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Alcohol-related brain injury

ADDICTION MEDICINE

Brain changes due to alcohol misuse are often misdiagnosed or not recognised.

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THE behavioural manifestations of alcohol and other drug use are well known, especially in the context of intoxication. However, the 'hidden', long-term effects are less well known.

Brain changes due to regular prolonged alcohol use at harmful levels are often misdiagnosed or not recognised by the general population and even, to a significant degree, by health professionals.

Alcohol-related brain damage has been estimated to occur in 10% of the alcohol-drinking adult population.

Biochemical and morphological changes in the brain due to prolonged alcohol and other drug use will impact a person's day-to-day life, even if only at a subtle level.

How drugs act on the brain

The pleasure centre

All drugs that affect mood act on a part of the brain termed the "pleasure centre" by Olds and Milner in 1954. They observed that rats would forgo food, liquid, sleep and sex in order to keep pressing a lever that stimulated a particular area of the brain, which was later found to correlate with the part of the brain that is stimulated by alcohol and other drug use in humans.

The release of dopamine in the pleasure centre produces the mood changes. Virtually all drugs that are misused either directly or indirectly augment dopamine. Prolonged drug use causes chronic dopamine depletion, which manifests as depressed mood, cravings, lethargy, anhedonia and an amotivational syndrome.

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Continued drug use, even when physiological tolerance has developed, is the person's attempt at trying to feel 'normal'. In other words, the person feels the need to take the substance to maintain the normal level of dopamine, rather than just wanting the mood altering effects they experienced when they first took the drug.

This phenomenon, along with wanting to avoid the physical withdrawal symptoms is a common motivation for continued drug use.

The depressed mood, anhedonia and amotivational syndrome are not often responsive to psychotropic medications, such as antidepressants, and efforts to induce enjoyment by 'getting a hobby' or interest that afforded them pleasure in the past can also be in vain. Discussion of 'stimulating' or meaningful activities is usually more productive.

Long-term effects of alcohol on the brain

Evidence over the past 40 years has shown that significant brain injury occurs with regular prolonged use of alcohol at harmful levels (more than 80g daily).

If a person is over 40 and has at least two of the following characteristics, there is a high probability that they will have alcohol-related brain changes that will impact cognition and day-to-day functioning:

- A history of drinking alcohol at harmful levels for the previous 10 years
- Poor nutrition
- A history of regular binge drinking
- Previous participation in medically assisted detoxification programs
- One or more episodes of Werneke's encephalopathy.

The manifestations of alcohol-related brain injury range from a subclinical syndrome to Korsakoff's syndrome.

Signs that may indicate alcohol-related brain injury include:

- Deterioration in day-to-day organisational abilities
- Deterioration in problem-solving skills and goal setting

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- Increase in 'concrete thinking' — the person's thinking style/processes become more rigid
- Reduced ability to manage change with a greater reliance on routine
- Reduced insight into their own behaviour and its impact on others
- Reduced learning capacity such as an inability to learn new instructions/procedures in the workplace.

The cognitive deficits become more apparent in new environments and can be masked by continuing routines or usual behaviours.

These abilities are generally referred to as executive functions and usually relate to frontal lobe function.

The affected individual may also experience short-term memory problems. However, the attention span is usually within normal limits. The patient may confuse blackouts related to alcohol binges with short-term memory problems, so it is important to distinguish between the two. Blackouts are related more to the rate of drinking rather than the amount consumed.

Visual-spatial and perceptual-motor abilities may also be impaired. This can manifest as problems in performing tasks that require eye-hand co-ordination. Reaction time may also be impaired.

People with significant alcohol-related brain injury may also:

- Invent stories in order to compensate for memory loss
- Confuse dates of significant events
- Display a lack of insight or acceptance of a link between their symptoms and their alcohol use
- Demonstrate a malleable reactivity in conversations
- Appear to have minimal empathy
- Prefer to live a very restricted lifestyle in a restricted environment.

People with alcohol-related brain injury may have slower reaction times ... and can get disoriented and panic more easily.

Well-learned behaviour patterns such as driving a car can remain intact. However, people with alcohol-related brain injury may have slower reaction times, have difficulty following traffic movements, and can get disoriented and panic more easily. Literary skills are not usually affected to any significant degree.

Imaging

While imaging (MRI or CT) is not required for a diagnosis of alcohol-related brain injury, and there is no evidence of a correlation between the imaging findings and the severity/nature of the injury, the following findings support the presence of the condition:

- Generalised cerebral atrophy
- Widening of the cortical sulci, in particular the Sylvian fissure
- Enlargement of the ventricles, in particular the third ventricle
- Demyelination of the corpus callosum (Macchiafava-Bignami disease).

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Wernicke's encephalopathy is a neurological condition associated with significant thiamine deficiency. It is often paired with Korsakoff's syndrome on the basis that if the former remains untreated, it can lead to the latter. However, they are two distinct conditions.

Signs of Wernicke's encephalopathy include:

- Disorientation with confusion of time and place
- Oculomotor disturbances — ophthalmoplegia, nystagmus
- Ataxia marked by wide-based gait (was known as 'sailor's walk'). Cerebellar atrophy, which also causes a wide-based gait, is a differential diagnosis.

The presence of one or more of the above suggests Wernicke's encephalopathy in the context of significant alcohol use and poor nutrition.

Thiamine is the recommended treatment for Wernicke's encephalopathy.

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Managing alcohol-related brain injury in general practice

Suspicion of alcohol-related brain injury should be followed by formal psychometric assessment, and comprehensive biographical and alcohol-use history.

Alcohol-related brain injury can occur in people with other mental health issues, such as mood disorder, congenital or acquired/traumatic brain injury and dementia.

Discussions with family members and significant others may be helpful in clarifying the clinical presentation.

Physical examination and blood tests can help identify signs of prolonged alcohol use in other areas of the body, in particular the liver.

Neurological examination can also be useful. However, alcohol-related neurological signs may not be present, even in the presence of significant alcohol use disorder.

Once identified, a patient with alcohol-related brain injury needs to be made aware of the condition, the impact it is having on their thinking, the effect their ongoing alcohol use will have on the brain, and in particular, the reversibility of their current symptoms. This needs to conducted in a calm, supportive manner.

Recovery of cognitive deficits is possible in many cases and occurs mainly in the first 6-8 weeks.

The patient should be encouraged to cease alcohol, if not forever, at least for the foreseeable future. This may require the involvement of psychological and support services, as well as the patient's family and friends.

The psychologist can also discuss such topics as memory retraining strategies, sleep hygiene, and motivation enhancement strategies regarding maintaining abstinence and lifestyle change.

Nutritional deficits should be addressed; in particular, thiamine should be prescribed.

The GP can, with the support of the psychologist, advocate for the patient at their workplace, if appropriate. The possibility of guardianship and power of attorney may also need to be considered given the results of cognitive assessment.

Follow-up

Ideally, patients with alcohol-related brain injury should be monitored at least monthly for a year after first presentation and initial detoxification. They should also be encouraged to continue ongoing psychotherapy as long-term use of alcohol at harmful levels can induce a protracted depression.

The patient may also experience symptoms of a generalised anxiety disorder and/or social phobia, as well as sleep problems, which may respond to antidepressant medications (for example, mirtazapine).

Recovery of cognitive deficits is possible in many cases and occurs mainly in the first 6-8 weeks. Given continued abstinence, daily thiamine, multivitamins and good nutrition, improvement can still occur up to a year later.

The support of significant others should be used and support groups, such as Alcoholics Anonymous, can be of great value.

Regular blood tests can be used to monitor recovery and provide incentive for continued abstinence. ●

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References

1. WHO. Neuroscience of psychoactive substance use and dependence. 2004; online. See: bit.ly/2alRoRF
2. Fals-Stewart W, et al. Neurobehavioural consequences of alcohol and substance abuse: A review of the findings and treatment implications. *Clinical Psychology Review* 1994; 14:755-78.