

## CHRONIC NEUROLOGICAL EFFECTS CAUSED BY ALCOHOL INGESTION: A LITERATURE REVIEW

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*Mariana Rossi Silva da Paixão*

IMEPAC-Araguari MG

<http://lattes.cnpq.br/2092998881967145>

*Marcos André Macedo do Vale Silva*

IMEPAC-Araguari MG

<http://lattes.cnpq.br/0441854499145468>

*Iago Araujo Marques*

Universidade Integrada Padre Albino  
(UNIFIPA) – Catanduva SP

<http://lattes.cnpq.br/9228536349489567>

*Karina de Lima Sobrinho*

IMEPAC-Araguari MG

<http://lattes.cnpq.br/0851164927926846>

*Stella Soares Mendonça Duarte*

IMEPAC-Araguari MG

<http://lattes.cnpq.br/5154183864152551>

*Giovanna Chiovato Socreppa*

IMEPAC-Araguari MG

<http://lattes.cnpq.br/6579189681597294>

*Mônica Lopes Vieira*

IMEPAC-Araguari MG

<http://lattes.cnpq.br/3154423705579687>

*Andressa de Paula Silva*

IMEPAC-Araguari MG

<http://lattes.cnpq.br/3102041731499174>

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**Jordanna Borges Oliveira**  
IMEPAC – Itumbiara GO  
<http://lattes.cnpq.br/7564961199107760>

**Alexia Geovanna Lourenço Cruz**  
IMEPAC – Itumbiara GO  
<http://lattes.cnpq.br/3042073449046776>

**Rafhael Fernandes Santos**  
IMEPAC – Itumbiara GO  
<http://lattes.cnpq.br/6211020136377742>

**Aline Daniela Vieira de Siqueira**  
IMEPAC-Araguari MG  
<http://lattes.cnpq.br/7859364823936968>

**Abstract:** Alcohol has been the first drug used, being the entry point in the trajectory of those who develop addictions (STEIN et al., 2012). Due to this implication, in 1976, the World Health Organization (WHO) recognized binge drinking as a chronic disease, with serious repercussions for health, whose non-treatment can be fatal for the individual (RIBEIRO, 2015). Due to the importance of this theme, the present work was carried out, being written through the analysis of numerous scientific articles, chosen from the PubMed, MedLine, LILACS and SCIELO databases, in the original languages English, Portuguese and Spanish. Through this, it is concluded that the abusive and chronic use of alcohol causes numerous serious consequences to the nervous system. The most common are polyneuropathy, cerebellar degeneration and dementia and the most serious are Wernicke-Korsakoff syndrome and Machiafava-Bignami syndrome.

**Keywords:** Alcoholism; neurological complications; insanity; Wernicke-Korsakoff alcoholic syndrome; alcoholic neuropathy.

## INTRODUCTION

In the mid-nineteenth century, in Europe, the term “alcoholism” was born as a result of social changes and the demand for cleaning up urban centers, technization and patriotism. Measures were taken to ward off alcoholism, given the demand for commitment, increased economy and political control of the masses (SILVA et al., 2021)

In 1976, the World Health Organization (WHO) recognized alcohol dependence as a chronic disease, with serious health repercussions, whose non-treatment can be fatal for the individual (RIBEIRO, 2015). In Brazil, alcohol is the first drug used, being the entry drug in the trajectory of those who develop addictions (STEIN et al., 2012).

The use of alcohol is a major public health problem in Brazil, consumption has greatly intensified. In 1996, the Brazilian population consumed 5.51 liters of pure alcohol per capita, estimating only individuals over 15 years of age. Between 1970 and 1996, alcohol intake grew by 74.53%. (HAES et al., 2010).

The pathological implications of alcohol use on the central nervous system (CNS) are well known, although the dose-response relationship between consumption and complications, as well as the explanations for its damages and the probability of reversing them, remain not fully elucidated. given the available scientific evidence. However, it is well understood that symptoms can arise from any level of the neuraxis, including the brain, peripheral nerves and muscles. Thus, a diagnosis of alcohol use disorder is associated with a series of complications in the central nervous system (HAES et al., 2010).

## **METHODOLOGY**

This article is a review of the literature on the main chronic neurological manifestations of excessive alcohol consumption on the central and/or peripheral nervous system. The present work was written through the analysis of numerous scientific articles, chosen from PubMed, MedLine, LILACS and SCIELO databases, in the original languages English, Portuguese and Spanish. The data study was carried out after translation, elimination of reviews and/or research that the abstract did not match the theme and, finally, reading the articles in full.

## **DISCUSSION**

Alcohol is present in alcoholic beverages, it is considered a licit drug that depressants the Central Nervous System (CNS) and is a psychoactive substance. The metabolism of these substances in the body occurs in the gastrointestinal tract, where it undergoes

a series of transformations and breaks down molecules until it is absorbed. The consumption of these drinks goes far beyond social issues, and can be used to deal with anxiety, due to their initial effect of euphoria followed by depressive symptoms (ROTH, Thais et al., 2020).

The immoderation of alcohol is an obstacle in public health, because in addition to its progressive growth, it generates numerous problems for the health of individuals, contributing to public spending. Chronic alcoholics have nutritional impairments, as the caloric intake of several food groups becomes insufficient, especially vitamins and minerals, due to reduced stocks and inefficiency in absorption. Thus, due to the lack of nutrients, harmful metabolic changes occur, which lead to pathological complications. (GUERRA and VIEIRA, 2019).

Complications from the abuse of alcohol intake become more visible during the chronicity of its use, in which the aggravation of pathologies occurs and permanent sequelae arise, especially in the CNS. These disorders can harm several aspects of individuals' lives, as they can compromise motor and cognitive areas in order to cause changes in speech, gait, reasoning, memory, feelings, among other comorbidities. (THOMAZ et al., 2014).

## **CHRONIC NEUROLOGICAL COMPLICATIONS DUE TO ALCOHOL CONSUMPTION**

### **Korsakoff Syndrome (KS)**

Although historically they were described as separate entities, currently, Wernicke's encephalopathy (WD) and amnesic Korsakoff syndrome are considered the acute phase and residual state, respectively, of the same disease process. Korsakoff syndrome is fundamentally related to alcoholic malnutrition, as a residual state of WE, although it can sometimes appear without this history or after undiagnosed

subacute episodes. However, it can also be a symptom of malnutrition from other causes or a symptom of diseases or lesions of the medial region of the thalamus, inferomedial temporal lobes, of ischemic etiology, tumor or others (PLANAS-BALLVÉ et al., 2017).

From a clinical point of view, it is characterized by a disproportionate deterioration of memory in relation to other cognitive functions, in an awake, attentive and responsive subject. There is impairment of previously acquired memory, especially recent (retrograde amnesia), as well as inability to acquire new information (anterograde amnesia), with relative preservation of memory for remote facts and other cognitive functions (ÁLVAREZ HERNÁNDEZ et al., 2021). Memory dysfunction (particularly anterograde) is thought to result from disruption primarily of the anterior thalamic axis of the hippocampus, especially the white matter connections between them (RAO and TOPIWALA, 2020). Immediate replay is preserved. Patients are typically disoriented in time and space, apathetic, do not recognize their deficit, and often confabulate (invent stories to fill in memory gaps), but language is preserved (HAES et al., 2010).

The diagnosis is clinical, and the relatively selective memory disorder must be distinguished from the Global Cognitive Impairment that occurs in dementia of various etiologies. Korsakoff syndrome is often perceived as intractable, but in fact, after thiamine treatment, only 25% experience no recovery, 25% experience slight improvement, 25% significant improvement, and 25% fully recover memory. 2017). There is no single, well-studied intervention that has proven effective as a primary treatment for cognitive impairment in KS. An approach to using environmental modifications in a well-structured living environment, combined with various cognitive interventions, such as

pictorial associations and perhaps a donepezil or memantine test, likely represents the best strategy for treating long-term cognitive impairment in KS (JOHNSON and Fox, 2018).

### **Alcohol-Related Dementia (ADR)**

It is a form of dementia attributable to the direct chronic effects of alcohol on the brain. Studies show that drinking 140 g or more of alcohol a day for a long period of time can cause moderate cognitive impairment. However, the disease has never been precisely defined from a clinical or pathological point of view, and therefore, in recent years, the diagnosis of alcoholic dementia has developed an important controversy about its entity (PLANAS-BALLVÉ et al., 2017 and MATEUS and WICK - 2021).

From a pathophysiological point of view, neuronal damage or loss is believed to be related to glutamatergic neurotoxicity, oxidative stress and disruption of neurogenesis, triggered by chronic alcohol abuse. Clinically, it is characterized by an insidious onset with a staggered progression of symptoms that overlap with other neurodegenerative dementias. In the early stages, the neuropsychological study usually reveals a cognitive impairment with a frontosubcortical profile with slowness, attention deficit, immediate or short-term memory impairment, visuospatial impairment and impairment of executive planning and organizational functions (MATEUS and WICK, 2021).

### **Machiafava-Bignami Disease**

Rare condition associated with chronic and abusive alcohol intake and characterized by progressive demyelination and necrosis of the corpus callosum, which may extend to the subcortical white matter, being generally attributed to a deficiency of B vitamins. Clinically, it presents itself in two forms: one acute form with severe impairment of

consciousness, seizures and muscle stiffness, often resulting in death, and a chronic form with varying degrees of cognitive impairment, accompanied by gait impairment (MELO et al., 2020).

The diagnosis is difficult due to the great variability of the clinical picture. A history of alcoholism, symptoms, and especially brain neuroimaging, specifically MRI, are essential to confirm the diagnosis. Typical lesions found on MRI are demyelination, edema and necrosis of the corpus callosum, a subcortical white matter of variable extent. As the etiology of the disease is uncertain, no specific treatment is available. Cessation of alcohol intake and vitamin supplementation is recommended. In addition, some cases of good response to high doses of corticosteroids have been reported (SHEN et al., 2019).

### **Alcoholic cerebellar degeneration**

Alcoholic cerebellar degeneration is a common complication that affects up to 25% of alcoholics and is one of the most common causes of acquired ataxia in adults. It may represent, like Korsakoff Syndrome, a long-term sequel of Wernicke's Encephalopathy, being also associated with nutritional deficiency, especially of thiamine, although some authors also consider the hypothesis of a direct toxic effect of alcohol on the cerebellum and even disorders electrolytics (HAES et al., 2010).

The neuropathological basis consists of the loss of cerebellar cortical neurons, mainly Purkinje cells, with a special predilection for the anterior and superior cerebellar vermis, as well as in Wernicke's Encephalopathy, and some authors consider Alcoholic Cerebellar Degeneration to be a variant of such encephalopathy (HAES). et al., 2010).

Clinically, cerebellar degeneration is characterized by ataxia and trunk instability from broad-based gait and varying degrees of lower limb dysmetria. Upper limb dysmetria,

dysarthria or oculomotor alterations are less frequent. In most cases, cerebellar syndrome progresses over a period of several weeks or months, after which it persists for years.

The diagnosis is clinical. In both anatomopathological and neuroimaging studies, degeneration of all neurocellular elements of the cerebellar cortex, but in particular of Purkinje cells, occurs on the anterior and superior surfaces of the vermis. Cerebellar atrophy is easily observed by CT and MRI of the brain. There is no specific treatment, although vitamin supplements and abstinence from alcohol are recommended. Patients with such an entity must also receive parenteral thiamine (PLANAS-BALLVE et al., 2017).

### **CHRONIC ALCOHOLIC POLYNEUROPATHY**

It is the most common chronic neurological disorder related to alcohol abuse. There are controversies as to the etiology of this condition, whether it is a direct effect of alcohol or secondary to nutritional deficiencies. The main pathogenic process is axonal degeneration, and there may also be demyelination secondary to axonal degeneration or concomitant nutritional deficiencies (GOUVEIA et al., 2019).

Clinically, it manifests as a symmetric, distal, sensory-motor polyneuropathy of gradual onset. Symptoms include weakness, pain, paresthesias, muscle cramps, gait ataxia, and burning dysesthesias, with greater involvement of the lower limbs. Globally reduced or abolished reflexes and reduced vibratory sensitivity are very common findings, and affected limbs may present with edema, bone deformities and skin changes such as ulcers and hyperpigmentation (GOUVEIA et al., 2019). The Achilles reflex is the first to be affected. Patients with alcoholic neuropathy are susceptible to compression of peripheral nerves, and radial nerve palsy may

occur after episodes of alcohol intoxication in which this nerve is compressed against the humerus (HAES et al., 2010).

Treatment includes alcohol abstinence and vitamin supplementation (B-complex), especially thiamine, and neuropathic pain must be addressed with anticonvulsants (carbamazepine) or tricyclic antidepressants (HAES et al., 2010).

## CONCLUSION

Through a review of the available literature, it is admissible to conclude that the abusive and chronic use of alcohol causes numerous serious consequences to the nervous system. The most common are polyneuropathy, cerebellar degeneration and dementia and the most serious are Wernicke-Korsakoff syndrome and Marchiafava-Bignami syndrome. All diseases have significant morbidity and mortality, and the rapid, correct diagnosis and the appropriate association of treatment are undoubtedly important in order to avoid or stop possible irreversible disorders.

## REFERENCES

- ÁLVAREZ HERNÁNDEZ, Nuria et al. Revisión de pacientes con sospecha de síndrome de Wernicke-Korsakoff en el servicio de urgencias. 2021.
- GOUVEIA, Ana Luísa Barbosa; FERREIRA, Ana Luísa de Andrade Seguti; GRIPPE, Talyta Cortez. Análise dos aspectos epidemiológicos, clínicos e eletrofisiológicos dos pacientes diagnosticados com polineuropatia alcoólica. Programa de Iniciação Científica-PIC/UniCEUBRelatórios de Pesquisa, 2019.
- GUERRA, Isabelle B. Rosas; VIEIRA, Milene Leivas. Efeitos intestinais do uso abusivo do álcool etílico. Revista Terra & Cultura: Cadernos de Ensino e Pesquisa, v. 34, n. 67, p. 84-94, 2019.
- HAES, Tissiana Marques et al. Álcool e sistema nervoso central. Medicina (Ribeirão Preto) , v. 43, n. 2, pág. 153-163, 2010.
- JOHNSON, Justin M.; FOX, Valerie. Beyond thiamine: treatment for cognitive impairment in Korsakoff's syndrome. Psychosomatics, v. 59, n. 4, p. 311-317, 2018.
- MATEUS, Raquel; WICK, Jeannette Y. Alcohol-Related Dementia: Rethink How Much You Drink. The Senior Care Pharmacist, v. 36, n. 7, p. 324-330, 2021.
- MELO, Ana Luzia et al. Marchiafava-Bignami Disease: The Importance of Early Diagnosis and Treatment by a Multidisciplinary Team. Revista Portuguesa de Psiquiatria e Saúde Mental, v. 6, n. 1, p. 31-35, 2020.

PLANAS-BALLVÉ, Anna et al. Manifestaciones neurológicas del alcoholismo. *Gastroenterología y Hepatología*, v. 40, n. 10, p. 709-717, 2017.

RAO, Rahul; TOPIWALA, Anya. Alcohol use disorders and the brain. *Addiction*, v. 115, n. 8, p. 1580-1589, 2020.

RIBEIRO, Marcelo. Tiamina e Álcool: Uma Revisão da Literatura Sobre Interações Nutricionais, Possíveis Complicações e Tratamento.

ROTH, Thais et al. Prejuízo na absorção de nutrientes pela ingestão de álcool: uma revisão. *Research, Society and Development*, v. 9, n. 1, p. e190911910-e190911910, 2020.

SHEN, Yao-Yao et al. Clinical and neuroradiological features of 15 patients diagnosed with Marchiafava-Bignami disease. *Chinese medical journal*, v. 132, n. 15, p. 1887, 2019.

SILVA, Brenda Carla Chaves Da; MOURA, Wiltamara Lacerda de; NOGUEIRA, Apolonia Maria Tavares. *Saúde Mental e Ações de Controle e Combate ao Alcoolismo*, 2021.

SM, Stein AT et al. *Abuso e Dependência de Álcool*.

THOMAZ, KÍSSILA DE CÁSSIA VIEIRA et al. Alcoolismo e deficiência de tiamina associada à síndrome de Wernicke-Korsakoff. *Uningá Review Journal*, v. 20, n. 3, 2014.

WORLD HEALTH ORGANIZATION. *Global status report on alcohol and health 2018*. World Health Organization, 2019. (WHO, 2019)