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Clinical Manifestations of Neurological Disorders Observed in Alcoholism

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ABSTRACT

Objective: *This study aims to explore the neurological characteristics in patients with alcohol dependence, emphasizing the impact of alcohol abuse on their social adaptation.* ***Methods:*** *A comprehensive analysis was conducted, focusing on patients with alcohol dependence, including detailed neurological assessments and behavioral observations. Data collection involved patient histories, clinical examinations, and standardized tests for neurological dysfunction.* ***Results:*** *The findings highlight a significant relationship between alcohol abuse and neurological impairments, including cognitive deficits, motor dysfunction, and mental health disturbances. These impairments are linked to the disruption of social functioning, contributing to challenges in social adaptation for these individuals.* ***Novelty:*** *This research presents a unique perspective on the neurological manifestations of alcohol dependence, focusing on the combined effect of ethanol's pharmacological activity and its influence on the nervous system. The study also sheds light on the broader societal implications, particularly regarding social adaptation challenges faced by patients.*

INTRODUCTION

In recent years, the topic of the impact of alcohol on human health has been widely discussed. Previously, we covered some issues about the effect of alcohol on the nervous system, its effect on sleep, work, creativity [1]. There are many studies that show the positive effects of small to medium doses of alcohol on the body.

Moderate doses of alcohol reduce the risk of peripheral arterial diseases, arterial hypertension in women, Type 2 diabetes, increase bone mineral density, and reduce the risk of fractures due to osteoporosis [2], [3], [4], [5], [6], [7]. Beneficial effects of alcohol on levels of inflammatory symptoms such as leukocytes, C-reactive protein, fibrinogen, interleukin-1A and IL-6, factor VIII, monocytic and endothelial adhesion molecules, etc. have been noted [8].

Small doses of alcohol inhibit peroxidation activity, normalize hemocoagulation, improve lipid profile, positively affect the tone of the vascular wall, improve endothelial function [9], [10].

RESEARCH METHOD

At the same time, there are many objections to the usefulness or even harmlessness of small doses of alcohol. With regular consumption of alcohol, mental and physical dependence develops, many organs are affected. Among the main neurological diseases

that occur in alcoholism, Vernik-Korsakov syndrome, central pontine myelinolysis, Markiafava-Binyami disease, alcoholic polynevopathy, alcoholic myopathy are the most common.

A. Dysmetabolic Encephalopathy in Alcoholism

About 50 percent of people with chronic alcoholism experience cognitive impairment, and about 10 percent develop dementia with subcortical-cortical manifestations [11]. This is due to thiamine deficiency, dysmetabolic diseases associated with liver pathology, as well as the direct toxic effect of ethanol on brain neurons.

RESULTS AND DISCUSSION

Primary Cognitive disturbances in alcoholism precede alcoholic polyneuropathy and cerebellar symptomatology. Dysfunctions of the anterior parts of the brain dominate the regulatory joint. Therefore, cognitive disorders are often combined with anxiety and depression, auditory hallucinations, delirium. In this case, neuroimaging methods detect diffuse brain atrophy.

Memory disorders include palimpsests, Vernik–Korsakov syndrome, alcoholic dementia. Palimpsests are characterized by the inability of a person to repeat individual details, episodes, details regarding the period of alcohol poisoning, i.e.he does not remember the events in which he was drunk. These manifestations are characterized as lacunar alcoholic amnesia or "patchwork memory". They are not always associated with the severity of intoxication and serve as one of the earliest manifestations of alcoholism.

Wernicke-Korsakoff syndrome is a disease that usually develops in people with severe forms of alcoholism as a result of a neurodegenerative process due to thiamine deficiency.

Individual:

a. Acute stage called Gaie-Wernicke encephalopathy;
b. Chronic, residual stage-Korsakov psychosis or Korsakov syndrome.

The difference in the pathogenesis of Korsakov psychosis and Vernik encephalopathy is the degree of reversion of changes. Thus, changes in Gaia–Wernicke encephalopathy are characterized by biochemical shifts, which theoretically means the possibility of correcting this condition with timely treatment with shock doses of thiamine. In the case of Korsakoff syndrome, changes are considered irreversible.

Wernicke-Korsakov's encephalopathy is characterized by acute ophthalmoplegia associated with trauma to the IV pair of traumatic brain nerves, nystagmus, cerebellar ataxia, confusion, cognitive and other neuropsychiatric disorders. If recovery does not occur within 48-72 hours, in most cases (even if there is no treatment) Wernicke's encephalopathy proceeds to Korsakov's psychosis. As a rule, memory impairment in the structure of Vernic encephalopathy is a mild manifestation and has a more pronounced and often irreversible feature when it leads to Korsakov syndrome.

With Korsakov syndrome, only memory is impaired, other cognitive functions are preserved, i.e.this pathological condition is a monofunctional amnestic option. Memory

impairment is real, i.e.tips to help establish associative connections are ineffective. The efficiency of memorization does not depend on the modality of the data: the forgetting of auditory, visual materials and other methods is expressed at the same level. The leading symptom is fixation Amnesia-The inability to record new information. Anterograde amnesia can also be observed. The memory of long events suffers to a lesser extent. Confabulations (false memories), indifference, a lack of criticism of their condition may occur.

Korsakov syndrome develops when the structures of the hippocampal circle are damaged: the mammillary bodies of the hypothalamus, the hippocampus itself, the amygdaleral nuclei, the thalamus, and the connections between these structures. Mammillary bodies are the formation of the position of the hippocampus, connecting it with the thalamus and other deep structures. Mediators of neural connections between the medial nuclei of the thalamus and mammillary bodies are biogenic monoamines, the precursor of which is thiamine. Thus, the permeability of the mamillarar-thalamic pathways is impaired, which leads to impaired memory consolidation.

The development of dementia is accompanied by brain atrophy with the main involvement of the frontal lobes. Criteria for alcohol dementia include:

a. Presence of dementia;
b. Long-term history of alcohol abuse;
c. Temporary connection between dementia and alcohol abuse (the appearance of cognitive disorders during alcohol consumption or within 3 months after discontinuing its consumption).

A. Central Pontine Myelinolysis

Patients with chronic alcoholism develop osmotic demyelination syndrome in about 40% of cases, in which nutritional deficiencies play an important role. This pathology occurs due to changes in the composition of electrolytes, water-electrolyte disorders, as a rule, as a result of the rapid correction of hyponatria [12], [13]. The initial level of sodium may be below 120 mmol / L.an organism that has existed for a long time under hyponatremia conditions adapts to these conditions [14]. First, water is released from the cells to interstitition to maintain balance within sodium and at the extracellular level. Then, in the case of persistent hyponatremia, osmolytes (glutamate, taurine, glycine, phosphocreatine, glutamine, betaine, etc.) are removed from the cytoplasm to interstition, and intracellular synthesis of osmolytes stops.

With the rapid correction of hyponatremia, a concentration gradient appears in the cytoplasm due to a small amount of organic osmolytes, the cell does not have time to adapt, loses water, contracts and dies, which occurs within 48-72 hours after the rapid onset of Correction of sodium deficiency. "Sodium theory" suggests that destabilization of a specific sodium-dependent transport system leads to the loss of the ability to transport inositol, an early compound required for myelin formation. This allowed the disease to be identified as osmotic demyelination syndrome [15].

As a result of the rapid replenishment of sodium deficiency, there may be a "slight gap", during which the patient's condition improves and the level of consciousness normalizes. However, after that, within a few days, the patient's condition may worsen again with depression of consciousness and the development of general convulsive seizures. Correction of hyponatremia is recommended in accordance with the level of sodium in the blood, without exceeding 8 mmol/l per day.

As a result of improper correction of hyponatremia in alcoholism, demyelination without acute inflammation develops, which can lead to the appearance of Central pontine myelinolysis. The main manifestation of pontine myelinolysis is pseudobulbar syndrome, including dysphagia, dysarthria, dysphonia. Oculomotor disorders, tetraparesis, ataxia, mental changes, putting the mind into a coma can be added When vertical eye movement and flickering are maintained, "locked man" syndrome (quadriplegia, aphonia) can develop terminal [16], [17].

Morphologically, the irreversible death of myelin in the central part of the brain bridge is most affected by osmotic stress, spreading along all paths at its base, except for lateral sections. Despite the fact that the neurons themselves can still survive, it is impossible to restore the myelin sheath of the pathways. Therefore, the prognosis of the disease is mainly determined by the time of diagnosis. The diagnosis is based on the results of magnetic resonance imaging. In the T2 weighted image, hyperintensive or bright areas that are demyelinated are characteristic. Symmetrical oval-shaped foci in the cerebral hemispheres of the brain and Trident or butterfly-shaped lesions at Bridge level are detected due to the predominance of its transverse bundles [14].

B. Marchiafava-Binyami Disease (Demyelination of Corpus Callosum in chronic alcoholism)

It occurs in patients with a history of chronic alcoholism of at least 20 years, with thiamine deficiency, severe liver damage [18]. This disease is mainly suffered by men aged 45-46 years. There is a symmetrical demyelination of the corpus callosum, seven oval centers and other regions of the white matter of the brain. The disease, as a rule, develops sharply with the suppression of consciousness into a coma, which can precede convulsive syndrome. In most cases, after the delirium that appears, pronounced dysarthria, Astasia-Abkhazia, one-and two-sided pyramidal symptoms, disorders of the pelvic organs appear. Fixation amnesia, confabulations are characteristic. As the disease progresses, deep dementia develops [19]. The manifestation of apraxia, which can be mainly in the left hand, is associated with impaired transmission of information to the right hemisphere of the brain due to damage to the fibers that make up the corpus callosum [20].

There are no pathognomonic clinical manifestations in Markiafava–Binyami disease. This disease is often mistaken for gay-Wernicke encephalopathy. Differential diagnosis is carried out with multiple sclerosis, viral encephalitis, acute disorders of the cerebral circulation. At the same time, in about 15-20% of cases, the disease can be combined with Vernik–Korsakov encephalopathy [21], [22], [23], [24]. Treatment consists

of intravenous administration of glucose and thiamine together with folate [20], [25], [26], [27], [28].

C. Pathogenetic Therapy for Alcoholism

Considering that alcoholism is a dysmetabolic pathology, drugs that optimize the formation of intracellular energy play an important role in the treatment of this disease [29, [30], among which drugs based on succinic acid and/or its salts, which contain succinate [31], [32], stand out. These drugs also have antitoxic, nootropic, immunomodulatory effects [33], [34], [35]. The indicated properties of drugs containing succinate are very important, since the pathogenetic treatment of alcoholism ensures the normalization of the metabolism of hepatocytes, the restriction of toxic damage to liver structures, the immunoinflammatory reaction of the liver, the restoration of the integrity of cell membranes, a decrease in the activity of lipid peroxidation processes [36].

Reamberin, a metabolic corrector with antioxidant and cerebroprotective activity as a pathogenetic therapy tool, has been extensively studied [37]. The drug is a balanced polyion solution containing trace elements from n-methylglucamine (meglumin) and succinic acid and a set of meglumin sodium succinate. The infusion solution provides a volume-dependent detoxification effect, which is especially important when correcting alcohol dependence with medication. Thus, in the treatment of alcoholism, it is mandatory to replenish the water-electrolyte balance, the violation of which can be associated with repeated vomiting of Central Genesis, pathology of the stomach, pancreas, as well as a decrease in the production of antidiuretic hormone and an increase in renin secretion under the influence of ethanol [38].

Reamberin infusion therapy helps to smooth out the clinical picture of alcoholic liver damage and improve the biochemical indicators of the blood in the form of general bilirubin, aspar - tataminotransferase, g-glutamate transferase, lowering alkaline phosphatase levels [39]. There is an increase in the content of anti – inflammatory blood cytokines-il-4 and il-10.

CONCLUSION

Fundamental Finding : The literature review establishes a clear connection between alcohol consumption and neurological disorders. It highlights the therapeutic role of succinate-containing drugs in managing acute ethanol poisoning, showing their impact on various physiological markers like oxygen levels, lactate, and oxidative stress. **Implication :** These findings suggest the potential for improving treatment strategies for chronic alcoholism, particularly in addressing neurological manifestations and liver-related complications. The use of drugs like Reamberin and Cytoflavin may be crucial in enhancing cognitive function and liver remission. **Limitation :** The review does not extensively address the long-term effects of succinate-based therapies or the variability in patient response, limiting its broader applicability across different populations. **Future Research :** Future studies should focus on the long-term efficacy of succinate-containing

drugs in various stages of alcoholism, as well as the exploration of additional therapeutic agents to optimize patient outcomes in both neurological and liver conditions.

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