Alcohol Use Disorder: Effect of Alcohol Consumption

Introduction

In the case scenario of David, he was diagnosed to have substance use disorder Those substances he used were cannabis, cocaine and alcohol. He expressed that if no stimulation by alcohol, he could easily reject to use the substances. Therefore, alcohol would be the first priority to keep abstinence in order to help him get rid of substances. In this assignment, diagnosis of Alcohol Use Disorder, effect of alcohol, its metabolism and withdrawal syndrome will be investigated. Then, treatment of it will be discussed.

Diagnosis of Alcohol Use Disorder

The diagnosis of alcohol use disorder (AUD) in the 5th edition of the Diagnostic and Statistical Manual of Mental Disorder (DSM-5) was the integration of alcohol abuse and dependence in its 4th edition, with an additionally grading system to sub-classify AUD into different severity: mild, moderate and severe (National Institute on Alcohol Abuse and Alcoholism, 2016). There are several diagnostic criteria in DSM-5, like unsuccessful effort to cut down, craving, physical hazardous, tolerance, withdrawal etc., and AUD was diagnosed if two or more of them occurred in the past 12 months (American Psychiatric Association, 2013).

Effect of Alcohol consumption

A common effect of alcohol ingestion

Alcohol intoxication is the circumstances of recent alcohol ingestion with an accumulation of alcohol and its metabolites in blood faster than its metabolization by liver (Jung & Namkoong, 2014). Same with benzodiazepine, alcohol act as central nervous system depressant to decrease gamma-aminobutyric acid (GABA) inhibitory function and increase N-methyl D-aspartate (NMDA) type glutaminergic activity (Krystal, Petrakis, Manson, Trevisan & D'Souza, 2003; Krystal et al., 2006). Drinking too much alcohol would bring about many harmful effects to our body. After ingestion, it would be rapidly absorbed into blood vessel and reach a peak after about 30 to 60 minutes. Alcohol can cross the blood-brain barrier and affect the brain cells to cause psychoactive and behavioral effects, like sedation, hallucinations, impaired judgement, slurred speech, loss of coordination, hangover etc. In case of extreme dose, respiratory depression, coma or death may result. If long-term high dose of alcohol intake, adverse consequences would appear, like cognitive impairment or brain damage. Tolerance would be developed for chronic alcohol consumption as neuroadaptation occurs which a higher dose of alcohol is needed to attain desired psychoactive effect (National Collaborating Centre for Mental Health, 2011).

Thiamine deficiency and Wernicke-Korsakoff syndrome

Thiamine deficiency can be resulted from chronic alcohol consumption causing risk of developing fatal condition called Wernicke-Korsakoff syndrome (WKS) (Donnelly, 2017).

Chronic alcohol consumption would lead to impair thiamine absorption from GI tract, weaken the processing and storage of thiamine in liver and increase the thiamine demand for high carbohydrate processing from a high-calorie alcoholic beverage. The lack of thiamine would impair the flow of electrolytes in and out of muscle and nerve cells. It also leads to lack of enzymes essential for producing adenosine triphosphate through Krebs cycle and toxic substances accumulate that can damage the brain. Acute Wernicke-encephalopathy is the first phase of WKS characterized by ocular disturbances (nystagmus and paralysis of eye muscle), mental state change (confusion and unable to concentration) and ataxia (damage in cerebellum and vestibular system). It then developed into Korsakoff syndrome that is the acute onset of serious memory impairment like anterograde and retrograde amnesia that can result in loss of important personal function.

Fetal alcohol syndrome

Apart from the alcoholic effect on adult, let's have some knowledge of fetal alcohol syndrome (FAS), which is the most severe abnormalities of the fetus due to drinking alcohol by mother during gestation. The fetus may have undeveloped liver and not be able to metabolize alcohol from which directly passing through the placenta and umbilical cord. The alcohol is then exposed directly to the developing fetus and cause serious developmental problems. Stewart (2012) summarized the effect of alcohol on fetal development. In cellular level, alcohol can dehydrate a cell and the cell growth will be interrupted, delayed or died. This particularly remarkable in brain development that leads to inborn brain damage and so learning disabilities like limited ability to memorize, control impulse, paying attention or judgement. It also results in congenital physical deformities like small head size, low birth weight or problems with the heart, kidney or bones. Facial characteristics of FAS fetus were identified like smooth philtrum, thin upper lip and short palpebral fissure lengths. Therefore, any alcohol must be strictly prohibited during all stage of pregnancy.

Metabolism of alcohol

There is always question that why do some people are able to drink more or appear to take a greater risk than others for developing adverse health consequences like hangover, hepatic damage or cancers. This can be explained in view of alcohol metabolism in our body. According to the publication of National Institute on Alcohol Abuse and Alcoholism (2007), the common alcohol metabolism pathways involve alcohol dehydrogenase (ADH) in the liver to breakdown most ethanol and transform them into a carcinogenic compound called acetaldehyde, then it is further breakdown into acetate by aldehyde dehydrogenase (ALDH), then breakdown into water and carbon dioxide for elimination. There are also other ways in body to convert alcohol into acetaldehyde like the use of enzyme cytochrome P450 2E1 and catalase. Some alcohol can also interact with fatty acids and forming fatty acid ethyl esters to damage the liver and pancreas. Nevertheless, acetaldehyde should take in focus as it is highly contributed to toxic effect in damaging the liver, pancreas, brain, and gastrointestinal tract, causing the intoxication sign and symptoms as mentioned before. Therefore, the main reason for variation of people in ability of drinking alcohol is the different ability of the enzymes involved. For example, a fast ADH or slow ALDH would cause acetaldehyde to accumulate and exert its toxic effect.

Alcohol withdrawal syndrome

In acute alcohol withdrawal syndrome, several symptoms are identified to have greater risk to develop if alcohol consumption that beyond healthy limit was stopped abruptly or reduced substantially (National Clinical Guideline Centre, 2010). Severity of alcohol withdrawal symptoms depends on the degree of alcoholism and personal differences. When alcohol is absent or inadequate, GABA receptors and NMDA system will take attempt to restore, and other pathophysiological changes occur like increase corticotropin-releasing hormone, decrease activity of dopamine activity or further increase homocysteine level. All these physiological changes contribute to the sign and symptoms of alcohol withdrawal. After 6 to 8 hours of abstinence, there were a series of symptoms that may occur simultaneously, like agitation, tremor, sweating, nausea, vomiting, palpitation, fever etc. These symptoms usually subside by second to third days. Some particular symptoms must be noted that special attention should be provided like seizure, hallucinations and delirium tremens (DTs). DTs is characterized by rapid onset of confusion and coarse tremor, and the symptoms above can be intensified. In severe cases, hyperpyrexia, ketoacidosis and circulatory collapse can occur which are fatal.

Treatment of AUD

Whether acute alcohol withdrawal patients need hospitalization is mainly depend on two considerations (National Clinical Guideline Centre, 2010). First, we need to evaluate the severity of the syndrome by a widely recognized assessment tool called Clinical Institute of Withdrawal Assessment for Alcohol scale, Revised (CIWA-Ar). Severity of repeated withdrawal episode would be higher due to the ‘kindling effect’, this increase the risk of treatment in the community. Second, we should have a comparison on the treatment between hospital and the community to meet the need of patient with alcohol withdrawal in both acute condition and long-term abstinence. If the patient is difficult to access required services or the community availability of the follow-up services is inadequate, the need for admission would increase.

For patient with alcohol intoxication, vital functions should be closely monitored, especially respiratory function as risk of respiratory depression; liquid replenishment should also be provided to prevent dehydration (Caputo et al., 2018). In more severe case, mechanical ventilation, parenteral fluid resuscitation and gastric lavage with activated charcoal should be prepared. In case of concurrent use of other sedatives, respective antidotes should be considered like naltrexone for opioids and flumazenil for benzodiazepines. Moreover, intravenous metadoxine can be considered for subside of symptoms more rapidly due to its effect on reduction of blood alcohol and acetaldehyde (Caputo et al., 2018).

For patient who developed alcohol withdrawal syndrome, vital parameters and withdrawal symptoms should be closely monitored, and continuous reassurance is required. Hydration is also necessary if symptoms of dehydration presents. Vitamin supplements like vitamin B1 (thiamine), B6, B12, C and folates are needed to prevent Wernicke’s encephalopathy. In case of CIWA-Ar score is greater than 8, benzodiazepine, like diazepine or chlordiazepoxide, should be administered to mimic the alcohol properties so as to mediate the withdrawal symptoms (Caputo et al., 2018). Anti-epileptic can be added in association with benzodiazepine for the development of seizure. If DTs developed, high dose of benzodiazepine is used; but if refractory DTs, adding anesthetic drugs, like barbiturates and propofol, may necessary after intubated with mechanical ventilation (Caputo et al., 2018).

Conclusion

Alcohol Use Disorder is a diagnosis in DSM-5 for patients with alcohol abuse. Ingestion of alcohol can cause many effects to our body and severe alcohol intoxication leads to development of fatal symptoms. Chronic alcohol consumption can bring about thiamine deficiency and increase the risk to have WKS. Alcohol must be forbidden to pregnant women as FAS. Alcohol is metabolized by the use of liver enzyme and toxic mid-produce, acetaldehyde, would accumulate if not able to further break down. Different withdrawal symptoms can appear if alcoholism stop alcohol abruptly. Treatment should be considered in community or hospital for different need of patients with AUD. Both alcohol intoxication and withdrawal have their medical management in the pharmacological and non-0pharmalogical way. Nurses should acquire this knowledge to carry out appropriate actions towards AUD patients who need professional assistance.

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