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COMPLICATIONS NEUROLOGICAL ACUTE FUR CONSUMPTION OF ALCOHOL: A LITERATURE REVIEW

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Abstract: Alcohol is the most used psychoactive substance in our society. The nervous system is a specific target tissue for alcohol. The substance interacts with numerous receptors of neurotransmission systems, particularly NMDA, GABA and glutamatergic receptors, inducing a depressant effect on the CNS, brain damage and cognitive deficits. The pathological effects of alcohol use on the central nervous system (CNS) are well known, and there are numerous neurological disorders of an acute nature. This article is a review of the available literature on the main complications of excessive alcohol consumption on the central and/or peripheral nervous system. The study reviews several scientific articles selected from PubMed, MedLine, LILACS and SCIELO databases. The most common complications are intoxication, alcohol withdrawal, Wernicke's emcephalopathy and hepatic encephalopathy. All these problems have significant morbidity and mortality, and early and correct diagnosis and the timely institution of treatment are essential in order to avoid or interrupt possible irreversible complications.
Keywords: Alcoholism; neurological complications; Wernicke's alcoholic syndrome; hepatic encephalopathy.

INTRODUCTION

Alcohol is the most used psychoactive substance in our society. Its abusive use constitutes a serious public health problem, due to the high potential of the substance to cause chemical dependence, associated with the ease of access and the social value added to its consumption (COSTA, 2003).

According to the 5th edition of the Diagnostic Statistical Manual of Mental Disorders (DSM5), of the American Psychiatric Association (APA), alcohol use disorders are defined as the repetition of

problems resulting from the use of alcohol that lead to harm and/or to clinically significant distress (APA et al., 2014). The diagnosis is made by the presence of two or more criteria at any time within a 12-month period. The severity of the condition is defined according to the number of symptoms presented, which range from the increase in the amount and period of consumption, the intense and urgent desire to consume the substance, psychosocial, interpersonal and occupational damage, to the symptoms of tolerance. and abstinence (APA et al., 2014).

A number of factors influencing brain damage are associated with the amount and frequency of exposure to the substance, age of onset and duration of consumption, age group, educational level, socioeconomic status and gender of the individual, as well as their genetic background and family history. of alcoholism (VOLKOW et al., 2019). Studies on the mechanisms underlying the modulatory influence of adverse social environments, childhood experiences and genetic variability are fundamental to understanding what makes individuals regularly exposed to a drug dependent or not on it (CARIM-TODD et al., 2016 and SPANAGEL et al., 2016 and SPANAGEL et al. al., 2014).

Neurological disorders caused by alcohol consumption can lead to acute complications, which include Wernicke's encephalopathy, traumatic brain injury, fainting, seizures, stroke, and hepatic encephalopathy. (RAO and TOPIWALA, 2020). As there is strong scientific evidence of the association between excessive alcohol consumption for long periods with increased risk of dementia and cognitive decline (LANGBALLE et al., 2015).

METHODOLOGY

This article is a review of the available literature on the main acute neurological manifestations of excessive alcohol consumption on the central and/or peripheral nervous system. The study reviews several scientific articles selected from PubMed, MedLine, LILACS and SCIELO databases, in the original languages English, Portuguese, Spanish and French. Data analysis was performed through translation and reading of the articles in full.

DISCUSSION

Excessive alcohol consumption is defined as alcohol that, without meeting the criteria of dependence, is capable of producing pathological changes in the individual, covering a level of alcohol consumption of 40-60 g/day in women or 60-100 g/day in women. men. Alcohol dependence disorder arises when the excessive consumption of this substance produces a deterioration in the individual's social, work and family relationships (APA et al., 2014). The World Health Organization report on excessive alcohol consumption identified more than 60 diseases related to the systemic effects of alcohol (WHO, 2019).

The nervous system is a specific target tissue for alcohol. The substance interacts with numerous receptors of neurotransmission systems, particularly NMDA, GABA and glutamatergic receptors, inducing a depressant effect on the CNS, brain damage and cognitive deficits. Thus, the state of alcohol dependence is associated with an increase in the activation threshold of dopaminergic neurons and a negative emotional state, in addition to a deficit in inhibitory control and an increase in the motivation to drink alcohol (NAASSILA et al., 2018).

Alcohol, among other effects, increases the firing of dopaminergic neurons in the ventral tegmental area, projecting to the nucleus accumbens, causing its disinhibition through the inhibition of GABA neurons (VOLKOW et al., 2019). Another neurotransmitter involved

is serotonin, whose levels are mitigated in the CNS by regular use of the substance. As a consequence, a cycle of depression and anxiety is established, which motivate the individual to drink, which causes a reduction in brain serotonin, reinforcing the anxious and depressive condition and the search for alcoholic effects (COSTARDI et al., 2015).

Therefore, acute intoxications result from episodes of excessive consumption, withdrawal syndromes or decompensation of nutritional deficiencies (DEMATTEIS and PENNEL, 2018). This article examines the different neurological manifestations resulting from excessive alcohol consumption.

ACUTE NEUROLOGICAL COMPLICATIONS FROM ALCOHOL CONSUMPTION

Acute Alcohol Intoxication

Symptoms of alcohol intoxication are the result of the depressant action of alcohol on the brain and spinal neurons. There is a relationship between symptoms derived from acute alcohol consumption and their blood levels, which varies according to inter-individual factors depending on sex, habitual consumption, genetic and metabolic factors Some of the early effects of acute alcohol consumption, such as euphoria , mood fluctuation, social disinhibition, mild ataxia, nystagmus, dysarthria, facial flushing, tachycardia, and mydriasis, and aggression appear to be due to inhibition of certain subcortical structures (such as the upper brain stem reticular formation) that modulate cerebral cortex activity (HAES et al., 2010).

With increasing amounts of alcohol, the depressant action extends to also include cortical neurons and other brainstem and spinal neurons, so that hyporeflexia, hypotension, decreased level of consciousness, and coma with respiratory depression can result. Amnesic gaps and seizures can easily occur in certain susceptible people after relatively mild intoxication. It is important to emphasize that alcoholic coma represents a medical emergency, mainly due to depression of respiratory function, which requires adequate life support measures (PLANAS-BALLVÉ et al., 2017).

Alcohol withdrawal syndrome

Withdrawal is the clinical expression of abruptly stopping or reducing alcohol intake in patients who have developed tolerance and dependence (PLANAS-BALLVÉ et al., 2017). The alcoholic patient is at risk for the development of alcohol withdrawal syndrome from alcohol consumption equal to or greater than 14 doses/week or four doses per occasion (for men) or equal to or greater than seven doses/week or three servings per occasion (for women). Patients at risk are those who ingest more than 14 doses/week (men) or more than 7 doses/week (women) (HAES et al., 2010).

Withdrawal syndrome manifestations range from distal hand tremor, anxiety, insomnia, and visual hallucinations, to psychomotor agitation, autonomic hyperactivity, seizures, or coma. Symptoms usually begin 6 to 24 hours after stopping or reducing alcohol dependence (MIRIJELLO et al., 2015). The most severe form, which usually appears 72 hours after withdrawal, is delirium tremens, which is characterized by disorientation, agitation, and visual hallucinations, accompanied by autonomic signs such as hyperventilation, tachycardia, and sweating. It may associate metabolic and electrolyte changes, such as hypomagnesemia. Mortality ranges from 5 to 15%, mainly due to metabolic, cardiovascular and infectious complications (OSÓRIO, 2020).

The treatment of choice is benzodiazepines, as they reduce the risk of epileptic seizures. Those with a long half-life, such as diazepam, are used. If the patient has hallucinations

and agitation that do not respond to benzodiazepines, antipsychotics may be added, only in combination with benzodiazepines, as these alone may increase the risk of seizures. Other drugs, such as alpha-2 agonists and beta-blockers, can be used as adjunctive treatments to control autonomic hyperactivity (MIRIJELLO et al., 2015).

Wernicke's Encephalopathy (WE)

Wernicke's encephalopathy (WE) is an acute syndrome that requires urgent treatment to prevent death and neurological comorbidities. Among its etiopathogenesis are c, drugs and chemical compounds, dialysis, malnutrition, chemotherapy and recurrent vomiting, the best known cause being chronic alcoholism. Alcoholism causes a very large thiamine (B1) deficiency, generating neuropsychiatric signs and symptoms (GALVÃO et al., 2020).

Thiamine plays a key role in carbohydrate metabolism as a cofactor of essential enzymes for the Krebs cycle. As these enzymes regulate brain energy metabolism, thiamine deficiency can cause damage, especially in regions with greater metabolic demand, such as the paraventricular region of the thalamus and hypothalamus, the mammillary bodies, the periaqueductal areas, the floor of the IV ventricle, and the vermis. cerebellar (PLANAS-BALLVÉ et al., 2017).

The classic triad of ophthalmoplegia, cerebellar damage (ataxia) and altered mental status occurs in only 16 to 19%, but mental confusion, characterized by mental slowness, disorientation, insufficient attention, agitation, and hallucinations, occurs in 80% of patients. Cerebellar dysfunction presents in 25% of WE as loss of balance, gait disturbance, truncal ataxia, dysdiadochokinesia, and occasionally full-body incoordination or dysarthria. (RAO e TOPIWALA, 2020).

WE is a medical emergency, as it is a potentially reversible disease and the absence of treatment or its delay can cause serious sequelae and even death. Its diagnosis is fundamentally clinical, although some complementary exams can help to confirm it or exclude other diagnostic alternatives. However, performing the tests must not delay the start of treatment. In this scenario, magnetic resonance imaging (MRI) is the most useful complementary test to confirm the diagnosis. In it, the most characteristic lesion is the reversible cytotoxic edema visualized on T2, FLAIR and DWI sequences in the periventricular and diencephalic regions. (PLANAS-BALLVÉ et al., 2017).

Treatment must be started immediately with the administration of thiamine in combination with other B vitamins, as this prevents the progression of the disease and reverses brain abnormalities that have not caused established structural damage. Daily oral administration of thiamine must be continued after completion of parenteral treatment and after hospital discharge until patients are no longer considered to be at risk. Magnesium and other vitamins are also replenished, along with other nutritional deficits, if present. (GALVÃO et al., 2020).

Hepatic encephalopathy (HE)

Hepatic encephalopathy (HE) is present in up to 50% of people with advanced cirrhosis, representing, therefore, a serious and frequent complication and an indicator of poor prognosis in these patients (OLIVEIRA et al., 2021). It is a complex syndrome of neuropsychiatric disorders that occurs in patients with advanced liver failure or portosystemic blood shunting (AASLD et al., 2014).

In the disease, there is a hepatic inability to metabolize substances, leading to hyperammonemia, considered one of

the pathogens of HE. It allows astrocytes to increase glutamine synthesis, causing edema, cellular and tissue degeneration that will culminate in acute neurocognitive dysfunction. In addition, excess ammonia in the bloodstream generates an imbalance between excitatory and inhibitory neurotransmitters, impairing the autoregulation of intracranial blood flow and producing clinical symptoms that are potentially irritating to the brain parenchyma (XU et al., 2019).

Symptoms are variable and fluctuating, from tremor and dysarthria to hepatic coma. It includes altered level of consciousness, which may progress from a mild confusional state to coma, neuropsychiatric symptoms such as behavioral changes, mental slowness, reversal of the sleep-wake cycle or psychomotor agitation, and neuromuscular signs, notably oscillating tremor (AASLD et al, 2014). Since the clinical manifestations of HE are nonspecific and can be seen in other diseases or metabolic disorders, the diagnosis is made after reasonable exclusion of other potential causes through complementary investigations (OLIVEIRA et al., 2021).

CONCLUSION

From the review of the available literature, it is possible to conclude that the abusive and chronic consumption of alcohol causes numerous serious changes and consequences to the human nervous system. The most common are Wernicke's encephalopathy, traumatic brain injury, fainting, seizures, stroke and hepatic encephalopathy. All syndromes have significant morbidity and mortality, and early and correct diagnosis and timely institution of treatment are essential in order to avoid or interrupt possible irreversible complications.

In this article, the most notable effects and with a more robust literature were described, but the existence of others must not be ignored. It is important to emphasize the need for further exploration of this theme, either through studies and/or scientific research, mainly with the aim of elucidating the mechanisms that cause neural damage, which include vitamin deficiencies, direct toxic effects of alcohol, changes in immunological and other unknown mechanisms.

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