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Clinical Pictures of Cognitive Dysfunctions Observed in Alcoholism

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ABSTRACT

Objective: *To explore the complications of the nervous system associated with alcoholism and address the gaps in understanding the pathogenesis and effects of ethanol on neural and muscular systems.* ***Methods:*** *A comprehensive review of historical and recent studies on alcoholic lesions of the nervous system was conducted. This involved examining the extent of damage caused by ethanol exposure across various nervous system components and muscles, as well as analyzing the diverse pathogenetic mechanisms underlying these toxic effects.* ***Results:*** *Findings indicate that ethanol exposure results in widespread damage to nearly all parts of the nervous system and muscular structures. The toxic effects are attributed to a variety of pathogenetic mechanisms, which collectively contribute to the complex clinical manifestations of alcohol-induced neurological disorders.* ***Novelty:*** *This study underscores the multifaceted impact of ethanol toxicity, emphasizing the need for a deeper understanding of its pathogenetic mechanisms. It highlights areas where current knowledge is insufficient, paving the way for future research to better address and manage alcohol-related neurological complications.*

INTRODUCTION

Alcohol is the most common exogenous toxin that causes encephalopathy. At the same time, progressive impairment of intellectual functions is a characteristic feature of alcoholism. Cognitive disorders in people with chronic alcoholism are detected in 60-80% of cases, in 12% of cases they have a clear character that reaches the level of dementia. Alcoholism-related dementia accounts for 5-10 percent of all dementia cases, especially in young people [1], [2], [3].

There are several pathogenetic mechanisms that can cause cognitive impairment in patients with alcoholism. The cause of cognitive disorders in alcohol abusers, in addition to the toxic effects of alcohol and Wernick-Korsakov encephalopathy (based on thiamine deficiency), other deficiency diseases (in particular pellagra caused by a lack of nicotinic acid), Markiafava-Binyami disease, toxic leukoencephalopathy, as well as hepatic encephalopathy, recurrent cranial diseases - brain damage, meningitis, Sleep Apnea Syndrome, Alzheimer's disease or vascular dementia associated with alcoholism [4], [5], [6], [7]. Also, vitamin B_{12} deficiency can be noted for several possible reasons – the presence of chronic gastritis, folate deficiency due to malabsorption, malnutrition, pancreatic pathology and the direct negative effect of alcohol on the absorption of vitamin B_{12} in the intestine. It is interesting to note that not all alcoholic beverages are associated with a risk of dementia: drinking beer increases the risk of dementia (Alzheimer's disease, vascular dementia, non-delicate dementia), and drinking red wine

reduces this risk. The authors interpret the findings very carefully and note that they only testify to the possible protective effect of flavonoids in red wine on oxidative stress, which is important in the pathogenesis of dementia of various origins. However, the protective effect of alcohol in terms of reducing the risk of dementia is not confirmed by all authors [8], [9], [10], [11].

Alcohol dementia is characterized by a predominance of impaired executive function caused by damage to the previous parts of the brain. Among the clinical features of this condition, one should also mention visual-spatial and perceptual disorders, memory disorders for events in one's own life. Often cognitive disorders are combined with emotional and personal disorders, with nearly 80% of patients having different levels of Depression [12].

RESEARCH METHOD

One of the causes of cognitive impairment in alcoholism is a lack of nicotinic acid or niacin (the latter term has been proposed to highlight the difference between nicotinic acid and nicotinic alkaloid). Clinically, this condition is known as pellagra (ital, pelle agra-rough or rough skin). A lack of the niacin tryptophan precursor can also lead to pellagra. In addition to a lack of nicotinic acid, vitamin B6 deficiency can also play a role in pellagra pathogenesis [13].

In addition to diarrhea, patients may also report phenomena such as stomatitis, glossitis, abdominal pain, anemia, and anorexia. Skin manifestations of pellagra range from erythema to the development of reddish–brown areas of hyperkeratosis, most often on the face, chest and back surface of the arms and legs. It is noted that since pellagra dermatitis is photosensitive, it may not manifest itself if the patient is constantly in the room. In the early stages of the disease, patients may experience disorders of memory, attention, agitation or apathy, depression, which with the development of the process can be replaced by psychotic diseases with the appearance of hallucinations, delirium and coma. In addition, in the neurological case, diplopia, dysarthria, dysphagia, retrobulbar neuritis and other cranial nerve damage (peripheral paresis of pair VII, damage of pair VIII), Parkinsonism, tremor, ataxia, pyramidal symptomatology (Babinsky's positive reflex, spastic), myoclonia (mainly in the muscles of the face and upper extremities) and axial reflexes. Paratonia is very characteristic of encephalopathy in the pellagra, with minimal manifestations in the neck and maximum in the extremities [14], [15], [16], [17].

The condition of patients with alcoholism can be complicated when hepatic encephalopathy occurs. The course of hepatic encephalopathy, as well as other encephalopathies of dysmetabolic Genesis, oscillate in nature. Advanced stages of hepatic encephalopathy are characterized by the appearance of pronounced disorders of attention and memory, constructive apraxia, acalculia, psychomotor diseases, dysarthria and dysphasia, disorientation, confusion, behavior disorders, apathy, epileptic seizures, in unfavorable cases a coma condition may develop. The worsening condition of patients may be associated with prescribing drugs (tranquilizers, thiazide diuretics, barbiturates,

etc.) that aggravate hepatic encephalopathy. The possibility of extrapyramidal disorders distinguishes hepatic encephalopathy from other variants of dysmetabolic encephalopathies. It should be noted that at the time of examination, motor phenomena characteristic of hepatic encephalopathy (asterixis, myoclonia) may not be present in the patient [18], [19], [20], [21].

There are several hypotheses explaining the phenomenology of the neuropsychological defect in alcoholism: the "right cerebral hemisphere" hypothesis, the "diffuse brain defect" or "premature aging" hypothesis, and the "preferential damage to the anterior parts of the brain" hypothesis. The" right hemisphere" hypothesis is based on the assumption of superior damage to the right hemisphere of the brain when exposed to alcohol. The" diffuse brain defect "or" premature aging " hypothesis is based on the similarity of cognitive disorders in severe alcoholism to cognitive disorders in aging. This means that damage to both hemispheres of the brain is expressed at the same level. The basis of the hypothesis of" dominant damage to the anterior parts of the brain" is the fact that in chronic alcoholism, signs of damage to the anterior parts of the brain are known to prevail. This hypothesis has many clinical and paraclinic affirmations. At the same time, a significant role is played by the violation of the connections of the previous parts of the brain with other parts of the cortex and subcortical structures, which manifests itself.divorce syndrome [22], [23], [24], [25].

Violations of executive functions due to separation syndrome are also characteristic of patients with alcoholism, without pronounced Mnestic diseases. A frontal defect often identified in alcoholic dementia may precede clinical manifestations of alcoholic polyneuropathy and cerebellar degeneration, as well as Vernik-Korsakov syndrome. In alcoholism, frontal lobes are more susceptible to injury, which is manifested by impaired executive function (ability to abstract and plan, inhibition of perceverative processes, alternation between different cognitive processes, speed of cognitive processes, etc.). Speech and praxis, confabulation disorders can also be detected in patients with dementia [26], [27], [28].

It should be noted that the concept of alcoholic dementia (dementia caused by the toxic effects of alcohol) is not very perfect from a nosological point of view and is a subject of debate. It is suggested that it is one of the variants of Vernik-Korsakov encephalopathy. However, clinical manifestations of alcoholic dementia are not limited to a Mnestic defect and are therefore distinct from manifestations of korsakoff syndrome. In particular, Korsakov syndrome has not been characterized by syndromes such as thought disorders and visual-spatial function disorders leading to alcoholic dementia [29].

Attention should be paid to such specific losses. Likewise, as in road transport crimes, when we talk about the immediate victims of the accident and the people who were injured in them, we should talk about those whose alcohol played a fatal role in life, although it did not directly lead them to death. Injury from alcohol abuse is such that we should talk about individuals such as limited legal (capable) members of society.

In addition, there are several times more people who actually abuse alcohol. High latency is associated with social criticism, possible stigmatization by society, rejection of the problem and many other factors [30], [31].

Three main stages are distinguished in the pathogenesis of alcoholism: the stage of repeated acute alcohol intoxication; the stage of intoxication (chronic alcohol intoxication); alcoholism (addiction to alcohol, including removal and complications in the form of psychosis). At the same time, we are talking about two, often adjacent, ways to damage the systems of the human body associated with alcohol consumption – these are the direct toxic effects of alcohol and cases of deficiency [32].

As part of this work, we are more interested in the mechanism of direct toxic effects of alcohol on the body and the functioning of its individual systems.

RESULTS AND DISCUSSION

According to the tenth revised International Classification of diseases and health problems (ICD-10), the toxic effects of alcohols represent poisoning with methyl, isopropyl alcohol, fusel oils and other alcohols. Acute alcohol intoxication is more common in poisoning with ethyl alcohol, which is used orally to get drunk. The dose of ethanol, which is life-threatening, varies from 4 to 12 grams per kilogram of body weight. The dangerous concentration of ethanol in the blood, which can lead to alcohol coma, starts from 3 g/l, the concentration of 6 g/l leads to death [31, [32], [33], [34].

These indicators are not absolute and can vary depending on individual tolerance to alcohol. Ethyl alcohol is absorbed very quickly in the gastrointestinal tract (about 80% of resorption occurs in the small intestine). About ninety minutes after oral administration of alcohol, the maximum concentration of ethyl alcohol in the blood appears.

After the absorption phase is almost complete (about 90% of the alcohol is resorbed), the elimination (release) phase begins. The average metabolic rate is 90-120 mg / kg of body weight per hour (varies individually). When taken orally, 90% ethyl alcohol is oxidized through the liver via alcohol dehydrogenase to CO_2 (carbon dioxide) and H_2O (Water). Residual ethylene (10%) is excreted through the lungs and kidneys for an average of 10-12 hours. Alcohol elimination occurs with urine and exhaled air, and ethylene in urine is detected longer than in blood.

Ethyl alcohol has an addictive effect on the central nervous system. Such an mechanism of action is associated with a decrease in the use of oxygen, a violation of the mediator systems, a change in the metabolism of brain cells, which leads to a weakening of excitation processes. The intensity of drug intoxication depends on individual tolerance, the concentration of alcohol in the blood at the resorption stage.

Hypoxia from the respiratory system is possible when taking alcohol, which causes a violation of homeostasis (acid-based condition, water-electrolyte balance).

From the point of view of hemodynamics, vascular tone disorders are possible, hypovolemia, microcirculation disorders due to acidosis, hypercoagulation, hypothermia, alcoholic cardiomyopathy are somewhat less common.

At the somatogenic stage of alcohol disease, we are talking about residual pathological diseases, damage to internal organs, infections that lead to impaired vital functions. Cognitive impairment of alcoholics is very diverse in terms of the type and extent of injury. Cognitive functions are usually understood to be the most complex functions of the brain, with which the process of rational knowledge of the world is carried out. Cognitive functions include thinking, memory, speech, perception, imagination.

In contrast to the concept of" cognitive function", it is recommended to use the concepts of" cognitive dysfunction "or"cognitive impairment". According to generally accepted terminology, cognitive dysfunctions in clinical psychology should be understood as a decrease in memory, mental performance, attention; speech disorders, perception and Baseline (individual norm).

From the point of view of nosology, the causes of cognitive disorders can be vascular diseases of the brain, dysmetabolic encephalopathies, neurodegerative diseases, neuroinfections, brain damage and tumors, among others.in this regard, poisoning and intoxication stand out a little, especially when it comes to alcohol poisoning. This is primarily due to the presence, discretion and legality of alcohol consumption, and it is impossible to talk about the same drugs and Psychotropic Substances (their consumption is actively condemned by society and stopped by the Legislature).

Cognitive dysfunctions in alcoholism are based on the direct toxic effects of ethanol and its metabolites on the central nervous system. "The effects on neurons are associated with decreased neurofilament protein production 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 (n-methyl - D-aspartic acid-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 that have the opposite effect on the cells of the nervous system; the dose-dependent effect of ethanol has been found to be the severity of nervous system damage" [24], [25], [26].

Damage to the central nervous system (CNS) is a mandatory sign of alcoholism and develops to one degree or another in all people with alcohol abuse (from mild reversible cognitive impairment to severe dementia).

Alcohol is one of the cheapest exogenous toxins that can lead to progressive impairment of intellectual functions that lead to encephalopathy. More than half (50-70 %) of alcoholics have cognitive disorders, in 10% of cases we are talking about acquired dementia (dementia). Every tenth case of dementia is alcoholic dementia. In 70% of alcoholics, diffuse brain atrophy is observed (about a third of them with damage to the frontal lobes) [27], [28], [29], [30].

It is not uncommon for alcohol withdrawal and therapy to lead to a recovery of cognitive impairment. Neuropsychological defects are explained from three perspectives:

a. "Right brain" hypothesis (the right hemisphere is primarily affected by alcohol consumption);
b. Hypothesis of" diffuse brain defect "or" premature aging " (similarity of cognitive lesions in severe alcoholism with aging - both hemispheres suffer equally);
c. Hypothesis of" preferential damage to the anterior parts of the brain" (predominance of signs of damage to the frontal parts of the brain in chronic alcoholism, separation syndrome-a violation of the connections of the anterior parts of the brain with other parts of the cortex and subcortical structures).

We have summarized the main cognitive disorders that occur with alcoholism, direct causes and their symptoms. Often, practice suffers as cognitive dysfunction in alcoholism. With alcohol abuse, almost always the ability to perform certain targeted actions suffers. Cognitive dysfunctions associated with attention dysfunction are also characteristic of many diagnoses and syndromes associated with alcoholism. Cognitive disorders in the form of memory disorders are characteristic of diagnoses such as encephalopathies (Vernike - Korsakov, liver, toxic), pellagra, Korsakov psychosis, alcoholic dementia, as well as severe cases of Alzheimer's disease, vascular dementia and vitamin B_{12} deficiency.

Gnosis as a cognitive function closely related to memory is more likely to suffer from pellagra, Markiafava - Binyami disease, toxic and chronic encephalopathy, Alzheimer's disease, separation syndrome and B_{12} deficiency. Speech disorders as cognitive dysfunction should be more associated with vascular and alcoholic dementia, Alzheimer's disease, toxic and hepatic encephalopathy [31], [32], [33].

It is necessary to talk about a wide range of cognitive disorders that occur as a result of alcohol abuse. In any case, even a slight exposure to alcohol negatively affects all systems of the human body. With alcoholism, we are talking about the chronic, constant excessive influence of alcohol on the body, which causes mental and physical dependence, which negatively affects the state of a person in society, his competence, position in society. Such a change in personality is primarily based on cognitive disorders that occur with alcohol illness, which we have learned within the framework of this work [34], [35], [36], [37].

CONCLUSION

Fundamental Finding : The study reveals that alcoholism leads to significant cognitive disorders, affecting both the individual and society. People suffering from alcohol addiction not only face physical impairments but also social and psychological challenges that render them incapable of participating as conscious and responsible members of society. **Implication :** The findings suggest that addressing alcoholism is crucial for the well-being of individuals and society. Alcohol abuse impairs cognitive

function, resulting in behavioral issues like aggression, autoaggression, and crime, highlighting the need for intervention and prevention strategies to mitigate these societal dangers. **Limitation :** The study is limited by the focus on cognitive disorders without exploring other potential health consequences of alcoholism, such as its impact on physical health, economic stability, and family dynamics. Further research is required to examine these aspects in greater detail. **Future Research :** Future research should explore the broader social, economic, and physical implications of alcoholism, as well as effective treatment methods and preventive strategies. Longitudinal studies on the cognitive recovery of alcoholics after intervention would provide more insight into potential rehabilitation pathways.

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