CODEN [USA]: IAJPBB ISSN: 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES

SJIF Impact Factor: 7.187

https://zenodo.org/records/10849891

https://www.iajps.com/volumes/volume11-march-2024/03-issue-03-march-24/

Available online at: http://www.iajps.com *Review Article*

A REVIEW ON ALCOHOLIC LIVER DISEASE

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Abstract:

Alcoholic liver disease is damage to the liver. The risk of developing the disease is related to the quantity and duration of alcohol consumption. The prevalence is highest in European countries. The most complications occur are Jaundice, Hepatic encephalopathy, Cirrhosis, Steatosis etc. This can be treated by liver biopsy, endoscopy. Alcohol intake increases NADHNAD+ in hepatocytes. The alcohol metabolism increases the production of NADH by reducing NAD in the body. Ongoing liver injury leads to irreversible liver damage.

Key words: *Alcoholic steatosis or Alcoholic fatty liver, Alcoholic hepatitis, Alcoholic cirrhosis*

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Please cite this article in press Reeba Roy et al., ***A Review On Alcoholic Liver Disease ,****, Indo Am. J. P. Sci, 2024; 11 (03).*

INTRODUCTION:

Alcoholic liver disease (ALD) is a complex multifactorial disease caused by chronic alcohol consumption and is a major cause of morbidity and mortality worldwide. According to the 2014 WHO report on global status report on alcohol and health, alcohol consumption is responsible for 3.3 million deaths and 139 million disability-adjusted life years (DALYs) in the world in 2012.

The chronic form of ALD progresses in stages of steatosis, steatohepatitis, fibrosis, and cirrhosis, which is the end stage of liver disease; heavy drinkers also have increased incidence of hepatocellular carcinoma. Chronic ALD typically develops over decades of alcohol abuse. Acute alcoholic steatohepatitis (ASH), a severe acute inflammatory disease, can occur at any stage of ALD and is associated with a very high short-term mortality rate. This high short-term mortality is due to liver failure, portal hypertension, and bacterial infections. Interestingly, many patients with ASH exhibit stage IV fibrosis, suggesting an increased susceptibility to ASH in patients with more advanced fibrosis . While it has long been appreciated that early stages of ALD, including steatosis and steatohepatitis, are reversible, recent evidence indicates that early stages of fibrosis may also be reversible, suggesting that specific therapeutic interventions targeting fibrosis may improve the long-term outcome in patients with ALD.

The spectrum of ethology is broad for chronic liver disease and include:

- Toxins
- Alcohol abuse for a prolonged time
- Infection
- Autoimmune diseases
- Genetic and metabolic disorders.

Alcoholic liver disease is damage to the liver and its function due to alcohol abuse. The liver serves a wide variety of body functions, including detoxifying blood and producing bile that aids in digestion. Liver disease is one of the five most common causes of premature death in the UK and is the only major cause of death that has a year by year increasing incidence. More than 90% of liver disease is preventable.

ALCOHOLIC LIVER DISEASE

The risk of developing alcohol liver disease is related to the quantity and duration of alcohol consumption. Alcoholic fatty liver disease is the most common alcohol – induced hepatic abnormality, occurring in 90-100% of chronic alcoholics. Complications from cirrhosis include encephalopathy, portal hypertension with bleeding at the oesophageal varies, portal vein thrombosis and hepato-renal syndrome.

EPIDEMIOLOGY

Alcohol is the most frequently misused drug throughout the entire world and in the United States of America. Alcohol is one of the main causes of end stage liver disease worldwide, and alcoholic liver disease is the second most common reason for liver transplantation in the United States.[1] Beginning in the 1970s, there was a gradual decline in alcoholic cirrhosis-related mortality in many countries. In the past few years, alcoholic liver disease mortality rates in several countries have stabilized or started to increase. There are significant ethic and gender differences in alcoholic cirrhosis-related mortality rates. Furthermore, alcohol use increases the risk for liver disease in those infected with hepatitis C.

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 60g/day, and cirrhosis occurs in 30% of individuals with long standing consumption of more than 40g/day.[1]

At risk drinking definitions are below:

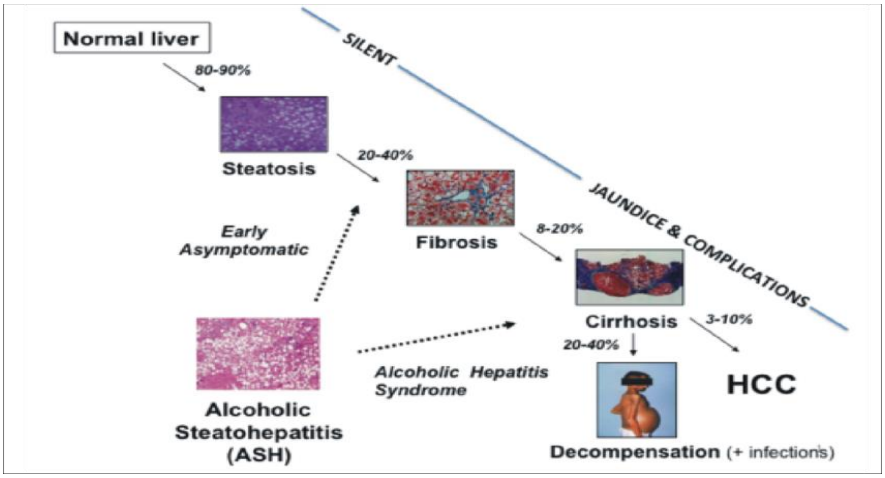
- Men over 14 drinks per week or more than four drinks per occasion
- Women and those over 65 years, over seven drinks per week or greater than three drinks per occasion
- On average about 27,000 deaths per year all around the world.
- Additional 10,000 deaths due to liver cancer secondary to cirrhosis.

This history is essential to differentiate non-alcoholic fatty liver disease (NAFLD) alcoholic fatty liver disease (AFLD).

DISEASE SPECTRUM AND PATHOGENESIS

ALD comprises different stages of liver disease because of susceptibility factors and duration of alcohol abuse. These stages include steatosis, alcoholic steatohepatitis (ASH), progressive fibrosis, cirrhosis, decompensated cirrhosis, and superimposed hepatocellular carcinoma (HCC) (Figure1. 2). Patients with underlying cirrhosis and ongoing alcohol abuse are predisposed to developing AH. With a mortality

rate of 30-50% at 3 months, alcoholic hepatitis represents one of the deadliest diseases in clinical hepatology.



Natural progressive along the spectrum of ALD, from steatosis to the inflammatory state of steatohepatitis, to progressive fibrosis and cirrhosis and finally, to decompensated cirrhosis and hepatocellular carcinoma (HCC). Exacerbations of alcoholic hepatitis (ah) occur at many of the later stages of disease. Predisposing risk factors to accelerated progression are listed.

Steatosis is defined histologically as the deposition of fat in hepatocytes. Alcohol intake increases NADHNAD+ in hepatocytes, thereby disrupting fatty acid oxidation.[2] Increased fatty acid and triglyceride synthesis, hepatic influx of free fatty acids from adipose tissue and chylomicrons from the intestinal mucosa, results in increased hepatic lipogenesis, decreased lipolysis, and mitochondrial and microtubule damage. Up to 90% of patients with heavy alcohol intakes have some degree of steatosis, which is usually asymptomatic and rapidly reversible with abstinence.

Continued heavy alcohol consumption leads to ASH, characterized by polymorphonuclear (PMN) cell infiltration and hepatocellular damage. Acetaldehyde, a byproduct of alcohol metabolism, is implicated for the hepatocellular injury. It binds proteins and DNA, forming adducts that promote glutathione depletion, lipid peroxidation and mitochondrial damage.[2]

Sustained alcohol misuse causes progression to liver fibrosis and cirrhosis, which leads to a high risk of complications (such as ascites, variceal bleeding, hepatic encephalopathy, renal failure, and bacterial infections). Acetaldehyde promotes fibrogenesis directly by increasing the expression of collagen in hepatic stellate cells (HSC). HSCs can also be activated by neutrophils, damaged hepatocytes, and activated Kupffer cells through various pro-fibro genic mediators including transforming growth factor β, platelet derived growth factor, IL-8, angiotensin II, and leptin. The activation and biological actions of these mediators are largely due to reactive oxygen species (ROS). Alcohol abuse contributes to dysbiosis and inflammation of the intestinal tract with resulting translocation of microbial products such as lipopolysaccharide (LPS) to the liver. LPS targets toll-like receptor-4 (TLR-4) signalling in HSCs and sinusoidal endothelial cells, resulting in HSC activation and promotion of fibrogenesis through regulation of angiogenesis.

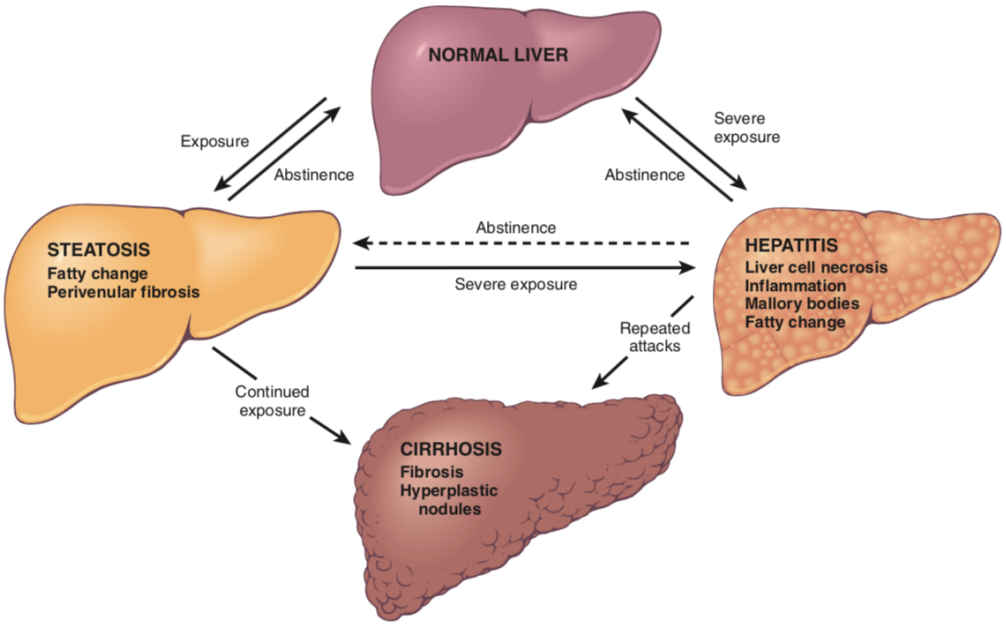
TYPES

Alcoholic Fatty Liver or Steatosis - At this stage, fat accumulates in the liver parenchyma.

Alcoholic Hepatitis - Inflammation of liver cells take 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.

Alcoholic Cirrhosis – Liver damage at this stage is irreversible and leads to complications of cirrhosis and portal hypertension.

Pathological changes



ETIOLOGY

- Alcoholic liver disease is caused by heavy use alcohol. The liver's job is to break down alcohol. If you drink more than it can process, it can become badly damaged. Fatty liver can happen in anyone who drinks a lot.[3] Alcoholic hepatitis and alcoholic cirrhosis are linked to the long-term alcohol abuse seen in alcoholics. The liver tolerates mild alcohol consumption, but as the consumption of alcohol increases, it leads to disorders of the metabolic function of the liver.
- The initial stage leads to fatty liver or steatosis. If the alcohol consumption 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.[3]
- The spectrum of aetiologies is broad for chronic liver disease, which includes toxins, alcohol abuse for a prolonged time, infection, autoimmune diseases, genetic and metabolic disorders. [3]

CLINICAL MANIFESTATIONS

SIGNS AND SYMPTOMS

The effects of alcohol on the liver how much and how long one has been drinking alcohol. These are the most common signs and symptoms:

Alcoholic steatosis

- Often causes no symptoms.
- Build-up of fat inside the liver cells enlarges the liver, causing upper abdominal discomfort on the right side.
- Tiredness and weakness
- Weight loss

Alcoholic hepatitis

- Pain over the liver
- Fever
- Weakness
- Nausea and vomiting
- Appetite loss
- Yellowing of the skin and eyes (jaundice)

Alcoholic cirrhosis, all the symptoms of alcoholic hepatitis and:

- Portal hypertension (increased resistance to blood flow through the liver)
- Enlarged spleen.
- Poor nutrition

- Bleeding in the intestines
- Ascites (fluid build-up in the belly)
- Kidney failure
- Confusion
- Liver cancer

DIAGNOSIS

Alcohol-related liver disease (ARLD) is often first suspected when tests for other medical conditions show a damaged liver. This is because the condition causes few obvious symptoms in the early stages. It's important to be totally honest about how often alcohol had been consumed to avoid further unnecessary testing.

Blood tests

- Blood tests used to assess the liver are known as liver function tests.
- But liver function tests can be normal at many stages of liver disease.
- Blood tests can also detect if you have low levels of certain substances, such as protein called serum albumin, which is made by the liver.
- A low level of serum albumin suggests your liver is not functioning properly.
- A blood test may also look for signs of abnormal blood clotting, which can indicate significant liver damage.

Further testing

If symptoms or liver function test suggest an advanced form

ARLD (either alcoholic hepatitis or cirrhosis), you may need further tests.

Imaging tests

Scans may be needed to produce detailed images of your liver. This may include:

- An ultrasound scans.
- A CT scans.
- An MRI scans.

Some scans may also measure the stiffness of the liver, which is a good indication of whether your liver is scarred.

Liver biopsy

During a liver biopsy, a fine needle is inserted into your body (usually between your ribs). A small sample of liver cells is taken and sent to a laboratory to be examined under a microscope. The biopsy is usually carried out under local anaesthetic, either as a day case or with an overnight stay in hospital. The liver tissue will be examined to determine the degree of scarring in the liver and the cause of the damage.

Endoscopy

An endoscope is a long, thin, flexible tube with a light and a video camera at one end. During an endoscopy, the instrument is passed down your oesophagus (the long tube that carries food from the throat to the stomach) and into your stomach. Pictures of your oesophagus and stomach are transmitted to an external screen. The doctor will be looking for swollen veins (varices) which are a sign of cirrhosis.

Liver function tests

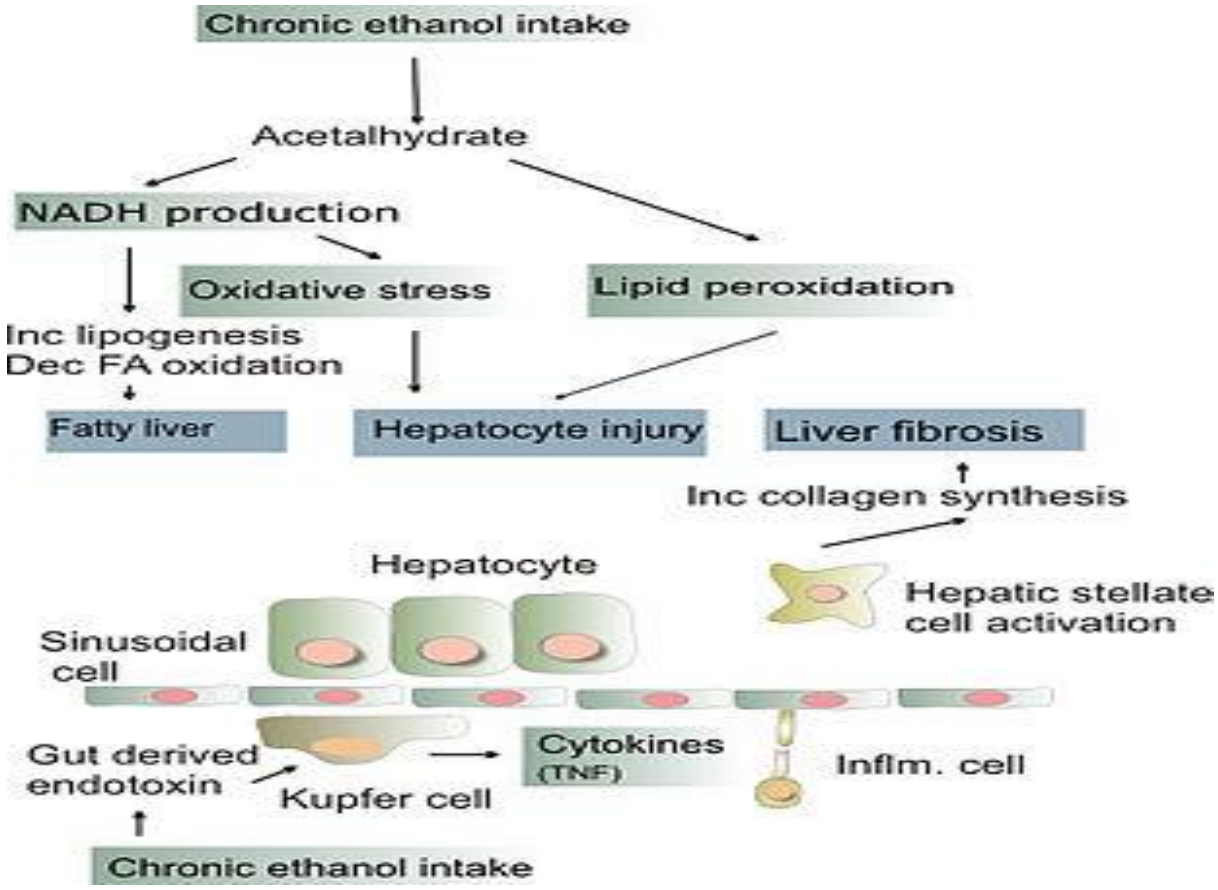
It checks the levels of certain enzymes and proteins in the blood. Levels that are higher or lower than usual can mean liver problems. The test may be performed to assess liver function is alanine transaminase (ALT) test.

PATHOPHYSIOLOGY

Alcohol metabolism by the liver is primarily through two enzymes:

a) Alcohol dehydrogenase
b) 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. [6]



Pathophysiology of Alcoholic Liver Disease

TREATMENT

PHARMACOLOGICAL TREATMENT

a) Alcohol abstinence

It decreases the progression during cirrhosis and improve the survival rate. Treatment for alcohol abstinence is:

- Disulfiram
- Naltrexone

b) Corticosteroid

It is highly valued in patients with severe hepatitis and increase the survival rate.

- Prednisolone

c) Anti cytokine therapy

Dysregulated cytokine like TNF plays a major role in ALD.

- Pentoxifylline

NON-PHARMACOLOGICAL TREATMENT

Stopping drinking alcohol

Treatment for ARLD involves stopping drinking alcohol. This is known as abstinence, which can be vital depending on what stage the condition is at. If a patient has fatty liver disease, the damage may be reversed if patient abstain from alcohol for a period (this could be months or years). If more serious of ARLD (alcoholic hepatitis or cirrhosis) is present, lifelong abstinence is recommended. This is because stopping drinking is the only way to prevent liver damage getting worse and potentially stop dying of liver disease. Stopping drinking is not easy, especially as an estimated 70% of people with ARLD have an alcohol dependency problem.[9]

Withdrawal symptoms

If patient is abstaining from alcohol, you may suffer withdrawal symptoms. These will be at their worst for the first 48 hours but should start to improve as body adjusts to being without alcohol. This usually takes 3 to 7 days from the time of last drink. Many people initially experience disturbed sleep when abstaining from alcohol, but in most cases their sleep pattern

returns to normal within a month. In some cases, they may be advised to reduce your alcohol intake in a gradual and planned way to help avoid withdrawal problems.

Preventing relapses

Once drinking had stopped, the patient may need further treatment to help ensure the patient do not start drinking again. The first treatment usually offered is psychological therapy. This involves seeing a therapist to talk about your thoughts and feelings, and how these affect your behaviour and well-being. If psychological therapy alone is not effective, you may also need medicine to help you abstain from alcohol, such as:

- Acamprosate
- Disulfiram
- Naltrexone
- Prednisolone
- Pentoxifylline

Diet and nutrition

Malnutrition is common in people with ARLD, so it's important to eat a balanced diet to make sure you get all the nutrients you need. Avoiding salty foods and not adding salt to foods you eat can reduce your risk of developing swelling in your legs, feet and tummy caused by a build-up of fluid. The damage to the liver also means, it's unable to store glycogen, a carbohydrate that provides short term energy. When this happens, the body uses its own muscle tissue to provide energy between meals, which leads to muscle wasting and weakness. This means you may need extra energy and protein in your diet.[9]

Liver transplants

In the most serious cases of ARLD, the liver loses its ability to function, leading to liver failure. A liver transplant is currently the only way to cure irreversible liver failure.

A liver transplant may be considered if:

- You develop progressive liver failure, despite not drinking alcohol.
- You are otherwise well enough to survive such an operation.
- You commit to not drinking alcohol for the rest of your life.

COMPLICATIONS

Portal hypertension and varices

Portal hypertension is a common complication of cirrhosis and, less commonly, alcoholic hepatitis. It occurs when the blood pressure inside the liver has risen to a potentially serious level. When the liver becomes severely scarred, it's harder for blood to move through it. This leads to an increase in the pressure of blood around the intestines. The blood must also find a new way to return to heart. It does this by using smaller blood vessels. But these vessels are not designed to carry the weight of blood, so they can become stretched out and weakened. These weakened blood vessels are known as varices. If the blood pressure rises to a certain level, it can become too high for the varices to cope with, causing the walls of the varices to split and bleed. This can cause long-term bleeding, which can lead to anaemia. Alternatively, the bleeding can be rapid and massive, causing to vomit blood and pass stools that are very dark or tar-like. Split varices can be treated by using an endoscope to locate the varices. A tiny band can then be used to seal the base of the varices.

Ascites

A person with portal hypertension may also develop a build-up of fluid in their abdomen (tummy) and around the intestines. This fluid is known as ascites. Initially, this can be treated with water tablets (diuretics). If the problem progresses, many litres of fluid can build up, this must be drained. This is a procedure known as paracentesis and involves a long, thin tube being placed into the fluid through the skin under local anaesthetic. One of the problems associated with the development of ascites is the risk of infection in the fluid (spontaneous bacterial peritonitis). This is potentially very serious complication and is linked to an increased risk of kidney failure and death.

Infection

Damage to the liver can weakens the immune system. This can make the body more vulnerable to infection, particularly urinary infections, and respiratory infections (pneumonia).

Hepatic Encephalopathy

One of the most important functions of the liver is to remove toxins from your blood. If the liver is unable to do this due to hepatitis or cirrhosis, the levels of toxins in the blood increase. A high level of toxins in the blood due to liver damage is known as hepatic encephalopathy.

Symptoms of hepatic encephalopathy include:

- Agitation

- Confusion
- Disorientation
- Muscle stiffness
- Muscle tremors
- Difficulty speaking
- In very serious cases, a coma

Hepatic encephalopathy may require hospital admission. In hospital, body functions are supported, and medicine is used to remove toxins from the blood.

Liver cancer

Liver damage due to heavy drinking over many years can also increase risk of developing liver cancer. Over the past few decades, rates of liver cancer in the UK have risen sharply due to increased levels of alcohol misuse. It's estimated 3% to 5% of people with cirrhosis will develop liver cancer every year.

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.

DRUG THERAPY OF ALCOHOLIC LIVER DISEASE (ALD)

The use of medicine to directly treat ARLD is controversial. For people with severe alcoholic hepatitis, treatment in hospital may be necessary. Specific treatment with corticosteroids may be used to reduce the inflammation of the liver in some people with this condition. Nutritional support is also an important part of treatment in these cases.[7]

MANAGEMENT

Cessation of alcohol consumption is the single most important treatment, without this, all other therapies are of limited value. Abstinence is even effective at preventing progression of liver disease and death when cirrhosis is present. Life-long abstinence is the best advice and is essential for those with more severe liver disease. Treatment of alcohol dependency, in the acute presentation of alcoholic liver disease, it is also important to identify and anticipate alcohol withdrawal and Wernicke's encephalopathy, which need treating in parallel with the liver disease.[10]

Corticosteroids

These are of value in patients with severe alcoholic hepatitis (Maddrey's discriminative score > 32) and increase survival. A similar improvement in 28-day survival from 52% to 78% is seen when steroids are given to those with a Glasgow score of > 9. Sepsis is the side-effect of steroids, and existing sepsis and variceal haemorrhage are the main contraindications to their use. If the bilirubin has not fallen 7 days after starting steroids, the drugs are ultimately to reduce mortality and should be stopped.[11]

Pentoxifylline

Pentoxifylline is a phosphodiesterase inhibitor used in the treatment of intermittent claudication. Pentoxifylline, which has a weak anti-TNF action, may be beneficial in severe alcoholic hepatitis. It appears to reduce the incidence of hepatorenal failure, and its use is not complicated by sepsis. Hepatorenal syndrome was the cause of death, thus, the benefit of pentoxifylline in treating alcoholic hepatitis appears to be related to a significant decrease in the risk of developing hepatorenal syndrome.

Colchicine

Colchicine is an inhibitor of collagen synthesis. Unfortunately, results regarding the use of colchicine in the treatment of liver cirrhosis are conflicting. In their study of colchicine for the treatment of all forms of cirrhosis, the 5-year survival rate was 74% in the colchicine-treated group compared with 34% in the placebo group. The 10-year survival rate was twice as great for the colchicine-treated group.[12]

Phosphatidylcholine

Encouraging results have been obtained with some super nutrients in the treatment of alcoholic liver disease. Phosphatidylcholine, purified from polyunsaturated lecithin, was discovered to oppose ethanol-induced fibrosis by decreasing the activation of stellate cells to transitional cells, and possibly also by stimulating collagenase activity.

For Alcoholic Hepatitis

- Prednisolone: 40 mg orally daily for 4 weeks, then taper the dose.
- Silymarin:
  - Antioxidative and Antifibrotic properties.
  - Believed to enhance liver regeneration and protect hepatocytes from toxicity.
  - Recommended dose is 140 mg 2-3 times/day.

For Cirrhotic Ascites

Bed rest and sodium restriction (60-90 meg/day to 1500-2000 mg of salt/day).

- Spironolactone: 100-400 mg/day.
- Furosemide: 40-160 mg/day.
- Hydrochlorothiazide: 50 mg/day.

PATIENT COUNSELLING OF ALCOHOLIC LIVER DISEASE

Lifestyle Modifications

- Cornerstone of long-term management of alcohol-related liver disease (ALD).
- Though the safe alcohol consumption level continues to be reviewed, patients with no liver disease should be advised to take no more than 2 standard drinks/day for males and no more than 1 standard drink/day for females.
  - Patients with ALD or other liver diseases (MASH, MASLD, viral hepatitis and hemochromatosis) should be advised to completely abstain from alcohol.
- Educate the patient regarding the nature of the disease and the benefit of discontinuing alcohol intake.
  - Abstinence or marked reduction in alcohol intake has been shown to improve histology and/or survival in all stages of ALD.
  - Abstinence can cause total resolution of alcoholic steatosis and improve long-term prognosis in alcoholic hepatitis.
  - Risk of liver-related complications and mortality is reduced complete alcohol abstinence in patients with alcohol-related cirrhosis.
- Assistance should be given to the patient to help them change their behaviour.
  - Psychosocial behavioural approaches may include counselling, group therapies or inpatient rehabilitation.
  - Other modalities include cognitive behavioural therapy, motivational interviewing, or motivational enhancement therapy.
- Patient may require consultation with a psychiatrist or addiction specialist.[8]
- Disulfiram, Naltrexone and Acamprosate are approved abstinence and relapse prevention medications for patients with alcohol use disorder (AUD).
  - Due to their potential for hepatotoxicity, Disulfiram and Naltrexone should be avoided in patients with ALD.
- Nalmefene is approved in Europe for reduction of heavy drinking in AUD and may be considered when abstinence is not feasible in patients with early-stage liver disease.
- Baclofen is used in patients with moderate alcohol withdrawal symptoms and to prevent alcohol relapse in patients with advanced ALD.
  - Psychosocial and behavioural approaches may include counselling, group therapies or inpatient rehabilitation.
  - Other modalities include cognitive behavioural therapy, motivational interviewing, or motivational enhancement therapy.
- Benzodiazepines are used in patients with ALD for alcohol detoxification to treat withdrawal symptoms. Inpatient detoxification provides the safest setting for treatment of alcohol withdrawal due to the unpredictability of withdrawal symptoms.[8]
  - Advise patients to refrain from concurrent use of alcohol and benzodiazepines.
  - Due to its potential for abuse and/or encephalopathy, benzodiazepines should not be given for >10-14 days.

Other Lifestyle Modifications

Smoking Cessation

- Smoking may increase rate of progression of fibrosis in ALD.[13]
- Patients should be encouraged to stop smoking.

Obese Patients

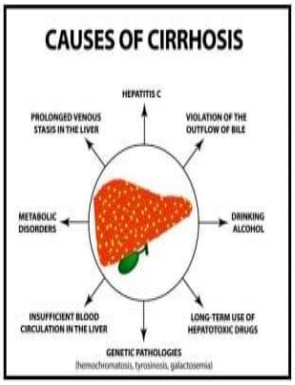
- Body mass index (BMI) has been shown to be an independent risk factor for ALD development.
- Physical activity and exercise are encouraged in obese patients.[13]

WHAT IS ALD?

Alcoholic liver disease is damage to the liver and its function due to alcohol abuse.

WHAT CAUSES ALD?

Alcohol-related liver disease (ARLD) is liver damage caused by alcohol intake.

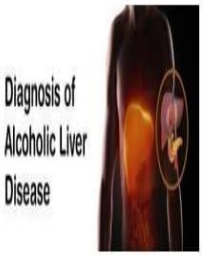


SYMPTOMS OF ALD:

- Feeling sick
- Weight loss
- Loss of appetite
- Confusion
- Drowsiness
- Vomiting blood

HOW IS ALD DIAGNOSED?

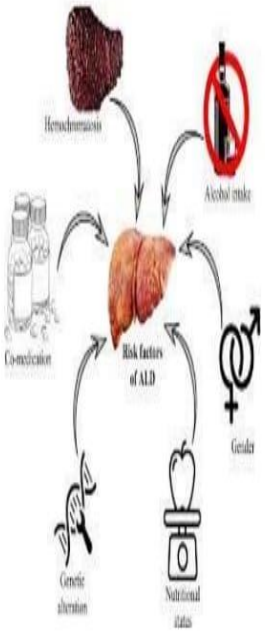
- Blood test
- Imaging tests
- Liver biopsy



HOW IS ALD TREATED?

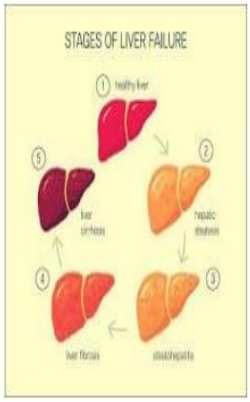
- Alcohol abstinence
- Nutritional support
- Liver transplantation
- Quit smoking
- Avoid hepatotoxic agents

RISK FACTORS OF ALD ARE:



COMPLICATIONS OF ALD ARE:

- Portal hypertension
- Jaundice and Cholestasis
- Hepatorenal syndrome
- Hepatic Encephalopathy



PHARMACOLOGICAL THERAPY

1) ALCOHOL ABSTINENCE

- Disulfiram
- Naltrexone

2) CORTICOSTEROID

- Prednisolone

3) ANTI CYTOKINE THERAPY

- Pentoxifylline

NON PHARMACOLOGICAL ROLE

- Avoid alcohol
- Avoid tobacco intake

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NAAC ACCREDITED INSTITUTION

ALCOHOLIC LIVER DISEASE (ALD)

A GUIDE FOR FAMILIES AND PATIENTS

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