Nutritional status in patients with sustained heavy alcohol use

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INTRODUCTION

Excessive alcohol use is highly prevalent and a major cause of nutritional deficiency in developed countries [1]. Alcohol causes nutritional complications from both its primary effects on the intake and metabolism of nutrients and secondary effects of end organ damage (eg, alcohol induced liver disease, pancreatitis) [1-8]. Modest alcohol use may be beneficial for cardiovascular and cerebrovascular risk reduction [2]; however, heavy alcohol use is associated with significant morbidity and mortality [3]. (See "Overview of the risks and benefits of alcohol consumption" and "Cardiovascular benefits and risks of moderate alcohol consumption" and "Risky drinking and alcohol use disorder: Epidemiology, clinical features, adverse consequences, screening, and assessment".)

Malnutrition results from sustained, heavy alcohol use, including what was described as alcohol dependence in the Diagnostic and Statistical Manual, fourth edition (DSM-IV) [9]. The diagnoses, alcohol abuse and alcohol dependence, were replaced by one diagnosis, alcohol use disorder, in DSM-5 [10]. Although the crosswalk between DSM-IV and DSM-5 disorders is imprecise, alcohol dependence is approximately comparable to the moderate to severe subtype of alcohol use disorder.

This topic addresses the epidemiology, pathogenesis, assessment, and treatment of malnutrition in individuals with sustained heavy alcohol use. Malnutrition in patients with

chronic liver disease, from alcohol or other causes, is discussed elsewhere. (See "Evaluating nutritional status in adults with cirrhosis".)

EPIDEMIOLOGY OF ALCOHOL-RELATED MALNUTRITION

With the exception of alcoholic steatosis without inflammation, the prevalence of malnutrition varies depending on the presence and degree of cirrhosis and ranges between 20 to 60 percent [11]. Individuals without liver disease may be nutritionally normal, deficient in micronutrients, or have significant protein-calorie malnutrition [12]. In one study, body protein and lean body mass were similarly reduced in alcoholic patients with or without liver disease [13]. There are no reliable predictors of who will develop malnutrition as a result of moderate to heavy alcohol use.

PATHOGENESIS OF MALNUTRITION

Both acute and chronic alcohol consumption can cause malnutrition by decreasing dietary caloric intake, impairing nutrient digestion and absorption, decreasing protein synthesis and secretion, increasing catabolism of gut proteins, and increasing breakdown and excretion of nutrients [14,15]. The degree of malnutrition depends on the amount of alcohol consumed, the quality of food intake, genetics, and the presence and severity of comorbid illnesses. The risk of developing micro- and macronutrient deficiencies increases significantly when alcohol makes up more than 30 percent of total caloric intake [14].

Effects of alcohol on dietary intake — The caloric content of an alcoholic beverage is comprised of calories from both alcohol (7.1 kcal/gram alcohol) and its non-alcohol components (alcohol calorie calculator) [16]. Individuals who drink modest amounts of alcohol (up to 20 grams per day) typically do not reduce their intake in food calories [17,18].

With increasing levels of sustained alcohol intake, however, the proportion of food calories consumed decreases as individuals consume up to 50 percent of their daily calories from alcohol [16,19]. Anorexia, nausea, vomiting, and abdominal pain related to gastritis can also contribute to reduced food intake. Alcohol increases serum leptin and cytokines (eg, serum TNF-alpha), which may contribute to anorexia [20,21].

Effects of alcohol on absorption and digestion — Alcohol is absorbed by simple diffusion in the stomach and, to a lesser degree, in the duodenum and jejunum. Acute alcohol ingestion causes mucosal erosions and villous-predominant epithelial loss [22]. The effects of chronic alcohol ingestion on intestinal mucosa are not well understood but may include fibrous tissue

accumulation [22]. Chronic alcohol ingestion reduces nutrient absorption [22,23], which along with bacterial overgrowth, increased intestinal permeability, and altered intestinal motility all contribute to malnutrition [24-27].

Effects of alcohol on energy metabolism — Individuals with modest alcohol consumption do not eat less to compensate for the calories provided by alcohol. Individuals with high levels of alcohol intake, however, paradoxically lose weight [28].

Chronic consumption of alcohol and a standard calorie diet reduces adipose accumulation and causes an energy deficit attributed to alcohol damage to mitochondria, increased thermogenesis, sympathetic nervous system activation, and oxidation of alcohol [29,30]. Additionally, if present, acute pancreatitis, alcoholic liver disease, and alcoholic hepatitis induce a hypermetabolic state [6,31]. (See "Evaluating nutritional status in adults with cirrhosis", section on 'Epidemiology and risk factors'.)

Effects of alcohol on macronutrient metabolism

Protein calorie malnutrition — Protein calorie malnutrition (PCM) occurs primarily in patients with advanced alcoholic liver disease and is most commonly identified in patients (8 to 30 percent) hospitalized for alcohol-related liver damage [4,32,33]. PCM is caused by inadequate protein intake and is a marker of advanced disease severity and poor prognosis. (See "Evaluating nutritional status in adults with cirrhosis", section on 'Epidemiology and risk factors'.)

NUTRITIONAL ASSESSMENT

Evaluation for the presence of malnutrition, including a focused history, physical examination, and laboratory testing, is indicated for individuals with heavy alcohol use (for men, more than four drinks per day or more than 14 drinks per week; for women, more than three drinks per day or more than seven drinks per week) [34] or who meet criteria for alcohol use disorder, moderate to severe subtype. (See "Risky drinking and alcohol use disorder: Epidemiology, clinical features, adverse consequences, screening, and assessment".)

The diagnosis of malnutrition, based on expert consensus, requires identifying two or more of the following six characteristics [35]:

- Insufficient energy intake.
- Weight loss.
- Decrease in muscle mass.

- Loss of subcutaneous fat.
- Edema (localized or generalized) that can mask weight loss.
- Diminished handgrip strength (measured by a dynamometer) [36,37].

Patients with sustained heavy drinking in whom malnutrition is suspected should be referred to a nutritionist for further evaluation.

Multiple validated measures exist for nutritional assessment, including the subjective global assessment (SGA), which combines historical cues (eg, weight loss, oral intake, weakness) with signs on physical examination (eg, muscle wasting, fat distribution, edema).

The assessment of nutritional status in patients with chronic liver disease is discussed separately. (See "Dietary assessment in adults" and "Evaluating nutritional status in adults with cirrhosis".)

Approach in patients with alcohol use disorder — For patients with a moderate to severe alcohol use disorder, nutritional assessment involves asking the patient about weight loss, fatigue, loss of muscle strength, edema, gastrointestinal symptoms, and dietary history. On physical examination, findings that support malnutrition include weight loss, decreased body mass index, muscle mass, and subcutaneous fat. Although laboratory testing is not required for the diagnosis of malnutrition, laboratory abnormalities may contribute to the assessment. Thus, we typically evaluate patients with alcohol use disorder with the following studies: complete blood count, liver function tests, international normalized ratio, albumin, full electrolyte panel, and serum folate and vitamin B12.

History — Individuals with heavy alcohol use or an alcohol use disorder should be asked about elements of history that support the presence of malnutrition: weight loss, dietary history, decreases in muscle strength, nausea, vomiting, abdominal pain, hematemesis, and diarrhea. In addition, there may be symptoms of liver disease, such as fatigue, lower extremity edema, easy bruising, fever, diarrhea, pruritus, increasing abdominal girth, confusion, or sleep disturbances.

Physical examination — Physical examination findings that indicate malnutrition include: weight loss, decreased body mass index (calculator 1), muscle wasting, loss of subcutaneous fat, edema, and decreased handgrip strength. Handgrip strength is measured by a dynamometer. In addition, there may be signs of cirrhosis (eg, hepatomegaly, splenomegaly, spider angiomata, ascites, nail changes, gynecomastia, testicular atrophy, jaundice). (See "Cirrhosis in adults: Etiologies, clinical manifestations, and diagnosis", section on 'Physical examination'.)

Deficiencies of thiamine, folate, iron, and vitamin B6 are common in individuals with chronic alcohol use. Both fat– and water-soluble vitamin deficiencies occur with moderate to severe alcohol use disorder [38]. The risk of deficiency increases with higher levels of alcohol intake. Some symptoms of vitamin deficiencies (eg, Wernicke's encephalopathy) do not manifest clinically until late-stage disease. Features that indicate certain micronutrient deficiencies are: pallor (iron deficiency), hyperkeratosis and dermatitis (vitamin A), bruising (vitamin C and K), and glossitis (folate, vitamin B12) (table 1). (See "Overview of vitamin D" and "Overview of vitamin K" and "Overview of vitamin A" and "Overview of vitamin E" and "Treatment of vitamin B12 and folate deficiencies" and "Overview of water-soluble vitamins".)

Laboratory findings — Although not required for a diagnosis of malnutrition, the following laboratory findings can support a diagnosis of malnutrition or the presence of liver disease and should be checked in all individuals with heavy alcohol use or an alcohol use disorder:

- Decreased hematocrit or hemoglobin
- Abnormal aspartate aminotransferase and alanine aminotransferase
- Elevated international normalized ratio
- Decreased albumin
- Decreased folate and vitamin B12 levels
- Abnormal electrolytes and creatinine
- Decreased calcium, magnesium, and phosphate levels

However, most of these laboratory markers are better indicators of liver disease severity than they are of nutritional status and should be used only as part of the global assessment of nutritional status [39].

SUPPLEMENTATION

The goal of supplementation is to restore adequate caloric intake and to prevent symptoms of micronutrient deficiencies. Nutritional therapy in malnourished patients **without** liver disease has not been studied. However, in line with consensus group guidelines for patients with alcoholic liver disease, we suggest frequent feedings, breakfast and nighttime snacks, an energy intake of 35 to 40 kcal/kg of body weight/day and protein intake of 1.2 to 1.5 g/kg of body weight/day [40,41].

Although there are no data from randomized trials of vitamin supplementation in alcoholic patients, we suggest empiric administration of an oral multivitamin including thiamine, folic acid, and vitamin B6 (pyridoxine) in all adults with sustained heavy alcohol use [42]. Since

multivitamin formulations contain variable doses of component vitamins, the selected multivitamin should contain: thiamine 100 mg, pyridoxine 2 mg, and folic acid 400 mcg to 1 mg. They should not exceed the tolerable upper intake levels (table 2). (See "Vitamin intake and disease prevention" and "Overview of water-soluble vitamins".)

Nutritional therapy for malnourished patients with alcoholic liver disease is discussed separately. (See "Management of alcohol-associated steatosis and alcohol-associated cirrhosis", section on 'Nutrition'.)

Fat-soluble vitamins

Vitamins A, D, E, and K — Vitamin A, D, E, and K levels are often deficient in patients with chronic pancreatitis or alcoholic liver disease [7]. Vitamin A and K deficiencies are rare in the absence of liver disease or chronic pancreatitis, thus we do not suggest routine supplementation with vitamins A, D, E, or K.

Manifestations of specific vitamin deficiencies include:

- Vitamin A: night blindness, sexual dysfunction, or immune deficiency (see "Overview of vitamin A", section on 'Replacement') [43].
- Vitamin D: can predispose patients to osteomalacia, osteopenia, and fractures [43].
- Vitamin E: hemolysis, myopathy, and retinopathy [44,45].
- Vitamin K: hemostatic disturbance.

(See "Overview of vitamin A" and "Overview of vitamin D" and "Overview of vitamin E" and "Overview of vitamin K".)

Water-soluble vitamins

Vitamin B1 (thiamine) — Thiamine deficiency is found in up to 80 percent of adults with chronic alcohol use [42,46] and is more common in those with advanced liver disease. Deficiency may be caused by insufficient intake, reduced gastrointestinal absorption, or reduced storage [47]. Thiamine deficiency can cause peripheral neuropathy, cardiomyopathy, and the Wernicke-Korsakoff's syndrome. Wernicke's encephalopathy is characterized by the triad of delirium, oculomotor abnormalities, and ataxia. Untreated Wernicke's encephalopathy can progress to Korsakoff's syndrome, which is characterized by anterograde and retrograde amnesia and confabulation. (See "Wernicke encephalopathy" and "Overview of the chronic neurologic complications of alcohol".)

As a preventive measure, oral thiamine 100 mg daily should be given to all adults with sustained heavy drinking. In hospitalized individuals with alcohol use disorder, it is especially important to

give thiamine supplementation prior to or along with glucose intravenous infusion to prevent precipitating Wernicke's encephalopathy. Patients admitted for alcohol withdrawal typically receive thiamine 100 mg daily. (See "Overview of water-soluble vitamins", section on 'Vitamin B1 (thiamine)' and "Wernicke encephalopathy", section on 'Prevention'.)

Vitamin B2 (riboflavin) — Riboflavin deficiency is less common than other vitamin deficiencies in patients with an alcohol use disorder and is primarily caused by reduced dietary intake. Symptomatic riboflavin deficiency is rare but can include oropharyngeal inflammation, behavioral change, neuropathy, anemia, or dermatitis. We do not supplement empirically. If deficient, then the recommended daily riboflavin replacement is 1.7 mg/day, found in most multivitamin preparations. (See "Vitamin intake and disease prevention".)

Vitamin B6 (pyridoxine) — Pyridoxine deficiency occurs in over 50 percent of alcoholic patients and is caused by reduced intake and increased breakdown of pyridoxine during ethanol metabolism [48,49]. Clinical manifestations are uncommon but include peripheral neuropathy, stomatitis, confusion, or depression [50]. For vitamin B6, we suggest oral supplement of 2 mg per day [42]. Some multivitamin preparations contain more than the recommended daily dosage. (See "Vitamin intake and disease prevention".)

Folate — Two-thirds of binge drinkers will have folate deficiency caused by malabsorption, reduced intake, and increased urinary excretion [22,48,51]. This can cause macrocytic anemia and intestinal malabsorption. We suggest supplementation of folic acid 400 mcg per day. Multivitamin preparations often contain 400 mcg of folic acid [42]. (See "Vitamin intake and disease prevention" and "Clinical manifestations and diagnosis of vitamin B12 and folate deficiency".)

Vitamin B12 (cobalamin) — In the absence of chronic pancreatitis, cobalamin deficiency is rarely present in adults with an alcohol use disorder [7,22]. Clinical manifestations of cobalamin deficiency include macrocytic anemia and neuropsychiatric symptoms such as cognitive impairment, irritability, gait abnormalities, and peripheral neuropathy. We do not routinely supplement vitamin B12. Oral doses of more than 50 mcg/day may be needed for replacement in patients with B12 deficiency; multivitamin preparations usually do not contain enough for replacement of cobalamin deficiency [42]. (See "Vitamin intake and disease prevention" and "Clinical manifestations and diagnosis of vitamin B12 and folate deficiency".)

Vitamin C — Vitamin C deficient patients present with scurvy, characterized by bleeding gums, ecchymoses, fatigue, and depression [52]. The recommended daily dose of vitamin C to prevent scurvy is 60 mg/day, which is found in most multivitamin preparations. (See "Vitamin intake and disease prevention" and "Overview of water-soluble vitamins".)

Minerals and trace elements

Calcium, magnesium, and phosphorus — Deficiency of calcium, magnesium, or phosphorus may be related to decreased intake, malabsorption, or renal losses. Calcium deficiency may also be secondary to magnesium or vitamin D deficiency [7,53].

Calcium, magnesium, and phosphorus levels should be checked in all patients with an alcohol use disorder or heavy alcohol use. In the presence of liver disease, total calcium may need to be adjusted for low albumin levels. Replacement of calcium, magnesium, or phosphorus is based on blood levels.

Magnesium replacement doses range from 100 to 400 mg daily. Intravenous replacement may be needed with symptomatic deficiency (weakness, lethargy, nausea) [42]. Oral phosphate is replaced at 15 to 20 mg/kg body weight per day, in the absence of significant renal impairment. If patients cannot tolerate oral phosphate supplementation or if the serum level is below 1 mg/dL (0.32 mmol/L) and patients are symptomatic, then intravenous phosphate should be administered. Phosphate therapy is discussed separately. (See "Hypophosphatemia in the patient with alcohol use disorder" and "Treatment of hypocalcemia", section on 'Therapeutic approach'.)

Iron — Iron stores are often increased with chronic alcohol consumption, even in the absence of liver disease. This is partly due to increased paracellular absorption of iron in the liver [54,55]. If iron deficiency occurs, potential sources of blood loss must be ruled out and iron should be replaced. (See "Treatment of iron deficiency anemia in adults".)

Water and electrolytes — Reduced sodium and water absorption in the small intestine with chronic alcohol use predispose to diarrhea [22]. In combination with inebriation, nausea, vomiting, and poor intake of water, reduced sodium and water absorption can predispose to dehydration and hyponatremia.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Alcohol use disorders and withdrawal" and "Society guideline links: Healthy diet in adults".)

SUMMARY AND RECOMMENDATIONS

- Malnutrition results from sustained, heavy alcohol use, including what was described as alcohol dependence in the Diagnostic and Statistical Manual, fourth edition (DSM-IV). The diagnoses, alcohol abuse and alcohol dependence, were replaced by one diagnosis, alcohol use disorder, in DSM-5. Although the crosswalk between DSM-IV and DSM-5 disorders is imprecise, alcohol dependence is approximately comparable to the moderate to severe subtype of alcohol use disorder. (See 'Introduction' above and "Risky drinking and alcohol use disorder: Epidemiology, clinical features, adverse consequences, screening, and assessment".)

- Sustained, heavy alcohol use, without liver disease, is associated with a range of macro- and micronutrient deficiencies. Adults with such intake are more likely to lose weight due to metabolic changes associated with excess alcohol intake. Alcohol causes malnutrition through effects on dietary intake, absorption, digestion, and resting energy expenditure. Malnutrition is associated with significant morbidity and mortality. (See 'Pathogenesis of malnutrition' above.)

- All individuals with a moderate to severe alcohol use disorder need to have a complete nutritional assessment. The primary care physician can initiate the evaluation with a focused history, physical examination looking for signs of malnutrition, and supplemented with laboratory findings. We ask about weight loss, fatigue, decrease in muscle strength, edema, gastrointestinal symptoms, and dietary history. On physical examination, findings that support malnutrition include weight loss, decreased body mass index, muscle mass, and subcutaneous fat. Although laboratory findings are not required for diagnosis of malnutrition, they contribute to the assessment. Thus, we typically evaluate patients with the disorder with the following studies: complete blood count, liver function tests, international normalized ratio, albumin, full electrolyte panel, and serum folate and vitamin B12. Nutritional consultation is appropriate for patients at higher risk for micronutrient deficiencies and malnutrition, especially those whose alcohol intake exceeds 30 percent of their total caloric intake. (See 'Nutritional assessment' above.)

- In adults with sustained, heavy alcohol use, the most common micronutrient deficiencies are deficiencies of thiamine (vitamin B1), pyridoxine (vitamin B6), and folate.

  Heavy alcohol use is considered to be more than four drinks per day or more than 14 drinks per week for men and more than three drinks per day or more than seven drinks per week for women. For adults with an alcohol use disorder or heavy alcohol use, we recommend supplementation with a multivitamin that contains a minimum of thiamine 100 mg daily, vitamin B6 2 mg daily, and folic acid 400 mcg to 1 mg daily (**Grade 1C**). (See 'Supplementation' above.)

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