StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-.

Alcohol-Associated Liver Disease

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Last Update: July 13, 2023.

Continuing Education Activity

The first stage of alcoholic liver disease is hepatic steatosis, which involves the accumulation of small fat droplets under liver cells approaching the portal tracts. More advanced disease is characterized by marked steatosis, hepatocellular necrosis, and acute inflammation, known as alcoholic hepatitis. There is a need for more effective treatment of alcoholic liver disease as the severe form of the disease is life-threatening. This activity reviews the evaluation and management of alcoholic liver disease and highlights the role of the interprofessional team in the recognition and management of this condition.

Objectives:

- Summarize the conditions and factors that aggravate alcoholic liver disease.
- Outline strategies for decreasing alcohol dependency and/or abuse in patients with alcoholic liver disease.
- Review the treatment options available for alcoholic liver disease.
- Describe interprofessional team strategies for improving care coordination and communication to ameliorate outcomes in patients with alcoholic liver disease.

Access free multiple choice questions on this topic.

Introduction

The alcoholic liver disease covers a spectrum of disorders beginning from the fatty liver, progressing at times to alcoholic hepatitis and culminating in alcoholic cirrhosis, which is the most advanced and irreversible form of liver injury related to the consumption of alcohol.

There are three histologic stages of alcoholic liver disease[1][2]:

1. **Alcoholic Fatty Liver or Steatosis -** At this stage, fat accumulates in the liver parenchyma.
2. **Alcoholic Hepatitis -** Inflammation of liver cells takes place at this stage, and the outcome depends on the severity of the damage. Alcohol abstinence, nutritional support, treatment of infection, and prednisolone therapy in severe cases can help in the treatment of alcoholic hepatitis, but more severe cases lead to liver failure.
3. **Alcoholic Cirrhosis -** Liver damage at this stage is irreversible and leads to complications of cirrhosis and portal hypertension.

Etiology

Different factors, such as metabolic, genetic, environmental, and immunological, collectively play a role in alcoholic liver disease.

The liver tolerates mild alcohol consumption, but as the consumption of alcohol increases, it leads to disorders of the metabolic functioning of the liver. The initial stage involves the accumulation of fat in the liver cells, commonly known as fatty liver or steatosis. If the consumption of alcohol does not stop at this stage, it sometimes leads to alcoholic hepatitis. With continued alcohol consumption, the alcoholic liver disease progresses to severe damage to liver cells known as "alcoholic cirrhosis." Alcoholic cirrhosis is the stage described by progressive hepatic fibrosis and nodules.

Quantity and duration of the patient's alcohol intake are the highest risk factors for the development of liver disease. The beverage type plays a minimal role. Women are more susceptible than men. Obesity and high-fat diet also increase the risk of alcoholic liver disease. Concurrent hepatitis C infection is associated with younger age of onset, more advanced histological damage, and decreased survival. Patatin-like phospholipase domain-containing protein 3 (PNPLAP3) is associated with alcoholic liver cirrhosis.

Epidemiology

Alcohol is the most frequently misused drug throughout the entire world and in the United States of America.

In the United States, it is the leading cause of liver disease. It involves 61 percent of the American population, and among the 61 percent, 10 to 12 percent are heavy drinkers.

Definition of one alcohol drink as per the Centers for Disease Control and Prevention (CDC) is a half-ounce or 13.7 g pure alcohol which is the amount of alcohol present in

- 12 oz beer (5% alcohol)
- 8 oz malt liquor (7% alcohol)
- 5 oz wine (12% alcohol)
- 1.5 oz 80-proof “hard-liquor” (40% alcohol)

The prevalence of alcoholic liver disease is highest in European countries. Daily consumption of 30 to 50 grams of alcohol for over five years can cause alcoholic liver disease. Steatosis can occur in 90% of patients who drink over 60 g/day, and cirrhosis occurs in 30% of individuals with long-standing consumption of more than 40 g/day.

At-risk drinking definitions are below:

- Men: over 14 drinks per week or more than four drinks per occasion
- Women and those over 65 years: over seven drinks per week or greater than three drinks per occasion

Definitions of significant drinking from a liver toxicity standpoint are as below (this history is essential to differentiate non-alcoholic fatty liver disease (NAFLD) from alcoholic fatty liver disease (AFLD)

- Men: more than 21 drinks per week
- Women: over 14 drinks per week

Pathophysiology

Alcohol metabolism by the liver is primarily via two enzymes:

1. Alcohol dehydrogenase
2. Aldehyde dehydrogenase

Alcohol dehydrogenase converts alcohol into acetaldehyde, and aldehyde dehydrogenase converts acetaldehyde into acetate. The metabolism of alcohol increases the production of NADH by reducing NAD in the body. This shifting of metabolic balance toward the production of NADH leads to the formation of glycerol phosphate, which combines with the fatty acids and becomes triglycerides, which accumulate within the liver. When lipid oxidation (lipolysis) stops due to alcohol consumption, fats accumulate in the liver and lead to "fatty liver disease." Continued alcohol consumption brings the immune system into play. Interleukins with the help of neutrophils attack the hepatocytes, and swelling of the hepatocytes known as the "alcoholic hepatitis" takes place. Ongoing liver injury leads to irreversible liver damage, the cirrhosis of the liver.

Histopathology

Histopathology at Different States of Alcoholic Hepatitis:

The first stage is hepatic steatosis. It involves the accumulation of small fat droplets around liver cells, specifically around the venules, and approaches the portal tracts. The altered intracellular redox potential leads to the accumulation of intracellular lipids. Fatty liver is generally considered a reversible condition.

On further progression, there is marked steatosis, hepatocellular necrosis, and acute inflammation. Eosinophilic fibrillar material (Mallory hyaline or Mallory-Denk bodies) forms in swollen (ballooned) hepatocytes. This stage is known as alcoholic hepatitis. Severe lobular infiltration of polymorphonuclear leukocytes (neutrophils) is abundantly present in this condition in contrast to most other types of hepatitis where mononuclear cells localize around portal triads.

The end-stage of liver disease is alcoholic cirrhosis. At this stage, fibrotic septae surround regenerative nodules in the liver.[2]

The deposition of collagen typically occurs around the terminal hepatic vein (perivenular fibrosis) and along the sinusoids, leading to a peculiar "chicken wire" pattern of fibrosis in alcoholic cirrhosis.

For the optimal assessment of liver fibrosis, it must be appreciated by specific stains, as Masson Trichrome or Sirius Red.

To note that the above stages are not absolute or necessarily progressive. An overlap of the above stages and features of all three histologic stages can be present in one individual with long-standing alcohol abuse. Discontinuation of alcohol intake may cause regression of all the above stages.

History and Physical

Drinking history is an essential component, which includes the number of drinks per day and the duration of drinking. Given the lack of a unique diagnostic test, the exclusion of other causes of liver injury is mandatory.

Personal and psychosocial factors are also important because excessive drinking is related to depression and other psychological diseases.

Patients should be asked about diet, alcohol consumption, caloric intake, risk factors for malnutrition, and risk factors for chronic liver diseases such as viral hepatitis.

Physicians should explore signs and symptoms, including:

- Nausea and vomiting
- Abdominal pain or discomfort
- Loss of appetite
- Weight loss or weight gain
- Increased thirst
- Yellowish discoloration of eyes
- Weakness
- Fever (in alcoholic hepatitis)
- Confusion
- Alteration of the sleep-wake cycle
- Mood swings
- Fainting

Physical Examination

The clinical definition of alcoholic hepatitis is a syndrome of liver failure where jaundice is a characteristic feature; fever and tender hepatomegaly are often present. The typical presentation age is between 40 and 50 yrs, and it occurs in the setting of heavy alcohol use. Patients often report a history of intake of at least 30 to 50 g alcohol/day though over 100 g/day is common. Patients may be abstinent for weeks before admission. The cardinal sign is the rapid onset of jaundice. Other signs and symptoms include fever, ascites (SAAG greater than 1.1), and proximal muscle loss. Patients presenting with severe alcoholic hepatitis may have encephalopathy. Typically, the liver is enlarged and tender.

General physical examination typically shows jaundice, hepatomegaly, splenomegaly, spider telangiectasias, Dupuytren contractures, testicular atrophy, decreased libido, parotid and lacrimal gland enlargement, white nails, Muecke lines, asterixis, and features of portal hypertension such as ascites, pedal edema, encephalopathy, and caput-medusae (distended and engorged superficial abdominal veins).[3]

Abdominal paracentesis should be performed in all patients with newly identified ascites.

Evaluation

Evaluation should include[3]:

- CBC (Complete blood count) to rule out the infection, look for complications of cirrhosis: anemia, thrombocytopenia, a leukemoid reaction in alcoholic hepatitis.
- LFTs (liver function tests): AST (aspartate aminotransferase) is markedly raised as opposed to ALT (alanine aminotransferase) in alcoholic liver disease. There is hypoalbuminemia, hyperbilirubinemia, and hypertriglyceridemia. Also, GGTP (gamma-glutamyl transpeptidase) is usually raised.
- Prothrombin time (PT) and INR (to assess liver synthetic function): an elevated value indicates more severe disease.
- Abdominal imaging (abdominal ultrasonography) is useful in looking for biliary obstruction and liver tumors.
- BMP (basic metabolic profile) should be ordered to look for renal failure and electrolyte disturbance (low levels of potassium, magnesium, and phosphorus).
- Ascitic fluid SAAG (serum-ascites albumin gradient) should be calculated to assess the reason for ascites if present.
- Screening blood tests for other causes of chronic liver disease, including viral hepatitis.
- Endoscopy to look for esophageal varices due to portal hypertension in patients with cirrhosis.
- A liver biopsy can lead to a definitive diagnosis in cases where the diagnosis is uncertain. More often than not, it is used for evaluation of severity, prognosis, staging, and treatment monitoring. For an accurate diagnosis of fibrosis, at least a 1.5 to 2 cm long sample of liver tissue is necessary. Liver biopsy has a risk of complication, including life-threatening hemorrhage, so it is reserved for cases where the results of a biopsy can make a difference in the treatment plan.
- Elevated CA-125 levels were noted in 85% of patients with cirrhosis in one study. The more advanced the degree of decompensation based on MELD score, Child's Turcotte-Pugh classification, and ALBI score, the higher the elevation in CA-125.[4]

Treatment / Management

Management of alcohol liver disease depends on the extent of the disease. **Medical Treatment**

- Alcohol abstinence, enrollment to detoxification programs
- Nutritional support
- Screening for hepatocellular carcinoma with ultrasonography every six months and screening for esophageal varices in those with cirrhosis
- Chronic alcoholics are more prone to develop hepatotoxicity from acetaminophen, so dosing should not exceed more than 2000 mg per day. An average person can tolerate up to 4000 mg of acetaminophen per day.
- Treatment of co-existing liver diseases such as Hepatitis B and C viral infections

Surgical Treatment

If liver damage is irreversible, definitive treatment is a liver transplant in those who have shown a commitment to continued alcohol abstinence.**Specific Treatment of Alcoholic Hepatitis** (as adapted from the chapter on Hepatitis, Alcoholic)

Abstinence, along with adequate nutritional support, remains the cornerstone of the management of patients with alcoholic hepatitis. An addiction specialist could help individualize and enhance the support required for abstinence. About 10% to 20% of patients with alcoholic hepatitis are likely to progress to cirrhosis annually, and 10% of the individuals with alcoholic hepatitis have a regression of liver injury with abstinence.

Patients with alcoholic hepatitis subdivide into mild-moderate alcoholic hepatitis or severe alcoholic hepatitis. A DF (discriminant function) is calculated as 4.6 x (patient's prothrombin time - control prothrombin time) + total bilirubin of the patient. MELD score is calculated as a 3.8 x log (serum bilirubin [mg/dL]) + 11.2 x log (INR) + 9.6 x log (serum creatinine [mg/dL]) + 6.4. For patients with MELD greater than 11, the score should be recalculated to account for serum sodium using the following equation: MELD(i) + 1.32 x (137 - Na) - [0.033 x MELD(i) x (137 - Na)], where MELD(i) is the initial MELD score calculated in step one. Patients with severe alcoholic hepatitis with or without hepatic encephalopathy are considered candidates for prednisolone (40 mg/day for 28 days). Prednisolone is preferred to prednisone as it does not require metabolism in the liver for its therapeutic efficacy. For patients unable to take it orally, methylprednisolone, 32 mg intravenously daily, is an option. However, failure to respond to steroids within a week, evident by a Lille score of greater than 0.45, suggests a lack of response to steroids, which should then get discontinued. For patients with a Lille score of less than 0.45 (Lille responders), prednisolone should continue for another three weeks. Glucocorticoids alter the expression of anti-inflammatory genes, thus promoting its anti-inflammatory role. Contraindications to steroid use include any active gastrointestinal (GI) bleeding, severe pancreatitis, uncontrolled diabetes, active infection, or renal failure. Such patients may receive pentoxifylline (400 mg orally, three times a day for 28 days). Hepatorenal syndrome is one of the leading causes of death in patients with alcoholic hepatitis. Patients with acute kidney injury or hepatorenal syndrome respond poorly to corticosteroid therapy. Patients with bacterial infection may be treated with corticosteroids after the infection is under control with antibiotics. Response to prednisolone is graded as complete if the Lille score is less than 0.16, partial if the Lille score is between 0.16 and 0.56), or null if the Lille score is greater than 0.56. A Lille score of more than 0.45 after one week of corticosteroid therapy is associated with 75% mortality at six months.[5]

Many recent trials, including the STOPAH trial and meta-analysis of the use of steroids and pentoxifylline, reveal only short-term (28-day) mortality improvements with steroids, no difference of 6-month, or 1-year mortality with steroid therapy, and no benefit with the use of pentoxifylline. In the STOPAH trial, however, patients with less severe alcoholic hepatitis were included, and most patients recruited had a clinical diagnosis of alcoholic hepatitis. Thus patients with decompensated alcoholic cirrhosis may have received a diagnosis of alcoholic hepatitis, which significantly alters the result of the trial. Also, patients with renal dysfunction met the exclusion criteria for the trial, which might have biased the results against the use of pentoxifylline as the previously reported benefits of pentoxifylline were because of the prevention or regression of hepatorenal syndrome. Attempted therapy with anti-TNF (tumor necrosis factor) agents like infliximab and etanercept demonstrate no proven survival benefits and may worsen outcomes.[6]

Patients with alcoholic hepatitis are prone to infections, especially when on steroids; this is particularly important as it might lead to a poor prognosis, acute renal injury, and multi-organ dysfunction. Patients with alcoholic hepatitis are at risk of alcohol withdrawal. Lorazepam and oxazepam are the preferred benzodiazepines for prophylaxis and treatment of alcohol withdrawal. Documentation of daily caloric intake is necessary for patients with alcoholic hepatitis, and nutritional supplementation (preferably by mouth or nasogastric tube) is an option if oral intake is less than 1200 kcal in a day.

Liver transplantation could be a consideration for patients not responding to steroids and with a MELD of greater than 26. However, varied barriers, including fear of recidivism, organ shortage, and social and ethical considerations, exist. A survey of liver transplant programs conducted in 2015 revealed only 27% of the programs offer a transplant to alcoholic hepatitis patients. Out of the 3290 liver transplants performed, 1.37% were on alcoholic hepatitis patients. The six months, one-year, and 5-year survival was 93%, 93%, and 87%, respectively, the outcomes of which are comparable to patients with similar MELD scores. The recidivism rates are similar (17%) to patients transplanted for alcohol-related cirrhosis.

Differential Diagnosis

Alcoholic hepatitis can be confused with other causes of hepatitis, such as viral, drug-induced, or autoimmune hepatitis. Clinical context and serum tests are fundamental to distinguish these entities.

Non-alcoholic fatty liver disease (NASH) represents the principal histological differential diagnosis since the three stages of liver disease (steatosis, hepatitis, and cirrhosis) are present in the two entities. Many other differential diagnoses include:

- Reye syndrome
- Cryptogenic liver disease
- Fulminant Wilson disease
- Emphysema
- Alpha-1 antitrypsin deficiency
- Ascending cholangitis
- Liver decompensation associated with hepatocellular carcinoma

Prognosis

Prognosis in early-stage liver disease is good since steatosis and steatohepatitis lesions may be reversible after alcohol cessation. However, up to 20% of patients with steatosis may still progress to cirrhosis.[7] Lesions associated with cirrhosis are irreversible, and the prognosis is poor. Screening for hepatocellular carcinoma is imperative at this stage. The presence of ascites, variceal bleeding, severe encephalopathy, and hepatorenal syndrome also indicate poor prognosis.

The Model for End-Stage Liver Disease (MELD) score was adopted by the United Network for Organ Sharing (UNOS) to prioritize patients waiting for a liver transplant due to its ability to predict 90-day mortality.[8] [9] In 2016 the Organ Procurement and Transplantation Network (OPTN) in conjunction with UNOS announced the inclusion of sodium into the MELD score for patients greater than 12 years of age with a MELD score greater than 11. 90-day mortality based on meld score is 1.9% for MELD <10, 6% for 10-19, 19.6% for 20-29, 52.6% for 30-39, and 71.3% for 40.

DF is also a useful predictive tool, and patients with alcoholic hepatitis and a DF greater than 32 have a >50% 30-day mortality.

Complications

Following are some of the significant complications of alcoholic liver disease:

- Variceal Hemorrhage: The presentation is usually with hematemesis or melena. Management options include endoscopic band ligation, sclerotherapy, and placement of transjugular intrahepatic portosystemic shunt placement (TIPS). TIPS increases the risk of hepatic encephalopathy.
- Ascites: This is the most common complication of alcoholic liver disease in which there is an accumulation of fluid in the peritoneal cavity. The patient usually presents with abdominal distension and pedal edema. It can be managed with sodium restriction, diuretics, paracentesis, and TIPS.
- Spontaneous Bacterial Peritonitis (SBP): It is an infection of ascitic fluid with no evidence of any other intraabdominal source (e.g., a perforated viscus) of infection. The diagnosis can be confirmed by positive ascitic fluid bacterial culture and ascitic fluid absolute neutrophil count of over 250/mm^3. The preferred antibiotic is cefotaxime, but ciprofloxacin can be an option if the patient is not able to take cefotaxime.
- Hepatorenal syndrome: This is the development of renal failure owing to advanced alcoholic liver disease after having excluded other causes of renal failure. Characteristically, it demonstrates a progressive rise in creatinine, low sodium excretion rate, oliguria, benign urinary sediment, and absence of proteinuria. Type 1 hepatorenal syndrome is a more severe type with at least a two-fold increase in creatinine in less than two weeks. It correlates with a high mortality rate. Type 2 is slower in onset and has a relatively better prognosis. Treatment for a critically ill patient includes norepinephrine and albumin. For noncritically ill patients treatment includes midodrine (oral alpha agonist), octreotide, and albumin. The ultimate treatment is liver transplantation
- Hepatic hydrothorax: This is the presence of pleural effusion, and excludes other causes of pleural effusion. Treatment is diuretics, thoracentesis, and TIPS.
- Hepatopulmonary syndrome: It characteristically presents with elevated alveolar-arterial oxygen gradient on room air and evidence of intrapulmonary vascular abnormalities. The patient typically presents with shortness of breath and hypoxia. There is no treatment option except liver transplantation.
- Hepatic encephalopathy: It is characterized by reversible neuropsychiatric abnormalities. Treatment includes lactulose, rifaximin, and correction of precipitating factors like infection, GI bleeding, etc.
- Other rare complications are cirrhotic cardiomyopathy, hepatocellular carcinoma, portal gastropathy, portopulmonary hypertension, and portal vein thrombosis.

Deterrence and Patient Education

Outside medical treatment, patient education is the key to treatment for patients with alcoholic liver disease.

Absolute abstinence from alcohol is crucial for preventing disease progression and complications. Sobriety is difficult to achieve without a rehabilitative program run by specialized staff. Psychological care is needed to act on the causes of alcohol addiction, and this may require the help of the patient's family.

It is important to encourage patients with alcoholic liver disease to participate in counseling programs and psychological assistance groups.

The education component also concerns the need to convince the patient to follow a screening program (to detect hepatocellular carcinoma) in case of severe liver damage.

Enhancing Healthcare Team Outcomes

All health professionals must coordinate their actions to improve the management of the patient with severe alcohol addiction, which is responsible for alcoholic liver disease. Psychologists and psychiatrists must be asked by clinicians to assess the psychological state of patients to determine the origin of alcohol intoxication (depression, post-traumatic shock).

A physician psychosocial belief questionnaire and a 3-day residential communication skills course increase interviewing styles. [Level 2]

Coordination starts with excellent communication between nurses, doctors, pharmacists, dietitians, psychologists, and rehab personnel. In the case of hospitalization of the patients, the prescriptions of the doctors can be discussed with the nurses, as regards the indications for the realization in practice, in a cooperative atmosphere. This communication could take place at the patient's bedside during visits or in staff meetings. Collaborative communication is associated with positive patient, nurse, and physician outcomes. Pharmacists can be consulted for medication therapy to guide the patient through the detoxification stage, performing medication therapy management, medication reconciliation, and verifying dosing, and reporting any concerns to the healthcare team. Nursing is on the front lines to assess the effectiveness of care during inpatient therapy. Psychological professionals must make this evaluation during any rehabilitative treatment, whether in or outpatient. A nutritionist or dietician should have input to bring about optimal caloric and nutrient intake. All members of the team need to evaluate abstinence compliance and report their findings to the rest of the interprofessional team. All the staff must maintain medical confidentiality.

The literature proposes several interventions that have been designed to improve the care delivered to the patient in terms of rapid recovery, the stability of health patient safety (compensated cirrhosis), and the performance of the patient.

Introducing video conferencing during the postoperative period ("telerounds") is associated with increased patient satisfaction and may decrease the routine during hospitalization of patients, especially if it is prolonged.[10] [Level 2]

It is crucial to assess the degree of satisfaction of physicians (gastroenterologists and surgeons) and all paramedical staff (nurses, secretaries) in their work environment to propose innovative activities that meet their expectations and target problems that are solvable with short-term interventions.[11] [Level 3]

Repeated collection of staff and patient satisfaction data through a questionnaire could contribute to the implementation of work improvement measures in the short and medium term. [Level 5] Video projections for staff and patients showing proper practice techniques (handwashing, ascites punctures, etc.) could reduce stress and reduce some of the problems inherent in patient management.[12] [Level 5]

Alcoholic liver disease requires an interprofessional team approach, including physicians, specialists, specialty-trained nurses, psychological/rehab personnel, and pharmacists, all collaborating across disciplines to achieve optimal patient results. [Level 5] Addressing the underlying misuse of alcohol is the primary objective.

Review Questions

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Disclosure: Roshan Patel declares no relevant financial relationships with ineligible companies.

Disclosure: Matthew Mueller declares no relevant financial relationships with ineligible companies.

Bookshelf ID: NBK546632 PMID: 31536239