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Assessment Of Management Strategies In Alcoholic Liver Disease Patients With Hypertension Or Diabetes Mellitus

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ABSTRACT

Objective: Analysis of different Treatment Strategies in ALD with Hypertension & ALD with Diabetes Mellitus and its Prescription Patterns Assessment with Comparison study of Alcoholic liver disease or Alcoholic Liver Disease with Hypertension or Diabetes Mellitus. To learn about the specific treatments given in patients of ALD

Methods: A retrospective observational analysis was conducted in a tertiary care center. The data of the patients undergoing treatment for ALD with hypertension or diabetes mellitus were obtained. Patients with ALD undergoing therapy. Data of 100 patients was gathered and examined from the Parul Sevashram Hospital. The data was reported and analyzed using graphical, chart, figures, and tabulations, and it was visually summarized in the data collection analysis form.

Results: Among 100 patients, 97% of patients were prescribed with oral ursodeoxycholic acid in both ALD with Hypertension & ALD with Diabetes mellitus. Furthermore, Oral Spironolactone was prescribed in 95% Patients for the treatment. And it was followed by Rifaximin & intravenous Human Albumin in some patients. It has also been noticed that oral Lactulose, intravenous Furosemide & various other supplements have been prescribed majorly in ALD alongside.

Conclusion: Ursodeoxycholic acid, which is an Hepatoprotective Agent, Spironolactone which is an Aldosterone receptor antagonist, Rifaximin which is an antibiotic are majorly used in the treatment of this ailment. However more research and large sample size is required for the assessment of management of the following.

Keywords: *Ursodeoxycholic acid, alcoholic liver disease, hypertension, Spironolactone, Rifaximin, human albumin.*

1. INTRODUCTION

The term "alcoholic liver disease" refers to a broad range of disorders that begin with fatty liver, can progress to encompass alcoholic hepatitis, and ultimately culminate in alcoholic cirrhosis, the most severe and irreversible form of liver damage caused by alcohol.

Alcoholic liver disease encompasses three histological stages: [1], [2]

1. A disease called alcoholic fatty liver, or ketosis, causes fat to accumulate in the parenchyma of the liver.

2. Alcoholic Hepatitis: This stage is defined by inflammation of the liver cells, leading to diverse consequences depending on the extent of damage. Alcoholic hepatitis can be treated with diet, infection management, liver failure prevention, and, in more severe cases, prednisolone therapy.

3. Alcoholic cirrhosis: This kind of irreversible liver damage results in portal hypertension and issues related to cirrhosis.

Etiology

A wide range of parameters, including immunological, metabolic, genetic, and environmental ones, combine to cause alcoholic liver disease. [3]

Small doses of alcohol are tolerated by the liver, but increasing use puts it under metabolic stress. The first stage, known as fatty liver or steatosis, is characterized by the accumulation of fat in liver cells. Alcoholic hepatitis could develop with continued alcohol consumption. Alcohol-related liver damage progresses over time, leading to severe damage described as "alcoholic cirrhosis," which is characterized by worsening hepatic fibrosis and nodules.[3]

The amount and duration of alcohol usage, rather than the type of beverage, is the main risk factor for liver injury in patients. Compared to men, women are typically more vulnerable. Furthermore, a high-fat diet and obesity raise the risk of alcoholic liver disease. Lower death rates, more extensive histological damage, and an earlier beginning of the disease are linked to concurrent hepatitis C infection. The existence of patatin-like phospholipase domain-containing protein 3 (PNPLAP3) has been connected to alcoholic liver cirrhosis.[3]

Epidemiology

The most widely abused drug, both domestically and internationally, is alcohol. It is the primary cause of liver disease in the United States, affecting about 61% of people, of whom 10 to 12% consume large amounts of alcohol. Half an ounce, or 13.7 grams, of pure alcohol makes up a normal drink, according to the Centers for Disease Control and Prevention (CDC). This is equivalent to:

i. Twelve ounces of 5% alcohol-containing beer.
ii. Eight ounces of 7% alcohol-content malt liquor.
iii. Five ounces of wine with a volume percentage of 12% alcohol.
iv. One and a half ounces of 80 proof ("hard liquor") (40% alcohol).

The highest frequency of alcoholic liver damage is seen in Europe. Drinking 30 to 50 grams of alcohol a day for more than five years may cause damage to the liver. Alcohol consumption over 40 grams per day can cause 30 % of people to acquire cirrhosis, while alcohol consumption over 60 grams per day can cause 90 % of people to develop steatosis. [3]

Pathophysiology

The liver uses two enzymes principally for alcohol metabolism namely, Alcohol dehydrogenase and Aldehyde dehydrogenase. Acetaldehyde is formed from alcohol due to catabolism by alcohol dehydrogenase, and acetate is produced when acetaldehyde is dehydrogenated by aldehyde dehydrogenase. Alcohol metabolism lowers NAD levels in the body, which boosts NADH synthesis. Glycerol phosphate is produced when the metabolic balance is changed to favor the production of NADH. When the liver accumulates a certain molecule and mixes it with fatty acids, triglycerides are created. Drinking alcohol stops the oxidation of lipids, which causes a lipid accumulation in the liver and "fatty liver disease." Regular alcohol use sets off the immune system reaction. The hallmark of "alcoholic hepatitis" is the swelling of liver cells brought on by interleukins attacking them with neutrophil assistance. Cirrhosis is an incurable liver disorder brought on by prolonged liver injury. [3]

Histopathology

Stage 1: Hepatic steatosis is the initial stage. It kickstarts in the portal tracts and entails the buildup of tiny fat droplets surrounding the venules and liver cells. Intracellular lipid accumulation is caused by the changed intracellular redox potential.

Stage 2: As the illness progresses, there is noticeable steatosis, hepatic necrosis, and acute inflammation. This stage is called "alcoholic hepatitis" and is characterized by enlarged hepatocytes that include eosinophilic fibrillar material known as Mallory hyaline or Mallory-Denk bodies. A significant infiltration of polymorphonuclear leukocytes, or neutrophils, within the lobules is a hallmark of alcoholic hepatitis, in contrast to many other types of the disease where mononuclear cells congregate near portal triads.

Stage 3: Alcoholic cirrhosis is the last stage, where fibrotic septae encircle the liver's regenerating nodules. Collagen accumulation usually happens around the terminal hepatic vein and along the sinusoids in cases of alcoholic cirrhosis, resulting in a characteristic fibrosis pattern called "chicken wire." Liver fibrosis must be recognized by particular stains, such as Masson Trichrome or Sirius Red, in order to be assessed as best possible.[2]

The advancement of steatosis to steatohepatitis, liver fibrosis, and cirrhosis is linked to long-term alcohol use, heavy alcohol consumption, and particular drinking habits. Even after prolonging alcohol consumption, most ALD patients do not develop cirrhosis.

Management of ALD

The main strategy for managing ALD at any stage is abstaining from alcohol entirely. It has been shown that prolonged alcohol consumption increases portal pressure and aggravates symptoms of portal hypertension, including variceal hemorrhage. But after giving up alcohol, the histology of fatty liver can improve in as little as two weeks. A recent meta-analysis found that the overall survival rates of cirrhotic persons who had stopped drinking alcohol for at least 1.5 years were significantly higher. In addition to being required for liver donation, alcohol abstinence is also essential for improving health

outcomes; most transplant facilities need at least six months of verified abstinence prior to listing for transplantation.[4]

Protein and calorie deficiencies are common in ALD patients. It's critical to evaluate their nutritional status in detail and stress the need to eat a balanced diet. Vitamin and trace mineral deficiencies, such as those in zinc, pyridoxine, folate, vitamin D, thiamine, and protein intake (measured per kilogram of body weight), should be addressed, according to the American Association for the Study of Liver Disease (AASLD). It's also advised to keep your daily intake of protein at 1.2–1.5 grams per kilogram and calories at 35–40 per kilogram. The goal of these actions is to enhance nitrogen balance. [5]

People who suddenly cut back or stop drinking run the danger of developing alcohol withdrawal syndrome; a serious illness that can make long-term alcoholism worse. Within the first day after alcohol withdrawal, symptoms including hyperreflexia, irritability, raised heart rate, and blood pressure may appear. Over the course of the next few days, these symptoms may intensify and may result in serious consequences such as seizures and delirium tremens. In addition to being widely used to treat alcohol withdrawal, drugs such as baclofen, gabapentin, clonidine, and topiramate are also routinely used to counteract the potential side effects of benzodiazepines.[6][7]

2. MATERIALS AND METHODOLOGY

This retrospective observational study was conducted at Parul Sevashram Hospital, Vadodara, over a 6-month period, utilizing data from 100 patients diagnosed with alcoholic liver disease (ALD) or ALD with comorbidities such as hypertension or diabetes mellitus. Data were extracted from hospital medical records, including patient profile forms (PPF), case sheets, and electronic health records, following ethical approval and acquisition of written informed consent from participants. Inclusion criteria encompassed patients aged 18 years or older, of both sexes, who were diagnosed with ALD and consented to participate. Individuals unwilling to provide consent or under 18 years of age were excluded. The study focused on analyzing social determinants of health (e.g., socioeconomic status, lifestyle factors, and access to healthcare) through a structured review of clinical and demographic data. Retrospective analysis was performed to identify patterns and associations, with findings represented graphically to illustrate trends in disease presentation, comorbidities, and contributing social factors. All data were anonymized to ensure patient confidentiality, and ethical guidelines for retrospective studies were adhered to throughout the research process.

3. RESULTS

3.1. Age of patients

Table 1: Shows graphical representation of age groups.

Age	No. of patients	Percentage %
18 – 30	28	28 %
31-45	63	63 %
46-70	9	9 %
Total	100	100%

According to the data, we found that Alcoholic Liver Disease was most prevalent in the age group 31 – 45.

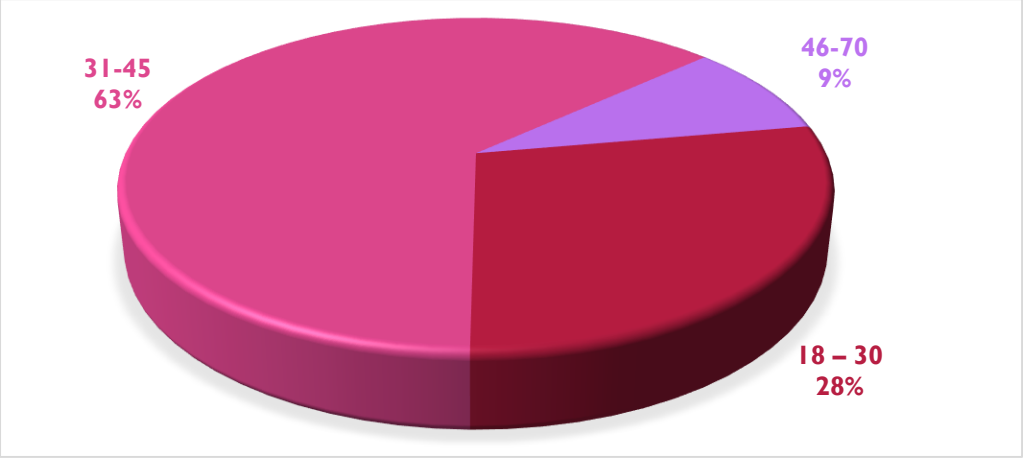

Fig 1: Shows graphical representation of age groups.

3.2. Area wise distribution

Table 2. Shows the ratio of Area of the Patient taking part in the survey.

Area	No of patient	Percentage %
Urban Area	32	32 %
Rural Area	68	68 %
Total	100	100 %

According to the Data, we found out Alcoholic Liver Disease Patients mostly come from the rural area.

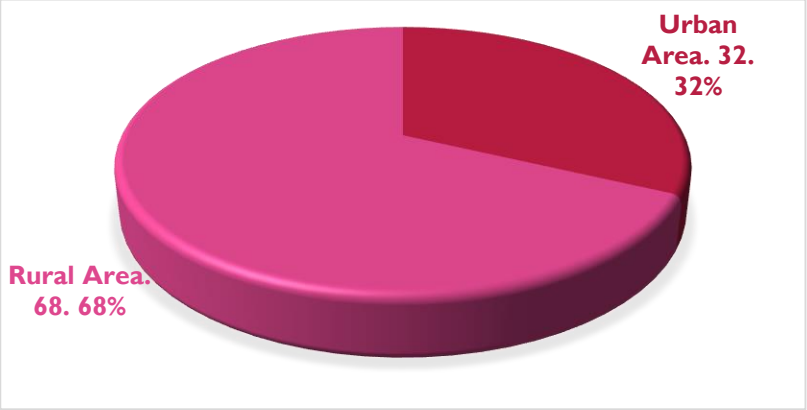

Fig 2: Shows the ratio of Area of the Patient taking part in the survey.

3.3. Gender categorization

Table 3: Shows the ratio of males and females taking part in the survey.

Gender	No. of patient	Percentage%
Male	91	91 %
Female	9	9 %
Total	100	100 %

According to the Data, we found out Alcoholic Liver Disease is more common in males as compared to females.

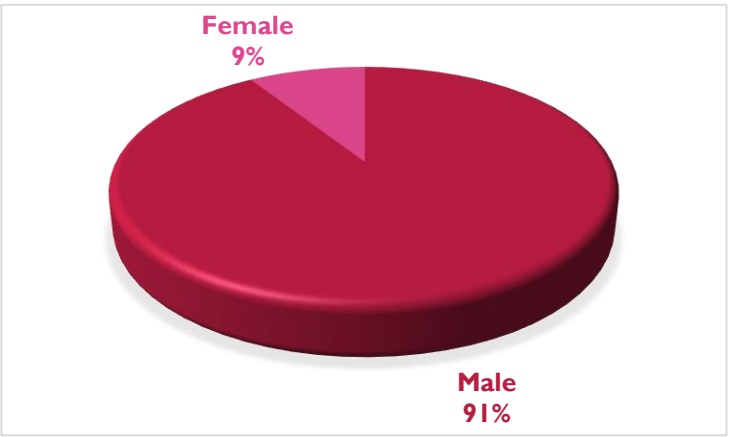

Fig 3: Shows the ratio of males and females taking part in the survey.

3.4. Comorbidities wise distribution

Table 4: Shows the ratio of comorbidities in sample of the survey.

Comorbidities	No. of Patient	Percentage
Diabetes	30	30 %
Hypertension	70	70 %
Total	100	100 %

ccording to the Data, we found that Hypertension is more common than Diabetes.

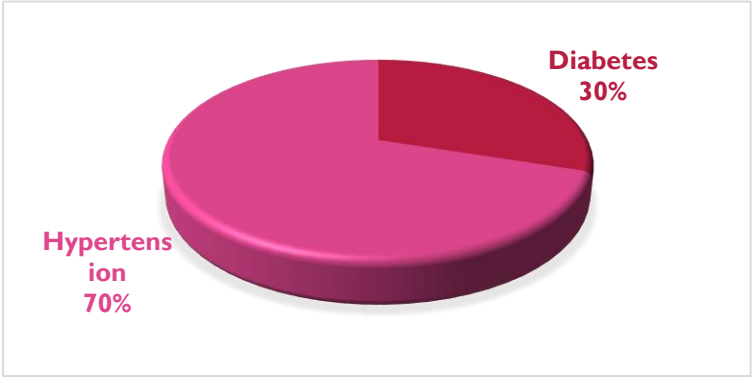

Fig 4: Shows the ratio of comorbidities in sample of the survey.

3.5. Number of Daily drinks

Table 5: Shows a ratio of patients as per number of daily drinks

Daily Drinks	No. of Patient	Percentage
1 – 7 Pegs	71	71 %
8 – 14 Pegs	26	26 %
15 and above	3	3 %
Total	100 %	100 %

According to the Data, we found that most of the patients had 1-7 pegs per day.

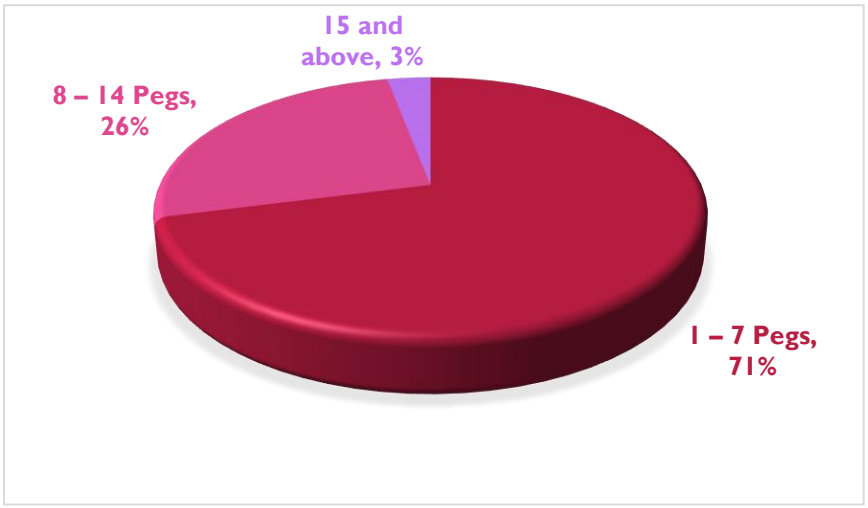

Fig 5: Shows a ratio of patients as per number of daily drinks

3.6. Treatment of Alcoholic Liver Disease

Table 6: Shows a graphical representation of Treatment of Alcoholic liver disease.

Drugs	No. of Patient	Percentage
Inj. Cefotaxime	16	16 %
Inj. Pantoprazole	75	75 %
Inj. Ondansetron	75	75 %
T. Spironolactone	95	95 %
C. Rifaximin	94	94 %
Inj. H. Albumin	81	81 %
T. Ursodeoxycholic acid	97	97 %
T. Folic Acid	93	93 %
Inj. Vitamin B 12	76	76 %
Syp. Lactulose	89	89 %
Inj. Ceftriaxone	72	72 %
Inj. Furosemide	71	71 %
Inj. Metronidazole	18	18 %
Inj. NS	4	4 %
Inj. Vitamin K	8	8 %
Inj. Multivitamins	5	5 %
Inj. Paracetamol	6	6 %

According to the data, the most used drug in alcoholic liver disease is Ursodeoxycholic acid followed by Spironolactone, Rifaximin. Conversely, the least used drug was normal saline.

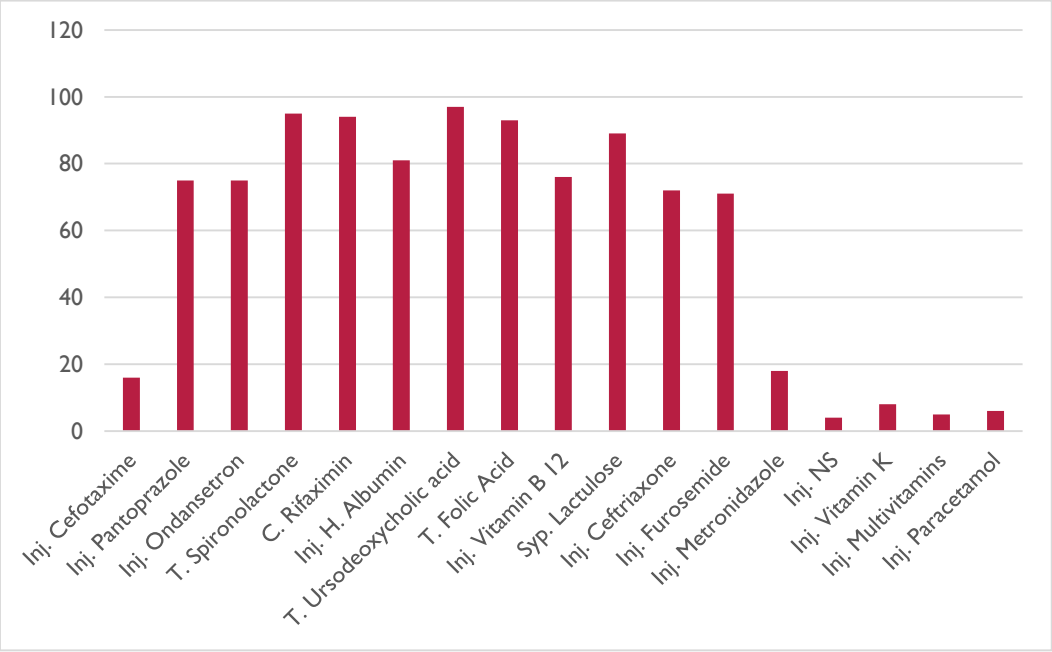

Fig 6: Shows a graphical representation of Treatment of Alcoholic liver disease.

3.7. Duration of ALD treatment

Table 7: Shows a graphical representation of duration of ALD treatment

Duration	No. of Patient	Percentage
7 Days	29	29 %
15 Days	48	48 %
1 Month	4	4 %
2 Month	6	6 %
3 Month	6	6 %
6 Month	4	4 %
2 Year	2	2 %
5 Year	1	1 %
Total	100	100%

According to the data, we found that patients have been receiving treatment for ALD since the past 15 Days.

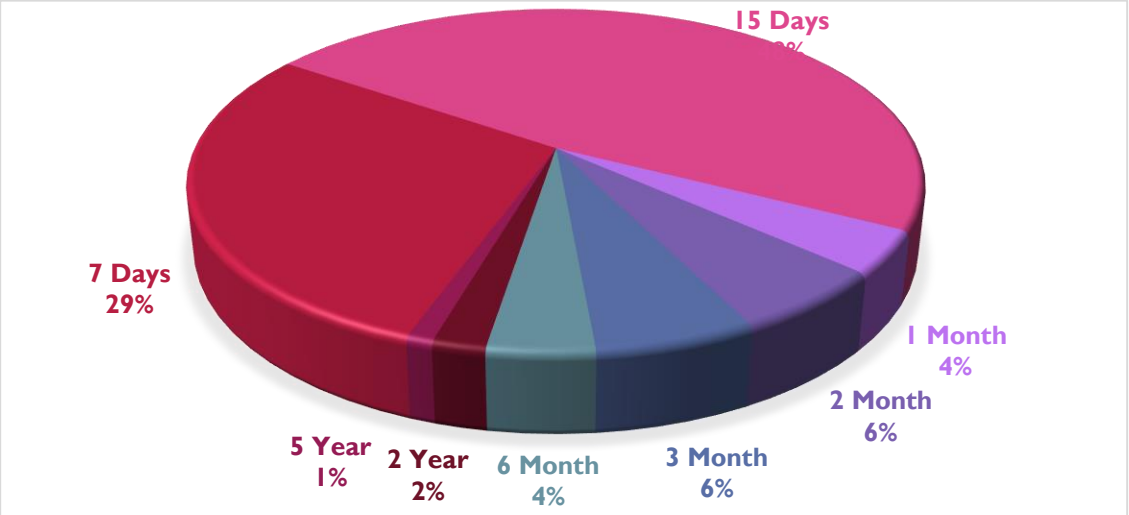

Fig 7: Shows a graphical representation of duration of ALD treatment

3.8. Drugs for comorbidities

Table 8: Shows a graphical representation of drugs for comorbidities

Drugs of Disease	No. of Patient	Percentage
Dapagliflozin	2	2.10 %
Sitagliptin	2	2.10 %
Metformin	25	26.31 %
Amlodipine	42	44.21 %
Atenolol	18	18.94 %

Telmisartan	15	15.78 %
Glipizide	3	3.15 %

According to the Data, we found that Amlodipine and Metformin were most used medication other than those medicine used in Alcoholic liver disease.

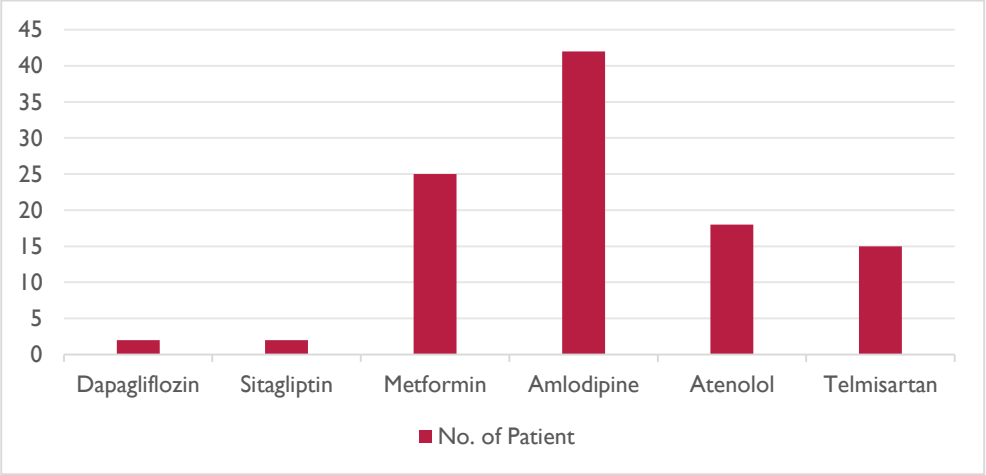

Fig 8: Shows a graphical representation of drugs for comorbidities

3.9. Symptoms During Treatment

Table 9: Shows a graphical representation of symptoms countered during treatment

Symptoms	**No. of Patient**	**Percentage**
Fatigue	26	31.70 %
Abdominal Pain	28	34.14 %
High Blood Pressure	19	23.17 %
Weakness	22	26 .82%
Leg Pain	22	26.82 %
Muscle Stiffness	18	21.95 %
Vertigo	2	2.34 %
Blur Vision	2	2.34 %
Loss of appetite	5	6.09 %
Constipation	8	9.75 %
Nausea	5	6.09 %
Body Pain	9	10.97 %
Chest pain	3	3.65 %
Back pain	5	6.09 %

According to the data, most of the patients experienced Abdominal pain, followed by fatigue, weakness, and leg pain. Conversely, only 2 patients had vertigo and blurred vision.

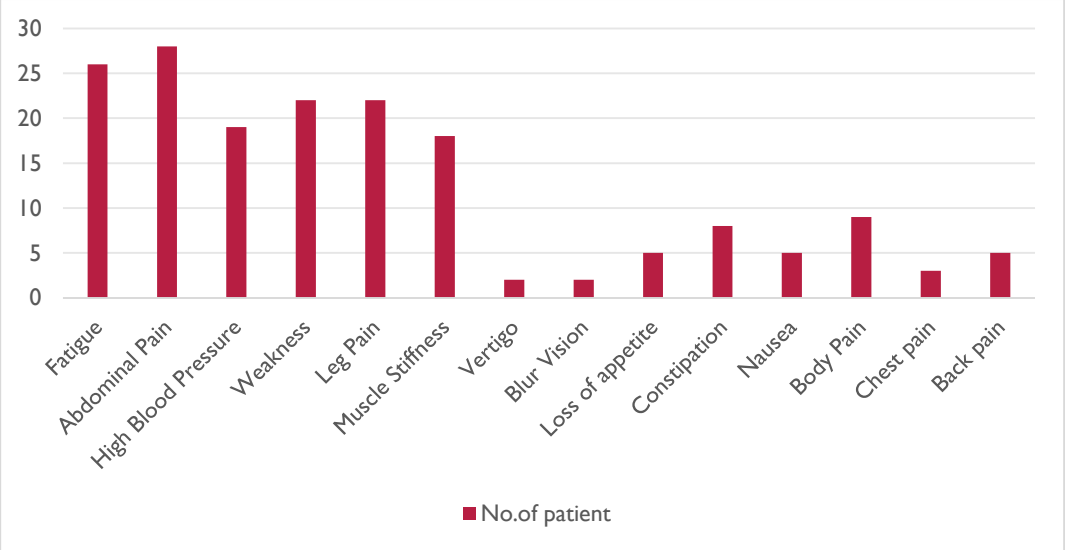

Fig 9: Shows a graphical representation of symptoms countered during treatment.

3.10. Adherence to specific diet alongside treatment.

Table 10: Shows a graphical representation of the number of patients adhering to specific diet alongside treatment.

Following Specific Diet	No. of Patient	Percentage
Yes	78	78 %
No	22	22 %
Total	100	100 %

According to the Data, we found that most of the Patients are following a specific diet

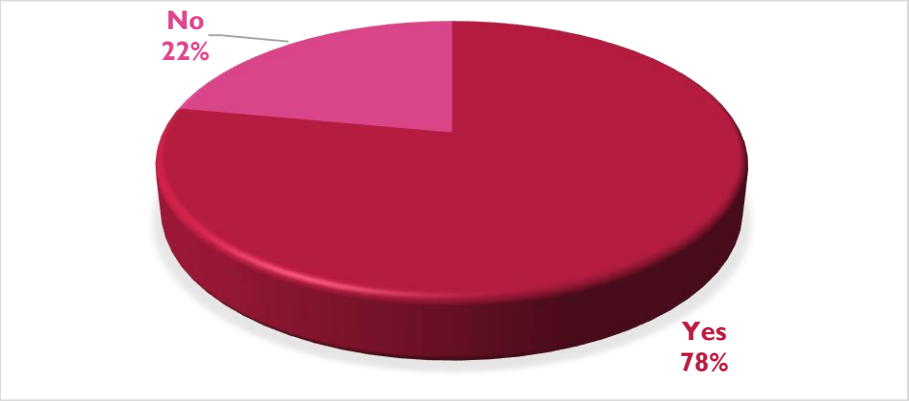

Fig 10: Shows a graphical representation of number of patients adhering to specific diet alongside treatment.

3.11. Affected Daily Life

Table 11: Shows a graphical representation of Affected daily life

Affected Daily Life	No. of Patient	Percentage
Fatigability	65	65 %
Anger	84	84 %

Lack of Attention to Work	18	18 %
Loss of Appetite	29	29 %
Irritability	58	58 %
Fight with Family and Friends	42	42 %
Protein Calories Malnutrition	25	25 %

According to the data, after alcohol addiction, majority of patients experienced anger followed by fatigue and irritability.

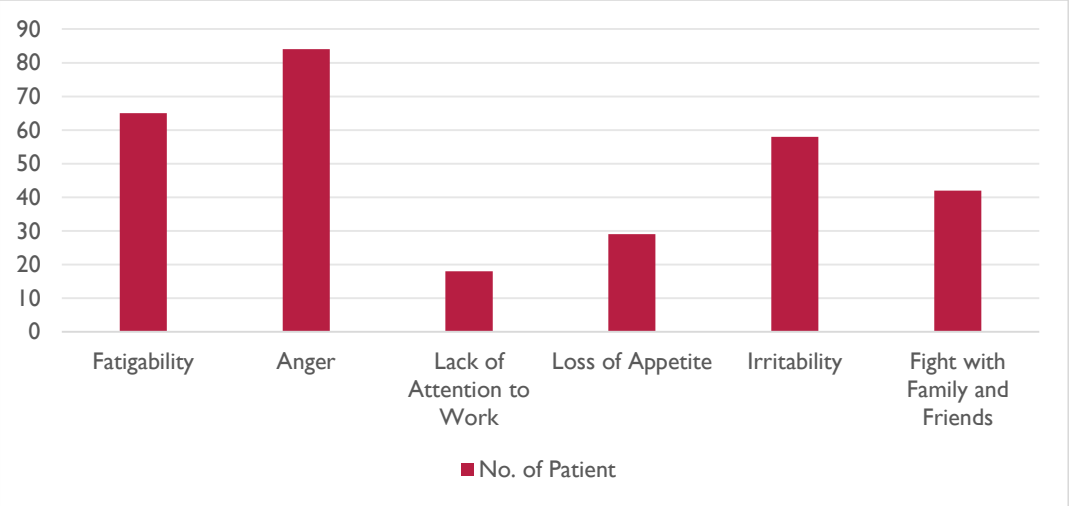

Fig 11: Shows a graphical representation of Affected daily life

4. DISCUSSION

This study highlights key demographic and clinical trends in ALD within Indian population with rural population being afflicted the most. The predominance of ALD in males aged 31–45 aligns with global patterns of alcohol consumption peaking during productive adulthood. The rural skew may reflect socioeconomic factors, such as limited healthcare access, cultural norms, or occupational stressors influencing alcohol use. The higher prevalence of hypertension over diabetes among ALD patients underscores alcohol's role in exacerbating metabolic dysfunction, particularly blood pressure dysregulation. Frequent abdominal pain and fatigue as primary symptoms correlate with advanced hepatic inflammation and portal hypertension, while the low incidence of vertigo/blurred vision suggests fewer neurological complications in this cohort.

The widespread use of ursodeoxycholic acid and spironolactone reflects standard protocols for cholestasis and ascites management. However, limited prescriptions for lactulose and furosemide indicate potential underutilization of therapies for hepatic encephalopathy and fluid overload, warranting further evaluation. Short-term treatment duration (15 days) raises concerns about long-term adherence and outcomes, emphasizing the need for structured follow-up. Dietary compliance and withdrawal-related anger/fatigue highlight psychosocial dimensions of ALD management, stressing the importance of integrating mental health support. While abstinence remains central, the interplay of social determinants (e.g., rurality, gender roles) necessitates community-based interventions to reduce alcohol dependency and improve outcomes.

5. CONCLUSION

This study underscores ALD's disproportionate impact on young males, with hypertension as a key comorbidity. The rural population suffers the most from this ailment. Standard therapies like ursodeoxycholic acid and spironolactone dominate management, though gaps in addressing complications like encephalopathy persist. Short-term treatment durations and psychosocial withdrawal symptoms highlight challenges in sustaining recovery. Integrating nutritional support, mental health services, and tailored abstinence programs is critical. Addressing rural healthcare disparities and alcohol-related stigma through education and policy reforms could mitigate ALD burden. Future research should explore longitudinal outcomes and culturally adaptive interventions to enhance holistic care for this high-risk population.

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