**Scholars Journal of Applied Medical Sciences**
Abbreviated Key Title: Sch J App Med Sci
ISSN 2347-954X (Print) | ISSN 2320-6691 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

**Medical Sciences**

# Alcoholic Liver Disease

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**DOI:** https://doi.org/10.36347/sjams.2025.v13i07.007 | **Received:** 27.05.2025 | **Accepted:** 02.07.2025 | **Published:** 07.07.2025

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**Abstract** **Review Article**

Alcoholic liver disease is a fatal condition and leading cause of morbidity and mortality caused by excessive alcohol consumption with an average weekly intake of 7-14 units for men and 8.75-26.25 units for women. In UK, it is advised not to consume more than 6 pints of beer or 6 medium wine glasses. ALD presents with liver inflammation and decompensation or fibrosis progression along with comorbidities. In advanced stages, patients develop cirrhosis and related complications leading to poor prognosis that can reduce life expectancy and irreversible liver damage. ALD is treatable at early stages with administration of corticosteroids. Recent studies have reported an association between ALD and tumor necrosis factor-alpha, hence latest research focuses on antitumor antibodies. There is a need for effective management and timely identification of this condition especially in males and individuals aged 40-50 years. This article provides a comprehensive insight regarding risk factors, clinical and laboratory features, and treatment of alcoholic liver disease.

**Keywords:** Alcoholic Liver Disease (ALD), Alcohol consumption, Liver damage, Women's susceptibility, Tumor necrosis factor-alpha (TNF-alpha).



## Risk Factors of Alcoholic Liver Disease

The major cause of ALD is attributed to alcohol consumption. Commonly consumed beverages include beer (42.5 units) and wine (14.2 units) contains 2.1 units of pure alcohol. Therefore, 7.5-10 units intake of alcohol daily for a decade in men and 2.5-5 units intake in women increases the chances of developing ALD significantly (Kang *et al*., 2020). Women are at higher risk of developing ALD with advanced liver injury even after less alcohol intake as compared to men (Bizzaro *et al*., 2023). This is due to the fact that alcohol metabolizes differently in the gastrointestinal tract and liver in women which makes them highly susceptible to the condition. In addition, the overexpression of tumor necrosis factor-alpha in women due to difference in alcohol metabolism triggers a pro-inflammatory response and increases reactive oxygen species, gut permeability and immune activation, contributing to liver toxicity (Diab *et al*., 2020). Alcohol consumption affects women more due to hormonal factors and body composition differences as compared to men. Estrogen enhances the intestinal permeability, allowing endotoxins to get absorbed in the gut leading to liver inflammation. It also impacts the ethanol metabolism by suppressing the expression of alcohol dehydrogenase and promoting expression of Cytochrome P450 2E1. Additional progesterone may prolong alcohol absorption, increasing its concentration in blood, thus highly contributing to liver damage even at lower levels of alcohol consumption.

With regards to body composition, women have higher fat index and lower water percentage in them in comparison to men. Hence, as Marshall *et al*., showed that the mean peak ethanol levels in women were $88 \pm 3$ mg per 100 ml which was significantly high than $75 \pm 4$ mg per 100 ml in men after consuming same alcohol amount (Marshall *et al*., 1983).

Hepatitis C is also a major comorbidity that is prone to progress ALD to pathological conditions like liver cancer and cirrhosis. Chronic drinkers (28-35 units per week) with HCV infection have a significantly quicker progression from ALD to cirrhosis than light alcoholics (≤14 units per week). HCV speeds up the progression by adding to oxidative stress and liver inflammation, establishing a synergy with alcohol-induced damage. It triggers continuous stimulation of immune system which leads to ongoing hepatocyte damage and release of TNF- α and interleukins like IL-6 and IL-1 β. At the same time, metabolism through CYP2E1 intensifies oxidative stress and lipid peroxidation. These simultaneous mechanisms accelerate fibrosis, steatosis and hepatocellular apoptosis

which increases the susceptibility of HCC and cirrhosis. Patients with HCV have a high risk of developing cirrhosis in case of daily alcohol intake of more than 6.25 units (Wieland and Everson, 2018). The coexistence of HCV and alcohol abuse leads to poor prognosis and survival, like development of cirrhosis within 10-15 years, risk of liver failure and hepatocellular carcinoma due to which most patients are left with a compensated liver at an early age.

Patients with ALD can develop a high liver iron burden as a sequala to the comorbidity of alcohol disorder and HCV and in rare cases lead to porphyria cutanea tarda (Usta Atmaca and Akbas, 2019). Liver cancer is also considerably common in HCV patients with cirrhosis and ALD with the risk being 5-10 folds higher than patients with HCV alone. Obesity and fatty liver caused by high carbohydrate intake leading to lipid transport and synthesis dysfunction can also cause alcohol injury. Metabolic syndrome exacerbates the progression of ALD and when it occurs in combination with chronic alcohol consumption it amplifies hepatotoxicity through increased oxidative stress, inflammation and insulin resistance leading to synergistic fibrosis progression. According to the 'multiple hit' model, liver disease is a result of concurrent hits by metabolic syndrome (inflammation) and alcohol consumption and then factors like gut dysbiosis, genetics and lifestyle hasten fibrosis progression (Leal-Lassalle *et al*., 2025). Among genetic factors, the presence of polymorphic genes including cytochrome P4502E1, alcohol hydrogenase, BNDF, etc. shows a linkage between ALD and genetics. Expression of the RS738409 gene has been reported to cause severe hepatic injury that progresses to cirrhosis. A flowchart of risk factors of ALD has been shown in Figure 1.

**Clinical Case Scenario**

A 60-year-old male presents with yellowish skin discoloration, fatigue, distended abdomen and constipation. He has decreased appetite, decreased bowel and bladder movements and swelled in lower legs. The patient has a history of asthma and depression. He is a light smoker for 12 years and smokes 1-3 cigarettes daily. He is also an alcoholic for eight years consuming 4-5 units of alcohol every day. He seeks diagnosis of his condition and wants to explore treatment option. You order laboratory and radiological confirmation. LFTs reveal elevated total bilirubin and reduced albumin levels coagulation profile shows prolonged INR. Patient was mildly anemic, had elevated leukocytes and thrombocytopenia. A MELD score of 18 and GAHS score of 7 showed mild to high risk. Lastly abdominal ultrasound confirmed cirrhosis, ascites and moderately enlarged liver. A 3-day treatment for alcoholic liver disease including IV hydration and nutrition support with high-protein diet and multi-vitamins was started. Drug prescriptions of 40 mg Prednisolone adjuvant with N-acetylcysteine, lactulose and 100 mg Spironolactone were given. At day 3, patient's symptoms approve significantly with reduced jaundice, abdominal distention and swelling and improved appetite and bowel function indicating that disease progression is halted. Patient is discharged and advised about lifestyle changes including referral to alcohol cessation programs and intake of protein diet and low sodium. In addition, tapered dose of Prednisolone and continued administration of spironolactone along with multi-vitamins were also prescribed.

**Clinical Features**

ALD clinically manifests in various forms. Alcohol-induced fatty liver presents very subtly and is mostly diagnosed during routine or incidental screening or examination for an unrelated condition. Hepatomegaly is often the only characteristic that hints at suspected occurrence but its absence does not exclude the possibility of ALD as some patients with ALD have significant liver damage while maintaining the normal liver size. Patients with fatty liver may complaint of right upper quadrant discomfort and nausea present with jaundice and tender hepatomegaly. The source of the fatty liver being alcoholic can be determined by assessing the intake quantity, pattern, and drinking habits. Other causes of abnormal liver function tests like drug induced liver injury, hepatitis and non-alcoholic fatty liver disease must be considered.

Alcohol hepatitis is characterized as a systematic manifestation triggered by cytokine production. While some patients remain asymptomatic, others present with jaundice, fever, acute abdominal pain, and spider nevi. ALD, if persistent and untreated, can advance to cirrhosis which presents as weight loss, weakness, and fatigue which can potentially worsen and cause GI bleed, confusion, jaundice, and abdominal swelling (Tsochatzis, 2014).

**Laboratory Features**

ALD can currently be diagnosed by physical assessment, evaluation of history of alcoholic intake, and lab analysis testing ALT, AST, and gamma-glutamyl transpeptidase of the patient. The laboratory results are inconclusive and non-specific about the abnormalities; typically showing AST levels 2–6 times the upper limit of normal, ALT less than 2 times the ULN, and an AST:ALT ratio of ≥2:1, with variable GGT elevation. In addition, increased levels of cholesterol, triglycerides, and bilirubin are reported. Hyperbilirubinemia is frequent in ALD patients and is mostly paired with slight elevations of alkaline phosphatase.

A dysfunction in hepatocyte production indicates that the ALD has advanced to a fatal degree (Gissen and Arias, 2015). In addition, decreased levels of albumin and coagulation disorder are common signs of severe hepatic injury. An elevated neutrophil number of more than 5500mL can indicate a neutrophil

infiltration which is a sign of alcohol hepatitis. Ultrasonography should be performed to examine liver size and fatty liver infiltration. The presence of ascites, hepatofugal flow, and abdominal collaterals on ultrasound point towards the injured liver with little to no chance of complete recovery (Subramaniyan and Middha, 2016).

However, the diagnostic accuracy of these above variables is not satisfactory to predict the severity of liver inflammation. The latest research highlights the novelty of clinical scores and biomarkers that act as better diagnostic and prognostic predictors of ALD that help to seek adequate treatment and therapy options as shown in Table 1. Hepatotoxicity can be measured by evaluating aminotransferases, cytokines, DNA fragmentation, and histopathological examination. Some validated and tested scores include the Model of End-Stage Liver Disease, Glasgow Alcoholic Hepatitis Score, and Discriminate Function Index (Gala and Vatsalya, 2020). Recent studies have also developed new biomarkers that measure alcohol consumption and liver injury caused by it non-invasively.

**Treatment**

Ongoing research for the treatment of ALD has revealed many drugs to be considered as a therapeutic target, however, no drug has yet been declared safe for use since it has been reported that they worsen liver toxicity. EASL guidelines about the treatment algorithm for alcohol hepatitis are shown in Figure 2.

Baclofen has been tested and approved as a safe drug for the treatment of ALD by the AASLD and EASL, however, drugs including disulfiram and naltrexone have been reported to enhance liver toxicity. Case reports including Ramer et al provide strong evidence for disulfiram induced fulminant hepatitis and liver injury (Ramer *et al*., 2020). Some studies suggest the safety of moderate doses of naltrexone (Ayyala *et al*., 2022, Springer, 2014) but this is not backed by results from systematic reviews yet. Hence, no treatment option has been deemed appropriate for curing alcoholic liver disease. However, for the management of ALD, several drugs are available for targeted action. Recent studies have highlighted the antiviral properties of oral pentoxifylline that help to reduce the severity of steatohepatitis in ALD subjects, however, it has not been recognized by AASLD or EASL as first-line treatment. In addition, corticosteroids adjuvant with N-Acetyl Cysteine has also shown good recovery outcomes and has been supported by AASLD for severe alcoholic hepatitis. Recent research focuses on experimental treatments through ASK1 inhibitors like Selonsertib and PPAR agonists like Elafibranor to prevent progression of fibrosis to ALD.

The current targeted treatments for ALD are as follows: Mycophenolate mofetil, an immunosuppressant, acts as a hepatic anti-inflammatory when administered to ALD patients, Emricasan inhibits apoptosis, Lactobacillus rhamnosus inhibits bacterial overgrowth to prevent inflammation, Obeticholic acid improves cholestasis, Rifaximin, Ciprofloxacin, and Amoxicillin clavulanate act as antibiotics and prevents infection. A detailed information of treatments is shown in Table 2.

**Supplementation of Nutritional Substances**

Physicians suggest that the best way to prevent and progression of ALD is abstinence to inhibit hepatic toxicity. However, since many patients of ALD are heavy drinkers for a long duration, this is often not possible without rehabilitation. Another adverse effect of excessive alcohol intake is malnutrition which significantly contributes to the advancement of liver disease.

Majority of the heavy drinkers are malnourished which can be caused by inadequate amounts of essential nutrients such as vitamins, proteins, fats, and carbohydrates in the body. Alcohol metabolism also plays an important role in this regard as it inhibits digestion and absorption of essential nutrients. Studies also found that more than 50% of cirrhotic patients were severely malnourished (Traversy and Chaput, 2015).

The management of ALD is done depending on the degree of liver disease. In order to prepare the patient to withstand the treatment, nutritional supplements of vitamins, zinc, magnesium, and other essentials as listed in Table 3 are administered. In the case of advanced cirrhosis, patients are recommended for orthotopic liver transplant as drugs are less effective in such patients. Nutritional supplements protect the liver from toxicity, provide protein balance, and inhibit alcohol-induced encephalopathy, especially by amino acids like Isolucine, asvaline, and leucine (Konstantis *et al*., 2022).

A daily intake of 1.2-1.5 g proteins and 2000-2500 kcal calories is recommended daily under recent guidelines. ALD patients frequently present with values lower than the suggested range with common deficiency of zinc, magnesium, selenium, and iron. Whereas, manganese and copper are often elevated. To manage these deficiencies, nutritional supplements are recommended to prevent the development of cirrhosis (Table 3). Almost all the patients with severe alcoholic hepatitis are malnourished, hence nutritional support is necessary for recovery.

**Liver Transplant**

Advanced alcoholic liver disease is commonly treated with an orthotopic liver transplant provided a strict eligibility criterion. Patients must demonstrate at least 6 months of abstinence to considered a candidate to allow for recovery alcohol induced damage to the liver and to reduce chances of recidivism after transplant.

Patient selection also involves evaluating the psychosocial stability and screening for comorbidities such as cardiovascular diseases or infections to reduce risk of relapse and graft failure. A high 1-year survival rates of 85-90% and 5-year survival rates of 70-80% have been reported after the transplantation (Bergsmark *et al.*, 2023), which is similar to outcomes of patients with hepatitis C or autoimmune diseases (Musto *et al.*, 2024). A pattern of smoking has been observed in transplanted patients as the survival analysis revealed an association between survival outcomes and cardiovascular disease and de nova malignancies. Hence, OLT patients must be assessed and monitored for smoking-induced mortality before and after the transplant. Routine checkups, appropriate nutritional habits, and lifelong follow-ups are suggested for OLT patients to prevent adverse effects. Recently, the mandated 6-month rule has been excluded especially for patients with severe alcoholic hepatitis and early liver transplant has shown promising results with a 3-year survival rates of over 75% (Mathurin *et al.*, 2011, Musto *et al.*, 2024).

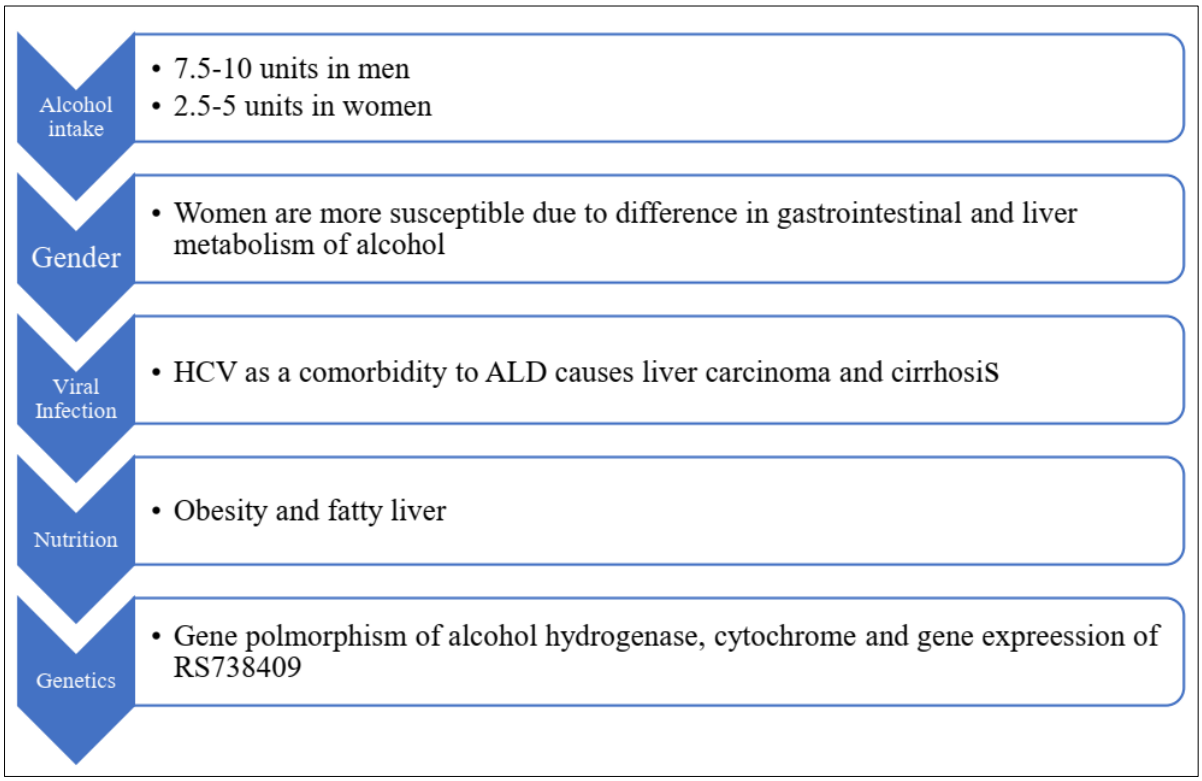


**Figure 1: Risk factors of Alcoholic Liver Disease Including Alcohol Intake, Gender, Viral Infection, Nutrition and Genetics**

**Table 1: Recently developed biomarkers for ALD**

| **Biomarkers** | **Uses** |
|---|---|
| Cytokeratin 18 | Diagnostic, prognostic and staging predictor of alcoholic hepatitis |
| Augmenter of Liver Regeneration | Not tested in human yet but could be useful for estimating severity of ALD |
| CD 163 | Can act as predictor of outcome and severity of alcoholic hepatitis |
| ST2 receptor | Is a predictor of hepatic inflammation and ALD severity and can be a possible therapeutic option |
| TNF-related Apoptosis inducing ligand | Suited to predict degree of alcohol hepatitis and can be a potential treatment target |
| Immunoglobulins | Predictor of severity of alcohol hepatitis |
| MicroRNAs | Risk factor for prognosis and grading of alcoholic hepatitis |
| Stearoyl-CoA desaturase 1 | Can be therapeutic option for treating early alcoholic liver disease |
| Magnesium | Possible predictor of ALD onset and grading |
| Acrolein | Possible predictor of degree of alcohol hepatitis |
| Resolvins | Potential predictor of liver inflammation and severity of alcoholic hepatitis |

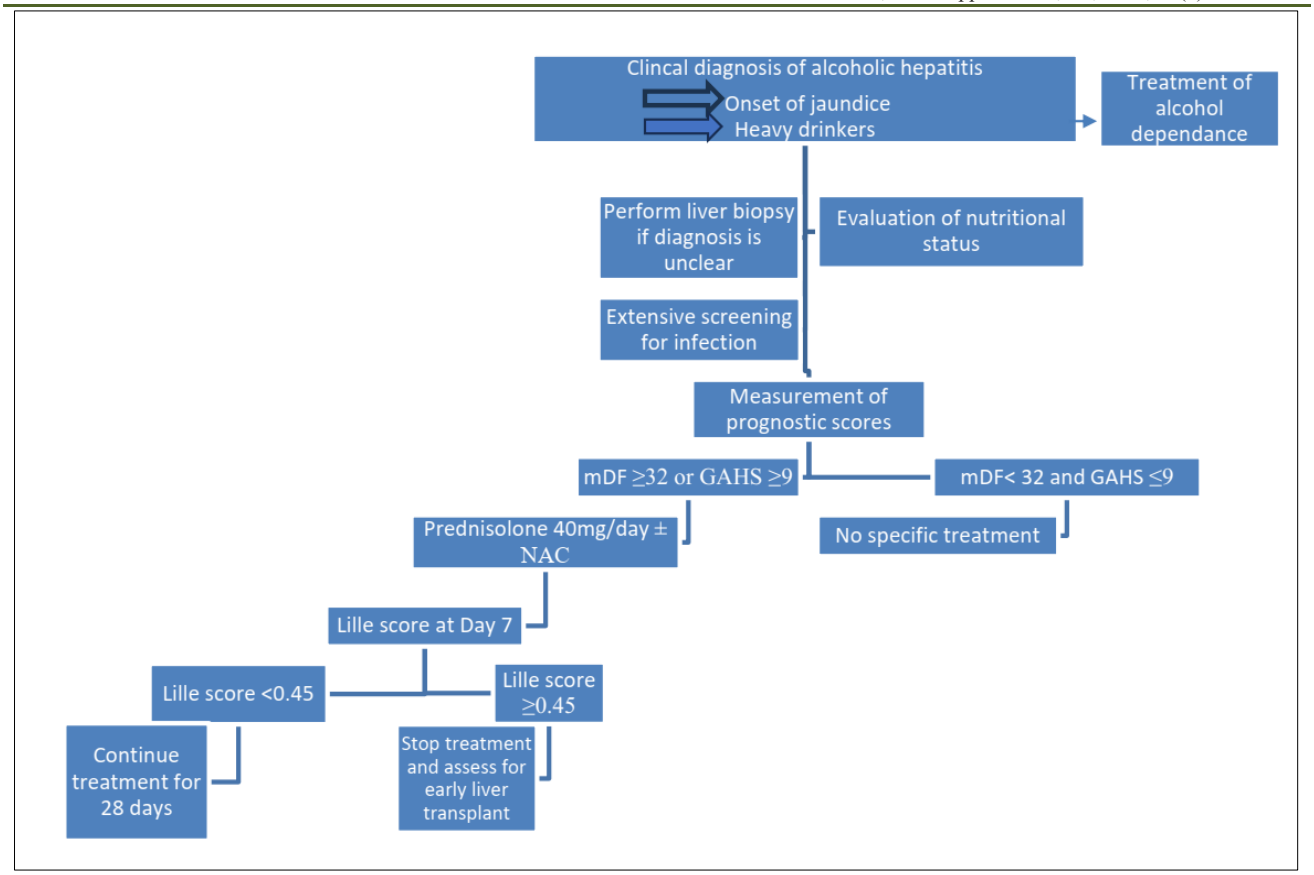


**Figure 2: Treatment Algorithm for Alcohol Hepatitis by EASL Guidelines**

**Table 2: Currently Targeted Drugs for Alcoholic Liver Disease, Their Drug Action and Applicability**

| Drugs | Pharmacodynamics | Clinical Application | Evidence for Effectiveness | Primary or Secondary Care |
|---|---|---|---|---|
| Mycophenolate mofetil | Suppresses immune system and interleukin-1 inhibitor | Hepatic anti-inflammatory | Experimental studies | Secondary |
| Rifaximin | Broad spectrum antibiotic | Prevents bacterial infections and imbalance | Strong evidence for hepatic encephalopathy | Secondary |
| Emricasan | Inhibits caspase enzymes | Inhibits apoptosis | Early-stage trials | Secondary |
| Lactobacillus rhamnosus | Supports gut microbiome | Inhibits bacterial overgrowth to prevent inflammation | Limited evidence for microbiome modulation | Primary |
| Zinc | Essential trace element | Gut anti-inflammatory and immune-stimulator | Limited evidence for deficiency correction | Primary and secondary |
| Obeticholic acid | Acts as bile acid analogue | Helps in bile flow improvement | Satisfactory evidence of use for liver disease but not a first-line treatment for ALD | Secondary |
| Amoxicillin clavulanate | Combination antibiotic | Resolves bacterial infections | Strong evidence for bacterial infections | Primary and secondary |
| Ciprofloxacin | Fluoroquinolone antibiotic | Resolution of bacterial infections | Strong evidence for spontaneous bacterial peritonitis | Secondary |

**Table 3: Nutritional supplements for common ALD symptoms and deficiencies**

| Symptoms | Deficiency | Daily recommended intake |
|---|---|---|
| Inflammation, diarrhea, immune insufficiency | Zinc | 8-11 mg |
| Impaired glucose tolerance and spasms | Magnesium | 310-420 mg |
| Low bone density and soft bones | Vitamin D | 600-800 International Unit |
| Antioxidative stress | Vitamin E | 15 mg |
| Anemia and cancer risk | Folate | 400 micrograms |
| Neuropsychiatric dysfunction and pellagra | Niacin | 14-16 mg |
| Myopathy and cardiomyopathy | Selenium | 55 micrograms |
| Cardiomyopathy and Wernicke-Korsakoff syndrome | Thiamine | 1.1-1.2 mg |
| Immune deficiency and night blindness | Vitamin A | 700-900 microgram |
| Hemorrhage and coagulopathy | Vitamin K | 90-120 microgram |
| Neuropathy, irritability and seizures | Vitamin B6 | 1.3-20 mg |
| Neurological dysfunction and macrocytic anemia | Vitamin B12 | 2.4 micrograms |

**Key Points**

- Excessive alcohol consumption leads to alcoholic liver disease in three stages; fatty liver disease, alcoholic hepatitis, and cirrhosis.
- Quantity of alcohol intake, gender, hepatitis C, nutritional habits, and genetics are common risk factors for ALD.
- Recent studies have developed new biomarkers for diagnostic, prognostic, and therapeutic accuracy for ALD.
- There is no cure for ALD yet but several drugs have been effective to manage and treat it.
- Most heavy drinkers are malnourished, hence nutritional supplements of zinc, magnesium, selenium, and vitamins can help manage ALD.

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