct action of ethanol on phospholipids of hepatocyte membranes and the production of anti – inflammatory cytokines-interleukins-1 and 6 (il-1, il-6), tumor necrosis factor alpha (tnf-a;
3. Activate complement components;
4. Causes inflammatory reactions in the liver;
5. Reduce glutathione levels and detoxification potential of liver cells by stimulating tissue damage [17], [18].

F. Stages of Treatment

The main factors that contribute to improving the effectiveness of treatment are listed below:

1. The direction of patients to completely abandon alcohol;
2. Continuity of the therapeutic process;
3. Differentiated but at the same time generalized approach to the application of one or another mode of treatment of chronic alcoholism;
4. Complexity of using different treatment methods;
5. Step-by-step and interrelationship of treatment measures [19].

The initial stage of the therapeutic treatment regimen for patients suffering from chronic alcohol intoxication syndrome includes targeted actions aimed at stopping alcohol withdrawal and alcohol withdrawal symptoms manifested by mental and somatovegetative symptoms 12-24 hours after alcohol withdrawal. In this case, detoxification therapy, nootropics, replacement therapy, psychotropic drugs are used [20], [21].

The most promising form of therapy at the stage of forming remission is the activation of treatment for the manifestation of pathological attraction to alcohol. At this stage, drugs are used, the effect of which is aimed at the development of alcohol aversion at the body level and the constant suppression of pathological desire for alcohol. It can be drugs that hate alcohol; able to reduce alcohol cravings; used together to treat depression. The final stage of treatment involves the use of antidepressant support doses, nootropics, vitamins, cerebroactive vasodilators, support doses of antipsychotics, adaptogens, and psychotherapy [22], [23], [24].

The main complication of chronic alcohol poisoning is damage to the cardiovascular system, which is due to a rapid, sharp increase in the level of catecholamines and, as a result, toxic effects on the heart muscle. Ka concentrated in the extraneyronal spaces of the heart can also cause the accumulation of biogenic amines in myocardial cells. This leads to instantaneous focal changes in the myocardium, with decreased contractility, arrhythmia, fibrillation, acute heart failure, and often serves as the leading cause of sudden death [25], [26], [27].

About 10% of patients in pharmacological therapy with neuroleptics or tranquilizers die each year due to Myo - card infarction and associated complications. The optimal explanation for this fact is the cardiotoxicity of the drugs in question, especially when prescribing high doses. Scientific studies have shown that in combination with deprimating agents (phenazepam, droperidol), ethyl methylhydroxypyridine succinate, increased sensitivity to stimuli presented in laboratory animals exposed to chronic alcohol poisoning and decreased aggressive preparation.unlike the group of animals where only deprimating agents are given [28], [29], [30], [31].

Histological examination of the myocardium of rats given Droperidol revealed edema, dystrophic degenerative changes, atrophy, hypertrophy of muscle tissue. And already in the myocardial microscope of animals, after pre - poisoning of alcohol and taking droperidol in combination with ethyl methyl hydroxypyride-succinate, the most noticeable decrease in ischemic zones in the heart muscle can be observed [32], [33], [34]. Drugs used for psychotropic effects are included in the treatment process at absolutely all stages of therapeutic prophylaxis of chronic alcohol dependence, therefore they have the most important effect on the body. At the same time, the effect of psychotropic drugs will directly depend on biorhythms in the case of chronic alcoholic intoxication [35], [36].

An important factor in the use of deprimation therapy is that not all patients tolerate this treatment regimen satisfactorily with the drugs in question. The dependence of the effect of psychotropic drugs on the biorhythms of the human body against the background of chronic alcoholism is such that you need to take into account the time of taking the drug, the degree of change in biorhythms against the background of alcohol poisoning and the effect of these changes on the action of used drugs, as well as their for successful treatment, a number of drugs are used that reduce alcohol cravings, block strengthening mechanisms, and eliminate euphoria due to alcohol consumption [37], [38], [39].

CONCLUSION

Fundamental Finding : The research highlights the importance of antioxidants in minimizing the detrimental effects of antipsychotics and tranquilizers on the myocardial structure. It also emphasizes the role of chronophysiological aspects in pathological processes, suggesting a new approach to cardiovascular therapy. This new paradigm could be crucial for treating alcohol withdrawal syndrome effectively. **Implication :** The findings imply that combining antioxidants with antipsychotics and tranquilizers may enhance therapeutic outcomes in cardiovascular protection. This approach could lead to improved treatment protocols for patients undergoing alcohol withdrawal, potentially reducing the side effects on the heart. **Limitation :** However, the study's limitations include the need for further exploration into the long-term effects of antioxidant use alongside these medications. There is also a lack of comprehensive clinical trials supporting the proposed therapy for alcohol withdrawal syndrome. **Future Research :**

Future studies should focus on large-scale clinical trials to confirm the efficacy and safety of combining antioxidants with antipsychotic treatments. Additionally, exploring the optimal timing and dosage for this combined therapy, particularly during sleep or within 22-24 hours, would be crucial for refining treatment strategies.

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