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Alcohol and the head-injured patient

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Abstract

Alcohol intoxication is a major predisposing factor for trauma in general and head injury in particular. The management of the head-injured patient is highly contingent on the accurate assessment of this patient's consciousness, which is invariably impaired if the patient is intoxicated. This complicates the decision-making process and impedes the promptness needed in management when the head injury is severe. Furthermore, the prognosis of the head injury can depend on the patient's degree and pattern of intoxication. This article presents some of the latest epidemiological data about the association of alcohol and head injury. It also highlights some of the challenges posed by alcohol intoxication in the management of head-injured patients, and examines the importance of documenting intoxication in head-injured patients.

Keywords

Alcohol, intoxication, head injury, traumatic brain injury, chronic alcoholism, Glasgow Coma Scale score

Introduction

Traumatic brain injury (TBI) remains one of the main causes of death and disability around the world today. The statistics vary by region and over time. However, from 2002 to 2006 in the United States, approximately 1.7 million people suffered from TBI annually. Nearly, 1.4 million were treated and released from emergency departments, 275,000 were hospitalized and discharged alive and 52,000 died (Faul et al., 2010). The most up to date report published in May 2011 by the Centers for Disease Control and Prevention estimates that the annual rate of deaths due to TBI is 18.4 per 100,000 people annually (Coronado et al., 2011). In Europe, the incidence of hospital referred TBI is 83.3 per 100,000 (Andelic et al., 2008), and that of fatalities is 15 per 100,000 (Tagliaferri et al., 2008). The incidence of death due to head injury in the UK is 6–10 per 100,000 per annum (DOH, 2001). This epidemic puts a huge-financial burden on the healthcare system. In the United States, the cost of TBI is estimated to be $48 billion annually (Rutland-Brown et al., 2006). Half of this cost goes

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towards long-term physical, psychological and psychosocial rehabilitation and care. When the cost of life-time loss of productivity is included, the numbers become staggering.

Alcohol is a major contributor to the global burden of disease, accounting for 4.0% of Disability Adjusted Life Years worldwide and 3.2% of mortality (Ezzati et al., 2002). A substantial portion of the burden of disease is attributed to acute alcohol-related injuries. The World Health Report 2002 states that injury is accountable for about one tenth of this disease burden (WHO, 2002). A collaborative study group from 12 countries found that the proportion of alcohol-related injury cases ranged from 6% to 45% (WHO, 2007).

In TBI, alcohol consumption is a strong predisposing factor (Kerr et al., 1971; Field, 1976; Parkinson et al., 1985). In studies addressing head injury and alcohol use specifically, elevated blood alcohol levels were present in more than 40% of the patients seen in emergency rooms or admitted to hospitals because of TBI (Galbraith et al., 1976; Rutherford, 1977; Brismar et al., 1983; Parkinson et al., 1985). This is comparable to a Swedish study showing a 35.8% rate of intoxication among those tested (Harr et al., 2011).

More than other injuries, the alcohol association with TBI represents a major challenge for clinicians. TBI cases require rapid intervention and decision making, and the ability to some extent to predict possible deterioration of the condition. Such decision making is dependent on the accurate assessment of the patient's consciousness, which is invariably impaired due to the effects of alcohol. Historically, the emergency department physician either over-diagnoses by interpreting signs of intoxication as indicating serious head injury, or under-diagnoses by attributing most or all neurological signs to intoxication, thereby missing possible co-existing and life-threatening conditions. Concern over the later scenario, combined with other legal, social and financial considerations have led to a spectrum of attitudes within the medical literature that is sometimes contradictory and confusing.

Some authors recognized the effect of alcohol intoxication on the level of consciousness a century ago (MacEwen, 1879) and stated that in evaluating patients of all grades of head injury severity, alcohol consistently had a depressive effect on the level of consciousness (Galbraith et al., 1976; Jagger et al., 1984; Sloan et al., 1989; Brickley and Shepherd, 1995) and associated intoxication with more severe injuries, and higher mortality (Pories et al., 1992; Cunningham et al., 2002). Others found no relation between alcohol in the presentation of TBI, the Glasgow Coma Scale (GCS) score, or severity and mortality (Huth et al., 1983; Nath et al., 1986; Sperry et al., 2006; Stuke et al., 2007). Finally, some authors have reported a positive association between high-blood alcohol concentration (BAC) on admission and lower in-hospital mortality rates in trauma patients (Ward et al., 1982; Blondell et al., 2002; Tien et al., 2006).

Alcohol intoxication is a complex issue and more research is needed. Nobody can ignore the consistent statistics that point to alcohol intoxication being the number one predisposing factor for head injury, intentional or unintentional, mild, moderate or severe and with any mechanism documented.

This article will examine the role played by alcohol in the presentation, and management of TBI cases, and discuss the implications of some of the literature concerning intoxicated head-injured patients.

Alcohol intoxication

Alcohol is a substance that is widely used. It also represents the oldest and the most universal substance of abuse. Ethanol is a water-soluble compound that rapidly crosses cell membranes, readily equilibrating between intra- and extracellular compartments. Absorption occurs mainly in the proximal intestinal tract, namely, in the stomach (70%) and in the duodenum (25%), with only a small percentage occurring in the remaining intestinal tracts (Marco and Kelen, 1990). Gastric alcohol dehydrogenase or

ADH is responsible for 10% of alcohol metabolism (first pass metabolism). The remaining 90% of ingested ethanol is metabolized to acetaldehyde by the following three liver enzymatic pathways: liver ADH (90%), microsomal ethanol oxidizing system (8–10%) and catalase (0–2%) (Lieber, 1991).

Symptoms and signs of alcohol intoxication are directly related to the BAC (Table 1). While any departure from zero BAC has some effects on the nervous system, effects on alertness and coordination start to occur at a BAC between 0.05% and 0.08% in adults. Significant motor and cognitive impairments occur between 0.1% and 0.15%, and amnesia may occur at levels above 0.2%. At BAC levels above 0.3%; disorientation and loss of consciousness are common (Moskowitz, 2000). Although the rate of elimination of alcohol is roughly 0.015–0.02 per hour, the effects of any given BAC on the level of consciousness vary by several factors including age, sex, gender, ethnicity (due to genetic variations in the types of ADH; Hurley et al., 2002), body weight, fat and water content, and frequency of consumption/tolerance. Even in the same person, the effects can vary from one occasion to another, depending on the amount and rate of consumption, and associated food and drug intake (Shahin et al., 2010). For this reason, a diagnosis of alcohol intoxication is usually based on the clinical picture rather than solely on BAC value.

Table 1. Main clinical symptoms in acute alcohol intoxication according to BAC.

Symptoms	BAC
Impairment in some tasks requiring skill (e.g. writing) Increase in talkativeness Relaxation	BAC 0.05–0.08%
Altered perception of the environment Ataxia Hyper-reflexia Impaired judgement Lack of coordination Mood, personality and behavioural changes, nystagmus Prolonged reaction time Slurred speech	BAC 0.1–0.15%
Amnesia Diplopia Dysarthria Hypothermia Nausea Vomiting	BAC 0.2–0.3%
Respiratory depression Coma Death	BAC > 0.3%

Alcohol intoxication and the presentation and management of head injury

TBI presentation, management and prognosis depend on a variety of factors, including the following: (1) the mechanism of injury i.e. penetrating, or closed head injury; (2) the severity of primary insults, such as diffuse axonal injury, intracranial haematomas and haemorrhage, or contusion. Which are usually detected by computed tomography (CT) scans of the head; (3) the presence of secondary insults such as hypotension and hypoxia; (4) the severity of coma associated with it, usually assessed by the GCS score. While several modifications have been suggested, the GCS score, first introduced by Teasdale and Jennett in 1974, remains the standard for objectively assessing the severity of TBI. Based on the GCS score, TBI is classified into mild (GCS 13–15), moderate (GCS 9–12) and severe (GCS 8 or less) (Valadka, 2000). The GCS score, particularly its motor component, correlates with survival and functional outcome. Addition of information about age, pupil reactivity, CT scan findings and the presence of secondary insults and other systemic injuries significantly improve the prediction reliability of the initial GCS score (Murray et al., 2007; Steyerberg et al., 2008).

However, the GCS score can be unreliable if the patient has been sedated or paralyzed in the

field to facilitate intubation and the verbal score cannot be assessed in intubated patients (Shahin et al., 2010).

Teasdale and Jennett postulated 3 years after their score's description; that the most reliable GCS score is done 6 h after the onset of the comatose state (Jennett et al., 1977). This time interval was chosen to stabilize the vital signs of patients and avoid temporary factors that can cloud consciousness, such as hypoxemia, hypotension and alcohol. This does not necessarily mean that obtaining a GCS score before 6 h is not helpful, but rather that scores obtained under a certain BAC, for example, should be documented the same way, a lack of the verbal component of the GCS score is documented in an intubated patient. One GCS score gives a snap shot of the patient's consciousness at a certain point, but decision making usually relies on a series of scores to determine deterioration, stability, or improvement. Some studies report that intoxicated head-injured patients' GCS score improves rapidly in a period of time that coincide with alcohol metabolism and elimination (Jagger et al., 1984; Shahin et al., 2010).

Guidelines for the management of head-injured patients specify numerous clinical decisions that have to be made rapidly and are contingent on the presence of predictor variables that identify at risk population. These variables when present are predictive of the presence of a clinically important brain injury (Vos et al., 2002). The variables include conditions that are not affected by intoxication such as: the presence of focal neurologic deficits and post injury seizures, patients with bleeding disorders and/or using blood thinners and anticoagulants, the presence of skull fractures or clinical signs that indicate skull fractures, age below 2 years and above 60, high-energy accidents and persistent headache. But more importantly, they include: loss of consciousness whether transient or consistent i.e. GCS score less than 15, post traumatic amnesia, vomiting that is persistent after the injury and irritability and/or abnormal behaviour. The later are signs that are very common with intoxicated patients with or without head injury. Some guidelines have included alcohol intoxication as one of these variables that should draw attention to the possibility of a clinically significant and a complicated brain injury (NIH, 2007). Critical decisions about head-injured patients' transport, admission, intubation, observation, CT scanning, surgical intervention, etc. are all dependant on the presence of loss consciousness and the patient's GCS score (NIH, 2007). Unfortunately, alcohol intoxication can confound both the diagnosis of brain injury as well as the classification of the seriousness of the injury.

It is difficult to imagine all the clinical scenarios where alcohol intoxication can complicate the decision-making process in TBI patients; however, these are some examples.

When the brain injury is mild, signs of alcohol intoxication can mimic TBI and confuse the diagnosis. An intoxicated patient might not be able to give accurate history. Confusion, amnesia, dizziness, nausea, vomiting and headache, can be easily interpreted as symptoms of mild TBI (Menon et al., 2010). With more severe degrees of brain injury, intoxication can lead to miscategorization of the severity of the injury. When the clinical picture suggests the presence of severe brain injury, where management includes invasive procedures such as intracranial pressure (ICP) monitoring, or where surgical evacuation of intracranial haemorrhage is more likely indicated, the contribution of alcohol intoxication to the impairment of consciousness is a critical judgement. For example, the current guidelines for management of severe TBI recommend ICP monitoring for patients with GCS score 3–8, who have abnormal CT scan findings (Bratton et al., 2007). If alcohol intoxication confounds the initial neurological examination, a patient who has mild or moderate brain injury may transiently have a GCS score <8. Such patients, however, will rapidly improve, and do not require ICP monitoring. The clinical judgement here is to distinguish such patients from one who truly has severe brain injury but also is intoxicated.

Indications for surgical management of certain traumatic haematomas, such as small epidural and subdural haematomas, are different for patients with GCS < 8 (Bullock et al., 2006). With more severe impairment of the neurological status, surgical evacuation is more likely to be recommended. When alcohol confounds the initial neurological examination, the decision can be more difficult. The severity of the CT scan findings, the nature of the brain injury, as well as, changes in the neurological examination during the first few hours after injury are important factors in making these distinctions.

In summary, the current approach to patients with TBI is rapid transportation, early intubation, prompt resuscitation, early CT scanning, immediate evacuation of intracranial mass lesions if indicated and aggressive intensive care management. These methods have helped in improving mortality rates in patients with severe TBI (Marshall et al., 1991). Because of the gravity of the clinical sequellae for delayed intervention in TBI, it is recommended not to delay any assessment or treatment intervention even if alcohol is suspected or detected.

Chronic alcoholism and head injury

Cunningham et al. (2002) reported that persons involved in motor vehicle accidents having tested positive for alcohol were approximately twice as likely to have more severe CT scan lesions, than those who tested negative for alcohol. Others found that alcohol abuse before the injury, rather than alcohol intoxication levels at the time of injury, had a significant effect on the severity of intracranial injuries (Andelic et al., 2010). Some explain this by the fact that chronic intoxication can lead to serious and persistent changes in the brain. This damage may be a result of the direct effects of alcohol on the brain or may result indirectly, from a poor general health status or from severe liver disease. Researchers have not found conclusive evidence that any one variable is solely responsible for the brain deficits found in alcoholics. Characterizing what makes some alcoholics vulnerable to brain damage whereas others are not remains the subject of active research (Oscar-Berman and Marinkovic, 2003).

Furthermore, the statistics establish a very solid link between increased alcohol consumption prior to head injury and the increased incidence of injury. More and more data are now emerging on the post injury incidence of substance abuse especially alcohol and chronic alcoholism. Most of the data suggest that at least, the pattern of intoxication does not really change post injury. In places where post head injury rehabilitation focused on alcohol abstinence completely or for a period of time, there is marked decrease in alcohol consumption. However, long-term surveys show return after 1 year (Ponsford et al., 2007). These data are consistent with previous data that reinforces the evidence that pre-injury alcohol intoxication is highly predictive of post-injury abuse (Kreutzer et al., 1996; Bombardier et al., 2003). A small percentage of the patients with no prior alcohol problems develop problems post injury (Ponsford et al., 2007). The obvious reasons for these results are the numerous psychological, behavioural and cognitive complications associated with post TBI recovery, however, the research has not been able to pin point TBI itself (rather than disability in general) as the cause of these problems (Bjork and Grant, 2009). What is clear is that alcohol abuse complicates head injury and head injury rehabilitation and renders the patient vulnerable for possible further head injury.

Alcohol as a treatment of head injury

The science is not yet complete on the issue of whether alcohol is neuroprotective after brain injury. Some animal studies have reported that alcohol has neuroprotective properties either alone or in combination with caffeine (Kelly et al., 1997; Janis et al., 1998; Strong et al., 2000; Dash et al., 2004; Tureci et al., 2004). This is thought to be mainly a result of

inhibition of N-methyl D-aspartate-mediated excitotoxicity (Chandler et al., 1993). Pre- or early post injury administration of ethanol had also demonstrated reduced intracellular calcium accumulation, hyperglycolysis, lesion size and improved functional recovery (Inglis et al., 1990; Shapira et al., 1990; Kawamata et al., 1992). These effects were not unique to alcohol but rather to any substance that inhibits N-methyl D-aspartate-mediated excitotoxicity. In both animal and clinical studies, higher doses of ethanol had many adverse effects on the central nervous system injury including: depressed haemodynamics and respiratory centres resulting in cerebral ischemia, (Kelly, 1995) decreasing regional cerebral blood flow, (Altura et al., 1983; Baughman et al., 1990) impairment of blood brain barrier function, with formation of brain oedema, (Persson and Rosengren, 1977; Brodner et al., 1981; Yamakami et al., 1995) impairment of platelet function and posttraumatic coagulopathy, (Elmer et al., 1984) and formation of free radicals (Flamm et al., 1977).

Documentation of alcohol intoxication after head injury

Although alcohol intoxication can confound the early neurological assessment of head-injured patients, it is important to note that the WHO study that involved 12 countries around the world found that there was no routine recording of alcohol within each of the participating study centres. Whatever collection and recording of information there was, was arbitrary and therefore widely variable (WHO, 2007). This included failure to record alcohol intoxication whether clinically assessed, or through questionnaires, or using the most reliable methods such as breathalyzers and BAC. This reluctance to document intoxication has been seen in previous studies in the United States (Maull et al., 1984; Soderstrom and Cowley, 1987; Simel and Feussner, 1988; Chang and Astrachan, 1988). The reasons cited are usually fears of legal and social implications of labelling a patient intoxicated and also due to the fear of the consequences of under treatment, i.e. assuming that the patient is intoxicated might lead to the assumption that this is his/her only medical problem or delaying treatment till alcohol is metabolized. While cases of under treatment had been reported (Golan et al., 2007), recent research has found that over treatment is much more common (Harr et al., 2011).

Documenting intoxication with head injury is important for several reasons: (1) it is important to document under which circumstances the patient's consciousness was evaluated, the same way it is indicated whether the GCS score was done under the effects of sedation, intubation, etc; (2) identifying intoxicated patients whether for early intervention to ameliorate alcohol addiction or to reduce the incidence of long-term disability associated with TBI and alcoholism is worthwhile; (3) not testing can mean missing life threatening conditions such as acute alcohol intoxication.

Some of the possible recommendations to establish consistency in accurate documentation is as follows: (1) ongoing training for emergency department staffs to raise awareness of the extent to which alcohol misuse and harmful use is contributing to the health of patients; (2) development of clinical practice guidelines for the emergency departments to ensure the routine documentation of alcohol involvement, and feed back about the usefulness and reliability of data collected.

Research in the field of early biomarkers for TBI could also be useful in identifying such cases rapidly and independently of any confounding substances. One attempt to use S100B levels was unsuccessful (Lange et al., 2010). Future research may be more successful.

Conclusions

Alcohol is a major predisposing factor for head injury. Understanding the short- and long-term effects of alcohol intoxication on the brain and its implications on head injury is important in the management of TBI.

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