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RESEARCH ARTICLE

A PROSPECTIVE STUDY ON CLINICAL PROFILES OF ALCOHOLIC LIVER DISEASE IN A TERTIARY CARE HOSPITAL

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Abstract

Background: There is a significant correlation in development of alcoholic liver disease with type of alcoholic beverage consumed. Country made alcoholic beverage showed a significant development of complications i.e. ascites and encephalopathy and poor prognosis[1].

Objective: To study on Clinical profiles of Alcoholic Liver Disease in tertiary care hospital.

Method: A hospital based observational, Prospective study on clinical profiles of ALD in tertiary care hospital for 6months duration in General medicine Department. Based on diagnosis, the study population was interviewed after obtaining written informed consent for assessing the severity, its treatment response, complications and prognosis of ALD using laboratory parameters.

Results And Discussion: In this prospective study, the clinical profiles of Alcoholic liver diseases in a tertiary care hospital was evaluated and determined in sixty patients. Among Sixty patients of ALD 95% patients were male and 5% patients were female. The most common age group was between 36-45yrs. Almost 50% of patients with ALD consume 90ml of Alcohol on daily basis. ALD positive were treated majorly with Ursodeoxycholic acid,L-Ornithine L-Aspartate,Thiamine,Chlodiazepoxide, Rifaximin and Thiamine. majority of patients had MELD score ranging between 10-19.Patients were monitored regularly and scores were recalculated accordingly.

Conclusion: Management of ALD relies on abstaining from alcohol while treating alcohol withdrawal, providing nutritional support, and managing cirrhosis-related complications. Patients with severe alcoholic hepatitis who fail medical therapy have very poor outcomes. Ascites has been the major complication of Alcoholic liver disease. spironolactone is the drug of choice for the initial treatment. Ursodeoxycholic acid was used in most number of patients(53.33%) in treatment followed by thiamine (31.66%). MELD score will be useful as a template to improve upon as an objective gauge of disease severity and assess risk of mortality.

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Introduction:-

Alcoholic liver disease one of the main causes of chronic liver disease worldwide and accounts for up to 48% of cirrhosis-associated deaths in the united state.

Excessive alcohol intake can lead to fatty liver, hepatitis and cirrhosis. Alcoholic hepatitis is characterized by acute or chronic inflammation and parenchymal necrosis of the liver induced by Alcohol. Alcoholic hepatitis is often a reversible disease but the most common precursor of cirrhosis in the United States. It is associated with four to five times the number of hospitalization and deaths as hepatitis C, which is the second most common cause of cirrhosis.

The frequency of alcoholic cirrhosis estimated to be 10-15% among persons who consume over 50 g of alcohol (4 oz of 100-proof whiskey, 15 oz of wine, or four 12-oz cans of beer) daily for over 10 years (although the risk of cirrhosis may be lower for wine than for a comparable intake of beer or spirits).the risk of cirrhosis is lower (5%) in the absence of other co-factors such as chronic viral hepatitis and obesity. Genetic factors may also account for differences in susceptibility to and severity of liver disease. Women appear to be more susceptible than men, in part because of lower gastric mucosal alcohol dehydrogenase levels. [2,3]

Complications of excessive alcohol consumption stem largely from excess hydrogen and from acetaldehyde. Hydrogen produces fatty liver and hyperlipidemia, high blood lactic acid, and low blood sugar. The accumulation of fat, the effect of acetaldehyde on liver cells, and other factors as yet unknown lead to alcoholic hepatitis. The next step is cirrhosis. The consequent impairment of liver function disturbs blood chemistry, notably causing a high ammonia level that can lead to coma and death. Cirrhosis also distorts liver structure, inhibiting blood flow, high pressure in vessels supplying the liver may cause ruptured varices and accumulation of fluid in the abdominal cavity. Response to alcohol differs among individuals, in particular, not all heavy drinkers develop hepatitis and cirrhosis.

Beverages:

Illustration of “standard drinks” in order of increasing ethanol content among currently available alcoholic beverages. According to the National Institute on Alcohol Abuse and Alcoholism, The amount of beverages containing approximately 14g of pure ethanol is defined as a standard drink.

The percent of pure alcohol, expressed as alcohol by volume (alc/vol),varies by beverages.Thus,12 ounces (360ml) of beer at 6 percent alc/vol,5 ounces (150ml)of wine at 12 percent alc/vol ,or 1.5 ounces (45ml)of distilled spirits at 40 percent alc/vol each are equivalent to a standard drink. Although the standard-drink amounts are helpful for following health guidelines, they may not reflect customary serving sizes. In addition, although the alcohol concentrations listed are typical, there is considerable variability in actual alcohol content within each type of beverages.[6]

Treatment

General measures:

Abstinence from alcohol is essential. Naltrexone, baclofen may consider with counseling to reduce the likelihood of recidivism. Fatty liver is quickly reversible with abstinence. Every effort should provide sufficient amount of carbohydrates and calories in anorectic patient to reduce endogenous protein catabolism, promote gluconeogenesis, prevent hypoglycemia. Nutritional support (30-40[no less than 21.5] kcal/kg with 1.0-1.5 g/kg as protein) improves liver disease, but not necessarily survival, in patient with malnutrition. The administration of micronutrients, folic acid,thiamine,zinc is indicated, when deficiencies are noted, glucose administration increases the thiamine requirement and can precipitate wernicke-korsakoff syndrome if thiamine is not co administered .nephrotoxic drugs should be avoided in severe Alcoholic hepatitis.[7]

Pharmacolgical Measures:

Methylprednisolone, 32 mg/day orally for 1 month may reduce short term (1-month but not 6-month) mortality with AH and encephalopathy or maddrey discriminate function index (defined by prothrombin time minus control prothrombin time times 4.6 plus the total bilirubin in mgldL) of 32 or more, or MELD score of 18 or more. Concomitant GI bleeding or infection not preclude treatment with corticosteroids if otherwise indicated, but treatment with prednisolone increase risk of serious infections during and after treatment is completed. Prophylactic antibiotic therapy is under study.

The combination of corticosteroid and N- acetylcystine reported to improve 1month not 6 month survival and reduce risk of hepatorenal syndrome and infections, combination superior to corticosteroid alone.

Pentoxifylline, 400 mg orally three times daily for 4 weeks decrease the risk of hepatorenal syndrome. The addition of pentoxylline to prednisolone does not improve survival but reduce frequency of hepatorenal syndrome compare with prednisolone alone.[8,9]

Prognosis:

Concomitant GI bleeding does not worsen survival. Failure of serum bilirubin level to decline after 7 days of treatment with corticosteroids predicts non response and poor long-term survival as does the LILLE MODEL (which includes age, serum creatinine serum albumin prothrombin time (or INR) serum bilirubin on admission and serum bilirubin on day7 .[10,11]

The MELD score used for cirrhosis and GLASGOW alcoholic hepatitis score (based on Age, WBC count, high BUN, thrombin time ratio and bilirubin levels.

Nutritional Support:

Several factors contribute to the malnutrition that is common in chronic alcoholics with liver disease :

1. Alcohol can replace food in the diet of moderate and heavy drinkers, displacing the intake of adequate calories and nutrients, In light drinkers it is usually an additional energy source, also called empty calories. Although alcohol yields 7.1kcal/g, when it is consumed in large amounts it is not used efficiently as a fuel source. when individuals consume alcohol on a regular basis but do not fulfill criteria for alcohol abuse, they are often overweight because of increase in calories from alcohol. This is different from the heavy drinker who replaces calories with alcohol.
2. In the alcoholic impaired, digestion and absorption are related to pancreatic insufficieny, as well as morphologic and functional alterations of the intestinal mucosa. Acute and chronic alcohol intake impairs hepatic amino acids and synthesis into proteins, reduces protein synthesis and secretion from the liver, and increase catabolism in the gut.
3. Use of lipids and carbohydrates is compromised. And excess of reduction equivalents (eg. NADPH) and impaired oxidation of triglycerides result in fat deposition in the hepatocytes and an increase in circulating triglycerides. Insulin resistance is also common among alcoholics.
4. Vitamin and mineral deficiencies occur in alcoholic liver disease as a result of reduced intake and alterations in absorption, storage and ability to convert the nutrients to their active forms. Steatorrhea resulting from bile acid deficiency is also common in alcohol liver disease affecting fat-soluble vitamin absorption. Vitamin A deficiency can lead to night blindness. Thiamin deficiency is the most common vitamin deficiency in alcoholics and is responsible for Wernicke's encephalopathy. Folate deficiency can occur as a result of poor intake, impaired absorption, accelerated excretion, and altered storage and metabolism. Inadequate dietary intake and interactions between pyridoxal-5'-phosphate (active coenzyme of vitamin B6) and alcohol reduce vitamin B6 nutriture. Deficiency of all B vitamins and vitamins C,D, E, K is also common. Hypocalcemia, hypomagnesemia, and hypophosphatemia are not uncommon among alcoholics; furthermore, zinc deficiency and alterations in other micronutrients can accompany chronic alcohol intake.

Objectives:-

1. To study alcoholic liver disease and its clinical features .
2. To access the severity and its treatment response.
3. To evaluate the extent of complications and prognosis of ALD using laboratory parameters.

Methods And Methodology:-

Inclusion Criteria Includes:

1. Patient above 18 years.
2. Patients of both the genders.
3. Patients who are willing to give Informed consent.
4. Presence of severe concurrent diseases (cardio-respiratory,renal,hepatic,neurological disorders etc).

Exclusion Criteria:

1. Patients with less than 18 years of age.

2. Pregnant and lactating women.
3. Patients not expected to cooperate and comply with the treatment.
4. Immuno- compromised patients.

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Results:-

Table 1:- Distribution of patients based on gender.

S.NO	SEX	PATIENTS	PERCENTAGE
1	▪ Male	57	95
2	Female	3	5
3	Total	60	100

Figure 1:- Distribution of patients based on gender.

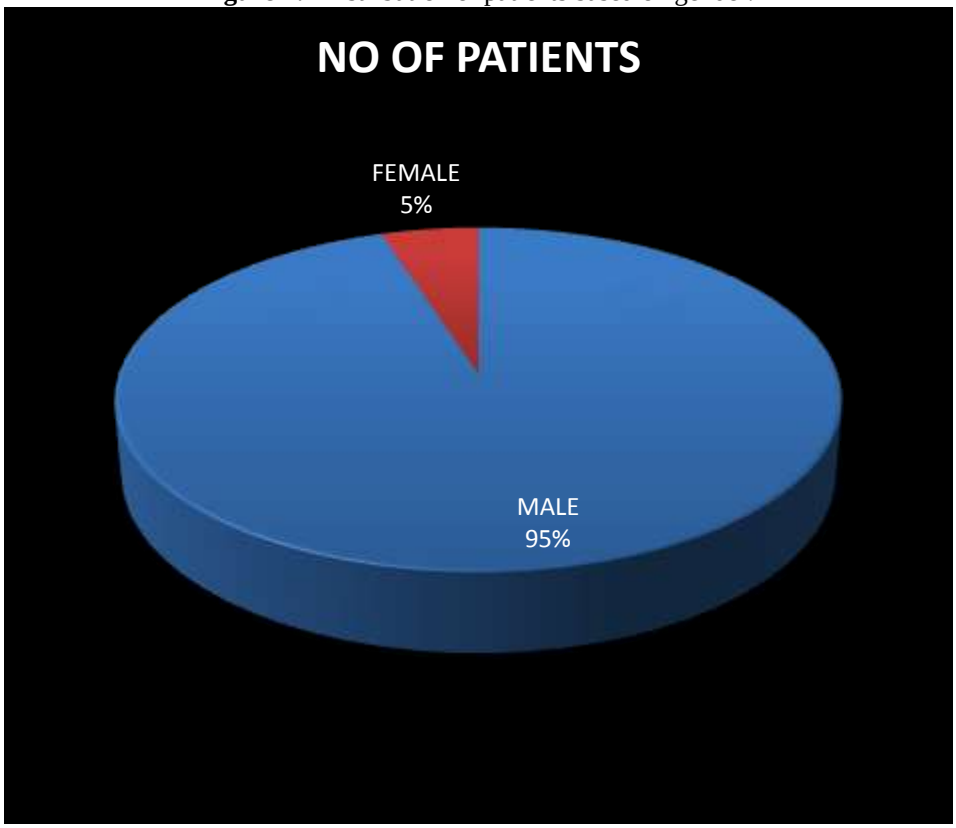


Table 2:- distribution of patients based on age group.

AGE(Yrs)	18-25	26-35	36-45	46-55		56-65
MALE	3	11	23	10	10	
FEMALE	0	0	1	0	2	

Figure 2:- distribution of patients based on age group.

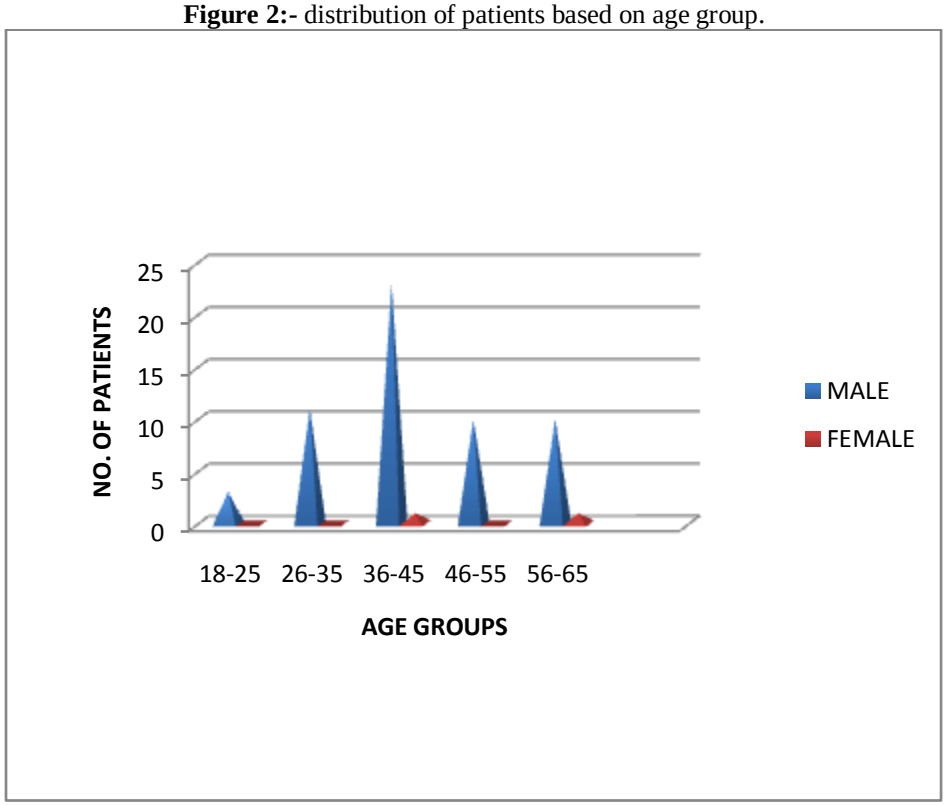


Table 3:- Distribution of patients based on diagnosis.

S.NO	DIAGNOSIS (USG)	NO. OF PATIENTS
1	Hepatomegaly with fatty changes	30
2	Splenomegaly	16
3	Jaundice	4
4	Calculus cholecystitis	1
5	Edematous gallbladder	4
6	Bulky pancreas	2
7	Mild hepatomegaly	7
8	Grade I fatty liver	6
9	Liver abscess	1
10	Grade III fatty liver	1
11	Hepatic encephalopathy	1
12	Acute pancreatitis	4
13	Ascites	12
14	Portal hypertension with esophageal varices	10
15	Liver cirrhosis	3
16	Grade II fatty liver	2
17	Alcoholic Hepatitis	9
18	Lymphadenopathy	1

Figure 3:- Distribution of patients based on diagnosis.

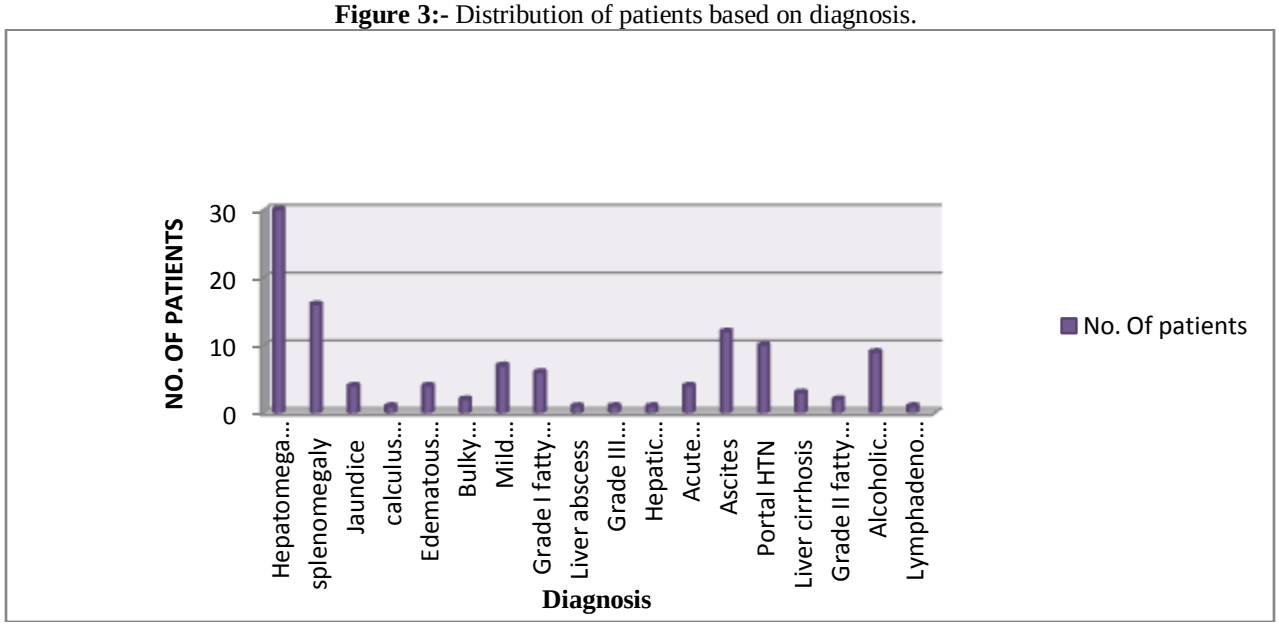


Table 4:- Distribution of patients based on complications.

complication	No.of patients
Jaundice	4
Hepatic Encephalopathy	1
Ascites	12
Portal hypertension with esophageal varices	10

Figure 4:- Distribution of patients based on complications.

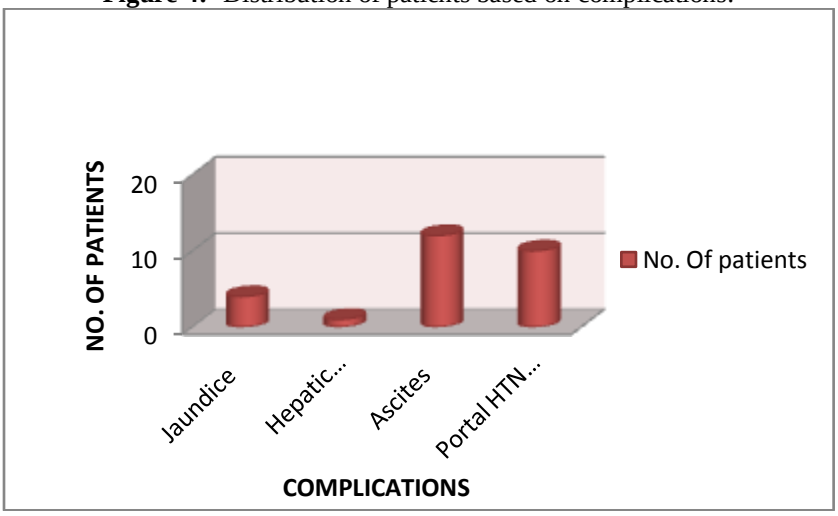


Table 5:- Distribution of patients based on Alcohol Consumption.

ALCOHOL CONSUMPTION		
S.no	**ml/day**	**No. of patients**
1	**Upto 90ml**	**30**
2	**90 – 180 ml**	**26**
3	**> 180ml**	**4**

Figure 5:- Distribution of patients based on Alcohol consumption.

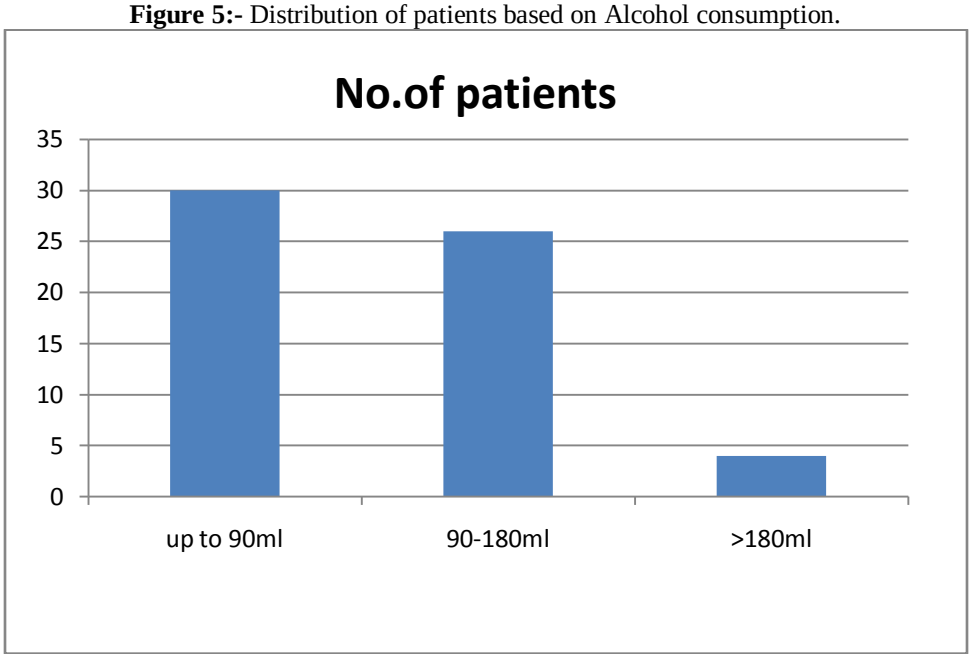
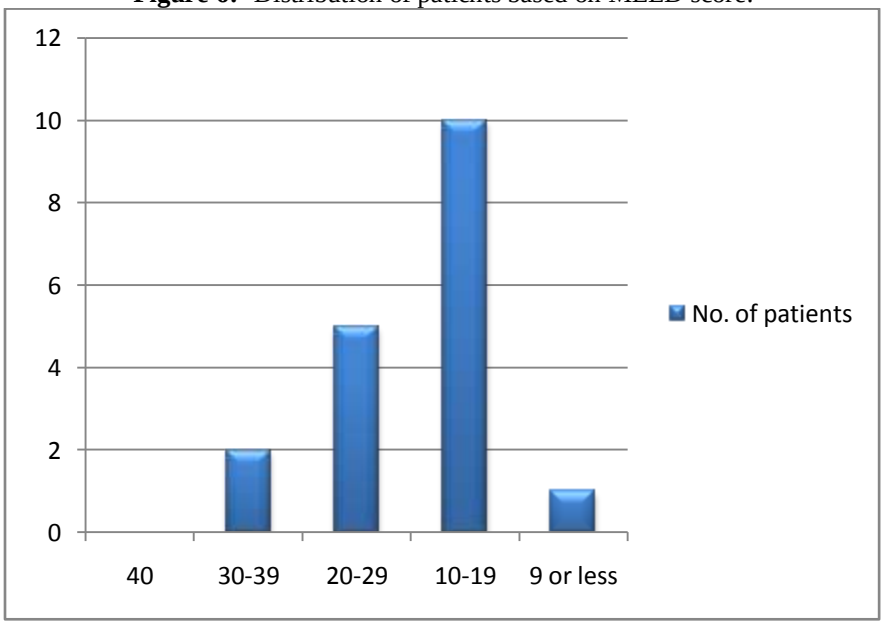


Table 6:- Distribution of patients based on MELD score.

MELD Score	No. of Patients
40	0
30-39	2
20-29	5
10-19	10
9 or less	1

Figure 6:- Distribution of patients based on MELD score.



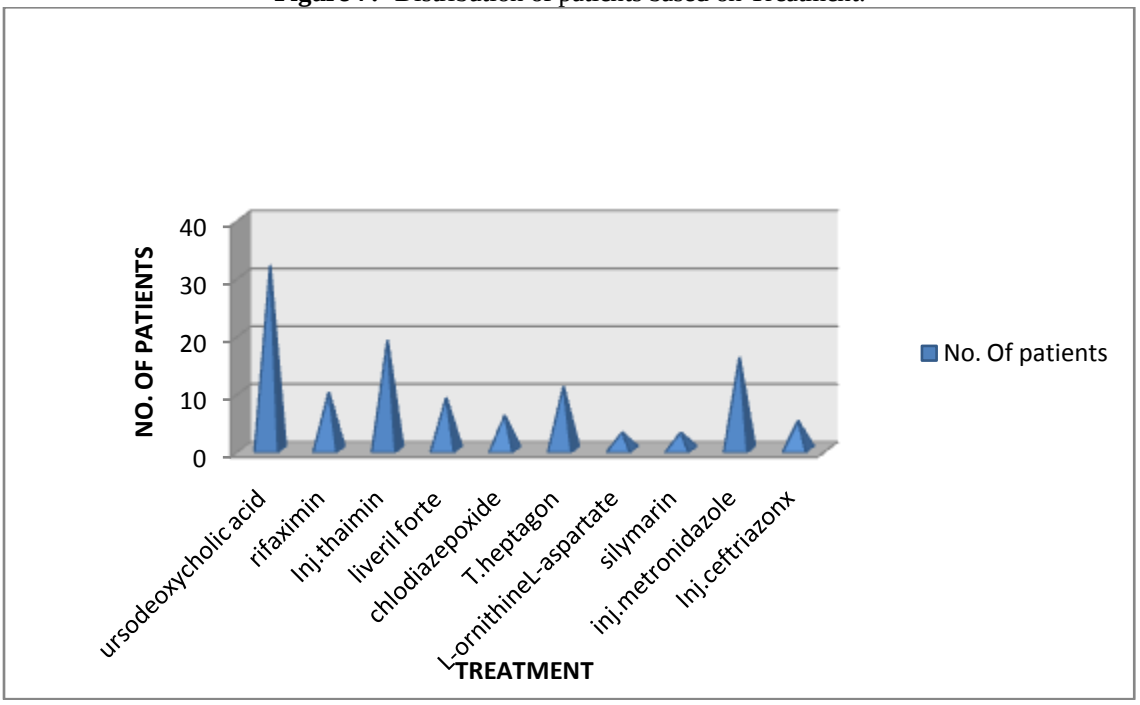
MELD Score	Mortality probability(%)
40	71.3
30-39	52.6

20-29	19.6
10-19	6.0
9 or less	1.9

Table 7:- Distribution of patients based on Treatment.

Treatment(drugs)	No. of patients		Percentage(%)
	male	female	
Ursodeoxycholic acid	29	3	53.33
Rifaximin	10	-	16.66
Inj.Thiamin	16	3	31.66
Liveril forte	9	-	15
chlordiazepoxide	6	-	10
T.Heptagon	10	1	18.33
L ornithine L aspartate	3	-	5
silymarin	2	1	5
Inj.Metronidazole	14	2	26.66
Inj.ceftriaxone	18	3	35

Figure 7:- Distribution of patients based on Treatment.



Conclusion:-

Excessive alcohol consumption is a global healthcare problem. The liver sustains the greater degree of tissue injury by heavy drinking because it is the primary site of ethanol metabolism. Chronic and excessive alcohol consumption produces a wide spectrum of hepatic lesions, the most characteristic of which are steatosis, hepatitis and fibrois/cirrhosis.

Major presenting complaints were Abdominal pain, fever, nausea and vomiting, yellowish discoloration of eyes and pedal edema in patients with ALD. On evaluating the Ultra sound abdomen and Liver functions tests such as Bilirubin levels, SGOT and SGPT, serum amylase and serum lipase levels, GGT ,PT and APTT it was confirmed that all patients had ALD. On enquiring for addiction in the present population, it was found that majority of them were smokers that is 21% of people with ALD has their personal history as smoking. Alcohol consumption varies from patient to patient .Almost 50% of patients with ALD consume 90ml of Alcohol on daily basis.

Management of ALD relies on abstaining from alcohol while treating alcohol withdrawal, providing nutritional support, and managing cirrhosis-related complications. Liver transplantation is the best available option for patients with alcoholic cirrhosis as long as they abstain from alcohol. Alcoholic hepatitis is a severe form of ALD associated with Hepatomegaly and Splenomegaly. Patients with severe alcoholic hepatitis who fail medical therapy have very poor outcomes. Ascites has been the major complication of Alcoholic liver disease. spironolactone is the drug of choice for the initial treatment. Ursodeoxycholic acid was used in most number of patients(53.33%) in treatment followed by thiamine(31.66%).

In our study we used The Model for End Stage Liver Disease or MELD, which is used for assessing the severity of chronic liver disease and prognosis in patients with liver cirrhosis. Majority of Patients (56%) were reported to have MELD score of 10-19 points. An increase in MELD score is associated with decrease in residual liver function.

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