International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2024: 7.101

The Pathophysiology and Psychosocial Consequences of Alcohol Consumption

Dr. Gururaj Meldapur[1], Dr. Anita Sharma[2]

[1]P. G. Scholar, Department of Agada Tantra, National Institute of Ayurveda (DU), Jaipur

[2]Professor & HOD, Department of Agada Tantra, National Institute of Ayurveda (DU), Jaipur
Email: *gurum285[at]gmail.com*

Abstract: ***Alcohol is one of the most widely consumed psychoactive substances globally, with deep cultural and social integration. While moderate use may offer some health benefits—such as reduced risk of coronary artery disease and improved appetite—excessive consumption leads to significant physical, psychological, and social harm. Alcohol use exists on a spectrum ranging from abstention to addiction, with increasing levels of harm corresponding to increased consumption and dependency. Chronic alcohol use is associated with a range of disorders affecting multiple organ systems including cardiovascular disease (e.g., hypertension, arrhythmias, dilated cardiomyopathy), liver disease (fatty liver, hepatitis, cirrhosis), gastrointestinal issues, endocrine disturbances (e.g., diabetes, pancreatitis), immunosuppression, hematologic abnormalities, and neurological impairments such as alcoholic neuropathy and Wernicke-Korsakoff syndrome. Additionally, alcohol impairs nutritional status and poses significant risks during pregnancy, potentially leading to foetal alcohol spectrum disorders. Acute effects include euphoria, loss of inhibition, impaired judgment, and in higher doses, coma or death. Chronic psychiatric effects can lead to irreversible neurocognitive dysfunction. The extensive impact of alcohol on health emphasizes the need for awareness, early intervention, and public health strategies to mitigate its harmful consequences. Understanding the complex clinical picture of alcohol-related disorders is crucial for both prevention and effective management.***

Keywords: Alcohol Use Disorder, Alcohol-Related Diseases, Organ System Effects, Foetal Alcohol Spectrum Disorders

1. Introduction

Alcohol is both the oldest and the most widely used psychoactive substance in the world. The use of alcohol is a part of most cultures worldwide, and it is recognized that there are both positive and negative aspects of alcohol consumption. Positive aspects might include the stimulation of appetite, aiding in sleep, and reduction in the incidence of heart disease. The negative aspects include poor judgment, liver disease, hypertension, memory problems, and even death. Of course, as with all drugs, there is a risk of addiction to alcohol, which exacerbates the negative aspects of alcohol use and leads to its own sequelae of complications and disorders.[1] There are six levels of alcohol use: abstention, experimentation, social or recreational use, habituation, abuse, and, finally, addiction. **Abstention** is non-use. **Experimentation** is the use of alcohol for curiosity and without any subsequent drug-seeking behaviour. **Social or recreational use** of alcohol involves sporadic infrequent drinking without any real pattern. **Habituation** involves drinking with an established pattern, but without any major negative consequences. **Abuse** of alcohol is the continuation of drinking despite negative consequences. Finally, **addiction** to alcohol involves a compulsion to drink, an inability to stop drinking, and the progression of major life dysfunction with continued use.[2]

Alcohol-Related Disorders: Alcohol is associated with many physical and mental disorders. Perhaps the most well documented physical disorder is alcohol-related liver disease. Alcohol-induced fatty liver disease and obesity are both associated with progression to cirrhosis.[3] Individuals with mental illness are susceptible to alcohol abuse and dependence. This, in part, may be due to attempts to self-medicate anxiety, mania, or depression. Drinking alcohol in excess tends to worsen underlying psychiatric illness. Excessive use of alcohol is associated with a poorer chance of recovery from anxiety and depressive disorders.[4]

Clinical Picture: As the body adapts to excessive alcohol consumption, tolerance develops. With tolerance, an increasingly greater amount of alcohol consumption is needed to obtain the same physiologic effects. Heavy drinking can lead to blackouts, a failure to recall the events around the intoxication, due to the brain's inability to process and lay down the memory in the hippocampus. Hangovers, which are associated with headaches and nausea, can manifest the next morning after a bout of heavy drinking. Often, as duties and responsibilities lapse, attention to hygiene can wane, and the chronic drinker's manner and behaviour change. Memory lapses or forgetfulness may become more evident.

2. Literature Survey

Alcohol consumption and its health implications have been extensively studied across diverse fields including medicine, psychology, and public health. The literature consistently underscores alcohol as a pervasive psychoactive substance, with historical, cultural, and recreational significance. While moderate consumption has been linked to certain cardiovascular benefits, a broad body of research highlights the profound risks associated with chronic and excessive alcohol use.

3. Method/ Approach

Signs and Symptoms

The first stage of alcohol intoxication is characterised by exhilaration, passion, proper manifestation of the attributes of food and drinks, and creativity of music, song, humour and stories. It does not impair the wisdom and memory and does not cause inability for the senses to perceive their objects.

This first stage of intoxication results in sound sleep and post-waking feeling of freshness. Thus, this stage of intoxication is conducive to happiness.[5]

1) Cardiovascular System:

While it has been consistently shown that light to-moderate drinking reduces the risk of coronary artery disease, there still remain severe risks to the cardiovascular system for people who are heavy alcohol drinkers.[6] Cardiovascular conditions that may result from heavy drinking include hypertension, cardiac arrhythmias, and dilated cardiomyopathy. The incidence of cardiac arrhythmias following excessive alcohol consumption is commonly known as "holiday heart phenomenon" following the observation that supraventricular arrhythmias in alcoholics most often occur on Mondays.[7] While the direct cause of arrhythmias following heavy drinking is not explicitly known, it has been suggested that it could be due to myocardial damage, vagal reflexes, electrolyte or metabolic effects, or changes in conduction and refractory periods. Regardless of the root cause, the incidence of cardiac arrhythmias doubles for heavy drinkers compared with light drinkers.[8] Dilated cardiomyopathy is characterized by an enlarged heart with weakened contraction. Sustained heavy alcohol use is thought to be a major contributing factor to dilated cardiomyopathy.[9]

2) Gastrointestinal System:

Excessive alcohol consumption can cause gastroesophageal reflux disease, gastritis, or ulcers in the lining of the stomach. These can manifest as a burning in the throat or stomach or complaints of dark stools (i.e., melena). In individuals who present with a long history of gastroesophageal reflux disease, there is an increased incidence of Barrett's oesophagus. Chronic excessive alcohol consumption can cause varices, both gastric and oesophageal. When varices rupture, often during severe retching, the individual may present with bright red blood.

3) Hepatic System:

Chronic excessive alcohol consumption is associated with an increased risk for the development of liver disease. Fatty liver is the accumulation of fatty acids in the liver. The pathogenesis of fatty liver is due to the overproduction of protonated nicotinamide adenine dinucleotide from alcohol dehydrogenase, which, in turn, leads to the inhibition of fatty acid oxidation, the citric acid cycle, and gluconeogenesis.[10] It is the inhibition of fatty acid oxidation, as well as an increased synthesis of triglycerides, followed by the inhibition of the secretion of lipoprotein from the liver, which all contribute to fatty liver.[11] Alcoholic hepatitis causes inflammation of the liver along with areas of fibrosis and necrosis. Cirrhosis is characterized by progressive scarring of the liver due to the toxic effects of excessive alcohol use and alcohol's metabolites. Cirrhosis, the most advanced form of alcoholic liver disease, is the leading cause of death among alcoholics. Individuals with a diagnosis of both alcoholic hepatitis and cirrhosis have a death rate of more than 60% over a 4-year period. Most individuals die within the first 12 months of receiving the diagnosis.[12] Whilst the progression of cirrhosis might be halted by abstinence, cirrhosis is very difficult to treat, and the damage to the liver cannot be reversed.

4) Endocrine System:

Pancreatitis, both acute and chronic, is another complication of excessive alcohol use. Pancreatic insufficiency or malabsorption presents with gray, foul-smelling stools that float. Individuals with chronic pancreatitis may have calcifications that can be seen on a plain radiographic film. Diabetes, both Type I and Type II, can be a consequence of excessive alcohol use. The development of Type I diabetes is rare and due to almost complete destruction of the pancreas. Type II diabetes is more common and due to weight gain from carbohydrate ingestion. Hypogonadism and osteoporosis are other complications. Thyroid disease also can be a sequela of excessive alcohol use, abuse, or dependence.

5) Rheumatic and Immune System:

Chronic excessive alcohol consumption has been linked with an increase in illness and death from infectious diseases. Due to alcohol's immuno-suppressive effects, there is an increased susceptibility to bacterial pneumonia, pulmonary tuberculosis, and hepatitis C. There is even some speculation that chronic excessive alcohol users are at increased risk for HIV infection due to lowered immune response, and those with HIV may have a quicker progression from HIV to full-blown AIDS.[12] Gout is a common complication of chronic excessive alcohol consumption. Podagra (pain in the big toe) is a typical complaint. Alcohol use appears to mitigate certain autoimmune conditions such as systemic lupus erythematosus and rheumatoid arthritis.

6) Hematopoietic System:

Anaemias, both macrocytic and microcytic, are possible. Macrocytic anaemia can be due to folate or vitamin B12 deficiency. An increased mean corpuscular volume can reflect macrocytic anaemia. Of note, an increased mean corpuscular volume can also be a result of liver disease when the lipid bilayers that hold the red cell do not form correctly. When liver disease is severe, platelets can be destroyed or can sequester in an enlarged spleen. Microcytic anaemias are related to active bleeding or blood loss and should prompt evaluation for a gastrointestinal disorder or lesion. Sideroblastic anaemia can also occur.

7) Central Nervous System:

The brain is sensitive to alcohol's toxic effects. Areas that are particularly sensitive include the hippocampus and the cerebellum, which can result in memory deficits and dementias as well as abnormal gaits and intention tremors. Rarely, central pontine myelinolysis can occur.

8) Peripheral Nervous System:

Changes in position and vibration sense occur after prolonged excessive alcohol use and are due to vitamin B12 or folate deficiencies, or both. Myopathy can be a rare manifestation of alcohol dependence.

9) Integumentary System (Skin):

Psoriasis vulgaris, acne rosacea, and erythropoietic protoporphyria are all common skin conditions associated with excessive alcohol use. With liver disease, spider nevi, telangiectasias, palmar erythema (reddened palms), spider angiomas, and hepatic porphyrias, particularly porphyria cutanea tarda (bullous erosions, blistering, crusting lesions, and scarred healing with hyperpigmentation or

depigmentation on the face, the side of the neck, and the back of the hands), might be found.

10) Nutritional Status:

Low levels of potassium, magnesium, and phosphorus are common among individuals with severe alcohol dependence. On many blood chemistries, magnesium and phosphorus are not part of the panel. Therefore, it is prudent to check these electrolytes in an alcohol-dependent individual who appears nutritionally compromised. Low levels of potassium can cause additional medical complications (particularly cardiovascular) if not replaced; however, this can be difficult to achieve in the setting of low magnesium. Therefore, magnesium and potassium need to be replenished simultaneously. Thiamine replacement is also often required.

11) Fetal Development:

The consumption of alcohol during pregnancy has been linked with poor birth outcomes, the potential for long-term developmental disabilities, and the manifestation of foetal alcohol spectrum disorder.[13] The clinical manifestations of foetal alcohol exposure fall under the classification of foetal alcohol spectrum disorders. Foetal alcohol spectrum disorders can be further subdivided into four categorical syndromes:[14]

a) **Foetal alcohol syndrome:** A clinical diagnosis of foetal alcohol syndrome requires alcohol exposure, a recognizable facial pattern that includes short palpebral fissures, thin upper vermilion lip, and smooth philtrum, evidence of growth retardation or malformation, and evidence of neurocognitive defects. Foetal alcohol syndrome newborns may exhibit irritability, tremors, hypotonia, and even withdrawal symptoms.
b) **Partial foetal alcohol syndrome:** It is diagnosed when there is confirmation of alcohol consumption during pregnancy and, while not all the features of foetal alcohol syndrome are present, neurocognitive and some craniofacial features are present.
c) **Alcohol-related neurodevelopmental disorder:** Children diagnosed with alcohol-related neurodevelopmental disorder do not typically have the growth retardation or facial features characteristic of foetal alcohol syndrome, but the resulting neurocognitive defects are more pronounced.
d) **Alcohol-related birth defects:** A diagnosis of alcohol-related birth defect requires some of the facial features characteristic of foetal alcohol syndrome, but it is the behavioural features or structural abnormalities that are more prominent.[15] In addition to the physical impairments inflicted by alcohol, there is a spectrum of cognitive problems that children diagnosed with foetal alcohol spectrum disorders exhibit. These problems include difficulties with hyperactivity, sustained and focused attention, cognitive flexibility, learning and memory, and social understanding.[16]

Alcohol affects almost all organ systems through the natural progression of the disease. These are characterized as acute, chronic, and withdrawal effects. The metabolism of alcohol occurs at a rate of about 1 ounce of pure alcohol (2 drinks) eliminated from the body every 3h. Following alcohol consumption, it takes about 15–20 min for alcohol to reach the brain and cause impairment. The maximum blood alcohol concentration is reached 30–90 min following the ingestion of alcohol.[17]

It is generally accepted that the consumption of a standard serving of alcohol (14 g, or 17.74 ml ethanol content) will increase the average person's blood alcohol concentration by 0.02–0.05%. The average person's blood alcohol concentration decreases approximately 0.015% per hour following complete cessation of alcohol intake. A blood alcohol concentration of 0.20% represents very serious intoxication. A blood alcohol concentration ranging between 0.35 and 0.40% could be potentially fatal alcohol poisoning. The accepted LD50 for alcohol—i.e., the dose that is lethal for 50% of the adult human population—is 0.40%.[18]

Acute Effects:

Table 1: The progressive effects of alcohol[19]

Blood alcohol concentration	Changes in behaviour	Activity impairment
0.01–0.05	Relaxation Feeling of well-being Loss of shyness Loss of inhibitions Exaggerated behaviours	Impaired alertness Impaired judgment Minor impairment of memory Minor impairment of reasoning
0.06–0.10	Feeling of euphoria Feeling of pleasure Numbness of feelings Nausea and sleepiness	Impaired coordination Impaired balance Impaired speech Impaired vision Slow reaction time
0.11–0.20	Anger Mood swings Feeling of sadness Confusion Feeling of restlessness Nausea and vomiting Disorientation	Impaired reasoning Impaired depth perception Inappropriate social behaviour Impairment of motor coordination Slurred speech Severely impaired judgment Severe memory impairment Blackouts
0.21–0.30	Aggression Depression Stupor Reduced sensations Nausea and vomiting	Loss of balance Loss of temperature regulation Loss of consciousness May be difficult to awaken

0.31–0.40	Unconsciousness Coma Death possible	Loss of bladder control Difficulty breathing Slowed heart rate
0.41 and greater	Death	

Chronic Effects:
Although the physical effects of chronic alcohol abuse or dependence are well characterized, the psychological and psychiatric consequences are less familiar. Such chronic complications from chronic alcohol abuse or dependence include Wernicke's encephalopathy, Korsakoff's psychosis, alcoholic neuropathy, chronic alcoholic myopathy, and alcoholic dementia. **Wernicke's encephalopathy** is caused by thiamine (vitamin B1) deficiency and is usually diagnosed by a triad of symptoms: ataxia, oculo-motor abnormalities, and global confusion.[20]

Korsakoff's psychosis is a chronic amnesic disorder that can occur in individuals who have had Wernicke's encephalopathy. Like Wernicke's encephalopathy, Korsakoff's psychosis is the result of thiamine deficiency. It is manifested by retrograde and anterograde amnesia, the latter caused by an inability to lay down new memories. While immediate recall remains intact, short-term memory is impaired. Individuals are unaware of their memory deficits, and confabulation is common.[21]

Alcoholic neuropathy is the most commonly reported neurologic complication in people addicted to alcohol. These individuals present with paresthesias, pain, and weakness. These individuals also can have reduced pain and reduced temperature sensations. Typically, there is axonal degeneration and demyelination, possibly due to a neurotoxic effect of ethanol on the peripheral nerves.[22]

Chronic alcoholic myopathy manifests as a painless syndrome wherein the individual has muscle weakness. The severity of the myopathy is related directly to the amount of alcohol consumed. It is thought that chronic alcoholic myopathy is the result of the toxicity of ethanol and its metabolites, such as acetaldehyde, as opposed to nutritional deficiencies.[23]

4. Discussion

The impact of alcohol on human health is complex, multifaceted, and far-reaching. This article highlights the dual nature of alcohol use—ranging from socially accepted, low-risk consumption to pathological, life-threatening addiction. While moderate drinking may confer certain cardiovascular benefits, the overwhelming evidence points to the detrimental effects of chronic and excessive alcohol consumption on nearly every organ system. A significant concern is the development of alcohol-related liver disease, including fatty liver, alcoholic hepatitis, and cirrhosis, which remain among the most common and fatal complications of alcohol misuse. Cardiovascular complications such as hypertension, arrhythmias, and cardiomyopathy demonstrate how even systems that may benefit from moderate intake are severely compromised with higher consumption levels. Furthermore, the psychological and neurological effects, such as memory impairments, blackouts, Wernicke's encephalopathy, and Korsakoff's psychosis, emphasize alcohol's toxic impact on the brain. These disorders not only impair quality of life but often remain underdiagnosed and undertreated due to overlapping psychiatric symptoms.

The systemic consequences—ranging from immune suppression, nutritional deficiencies, endocrine disturbances, and gastrointestinal issues—underscore alcohol's broad physiological impact. Particularly concerning are the effects on foetal development, with alcohol consumption during pregnancy leading to foetal alcohol spectrum disorders (FASD), which have lifelong consequences on physical, cognitive, and behavioural development. Given the breadth of complications associated with alcohol, this discussion reinforces the urgent need for comprehensive public health strategies, early screening, education, and intervention programs. Clinicians should be vigilant in recognizing early signs of abuse, addressing co-morbid psychiatric conditions, and providing support for cessation. Public awareness campaigns and policy-level interventions are essential to mitigate the burden of alcohol-related diseases globally.

5. Conclusion

Alcohol-related disorders are an important global health problem. Not only is there a significant economic burden, but the negative personal effects of excessive alcohol consumption may be both physically and psychologically devastating. The clinical picture of alcohol is different for everyone, but there are consistent themes based on the pathophysiology of alcohol. Intoxication, blackouts, and hangovers are all typical clinical manifestations of excessive alcohol use. Many organ systems may be negatively affected by alcohol consumption. Alcohol related liver disease, holiday heart phenomenon, gastroesophageal reflux disease, and anaemia may all result from the prolonged use of alcohol, especially in excessive amounts. Furthermore, the excessive consumption of alcohol not only harms the individual who is drinking but may also have serious physical effects on the developing foetus. The psychological and psychiatric picture of alcohol consumption can be divided into acute and chronic effects. The acute effects of alcohol consumption, such as a loss of inhibitions and feelings of pleasure and euphoria, are well known, and these well-known effects entice individuals to consume alcoholic beverages. Finally, the continued excessive use of alcoholic beverages may result in severe chronic psychological and psychiatric effects such as Wernicke's encephalopathy or Korsakoff's psychosis.

6. Future Scope

The pervasive effects of alcohol on human health highlight a critical need for ongoing research, intervention, and policy development. Future studies should focus on early biomarkers for alcohol-related organ damage, allowing for timely diagnosis and treatment before irreversible harm occurs. Advancements in neuroimaging and genetic research may help identify individuals most at risk for addiction and related complications, facilitating more personalized and

preventive approaches. There is also scope for improving therapeutic interventions, particularly for managing alcohol dependence and withdrawal. Pharmacological research should explore safer and more effective medications to reduce cravings, improve abstinence rates, and repair alcohol-induced tissue damage, especially in the liver and nervous system.

Another vital area is the development of comprehensive public health strategies to reduce alcohol misuse. These may include educational campaigns, community-based outreach programs, and the integration of alcohol screening in primary care settings. Additionally, research into the socio-economic determinants of alcohol abuse can inform more targeted and culturally sensitive interventions. In summary, the future scope encompasses clinical, preventive, and public health dimensions, aiming to reduce the global burden of alcohol-related diseases through innovation, awareness, and evidence-based care.

References

[1] National Institute on Alcohol Abuse and Alcoholism (2007) Helping patients who drink too much: a clinician's guide
[2] Inaba DS, Cohen WE (2004) Uppers, downers, all arounders: physical and mental effects of psychoactive drugs, 5th edn. CNS Publications, Ashland, OR
[3] Day CP (2000) Who gets alcoholic liver disease: nature or nurture? J R Coll Physicians Lond 34:557–562
[4] Haynes JC, Farrell M et al (2008) Alcohol consumption as a risk factor for non-recovery from common mental disorder: results from the longitudinal follow-up of the National Psychiatric Morbidity Survey. Psychol Med 38: 451–455
[5] R.K.Sharma, Bhagwan Dash. (2000). Charaka Samhita: Text with English translation (Vol. 4, Chikitsa Sthana). Chowkhamba Sanskrit Series Office. Pn 394
[6] Rehm JT, Bondy SJ, Sempos CT, Vuong CV (1997) Alcohol consumption and coronary heart disease morbidity and mortality. Am J Epidemiol 146:495–501
[7] Ettinger PO, Wu CF, De La Cruz C Jr, Weisse AB, Ahmed SS, Regan TJ (1978) Arrhythmias and the holiday heart: alcohol-associated cardiac rhythm disorders. Am Heart J 95:555–562
[8] Cohen EJ, Klatsky AL, Armstrong MA (1988) Alcohol use and supraventricular arrhythmia. Am J Cardiol 62:971–973
[9] Kasper EK, Willem WRP, Hutchins GM, Deckers JW, Hare JM, Buaghman KL (1994) The causes of dilated cardiomyopathy. Clinicopathologic review of 673 consecutive patients. J Am Coll Cardiol 23:586–590
[10] Lieber CS (1994) Alcohol and the liver: 1994 update. Gastroenterology 106:1085
[11] Seitz HK, Sutter PM (1994) Ethanol toxicity and nutritional status. In: Kotsonis FN, Mackey M, Hjelle J (eds) Ethanol toxicity and nutritional status. Raven, New York
[12] National Institute on Alcohol Abuse and Alcoholism (2000) Medical consequences of alcohol abuse. Alcohol Res Health 24:27–31
[13] Bailey BA, Sokol RJ (2008) Pregnancy and alcohol use: evidence and recommendations for prenatal care. Clin Obstet Gynecol 51:436–444
[14] Burd L, Deal E, Rios R, Adickes E, Wynne J, Klug MG(2007) Congenital heart defects and foetal alcohol spectrum disorders. Congenital Heart Disease 2:250–255
[15] Mukherjee RAS, Hollins S, Turk J (2006) Foetal alcohol spectrum disorder: an overview. J R Soc Med 99:298–302
[16] Jacobson JL, Jacobson SW (2002) Effects of prenatal alcohol exposure on child development. Alcohol Res Health 26:282–286
[17] National Institute on Alcohol Abuse and Alcoholism (1997) Alcohol metabolism. Alcohol Alert No. 35
[18] National Institutes of Health: Understanding alcohol [Online]. Available at http://science.education.nih.gov/supplements/nih3/alcohol/guide/info alcohol.html (2006). Accessed 22 June 2009
[19] National Institute on Alcohol Abuse and Alcoholism (2003) Understanding alcohol: investigations into biology and behaviour
[20] Victor M, Adams RD, Collins GH (1989) The Wernicke's-Korsakoff syndrome and related neurologic disorders due to alcoholism and malnutrition. FA Davis, Philadelphia, PA
[21] Shimamura AP, Jernigan TL, Squire LR (1988) Korsakoff's syndrome: radiological (CT) findings and neuropsychological correlates. J Neurosci 10:561–565
[22] McLane JA (1987) Decreased axonal transport in rat nerve following acute and chronic ethanol exposure. Alcohol 4:385–389
[23] Urbano-Marquez A, Estruch R, Navarro-Lopez F, Grau JM, Mont L, Rubin E (1989) The effects of alcoholism on skeletal and cardiac muscle. N Engl J Med 320:409–415

Author Profile

Dr. Gururaj Meldapur from Vijayapura Dist, Karnataka. Graduated from Ayurveda Mahavidyalaya, Hubballi, Karnataka in 2022. Currently pursuing M.D. in Agada Tantra department from National Institute of Ayurveda, Jaipur, Rajasthan since 2023