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REVIEW

Neurological manifestations of excessive alcohol consumption☆

Anna Planas-Ballvé[a,*], Laia Grau-López[a], Rosa María Morillas[b], Ramón Planas[b]

[a] *Unidad de Neurociencias, Servicio de Neurología, Hospital Germans Trias i Pujol, Badalona (Barcelona), Spain*
[b] *Unidad de Hepatología, CIBERehd, Servicio de Aparato Digestivo, Hospital Germans Trias i Pujol, Badalona (Barcelona), Spain*

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Abstract This article reviews the different acute and chronic neurological manifestations of excessive alcohol consumption that affect the central or peripheral nervous system. Several mechanisms can be implicated depending on the disorder, ranging from nutritional factors, alcohol-related toxicity, metabolic changes and immune-mediated mechanisms. Recognition and early treatment of these manifestations is essential given their association with high morbidity and significantly increased mortality.

PALABRAS CLAVE
Alcoholismo;
Complicaciones neurológicas;
Encefalopatía hepática

Manifestaciones neurológicas del alcoholismo

Resumen En este artículo se revisan las distintas manifestaciones neurológicas del consumo excesivo de alcohol, que pueden ser agudas o crónicas y afectar al sistema nervioso central o periférico. El mecanismo por el cual se producen varía de un grupo de trastornos a otro. Destacan factores nutricionales, efectos tóxicos del alcohol, factores metabólicos e incluso inmunológicos. Estas manifestaciones pueden conllevar una gran morbilidad y un aumento significativo de la mortalidad, por lo que es importante reconocerlas y tratarlas precozmente.

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* Corresponding author.
E-mail address: annaplanasb@gmail.com (A. Planas-Ballvé).

Introduction

Excessive or harmful alcohol consumption is defined as the consumption of 40–60 g/day of alcohol in women or 60–100 g/day in men. Although it does not meet the criteria for alcohol dependency, this level of consumption can produce clinical changes. Alcohol use disorder appears when excessive alcohol consumption causes the deterioration of an individual's social, work and family relationships.[1]

The World Health Organization's report on excessive alcohol consumption identified more than 60 alcohol-related diseases.[2] The systemic effects of alcohol include changes in the digestive tract and the liver, the heart and vascular system, the skeletal and muscular systems, nutritional status, the immune, endocrine and haematological systems, and in the central and peripheral nervous systems.

Several alcohol-related neurological complications have been described (Table 1), and pathogenesis varies greatly among the different disorder groups, although one of the most frequent causes is nutritional deficiency. Alcohol can produce alcoholic liver disease that can be accompanied by a wide variety of neurological manifestations, including hepatic encephalopathy (HE).

In this article, we will examine the different neurological manifestations of excessive alcohol consumption, the neurological alterations most frequently found in alcoholic liver disease – such as HE – and the best diagnostic approach in clinical practice.

Central nervous system involvement

Acute complications

Acute intoxication

The symptoms of alcohol intoxication are the result of the inhibitory effect of alcohol on the nerve cells of the brain and spinal cord. Some of the immediate effects of acute alcohol ingestion – such as loquacity, loss of social inhibition, and aggressiveness – appear to be due to the inhibition of certain subcortical structures (perhaps the midbrain reticular formation) that modulate the activity of the cerebral cortex.[3] However, as more alcohol is consumed, this inhibitory action extends to cortical and other brain stem and spinal neurons, and can cause decreased alertness and coma with respiratory failure. Some susceptible individuals may experience amnesic lacunae and seizures after relatively mild alcohol intoxication.[4]

The severity of symptoms of acute alcohol intoxication are related to blood alcohol levels[5] (Table 2). These levels should be taken merely as a guide, and will vary between individuals according to sex, habitual consumption, and genetic and metabolic factors.

Acute alcohol intoxication should be treated with supportive measures and monitoring of the individual's level of consciousness. Alcoholic coma, with its associated respiratory depression, is a medical emergency that requires appropriate life support measures.

Alcohol withdrawal syndrome

Alcohol withdrawal syndrome, or abstinence syndrome, is the clinical manifestation of abruptly terminating or substantially reducing intake in patients who have developed tolerance and dependence. Alcohol acts basically through 2 specific neuronal receptors. On the one hand, it regulates the neurotransmitter gamma-aminobutyric acid type A receptor that inhibits neuronal excitability, which explains its sedative and hypnotic effects. On the other hand, alcohol increases glutamate N-methyl-D-aspartate receptor expression, which in turn increases glutamate activity and causes hyperexcitation. Chronic alcohol consumption induces neuroadaptive changes (tolerance), increases glutamate N-methyl-D-aspartate receptor expression, and desensitises the response and the expression of gamma-aminobutyric receptors.[6]

The manifestations of withdrawal syndrome (a wide range of severe symptoms, ranging from distal hand tremor, anxiety, insomnia and visual hallucinations to psychomotor agitation, autonomic hyperactivity, seizures or coma)

Table 1 Neurological manifestations secondary to excessive alcohol consumption.

Central nervous system	Peripheral nervous system
Acute	
Acute intoxication	Acute alcoholic neuropathy
Withdrawal syndrome and delirium tremens	Alcohol-related compressive neuropathy
Wernicke's encephalopathy	
Chronic	
Korsakoff syndrome	Chronic alcoholic neuropathy
Alcohol-related dementia	Disulfiram neuropathy
Marchiafava–Bignami disease	
Cerebellar degeneration	

Table 2 Manifestations of acute alcohol intoxication.

Blood alcohol, in mg/dl	Effects
<50	Difficulty in performing tasks that require skill, euphoria, loquacity
>100	Loss of self-control, loss of coordination, mental slowness, mild dysarthria, ataxia, altered perception
>200	Amnesia, confusion, diplopia, dysarthria, hypothermia, nausea, vomiting
>400	Stupor, respiratory depression, coma