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Acute ethanol–related nervous system injuries

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ABSTRACT

Acute ethanol intoxication has a diversity of clinical pictures that extend from mere euphoria to severe neurological impairment culminating in coma. Although the majority of cases have a reversible and benign course, the situation needs a careful medical monitoring. Authors describe pharmacological aspects of the acute ethanol intoxication, with organs and systems affected during the consumption of exaggerated quantities. Correlations between blood alcohol concentration and neurological symptomatology are given, and a brief discussion of the criteria for the acute intoxication is made. The fact that this occurrence has been of little attention is due not only to the reversibility of the majority of symptoms, but also because that medical research has been ever since focused more on chronic diseases related to alcohol abuse, withdrawal syndrome and recently with hangover as an independent situation. With no specific antidote actually at hand, treatment is symptom-oriented and the pharmacological armamentarium is richer when it comes to dealing with withdrawal, abstinence and other chronic complications of ethanol abuse.

1. Introduction

Ethanol is a constant and strong depressant of the central nervous system, a system that seems directly and mostly involved during acute intoxication[1]. Autonomous and vegetative nervous mechanisms might even introduce the depressant effects on the myocardium, albeit ethanol has straightforward toxic effects upon a variety of cells and tissues[2].

Ethanol provokes sedation and suppresses anxiety, hence it has high potential of reward, reuse and abuse. During binges, whose clear definition is still debatable, the speech of the consumer becomes slurred; judgment and social interactions are gradually severed with asocial behaviors coming frequently forward, and senses acuity is consistently decreased[3-5].

It might seem to be such a common occurrence that binges as a rule rarely come under special focus from medical staffs. The consumer is simply 'allowed to sleep' and that will be sufficient sober him up if the episode is isolated. However, severe intoxications will induce loss of conscience, coma; and autoptic data albeit not pathognomonic suggest a diffuse brain edema in connection with these situations[6]. Among the variety of physiological and psychological changes in connection with ethanol consumption,

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even the immune system has been studied and alterations have been suggested[7]. There is no doubt about the immediate effects of alcohol consumption on cognition. Learning will become difficult, with deficient attention and concentration; movements will be clumsy and reasoning superficial, with older ages being more prone to adverse effects[8]. The effect of a binge just before sleeping is more straightforward. The sleep will become fragmented, with earlier and frequent periods of wakefulness, although initially ethanol acts as a sleep-inducing agent. Since ethanol and nicotine are routinely co-consumed, patients with obstructive pulmonary disease will be more prone to periods of sleep apnea and probably prolonged and severe hypoxia[9].

Obviously acute ethanol intoxication will not affect simply and only the nervous system. Even moderated ethanol consume will decrease myocardial contracting ability[2]. Immediate vascular effects will include cutaneous vasodilatation and hyperemia, but contrarily to common beliefs, the coronary blood flow will not be affected. Shakespeare intuitively granted to ethanol consumption immediate effects such as 'sleep', 'nose- painting', 'lechery' and 'urine'[10]. Actually the diuretic effects of alcohol consumption and abuse will be of major importance regarding the sleep fragmentation due to repeated nocturnal urination, and seem to be related to the direct inhibitory role of ethanol vis-à-vis the release of antidiuretic hormone from the posterior pituitary[11]. We should also bear in mind that effects of ethanol intoxication do not limit their scope only within a single organ or hormonal structure, but studies suggest that almost the entire endocrine activity is affected[12].

2. Acute intoxication: Pharmacology and beyond

Connecting the binge with the following different antisocial and delinquent behaviors has been a constant issue to lawyers and toxicologist. The same might be true for medical doctors working during periods before the nineteenth century. Only Wernicke and Korsakoff at a certain time were able to make a causative link[13]. Blood alcohol concentration (BAC) is actually a very important parameter, not only because clear connections are made in between BAC and neurological signs, but also because laws and bylaws that are gradually being unified internationally, prohibit some actions (like for example, driving) under ethanol when limits of sobriety are not respected.

The Swedish scholar Widmark made important contributions with regard to the alcohol kinetics inside the human body. Some interesting formulas were suggested and still are used when trying to estimate BAC, given the number of drinks consumed[14].

Maling offered an interesting table correlating BAC with the neurological signs. We are reproducing some of these correlations in a modified form here below in Table 1[15]. Some of the symptomatology might be delayed if not absent in chronic alcoholics, and it is presumed that the whole array of these signs and symptoms will be entirely and gradually manifested in sober consumers, non-alcoholics albeit maybe not entirely naïve to the agent.

Table 1
Correlations between BAC and clinical-neurological signs.

Ethanolemia (mg /100 mL)	Consequences
20-99	A. Alteration of sensory functions:
	-decrease of visual acuity
	-hyposmia and hypogeusia
	-euphoria
	-decreased pain perception
	B. Motor coordination:
	-nystagmus
	-positive Romberg sign
	-difficult fine movements
	-impaired driving ability
	C. Personality, mood and behavioral changes:
	-less fatigue perception
	-enhanced self-esteem
	-loss of inhibition
	-talkative, loud speech
100-199	A. Staggering gait
	B. Poor performance of psychometric tests
	C. Highly impaired driving ability
	D. Increased reaction time
	E. Risk of blackouts, accidents, and hangovers
200-299	---Nausea and vomiting
	---Diplopia
	---Severe ataxia
	---Antisocial behavior
300-399	---Hypothermia
	---Loss of speech
	---Amnesia
	---Anesthesia (loss of pain perception)
	---Polypnea

Such a mixture of psychiatric, neurological and systemic signs and symptoms will reflect the severity of the intoxication, probably paralleling the blood alcohol levels and leading to coma, that is not as rare as it might seems, although benign in its course in the overwhelming majority of cases.

3. Discussion

With the amount of allowed or 'social' alcohol use decreasing gradually from the initial Anstie's limit to the actual much less of this proposed figure, other authors have also tried to define drinks and drinking levels in more practical terms[16,17].

It is a fact however, that acute alcohol intoxication has not received much of attention among medical professionals. The focus has been mainly shifted, and probably in a justified way, on the chronic disorders / diseases that are clearly related to alcohol abuse, as well as on alcohol withdrawal syndromes and recently, on the alcohol hangover as another independent entity[18,19].

DMS V and other versions have included alcohol intoxication as an entity whose complete symptomatology will encompass

probably the majority of the elements already included in the Table 1[20]. Coded under the ICD-9-CM coding system as 303.00 (International Classification of Diseases), some of the diagnostic criteria are as following:

1) Recent ingestion of alcohol;

2) Important behavioral and psychological changes (aggressive behavior, mood lability, impaired judgment);

3) Signs and symptoms during or shortly after consumption (slurred speech, motor incoordination, gait instability, nystagmus, attention and memory impairment, stupor and eventually coma);

4) No other medical condition or substance abuse attributable to the occurrence.

The benign character of an alcoholic binge has ever since been as a justifier for treating intoxications only symptomatically. Given the frequency of the situation, the event still receives little attention even in emergency departments, and it is prone to believe that the next day will sweep away all of the signs. However, pathologic studies have found brain edema following severe ethanol intoxications and coma, and the finding cannot be underestimated[6]. However, it is a fact that to date no specific antidote exists for the ethanol, and although hemodialysis has rarely been used in severe intoxications as an extreme but efficacious therapeutic measure[21,22].

On the other hand, if by far peripheral polyneuropathy is the most frequent long-term complication of chronic alcoholism, cases of acute neuropathy are reported. These are frequently related with neurapraxia of peripheral nerves in anatomic sites where elsewhere entrapment syndromes can be encountered. The so-called “Saturday night palsy” has for long been a synonym of radial nerve compression following a deep sleep. The event is generally associated with an alcohol binge for the weekends, although some sources consider it a misnomer[23]. In fact, the consumption of large doses of ethanol during particular days of the week, that remind us the beta-alcoholism of Jellinek, might not be necessarily restricted to alcohol abuse solely during the eve of holidays[24,25]. The radial nerve is frequently prone to compression for a diversity of reasons[26,27]. The common peroneal nerve as well might show clinical signs of an important compression at the head of fibula after acute alcohol intoxications[28,29]. Whether such cases are isolated or suggest the initiation of a polyneuropathy that will probably follow the repeated binges or the chronic abuse, this is still debatable.

4. Conclusion

It might be erroneous to declassify acute ethanol intoxication as a medical condition of non-emergency. Although changes are evident in several systems and organs, the fact that those are generally reversible, will not warrant a complete functional recovery[30]. Furthermore, lethal outcome might follow severe ethanol intoxications, with clear legal implications[31]. Since the majority of research has been focused on chronic alcoholism and its complications, people with acute intoxication still receive no an ad hoc therapeutics. Strangely enough even supportive measures, such as intravenous crystalloid fluids that are largely applied during intoxications, have an uncertain efficacy or can even be counterproductive[32]. Thiamine has ever since been a part of chronic alcoholism treatment, and studies have tested its efficacy in acute cases as well; some new drugs have been experimented in animal models, with application in humans yet to come[33]. Logically, the approach and the treatment of acute ethanol intoxication must be holistic, not merely oriented from the symptomatology, and aiming towards the prevention of immediate and long-term physical and psychological injuries.

Conflict of interest statement

The authors declare that they have no conflict of interest.

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