Research Article

Clinical Profile in Diagnosed Cases of Alcoholic Liver Cirrhosis by Using Model of End Stage Liver Disease (MELD) Score

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Abstract

Alcoholic liver disorder divided into three types mainly alcoholic fatty liver ,acute hepatitis and alcoholic liver cirrhosis but study of complication of alcoholic liver cirrhosis always challenging for clinicians since many years for that many scores are recommended for explanation of prognosis of alcoholic liver cirrhosis but here we are using first time MELD score to study of clinical profile of alcoholic liver cirrhosis which initially used for sorting ALC patient for liver transplant and our study show significant relationship between MELD score and prognosis of ALC and compared with other 2 scores i.e Child pugh and Meddryes discrimination score.

Keywords: Alcoholic Liver Disease (ALD); Child Pugh score; Maddry's Discrimination Score; MELD score

Introduction

The World Health Organization (WHO) defines cirrhosis of the liver as a diffuse process marked by liver necrosis, fibrosis, and the transformation of normal liver architecture into structurally aberrant nodules lacking normal lobular organization. As liver is the major site of alcohol metabolism, it suffers the most tissue damage from heavy drinking; Alcoholic liver disease is caused by chronic and severe alcohol drinking and includes three types of disease:

- Alcoholic fatty liver (Steatosis)
- Alcoholic Hepatitis (AH)
- Fibrosis/Cirrhosis [1].

Steatosis develops in drinkers after consuming 4-5 standard drinks per day for up to a decade. Fatty liver can progress to alcoholic hepatitis after chronic use of alcohol, which is a more severe, inflammatory type of alcoholic liver injury. Alcoholic hepatitis can progress to Alcoholic Fibrosis or Cirrhosis (ALC), which is characterized by excessive extracellular matrix protein deposition. The fibrotic response starts with active peri cellular fibrosis, which can lead to liver cirrhosis, which is marked by severe liver scarring, vascular changes, and eventually liver failure [1].

20% of alcoholics who consume more than 70 drinks per week for more than 20 years develop Alcoholic Liver Disease (ALD), while 7% develop Alcoholic Liver Cirrhosis (ALC). Females require lesser drinks than males for the development of Alcoholic Liver Disease (ALD), (7-13 drinks per week for females versus 14-27 drinks per week for men). According to the National Institute on Alcohol Abuse and Alcoholism of United state defined the amount of alcoholic beverage containing approximately 14 gram of pure alcohol is defined as a standard drink, The percentage of pure alcohol, expressed as alcohol by volume (alcohol/volume), varies by beverage; According to the National Institute on Alcohol Abuse and Alcoholism of United state defined the amount of alcoholic beverage containing approximately 14 gram of pure alcohol is defined as a standard drink, The percentage of pure alcohol, expressed as alcohol by volume varies by beverage [2,3] [Table 1].

Table 1: Quantity of alcohol for standard drink in each beverages.

Beverages	Quantity Ounce (ml)	% Alcohol/Volume
Beer	12 (360ml)	5%-6%
Malt Liquor	09 (270ml)	7%
Wine	05 (150ml)	12%
Brandy	1.5 (45ml)	40%
Distilled Spirit	1.5 (45ml)	40%

Liver cirrhosis complications mainly divided in to two categories that is (A) Direct complications and (B) Indirect complications

- Direct complications
  - Jaundice
  - Hepatic Encephalopathy(HE),
  - Ascites
  - Upper Gastrointestinal bleeding (UGI bleeding), all these occurs due to loss of liver function and portal hypertension.
- Indirect complications includes
  - Hepatorenal Syndrome(HRS),

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- Spontaneous Bacterial Peritonitis(SBP)
- Hepatocellular Carcinoma (HCC) and infections [4].

In health-care settings, a variety of prognostic models are used. Some are generalized in use, while others are disease-specific. The Child-Turcotte-Pugh (CTP) score, the Model for End-stage Liver Disease (MELD) score, and Maddrey's Discriminant Function (MDF) are commonly used models in the treatment of patients with Alcoholic Liver Disease (ALD). The original MELD score is a prospectively established and validated chronic liver disease severity grading system that employs a patient's serum bilirubin, serum creatinine, and Prothrombin Time(PT), International Normalized Ratio (INR) to predict three-month survival (original MELD score). An increasing MELD score is linked to worsening hepatic dysfunction and a higher three-month mortality risk in individuals with cirrhosis.

MELD was created to predict 3-month mortality in patients receiving Trans Jugular Intrahepatic Portosystemic Shunt (TIPS) operation to treat refractory bleeding of esophageal varices Conversely, the MELD has been confirmed to be a valuable predictor of death in patients with End Stage Liver Disease (ESLD) without a TIPS procedure. Currently, the MELD is utilized to help predict short term mortality for patients with ESLD and prioritize organ allocation on the liver transplant waiting list [5].

The MELD is measured using total bilirubin, creatinine, an International Normalized Ration (INR). These laboratory values are calculated using an algorithm to produce a numeric score. Scores range from 6 to 40, with 40 being demonstrative of the most serious stage of end stage liver disease [6].

The original mathematical formula for MELD score [7].

3.78 x loge (Bilirubin in mg/dl)+11.2 loge (INR)+9.57 loge (Creatinine in mg/dl)+6.43

My study was conducted studied on 100 cases of alcoholic liver cirrhosis with an aim to evaluate the outcome of alcoholic liver cirrhosis by applying MELD score and to compare of MELD score with other scores for outcome of alcoholic cirrhosis like Child-Turcotte-Pugh (CTP) and Maddrey Discriminant Function (MDF) .

Materials and Methods

The production of clotting factors along with essential proteins and detoxification of harmful metabolic products with excretion of bile these all are functions of the liver. Chronic liver disease is defined as the progressive worsening of liver function for more than 6 month approximately. This is a continuous process of liver parenchyma Inflammation, destruction and regeneration that leads to fibrosis or cirrhosis. Once fibrosis develops, hepatocytes loses their ability to regenerate.

The etiology affects the pattern of liver fibrosis, In case of alcoholic liver disease cirrhosis seen at centrilobular and perivenular area with periportal distribution. Chronic hepatotropic viruses like HBV and HCV causes cirrhosis of liver by periportal fibrosis and bridging fibrosis , Biliary tract disease related liver cirrhosis shows specific pattern of feathery degeneration of periseptal hepatocyte leading to presence of prominent halos and irregular shaped nodules. A study by Sarin et al reported HBV as the commonest cause of cirrhosis in adults (44.3%) as well as the young (50.8%), followed by alcohol in both adults (35.8%) and the young (15.9%). A study by Ray et al., from eastern India including 175 patients with chronic liver disease reported HBV (35.4%), HCV (14.9%), autoimmunity (2.8%), Wilson's disease (2.8%) and alcohol (1.7%) as the commonest risk factors of cirrhosis [8].

Alcoholic Liver Disease (ALD) includes three stages of disease which includes

- Alcoholic fatty liver-occurs due to fat deposition in hepatocyte
- Alcoholic Hepatitis (AH)
- Alcoholic Liver Cirrhosis (ALC)

Epidemiology of alcoholic liver disease

Heavy alcohol consumption is particularly common among persons with poor educational levels and income, the jobless and those who work in jobs that are stressful and low-paying [8].

Many factors play key role in development of alcoholic liver cirrhosis like age, sex, race, nutritional deficiency, genetic factors and other associated chronic disease like Hepatitis B and C.

- Age-The most common age group for alcoholic hepatitis is 4th to 5th decade of life [9].
- Sex-The relative risk for development of alcoholic liver disease is more in women compared to men with increasing alcohol intake, This type of difference is due to several factors such as differences in gastric Alcohol Dehydrogenase (ADH) levels, and a higher proportion of body fat in women [10,11].
- Obesity-Long-term obesity in alcoholics is an independent risk factor for alcoholic liver disease and cirrhosis
- Malnutrition-The majority of AH patients are malnourished and the risk of death is directly proportional to the degree of malnutrition
- Hepatitis B and C-When alcohol and HCV are combined together, they increase the risk of cirrhosis and Hepatocellular Carcinoma (HCC), as well as a faster progression to fibrosis and cirrhosis and decreased life expectancy.

Results

Pathophysiology of Chronic Liver Disease (CLD)

The pathophysiology of Chronic Liver Disease (CLD) is based on the idea that an acute triggering event in a patient with chronic liver disease,which injures hepatocyte, triggers a cascade of inflammatory cytokines, which leads to subsequent hepatic injury in case of hepatocyte regeneration failure. This syndrome causes immune system impairment and hepatic decompensation ,which leads to increased infection risk, multiple organ failure, and death. Cirrhosis of the liver is an end-stage organic disease caused by immunological dysfunction caused by macrophage loss of antimicrobial detection and clearance and decreased

antigen presenting capacity by monocytes [12]. Albumin's anti-inflammatory, antioxidative, plasma expanding, and endothelium stabilizing properties are beneficial in patients with alcoholic liver cirrhosis who have spontaneous bacterial peritonitis and hepatorenal syndrome (a kind of acute injury to kidney) (Tables 2 and 3)

Table 2: Clinical feature of liver cirrhosis.

Clinical findings	How to examine	Cause for clinical findings
Jaundice	Yellow discoloration of sclera and mucus membrane	Serum total bilirubin >2mg/dl
Cruveilhier Baumgarten syndrome	Epigastric vascular murmur by auscultation	Development of shunts from portal vein to umbilical vein branches
Caput medusa	Dilated vein around umbilicus	As a result of portal vein hypertension reopening of the umbilical vein causes blood to shunt from the portal vein.
Palmer erythema	Redness of palm with sparing of central area	Increased estradiol due to decreased it degredation
Splenomegaly	By palpitation or by ultra sonography	Portal hypertension
Ascites	By palpitation or by ultra sonography	Portal hypertension and low serum albumin
Spider angioma	Central arteriole with radiating vessel	Increased estradiol due to decreased it degredation
Flapping tremor (asterixis)	Jerky movement of outstretched palms	Increased serum ammonia leads to disinhibition of motor neurons
Gynecomastia	Holding nipple around aerola area in hand pinch	Conversion of androstenedione to estrone and estradiol and decrease in estradiol breakdown
Anorexia, Fatigue and weight loss	By asking proper history	Due to increased catabolic phenomenon & decreased appetite
Dupuytren's contracture	Contracture and fibrosis palmer fascia	Due to increased oxidative stress
Nodular liver	Irregular liver on palpation	Due to irregular Fibrosis
Finger clubbing	Softening of nail bed	Hypoxia ,Porto-pulmonary hypertension
Diabetes mellitus	By measuring blood sugar level	Decresed use of glucose by liver
Testicular atrophy	Palpating scrotum	Due to increased estradiol

Table 3: Laboratory finding of liver cirrhosis.

Laboratory parameters	Significance	Etiology
AST AND ALT	May be normal or elevated Ratio of AST/ALP >2 in ALD	Leakage from damaged hepatic cells
Bilirubin	Elevated (Important indicator of mortality)	Decreased hepatic andrenalexcretion(exacerbated by systemic inflammatory reaction
Albumin	Decreased	Sequestration into ascetic fluid and interstitium (exacerbated in systemic inflammatory reaction) and decreased hepatic production
Prothrombin time	Decreased	Decreased production of clotting factor V,VII and Vitamin K also
Sodium imbalance	Hyponatremia	Decreased water excretion by kidney due to enhanced activity of antidiuertic hormone
Anemia	Macro or microcytic	Folate and vitamin B -6,B-12 deficiecy ,UGI bleed
Alkaline phosphatase	Elevated but <3 times normal Value	Cholestasis
Albumin/ globulin ratio	Reversed in liver cirrhosis (Normal value=1.5-3/1)	Decreased albumin production by liver cells

Complications of Chronic Liver Disease (CLD)

Common complication of alcoholic liver cirrhosis were

- Ascites (70%-74%)
- Hepatic encephalopathy (HE) (59%-60%)
- Upper gastro intestinal bleed (UGI bleed) (59%-60%)
- Hepatorenal syndrome(HRS)(35%-38%),
- Hepatopulmonary syndrome (HRS)(18%-20%)

Common symptoms seen during alcoholic liver cirrhosis are

- Abdominal pain(55%-60%) and distension(78%)
- Jaundice(60%)
- Ascites(72%-74%)
- Icterus(60%-62%)
- Pedal oedema (60%-64%) are common clinical signs.

Scores used for prognosis of chronic liver diseases used in this study

- Child Pugh score (CP score)
- Model of End Stage Liver Disease Score (MELD)
- Maddrey's Discriminant Function (MDF)

Child Pugh score (CP score)

CTP score includes five factors that is Ascites, Encephalopathy, Serum bilirubin, Serum albumin and Prothrombin Time (PT) and assigned score for each variable from 1 to 3 and Chronic Liver Disease (CLD) is classified into CTP classification in to three category A (Score up to 6), B (7 to 9) and C (10-15), using the final score derived from variables , with the resulting classes having different prognostic implications as shown below (Table 4).

Table 4: CTP score and survival.

Score	Class	1 Year survival	2 year survival
5-6	A	100%	85%
07-09	B	81%	57%
10-15	C	45%	36%

Model of End Stage Liver Disease Score (MELD)

Patients with end-stage liver disease who are candidates for Liver Transplant (LT) had a linear relationship between mortality and MELD score uses the patient's values for Serum bilirubin, Serum creatinine, and International Normalized Ratio for prothrombin time (INR) to predict survival. It is calculated according to the following formula

3.78 x loge (Bilirubin in mg/dl)+11.2 loge (INR)+9.57 loge (Creatinine in mg/dl)+6.43

According to this modified score, patients with bilirubin and creatinine values below 1 mg/dL are rounded off to 1 mg/dL to avoid negative logarithmic values. Patients with an INR of less than 1 are also rounded up to one. The score is empirically capped at a value of 40, regardless of the individual values. As a result, MELD score is a continuous variable with a range of 6 to 40. From 2002, the MELD score has been used to allocate organs to patients awaiting liver transplantation .Patients with the highest MELD score have priority for organ allocation under the MELD-based policy (Table 5).

Table 5: MELD score and survival.

Score	Mortality at 3 months
<9	4% mortality
Oct-19	27% mortality
20-29	76% mortality
30-39	83% mortality
40 or >	100% mortality

Maddrey's Discriminant Function (MDF)

The Maddrey Discriminant Function (MDF) score is a measure of disease prognosis in Alcoholic Hepatitis (AH) that is used to identify individuals with the highest risk of mortality and to determine when pharmacologic therapy should be initiated. Patients with severe AH, defined as a modified Maddrey's discriminant function (mDF>32), have a high short-term death rate and may benefit from certain medications that improve short-term survival, such as corticosteroids. Pharmacologic therapy, on the other hand, is not indicated for less severe patients (mDF<32), particularly those without hepatic encephalopathy, because they are unlikely to benefit from it.Patients with AH with an MDF<32 have a 1-month fatality rate of less than 10% and are usually not evaluated for specific therapy.

Benefits of MELD score-MELD score offers convincing advantages over Child–Pugh. MELD score relies on a triad of simple and objective biological variables, which facilitates comparisons between populations. These biological variables, in contrast to Ascites and encephalopathy are not influenced by individual judgment and may only be slightly altered by external factors. The score itself is a continuous variable which helps ranking appropriately and precisely patients within large populations. It proven to be a reliable prognostic indicator across a wide range of cirrhosis severity and etiology. The addition of a renal function marker in the score is likely one of the deciding factors in the MELD score's superiority over the Child–Pugh score in some domains. Moreover, the MELD measure has been prospectively confirmed in the context of organ allocation prioritization and post-TIPS survival. Its capacity to forecast early death after listing is especially useful when liver transplant allocation is involved.

Limitations of MELD score-All the variables in the MELD model were chosen empirically, because they were thought to have an impact on the result or because they were easily accessible. The three variables in the MELD score (Bilirubin, Creatinine, and INR) are objective variables depending on the methodologies used for determination, there are significant differences in INR amongst laboratories. The multiplicative value of INR is the highest of the three MELD score variables. As a result, fluctuations in INR might result in MELD score discrepancies of up to 20%. Significant variations in serum Creatinine can occur in people with cirrhosis, especially when serum bilirubin levels rise.

Discussion

Distribution of study subjects according to age (Figure 1)-Out of 100 cases studied Majority 32(32%) were in age group of 41-50 years followed by 31(31%) cases in age group of 30-40 years followed by 22(22%) in age group of 51-60 years .Mean age was 48.63 ± 10.63 years ranging between 32-76 years.

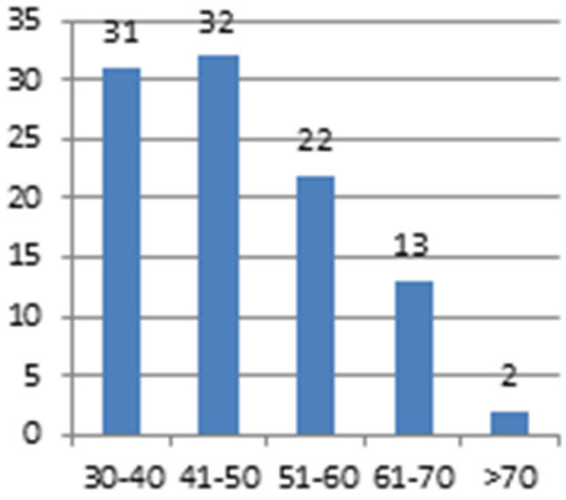

Figure 1. Age wise distribution; **Note:** (▪) percentage

Distribution of study subjects according to gender-All 100 (100%) cases were males as female who was coming to my hospital are non alcoholic.

Distribution of study subjects according to Child Pugh score (Figure 2)-Majority 52 (52%) cases were in class B of Child Pugh score followed by 39 (39%) in class C and rest 09(9%) were in class A of CTP score (Figure 2).

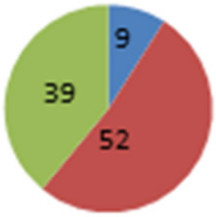

Figure 2. Distribution according to Child Pugh score; **Note:** (■) A, (■) B, (■) C

Distribution of study subjects according to complications (Figure 3)-There were multiple complications in cases and maximum 32 (32%) cases had hepatic encephalopathy as most common complication followed by refractory ascites in 29 (29%) cases. Hepato renal syndrome was seen in 22 (22%) cases (Figure 3).

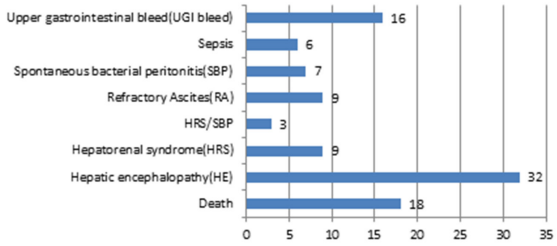

Figure 3. Distribution according to outcome; **Note:** (■) percentage

Distribution of study subjects according to final outcome (Figure 4)Out of 100 cases, 18 (18%) cases died during study period. Hepatic encephalopathy was final outcome in 32 (32%) cases. Hepatorenal syndrome and refractory ascites was seen in 09 (9%) cases respectively. Spontaneous bacterial peritonitis was seen in 07 (7%) cases (Figure 4).

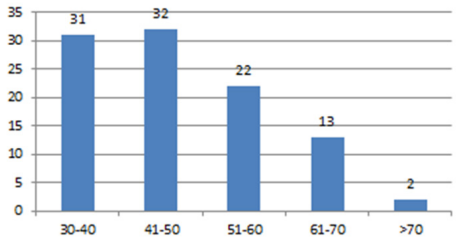

Figure 4. Distribution according to age and mean MELD score; **Note:** (■) n (%)

Distribution of study subjects according to age and mean MELD score (Figure 5)-Mean MELD score was maximum (24.22 ± 8.78) in age group of 51-60 years followed by 23.84 ± 8.71 in age group of 41-50 years followed by 21.25 ± 7.91 in age group of 30-40 years (Figure 5).

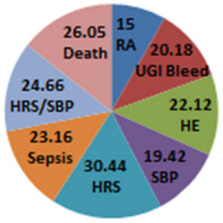

Figure 5. Distribution according mean MELD score; **Note:** (■) RA, (■) UGI bleed, (■) HE, (■) SBP, (■) HRS, (■) Sepsis, (■) HRS/SBP, (■) Death

Distribution of study subjects according to final outcome and mean MELD score (Table 6) When mean MELD values were compared in different final outcomes, it was found to be statistically significant. (p value=0.001) (Table 6), (Figure 5).

Distribution according to mean MELD score range and Final outcome (Table 7) Death was more in cases with MELD score between 21-30 followed by 11-20 score (Table 7).

Distribution of study subjects according to final outcome and Child Pugh score (Table 8). Out of 9 cases in class A,5 cases had UGI bleed,3 cases had refractory ascites and 1 with spontaneous bacterial peritonitis, Out of 52 cases of class B, 19 cases had hepatic encephalopathy and 10 had death. Out of 39 cases of class C, 13 cases had hepatic encephalopathy.

Distribution of study subjects according to final outcome in manner of death or alive and mean MELD score (Table 9)-When MELD scores in alive and deceased were compared by applying t test, it was found to be statistically significant. (p value=0.04)

Distribution of study subjects according to final outcome and mean Maddrey's discriminate function (Table 10)-Mean DF was not significantly different in alive and deceased patients.

Comparison of severity of score and mortality between MELD and CTP score (Table 11)-Only 2 mortality matched according to severity of both scores.

Table 6: Distribution of study subjects according to final outcome and mean MELD score.

Final outcome	Number	Mean MELD score	Standard deviation	t test P value
RA	9	15	4.63	
UGI bleed	16	20.18	7.22	
HE	32	22.12	8.03	
SBP	7	19.42	4.92	0.001*
HRS	9	30.44	10.46	
Sepsis	6	23.16	3.76	
HRS/SBP	3	24.66	5.85	
Death	18	26.05	7.99	

Table 7: Distribution according to mean MELD score range.

MELD score	RA	UGI Bleed	HE	SBP	Sepsis	HRS	HRS/SBP	Death	Total
05-10	1	2	0	0	0	0	0	0	3
11-20	7	6	17	5	2	1	1	5	44
21-30	1	7	12	2	4	3	2	8	39
31-40	0	1	2	0	0	4	0	4	11
>40	0	0	1	0	0	1	0	1	3

Table 8: Distribution of study subjects according to final outcome and Child Pugh score.

Final outcome	N	Child Pugh score		
		A (5-6) (n)	B(7-9) (n)	C(10-15) (n)
Refractory Ascites	9	3	4	2
UGI bleed	16	5	7	4
HE	32	0	19	13
Hepatorenal syndrome	9	0	2	7
SBP	7	1	3	3
Sepsis	6	0	5	1
HRS/SBP	3	0	2	1
Death	18	0	10	8
Total	100	9	52	39

Table 9: Quantity of alcohol for standard drink in each beverages.

Final Outcome	Number	Mean ± SD MELD score	T test p value
Alive	82	21.81± 8.10	0.04*
Dead	18	26.05 ± 7.99	

Table 10: Distribution of study subjects according to final outcome and mean Maddrey's discriminate function.

Final Outcome	Number	Mean ± SD MELD score	T test p value
Alive	82	54.044 ± 43.61	0.9
Dead	18	50.44 ± 37.21	

Table 11: Comparison of severity of score and mortality between MELD and CTP score.

Sr no	Duration of mortality from diagnosis in months	MELD score	CTP score	Comparison of mortality in two scores
1	2month	33	B	Not matched
2	4month	38	C	Matched
3	1month	43	C	Matched
4	2.5month	31	B	Not matched
5	1month	20	B	Not matched
6	5month	26	C	Not matched
7	3month	11	B	Not matched
8	1month	20	B	Not matched
9	1month	29	B	Not matched
10	2month	28	C	Not matched
11	1month	18	C	Not matched
12	2month	15	B	Not matched
13	1month	23	A	Not matched
14	1month	12	B	Not matched
15	1month	34	B	Not matched
16	1month	27	C	Not matched
17	1month	23	C	Not matched
18	2month	25	C	Not matched

Measures of central tendency for MELD score in study subjects (Table 12)-Central tendency for MELD score in study subjects. Mean, Median and mode MELD score was 22.58 ± 8.21, 22 and 18 respectively

Table 12: Measures of central tendency for MELD score in study subjects.

Measures of central tendency	MELD score values
Mean	22.58 ± 8.21
Median	22
Mode	18

Summary of study

Total 100 cases of alcoholic liver cirrhosis where studied during this study for 2 years at MGM hospital, Aurangabad, Maharashtra, India

- Mean age was 48.63+/- 10.63 years ranging between 32-76 years.
- All 100 (100%) cases where male.
- Majority 52% cases were in class B of Child Pugh score followed by 39 (39%) in class C
- Majority 51 (51%) cases had duration of illness between 1-4 month followed by 35% cases with 4-18 month
- There are multiple complication in case and maximum 32% cases had hepatic encephalopathy as most common complication followed refractory Ascites.
- Mean, Median and Mode of MELD score was 22.58+/- 8.21,22 and 18 respectively.
- Out of 100 cases 18 (18% cases) died during study period
- Mean MELD score was maximum (24.22+/- 8.78) in age group of 51-60 years followed by 23.84+/- in age group of 41-50 years
- When MELD score in alive and dead patient were compared by applying t test, it was found to be statistically significant. (p value=0.04)
- Mean DF was not significantly different in alive and dead patient.

Conclusion

The MELD score confirmed as predictor of survival in patient of alcoholic liver cirrhosis, Alcoholic hepatitis and alcoholic liver failure ,The said theme of MELD score in relation to degree of sickness is studied and analysed in present study. The clinical profile and MELD score quantile justified in the present study and does not show any discrepancy related to seriousness of complication of MELD score.

The mortality as stated by MELD quantile Criteria (31-40 and >40) is observed. In this quantile 5 patient died within period 2.1 month from diagnosis of alcoholic liver cirrhosis, Same 5 patient when observed by applying CTP score criteria (Class B and C) did not survived for period of 1 year, CTP score delineates survival rate of 2 year(Class B 57% and C-36%) and 1 year(Class B 81% and C-45%) respectively, Therefore it is suggested while evaluating any case of chronic liver disease including alcoholic liver cirrhosis ,for prediction of survival and mortality rate combined application of these system and there score should be adopted reason being the seriousness of illness with CTP score is symptoms complex based while MELD score is laboratory based.

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