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# A STUDY ON RELATIVE EFFECTS OF ALCOHOL USE IN DECOMPENSATED CHRONIC LIVER DISEASE IN A TERTIARY CARE HOSPITAL

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## ABSTRACT

**Background:** Liver disease can progress from a compensated state to decompensated cirrhosis. Alcohol consumption accelerates liver injury in both stages, but its impact is particularly pronounced in decompensated chronic liver disease (DCLD). **Objective:** To estimate the relative effects of heavy and moderate alcohol consumption in patients with DCLD and CLD, to determine the prevalence of alcohol use among DCLD patients, and to evaluate its impact on disease progression and severity. **Methods:** A prospective observational study was conducted over six months in a government general hospital, Kurnool [Dist]. Patients aged ≥18 years were enrolled and randomized into two groups DCLD and CLD. Patients were evaluated for changes in systolic and diastolic blood pressure, renal function markers, and incidence of adverse effects. Alcohol consumption patterns were categorized as heavy, moderate, or low. **Results:** Heavy alcohol intake was the major

contributing factor for progression from CLD to DCLD. Males exhibited a higher prevalence of DCLD compared to females. No significant difference in DCLD prevalence was observed between moderate alcohol consumers and non-drinkers. Patients over 40 years had a higher susceptibility to DCLD. **Conclusion:** Excessive alcohol consumption is strongly associated with the development and progression of DCLD. Age above 40 years and male gender were identified as additional risk factors.

**KEYWORDS:** Chronic liver disease (CLD), Decompensated chronic liver disease (DCLD), End Stage Liver Disease (ESLD), Non-alcoholic fatty liver disease (NAFLD), Model for End-Stage Liver Disease (MELD)

## INTRODUCTION

Decompensated liver disease is also known as decompensated cirrhosis. Cirrhosis is a chronic liver disease that's commonly the result of hepatitis or alcohol use disorder. Cirrhosis is the severe scarring of the liver seen at the terminal stages of chronic liver disease. When your liver is damaged, scar tissue is formed as it tries to repair itself.

### Compensated Liver Disease (CLD)

Compensated liver disease (CLD) represents a stage in chronic liver damage where the liver remains functionally stable despite having undergone substantial injury over months or years. This damage typically arises from persistent inflammation leading to fibrosis and eventually cirrhosis, though the liver can still perform most of its tasks. Common causes include alcoholic liver disease, where heavy and prolonged alcohol intake induces fatty changes, hepatitis, and eventually irreversible scarring. Non-alcoholic fatty liver disease (NAFLD), often driven by metabolic syndrome—which includes obesity, diabetes, and hyperlipidemia—can also progress into liver inflammation and cirrhosis. Chronic viral hepatitis, especially types B, C, and D, is a major contributor globally, with genotypes differing by region and influencing disease patterns and response to therapy. Genetic conditions like alpha-1 antitrypsin deficiency, Wilson disease (causing copper buildup), and hereditary hemochromatosis (iron overload) initiate liver injury at the molecular level. Autoimmune conditions, including autoimmune hepatitis, primary biliary cirrhosis, and primary sclerosing cholangitis, involve the immune system mistakenly attacking liver structures, often leading to progressive scarring. Additionally, certain drugs—such as methotrexate, phenytoin, and isoniazid—can cause liver toxicity with chronic use, while vascular problems like Budd-Chiari syndrome disrupt blood flow and exacerbate liver damage. Around 15% of cases

remain idiopathic, with no clearly defined cause. Though patients in the compensated stage may not show overt symptoms, the underlying damage poses a serious risk for progression to liver failure or cancer, making early diagnosis and management critically important.

Chronic liver disease (CLD) progresses gradually and may manifest subtly in the compensated phase, yet the symptoms can worsen over time as liver function declines. Common signs include persistent fatigue, reduced appetite, diminished libido, jaundice (yellowing of the skin and eyes), itching, and flu-like feelings. Neurological changes are also notable—ranging from poor concentration and memory lapses to disorientation, euphoria, or unusual behavior, reflecting potential hepatic encephalopathy. If left unchecked, CLD can lead to serious complications such as complete liver failure, hepatorenal syndrome (loss of kidney function), esophageal varices (swollen veins at risk of bleeding), hyperestrogenism, portal hypertension, spontaneous bacterial peritonitis, and even coma or death. Diagnosis involves a combination of clinical evaluation and tests: review of symptoms and history, physical examination, liver-specific blood panels (checking enzyme levels, iron, cholesterol, and hepatitis markers), autoimmune and genetic screens, urine analysis, and imaging techniques like ultrasound, CT scan, transient elastography, and endoscopy. In some cases, a liver biopsy provides definitive insight into the extent and nature of liver damage. Early detection and monitoring are essential to prevent progression toward decompensated liver disease.

**Decompensated Liver Disease (DCLD)**

Decompensated liver disease (DCLD), also known as decompensated cirrhosis, marks a critical phase in chronic liver disease where the liver's ability to function becomes severely impaired due to extensive scarring. This condition often arises from long-standing liver damage caused by hepatitis B or C, alcohol use disorder, or non-alcoholic fatty liver disease. Histologically, cirrhosis is defined by fibrous tissue surrounding regenerative nodules, formed during chronic inflammation. As the liver deteriorates, complications such as fluid accumulation (ascites and edema), jaundice, bleeding disorders, reduced platelet count, hepatorenal syndrome, hyponatremia, hepatic encephalopathy, and even coma may develop. Symptoms include fatigue, bruising, abdominal pain, nausea, fever, dark urine, and cognitive disturbances. Besides common causes, inherited conditions (like Wilson's disease and hemochromatosis), autoimmune and genetic disorders, poorly formed bile ducts, and certain medications also contribute. Unlike compensated cirrhosis—which may be asymptomatic—

decompensated cirrhosis signals end-stage liver disease, often requiring liver transplantation to prevent fatal outcomes.

Diagnosing decompensated liver disease (DCLD) involves recognizing both visible symptoms and conducting detailed evaluations to confirm the severity of liver dysfunction. As patients progress from compensated to decompensated cirrhosis, they often develop clear signs such as jaundice, fluid retention, confusion, and gastrointestinal bleeding. Physicians begin with a clinical assessment, noting the patient's medical history, risk factors (like alcohol use or viral hepatitis), and physical changes such as abdominal distension or skin discoloration. Blood tests are fundamental, measuring liver enzymes, bilirubin, coagulation markers, and kidney function—especially because conditions like hepatorenal syndrome can emerge during decompensation. A key component of diagnosis is the MELD (Model for End-Stage Liver Disease) score, which uses values such as creatinine, bilirubin, and INR to estimate disease severity and prioritize liver transplant eligibility, with scores ranging from 6 (mild) to 40 (critical). Imaging plays a vital role: ultrasound is used to detect ascites and evaluate liver texture, CT and MRI scans provide detailed views of the liver and surrounding organs, and elastography tests measure liver stiffness, helping gauge the degree of fibrosis or cirrhosis. In certain cases, a liver biopsy may be performed to analyze tissue and distinguish between overlapping or co-existing conditions. Altogether, these diagnostic tools help physicians determine how far the disease has progressed, assess complications, and make crucial decisions regarding patient management and potential transplant referral.[1]

**Stages of Liver Damages**

Liver damage progresses through four distinct stages, each representing a worsening impact on the liver’s ability to function. The first stage, inflammation, occurs when the liver becomes irritated due to infections, toxins, or metabolic stress. This may not cause noticeable symptoms but signals the beginning of liver injury. If the inflammation persists, it leads to fibrosis, where scar tissue starts replacing healthy liver cells. Although some fibrosis can be reversible, it begins to interfere with blood flow and liver efficiency. The third stage is cirrhosis, marked by extensive and permanent scarring that severely disrupts liver architecture and function. At this point, symptoms often become more apparent, and complications may arise. The final stage is End-stage liver disease (ESLD), where the liver can no longer perform its vital roles, leading to life-threatening conditions such as hepatic

encephalopathy, ascites, and multi-organ failure. Each stage builds upon the previous one, making early detection and intervention crucial to prevent irreversible damage.

Liver damage progresses through four key stages, each representing a deeper level of impairment. The first stage, inflammation, involves swelling or enlargement of the liver, often without symptoms. If untreated, it can lead to fibrosis, where scar tissue replaces healthy liver cells, subtly reducing liver efficiency. In the third stage, cirrhosis, extensive scarring severely disrupts liver architecture, making it difficult for the organ to function properly—this is when symptoms typically begin to appear. The final stage, end-stage liver disease (ESLD), includes conditions like decompensated cirrhosis, stage 4 hepatitis C, and chronic liver failure. At this point, liver function is critically compromised, and complications such as ascites and hepatic encephalopathy may develop. Liver failure, which can be acute or chronic, occurs when the liver can no longer perform essential tasks like detoxifying blood or producing bile. While not all liver damage leads to failure, ESLD often requires a liver transplant to restore function and prevent fatal outcomes.

Liver damage and failure can result from a wide variety of causes, both acute and chronic. Common triggers include viral infections such as hepatitis A, E, and especially chronic forms of hepatitis B and C. Genetic conditions like Wilson disease, autoimmune hepatitis, and disorders affecting bile ducts or liver veins (such as cholangitis or Budd-Chiari syndrome) also contribute significantly. Alcohol misuse, non-alcoholic fatty liver disease, reactions to medications (including acetaminophen overdose and certain antibiotics or anti-inflammatory drugs), herbal supplements, and exposure to toxins (from chemicals or poisonous mushrooms) round out the list. While chronic liver failure develops over time due to progressive liver disease, acute liver failure may strike suddenly—even in individuals without prior liver issues—and sometimes its cause remains unknown. Symptoms in early stages like inflammation and fibrosis are often absent, but as liver function deteriorates, signs such as fatigue, appetite loss, and weight changes emerge. In cirrhosis, nausea, vomiting, and abdominal discomfort become more common. End-stage liver disease (ESLD) brings pronounced symptoms like jaundice, skin itching, bruising, fluid retention, digestive bleeding, and mental confusion, particularly in conditions such as decompensated cirrhosis or chronic hepatitis C. Prompt recognition and diagnosis are vital to managing progression and preventing life-threatening complications.[2]

**Effect Of Alcohol on Liver**

Alcohol has a profound impact on liver health due to the organ's central role in metabolizing and detoxifying substances. The liver converts nutrients into usable forms, filters toxins from the blood, and produces bile for fat digestion. When alcohol is consumed—especially in large amounts or over extended periods—the liver prioritizes breaking down ethanol, which can overwhelm its capacity and lead to damage. This damage may begin as fatty liver, progress to alcoholic hepatitis, and eventually result in cirrhosis, a condition marked by irreversible scarring. However, these stages don't always follow a linear path; any one of them can occur independently and pose serious health risks, including liver failure or death if untreated.

The type of alcohol in beverages is ethanol, produced through fermentation. Other forms like isopropyl or butyl alcohol are toxic and not safe for consumption. Alcohol content is measured by Alcohol by Volume (ABV), and serving sizes are standardized to contain about 0.6 ounces of pure alcohol. For example, a standard drink includes 5 oz of wine (12% ABV), 12 oz of beer (5% ABV), or 1.5 oz of spirits (40% ABV). Understanding these measurements is crucial for monitoring intake and reducing the risk of liver-related complications.[3]

**Alcoholic Fatty Liver Disease**

Alcoholic fatty liver disease (AFLD), also known as steatosis, is one of the earliest and most common liver conditions caused by chronic or excessive alcohol consumption. It occurs when fat accumulates abnormally within liver cells, surpassing healthy levels and interfering with liver metabolism. AFLD develops in the majority of individuals who consume large amounts of alcohol regularly—over 90% in those drinking 4–5 standard drinks daily for years—or even after short-term binge drinking episodes. Despite its prevalence, AFLD often remains silent, with few noticeable symptoms, although some people might experience mild discomfort in the upper right abdomen. The condition is classified as entirely preventable and reversible if diagnosed early and the person ceases alcohol intake. However, if ignored, AFLD can progress to more severe forms such as alcoholic hepatitis and ultimately cirrhosis, both of which carry risks of liver failure and long-term health consequences. Recognizing this disease early through medical evaluation and lifestyle changes is key to preventing irreversible damage.[4]

**Alcoholic Cirrhosis of the Liver**

Alcoholic hepatitis is a serious inflammatory liver condition that affects up to 35% of heavy drinkers, typically developing from untreated alcoholic fatty liver disease (AFLD). It presents

with sudden symptoms like jaundice, general discomfort (malaise), and liver enlargement (tender hepatomegaly). While milder cases can potentially be reversed with sustained alcohol abstinence, severe forms may result in short-term mortality and further progression to alcoholic cirrhosis. Alcoholic cirrhosis is the most advanced form of alcohol-related liver disease, marked by widespread fibrosis that obstructs blood flow and liver function. This leads to toxin accumulation, nutrient deficiencies, and diminished bile production—eventually causing liver failure. Symptoms range from fatigue and nausea to jaundice, skin itching (urticaria), swelling in limbs, abdominal fluid buildup (ascites), and mental decline (hepatic encephalopathy). Though early-stage cirrhosis may be silent, advanced disease is associated with high mortality, especially among young adults. While no curative treatments currently exist for alcohol-induced cirrhosis, supportive care and management of complications can help improve survival and quality of life.[5]

**Risk Factors for Liver Damage from Alcohol**

Several factors influence an individual's risk for developing alcohol-related liver damage and how quickly it progresses. Patterns of alcohol consumption are a major contributor—regular intake of 1 to 3 ounces of ethanol per day over a decade or more, and binge drinking episodes, significantly elevate the risk of developing severe liver conditions like alcoholic hepatitis and cirrhosis. Age also plays a role, with older adults being more vulnerable due to a decreased ability to metabolize alcohol efficiently. Ethnicity can affect the onset and severity of alcohol-induced liver disease; notably, white Hispanic men have shown the highest rates of alcoholic cirrhosis, although the reasons remain unclear. Additionally, obesity compounds the harmful effects of alcohol on the liver, with a high body mass index (BMI) being associated with greater susceptibility to liver damage. These risk factors highlight the importance of considering personal health profiles when evaluating the impact of alcohol consumption.[6]

**AUDIT Scale**

The Alcohol Use Disorders Identification Test (AUDIT) is a 10-item screening tool developed by the World Health Organization (WHO) to assess alcohol consumption, drinking behaviours, and alcohol-related problems. Patients should be encouraged to answer the AUDIT questions in terms of standard drinks. A chart illustrating the approximate number of standard drinks in different alcohol beverages is included for reference. A score of 8 or more is considered to indicate hazardous or harmful alcohol use. The AUDIT has been validated

across genders and in a wide range of racial/ethnic groups and is well suited for use in primary care settings.[7]

## MATERIALS AND METHODS

**Study Design:** Prospective observational study

**Setting:** Government General Hospital, Kurnool

**Duration:** November 2024 to April 2025

**Participants:** 97 inpatients (≥18 years)

**Data Collection:** Information was gathered from case report forms, laboratory investigation reports, and patient prescriptions. An annexure was employed to ensure standardization of data collection across participants.

**Ethical Approval:** Approved by Institutional Ethics Committee (SJCPS/IEC/V PD/ 2024-25/043A)

## RESULTS

A total of 112 patients who met the selection criteria were enrolled in the study. Among them, 7 from Group A (DCLD) and 8 from Group B (CLD) were dropped out because of loss of follow up. Therefore, a total of 97 patients, 29 from group A and 68 patients from group B completed the study.

**Table 1 and Figure 1** depicts Gender-wise distribution in our study in DCLD is among 68 patients 45 (67%) patients are males and 23 (33%) patients are females and in CLD is among 29 patients 11 (37%) patients are males and 18 (63%) patients are females.

**TABLE 1: GENDER WISE DISTRIBUTION.**

| Gender | DCLD | CLD |
|---|---|---|
| **Male** | 45 | 11 |
| **Female** | 23 | 18 |

Table 1 represents Gender wise distribution in both groups.

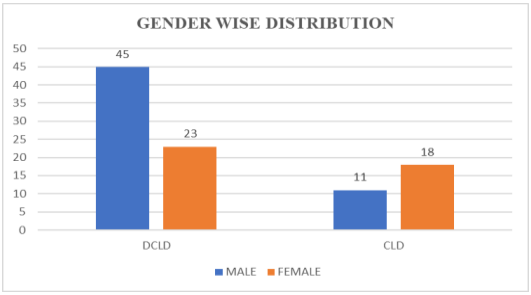


**Figure 1: Gender wise distribution in both groups.**

**Table 2 and Figure 2** indicates the gender in different age groups in CLD patients is as follows years, 11-20 years age group male are 0 and female are 0, 21-30 years age group males are 2 and females are 2, 31-40 years age group male are 2 and females are 3, 41-50 years age group male are 3 and females are 3, 51-60 years age group male are 2 and females are 4, 61-70 years age group male are 2 and females are 6.

**TABLE 2: AGE-GENDER WISE DISTRIBUTION IN CLD.**

| Age | Compensated liver disorder | |
|---|---|---|
| | Male | Female |
| **11--20** | 0 | 0 |
| **21-30** | 2 | 2 |
| **31-40** | 2 | 3 |
| **41-50** | 3 | 3 |
| **51-60** | 2 | 4 |
| **61-70** | 2 | 6 |

Table 2 represents Age-Gender wise distribution in patients with CLD.

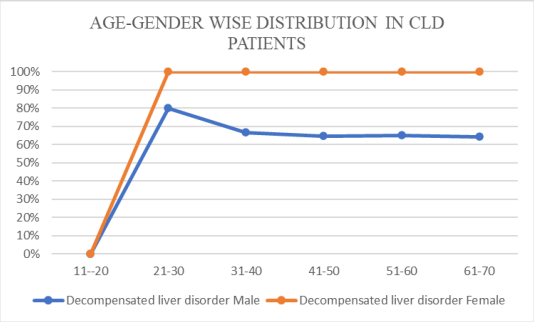


**Figure 2: Age-Gender wise distribution in patients with CL.**

**Table 3 and Figure 3** gender in different age groups in DCLD patients is as follows years, 11-20 years age group male are 0 and female are 0, 21-30 years age group males are 4 and females are 1,31-40 years age group male are 8 and females are 4, 41-50 years age group male are 11 and females are 6, 51-60 years age group male are 13 and females are 7, 61-70 years age group male are 9 and females are 5.

**TABLE 3: AGE-GENDER WISE DISTRIBUTION IN DCLD.**

| Age | Decompensated liver disorder | |
|---|---|---|
| | Male | Female |
| **11--20** | 0 | 0 |
| **21-30** | 4 | 1 |
| **31-40** | 8 | 4 |
| **41-50** | 11 | 6 |
| **51-60** | 13 | 7 |
| **61-70** | 9 | 5 |

Table 3 represents Age-Gender wise distribution in patients with DCLD.

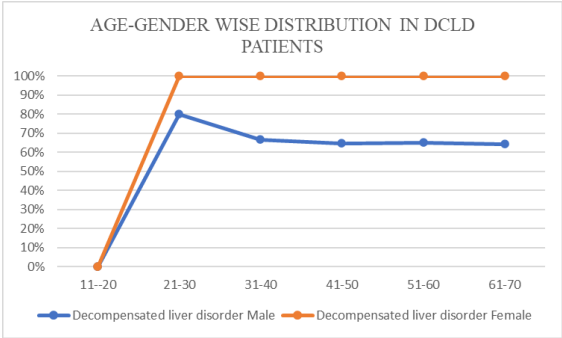


**Figure 3: Age-Gender wise distribution in patients with DCLD.**

**Table 4 and Figure 4** shows that in a total of 97 patients, 31(31.96%) male and 12(12.37%) female patients are alcoholics in DCLD and 5(5.15%) male and 1(1.03%) female patients are alcoholics in CLD whereas 12(12.37%) male and 13(13.40%) female patients are Non-Alcoholics in DCLD and 9(9.28%) male and 14(14.43%) female patients are Non-Alcoholics in CLD.

**TABLE 4: GENDER WISE ALCOHOL USAGE DISTRIBUTION IN DCLD AND CLD PATIENTS.**

| Alcohol Usage | DCLD | | P-Value | CLD | | P-Value |
|---|---|---|---|---|---|---|
| | Male | Female | | Male | Female | |
| **Alcoholics** | 31 | 12 | 0.046949 | 5 | 1 | 0.05365 |
| **Non-Alcoholics** | 12 | 13 | | 9 | 14 | |

Table 4 represents Gender Wise Alcohol Usage Distribution in DCLD and CLD patients.

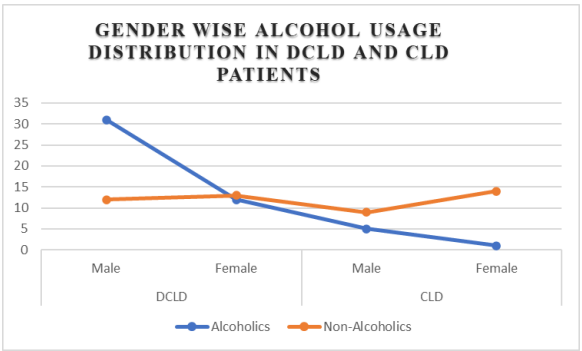


**Figure 4: Gender Wise Alcohol Usage Distribution in DCLD and CLD patients.**

**Table 5 and Figure 5** shows that in a total of 97 patient's data, there were patients with different stages of liver damage in which patients with Fatty liver were 10 patients in DCLD and 2 patients in CLD, patients with Fibrosis were 9 patients in DCLD and 04 patients in

CLD, patients with cirrhosis were 34 patients in DCLD and 3 patients in CLD, patients with ESLD were 2 patients in DCLD and 0 patients in CLD.

**TABLE 5: DISTRIBUTION OF PATIENTS ACCORDING TO HISTOLOGICAL DATA.**

| | Fatty Liver | Fibrosis | Cirrhosis | ESLD |
|---|---|---|---|---|
| **DCLD** | 10 | 09 | 34 | 2 |
| **CLD** | 02 | 04 | 03 | 0 |

Table 5 represents Distribution of patients according to histological data.

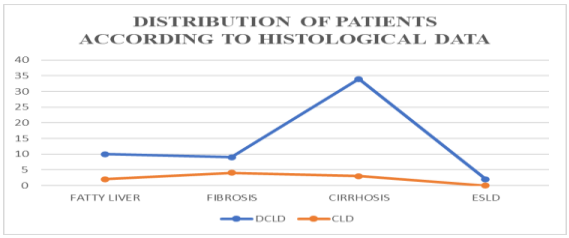


**Figure 5: Distribution of patients According to Histological Data.**

**Table 6 and Figure 6** represents that patients were categorized under 4 zones according to the AUDIT questionnaire i.e. Low risk, Risky, Harmful, Severe.

According to the questionnaire 2 patients with CLD and 6 patients with DCLD comes under low-risk zone, 3 patients with CLD and 8 patients with DCLD comes under risky zone, 1 patient with CLD and 13 patients with DCLD comes under harmful zone, 0 patients with CLD and 16 patients with DCLD comes under severe zone.

**TABLE 6: DISTRIBUTION OF PATIENTS ACCORDING TO THE AUDIT QUESTIONNAIRE.**

| | Low risk | Risky | Harmful | Severe |
|---|---|---|---|---|
| **DCLD** | 6 | 8 | 13 | 16 |
| **CLD** | 2 | 3 | 1 | 0 |

Table 6 represents Distribution of Patients According to the AUDIT Questionnaire.

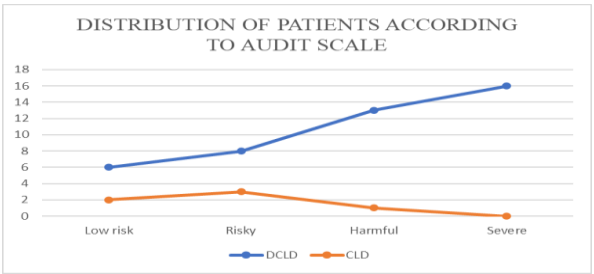


**Figure 6: Distribution of Patients According to the AUDIT Questionnaire.**

**Table 7 and Figure 7** represent by using the AUDIT Questionnaire as the basis, the alcohol intake can be categorized into three categories. They are as follows:

Low alcohol intake. (in which low risk zone patients were included)

Moderate alcohol intake. (in which risky zone patients were included)

Heavy alcohol intake. (in which harmful and severe zone patients were included)

**TABLE 7: DISTRIBUTION OF PATIENTS-BASED ALCOHOL INTAKE.**

| | Low Alcohol Intake | Moderate Alcohol Intake | Heavy Alcohol Intake |
|---|---|---|---|
| **DCLD** | **6** | **8** | 29 |
| **CLD** | **2** | **3** | 1 |

Table 7 represents Distribution of Patients-based alcohol intake.

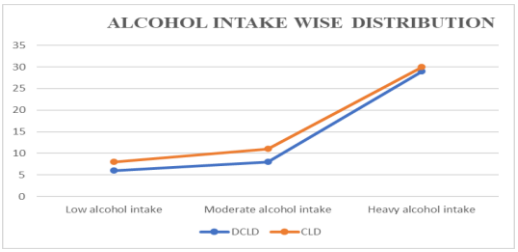


**Figure 7: Distribution of Patients-based alcohol intake.**

## DISCUSSION

The study presented here utilized a prospective cross-sectional approach to examine the relative effects of alcohol use on decompensated chronic liver disease (DCLD) by comparing it with compensated liver disease (CLD) in patients at a tertiary care hospital. The primary objective of the research was to determine the prevalence of alcohol use among patients with DCLD. Findings revealed that heavy alcohol consumption significantly increases the likelihood of CLD progressing to decompensation compared to low or moderate alcohol intake. The prevalence of heavy alcohol use was notably higher in individuals with decompensated liver cirrhosis than in those with compensated liver disease. However, the difference was less pronounced among patients who did not consume heavy amounts of alcohol. Furthermore, the study observed that DCLD prevalence is higher among males than females, which aligns with other studies showing that both CLD and DCLD are more common in men compared to women. The study conducted by P. Mankal et al. (2014) sought to examine the relative impact of heavy alcohol consumption and hepatitis C virus (HCV) infection on decompensated chronic liver disease (CLD). The research included 904 patients diagnosed with cirrhosis who were admitted to hospitals between January 2010 and

December 2012. The findings of the study indicated that the prevalence of CLD decompensation was significantly higher among patients with heavy alcohol intake compared to those who consumed alcohol in moderate amounts or abstained altogether. This conclusion aligns closely with the results obtained in our own study. The study conducted by Wong RJ et al. (2024) investigated the long-term effects of alcohol consumption on the risk of cirrhosis in veterans with steatotic liver disease. Analyzing data from January 2010 to December 2022, it categorized alcohol use into three groups—no alcohol, low-risk, and high-risk—based on AUDIT-C scores. Among 1,156,189 veterans, those identified as high-risk alcohol users had a 43% greater incidence of cirrhosis compared to those who abstained. Importantly, reducing alcohol consumption significantly lowered the likelihood of developing cirrhosis. The study highlights the need for timely alcohol use assessments and early intervention to mitigate high-risk drinking behaviors in individuals with steatotic liver disease. These findings align with our conclusion that moderate alcohol intake and abstinence show no significant difference in disease prevalence, while heavy alcohol consumption is strongly associated with CLD decompensation.

## CONCLUSION

Thus, this study concludes that excessive alcohol intake is associated with increased risk of developing DCLD compared to low and moderate alcohol intake. Besides, patients with age group above 40 years were prone to develop DCLD than their younger ones. Further, the chances of development of DCLD is higher in males than in females.

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