Problem based review: Alcohol-use disorders on the Acute Medical Unit

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Abstract

Alcohol-use disorders including acute intoxication and withdrawal are common in the acute medical setting. Acute physicians should be aware of the indications for inpatient detoxification, and be able to liase with specialist alcohol services in the hospital and in the community to determine those patients for whom community-based detoxification may be beneficial. Additionally, it is important to recognise the benefit of Brief Interventions for higher-risk drinkers who are not yet dependent. For patients with confusion and a possible history of high alcohol intake and malnutrition, acute physicians should maintain a high index of suspicion for Wernicke's Encephalopathy and treat appropriately with parenteral thiamine.

Introduction

No acute medical take would seem complete without at least one admission directly related to alcohol use, and all acute physicians should be aware of the importance of alcohol-use disorders. Recent Government statistics indicate that there were just over one million alcohol-related admissions in England in 2009/10, an increase of 12% on the previous year and more than double the number seen in 2002/3.[1] While in many cases alcohol use is a complicating factor in the assessment and treatment of patients presenting with other primary diagnoses, problems primarily related to alcohol are common and failure to diagnose and subsequently treat correctly can lead to severe disability or even death.

The following clinical scenarios, which are fictitious but based on several different real cases managed by the authors, are presented to illustrate some of the potential problems associated with alcohol-use disorders that are commonly encountered in the acute medicine setting.

Case History 1

A woman in her late 30s presented to the emergency department with palpitations, insomnia and feelings of anxiety that seemed to occur in the mornings. There was no significant past medical history, no regular medication and no allergies. She had an office job and lived with her 8-year-old daughter. On further questioning she reported that she had been drinking 2 bottles of red wine per night for the past few months since becoming estranged from her partner. She added that she had started drinking smaller amounts earlier in the day as she felt that it helped her anxiety.

On examination she was tremulous and sweaty. She was mildly tachypnoeic and tachycardic and her blood pressure was 155/85. She had facial erythema, but no signs of chronic liver disease. Her ECG showed a sinus tachycardia with occasional premature ventricular complexes. She was diagnosed with an alcohol withdrawal syndrome and referred to the community alcohol team for an urgent assessment and management plan. Child safety and protection issues were identified and referral to child and family services were made.

What are the key features of alcohol withdrawal syndromes?

In the brain, ethanol promotes activity in the inhibitory gamma-aminobutyric acid (GABA) system and diminishes the excitatory activity of the N-methyl D-aspartatic acid (NMDA) system, resulting in the sedative effects of acute intoxication. Regular use of alcohol promotes the downregulation of GABA receptors and upregulation of NMDA receptors, leading to tolerance.

As plasma ethanol concentration declines when a patient stops drinking, reduced GABA system activity contributes to anxiety and insomnia, while reversed inhibition of the NMDA system promotes neuronal excitation, reducing the seizure threshold. Additionally, noradrenaline release during acute withdrawal results in sympathetic hyperactivity symptoms such as tremor, anxiety, agitation and diaphoresis. Increased dopaminergic transmission may be responsible for withdrawal hallucinations.[2] Features of acute alcohol withdrawal syndromes are shown in Table 1.

At their most severe, these physiological changes can manifest in seizures, and / or *delirium tremens* (DTs), characterised by sympathetic hyperactivity, delusions and hallucinations. This may be followed by hyperpyrexia, ketoacidosis and circulatory collapse.

What are the management issues for this patient?

The patient has developed alcohol dependence and has started drinking to relieve withdrawal symptoms.

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Table 1. Features of acute alcohol withdrawal syndromes.

Autonomic hyperactivity	Gastrointestinal	Cognitive and perceptual changes
Sweating	Anorexia	Anxiety
Tachycardia	Nausea	Vivid dreams
Hypertension	Vomiting	Delusions
Insomnia	Dyspepsia	Hallucinations
Tremor		Delirium
Fever		

Medication will be required both to control these symptoms and to prevent complications such as DTs and seizures.[3] This could be provided at home, but since her home environment is likely to be contributing to her drinking, an inpatient detoxification is likely to be a better option.

In this case the community alcohol team would be best placed to organise this, as a residential unit is usually a better environment than an acute hospital to provide the psychological and emotional support required. Patients need to be supported by trained staff in making changes to their drinking and coping behaviours to sustain abstinence and prevent relapse. Detoxification in community settings is also cost effective.[4] Therefore all hospitals should have pathways with their local alcohol services to enable rapid referral.

Additionally, there are social issues that need to be addressed. The patient has a dependent child, who will need to be looked after while she is in residential detoxification and who may need additional social services input on a longer-term basis. Clinicians need to identify child protection issues and make appropriate referrals to designated hospital staff and Child and Family Services.

Which individuals are at risk of alcohol withdrawal?

Patterns of alcohol use can be described as lower risk, increasing risk, higher risk and dependent. The definitions for levels of risk used in the NHS[5] are provided in Table 2. An alcohol history will indicate the pattern of use and the most appropriate clinical management for the individual circumstance.

Patients who are physiologically dependent on alcohol will experience some form of withdrawal syndrome within 24 hours of stopping drinking. Symptoms are likely to peak by 30 hours and usually begin to subside by 50 hours,[6] although DTs may start to develop from 48-72 hours after the cessation of drinking. Maintaining a high index of suspicion for alcohol dependence will help ensure that withdrawal symptoms are not missed or misdiagnosed, since many of the patients most at risk will not be able to give a history.

Dependence should be considered in patients with conditions that could be alcohol-related (Table 3). The Alcohol Use Disorders Identification Test (AUDIT)[7] has been shown to be effective in identifying alcohol dependence, and performs better than the easily-memorised CAGE[8] questionnaire.[3] Due to the AUDIT tool's unwieldy length, the Scottish Intercollegiate Guidelines Network (SIGN) recommends that shortened versions such as the Paddington Alcohol Test (PAT)[9] or Fast Alcohol Screening Test (FAST)[10] be used instead in the emergency department for people with a potentially alcohol-related presentation.

Dependent drinkers are more likely to be males who are divorced, separated or never married, who started drinking and/or smoking cannabis at a young age as well as being involved in antisocial behaviour.[11] A positive family history of dependence,[12] genetic influences,[13] and coexisting severe depression and anxiety[14] are also predictors.

In 2007, 9% of UK adult men and 4% of women showed indications of alcohol dependence.[1] These patients often have other substance abuse problems or psychiatric diagnoses, and their presentation with withdrawal symptoms can be a good opportunity to consider appropriate referrals and/or treatment for these problems.

What is the appropriate management for alcohol withdrawal?

Medical management of acute alcohol withdrawal should aim to prevent the development of potential complications. SIGN guidelines[3] suggest that medication may not be required if the patient does

Table 2. Definitions for levels of risk.

Risk	Men	Women
Lower Risk	No more than 3-4 units per day on a regular basis	No more than 2-3 units per day on a regular basis
Increasing Risk	4 or more units per day on a regular basis	3 or more units per day on a regular basis
Higher Risk	8 or more units per day on a regular basis or 50+ units per week	6 or more units per day on a regular basis or 35+ units per week

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Table 3. Chronic alcohol consumption features.

Direct toxicity of alcohol:
Dyspepsia/Gastritis/Peptic ulcer disease
Hypertension
Immunocompromise
Cardiomyopathy
Proximal metabolic myopathy
Peripheral neuropathy
Osteoporosis
Hyper LDL cholesterolaemia
Cancer (of head and neck, oesophagus, breast, rectum, amongst others)
Pancreatitis
Alcoholic liver disease (fatty infiltration, alcoholic hepatitis, cirrhosis)
Haematopoietic abnormalities (anaemia, thrombocytopaenia)
Adverse foetal effects (low birthweight, miscarriage, prematurity, foetal alcohol spectrum disorders)
Signs of chronic alcohol misuse:
Spider telangiectasias or angiomas
Caput medusa
Facial and palmar erythema
Hyperpigmentation
Jaundice from alcoholic liver disease
Gingival inflammation and parotid swelling
Nutritional deficiencies including nail changes
Enlarged or shrunken liver
Ascites
Liver flap/encephalopathy
Tremor
Neuropsychiatric effects:
Depression
Insomnia/frequent late-night awakening
Anxiety disorders
Absenteeism
Relationship difficulties
Paranoia/psychosis
Cognitive impairment
Laboratory markers of heavy drinking
Elevated MCV
Elevated γGT/ALT/AST (typically with raised ALT:AST ratio)

not develop withdrawal symptoms and has been drinking < 15 units/day, for men, or <10 units/day for women.

For the hospital physician faced with a patient experiencing acute alcohol withdrawal, the question is whether acute admission is necessary or desirable, or whether advice to continue drinking while a planned detoxification is arranged in liaison with the specialist alcohol service might be preferable.

National Institute for Health and Clinical Excellence (NICE) guidance (2010) recommends that patients experiencing alcohol withdrawal who have developed, or are at high risk of developing withdrawal seizures or DTs, should be admitted to hospital for unplanned detoxification.[6] Features of a high-risk presentation were reported as having a history of withdrawal seizures or delirium tremens, and the presence of signs and symptoms of autonomic over-activity with a blood alcohol concentration greater than 100mg/100ml. Additionally, patients younger than 16 years presenting in acute alcohol withdrawal should be admitted, while the threshold for admission should be lower in patients who are 16-18 years old or who are frail and therefore more vulnerable to injury.

Where the acute physician decides that unplanned detoxification is unnecessary, the patient must be given advice regarding the dangers of sudden cessation of drinking, and should be put into contact with specialist alcohol services and encouraged to arrange a planned detoxification.

What medication should be used for detoxification?

Detoxification aims to prevent complications and reduce symptoms of withdrawal, usually by giving a reducing dose of a drug active on the GABA system. Benzodiazepines are the most commonly used agents, although as noted in both 2010 NICE guidance[6] and a recent overview of Cochrane reviews,[15] there is little evidence for the superiority of any one benzodiazepine over another, or of this class of drugs over clomethiazole or carbamazepine. Consequently, NICE recommends that withdrawal symptoms should be controlled with either benzodiazepines or carbamazepine. In view of previous concerns about the safety of clomethiazole, which can cause dependence and excessive sedation (particularly if taken with alcohol), the guideline group recommended that this agent only be considered in an inpatient setting.

Benzodiazepines, such as chlordiazepoxide, diazepam or lorazepam, can also reduce a patient's level of consciousness, as well as causing hypoventilation or even paradoxical agitation. A shorter-acting agent such as lorazepam (which is not metabolised in the liver) can be used in the elderly, or where there is evidence of advanced cirrhosis or hepatic encephalopathy.

Other drugs may also have a place in management of alcohol withdrawal, but evidence for their efficacy and safety is currently lacking. Both gamma hydroxy butyric acid (GHB) and baclofen have been suggested as alternative GABA agonists, although baclofen may be less prone to dependence.[15] Alpha-2 adrenergic agonists such as clonidine may be useful as adjuncts to GABA agonists, specifically helping to reduce symptoms of sympathetic overactivity[17] and thus allowing lower doses of other medications.

What is the best withdrawal regimen?

Detoxification regimens in hospital can broadly be split into those that consist of a fixed reducing dose and those that are symptom-triggered (e.g. by the Clinical Institute Withdrawal Assessment for Alcohol [CIWA-Ar]).[16] Most studies comparing the two have used benzodiazepines for symptom control.

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There is evidence that symptom-triggered dosing regimens are associated with lower cumulative doses of benzodiazepines and shorter overall treatment duration, without increased severity of withdrawal symptoms or greater risk of seizures or DTs. However, most of this research has assessed planned withdrawals in a specialist addiction setting, rather than unplanned withdrawals on busy acute medical wards. Accordingly, NICE has recommended the use of symptom-triggered regimes when patients are in hospital (or where 24-hour assessment and monitoring is available) with the caveat that there needs to be adequate numbers of nursing and medical staff who are familiar with the signs and symptoms of alcohol withdrawal and the use of severity scoring systems such as CIWA-Ar.

How should alcohol withdrawal seizures and other complications be managed?

Those with established withdrawal seizures should be treated with intravenous or oral lorazepam, according to NICE guidance. Phenytoin should not be used to control seizures caused by alcohol withdrawal. Established DTs should also be treated with oral or parenteral lorazepam.

Olanzapine and haloperidol are sometimes used in patients with DTs and severe psychosis/hallucinations although both of these antipsychotics may increase the risk of withdrawal seizures. The NICE guideline group preferred the side effect profile of olanzapine to haloperidol, which can cause hypotension and extrapyramidal symptoms.

Patient presenting with both encephalopathy from decompensated liver disease and alcohol withdrawal symptoms can be very difficult to manage; NICE guidance recommends early involvement of a specialist gastroenterology or hepatology team.

When is community detoxification indicated?

A patient's past periods of abstinence, level of alcohol dependence, level of motivation and social support will inform the clinician of the level of intervention required and most appropriate service to which to refer the patient.

Male patients who have limited personal and social resources, those with more severe and longer-term alcohol use, and those with limited coping strategies are more likely to relapse quickly following detoxification. In addition patients who do not engage in aftercare are at greater risk of relapse.[18] These findings support local data gathered by our alcohol service. We recommend that this cohort should be referred to community alcohol teams rather than being admitted for unplanned detoxification due to their greater ongoing support needs following discharge.

Case History 2

A 19-year-old student was brought by ambulance to the emergency department acutely intoxicated after being found lying in the street. He had Glasgow Coma Score (GCS) of 12/15 and some minor skin abrasions were noted, but there were no other external signs of injury and no signs of malnutrition or chronic liver disease. His breath smelt strongly of alcohol-containing beverages. A CT scan of the head was unremarkable.

What are the management options for this patient?

This patient has presented after a drinking binge, but there are no indications as yet of alcohol dependence and so he does not need to be admitted for detoxification. He should be observed and discharged once his consciousness improves.

After establishing that he is not dependent on alcohol and does not need to be admitted for management of this, the key question is whether he would benefit from a brief intervention to reduce the likelihood of his developing dependence in the future.

Alcohol Screening and Brief Intervention (SBI) is a strategy designed to identify increasing and higher risk drinking. The screening varies from setting to setting with the AUDIT screening tool used most in primary care and the PAT or FAST tools preferred in acute settings. A screening result suggestive of problem drinking will trigger an offer of brief advice. In an acute setting this usually consists of health information from the screening clinician connecting the hospital presentation to the patient's alcohol consumption, along with a Department of Health brochure. It is worth noting that only a third of people in a recent UK government survey were aware of the recommended maximum alcohol consumption limits of 3-4 units/day for men and 2-3 units/day for women.[19]

The patient is then offered an outpatient appointment with the alcohol specialist for Brief Intervention sessions. The purpose of these sessions is to more fully assess the patient more completely, and formulate an agreed treatment plan. This may include assisting the patient to cut down on their drinking or stop altogether. The aim is to reduce harm and hospital admissions while ensuring the patient is referred to follow on services as required.[20]

For eight patients screened and provided with brief advice, one will reduce their drinking to within lower risk levels.[21]

Case history 3

A middle aged male patient presented to the emergency department with confusion. No collateral history, including his medical history, was available.

On examination he was tremulous, had a left hemiparesis suggestive of a previous cerebrovascular event, and had signs of chronic liver disease.

A CT scan of the head showed no acute changes. The full blood count showed a macrocytosis, but other routine blood tests were unremarkable. Urine dipstick examination was positive for leukocytes and the patient was treated with trimethoprim for a presumed urinary tract infection.

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The patient became agitated at night and his confusion worsened. Six days after admission his movements were observed to be ataxic and he developed a lateral gaze palsy on the right side. A family member visited for the first time, and expressed concern that the patient was normally lucid and had deteriorated dramatically since they had last seen him. This family member also reported that the patient had been regularly drinking several cans of extra strong lager per night.

What is the cause of the patient's deterioration?

This patient has developed confusion and a focal neurological sign in the form of an ophthalmoplegia. The differential diagnosis is broad and his initial workup was designed to exclude a recent stroke with subsequent treatment for presumed infection.

However, where patients are at risk of malnutrition, especially those with a high alcohol intake, Wernicke's encephalopathy (WE), caused by depletion of intracellular thiamine must also be considered. Derivatives of thiamine are required for many processes including various stages of aerobic respiration. High metabolic demands placed on neurones, such as during acute alcohol withdrawal, combined with inadequate thiamine stores, can lead to cellular acidosis, regional glutamate release and cell death.[22] WE is an acute insult that is initially reversible if adequately treated with thiamine replacement, but if inadequately treated, WE can progress to permanent brain damage characterised by the inability to form short term memories (Korsakoff's psychosis, KP), or death.

Chronic alcohol abusers suffer from reduced absorption of thiamine in the small intestine, and additionally often have vitamin-poor diets, making them more likely to become thiamine deplete.

Ultimately, failure to treat WE and development of KP can have devastating consequences for a patient. 25% of those with KP are so cognitively impaired as to require long-term institutional care. Those patients with impaired mental capacity who pose a risk to themselves or others due to withdrawal from alcohol may need to be held against their will under mental health legislation. The Mental Capacity Act in England and Wales makes provision for the assessment and treatment of such patients if this is deemed to be in their best interests. It should be remembered that this requires discussion with their responsible clinician.[23]

For this patient, the specialist alcohol liaison team was contacted for advice, and a diagnosis of Wernicke's encephalopathy-Korsakoff's syndrome was made. The patient was treated with parenteral thiamine replacement. Unfortunately he developed persistent short-term memory impairment and was unable to return to his previous accommodation. He was transferred to a residential unit for patients with Korsakoff's psychosis.

Table 4. (Features of and risk factors for WE).

Features suggestive of a diagnosis of WE:
Evidence of chronic alcohol misuse and:
acute confusion, decreased conscious level, ataxia, ophthalmoplegia, memory disturbance, hypothermia with hypotension
Risk factors for development of WE:
Acute alcohol withdrawal
Malnourishment
Alcohol-related liver disease
High alcohol intake
Medically-assisted alcohol withdrawal
Hospitalisation for acute illness or other comorbidity

How should WE be managed?

Since patients at greatest risk of WE are usually also those about whom little is known, who are unable to give a history and who cannot be reliably examined, it is vital to maintain a high index of suspicion. Risk factors are listed in Table 4.

Where there is clinical suspicion of incipient WE, the patient must be treated with a high dose IV thiamine preparation (eg. Pabrinex I+II x2 three times daily), according to NICE guidelines (2010). Treatment with parenteral thiamine should continue for a minimum of five days or until a diagnosis of WE has been excluded.

Additionally, prophylactic oral thiamine should be offered to selected harmful or hazardous drinkers and parenteral thiamine should be offered to these drinkers where they appear malnourished or have decompensated liver disease (broadly those with evidence of portal hypertension, such as varices, ascites or hepatic encephalopathy). Local policies vary and in some hospitals parenteral thiamine is used more widely.

Oral supplementation is severely limited by poor compliance and slow intestinal absorption rates. As such, parenteral supplementation is preferred where there is greater need for replacement. Pabrinex (Archimedes Pharma Ltd, Reading, UK) is the only parenteral thiamine preparation currently listed in the British National Formulary. The most important potential side effect of Pabrinex is anaphylaxis.

The administration of intravenous glucose where a patient has a sub-clinical thiamine deficiency can precipitate overt WE, and so intravenous thiamine supplementation should precede the administration of IV dextrose whenever possible, and where IV dextrose must be given immediately, as in hypoglycaemic coma, there should be concomitant parenteral thiamine replacement.

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