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The Effects of Alcohol on the Chronic Kidney Disease Population

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There have been numerous clinical studies evaluating the consequences and possible benefits of alcohol consumption in the general population. However, few studies have focused on the chronic kidney disease (CKD) population. The relationship between heavy or abusive alcohol intake and negative health outcomes, such as elevated blood pressure, some cancers, liver cirrhosis, pancreatitis, injury from automobile accidents and violence, and death, is well documented (1). The possible health benefits of moderate alcohol consumption, such as cardiovascular protection, have been widely studied, but remain controversial.

With regards to alcohol consumption, the terms moderate and heavy are unstandardized, however physicians surveyed defined moderate alcohol intake as 2.2 drinks per day, heavy alcohol intake as 3.5 drinks per day and abusive intake as 5.4 drinks per day (2). One drink refers to either a 12-oz beer, a 5-oz serving of wine or 1.5-oz of distilled spirits. In 2003, approximately 59% of adults in the United State were current drinkers, defined as consumption of at least one alcoholic beverage during the previous month (3).

The 2005 Dietary Guidelines for Americans recommend that those who consume alcohol should drink in moderation. Moderate consumption is defined as up to one drink per day for women and up to two drinks per day for men (4). In addition, it is recommended that some groups abstain from consuming alcohol, including children and adolescents, pregnant and lactating women, women who may become pregnant, people with conditions such as alcoholism and depression, and individuals taking certain medications which will be discussed in a later section.

Most patients undergoing dialysis take multiple medications daily to treat kidney disease and other comorbidities including diabetes, heart disease and hypertension. A patient who consumed alcohol prior to kidney failure may inquire whether alcohol consumption is contraindicated on dialysis. Due to the complicated health condition and medical treatment of those with CKD, patients should consult their physicians about alcohol intake. Once a patient makes an informed decision to drink alcohol, the renal dietitian is an appropriate source for evidence-based clinical recommendations regarding safe and moderate alcohol consumption for the CKD patient.

The following article addresses key counseling considerations for the renal dietitian and other healthcare practitioners regarding alcohol consumption in patients undergoing dialysis.

Key Counseling Considerations

Currently there is a scarcity of nutrition literature on the consumption of alcohol within the CKD population. One significant role of the renal dietitian is to educate patients regarding a modified nutrition plan that may limit

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phosphorus, potassium, sodium and fluid to improve health outcomes. Patients are instructed on how to make appropriate nutrition choices to meet these restrictions while maintaining optimal nutritional status. When discussing alcohol intake with CKD patients, the renal dietitian should reinforce these nutritional limitations as well as the effects of alcohol on co-morbid conditions the patients may have.

Nutrient Restrictions

Patients should be educated to make informed decisions consistent with renal nutrition plan modifications when choosing an alcoholic beverage. While some drinks may be nearly free of phosphorus, potassium and sodium, other beverages may be a hidden source of one or more of these nutrients (Table 1).

Fluid and Portion Sizes

Alcoholic beverages provide kilocalories and fluid with minimal nutritional value. One serving of alcohol equals 12-oz beer, 5-oz wine or 1.5-oz distilled spirits. Each serving contains approximately 15 grams of alcohol. Interdialytic weight gain is a concern for patients undergoing dialysis, therefore focusing on the appropriate serving sizes of alcoholic beverages allows patients to plan fluid intake and avoid exceeding daily fluid allowances.

Multiple factors play a role in determining an acceptable alcohol intake for the individual including body weight, body composition and gender. Studies show that women and men process the same amount of alcohol per hour, but women eliminate more alcohol per unit of lean body weight compared with men (6). Since women have a higher body fat to body water ratio than do men, women reach higher blood alcohol concentrations after ingesting an equivalent number of servings of alcohol (7, 8). Thus, acceptable alcohol intake for a small woman may be significantly less than what is acceptable for a large man.

Alcohol Metabolism and Drug Interactions

There is an abundant amount of literature pertaining to alcohol metabolism and drug-alcohol interactions. The following is a synopsis of alcohol metabolism and information to consider when counseling patients to avoid drug-alcohol interactions.

Alcohol has the potential to interact adversely with numerous medications (9). The metabolism of alcohol occurs mainly in the liver. The two enzymes alcohol dehydrogenase and cytochrome P450 are responsible for the breakdown of alcohol to acetaldehyde (10). Acetaldehyde is further metabolized and eliminated from the body. Cytochrome P450, also known as microsomal ethanol oxidizing system (MEOS), is central in drug- alcohol interactions (11). The two main com-

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Table 1: Nutrient Content of Portions of Common Alcoholic Beverages (5)

Beverage	K+ (mg)	P04 (mg)	Na+ (mg)	Serving Size (fl oz)
Beer, light	74	42	14	12 oz
Beer, regular	96	50	14	12 oz
Beer, Budweiser®	118	46	11	12 oz
Beer, Bud Light®	92	39	11	12 oz
Beer, Michelob® Ultra™	60	28	11	12 oz
Crème de menthe	0	0	2	1.5 oz
Daiquiri, from recipe	13	3	3	2 oz
Daiquiri, from can	11	2	40	6.8 oz
Gin, rum, whiskey, vodka	1	2	0	1.5 oz
Liqueur, coffee	16	3	4	1.5 oz
Liqueur, coffee with cream	15	24	43	1.5 oz
Martini	5	1	1	1 oz
Piña Colada, from recipe	100	10	8	4.5 oz
Piña Colada, from can	184	80	158	6.8 oz
Sake, rice	7	2	1	1 oz
Tequila Sunrise, from can	21	21	120	6.8 oz
Whiskey Sour, from mix	19	5	65	3.5 oz
Whiskey Sour, from can	23	13	92	6.8 oz
Wine, dessert	95	9	9	3.5 oz
Wine, red	115	14	5	3.5 oz
Wine, white	82	14	5	3.5 oz
NONALCOHOLIC BEVERAGES/ MIXERS				
Club soda	7	0	75	12 oz
Cola	4	48	15	12 oz
Cranberry juice	46	5	5	8 oz
Cream, half & half	39	29	12	1 oz
Eggnog	329	209	150	8 oz
Ginger Ale	4	0	26	12 oz
Grapefruit juice	378	27	2	8 oz
Lemon-lime soda	4	0	40	12 oz
Lime juice	288	34	5	8 oz
Pineapple juice	335	20	2	8 oz
Tomato juice, no added salt	556	44	24	8 oz
Tomato juice, added salt	556	44	654	8 oz
Wine, nonalcoholic	91	14	7	3.5 oz

The Effects of Alcohol *continued from page 4*

Table 2: Interaction of Drug Classes and Alcohol (10, 12, 13)

Drug Classification	Type of Interaction
Analgesics (aspirin and acetaminophen)	Alcohol intake with aspirin may increase side effects including gastric bleeding and anticoagulation. Increased gastric emptying with aspirin may cause faster alcohol absorption. Chronic alcohol intake with acetaminophen may cause liver damage.
Anesthetics (propofol)	Chronic alcohol intake increases dose needed to induce loss of consciousness. May increase risk of liver damage.
Antibiotics (furazolidone, griseofulvin, metronidazole, erthromycin, isoniazid and quinacrine)	Acute alcohol intake may cause nausea, vomiting, headache, and convulsions. Erthromycin may increase gastric emptying causing faster alcohol absorption. Increased risk of isoniazid-related liver disease.
Anticoagulants (warfarin)	Chronic alcohol intake decreases anticoagulation. Acute alcohol intake increases anticoagulation by reduces drug's metabolism.
Anticonvulsants (phenytoin)	Acute alcohol intake increases availability and the risk of side effects. Chronic alcohol intake decreases availability, even during abstinence, decreasing drug effectiveness.
Antidepressants (tricyclics and Monoamine oxidase inhibitors including amitriptyline, clomipramine, desipramine, doxepin, imipramine, and nortriptyline)	Acute alcohol intake increases availability, increasing sedative effects with tricyclics. Tyramine, in some beers and wine, may cause a severe alteration in blood pressure with MAO inhibitors.
Antihistamines (diphenhydramine)	May cause dizziness and increase side effects on CNS causing drowsiness, sedation and decreased motor skills. The elderly may be more prone to these interactions.
Antipsychotics (chlorpromazine)	Acute alcohol intake enhances sedative side effects, may cause impaired coordination and fatal breathing complications.
Cardiovascular (nitroglycerin, reserpine, methyldopa, Isosorbide dinitrate, hydralazine, and guanethidine)	Acute alcohol intake may cause dizziness or fainting when standing up. Chronic alcohol intake decreases availability, reducing effectiveness.
Herbal remedies (chamomile, echinacea and valerian)	Alcohol may intensify the drowsiness associated with these supplements.
Histamine H_2 receptor antagonists (cimetidine, nizatidine, and ranitidine)	These drugs decrease gastric ADH and increase gastric emptying, leading to higher than expected blood alcohol levels per alcohol serving.
Narcotic pain relievers (morphine, codeine, propoxyphene, and meperidine)	Combining alcohol with opiates increases side effects of both drugs, affecting the CNS causing drowsiness, sedation and decreased motor skills; may increase risk of death by overdose.

ponents of cytochrome P450 are the enzymes cytochrome P450 reductase and CYP2E1 (11). CYP2E1 metabolizes both alcohol and numerous drugs. Alcohol may alter the effectiveness of a drug by interfering with its availability or the extent to which the drug reaches the target site at a specific dose (9). The following are common mechanisms of pharmacokinetic alcohol-drug interactions:

1) Acute alcohol ingestion (moderate) may inhibit a drug's metabolism by competing with the drug for cytochrome P450, which increases and prolongs the drug's availability.

2) Chronic heavy alcohol intake (in nonintoxicated state) enhances cytochrome P450 activity, leading to inadequate drug levels and decreased effectiveness. In addition, enzymes associated with chronic alcohol use can breakdown medications into toxins, which may accumulate and cause organ damage.

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3) Chronic heavy alcohol intake (during intoxicated state) requires activation and primary involvement of CYP, reducing drug metabolism and excretion (11).

While pharmacokinetic interactions are of a primary concern in chronic heavy drinkers, pharmacodynamic interactions may occur during a single episode of moderate drinking (10). Pharmacodynamic drug- alcohol interactions often occur in the central nervous system when alcohol intake changes a drug's effect without altering serum drug levels. The type of interaction depends on the class of medication involved (Table 2).

Vitamin Deficiency

Vitamin deficiencies are common with excessive alcohol consumption (14). Two common vitamin deficiencies in people with alcoholism are folate and thiamine deficiencies, however multiple nutrient deficiencies may occur as alcohol interferes with the conversion and storage of vitamins in the liver (14, 15). In addition, the replacement of nutrient dense foods with alcohol further contributes to these deficiencies. Folate deficiency is likely caused by an increased need for nucleic acids to regenerate damaged liver cells (14). One study using both humans and experimental primates found an association of chronic excessive alcohol intake with intestinal malabsorption, decreased uptake by the liver and increased urinary excretion of folate, which led to low liver folate levels (16).

One study found 20% of "persistent alcohol misusers" to have abnormally low thiamine-dependent transketolase activity, suggesting an increased requirement for thiamine (15). Thiamine deficiency may lead to Wernicke-Korsakoff Syndrome, a degenerative brain disorder, which affects the heart, vascular system and nervous system. This syndrome is associated with cognitive memory deficit, impairing the retrieval of previous memories and the capacity to create new memories. In addition, vision impairment, coma and ataxia leave the patient further disabled (17). The registered dietitian plays a significant role in screening patients in the prevention and treatment of vitamin deficiencies.

Alcohol Intake and Diabetes

Currently, 43% of the CKD Stage 5 population in the United States has diabetes (18). Experimental studies have been conducted to assess the relationship between alcohol ingestion and glycemic control in individuals with type 1 and type 2 diabetes (19, 20, 21, 22). When subjects with type 1 and type 2 diabetes consumed one gram of alcohol per kilogram body weight with food, subjects with type 2 diabetes had slightly lower blood glucose concentrations the following morning (19). Many studies have compared the effects of alcohol when fasting versus with food in subjects with type 2 diabetes (20, 21, 22). Hypoglycemia was not experienced in subjects with diabetes after consuming alcohol with food. The studies of light to moderate alcohol doses with or without food showed no short-term effect of alcohol on glycemic control. A statistically significant relationship was found between low blood glucose in persons with type 2 diabetes and alcohol infusion while fasting (22). These findings were consistent with an early study of alcohol and low blood glucose in males with type 1 diabetes (23).

The relationship between alcohol and carbohydrate metabolism is complex and varies according to the amount of alcohol consumed, timeframe of consumption, nutritional status of the individual and presence or absence of food (24). Alcoholic hypoglycemia has been attributed to the inhibition of gluconeogenesis by alcohol (25). Furthermore, alcohol may affect the utilization and production of glucose by interfering with the uptake of lactate (26).

The American Diabetes Association recommends that alcoholic beverages be planned in conjunction with a regular meal (27). In addition, to avoid the risk of hypoglycemia individuals with diabetes should not substitute food with alcohol.

Cardiovascular and Lipid Profile Benefits

Nearly 50% of the deaths of CKD patients may be attributed to cardiovascular disease (28). The "French Paradox," which first captured mainstream attention when highlighted on a *60-Minutes* broadcast in 1992, catalyzed studies of the potential benefits of light to moderate alcohol intake, especially red wine, on the progression of cardiovascular disease (29). Alcohol is known to increase the level of serum high-density lipoprotein (HDL) and lower serum lipoprotein a (30, 31). HDL is protective against atherogenesis while high amounts of lipoprotein a promote the process (14). One study of alcohol consumption and atherosclerotic

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risk in type 2 diabetes found that heavy alcohol intake was associated with both increased serum HDL levels (negative risk factor) and high serum triglyceride levels (positive risk factor). However, there was no difference in these two factors between nondrinkers and light drinkers (32). This suggests that the potential beneficial changes in serum lipid profile with light to moderate alcohol intake in nondiabetics may not be translated to the diabetic population.

There have been numerous studies of the cardioprotective value of moderate wine consumption related to antioxidant properties. Some experts are examining the role of oxidative stress, endothelial dysfunction and the intake of wine among CKD patients (33). Currently there is not conclusive evidence to recommend the intake of alcohol, a nonessential nutrient, as a cardioprotective diet modification for individuals abstaining from alcohol.

Cultural and Ethnic Considerations

Heavy drinking is most prevalent among American Indians and Alaska Natives and lowest among Asian Americans and Pacific Islanders (34). Among adolescents, Hispanics have the highest annual prevalence of heavy drinking (35). However, acculturation of ethnic groups causes individuals to assume the drinking patterns of the general population.

There appears to be genetic factors, which affect the body's ability to metabolize and eliminate alcohol (36). It is common among Asian subpopulations to have inactivity of the enzyme aldehyde dehydrogenase-2, leading to flushing of the skin, nausea and uncomfortable symptoms. The potential variation in the enzyme alcohol dehydrogenase-2 among some African Americans may affect the vulnerability of this population to alcoholic cirrhosis and alcohol-related fetal defect (36). There are ethnic differences in drinking patterns, and an understanding of the ethnic context of alcohol consumption should be utilized when working in the community.

Summary

The renal dietitian has the training and expertise to educate dialysis patients about the appropriate consumption of alcohol within the parameters of the prescribed renal nutrition plan. Patients with CKD should discuss the consumption of alcoholic beverages with attending physicians. The current literature provides evidence that the negative affects of alcohol outweigh any potential health protective value. In addition, there is a lack of research on alcohol ingestion specific to the CKD population. By providing patients who choose to use alcohol with recommendations and encouraging moderate and safe alcohol consumption, renal healthcare professionals may empower patients to make responsible choices leading to improved quality of life and better health outcomes.

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