Chronic Alcohol Abuse and Long Term Complications

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INTRODUCTION

Alcohol (aka, ethanol, ethyl alcohol) is the most commonly abused drug in the United States. Approximately 10% - 15% of all Americans are considered to be alcohol dependent. Alcohol dependence is defined in the DSM-IV as repeated alcohol-related difficulties in at least three of seven areas of functioning that cluster together over a 12-month period. A simpler definition is: alcohol abuse is **repetitive problems in four life areas – social, legal, interpersonal, and occupational – caused by drinking.**

Most nurses are familiar with the potentially serious effects of acute alcohol intoxication. However, the effects of chronic alcohol abuse are perhaps more severe, and there are several complications of chronic alcohol abuse that can be life-threatening.

OBJECTIVES

When the learner has finished this module, she/she will be able to:

1. Identify the differences in alcohol metabolism between non-drinkers and alcoholics.
2. Identify the amount of alcohol that is considered to be a health risk if ingested daily.
3. Identify two types of neurological damage associated with heavy drinking.
4. Identify three gastrointestinal complications associated with heavy drinking.
5. Identify two cardiac complications associated with heavy drinking.
6. Identify the basic cause of alcohol withdrawal syndrome.
7. Identify three signs/symptoms of the alcohol withdrawal syndrome.

8. Identify two serious complications associated with the alcohol withdrawal syndrome.
9. Identify the optimum treatment for delirium tremens.
10. Identify the optimum treatment for Wernicke-Korsakoff syndrome.

THE BASICS ABOUT ALCOHOL

Alcohols are chemical compounds that are composed of a hydrocarbon and a hydroxyl group. Ethyl alcohol – the intoxicating component of alcoholic beverages – is the most commonly used and abused alcohol. Ethyl alcohol causes acute intoxication by increasing the activity of a potent inhibitory neurotransmitters, γ-aminobutryic acid (**GABA**) and, and interfering with the activity of a potent excitatory neurotransmitters, **glutamate.**

Alcohol is rapidly absorbed and most ethanol is metabolized in the liver by the enzyme alcohol dehydrogenase (ADH). **People who chronically abuse alcohol metabolize alcohol differently. They metabolize alcohol at a faster rate than non-drinkers. They also develop a functional tolerance: the greater the number of years someone has been drinking, the more alcohol that person will need to drink to become functionally impaired.**

The national standard for acute intoxication is an ethyl alcohol level of 80 mg/dL. This is considered the level that will alter consciousness to the degree that the individual loses the capacity to act with reason and caution.

WHAT ARE THE LONG-TERM EFFCTS OF CHRONIC ALCOHOL ABUSE?

Chronic alcohol abuse causes many well known social problems such as driving while intoxicated, family and personal trauma, suicide, workplace accidents, and interpersonal violence. The Centers for Disease Control estimates that the excessive alcohol use is the third leading cause of death in the United States.

Fewer people are aware of the *long-term* medical complications associated with chronic alcohol abuse. However, when alcohol is abused chronically, it affects virtually every organ system. Excessive, chronic alcohol use also worsens and exacerbates pre-existing medical conditions, it significantly increases the risk of developing many diseases, and it shortens life expectancy by at least a decade.

Key Point: How much alcohol will cause irreparable harm to the body? That depends on many factors such as individual tolerance, genetics, pattern and length of use, etc. **But the consumption of more than**

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two standard drinks a day (a standard drink is 1.5 ounces of spirits, 12 ounces of beer, or five ounces of wine) **is associated with a significant risk for developing alcohol-related health issues.**

Damage to Organ Systems

- Nervous system: Chronic alcohol abusers frequently experience **blackouts.** These are episodes of **antegrade amnesia.** The sleep patterns of alcohol abusers are disturbed, and this is a risk factor for the development of cardiac disease. Chronic, high intake of alcohol also causes **peripheral neuropathy** in approximately 5% – 15% of alcohol-dependent individuals. Approximately 1% of alcohol-dependent individuals will develop clinical signs of **cerebellar degeneration** or **atrophy**, and 50% will have evidence of atrophy as seen by a CT or MRI scan.

- Gastrointestinal system: Alcohol is very irritating to the gastric mucosa, and alcohol is a common cause of **hemorrhagic gastritis.** Alcoholics are at a higher risk than the general population for developing **Mallory-Weiss tears**. Alcoholic cirrhosis is one of the two leading causes of **esophageal varices**. Approximately 30% of all cases of **acute pancreatitis** are caused by alcohol abuse, and approximately 70% - 80% of all cases of **chronic pancreatitis** are caused by alcohol abuse. Chronic alcohol abuse is the second leading cause of **cirrhosis.** Cirrhosis is characterized by slow destruction of hepatocytes and replacement of functioning liver tissue with scarring and fibrosis. Approximately 30% of all people who chronically abuse alcohol will develop alcoholic cirrhosis. These patients will have jaundice, ascites, bleeding disorders, iciteral sclera, and they may develop esophageal varices and/or hepatic encephalopathy.

- Heart: Chronic drinkers are almost twice as likely as abstainers to develop **atrial fibrillation**, and heavy drinking increases the risk of developing **coronary artery disease** by sixfold. Alcohol abuse also increases the risk of **hypertension**. **Cardiomegaly** and **cardiomyopathy** are relatively common consequences of heavy drinking.

- Hematopoietic: Chronic, heavy drinking **decreases the production of white blood cells** and can affect their function.

- Metabolic: Heavy drinking increases the risk of developing **non-insulin dependent/type 2 diabetes.** The **metabolic**

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syndrome is more common among heavy drinkers. Chronic drinkers are often **malnourished**; the lack of vitamin B1 (thiamine) in the diet of an alcoholic can have very serious consequences.

THE PATHOPHYSIOLOGY OF ALCOHOL-RELATED ORGAN DAMGE

There has been an enormous amount of research on the toxic effects of alcohol. However, although there are many theories as to why alcohol damages the liver, the brain, the heart, etc., no one found the definitive answer. The neurological damage caused by drinking is *probably* caused by damage to myelin and axons, and this is *probably* caused by thiamine deficiency. Cardiac damage is *probably* caused by a direct toxic effect of ethanol on the heart, a chronic low level of inflammation that predisposes to atherosclerosis, or free radical damage caused by shifts in ethanol metabolism. Liver damage *may* be caused by an endotoxin produced in the gut in response to heavy drinking, or it *may* be caused by activation of inflammatory mediators. There may also be genetic variations in susceptibility to alcohol-induced organ damage, and it is difficult to know how much these morbidities are due to alcohol or the alcoholic lifestyle.

Key Point: Although it is k not known exactly how alcohol causes organ damage, it is *very* clear is that **the more you drink and the longer you drink the greater the amount of damage.**

ACUTE EFFECTS OF CHRONIC ALCOHOL ABUSE

The conditions discussed here – alcohol withdrawal and Wernicke-Korsakoff syndrome – are caused by chronic alcohol abuse, but they have often an acute and dramatic presentation.

Alcohol Withdrawal and Delirium Tremens

Alcohol is a physically addictive drug, and the abuser must maintain a certain blood level or he/she will suffer withdrawal. Alcohol withdrawal can be relatively moderate, the alcoholic may experience very little or no discomfort at all (approximately 50% of all chronic, heavy drinkers do not need medical care), or it can be potentially life threatening. **Alcohol withdrawal is basically a state of sympathetic excess that is caused by a) the same mechanisms by which alcohol produces intoxication, and b) the effects of prolonged drinking on the nervous system.**

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Alcohol produces intoxication by its effects on GABA, and glutamate (the latter at the *N*-methyl-D-aspartate type glutamate receptor, also called the NDMA receptor). Chronic alcohol abuse overstimulates the GABA receptors and eventually the number, responsiveness, and activity of the GABA receptors diminishes (This phenomenon is called **receptor downregulation**, and it is one of the reasons why alcoholics can tolerate large amounts of alcohol and why they need large amounts to become intoxicated). Chronic alcohol abuse also affects the NDMA receptors, but in the opposite way: they are **upregulated** and their number and their activity and responsiveness increases. When a chronic, heavy drinker suddenly stops drinking, two things occur: a) there is no more inhibition of glutamate by ethanol, and there are greater numbers of highly active excitatory glutamate NDMA receptors, and b) there is no more stimulation of the far fewer and far less active and responsive inhibitory GABA receptors. The result: over-stimulation and no inhibition. To put it simply, the pedal is to the metal and the brakes are disconnected.

Key Point: The basic cause of alcohol withdrawal is glutamate stimulation and GABA inhibition.

The onset of signs and symptoms of alcohol withdrawal can start within six hours after the last drink, and the serious, life-threatening effects usually occur three to seven days later. Most people will develop headache, tremors, anxiety, palpitations, diaphoresis, and abdominal pain. If the patient remains abstinent, his/her condition steadily deteriorates. Visual and auditory hallucinations are experienced, tonic-clonic seizures can occur, and the patient may eventually become **delirious, disoriented, febrile, tachycardic, and hypertensive.** At that point, the patient is said to have **delirium tremens: delirium tremens (aka DTs, rum fits) is the most severe form of alcohol withdrawal. It usually occurs 48 to 96 hours after the last drink.**

Key Point: Alcohol withdrawal can be misdiagnosed if the patient was not candid about his/her drinking habits.

The patient with mild to moderate signs/symptoms of alcohol withdrawal will usually recover in several days. Someone with a serious case of DTs may be incapacitated for several weeks. The severity of alcohol withdrawal appears to be related to how much and how long: the more someone drinks and the more years that person has been drinking, the more intense the signs and symptoms. If the patient has multiple episodes of withdrawal

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through the years as he/she tries repeatedly to stop drinking, each withdrawal episode is usually worse than the preceding one.

If the patient receives treatment, most will survive, but the mortality rate can be as high as 35% if the patient has complications. Few patients with alcohol withdrawal will progress to delirium tremens, and seizures are usually single, self-limiting, and have no sequelae. Treatment consist of hydration, safety measures, monitoring for complications, replacing thiamine stores, and **keeping the patient well sedated with benzodiazepines.**

Wernicke-Korsakoff Syndrome

Wernicke's encephalopathy and Korsakoff's psychosis are two distinct, but closely related neurological conditions that occur in patients who are heavy drinkers and are malnourished. Wernicke's encephalopathy can sometimes lead to Korsakoff's psychosis, there are some similarities in their clinical presentations, and they are both caused by heavy drinking and vitamin B1 (thiamine) deficiency, so the two are often described as a single pathological event, Wernicke-Korsakoff syndrome.

Thiamine serves as cofactor for enzymes that are responsible for glucose metabolism, lipid and carbohydrate metabolism in the brain, production of neurotransmitters, and nerve conduction. Alcohol interferes with thiamine absorption, transformation of thiamine to its active form, and thiamine storage. In addition, the body only stores a little more than two weeks of thiamine, and chronic heavy drinkers on a binge at times consume little more than alcohol for weeks a time. When thiamine deficiency becomes acute, brain and nerve cells can be damaged.

Wernicke-Korsakoff syndrome is characterized by **ocular abnormalities, gait disturbances, and mental status changes**: double vision, ataxia, hallucinations, anterograde and retrograde amnesia, confusion, stupor, and even coma can be seen. For most patients, the ocular and gait abnormalities will correct, but if the neurological deficits are usually permanent.

Wernicke-Korsakoff syndrome is considered a medical emergency. Prompt treatment with 100 mg thiamine, IV, can correct the ocular and gait problems and prevent progression of the neurological deficits. Thiamine supplementation and supportive care are the mainstays of continued therapy.

Key Point: Thiamine is often given empirically – along with naloxone, oxygen and hypertonic dextrose – to patients who patients who present with altered consciousness. Dextrose given to a patient with

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Wernicke-Korsakoff syndrome could be potentially dangerous; it is preferred to give thiamine first, then dextrose, or give them concurrently.

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