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Alcohol Use Disorder (AUD) in the Elderly Population

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Introduction

There is a substantial and growing population of elderly adults (65 years and older) who misuse alcohol, psychoactive prescription drugs, and/or other substances (Blow & Barry, 2016). Alcohol dependence and alcohol abuse have now been combined into one entity called Alcohol use disorder – AUD (American Psychiatric Association, 2013).

This topic was chosen because studies have shown that an average of 2.8 million adults over the age of 50 suffered from substance use disorders, including alcohol in the United States. That number is projected to double, totaling roughly 5.7 million seniors in five years (Blow & Barry, 2016).

A variety of factors may contribute to AUD in elderly (Blow & Barry 2016), they include:

- Empty nest syndrome (when children grow up and move away)
- Loss of friendships due to moves, health complications or death
- Deteriorating health conditions (cardiovascular disease, vision/hearing loss, and diabetes)
- Traumatic events like a spouse's illness or death
- Sadness after downsizing a home
- Boredom from retirement or lack of socialization

Signs and Symptoms of Alcohol Use Disorder (AUD)

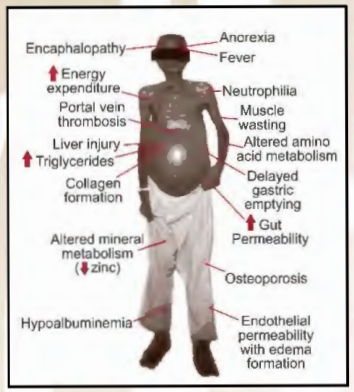

Figure 1. Signs/Symptoms of AUD (Frazier et al., 2011).

Pathophysiological Processes

Underlying Pathophysiology

1. Mechanism of ETOH induced depression and the dopamine reward system (Burchum & Rosenthal, 2019)

ETOH induces general depression and activation of the reward circuit via interaction with 1. Receptors for gamma-aminobutyric acid (GABA) – the principal inhibitory neurotransmitter in the CNS. 2. Receptors for glutamate – a major excitatory neurotransmitter in the CNS. 3. The 5-HT subset of receptors that mediate the release of dopamine, the major neurotransmitter of the reward system.

When alcohol binds with GABA receptors, it enhances GABA-mediated inhibition thereby causing widespread depression of CNS activity.

Whereas, when alcohol binds with N-methyl -D-aspartate (NMDA)-type of glutamate receptors, they inhibit the flow of ions thereby blocking glutamate-mediated excitation and hence reduces the overall activity of the CNS (Burchum & Rosenthal, 2019).

These pathophysiologic concepts explain why alcohol may be used by the elderly as an escape pathway from the various problems associated with aging because it is a CNS depressant and it also feeds the reward system.

2. Mechanism of alcohol metabolism in the liver

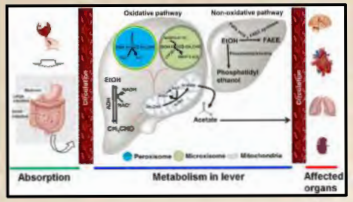

Figure 2. Alcohol metabolism in the liver. Alcohol dehydrogenase (ADH); Cytochrome Enzymes (P450 IIE1, CYP2E1); Aldehyde dehydrogenase2 (ALDH2); Fatty acid ethyl ester (FAEE); Acetaldehyde (CH3CHO); Hydrogen peroxide (H2O2); Nicotinamide adenine dinucleotide (NAD/NADH); Reactive oxygen species (ROS). (Mandal et al., 2017).

3. Epigenetic Mechanisms of Alcoholism

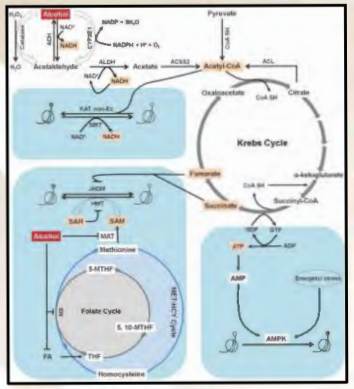

Figure 3. DNA methylation, histones modifications, and folate deficiency. Acyl-CoA synthetase 2 (ACSS2); ATP-citrate lyase (ACL); S-adenosylmethionine (SAM); S-adenosyl homocysteine (SAH); Histone methyl transferase (HMT); Jumonji-C domain-containing histone demethylases (JHDMs); AMP-activated protein kinase (AMPK); Alcohol dehydrogenase (ADH); Nicotinamide adenine dinucleotide (NAD/NADH); Cytochrome Enzymes (CYP2E1, P450 IIE1); Aldehyde dehydrogenase (ALDH); CoA synthase (CoA-SH); Lysine acetyltransferase (KAT); Sirtuin (SIRT); Folic acid (FA); Tetrahydrofolate (THF); Methylene tetrahydrofolate (MTHF); Methionine synthase (MS); (MAT); Guanosine diphosphate (GDP); Guanosine triphosphate (GTP); Adenosine triphosphate (ATP); Adenosine diphosphate (ADP); Adenosine monophosphate (AMP). (Mandal et al., 2017).

4. Mechanism of ETOH induced liver disease

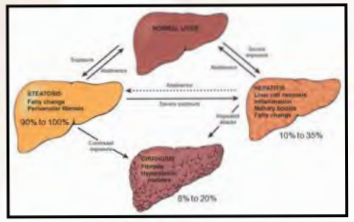

Figure 4. Inter-relationships among hepatic steatosis, hepatitis, and Cirrhosis (Medchrome, n.d.).

Significance of Pathophysiology

Two neuropsychiatric syndromes are exacerbated with AUD - Wernicke's encephalopathy and Korsakoff's psychosis (Burchum & Rosenthal, 2019).

Wernicke's encephalopathy and Korsakoff's psychosis are caused by thiamine deficiency secondarily to poor diet and alcohol-induced suppression of thiamine absorption.

Wernicke's encephalopathy is characterized by confusion, nystagmus, and abnormal ocular movements. This syndrome is reversible with thiamine.

Korsakoff's psychosis is characterized by polyneuropathy, inability to convert short-term memory into long-term memory, and confabulation. Korsakoff's psychosis is not reversible.

Long-term alcohol consumption leads to cerebral atrophy and enlargement of the ventricles. It is associated with impairment of memory and intellectual function (Burchum & Rosenthal, 2019).

The acetate that is a by-product of ETOH metabolism is used to synthesized cholesterol and fatty acids by the liver. That explains why excessive alcohol consumption can lead to the alcohol-induced fatty liver which can progress to non-viral hepatitis and cirrhosis

AUD can lead to DNA hypomethylation, histones acetylation, methylation, phosphorylation, ubiquitination, and crotonylation. Taken together, these events can lead to gene silencing, and a prelude to ETOH induced liver diseases, and hepatocellular carcinoma.

Diagnosis

Several screening tools are available to make AUD diagnosis. (Berks & McCormick 2018; Rumpf et al., 2016).

Alcohol Use Disorders Identification Test (AUDIT) is used to assess whether or not there is a problem with alcohol dependence.

Severity of Alcohol Dependence Questionnaire (SADQ) is used where alcohol dependence has been identified, and to measure the severity of dependence.

The revised Clinical Institute Withdrawal Assessment for Alcohol scale assessment (CIWA-Ar) is used to assess the severity of alcohol withdrawal syndrome.

General medical workup should include Vital signs, CBC, BMP, LFTs; it should be done to rule out any medical problems.

Treatment

The best outcome as supported by evidence is combining psychosocial treatments with medications.

Psychosocial treatments include brief interventions, motivational enhancement therapy, cognitive-behavioral therapy, behavioral approaches, family therapies, and 12-step facilitation (Pearson & Duff 2019).

Medications include Food and Drug Administration (FDA) approved and non-FDA approved medications. There are only four FDA approved medications to treatment AUD (Kranzler & Soyka 2018), and they are as follow

1. Disulfiram inhibits aldehyde dehydrogenase, an enzyme that metabolizes acetaldehyde, a toxic metabolite of alcohol. Inhibiting the enzyme rapidly increases the concentration of acetaldehyde and produces a disulfiram-ethanol reaction (DER) characterized by nausea, flushing, vomiting, sweating, hypotension, palpitations, and rarely, serious reactions including cardiovascular collapse (Kranzler & Soyka 2018)
2. Naltrexone is a non-selective antagonist of mu, kappa, and delta-opioid receptors which was initially approved to treat opioid dependence. It reduces mesolimbic opioidergic activity thereby modulating the dopamine-mediated rewarding effects of alcohol; it reduces alcohol consumption (Kranzler & Soyka 2018)
3. Acamprosate modulates glutamatergic neurotransmission, which explains its efficacy in treating AUD (Kranzler & Soyka 2018)
4. Long-acting injectable Naltrexone is used to treat alcohol dependence in patients who can abstain from alcohol in an outpatient setting (Kranzler & Soyka 2018)

Non-FDA-approved medications for treating AUD are often used off-label. Typical representatives are Nalmefene, Baclofen, gabapentin, and Topiramate (Kranzler & Soyka 2018)

Implications for Nursing Care

RNs should be aware that elderly people who have AUD are significant for the following:

- Ineffective coping that may be related to the stress of bereavement
- Impaired adaptive behavior and problem-solving skills
- Self-neglect related to alcohol misuse
- Risk for malnutrition-related to alcohol misuse
- Risk for injury related to alcohol abuse

Nursing Interventions

- Explore alternative coping strategies such as the use of relaxation skills, and guided imagery (Stuart, 2019). Rationale - Patient may have little or no knowledge of adaptive responses to stress and needs to learn other options for managing time, feelings, and relationships without drugs. Alternative coping strategies will help patient to relax, develop new ways to deal with stress, and problem-solving
- Encourage verbalization of feelings, fears, and anxiety (Stuart, 2019). Rationale - This may help the patient to begin to come to terms with long-unresolved issues.
- Encourage involvement with self-help associations (Alcoholics Anonymous) (Stuart, 2019). Rationale - Alcoholics Anonymous will put the patient in direct contact with the support system necessary for managing sobriety and live an alcohol-free life.
- Use peer support to find ways of spending time on interesting life leisure (Townsend & Morgan, 2017). Rationale - Understanding and support from his peer group will help to promote abstinence
- Discuss with the patient the long-term effect of excessive alcohol use including Wernicke's encephalopathy and Korsakoff's psychosis (Townsend & Morgan, 2017).
- Discuss treatment referral where he will have the opportunity to be managed with medications that will diminish the crave for alcohol (Townsend & Morgan, 2017).

Conclusion

There are several reasons why AUD can lead to negative outcomes for the elderly:

Changes in body composition with aging result in higher blood alcohol concentrations for a given alcohol intake (Shenvi et al., 2020).

Older adults tend to have more chronic medical conditions, take more prescription medications (polypharmacy), and are at higher risk of falls, all of which can be complicated or worsened by high-risk alcohol use (Shenvi et al., 2020).

Symptoms of excess alcohol use, such as gait disturbances, falls, or confusion may be interpreted as signs of aging or other conditions (Shenvi et al., 2020).

Chronic use of alcohol produces physical dependence, and abrupt withdrawal produces abstinence symptoms that may include hallucinations, intense tremors, tonic-clonic seizures, elevated heart rate, blood pressure, and temperature (Burchum & Rosenthal 2019).

Individuals who have a physical alcohol dependence have cross-dependence to other CNS depressants such as barbiturate, benzodiazepines.

Chronic alcohol consumption produces tolerance – requirement for larger amount of alcohol to produce the same effects. They also develop cross-tolerance to general anesthetics, barbiturates, and other CNS depressants (Burchum, J. R., Rosenthal, L. D., 2019).

References

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