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Clinical Course of Alcoholic Polyneuropathy and Alcoholic Myopathy Observed in Alcoholism

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ABSTRACT

Objective: *This article aims to explore the most prevalent neurological manifestations associated with alcoholism, including dysmetabolic encephalopathy, central pontine myelinolysis, demyelination of the corpus callosum, alcoholic polyneuropathy, and alcoholic myopathy.* ***Methods:*** *The study examines the potential therapeutic role of drugs containing succinate, focusing on their application in pathogenetic treatment approaches to address these neurological conditions.* ***Results:*** *Key findings indicate that succinate-containing drugs may offer significant benefits in mitigating the pathological effects of alcoholism on the nervous system by targeting specific mechanisms underlying these disorders.* ***Novelty:*** *The article provides novel insights into the therapeutic potential of succinate-based treatments, presenting an alternative strategy for addressing complex neurological disorders linked to chronic alcohol consumption.*

INTRODUCTION

The proportion of alcoholic polyneuropathy is about 36% among all polyneuropathies, which occurs in 9-76% of alcohol-dependent patients for more than 5 years [1]. With this disease, symmetrical damage is caused to the motor and sensory fibers of the peripheral nerves. First of all, axial cylinders of nerve fibers (axonopathy) suffer, then myelinopathy is added during Demi - lynization of nerve fibers [2].

Alcoholic polyneuropathy can have an acute, subacute and chronic course. Thiamine deficiency plays a key role in acute and subacute courses. Asymptomatic forms of the disease confirmed using electroneuromyography are also known in 97-100% of people with chronic alcoholism [3]. Alcohol plays a key role in the pathogenesis of polynev-Patia: 1) the direct toxic effect of ethanol and its metabolites on the fibers of the peripheral nervous system; 2) thiamine deficiency.

Ethanol affects neuronal metabolism by activating glutamate receptors in the spinal cord, leading to increased glutamate neurotoxicity, free radical lipid peroxidation processes, and increased production of anti-inflammatory cytokines [4]. Ethanol also reduces synthesis and disrupts the normal configuration of nerve fiber cytoskeletal proteins and slows axonal transport [5]. Experimental studies have found that ethanol activates spinal microglia cells, increasing the functional activity of the hypothalamic-pituitary adrenal and sympathoadrenal systems. These changes, together with alcoholic oxidative stress, play an important role in the formation of central sensitization in the

spinal cord and, consequently, in the development of neuropathic pain syndrome in alcoholic polyneuropathy [6].

Thiamine deficiency in patients with alcoholism is associated with a lack of its intake with food and a decrease in the absorption of thiamine in the small intestine during ethanol intake. Phosphorylation of thiamine and its active form is the formation of thiamine pyrophosphate, which is the coenzyme of the most important multicomponent enzyme complexes involved in carbohydrate metabolism (Krebs cycle, adenosine triphosphate formation), biosynthesis of certain components of the cell, components of an endogenous antioxidant system, synthesis of nucleic acid precursors in the pentose phosphate pathway, and NADPH – oxidases (NADPH-nicotinamide adenine dinucleotide phosphate). As a result, the addition of lipids to myelin is reduced, the biosynthesis and metabolism of neurotransmitters is disrupted, zones are formed in neurons with lactate-acidosis and intracellular accumulation of calcium, which enhance the neurotoxic effects of alcohol [7], [8], [9], [10], [11].

RESEARCH METHOD

Sensitive manifestations of polyneuropathy are characteristic of distal parts of the extremities such as burning, numbness, paresthesia, feeling cold, painful spasms of the lower leg muscles, pain, often in the lower extremities. The examination reveals the phenomena of hyperalgesia, hyperpathy, dysesthesia. Touching the skin dramatically increases pain (allodynia). The diagnosed sensitivity disorder (hypo - or hyperesthesia of pain and temperature sensitivity), as a rule, is symmetrical in the area of the palms and feet of the "glove" and "sock" type, and then to the proximal parts of the limbs – "high glove", "golf", "socks". Dissociated sensitivity may be impaired [12], [13], [14], [15], [16]. Subtle disorders are often combined with vegetative-vascular changes: disorders of pupil reactions, hyperhidrosis, acrocyanosis, Ebru, cyanosis, swelling and hyperpigmentation of the skin of the palms and feet, dystrophic changes of the nails. In this category of patients, menstrual irregularities and impotence are possible. Inhibition of the Tendon and periosteal reflexes is detected. In the pre-clinical stage, Achilles reflexes disappear first [17]. Sensitive ataxia develops when deep sensitivity fibers are involved. Atactic form of alcoholic polyneuropathy (peripheral pseudotabes) is also distinguished, in which violation of the Coordination of walking and movements is accompanied by drowsiness and hypesthesia of the distal extremities, the absence of Achilles and knee reflexes, pain during palpation in the area of the nerve trunks [18], [19], [20], [21].

The motor form of alcoholic polyneuropathy is characterized by the development of peripheral paresis of the lower extremities of various degrees. With the appearance of symmetrical lethargic paresis, mainly the tibial and peroneal nerves suffer. When the Tibial nerve is damaged, the plantar flexion of the feet and fingers is disturbed, the foot is turned inward, it is impossible to walk on the toes [22]. Damage to the Peroneal nerve is characterized by dysfunction of the extensors of the feet and fingers. The foot hangs and turns inward, the patient raises his feet high so as not to touch the ground with his

toes (peroneal walking). Hypotonus and hypotrophy or atrophy of the muscles of the legs and feet are observed in the form of a deposition of interosseous spaces - "clawed foot". Sometimes atrophy also spreads to the hips and muscles. Most often, hypermobility and deformation of the ankle joints are determined. In some cases, peripheral nerve damage with enmg is not detected, which does not indicate the absence of pathology. This is because ENMG data characterize the condition of thick myelinated fibers, and in the chronic toxic form of alcoholic polyneuropathy, mainly thin, weakly myelinated or myelinated fibers are affected, so in such cases, ENMG indicators remain within normal limits [23], [24], [25], [26].

Alcoholic myopathy. Acute and chronic forms of skeletal muscle injury are distinguished [26]. Acute alcoholic myopathy develops in 1-5% of cases and is manifested by weakness, acute pain and swelling of mainly proximal muscle groups, as well as a significant increase in the level of creatine phosphokinase in blood plasma, accompanied by myoglobinuria. A morphological examination reveals necrosis of different types of skeletal muscle fibers (rhabdomyolysis), so this clinical form of myopathy is interpreted as acute necrotic [27], [28], [29].

RESULTS AND DISCUSSION

Acute alcoholic myopathies also include acute hypokalemic myopathy, characterized by a general weakness of the muscles and a lack of muscle pain and swelling affected by the necrotic form. CPK levels also increase significantly, with blood plasma potassium levels decreasing to 1,4–2,1 mmol/l (norm 3,6–6,3 mmol/l). Morphological examination reveals necrosis of individual muscle fibers [30].

Chronic alcoholic myopathy occurs in 40-60% of patients with chronic alcoholism [31]. It is characterized by proximal paraparesis, hypotrophy and painful muscle cramps of the lower extremities. With this form of skeletal muscle damage, the level of CPK in the blood does not increase.

For a long time, it was believed that the development of muscle weakness in such patients is associated with denervative changes in muscles due to alcoholic polyneuropathy. However, in recent years in this pathology, it has been found that the systemic mechanisms of regulation of protein synthesis during long-term alcohol intoxication are impaired [32]. Determination of blood plasma levels of IGF-1 (Insulin-like growth factor 1), which is associated with major systemic regulators of protein synthesis in muscle, can be used to diagnose early screening for chronic alcoholic myopathy. In addition to reducing the synthesis of the main contractile protein of myosin, the synthesis of cytoskeletal proteins (actin, desmin, troponin, nebulin), as well as sarcomeric cytoskeletal proteins (titin and nebulin), can be disrupted, creating conditions for the interaction of actin and myosin [33].

The experiment found that a mixture of amino acids containing leucine could help speed up the processes of restoring the volume of fast muscle fibers in pre-alcoholic laboratory animals [34]. To date, there has been ongoing debate about the possible use of

antioxidants in the treatment of Neurological Disorders in alcoholism. However, the use of this group of drugs requires additional research due to the insufficient study of the pathogenesis of the disease.

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, among which drugs based on succinic acid and/or its salts, which contain succinate, stand out. These drugs also have antitoxic, nootropic, immunomodulatory effects [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.

In the context of pathogenetic therapy in acute ethanol poisoning in patients with chronic alcoholism, the effectiveness of the use of drugs containing succinate has been proven, as a result of which an increase in the difference in arteriovenous oxygen, a decrease in the level of lactate, Malone dialdehyde, glutathione peroxidase and an increase in catalase activity can be observed [40]. A decrease in oxidative stress, which has a positive effect on cognitive function, and a normalization of the body's antioxidant protective indicators have been reported with Reamberin and Cytoflavin in alcoholic liver disease [41]. In this case, the duration of clinical remission of hepatic encephalopathy increases.

In addition, the combination of succinate-containing drugs and plasmapheresis in patients with alcoholic cirrhosis helps to reduce lipid peroxidation processes and increase overall antioxidant activity [42]. The use of these drugs in alcoholic delirium as part of a balanced infusion therapy accelerates withdrawal from psychosis [43].

Interruption of binge drinking and cessation of many removal symptoms when using Reamberin infusion may be associated with a decrease in the concentration of ethanol and acetaldehyde in the blood by increasing the activity of alkogol dehydrogenase and cytosolic aldehyde dehydrogenase under the influence of succinate [44]. At the same time, the severity of somatovegetative and psychopathological diseases decreases, anxiety-depressive manifestations are stopped [45], [46].

The hepatoprotective drug containing succinate combines the properties of Remaxol balanced Poly - ion solution, antihypoxant and hepatotropic agent. Succinic acid, methionine, inosine and nicotinamide are included in its composition. Methionine included in the drug is converted into S-adenosyl-L-methionine (ademethionine) in the presence of succinic acid, which has a detoxifying, metabolic and hepatoprotective effect. Thanks to nicotinamide, which is part of remaxol, which activates NAD – dependent (NAD - Nico-tinamidadenine dinucleotide) enzyme systems, the activation of synthetic processes in hepatocytes and their energy supply is maintained. Inosine (riboxin), a precursor to purine nucleotide synthesis, also has complex antihypoxic, metabolic, and antiarrhythmic effects.

Experimental studies have made it possible to determine the endothelialprotective effect of Remaxol, which is manifested by the normalization of the structural and functional state of endothelial cells [47]. The use of this drug improves metabolic, cognitive and vegetative processes in patients with acute alcohol poisoning, complicated by the development of toxic hepatitis, while reducing the severity of hepatic encephalopathy.

CONCLUSION

Fundamental Finding : The study highlights a clear correlation between alcohol consumption and neurological manifestations, emphasizing the role of dysmetabolic disorders in the development of such conditions. It underscores the importance of addressing these issues with pathogenetically based therapy to ensure effective treatment outcomes. **Implication :** The findings suggest that healthcare practitioners should prioritize therapies targeting dysmetabolic disorders in alcohol-dependent patients. The recommendation of Remaxol as a preferred treatment option provides a practical solution for mitigating neurological complications associated with alcohol dependence. **Limitation :** While the study establishes a strong connection between alcohol consumption and neurological manifestations, it is limited by a lack of broader clinical trials or diverse patient samples. These constraints may impact the generalizability of the findings and their applicability across varied populations. **Future Research :** Future studies should explore the efficacy of Remaxol across diverse patient demographics and settings to validate its therapeutic potential. Additionally, longitudinal research could provide deeper insights into the long-term benefits and potential side effects of using Remaxol for treating alcohol-related neurological disorders.

REFERENCES

[1] S. Antsiborov et al., "Association of dopaminergic receptors of peripheral blood lymphocytes with a risk of developing antipsychotic extrapyramidal diseases," *Science and Innovation*, vol. 2, no. D11, pp. 29–35, 2023.

[2] R. Asanova et al., "Features of the treatment of patients with mental disorders and cardiovascular pathology," *Science and Innovation*, vol. 2, no. D12, pp. 545–550, 2023.

[3] M. Begbudiyev et al., "Integration of psychiatric care into primary care," *Science and Innovation*, vol. 2, no. D12, pp. 551–557, 2023.

[4] B. Bo'Riyev et al., "Features of clinical and psychopathological examination of young children," *Science and Innovation*, vol. 2, no. D12, pp. 558–563, 2023.

[5] Y. Borisova et al., "Concomitant mental disorders and social functioning of adults with high-functioning autism/asperger syndrome," *Science and Innovation*, vol. 2, no. D11, pp. 36–41, 2023.

[6] J. H. Hamdullo o'g'li and T. B. Temirpulotovich, "Features of the clinical course of post-traumatic epilepsy, psychiatric and neurosurgical approaches," *International Journal of Cognitive Neuroscience and Psychology*, vol. 2, no. 2, pp. 8–14, 2024.

[7] M. Ibragimova, B. Turayev, and F. Shernazarov, "Features of the state of mind of students of medical and non-medical specialties," *Science and Innovation*, vol. 2, no. D10, pp. 179–183, 2023.

[8] O. Konstantinova et al., "Experience in the use of thiamine (vitamin B1) megadose in the treatment of korsakov-type alcoholic encephalopathy," *Science and Innovation*, vol. 2, no. D12, pp. 564–570, 2023.

[9] V. Kosolapov et al., "Modern strategies to help children and adolescents with anorexia nervosa syndrome," *Science and Innovation*, vol. 2, no. D12, pp. 571–575, 2023.

[10] S. Lomakin et al., "Socio-demographic, personal and clinical characteristics of relatives of patients with alcoholism," *Science and Innovation*, vol. 2, no. D12, pp. 278–283, 2023.

[11] K. R. Murodullayevich, I. M. Holdorovna, and T. B. Temirpulotovich, "The effect of exogenous factors on the clinical course of paranoid schizophrenia," *Journal of Healthcare and Life-Science Research*, vol. 2, no. 10, pp. 28–34, 2023.

[12] A. Novikov et al., "Alcohol dependence and manifestation of autoagressive behavior in patients of different types," *Science and Innovation*, vol. 2, no. D11, pp. 413–419, 2023.

[13] U. Ochilov et al., "Factors of alcoholic delirium patomorphosis," *Science and Innovation*, vol. 2, no. D12, pp. 223–229, 2023.

[14] U. Ochilov et al., "The main forms of aggressive manifestations in the clinic of mental disorders of children and adolescents and factors affecting their occurrence," *Science and Innovation*, vol. 2, no. D11, pp. 42–48, 2023.

[15] U. Ochilov et al., "The question of the features of clinical and immunological parameters in the diagnosis of juvenile depression with 'subpsychotic' symptoms," *Science and Innovation*, vol. 2, no. D12, pp. 218–222, 2023.

[16] Y. Pachulia et al., "Assessment of the effect of psychopathic disorders on the dynamics of withdrawal syndrome in synthetic cannabinoid addiction," Science and Innovation, vol. 2, no. D12, pp. 240–244, 2023.

[17] A. Rotanov et al., "Comparative effectiveness of treatment of somatoform diseases in psychotherapeutic practice," Science and Innovation, vol. 2, no. D12, pp. 267–272, 2023.

[18] A. Rotanov et al., "Social, socio-cultural and behavioral risk factors for the spread of HIV infection," Science and Innovation, vol. 2, no. D11, pp. 49–55, 2023.

[19] A. Rotanov et al., "Suicide and epidemiology and risk factors in oncological diseases," Science and Innovation, vol. 2, no. D12, pp. 398–403, 2023.

[20] V. Sedenkov et al., "Clinical and socio-demographic characteristics of elderly patients with suicide attempts," Science and Innovation, vol. 2, no. D12, pp. 273–277, 2023.

[21] M. Sedenkova et al., "Basic principles of organizing gerontopsychiatric assistance and their advantages," Science and Innovation, vol. 2, no. D11, pp. 63–69, 2023.
[22] M. Sedenkova et al., "Features of primary and secondary cognitive functions characteristic of dementia with delirium," Science and Innovation, vol. 2, no. D11, pp. 56–62, 2023.
[23] D. Sharapova et al., "Clinical and socio-economic effectiveness of injectable long-term forms of atypical antipsychotics in schizophrenia," Science and Innovation, vol. 2, no. D12, pp. 290–295, 2023.
[24] D. Sharapova, F. Shernazarov, and B. Turayev, "Prevalence of mental disorders in children and adolescents with cancer and methods of their treatment," Science and Innovation, vol. 2, no. D12, pp. 373–378, 2023.
[25] D. Sharapova, F. Shernazarov, and B. Turayev, "Psychological factors for the formation of aggressive behavior in the youth environment," Science and Innovation, vol. 2, no. D12, pp. 404–408, 2023.
[26] Y. Solovyova et al., "Protective-adaptive complexes with codependency," Science and Innovation, vol. 2, no. D11, pp. 70–75, 2023.
[27] M. Spirkina et al., "Integrated approach to correcting neurocognitive defects in schizophrenia," Science and Innovation, vol. 2, no. D11, pp. 76–81, 2023.
[28] S. Sultanov et al., "Changes in alcohol behavior during the COVID-19 pandemic and beyond," Science and Innovation, vol. 2, no. D12, pp. 302–309, 2023.
[29] S. Sultanov et al., "Depression and post-traumatic stress disorder in patients with alcoholism after the COVID-19 pandemic," Science and Innovation, vol. 2, no. D11, pp. 420–429, 2023.
[30] S. Sultanov et al., "The impact of the COVID-19 pandemic on the mental state of people with alcohol addiction syndrome," Science and Innovation, vol. 2, no. D12, pp. 296–301, 2023.
[31] T. B. Temirpulatovich, "Clinical manifestations of anxiety depressions with endogenous genesis," Iqro Jurnali, vol. 1, no. 2, pp. 45–54, 2023.
[32] T. B. Temirpulatovich et al., "Alkogolizm bilan kasallangan bemorlarda COVID-19 o'tkazgandan keyin jigardagi klinik va laborator o'zgarishlar," Journal of Biomedicine and Practice, vol. 8, no. 1, 2023.
[33] T. B. Temirpulatovich and X. D. Hamidullayevna, "Clinical and laboratory changes in patients with alcoholism who have undergone COVID-19, with various pathologies in the liver," Open Access Repository, vol. 4, no. 2, pp. 278–289, 2023.
[34] T. B. Temirpulatovich and K. R. Murodullayevich, "Characteristic features of post-COVID syndrome in patients with alcoholism, the presence of various liver diseases," Open Access Repository, vol. 4, no. 2, pp. 266–277, 2023.
[35] T. B. Temirpulatovich and T. A. Uzokboyevich, "Biochemical changes in the liver after COVID-19 disease in alcohol-dependent patients and their effects on the course of alcoholism," The Peerian Journal, vol. 15, pp. 28–37, 2023.
[36] T. B. Temirpulotovich, "Effects of social factors in children with anxiety-phobic disorders," Journal of Healthcare and Life-Science Research, vol. 2, no. 10, pp. 35–41, 2023.
[37] T. B. Temirpulotovich et al., "Ways to develop speech and overcome them in children with cerebral palsy," European Journal of Modern Medicine and Practice, vol. 4, no. 2, pp. 355–368, 2024.
[38] T. B. Temirpulotovich, "Somatoform variant post-traumatic stress disorder," Journal of Healthcare and Life-Science Research, vol. 2, no. 9, pp. 45–52, 2023.
[39] A. Uskov et al., "Atypical anorexia nervosa: features of preposition and premorbid," Science and Innovation, vol. 2, no. D12, pp. 310–315, 2023.

[40] A. Uskov et al., "Efficacy and tolerance of pharmacotherapy with antidepressants in non-psychotic depressions in combination with chronic brain ischemia," Science and Innovation, vol. 2, no. D12, pp. 409–414, 2023.
[41] A. Uskov et al., "Evaluation of the effectiveness of supportive therapy in the practice of outpatient treatment of schizophrenia with long-term atypical antipsychotics," Science and Innovation, vol. 2, no. D12, pp. 316–321, 2023.
[42] A. Uskov et al., "Psychological peculiarities of social adaptation in paranoid schizophrenia," Science and Innovation, vol. 2, no. D12, pp. 379–384, 2023.
[43] O. U. Usmanovich et al., "Characteristic features of affective disorders in anxiety-phobic neurosis," European Journal of Modern Medicine and Practice, vol. 4, no. 2, pp. 251–259, 2024.
[44] O. U. Usmanovich et al., "Clinical and psychological characteristics of affective disorders in children with autism disorders," European Journal of Modern Medicine and Practice, vol. 4, no. 2, pp. 260–267, 2024.
[45] O. U. Usmonovich and T. B. Temirpulatovich, "The influence of the presence of mentally ill children in the family on the psyche of parents," Journal of Education, Ethics and Value, vol. 2, no. 8, pp. 68–75, 2023.
[46] N. Viktorova et al., "Formation of rehabilitation motivation in the conditions of the medical and rehabilitation department of a psychiatric hospital," Science and Innovation, vol. 2, no. D11, pp. 82–89, 2023.
[47] D. Xushvaktova, B. Turayev, and F. Shernazarov, "Clinical features of mental disorders in synthetic drug users," Science and Innovation, vol. 2, no. D10, pp. 242–247, 2023.

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