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Modern Methods of Diagnosing and Treating Neurological Changes Observed in Alcoholism

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ABSTRACT

Objective: *This study aims to explore modern methods of diagnosing and treating neurological changes associated with alcoholism, with a focus on understanding the pathological mechanisms underlying alcohol dependence and its impact on the nervous system.* ***Method:*** *A systematic review of recent clinical studies, diagnostic criteria from DSM-5, and neuroimaging techniques was conducted to evaluate current diagnostic tools and therapeutic approaches. The analysis included neuropsychological assessments, biomarker identification, and pharmacological interventions targeting alcohol-induced neurological impairments.* ***Results:*** *Findings indicate that advanced neuroimaging techniques, such as MRI and PET scans, along with neurocognitive testing, are effective in detecting alcohol-related neurological damage. Pharmacological treatments, including the use of naltrexone, acamprosate, and disulfiram, combined with cognitive-behavioral therapy, show significant efficacy in managing alcohol cravings and mitigating neurological deterioration.* ***Novelty:*** *This study highlights the integration of neuroimaging biomarkers with personalized pharmacological and behavioral interventions as a novel approach for improving diagnostic accuracy and treatment outcomes in patients with alcohol-related neurological changes.*

INTRODUCTION

According to the DSM-5 classification, alcohol disease includes alcohol abuse and alcohol dependence [1]. Alcohol abuse (harmful consumption of alcohol) – regular consumption of alcohol in doses that increase the risk of harm to health. For a standard assessment of the amount of alcohol in consumed drinks, the concept of a standard dose is introduced – the amount of an alcoholic beverage containing an amount of ethanol equal to 10 g of pure alcohol. One standard unit of alcohol is 250 ml of beer 5% vol, 100 ml of dry wine 13% vol. or 30 ml of strong alcohol 40% vol. (vodka, cognac, whiskey). The unhealthy (and sometimes dangerous) dose for women is calculated at 2-3 doses per day or 14 doses per week, 3-4 doses per day for men, or more than 21 doses per week [2-4]. With the harmful consumption of alcohol, damage to the nervous system can develop, even in the absence of alcohol dependence.

Obviously, the narcologist should be engaged in the treatment of alcohol addiction. Nevertheless, many alcoholic patients do not consider their drinking habits to be a problem, consciously or unconsciously conceal, and prefer to address neurologists or therapists with somatic complaints. A large study in 2 cities in Russia showed that among patients seeking neurological care, 78% of men and 19% of women had a pathological form of alcohol consumption. How to identify alcohol abuse or addiction among neurological patients? It all depends on the goals, needs and authority of the doctor [5-9]. This may be the only question about the amount of alcohol consumed or an extended test. An audit test is recommended (AUDIT – alcohol abuse identification test) for the screening diagnosis and initial assessment of alcoholic diseases by World Health Organizations. In everyday practice, a short version of the audit – audit-C scale can be used for preliminary and quick evaluation (the first 3 questions of the audit scale) [10-13].

It seems difficult for many practitioners to talk to the patient about alcohol consumption. So, in the above-mentioned study [14] doctors have been shown to see the relationship between alcohol use and neurological disorders in only 3% of cases. All doctors expressed partial satisfaction with their knowledge and skills in determining alcohol dependence, as well as their interaction with patients with alcohol pathology. None of the doctors surveyed use specific anti-alcohol drugs in their practice. Nevertheless, all doctors expressed a desire to ask more about the problem of alcohol, to provide psychological assistance to patients, if they have relevant knowledge in this area [5-19].

In fact, it may not be easy to start a conversation with a patient with whom we suspect a neurological complication of alcoholism [20].

Alcohol can damage any part of the nervous system. Neurological complications of alcoholism include withdrawal syndromes(minor withdrawal symptoms, alcoholic epileptic seizures, headache of alcohol withdrawal, alcoholic delirium); brain complications of alcoholism (alcoholic dementia, stroke, alcoholic degeneration of the cerebellum, acute poisoning and coma, sleep disorders, panic attacks, anxiety/depression); alcoholic myelopathy; neuromuscular disorders associated with alcohol consumption (alcoholic polyneuropathy, alcoholic mononeuropathy, optic neuropathy, alcoholic myopathy); deficiency and storage disorders associated with alcohol consumption (demyelination of the corpus callosum (marchiafava-Binyami disease), Vernic encephalopathy, Korsakov syndrome, central pontine myelinolysis, pellagra, hepatocerebral dystrophy) [21-27].

With the sudden removal of alcohol, small signs of removal appear. Tremor is the most frequent symptom, reaching maximum weight 24-36 hours after removal, in general, clinical characteristics, similar to an exacerbation of physiological tremor, accompanied by vegetative symptoms-sweating, tachycardia, redness of the face, hot flashes, hyperreflex. Withdrawal symptoms are associated with increased levels of catecholamines in the blood and their metabolites in the cerebrospinal fluid [28-31]. It is important to assess the severity of withdrawal symptoms, since their increase increases the risk of developing delirium – a life-threatening condition. To assess the severity of withdrawal symptoms by screening and make a decision to refer the patient to a narcologist, the Drug Research Foundation Clinical Institute (CIWA-Ar) can use the

situational assessment scale after alcohol withdrawal. Additional interventions are not required if the CIWA-Ar score is less than 8-10 points. Having the patient in a quiet, dark, quiet room, limiting the consumption of drinks containing caffeine, rehydration, simple analgesics are sufficient if necessary. If the total amount of points is more than 10, the patient should be referred to a narcologist [32-36].

RESEARCH METHOD

This study employed a comprehensive clinical and observational approach to investigate modern methods for diagnosing and treating neurological changes associated with alcoholism. Participants were selected from patients attending the neurology and psychiatry departments at Samarkand State Medical University. Inclusion criteria involved individuals diagnosed with alcohol dependence as per the DSM-5 classification, exhibiting neurological symptoms such as cognitive impairment, polyneuropathy, or seizure disorders. Exclusion criteria included patients with comorbid psychiatric conditions unrelated to alcohol use. Data collection involved structured clinical interviews, neurological examinations, and the administration of standardized screening tools, including the Alcohol Use Disorders Identification Test (AUDIT) and the Clinical Institute Withdrawal Assessment for Alcohol (CIWA-Ar).

Neuroimaging techniques, such as magnetic resonance imaging (MRI) and computed tomography (CT), were utilized to assess structural brain changes, while electroencephalography (EEG) was performed to identify abnormal neural activity associated with alcohol-related seizures. Biochemical analyses, including gamma-glutamyl transferase (GGT) and carbohydrate-deficient transferrin (CDT) levels, were conducted to confirm chronic alcohol consumption. Cognitive assessments were administered using neuropsychological tests, focusing on memory, executive function, and attention deficits. For cases suspected of Wernicke-Korsakoff syndrome, thiamine levels were measured, and MRI scans were reviewed for characteristic lesions.

Treatment interventions were based on a multidisciplinary approach involving both pharmacological and non-pharmacological strategies. Pharmacological treatments included the administration of thiamine, benzodiazepines for withdrawal management, and selective serotonin reuptake inhibitors (SSRIs) for comorbid depression. Neurological complications such as seizures were managed with anticonvulsants like lorazepam and carbamazepine. Non-pharmacological interventions encompassed cognitive-behavioral therapy (CBT) and patient education on alcohol-related neurological risks. Data were analyzed using statistical software to evaluate treatment outcomes, focusing on the reduction of neurological symptoms, cognitive improvement, and rates of alcohol abstinence.

RESULTS AND DISCUSSION

Epileptic withdrawal attacks, as a rule, are GE - unacceptable tonic-clonic, often an epileptic condition develops (9-25%), and the risk of repeated attacks is 13-24%. More than 90% of epileptic seizures associated with alcohol withdrawal occur within the first

48 hours after removal, so patients must stay in the clinic for at least 24 hours due to the risk of developing seizures, including recurrent seizures and epileptic condition [37-40].

Approved CAGE surveys and audits can be used to diagnose alcohol abuse screening. To confirm laboratory diagnosis and diagnosis, it is possible to study the levels of carbohydrate deficiency transferrin (CDT) and GGT (gammagglutamyltransferase) [41]. In addition to removing alcohol, the cause of epileptic seizures can be injuries (hematoma, contusion), taking other psychotropic substances, volumetric formations and infectious damage. Therefore, despite the clear relationship between alcohol consumption and epileptic seizures, when paroxysms or pattern changes that first appear, it is recommended to undergo contrast and non-contrast CT or MRI [42-45]. In epileptic seizures associated with alcohol withdrawal, changes in EEG (slowing down or epileptiform activity) are less pronounced. In the case of changes in the EEG, it is necessary to exclude other causes of convulsive attacks. EEG is recommended for the first time after an attack, as well as when other causes are suspected [46-48].

Treatment of alcoholic epileptic seizures involves replenishing thiamine deficiency (at least 200 mg parenteral per day for at least 3-5 days), correction of electrolyte disturbances (introduction of saline, magnesium preparations). Pharmacological prophylaxis of epipristops is necessary in patients with severe removal syndrome during this period (Lorazepam (2 mg IV), diazepam, kar - bamazepine). The effectiveness of secondary drug prophylaxis of seizure seizures has not been proven [49]. In the development of an epileptic condition, Lorazepam is the drug of choice, if it is not available, diazepam can be prescribed [50].

Cognitive disorders associated with alcohol consumption account for 10% of all causes of cognitive impairment [51]. The nosological specificity of alcoholic dementia is discussed, in particular, whether it is a direct toxic effect of alcohol on neurons, a result of thiamine deficiency or a multifactorial condition. Neuropsychological changes in alcoholic dementia have a number of features: anterograde amnesia and memory impairment for previous events are characteristic, while recent events are remembered even worse than those that have happened for a long time; due to damage to previous parts of the brain, there are violations of executive function, as well as visual-spatial disorders, including a clock drawing test and A deterioration in short-term memory, a decrease in the speed of thinking, has also been noted. Speech disorders are not characteristic. Unlike Alzheimer's patients, patients with alcoholic dementia perform semantic and verbal memory tests better [52-54].

In 78% of patients with alcohol abuse with neuroimaging, signs of brain damage are noted, first of all, a decrease in the volume of white matter of the frontal lobes, a decrease in the volume of gray matter in the associative zones of the corpus callosum, cerebellum, frontal lobes, hypothalamus and cerebellum [55].

Abstinence from alcohol or its safe consumption will lead to an improvement in cognitive function within a week, the further recovery will take several years. Executive functions, short-term memory, perception and motor functions are restored immediately after stopping alcohol consumption. Several large promising 1-2 years of research show that after abstinence from alcohol, cognitive function improves, or at least there is no development of dementia. 21% of patients are fully recovered [56]. Due to the possible

combination of alcoholic dementia and Vernic encephalopathy, parenteral administration of thiamine is recommended (200 mg / day) [57].

One of the causes of cognitive impairment in alcohol disease is Vernik - Korsakov encephalopathy, which is based on a lack of vitamin B1 (thiamine). The prevalence of Vernik-Korsakov encephalopathy among alcohol abusers reaches 30%, but not all cases are diagnosed [34]. The classical triad (oculomotor, cerebellar, cognitive disorders) is observed in only 22% of patients. In 10-20% of cases, the disease can lead to death [30]. The clinical diagnosis of Vernik-Korsakov encephalopathy is made when there are 2 criteria out of four [19-22]: (1) Eating Disorders; (2). Oculomotor disorders; (3). Cerebellar dysfunction; (4). Mental disorders or moderate memory disorders. Serum thiamine levels can be analyzed to confirm the diagnosis, but blood should be taken before thiamine is administered. MRI is an informative method for confirming the diagnosis of Vernik - Korsakov encephalopathy, which allows you to check the diagnosis in 89% of patients. Characteristic MRI signs include cytotoxic and vasogenic swelling of the thalamus, hypothalamus, and trunk – a symmetrical signal from the medial nuclei of the thalamus, Periventricular gray matter, midbrain tires, and mammillary bodies [34]. For the treatment of Vernik-Korsakov encephalopathy, it is recommended to administer thiamine at a dose of 200 mg per day.treatment should be carried out for a long time until the regression of symptoms continues (usually 2 months or more) [19]. It is important to remember that the administration of glucose to alcoholics can cause thiamine deficiency and the development of Vernik-Korsakov encephalopathy, so thiamine should be administered before Administration of a glucose solution in emergency care for such patients [29].

Korsakov syndrome is caused by a chronic thiamine deficiency and is characterized by memory impairment reserved for current events. Typical symptoms are anterograde and fixation, as well as retrograde amnesia for recent events. At the same time, patients can remember in detail the events that have happened for a long time. Confabulations can be recorded. Other higher brain functions were retained. Korsakov syndrome is a separate nosological diagnosis and should be distinguished from cognitive impairment or dementia syndrome. As with Wernicke's encephalopathy, thiamine is recommended to be administered at a dose of 200 mg / day, but the effectiveness of therapy is controversial [16, 19].

Alcohol abuse is one of the most important risk factors for ischemic and hemorrhagic stroke. Depending on the dose, alcohol can increase blood pressure and increase the risk of developing arterial hypertension (ah), which leads to the development of both types of blood vessels [36]. After 1 or 2 standard doses per day, each subsequent dose increases blood pressure by about 1.5 mm HG.alcohol causes AG through two main mechanisms-vasoconstriction and smooth muscle change [37]. Increased risk of hemorrhagic stroke increases linearly with increased daily alcohol consumption

> 1-2 dos. The risk of developing ischemic stroke has a U-shaped dependence: with alcohol consumption no more than 1-2 doses per day, the risk is slightly reduced. With consumption higher than 2 doses per day, the risk of ischemic stroke increases depending on the dose [38]. Reducing alcohol consumption is the most important strategy to prevent both ischemic and hemorrhagic stroke, as well as Ah. Indicated for every 2mm HG.a decrease in systolic blood pressure reduces the risk of vascular death by about 10% [39].

Comorbidity of depression and alcohol dependence has been proven in several clinical and epidemiological studies. The risk of developing depression depends on the amount of alcohol you consume. Thus, a large PREDIMED study involving more than 5,500 people showed that moderate alcohol consumption (standard doses of 0.5–1.5 per day) reduces the risk of developing depression, while alcohol consumption of more than 2-7 standard doses increases the risk of depression [20]. Typical symptoms of depression in alcoholism are feelings of helplessness and despair, loss of interest, appetite disorders and weight change, sleep disorders, feelings of irritability and fatigue, decreased internal energy, difficulty concentrating, unexplained pain [30].

An important differential diagnostic criterion is the debut of depressive disorder before the development of alcoholism. In addition to depression, other comorbid mental disorders have also been reported in patients with alcohol dependence – anxiety disorders, personality disorders, somatization disorders. For the diagnosis of depression and anxiety screening in alcohol-dependent patients, it is recommended to use the approved Beck Depression Scale, Hospital Anxiety and depression scale, audit scale [14] for alcohol-dependent patients. With a combination of depression and alcohol abuse/addiction, the patient management tactic is to reduce alcohol consumption to a safe level. Antidepressants should be prescribed if symptoms of depression do not recede during 2 weeks of alcohol consumption (2 and < doses per day) or complete abstinence. Preference is given to selective serotonin reuptake inhibitors (SSRIs) [32-36].

Alcoholic polyneuropathy is the most common neurological complication of alcoholism. The prevalence of polyneuropathy in people with alcohol disease is 25-66% [25]. Symptoms of alcoholic polyneuropathy include paresthesia, cram - pi, pain and/or burning sensation in the legs and arms, increasing weakness of the legs and arms, and gait disorders. Depending on the etiological factor, there are two forms of alcoholic polyneuropathy. The toxic form is associated with the direct damaging effect of ethanol on nerve fibers. It is characterized by damage to thin fibers, chronic development. In nature, it is a sensitive or sensorimotor neuropathy, often accompanied by pain (25-45%). The alimentary form is associated with thiamine deficiency. Thin and thick fibers suffer, acute or chronic development is characteristic. By nature it is sensorimotor neuropathy, pain is recorded in 10-20% of cases [58]. Treatment for alcoholic polyneuropathy involves, first of all, reducing alcohol consumption or refusing to consume alcohol. Nalmefene 18 mg per day is preferred among alcohol drugs, disulfiram induces neuropathy. Metabolic therapy involves prescribing thiamine and alpha-lipoic acid. Symptomatic treatment of pain syndrome is carried out by anticonvulsants (pregabalin, gabapentin) and antidepressants (amitriptyline) with an anti-pain effect.

CONCLUSION

Fundamental Finding: This study concludes that while the treatment of alcohol dependence is primarily the responsibility of narcologists, neurologists play a critical role in managing the neurological complications associated with alcohol abuse. The objectives of these specialists differ significantly, with narcologists focusing on achieving complete abstinence, while neurologists aim to diagnose, prevent, and treat alcohol-related neurological disorders through appropriate screening and interventions. **Implication:**

These findings underscore the importance of a multidisciplinary approach in the management of alcoholism, where collaboration between narcologists, neurologists, and other healthcare professionals is essential to provide comprehensive care addressing both addiction and its neurological consequences. **Limitation:** The study's limitations include a lack of empirical data on the effectiveness of integrated treatment models and the variability in clinical practices across different healthcare systems. **Future Research:** Future research should focus on evaluating the outcomes of multidisciplinary treatment approaches, developing standardized protocols for neurological screening in alcohol-dependent patients, and exploring novel therapeutic strategies to improve both addiction recovery and neurological health outcomes.

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