IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429
Volume 5, Issue 2, December 2025
Impact Factor: 7.67

Alcoholic Liver Disease (ALD)

Mr. Swapnil Digambar Patil, Asst. Prof. Brigmohan Sagane,
Dr. Avinash S. Jiddewar, Mr. Prachi H. Rathode, Mohini V. Kashikar
NSPM College of Pharmacy, Darwha, Yavatmal

Abstract: *Alcohol-induced liver disease, which includes a variety of conditions such as simple steatohepatitis and superimposed hepatocellular carcinoma, is a leading cause of chronic liver disease globally. The pathophysiology of alcohol liver disease is still unknown, and there are presently no FDA-approved medications for its therapy, despite the fact that the field has made enormous strides in the last 21 years. Through the use of multiple research and other state-of-the-art techniques, we address novel insights into the pathophysiology and potential treatment targets of alcohol-related liver disease in this review. It is discussed how these studies might be applied to therapy and clinical practice. Additionally, we go over alcohol-associated liver disease, the interaction between alcohol liver disease, and preclinical models of alcohol liver disease.*

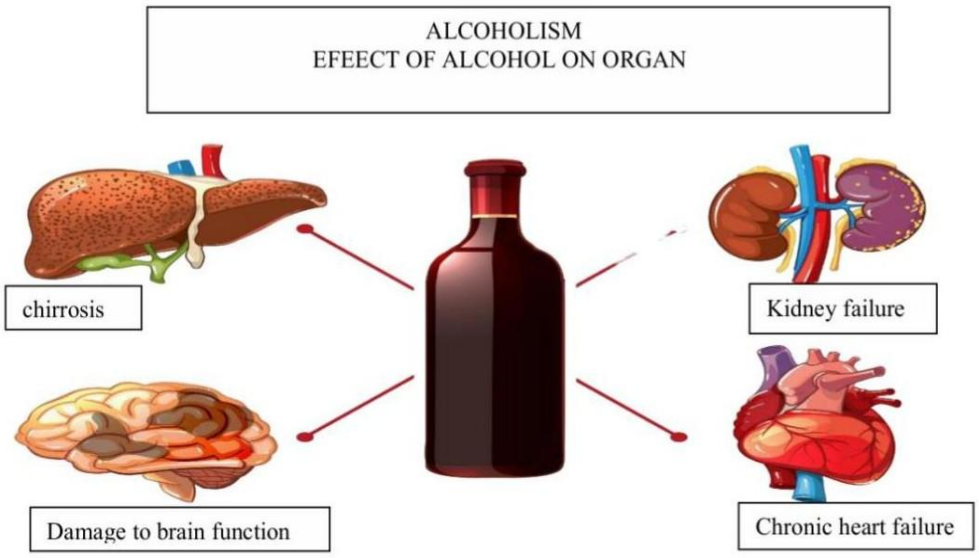
Keywords*:* Alcohol metabolism, alcoholic liver disease, alcoholic fatty liver, alcoholic steatohepatitis, alcoholic hepatitis, alcoholic cirrhosis, hepatocellular carcinoma, intestinal microbiome, MEOS, microsomal ethanol-oxidizing system, alcohol dehydrogenase.

I. INTRODUCTION

It is a condition of a structural and functional changes in the liver which is mostly caused Due to excessive consumption of alcohols. is a spectrum of diseases that encompasses alcoholic cirrhosis, hepatic steatosis, steatohepatitis with or without fibrosis, and AH. The most prevalent symptom is steatosis. The majority of alcohol users do not get ALD, and only a small percentage of heavy drinkers—who are impacted by environmental, sex, and genetic factors—progress to severe ALD. Worldwide, alcohol use disorders are a major source of avoidable disease, and among individuals who abuse alcohol for an extended period of time, alcoholic liver disease causes a considerable amount of liver-related morbidity and mortality. ALD is the leading cause of advanced liver disease worldwide, accounting for half of all instances of liver cirrhosis. With greater rates of complications and death, ALD is typically diagnosed at an advanced stage of the illness. It is important to support the early identification of early stages of ALD in the primary care context as well as any behavioral therapies that follow. Nevertheless, the early phases of ALD in humans are not well described. Furthermore, defining the natural history and prognostic variables is obviously necessary. prognostic determinants into to develop trustworthy non-obtrusive flags for ALD. The administration of patients accompanying ALD has developed little on account of many determinants including troubles of attending dispassionate tests in patients accompanying an alive alcohol abuse, the boredom from drug companies, public capital for research and the disadvantages of existent exploratory models. As a consequence, there are banned mean cures to treat patients accompanying harsh ALD [3]. The incident of aforementioned therapies demands translational studies cruel samples and appropriate animal models that photocopy clinical and histological countenance of alcoholic hepatitis (AH). In current age, new animal models that imitate some of the facial characteristics of human AH have existed grown, and translational studies utilizing hu man samples have identified potential pathogenic determinants and histological limits that foresee continuation.

Copyright to IJARSCT
www.ijarsct.co.in
DOI: 10.48175/568

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429
Volume 5, Issue 2, December 2025
Impact Factor: 7.67



ALD divided into three stages:

1. Alcoholic fatty liver : It is the initial and mildest stage which is caused by prolonged consumption of small amount of alcohols.
2. Alcoholic hepatitis : It Is the second stage which is caused due to excessive consumption of alcohol which increases fatty changes and liver cell necrosis.
3. Alcoholic cirrhosis : It is the third and most advanced stage, which obstructs liver function and reduces blood facus through lives.

1. Alcoholic fatty liver:

Heavy alcohol use is the cause of alcoholic fatty liver disease. The majority of the alcohol you consume is broken down by your liver, allowing your body to eliminate it. However, the breakdown process may produce toxic compounds. These drugs can impair your body's natural defenses, cause inflammation, and harm liver cells. The more alcohol you consume, the more harm it does to your liver. The first stage of alcohol-related liver damage is called alcoholic fatty liver disease.

2. Alcoholic hepatitis

Hepatitis is irritation of the liver. It`s a signal of contamination or harm to the tissues. Several matters can motive hepatitis, together with viruses and toxins. Heavy alcohol use is likewise certainly considered one among them. Acute alcohol-triggered hepatitis (previously called alcoholic hepatitis) would possibly best be a transient reaction to overindulgence. But while alcohol-triggered hepatitis turns into a continual condition, it threatens.

3. Alcoholicirrhosis

Cirrhosis is extreme scarring of the liver. This extreme circumstance may be because of many sorts of liver sicknesses and conditions, including hepatitis or persistent alcoholism. Each time your liver is injured — whether or not via way of means of immoderate alcohol intake or any other cause, including infection — it attempts to restore itself. In the process, scar tissue forms. As cirrhosis receives worse, increasingly scar tissue forms, making it tough for the liver to do its job. Advanced cirrhosis is life-threatening

4. Pathophysiology :

Alcohol metabolism via way of means of the liver is basically through enzymes:

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429
Volume 5, Issue 2, December 2025
Impact Factor: 7.67

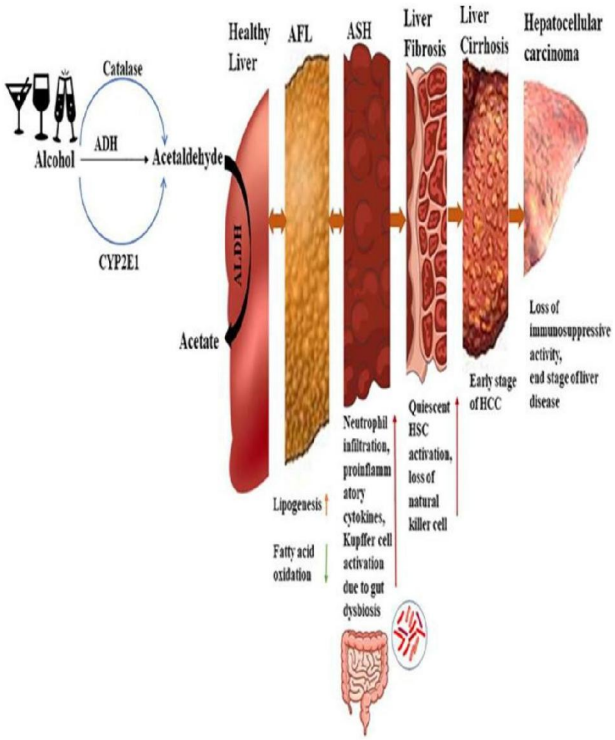
1. Alcohol dehydrogenase

2.Aldehy dedehydrogenase

Alcohol dehydrogenase converts alcohol into acetaldehyde, and aldehyde dehydrogenase converts acetaldehyde into acetate. The metabolism of alcohol will increase the manufacturing of NADH via way of means of decreasing NAD withinside the body. This moving of metabolic stability closer to the manufacturing of NADH results in the formation of glycerol phosphate, which mixes with the fatty acids and turns into triglycerides, which collect withinside the liver. When lipid oxidation (lipolysis) stops because of alcohol intake, fat collect withinside the liver and lead to "fatty liver disease." Continued alcohol intake brings the immune machine into play. Interleukins with the assist of neutrophils assault the hepatocytes, and swelling of the hepatocytes referred to as the "alcoholic hepatitis" takes place.

5. Histology:

Since alcoholic fatty liver is a benign and curable condition, liver biopsy is typically not required for diagnosis. To ascertain the extent of alcoholic liver disease progression and rule out cirrhosis, a biopsy may be necessary in certain patients. Alcoholic hepatitis is frequently diagnosed based on clinical and laboratory findings, though there may still be some diagnostic uncertainty in the absence of histological confirmation due to the advancements in serologic and genetic diagnoses of infectious and metabolic hepatitis over the past ten years. 22, 23 Although liver biopsies are frequently helpful in confirming the diagnosis and assessing the degree of liver damage, they are risky for patients who have coagulopathy thrombocytopenia. Understanding the Pathogenesis of Alcoholic Liver Disease (ALD) 2.1. Ethanol Metabolism or Ethanol, a polar molecular compound, exhibits solubility in both aqueous and lipid environments. Once ingested, it is absorbed into the bloodstream via the gastrointestinal tract. Typically, over 95% of ingested ethanol is metabolized by the liver, while a minor portion is directly eliminated through respiration, urination, and perspiration [11].



As indicated by prior research, three primary metabolic pathways are involved in the oxidative conversion of ethanol into acetaldehyde. The first is the alcohol dehydrogenase (ADH) pathway found in hepatocytes, which oxidizes ethanol

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429 Volume 5, Issue 2, December 2025 Impact Factor: 7.67

into acetaldehyde with the assistance of nicotinamide adenine dinucleotide (NAD+) as a co factor. Among its various isoenzymes, ADH1 is crucial for ethanol metabolism within the liver. Acetaldehyde produced by ADH is subsequently converted to acetate by acetaldehyde dehydrogenase (ALDH) [12,13]. The second major metabolic pathway is mediated by cytochrome P450 2E1 (CYP2E1)enzymes, which transform chemical substances into polar metabolites before excretion. C YP2E1 serves as the primary element of this system. Under standard physiological circumstances, CYP2E1 facilitates the oxidation of a minor fraction of ethanol, about 10%, into acetaldehyde. However, in cases of chronic alcohol consumption, the expression of C YP2E1 is induced, leading to increased activity. The third metabolic pathway consists of the catalase endoplasmic reticulum (CAT) system, regarded as another vital metabolic pathway that utilizes NADPH for oxidative breakdown. This catalase enzyme, which contains heme, typically functions to eliminate H2O2 but is also capable of facilitating the oxidation of ethanol to acetaldehyde.

6. Signs:

1) Loss of vitality
2) Decreased appetite and unintentional weight loss
3) Feeling nauseous
4) Abdominal discomfort
5) Tiny, red spider-like vascular lesions on the skin
6) As liver function deteriorates, signs may manifest
7) Fluid accumulation in the legs (edema) and in the abdomen (ascites)
8) Yellowing of the skin, mucous membranes, or eyes (jaundice)Redness of the palms
9) In men, sexual dysfunction, reduction in testicle size, and breast enlargement
10) Easy bruising and unusual bleeding
11) Confusion or cognitive difficulties
12) Pale or light-colored stools
13) Bleeding within the gastrointestinal system

7. Assessments:

Your healthcare professional will conduct a physical examination to identify:

1) An enlarged liver or spleen
2) Excessive breast tissue in males
3) Swelling of the abdomen due to fluid retention
4) Red palms
5) Spider-like blood vessels on the surface of the skin
6) Reduced size of testicles
7) Dilated veins in the abdominal wall
8) Yellowing of the eyes or skin (jaundice)
9) Possible tests may include:
10) Complete blood count (CBC)
11) Liver function assessments
12) Coagulation tests
13) Liver biopsy
14) Exclusions for other illnesses may involve:
15) Abdominal CT scan
16) Blood tests for alternate causes of liver issues
17) Abdominal ultrasound
18) Ultrasound elastography

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429
Volume 5, Issue 2, December 2025
Impact Factor: 7.67

8. TREATMENT :

Most main situation you can undertake search out forever stop quaffing intoxicating to give your liver the chance to rest and cure nearly likely. Depending on the severity of damage to your liver and your particular ailment.

II. CONCLUSION

Clinicians concede possibility screen all sufferers for harmful patterns of intoxicating use. All sufferers with intoxicating accompanying liver affliction should forgo intoxicating. For those with harsh ailment (i.e,DF ⩾32 or hepatic encephalopathy or two together), and no contraindications to their use, steroids should be thought-out. Liver transplantation concede possibility be considered as a situation alternative for patients accompanying decompensated intoxicating connected cirrhosis and severe alcoholic hepatitis.

REFERENCES

[1]. Ambade A, Mandrekar P. Oxidative stress and inflammation: Essential partners in alcoholic liver disease. International Journal of Hepatology. 2012;2012;853175 doi: 10.1155/2012/853175. [DOI] [PMC free article] [PubMed] [Google Scholar]

[2]. Anstee QM, Seth D, Day CP. Genetic factors that affect risk of alcoholic and nonalcoholicFattyliverdisease.Gastroenterology.2016;150(8):17281744.e7.doi:10.1 053/j.gastro.2016.01.037. [DOI] [PubMed] [Google Scholar]

[3]. Aragon CM, Rogan F, Amit Z. Ethanol metabolism in rat brain homogenates by a catalase-H2O2 system. Biochemical Pharmacology. 1992;44(1):93–98. doi: 10.1016/0006-2952(92)90042-h. [DOI] [PubMed] [Google Scholar]

[4]. Bala S, Petrasek J, Mundkur S, et al. Circulating microRNAs in exosomes indicate hepatocyte injury and inflammation in alcoholic, drug-induced, and inflammatory liver diseases. Hepatology. 2012;56(5):1946–1957. doi: 10.1002/hep.25873. [DOI] [PMC free article] [PubMed] [Google Scholar]

[5]. Becker U, Deis A, Sorensen TI, et al. Prediction of risk of liver disease by alcohol intake, sex, and age: A prospective population study. Hepatology. 1996;23(5):1025–1029. doi: 10.1002/hep.510230513. [DOI] [PubMed] [Google Scholar]

[6]. Bergheim I, McClain CJ, Arteel GE. Treatment of alcoholic liver disease. Digestive Diseases. 2005;23(3–4):275–284. doi: 10.1159/000090175. [DOI] [PMC free article] [PubMed] [Google Scholar]

[7]. Bode C, Bode JC. Effect of alcohol consumption on the gut. Best Practices & Research. Clinical Gastroenterology. 2003;17(4):575–592. doi: 10.1016/s1521 6918(03)00034-9. [DOI] [PubMed] [Google Scholar]

[8]. Brooks PJ, Zakhari S. Acetaldehyde and the genome: Beyond nuclear DNA adducts and carcinogenesis. Environmental and Molecular Mutagenesis. 2014;55(2):77– 91. doi:10.1002/em.21824.[DOI] [PubMed] [GoogleScholar] 10.1093/alcalc/35.3.28 6. [DOI] [PubMed] [Google Scholar]

[9]. Kharbanda KK, Mailliard ME, Baldwin CR, et al. Betaine attenuates alcoholic steatosis by restoring phosphatidylcholine generation via the phosphatidylethanolamine methyltransferase pathway. Journal of Hepatology. 2007;46(2):314–321. doi: 10.1016/j.jhep.2006.08.024. [DOI] [PubMed] [Google Scholar]

[10]. Kharbanda KK, McVicker DL, Zetterman RK, Donohue TM., Jr Ethanol consumption reduces the proteolytic capacity and protease activities of hepatic lysosomes. Biochimica et Biophysica Acta. 1995;1245(3):421–429. doi: 10.1016/0304-4165(95)00121-2. [DOI] [PubMed] [Google Schlor]

[11]. Kharbanda KK, McVicker DL, Zetterman RK, Donohue TM., Jr Ethanol consumption alters trafficking of lysosomal enzymes and affects the processing of procathepsin L in rat liver. Biochimica et Biophysica Acta. 1996;1291(1):45–52. doi: 10.1016/0304-4165(96)00043-8. [DOI] [PubMed] [Google Scholar]

[12]. Kharbanda KK, Todero SL, Ward BW, et al. Betaine administration corrects ethanol-induced defective VLDL secretion. Molecular and Cellular Biochemistry. 2009;327(1– 2):75–78. doi: 10.1007/s11010-009-0044-2. [DOI] [PubMed] [Google Scholar]

IJARSCT
International Journal of Advanced Research in Science, Communication and Technology
International Open-Access, Double-Blind, Peer-Reviewed, Refereed, Multidisciplinary Online Journal
ISSN: 2581-9429
Volume 5, Issue 2, December 2025
Impact Factor: 7.67

[13]. Kim MS, Ong M, Qu X. Optimal management for alcoholic liver disease: Conventional medications, natural therapy or combination? World Journal of Gastroenterology. 2016;22(1):8–23. doi: 10.3748/wjg.v22.i1.8. [DOI] [PMC free article] [PubMed] [Google Scholar]

[14]. Kirpich IA, Miller ME, Cave MC, et al. Alcoholic liver disease: Update on the role of dietary fat. Biomolecules. 2016;6(1):1. doi: 10.3390/biom6010001. [DOI] [PMC free article] [PubMed] [Google Scholar]

[15]. Klatsky AL, Armstrong MA. Alcohol, smoking, coffee, and cirrhosis. American Journal Epidemiology.1992;136(10):12481257.doi:10.1093/oxfordjournals.aje.a11 6433. [DOI] [PubMed] [Google Scholar]

[16]. Lefkowitch JH. Morphology of alcoholic liver disease. Clinics in Liver Disease. 2005;9(1):37–53. doi: 10.1016/j.cld.2004.11.001. [DOI] [PubMed] [Google Scholar]

[17]. Levy RE, Catana AM, Durbin-Johnson B, et al. Ethnic differences in presentation and severity of alcoholic liver disease. Alcoholism: Clinical and Experimental Research. 2015;39(3):566–574. doi: 10.1111/acer.12660. [DOI] [PMC free article] [PubMed] [Google Scholar]

[18]. Lieber CS. Alcoholic liver disease: New insights in pathogenesis lead to new treatments. Journal of Hepatology. 2000;32(1 Suppl):113–128. doi: 10.1016/s0168- 8278(00)80420-1. [DOI] [PubMed] [Google Scholar]

[19]. Lieber CS. Alcoholic fatty liver: Its pathogenesis and mechanism of progression to inflammation and fibrosis. Alcohol. 2004;34(1):9–19. doi: 10.1016/j.alcohol.2004.07.008. [DOI] [PubMed] [Google Scholar]

[20]. Lieber CS, DeCarli LM. Ethanol oxidation by hepatic microsomes: Adaptive increase Afterethanolfeeding.Science.1968;162(3856):91918.doi:10.1126/science.1 62.3856.917. [DOI] [PubMed] [Google Scholar]

[21]. Lucey MR. Liver transplantation for alcoholic liver disease. Nature Reviews. Gastroenterology&Hepatology.2014;11(5):300307.doi:10.1038/nrgastro.2013.247. [DOI] [PubMed] [Google Scholar]

[22]. Chalasani NP, Maher J. Alcoholic and nonalcoholic steatohepatitis. In: Goldman L, Cooney KA, eds. Goldman-Cecil Medicine. 27th ed. Philadelphia, PA: Elsevier; 2024:chap138

[23]. Haines EJ, Thompson H. Liver and biliary tract disorders. In: Walls RM, ed. Rosen's Emergency Medicine: Concepts and Clinical Practice. 10th ed. Philadelphia, PA: Elsevier; 2023:chap 76.

[24]. Szabo G, McClain CJ. Alcohol-associated liver disease. In: Feldman M, Friedman LS, Brandt LJ, eds. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. 11th ed. Philadelphia, PA: Elsevier; 2021:chap 86.