ejpmr, 2023, 10(2), 15-27

SJIF Impact Factor 6.222

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Review article

ISSN 2394-3211

EJPMR

IMPACT OF ALCOHOL CONSUMPTION AND POISONING ON HUMAN HEALTH

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Article Received on 30/11/2022 **Article Revised on 20/12/2022** **Article Accepted on 11/01/2023**

ABSTRACT

The gravity of alcohol poisoning is profound and it is a leading cause of poisoning in many countries. Alcohol poisoning affects our organs and can lead to fatality. Recognition of alcohol intoxication is challenging and appropriate management is necessary to avoid significant patient morbidity. This review discusses the alcohol use, characteristics of customers, alcohol intoxication and associated disorders. Incidence of alcohol poisoning is gradually increasing across the world including Bangladesh. Previously, from November, 2012 to January, 2013, medicine unit of Dhaka Medical College and Hospital received 8 cases of alcohol poisoning with fatality. Most of the patients were presented without consciousness within 3-7 hours of consuming alcohol. Recently, on February 2, 2021, 12 people including 2 students have died from alcohol poisoning in Dhaka and Bogura district while around 50 people were admitted in hospital with alcohol poisoning. The use of antidote can save the precious lives. A national guideline should be uniformly practiced by physicians to fight against this curse and industrialists should maintain ethics and proper maintenance to prevent further catastrophe.

KEYWORDS: Alcohol, Health effects, Poison, Intoxication.

INTRODUCTION

For centuries, alcohol has been a part of human culture especially in social activities in many countries around the world. (WHO, 2018). As a stuff of abuse, alcohol is the earliest and most spread one (Vonghia *et al.*, 2008). Alcohol consumption have socioeconomic impacts, including the medical costs and health burdens (Room *et al.*, 2005). In recent years, harmful usage of alcohol has increased and its burden on human health, society and economy has risen significantly. (WHO, 2018). Throughout the world, harmful use of alcohol is one of the top 5 factors which can increase the chance of disease, disability and death (Monzavi *et al.*, 2015). Each year 3 million people die due to alcohol toxicity (WHO, 2018), which is also associated with more than 200 diseases (Dewan *et al.*, 2015). In the United States, it is the fourth major avertible reason of death (LaHood *et al.*, 2021).

Alcoholic beverages consist of a wide category of beverages such asgin, vodka, tequila, beer, wine, whisky, rum, wine, pisco. These beverages differ from each other based on ethanol content as well as their manufacturing methods (Cacho *et al.*, 2005). The oral ingestion of ethanol carrying beverages is known as alcohol consumption. For humans, ethanol is the solitary alcohol that is safe. In case of other alcohols, methanol, ethylene glycol and isopropanol are known as toxic alcohol (Collins *et al.*, 2013; Medscape, 2021).

Ethanol is produced by fermenting sugars as well as cereals (Medscape, 2021). In addition to ethanol's extensive availability as a beverage, it is used as an ingredient in mouthwashes, antiseptics, hair tonic, cosmetics, aftershave, dishwashing detergents etc. Ethanol is a popular solvent that is used in the industries (Vale *et al.*, 2007). In the modern world, ethanol is a widely used psychoactive drug. Though it reduces anxiety in moderate doses, ethanol is poisonous in higher doses. Respiratory depression and death can result from elevated doses of ethanol (Adinoff *et al.*, 1988).

If very high amount of alcohol is drunk by a person within a brief spell of time, alcohol poisoning takes place (NHS, 2021). Alcohol poisoning is known as alcohol overdose as well (Webmd, 2020). Based on blood alcohol concentration (BAC), signs and symptoms related to alcohol toxicity occur (LaHood *et al.*, 2021). Excessive alcohol ingestion can cause cessation of the regions of brain that regulates heart rate, breathing and temperature control (NIAAA, 2021). Based on the health condition, quantity and rate of alcohol ingestion,

consumption of other medicines and metabolic resistance of the person drinking, the reaction to alcohol may differ (Kanny *et al.,* 2015). Binge drinking is a very common cause behind alcohol poisoning. Binge drinking is defined as the consumption of 5 or over 5 drinks within two hours for men. The criterion is 4 or over 4 for women (Mayo Clinic, 2021). Binge drinking is eight times more common than drug abuse (Monzavi *et al.,* 2015). Binge drinking before a work day engender demises, which occur due to heart attacks (Shazzad *et al.,* 2013). Probability of bruises during activities rise after alcohol consumption. According to a study, patients without alcohol intoxication show less bruise severity, briefer stay in the hospital, lower treatment cost than those with alcohol intoxication (Peng *et al.,* 2016). In children below 5 years old, ethanol intoxication can lead towards unexpected death. Consumption of tiny amount of ethanol can result in permanent neurological injury due to hypoglycemia in young children who has been without food for 8-12 hours (Vale, 2007).

Methanol is a solvent that is used extensively in the industries. It also has use as paint remover (Medscape, 2021). If an alcoholic beverage is adulterated by adding methanol, methanol poisoning takes place (Doreen *et al.,* 2017). As a result, volume of alcohol increases and the drink becomes cheaper as ethanol is expensive than methanol. Either significant health complications or death may result from ingestion of liquors carrying toxic quantity of methanol (Destanoğlu *et al.,* 2019).

Bangladesh is a country of South Asia (Ministry of Foreign Affairs. Bangladesh An Introduction). Bangladesh is surrounded by The Bay of Bengal on the south and Myanmar on the southeast and also India on the east, west as well as north (Sharmin *et al.,* 2015). The economy of Bangladesh is built on agriculture (Uddin, 2009). There are laws in this country to control the manufacturing, purchase along with consumption of alcoholic beverages (Islam *et al.,* 2014). The majority of Bangladeshi population are Muslims (Population & Religion). Islam forbids drinking alcohol (Islam *et al.,* 2014). But people are consuming alcohol and majority of the drinkers are binge drinkers (Islam *et al.,* 2017). Methanol poisoning is being reported intermittently in recent years (The Business Standard, 2020). People are dying drinking toxic alcohol (New Age, 2021). Deaths because of alcohol abuse have also been reported in the past (Islam *et al.,* 2014).

The objective of this review is to summarize the impact of alcohol poisoning as well as the impact of spurious alcohol on human body and investigate the recent incidents of alcohol poisoning in Bangladesh to get a clear idea for a solution.

MATERIALS AND METHODS

Article search strategy: An extensive literature search was done using several online databases including Web of Science, Scopus, PubMed/Medline, ScienceDirect, Wiley Online Library, and Google Scholar during this review. We have used ‘Ethanol’, ‘Methanol’, ‘Methanol poisoning', ‘Alcohol poisoning’, ‘Alcohol in Bangladesh’ and ‘Alcohol poisoning in Bangladesh’ as key words to gather the related information. Considering peer reviewed and published articles only, describing alcohol consumption and complication and poisoning as inclusion criteria, 68 distinct articles were included in this review.

Chemistry and Pharmacology

Ethanol is a clear and colorless liquid. Apart from its strong taste, it has a smell that resembles wine (NOAA, 2021). It is soluble in water (Vonghia *et al.,* 2008). Mainly it is absorbed from the intestine (Drugs.com, 2021). The major part of metabolism of consumed ethanol happens in liver (Vonghia *et al.,* 2008). In the beginning of ethanol metabolism, ethanol is oxidized to acetaldehyde by Alcohol Dehydrogenase (ADH). The resulting acetaldehyde is again oxidized to acetic acid by Aldehyde Dehydrogenase (ALDH). NAD+ is needed to complete each one of these metabolic steps (Brunton *et al.,* 2018). Ethanol causes depression in the central nervous system (Drugs.com, 2021). There is a higher probability of respiratory depression and arrest at Blood Alcohol Concentration (BAC) of more than 65.1 mmol/L. Concentrations of more than 108.5 mmol/L are found to be associated with death in case of acute alcohol toxicity (Vonghia *et al.,* 2008).

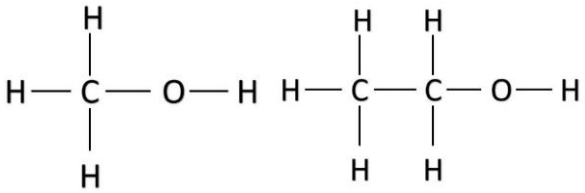

Figure-1: Structure of alcohols- a) methanol and b) ethanol.

Methanol is a liquid without color (CDC, 2020). It evaporates at normal temperature (Chemical Safety Facts, 2020). Methanol has a smell that is sharply strong (Occupational Safety and Health Administration). The flammability of methanol is on a high level (ICSC 0057). Methanol is fully miscible with water (CAMEO Chemicals). The absorption of methanol takes place in gastric mucosa (Medscape, 2021). If methanol is drunk with ethanol, metabolism of methanol occurs only after the total metabolism of ethanol (Amin *et al.,* 2017). Mainly, the metabolism of methanol occurs in liver where it is metabolized to formaldehyde (Medscape, 2021). Like ethanol, methanol also causes depression in the central nervous system (Perri *et al.*, 2022). Permanent sightlessness may result from consumption of only 10ml methanol. 30 ml methanol consumption can cause death (Tulashie *et al.,* 2017).

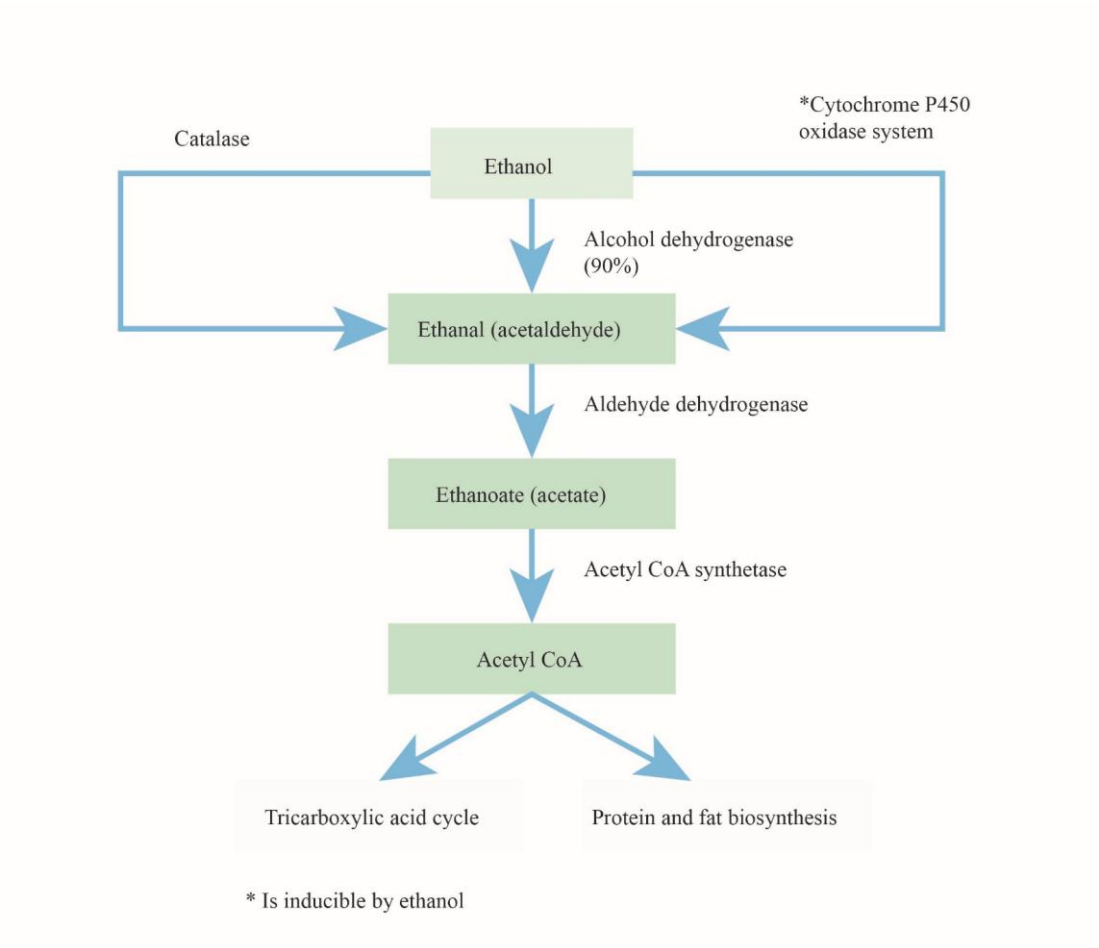

Figure-2: Metabolism of Ethanol.

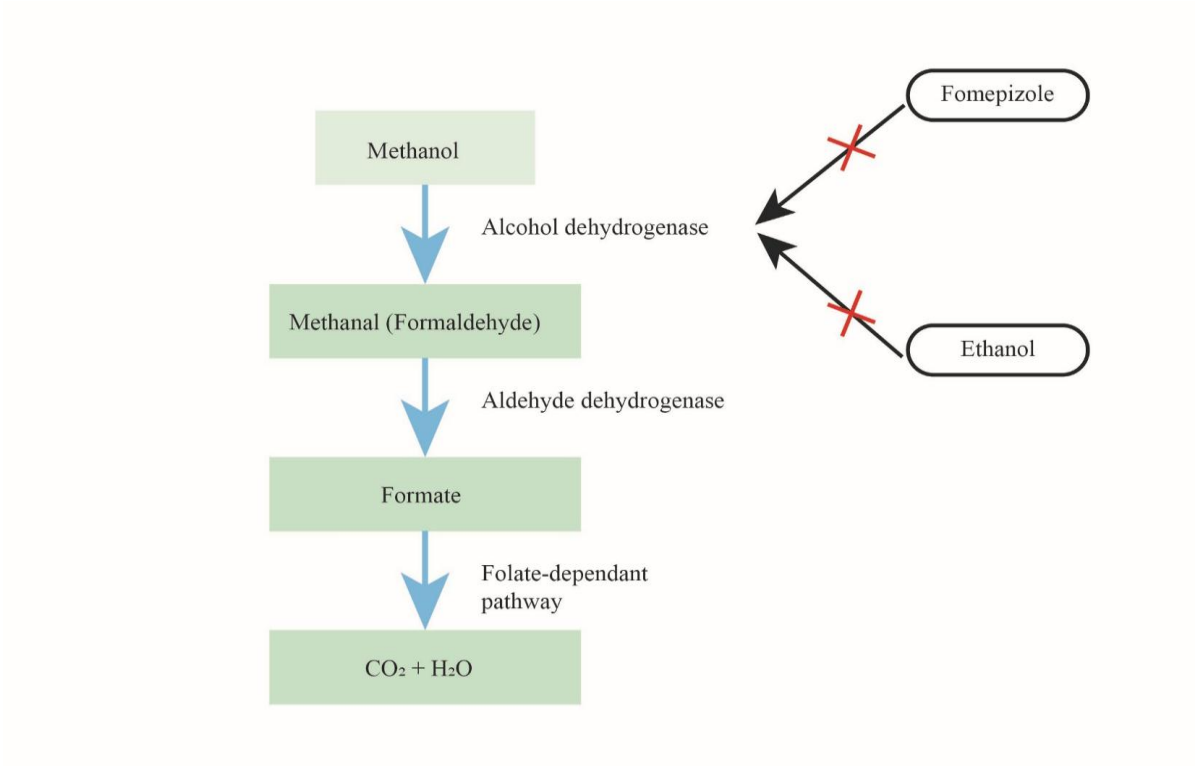

Figure-3: Metabolism of Methanol.

Pathophysiology

In case of acute alcohol (ethanol) poisoning, central nervous system (CNS) is the principal site of action. Ethanol enhances inhibition of CNS and causes reduction in excitation of CNS (LaHood *et al.,* 2021). Chloride ions penetrate the cell after γ-aminobutyric acid (GABA) binds to $GABA_A$ receptors. As a result, there is decrease in cellular excitability (Pitzele *et al.,* 2010). Alcohol binds to GABA receptors with great strength, which causes activation of the inhibitory cascade. Cognitive impairment, sedation, reduced coordination result from this activation (LaHood *et al.,* 2021). Ethanol inhibits glutamate (excitatory neurotransmitter) as well (Pitzele *et al.,* 2010). Rise in the number of NMDA receptor along with enhanced responsiveness of these receptors to glutamate is found in people who have been diagnosed with alcohol use disorder. These people may experience hallucinations and seizures after alcohol withdrawal due to this enhanced responsiveness (LaHood *et al.,* 2021).

In case of methanol poisoning, formaldehyde is formed in the liver due to metabolism of methanol. Getting rid of formic acid is hard and formic acid gathers together (Ashurst *et al.,* 2018). Mostly the toxicity of methanol is due to accumulation of formic acid (Medscape, 2021).

Ingredients That Make Alcohol Spurious

These are the most common ingredients used to produce spurious alcohol:

Table-1: Ingredients responsible for spurious alcohol.

Spurious Ingredient	Justification	Lethal Dose
Methanol	Diluent, Taste Enhancer, Denaturant	1-2 mL/kg
Ethylene Glycol	Sweetener	1.4 mL/kg
Denatonium Benzoate	Denaturant	≤150 g (2)
Pyridine	Denaturant	0.5-50 mg/kg (1)
Naphtha (Mineral Oil)	Denaturant	
Isopropyl Alcohol	Denaturant	2-4 mL/kg (3)
Butanone	Denaturant	50-100 mL (4)

(Moon, 2017; Brent, 2001; Slaughter *et al.,* 2014; Patnaik, 2007; OSHA Pyridine, 1990; Datasheet, 2008.)

Besides, there are other chemical substances which are added with alcohol and make the product unfit for human consumption. The additives will determine the function of denatured alcohol as it will be used as fuel, cosmetic or as a cleaner apart from drinking liquor. Common denaturants used solely or combined include methanol, gasoline, kerosene, chloroform, gasoline, naphthalene methyl alcohol benzene, butyl alcohol and pyridine. Denatured alcohol is usually legally available in three forms according to the concentration of alcohol:

1. Industrial denatured alcohol (IDA): Wood naphtha (methanol) is added.
2. Completely denatured alcohol (CDA): Isopropyl alcohol, methyl ethyl ketone and denatonium benzoate are added.
3. Trade-specific denatured alcohol (TDSA)

In a final denatured alcohol, it is nearly impossible to separate the alcohol and denaturants (methylated spirits). (Corrosionpedia, 2019)

Methanol Poisoning

Symptoms

Methanol intoxication typically involves two set of symptoms separated by a substantial latent period. The initial symptoms of methanol poisoning are usually less severe than that of similar dose of ethanol poisoning. They include mild to moderate CNS depression, headache and dizziness. The latent period lasts 12 to 24 hours, varying based partly on the dose of methanol (Røe *et al.,* 1946). Co-admission of ethanol may elongate this period. According to a case series of 323 patients who drank methanol-contaminated whiskey, the latent period lasted 40 minutes to as long as 72 hours for some patients, averaging at 24 hours (Bennet *et al.,* 1953). Another study suggested that the latent period is not a significant prognostic factor for methanol poisoning (Naraqi *et al.,* 1979). The second set of symptoms manifested after latent period and they included blurred vision, metabolic acidosis and in rare cases nystagmus and putaminal hemorrhages (Røe *et al.,* 1946; Permpalung *et al.,* 2013). These symptoms arise due to metabolic acidosis and accumulation of formate in blood and may progress to death by respiratory failure and sudden respiratory arrest (Bennet *et al.,* 1953).

Central Nervous System (CNS)

The CNS signs and symptoms can range from mild headache to coma and death. With headache, vertigo, weakness and confusion are common in mild to moderate poisoning. The survivors might develop extrapyramidal syndrome similar to patients with Parkinsonism. The extrapyramidal syndrome includes rigidity, masked faces, tremor, bradykinesia and mild dementia These clinical effects usually result from the necrosis in the putamen and subcortical white matter (Guggenheim *et al.,* 1971; Ley *et al.,* 1983). Other rare neurological complications of severe intoxication are transverse myelitis cognitive deficits and pseudobulbar palsy (McLean *et al.,* 1980; Anderson *et al.,* 1987; Naraqi *et al.,* 1979). In severe cases of intoxication cerebral edema leads to convulsions and coma (Scrimgeour, 1980). Amnesia was reported by some studies where several patients could not recollect anything regarding methanol ingestion (Røe *et al.,* 1946).

Ocular System

Methanol has immense effect on vision. The signs and symptoms range from impaired vision to total blindness. Blurring vision, decreased visual acuity are some of the most common complaints in the early stage of poisoning usually 30 hours after ingestion. In most methanol poisoning outbreaks there had been two types of visual abnormalities such as transient and permanent abnormalities. Transient abnormalities include peripapillary retinal edema, optic disc hyperaemia, diminished pupillary reactions to light, and central scotomata. Permanent ocular abnormalities included optic disc pallor, attenuation of arterioles, sheathing of arterioles, diminished pupillary reaction to light, diminished visual acuity, central scotomata, and other nerve fibre bundle defects and blindness. A significant correlation was found between the incidence of acidosis and the development of permanent ocular abnormalities (Dethlefs *et al.,* 1978). Blindness is usually permanent but some degree of recovery can be expected in several months after intoxication (Scrimgeour *et al.,* 1982). The correlation between patient's subjective perception of vision or objective ocular tests and the clinical findings of ocular abnormalities was very poor. The absence of visual dysfunctions may provide the wrong idea about prognosis (Swartz *et al.,* 1981).

Gastrointestinal Tract

Gastrointestinal symptoms are not universal complaints from all patients. Their absence does not rule out the possibility of toxicity (Hanston *et al.,* 2000). Methanol intoxication normally results in nausea, vomiting, abdominal pain. Abdominal pain could be associated with pancreatitis. Acute pancreatitis increases blood amylase level, which is an indicator of inflammation of the pancreas (Swartz *et al.,* 1981).

Kidney

The occurrence of renal dysfunction is a rare complication of methanol poisoning. In rare cases, myoglobinuria occurred, which led to kidney dysfunction. The symptoms peaked on the eighth day and subsided within a month (Grufferman *et al.,* 1985).

Respiratory System

Due to metabolic acidosis, dyspnea or respiratory distress might occur in some patients during some stage of the poisoning. Dyspnea or breathlessness is usually a minor complaint by the patients. What is remarkable is the fact that dyspnea is not a good marker of the severity of acidosis (Bennet *et al.,* 1953).

Physical Findings

General Appearance

Almost all patients with methanol poisoning appeared in the hospital with moist and cool skin, diaphoresis and a general feeling of discomfort. The sweating may suggest an onset of cardiovascular shock but the cardiac functions are usually well maintained (Bennet *et al.,* 1953). A study found the combination of cyanosis and rubeosis of the skin (Røe *et al.,* 1946). But another study suggested that the skin discoloration of methanol poisoned patients was pallor and cyanosis was not remarkable. Cyanosis was prominent in patients with respiratory failure who ultimately died (Bennet *et al.,* 1953). The change in body temperature was not notable. In patients with severe acidosis, deep and labored breathing was noticeable. This type of breathing was termed as Kussmaul type breathing and found in patients with plasma bicarbonate level below 10 mEq (Bennet *et al.,* 1953).

Eye

Mydriasis, accommodation and non-photosensitive pupils are common with or without any visual impairments. Most of the patients with methanol intoxication have some clinical evidence of ophthalmologic abnormalities, even in the absence of visual dysfunction (Benton *et al.,* 1952). The ophthalmic findings include hyperemia of the optic disc and reduced pupillary responses to light as the early signs of methanol poisoned patient (Onder *et al.,* 1999). Hyperemia of the disc is the earliest to develop followed by slow development of peri-papillary retinal edema and edema of the optic disc. Although the hyperemia usually subsides within three days, the retinal edema may persist for so long as two weeks. Edema can extend to macular area in rare cases (Bennet *et al.,* 1953).

Cardiovascular

The heart rate is usually normal in most patients. In some instances, tachycardia might manifest. Blood pressure also stays within normal range except for the patients with pre-existing hypertension or hypotension. Even the comatose patients maintain the normal blood pressure (Bennet *et al.,* 1953).

Neurologic Examination

Methanol intoxication has profound impact on the CNS. The macroscopic and microscopic examination of methanol intoxicated brain may demonstrate: moderate cerebral edema; temporal and parietal cortex, basal ganglia, hypothalamus and pons; hemorrhagic necrosis of white matter of thalamus, putamen, and globus pallidus and in cerebral cortex. In more rare cases acute hemorrhage in the putamen, cerebral edema, hemorrhagic necrosis in basal ganglia followed by ventricular compression, transverse myelitis might be found (Karayel *et al.,* 2010; McLean *et al.,* 1980).

Abdominal Examination

The epigastric pain is usually associated with increased rigidity of abdominal muscles. The elevated level of plasma amylase may indicate pancreatitis which also causes abdominal pain, nausea and vomiting.

Ethanol Poisoning

Symptoms

Ethanol poisoning can be categorized into acute and chronic. The acute poisoning can be referred as ethanol

intoxication and the chronic poisoning can be termed as alcohol addiction (Gupta, 2016). Long-term alcohol use or alcohol addiction has the potential to affect every system of the body. Acute poisoning however has profound effects on central nervous system, cardiovascular system, respiratory system, gastrointestinal tract. The major signs and symptoms are usually related to these systems and organs.

Central Nervous System

The most common consequence of ethanol poisoning regarding the nervous system is temporary anterograde amnesia. The loss of memory can be either total or partial loss of recollection of the events of alcohol ingestion (Goodwin, 1995). Amnesia is also termed as 'blackout' which is different from losing consciousness as the patient might act and converse normally. The sudden increase of blood alcohol concentration influences hippocampal cellular activity that accounts for the short-term memory loss (White *et al.*, 2000). Another major sign and symptom of ethanol intoxication is impaired judgment and awareness of illness. Ethanol also induces cognitive impairment regarding visual and verbal memory, information processing, inhibitory control, and working memory. The tendency of denying cognitive impairment in intoxicated patients is also prominent (Jung, 2014). Alcohol helps in falling asleep and is believed to improve amnesia. However, it disrupts the overall sleep throughout the night. It relaxes pharynx muscles which causes obstructive sleep apnea (Issa *et al.*, 1982). It results in sleep fragmentation and non-restorative sleep and ultimately shortens the duration of deep slow-wave sleep and rapid eye movement sleep (Jung, 2014). After the initial intoxication episode subsides, chronic alcoholics may disclose peripheral neuropathy, including limb numbness, tingling and burning sensations, and paresthesia (Peters *et al.*, 2006).

Gastrointestinal System

Heavy alcohol intake may cause spasm of pyloric valve of the stomach, resulting in nausea and vomiting. Ethanol intake also irritates the esophagus and stomach mucosa and can manifest esophagitis, gastritis, pancreatitis and gastric ulcers. All of these result in epigastric pain, nausea and vomiting. Vigorous vomiting can induce longitudinal laceration in the mucosa at the gastroesophageal junction, which is known as Mallory–Weiss lesion. This will result in hematemesis. Chronic alcohol consumption most certainly results in several liver diseases. Steatosis or fatty liver, which is the accumulation of fat in the liver due to the impaired gluconeogenesis and fatty acid oxidation in the liver. Continuous heavy drinking will commonly manifest into alcoholic hepatitis and hepatic cirrhosis, which are very serious liver diseases (Maddrey, 2000).

Cardiovascular System

Alcohol has both beneficial and protective action on the cardiovascular system. The regular heavy intake of ethanol usually gives a proportional rise of blood pressure that is also dose dependent. Pressure falls to the normal range with the gradual decline of drinking. Acute ethanol intoxication results in malignant ventricular arrhythmias. Arrhythmias usually subside without any other complications in heart. On the contrary, chronic alcoholism imposes risk for cardiomyopathy, which can further manifest into unexplained arrhythmias with left ventricular impairment, mitral valve regurgitation with associated mural thrombi and congestive heart failure (Uyarel *et al.*, 2005).

Respiratory System

High blood concentration of ethanol can cause life-threatening respiratory depression. Alcohol intoxication interferes with mucociliary clearance, airway sensitivity and gag reflex. It increases risk of aspiration, which is associated with decreased gag reflex and airway sensitivity (Vonghia *et al.*, 2008).

Reproductive System

Repeated heavy drinking in adolescence can hamper secondary sexual development and disturb the onset and progression of puberty. Light drinking can elevate sexual drive but can also cause erectile dysfunction in men. Even in the absence of liver cirrhosis, chronic alcoholic Men with chronic alcoholism may show testicular atrophy, gynecomastia, decreases in ejaculate volume, and changes in body hair (Van Thiel *et al*., 1975). Women with chronic alcoholism show alteration in sex hormone levels and ovarian function, resulting in amenorrhea, infertility, and an increased risk of spontaneous abortion (Gill, 2000).

Other Signs and symptoms

Long-term ethanol intoxication with nutritional deficiencies disturbs the bone marrow hematopoietic system and thus decreases the production of erythrocytes, leukocytes and thrombocytes resulting in anemia, neutropenia, thrombocytopenia respectively. Thrombocytopenia imposes the risk of hemorrhagic shock, which usually decline within a week without alcohol consumption. Increased corpuscular volume (enlarged red blood cell) is also observed in chronic alcoholics (Palmiery *et al*., 2006). Heavy drinkers can experience muscle cramps and weakness by acute alcoholic myopathy (Preedy *et al*. 2001). Ethanol also interferes with calcium absorption and metabolism in the skeletal system. Patients with chronic ethanol intoxication may express lower bone density, decreased growth in the epiphyses, which increase the risk of falling fractures and osteonecrosis of the femoral head (Kaukonen *et al*. 2006).

Physical Findings

General Appearance

The heavily intoxicated patients appeared to have flushed face, bloodshot or watery eyes, droopy eyelids, blank stare, twitching or body tremors, disheveled clothing. They usually act normally but their memories about the intoxication event are always blurry and unclear. Their

speech can range from slurred to noisy and the speed of speaking is impaired. They exhibit a mean and annoying attitude. Their behavior can be careless and disoriented. They have a distinctive ethanol-smelling breath (50 signs of Visible Intoxication, 2012).

Neurologic Examination

In vivo longitudinal neuroimaging has revealed the changes in brain tissue structure in parallel with drinking behavior (regular drinking and sobriety) (Rosenbloom *et al*. 2008). Structural change usually includes ventricular enlargement and cortical gray matter loss, especially in the frontal lobes, and the extent of cortical volume (Pfefferbaum *et al*., 1998). These changes will eventually subside with the abstinence from alcohol. Synaptic properties of neurons in the hippocampus are consistently altered by ethanol in intoxicating concentrations (Siggins *et al*. 1987). Ethanol alternates the hippocampal functions by antagonizing activity at NMDA receptors (Markwiese *et al*., 1998), decreasing the overall level of glutamate released at synapses (Shimizu *et al*., 1998) and potentiating the effects of GABA at some GABA receptor subtypes within the hippocampus (Paulsen *et al*., 1998). Thus, ethanol causes spatial memory deficit or retrograde amnesia. Alcohol intoxication also impairs the upper and lower-limb motor movement. Even in sobriety, risk of falling is imposed on alcoholics. Severe choric alcoholism can lead to: Alcoholic cerebellar degeneration (ACD). Patients with ACD is more susceptible to upper-limb motor impairment (Johnson-Greene *et al*., 1997).

Cognitive Examination

A significant correlation between blood alcohol (ethanol) concentration and cognitive impairment had been found. The rising blood alcohol level always hampers the reaction time regarding information processing, learning, working memories, selective attention, visual and verbal memories. The declining alcohol concentration usually is not as bad but it can affect the accuracy of the previously mentioned cognitive operations (Schweizer *et al*., 2008).

Gastrointestinal Examination

Ethanol poisoning can induce nonmalignant esophageal diseases. It decreases lower esophageal sphincter pressure and interferes with acid-clearing mechanisms (Van Thiel *et al*., 1981). Ethanol induces the structural changes in the gastric mucosa. Patchy hemorrhagic lesions appear first, often within 1 h of exposure. Such lesions are transient and resolve completely within 1-3 days (Davenport, 1967). Ethanol exposure changes the ultrastructural in gastric mucosa which, results in decrease in the density of the cytoplasm of epithelial cells, and a generalized disruption of cell membranes and intercellular contact points. Chronic alcoholism disintegrates gastric mucosal cells (Eastwood *et al*., 1974). A reduction in transmural potential difference is universal in either type of ethanol poisoning (Eastwood *et al*., 1978).

Effects on the Liver

There are three major alcoholic liver diseases: alcoholic fatty liver, alcoholic hepatitis, and alcoholic cirrhosis. All three of them generally manifest with chronic alcohol intoxication. Fatty liver is a reversible lesion of liver when fat accumulates in the liver and the liver is enlarged. It is the early stage of alcoholic liver disease and it does not lead to cirrhosis. The patients are usually asymptomatic liver function tests such as BSP, ASP or alkaline phosphatase may be normal (Rubin *et al*., 1968). The alcoholic hepatitis is the inflammation of the liver with the necrosis of hepatocytes. Alcoholic hepatitis is characterized by the swelling of hepatocytes, ballooned cells with alcoholic hyaline. Eventually necrosis of hepatocytes results. Finally, scarring by fibrous tissue occurs and disturbs the normal architecture and alters its function (Van Thiel *et al*., 1981). Alcoholic hepatitis can progress into cirrhosis even in the absence of drinking where tissue lymphocytes are responsible for the progression (Leevy *et al*., 1976).

Hormonal Examination

Ethanol intoxication induces hypogonadism in male resulting in testicular atrophy, gynecomastia, reduced ejaculation, and changes in body hair (Van Thiel *et al*., 1975). Chronic excessive alcohol consumption by women can affect sex hormone levels and alter ovarian function, resulting in amenorrhea, infertility, and an increased risk of spontaneous abortion (Gill, 2000). Hormonal changes associated with acute alcohol poisoning include increase in cortisol levels (Badrick *et al*., 2008), inhibition of vasopressin secretion, and decrease in serum thyroxine and triiodothyronine (Kolakowska *et al*., 1977).

Mechanism of poisoning by methanol

From the present report we can claim that methanol shows toxicity by two mechanisms. Firstly, it can enter into the body by ingestion, inhalation and absorption through the skin and then can show its fatality due to its CNS depressant properties like ethanol poisoning. Secondly, through a process of intoxication, methanol is metabolized into formic acid which contains formate ion, via formaldehyde with the help of an enzyme called alcohol dehydrogenase in the liver (Schep *et al*., 2009). Methanol is metabolized into formaldehyde via alcohol dehydrogenase and formaldehyde is metabolized into formic acid via aldehyde dehydrogenase and the later conversion proceeds with full completion, with no detectable formaldehyde remaining (McMartin *et al*., 1979). Formate from formic acid is the toxic compound which is responsible for inhibition of mitochondrial cytochrome c oxidase that cause hypoxia at the cellular level and also metabolic acidosis (Liesivuori *et al*., 1991). Methanol or its derivative depress the endogenous production of CO_2 by the interference with oxidative enzyme systems particularly those related with aerobic glycolysis. The accumulation of lactic acid in methanol poisoning seems to indicate that anerobic carbohydrate breakdown is going on. Finally, it can be claimed that

serial determinations of blood pH, respiratory output and identification of accumulated acids are most significant factors of methanol poisoning (Bennett *et al*., 1953). Fomepizole has shown potency against alcohol dehydrogenase in man (McMartin *et al*., 1980).

Alcohol in Bangladesh

Historic Background

Though history of alcohol use in this region started long time ago, Bangladesh has never been established as an alcohol consuming country. During the reign of Mughal dynasty in this region, a special tax wax acted upon alcoholic beverage production. In 1938, first industrial scale alcohol beverage company was established by British merchants in Darshana of Chuadanga district called "Carew and Company" which is now owned by Government of Bangladesh (Dewan *et al*., 2015). Bangladeshi Companies that produce different types of alcohol for human drinking are summarized in table 2 and 3.

Table-2: Bangladeshi Companies that Produce Drinking Alcohol.

Name of Company	Founding Year
Carew & Co (Bangladesh) Ltd	1938
Jamuna Distillery Limited	2003

(Dhaka Tribune, 2017; The Daily Star, 2009)

Table-3: Different types of alcoholic beverages.

Name	Main Ingredient	Alcohol by Volume
Vodka	Potatoes or fermented cereal grains	40-75%
Whiskey	Fermented grain mash including barley, corn, rye, and wheat	Up to 40%
Brandy	Grapes	35-60%
Rum	Fermented molasses	38-54%
Gin	Barley or other grain	35-60%
Wine	Fermented grapes	6-21%
Beer	Malted barley	4-6%

(The Economic Times, 2017)

Toxic Dose of Ethanol

In adults, alcohol intoxication may develop after ingestion of 50-100 mL of pure alcohol. Approximately 1 mL/kg of pure alcohol intake will result in blood ethanol level of 0.1 g/dL (27.1 mmol/L). Death may result from 80 to 90 mmol/L ethanol levels in blood. (Perri *et al.*, 2022).

Religious Background

Islam, Buddhism and Christianity fully prohibit alcohol consumption. On the other hand, Hinduism do not claim absolute ban on alcohol consumption (Das *et al.*, 2006; Gentry, 2001).

Alcohol and Government policies in Bangladesh

Alcohol is defined as any spirit or liquor of any sort (wine, beer) or any liquid having more than 0.5 percent alcohol, according to Bangladesh's "Intoxicant Control Act" of 1990. Establishing a distillery or winery necessitates the acquisition of a certain license. Brewery, alcohol possession, storage, and consumption. Alcohol consumption is prohibited unless: a) the person is a Muslim; b) the person is a non-Muslim; or c) the person is citizens who have been granted permission to consume alcohol due to a medical condition ground from either a civil surgeon (head of government) or a government official an associate professor (and a member of the district health authority) a) sewage cleaners, morgue employees, a tea estate coolie (day laborer), footwear makers, and Rangamati, Khagrachari, and other indigenous peoples Bandarban district (also known as Chittagong Hill) is a district of Chittagong, Bangladesh. c) overseas tourists and businessmen who use tracts or CHT d) non-Muslims who consume alcohol in a licensed bar civilians (with a license). However, the legislation is silent on this. A Muslim's health issues for which he or she may be allowed to fast use alcohol, and it is up to the approved physician to make the decision. Furthermore, due of the multiethnic background of the participants, Bangladesh, as well as to avoid tampering with existing customs and traditions. The usage of alcoholic beverages was declared as part of the local culture. After a change to this law, it is legal for indigenous peoples enacted in 2001 As a result, there is a partial prohibition on the consumption of alcoholic beverages. It's interesting that only Muslim citizens are subject to punishment. Others are not covered by this act, there is no such thing as a minimum alcohol consumption age limit (Dewan *et al*., 2015).

Alcohol poisoning in Bangladesh

From November 2012 to January 2013, medicine unit of Dhaka Medical College received 8 fatal cases of methanol poisoning.

Table-4 (a): Fatal cases of alcohol poisoning.

Case No.	Name	Age	Profession	District of Residence	Date of Hospital Admission	Time of Hospital Admission	Date of Death	Time of Death
1	Mr. T. A.	42	Day laborer	Dhaka	3.11.12	2.30 a.m.	Unknown	Unknown
2	Mr. M. R	35	Serviceman	Dhaka	4.11.12	7 p.m.	4.11.12	7.30 a.m.
3	Mr. D.	40	Businessman	Chandpur	5.11.12	11 p.m.	12.11.12	6 a.m.
4	Md. J. H.	40	Rickshaw puller	Sherpur	6.11.12	11.30 a.m.	6.11.12	12 p.m.
5	Mr. M. H.	55	Unknown	Dhaka	13.11.12	10 p.m.	13.11.12	2.15 a.m.
6	Mr. B. B.	55	Unknown	Comilla	17.11.12	7.10 a.m.	17.11.12	7.45 a.m.
7	Md. A. A.	35	Businessman	Dhaka	21.12.12	11 p.m.	22.12.12	2.15 a.m.
8	Mr. B. H.	50	Businessman	Dhaka	23.01.13	9.10 p.m.	24. 01.13	3.30 a.m.

(Amin *et al.*, 2017)

Table-4 (b): Fatal cases of alcohol poisoning.

Case Study No.	Name	History	Blood Pressure (BP) measured after Hospital Admission (mm Hg)	Pulse rate recorded after Hospital Admission (Beats per minute)	Glasgow Coma Scale (GCS) recorded after Hospital Admission
1	Mr. T. A.	Consumption of considerable amount of Methanol	80/60	84	Not recorded
2	Mr. M. R	Ingestion of alcohol seven hours before hospital admission	120/80	84	3
3	Mr. D.	Consumption of alcohol 6 hours prior to hospital admission	100/60	88	8
4	Md. J. H.	Ingestion of methanol 2 days before hospital admission	100/70	90	8
5	Mr. M. H.	Consumption of alcohol 6 hours before hospital admission	110/70	100	12
6	Mr. B. B.	Consumption of considerable amount of Methanol 7 hours earlier	90/60	96	4
7	Md. A. A.	Ingestion of Methanol 2 days earlier	Not recorded	16	5
8	Mr. B. H.	Complaints of alcohol poisoning 3 hours earlier	100/50	72	7

(Amin *et al.*, 2017).

On February 2, 2021, 12 people including 2 students have died from consuming counterfeit alcohol in Dhaka and Bogura district. In Dhaka, a group of 41 employees of the media organization went to Gazipur for an office tour and some had alcoholic beverages and got sick. Meanwhile in Bogura, around seven people got admitted to the hospital because of toxic liquor (The Daily Star, 2021).

CONCLUSION

A comprehensive training and awareness are mandatory to prevent this hazardous problem in Bangladesh. Law should be implemented properly and the industries should be aware of the law and also practice honesty. There should be adequate poisoning center distributed around whole country and also have skilled personnel to manage this situation. Enough medication and antidote should be stored. Above all, moral development is the key step to manage this type of unethical problem.

Funding
This work has been a self-finance activity.

Authors' Contributions
MSA has conceived the original idea, TH, MJS, ANR, KAR extensively reviewed the literatures. MAS edited the article and give intellectual input. All the authors read the review article meticulously and agreed to submit the article.

ACKNOWLEDGMENTS
We express our gratitude to the authors of the articles we have cited. TH has drawn the Figure-1, Figure-2 and Figure-3.

Conflicts of Interest
The authors declare no conflict of interest.

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