Alcohol Related Brain Damage (ARBD)

Clinical definition and diagnosis of alcohol related cognitive impairment

Wernicke-Korsakoff syndrome is a well-recognized syndrome, associated with thiamine deficiency and excessive alcohol ingestion. A classical presentation of the syndrome is rare, as presentation is frequently complex and varied (Harper et al 1989). Establishment of diagnosis is confounded by general ignorance concerning the cognitive effects of excessive alcohol ingestion, the related lack of expertise (Anderson et al 1999, Hillman et al 2001), high levels of stigmatization (Cox et al 2004) and the variable presentation of the syndrome (Jacques & Stevenson 2000). Less specific presentation of cognitive deficits caused by excessive alcohol ingestion have promoted the broader diagnostic concept of alcohol related dementia (Victor 1993). However, its nosological distinction remains ambiguous as reflected in both ICD and DSM IV classification systems. Oslin & Carey (2003) have described clinical criteria; informed through the work conducted in Liverpool (Saunders et al 1991), the NINCDS/Alzheimer’s Disease and related Disorders Association (NINCDS/ADRAD) and the California Criteria for Ischaemic Vascular Dementia. The criteria have recently been validated (Oslin & Carey 2003). The early establishment of more prolonged or permanent effect of alcohol excess and thiamine deficiency on the brain is frequently confounded by acute confusional states associated with intoxication, alcohol withdrawal and physical illness (Cox et al 2004). Hence, Oslin’s criteria for diagnosis of alcohol related dementia imposes a period of 60 days of abstinence (Victor et al 1971) so as to reduce the likelihood of including patients with more transient cognitive impairment. Also, Oslin specifically excludes other and often associated causes of cognitive damage including early cerebrovascular disease and brain trauma, which are both common in this population (Weinstein & Martin, 1995, Bleich et al 2000). Notably, Oslin’s criteria include the employment of the Mini Mental State Examination; utilising a cut-off score of 14 in order to establish a diagnosis of dementia. The use of this instrument and a specific cut-off has implications regarding sensitivity: Patients with relatively early ARBD will not be identified as many present with frontal lobe signs due to secondary damage (Chiang 2002) which the MMSE does not cater for. Alternative instruments are likely to be more sensitive; for example the Addenbrooks Cognitive Examination (Mioshi et al 2006).

More recently, the more pragmatic term of ‘alcohol related brain damage’ (ARBD) (Lishman 1998) has been adopted by Jacques and Stevenson (2000). This concept has the advantage in that it facilitates differentiation from ‘dementia’, which is usually defined as a ‘progressive’ syndrome. As most patients with ARBD do not deteriorate without further exposure to alcohol, this is an important distinction with potential implications for the commissioning of services (Smith & Atkinson 1995). ARBD is a clinical syndrome characterized by two seminal features; prolonged cognitive impairment and a causative link to excessive alcohol ingestion and thiamine deficiency. It covers a wide range of alcohol related cognitive and neurological syndromes, including Wernicke’s encephalopathy, Korsakoff’s syndrome, alcohol dementia, cerebella atrophy, hepatic encephalopathy and frontal lobe dysfunction involving elements of both cortical and subcortical dysfunction (Schmidt et al 2005).

Issues
From a paractical, clinical perspective there are a number of issues which need consideration when defining the condition and providing guidance relating to the diagnostic process:

1. Patients presenting with ARBD will present with cognitive dysfunction over and above that defined by a specific diagnosis of WE/KP definition.

2. Cognitive dysfunction associated with alcohol excess and thiamine deficiency can be classed into three domains:
   - Acute global confusional state associated with the presenting encephalopathy that may take up to two months to resolve
   - Non permanent cognitive dysfunction that may take up to 3-5 years to resolve
   - Permanent cognitive dysfunction

Any pathway of care must cater for these stages within the presentation; including the more transient confusional state that may resolve within the first 2-3 months.

3. A significant proportion of patients will present with evidence of cerebro-vascular disease and history of brain trauma (Wilson et al 2012). The long term effects of these concomitant disorders can only be resolved after a number of years during which more transient damage will improve. At the time of
presentation it is often very difficult to differentiate between the cognitive deficits directly associated with alcohol and thiamine deficiency, concommitant vascular damage which is often evident on scanning and cerebral trauma with which the patient may also present. The adoption of the more generic term ‘Alcohol Related Brain Damage’ can be employed to cover most of these presentations (fig 1).

Figure 1: ARBD components

The adoption of the more generic term ‘Alcohol Related Brain Damage’ can be employed to cover most of these presentations (fig 1).

The diagnosis of ARBD can be determined by adaptation of Oslns criteria;

**Proposed ARBD criteria (adapted from Oslns criteria)**

Principal; the main issue is to establish that excessive and long term alcohol ingestion significantly contributes towards the development of cognitive dysfunction. Clinicians may well be presented with patients with alcohol related brain damage, complicated by vascular change and/or trauma. In these situations, provided alcohol is recognised as the major cause and vascular disease or trauma is considered as secondary then the primary diagnosis should be ARBD.

A. Criteria for the clinical diagnosis of probable ARBD include the following;
   1. Evidence of cognitive impairment. (as demonstrated by clinical examination or use of appropriate instruments; e.g ACE-R)
   2. Significant alcohol use as defined by the minimum average of 35 standard drinks per week for men and 28 for women, for a period of greater than 5 years. The period of significant alcohol use must occur within three years of clinical onset of the cognitive deficits.

B. The diagnosis of ARBD is supported by the presence of the following:
   1. Alcohol related hepatic, pancreatic, gastrointestinal, cardiovascular or renal disease or other end organ damage.
   2. Ataxia or peripheral polyneuropathy (not attributable to other non alcohol related causes).
   3. Neuroimaging evidence of cerebellar atrophy, especially of the vermis
   4. Cognitive damage and evidence of ventricular or sulcal dilatation are likely to improve within the first 60 days, residual damage will be slower to improve and may be permanent.

C. The following clinical presentation indicates that there may be complicating conditions such as vascular or traumatic lesions
   1. The presence of language impairment, especially dysnomia or anomia
2. The presence of focal neurological signs or symptoms (except ataxia or peripheral sensory polyneuropathy)
3. Neuroimaging evidence of cortical or subcortical infarction, subdural haematoma or other focal brain pathology
4. Elevated Hachinski Ischemia scale score
References


Cox S, Anderson I, McCabe L. A fuller life; report of the expert committee on alcohol related brain damage. Stirling: Dementia Services Development Centre. 2004. 13-3-2010. Ref Type: Online Source


