BMJ Best Practice

Overview of chronic alcohol use

Straight to the point of care

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Introduction

Chronic alcohol use can be associated with a variety of medical and psychiatric sequelae. In 2019, harmful alcohol use resulted in an estimated 2.6 million deaths globally.[1] Young people are disproportionately affected by alcohol consumption - people aged 20-39 years made up 13% of alcohol-attributable deaths in 2019.[1] Unintentional injuries, digestive diseases, disabilities, and alcohol-use disorders are the leading contributors to the alcohol-related burden of disease.[1]

Related conditions

◊ Alcohol-use disorder

» see our comprehensive coverage of Alcohol-use disorder (https://bestpractice.bmj.com/topics/en-gb/198)

Alcohol-use disorder is a problematic pattern of alcohol use leading to clinically significant impairment or psychosocial stress in the previous 12 months.[2] An estimated 400 million people globally live with alcohol-use disorders.[1] Unhealthy alcohol use includes the spectrum of at-risk drinking and alcohol-use disorders. People with hazardous or harmful alcohol use are at higher risk of developing an alcohol-use disorder, but do not have to develop a diagnosable disorder to suffer harm. While the diagnosis of alcohol-use disorder is made by a comprehensive and compassionate history, physical examination can help establish stigmata of alcohol use. Early identification of unhealthy alcohol use can reduce morbidity and mortality. Routine screening for unhealthy alcohol use may help clinicians identify unhealthy alcohol use, perform brief interventions for those with at-risk use, and treat alcohol-use disorder. Engagement and retention in substance use disorder treatment can be a major clinical challenge; it is recommended that healthcare providers proactively engage people who would benefit from treatment at all stages of readiness for change, including those who are uninterested or ambivalent about receiving treatment.[3]

◊ Alcohol withdrawal

» see our comprehensive coverage of Alcohol withdrawal (https://bestpractice.bmj.com/topics/en-gb/3000096)

Alcohol withdrawal occurs in patients who are alcohol dependent and who have stopped or reduced their alcohol intake within hours or days of presentation. Identify any patient with features of severe alcohol withdrawal early. These patients need urgent treatment. Symptoms such as anxiety, nausea or vomiting, autonomic dysfunction, and insomnia typically begin 6 to 24 hours after the patient's last alcoholic drink.[4] These may progress to severe withdrawal with seizures, psychiatric disturbance, and delirium tremens.[2] [5]

◊ Alcohol-related liver disease (ARLD)

» see our comprehensive coverage of Alcohol-related liver disease (ARLD) (https://bestpractice.bmj.com/topics/en-gb/1116)

ARLD has three stages of liver damage: fatty liver (steatosis), alcohol-related hepatitis (inflammation and necrosis), and alcohol-related liver cirrhosis. All are caused by chronic heavy alcohol ingestion. One systematic review and meta-analysis found that the prevalence of ARLD and proportions of alcohol-attributable cirrhosis and hepatocellular carcinoma are lower in Asia, compared with western countries.[6] Clinical presentation is highly variable. A detailed history of the quantity and duration of alcohol ingestion, together with a physical examination and appropriate laboratory tests, is essential in the diagnosis of ARLD. Key risk factors include prolonged heavy alcohol consumption, presence of hepatitis C, and female sex.

◊ Cirrhosis

» see our comprehensive coverage of Cirrhosis (https://bestpractice.bmj.com/topics/en-gb/278)

The pathological end stage of any chronic liver disease. The most common causes of cirrhosis are ARLD, metabolic dysfunction-associated steatotic liver disease (MASLD [previously known as non-alcoholic fatty liver disease] and associated steatohepatitis), and chronic viral hepatitis.[7] [8] In 2019, alcohol was responsible for 42% of global deaths of people with liver cirrhosis.[1] The evaluation of a patient with suspected chronic liver disease and cirrhosis should begin with a detailed history identifying the presence of risk factors for the different causes of cirrhosis. Patients should then undergo a thorough physical examination in order to elicit any signs of chronic liver disease or complications of cirrhosis. A full panel of blood tests should be undertaken to ascertain the degree of disease severity. The main complications of cirrhosis are related to the development of liver insufficiency and portal hypertension, and include ascites, variceal haemorrhage, jaundice, portosystemic encephalopathy, acute kidney injury and hepatopulmonary syndromes, and the development of hepatocellular carcinoma.

◊ Acute liver failure (ALF)

» see our comprehensive coverage of Acute liver failure (ALF) (https://bestpractice.bmj.com/topics/en-gb/1010)

ALF is a rare, life-threatening, potentially reversible condition defined by a rapid decline in hepatic function, characterised by jaundice, coagulopathy (INR >1.5), and hepatic encephalopathy in patients with no evidence of prior liver disease.[9] [10] [11] ALF is a rare event with fewer than 10 cases per million people per year in the developed world.[12] Chronic alcohol misuse is a significant risk factor for the development of ALF. The diagnosis of ALF is established through a careful history, including chronology of events prior to presentation, physical examination, laboratory studies. Early recognition, diagnosis, and establishment of prognosis are paramount to providing an optimal management strategy in patients with ALF.

◊ Hepatic encephalopathy

» see our comprehensive coverage of Hepatic encephalopathy (https://bestpractice.bmj.com/topics/en-gb/294)

A brain dysfunction caused by liver insufficiency and/or portosystemic shunt. It manifests as a wide spectrum of neurological or psychiatric abnormalities ranging from subclinical alterations to coma.[13] Causation is thought to be multi-factorial, resulting in brain exposure to ammonia that has bypassed the liver through portosystemic shunting. Diagnosis is based on reported neurological deficits combined with laboratory abnormalities showing severe liver dysfunction.

◊ Acute pancreatitis

» see our comprehensive coverage of Acute pancreatitis (https://bestpractice.bmj.com/topics/en-gb/3000118)

A disorder of the exocrine pancreas and associated with acinar cell injury with local and systemic inflammatory responses.[14] The most common presenting symptom is mid-epigastric or left upper quadrant pain that radiates to the back. Epigastric tenderness is typical. Gallstones and excessive alcohol consumption account for approximately 70% to 80% of acute pancreatitis cases.[15] The average amount of alcohol intake in patients with acute pancreatitis is 150 to 175 g per day.[16] [17] The diagnosis is confirmed in most patients by raised serum lipase or amylase (>3 times upper limit of normal). Contrast-enhanced computed tomography (CECT) is only required where there is diagnostic doubt or a failure to improve within 72-96 hours from onset of symptoms.

◊ Chronic pancreatitis

» see our comprehensive coverage of Chronic pancreatitis (https://bestpractice.bmj.com/topics/en-gb/67)

Chronic pancreatitis is most commonly associated with chronic alcohol ingestion (>75%). Unlike recurrent acute pancreatitis, chronic pancreatitis is characterised by epigastric abdominal pain radiating to the back, steatorrhoea, malnutrition, reduced pancreatic exocrine function, malabsorption, diabetes mellitus, and pancreatic calcifications. Diagnosis is based on clinical findings and imaging. Co-factors required to induce alcoholic pancreatitis include anatomical, environmental, and/or genetic factors, as few people with chronic alcohol dependence develop the disease (no more than 10%, and likely <3%).[18] [19]

◊ Wernicke's encephalopathy

» see our comprehensive coverage of Wernicke's encephalopathy (https://bestpractice.bmj.com/topics/en-gb/405)

A neurological emergency resulting from thiamine deficiency with varied neurocognitive manifestations, typically involving mental status changes, gait, and oculomotor dysfunction (ophthalmoplegia). This triad may only be present in up to 16% of patients with Wernicke's encephalopathy; around 19% of patients may present without any classical signs.[20] In people with chronic alcohol dependence, thiamine deficiency is a result of a combination of factors: poor intake, low content of vitamins in alcohol, low storage capacity of the liver, decreased intestinal absorption, impaired conversion of thiamine to its active form (thiamine pyrophosphate), and increased demand to metabolise the carbohydrates in alcohol.[21]

◊ Fetal alcohol spectrum disorders

» see our comprehensive coverage of Fetal alcohol spectrum disorders (https://bestpractice.bmj.com/topics/en-gb/1141)

Refers to a group of conditions that may result from fetal exposure to alcohol.[22] Disorders include fetal alcohol syndrome (FAS), partial FAS, alcohol-related neurodevelopmental disorder, and alcohol-related birth defects. In one 2017 meta-analysis, global prevalence of fetal alcohol spectrum disorders was estimated at 8 of 1000 in the general population, with the prevalence exceeding 1% in 76 out of 187 countries.[23] FAS is characterised by antenatal and postnatal growth restriction, specific facial dysmorphology and structural and/or functional abnormalities of the central nervous system. Prevention is a priority, as brain injury sustained in utero is permanent and is associated with severe physical, behavioural, learning, and mental health problems. Early diagnosis may prevent secondary disabilities, but many clinicians are unaware of, or are confused by, existing diagnostic criteria and terminology.

◊ Assessment of liver dysfunction

» see our comprehensive coverage of Assessment of liver dysfunction (https://bestpractice.bmj.com/topics/en-gb/1122)

Assessment of patients with abnormal liver tests should be guided by history, risk for liver disease, duration and severity of clinical findings, presence of comorbidities, the nature of the liver test abnormality noted, and relevant scans and biopsies.[24] Chronic alcoholic liver disease and acute alcoholic hepatitis are associated with elevation of serum aminotransferases.[25] Elevated gamma-GT correlates with excessive alcohol consumption; however, isolated elevations in gamma-GT are common, and often unhelpful, that many institutions have chosen to delete this test from their liver test panel.

◊ Assessment of delirium

» see our comprehensive coverage of Assessment of delirium (https://bestpractice.bmj.com/topics/en-gb/241)

Delirium is an acute, fluctuating change in mental status, with inattention, disorganised thinking and altered levels of consciousness.[26] Delirium can result from alcoholic ketoacidosis (seen after binge drinking and with chronic alcohol abuse) and can occur in Wernicke's encephalopathy and Korsakoff's psychosis, associated with thiamine deficiency. Delirium can be associated with drug withdrawal (benzodiazepine, alcohol) and this should be considered in every case.[27]

◊ Assessment of polyneuropathy

» see our comprehensive coverage of Assessment of polyneuropathy (https://bestpractice.bmj.com/topics/en-gb/158)

Polyneuropathy is a generalised disease of the peripheral nerves due to damage to the axon and/or the myelin sheath. It most commonly presents as symmetric numbness, paraesthesias, and dysaesthesias in the feet and distal lower extremities (distal symmetrical sensorimotor polyneuropathy). In severe cases, sensory symptoms and signs progress proximally to fit a stocking-glove distribution. Balance and gait may be impaired. Alcohol-use disorder is a risk factor for thiamine and pyridoxine deficiencies, which are possible causes of polyneuropathy. Ethanol-polyneuropathy, associated with alcohol-use disorder, may be caused by direct toxicity of ethanol on the nerve and/or concomitant nutritional deficiencies.

◊ Assessment of upper gastrointestinal bleeding

» see our comprehensive coverage of Assessment of upper gastrointestinal bleeding (https://bestpractice.bmj.com/topics/en-gb/456)

Upper gastrointestinal bleeding refers to gastrointestinal blood loss whose origin is proximal to the ligament of Treitz at the duodenojejunal junction. Causes are multiple, but in developed countries bleeding is usually secondary to peptic ulcer disease, erosions, oesophagitis, or varices. Any history of chronic and excessive alcohol use, intravenous drug use (or other behaviour that places people at risk of contracting hepatitis), or underlying liver disease strongly suggests a variceal bleed. The importance of a thorough history and physical examination cannot be emphasised enough, as they allow rapid triage of patients who need care on the medical ward or intensive care unit and will also prioritise patients for urgent versus emergency endoscopy. Mortality is secondary to hypovolaemic shock.

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This approach is in line with the guidance of the International Bureau of Weights and Measures Service. https://www.bipm.org/en/about-us/

Figure 1 – BMJ Best Practice Numeral Style

5-digit numerals: 10,000

4-digit numerals: 1000

numerals < 1: 0.25

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