IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 17, Issue 9 Ver. 13 (September. 2018), PP 58-61
www.iosrjournals.org

Clinical Profile and Etiology of Cirrhosis of Liver In Tertiary Care Hospital, AP.

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Abstract: ***Introduction:*** *Cirrhosis is defined by the World Health Organization (WHO) as a diffuse process characterized by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules.[3,4] The progression of liver injury to cirrhosis may occur over weeks to years. Chronic liver diseases and cirrhosis result in 26,000-35,000 deaths each year in the United States (US). Cirrhosis is the 9th leading cause of death in the US and is responsible for 1.2% of all US deaths.[5]*

Materials and Methods: *Three hundred consecutively diagnosed adult cirrhosis patients were prospectively studied at the Department of Gastroenterology, Kurnool Medical College and hospital, a tertiary care hospital of AP from June 2016- May 2017 for their clinical characteristics, prognosis and mortality at one month*

Results: *Commonest age group was 35-54years, mean age 45.8+ 10.4 years; M: F ratio 7.5:1. Symptoms were ascites (74.3%), gastrointestinal bleeding (43.4%), jaundice (36.3%), low urine output (31%) and altered sensorium in 23%. 37.1% patients had severe malnutrition. Aetiology were alcohol related (72.2%), HBV (8.9%), HCV (3.2%), Autoimmune Hepatitis (0.9%), Cryptogenic cirrhosis (17.2%) and NASH (1%). Complications were ascites (78.6%), variceal bleeding (43.4%), hepatic encephalopathy (21.6%), Spontaneous bacterial peritonitis 4.2%, Hepatorenal syndrome (2.7%) and Hepatocellular carcinoma (1.3%). 50% had Child C disease, 83% had MELD between 10-29 and APRI (AST to Platelet ratio index) >2.5 in 38.5% patients. Mortality was 7.8% and highest among alcoholic cirrhosis (6.8%).*

Conclusion: *Cirrhosis is common in the most productive age and the commonest cause was alcohol cirrhosis which is preventable through proper education and legislation. Proper awareness will lead to prevention of long term morbidity.*

Key words: *Cirrhosis, etiology, complications, prognosis, mortality*

Date of Submission: 15-09-2018 Date of acceptance: 29-09-2018

I. Introduction

Cirrhosis named by Laennec in 1826 means orange or twany in Greek.[1] Many forms of liver injuries are marked by fibrosis. This response to liver injury is potentially reversible. In contrast, cirrhosis is not a reversible process.[2] Cirrhosis is defined by the World Health Organization (WHO) as a diffuse process characterized by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules.[3,4] The progression of liver injury to cirrhosis may occur over weeks to years. Chronic liver diseases and cirrhosis result in 26,000-35,000 deaths each year in the United States (US). Cirrhosis is the 9th leading cause of death in the US and is responsible for 1.2% of all US deaths.[5]

Cirrhosis can be classified as follows: 1) alcoholic; 2) cryptogenic or post hepatic; 3) biliary; 4) cardiac; 5) metabolic 6) inherited and 7) drug related.[6] The clinical presentation of cirrhosis is variable depending on the aetiology and whether hepatocellular or portal hypertension predominates.[6] However, severe liver injury may be present without any clinical signs. The diagnosis of cirrhosis is based on the clinical features, laboratory investigations, histology and radiologically.

The main complications of cirrhosis are gastrointestinal haemorrhage, hepatic failure, hepatocellular carcinoma, and bacterial infection. The relative frequencies of these complications in different forms of cirrhosis are difficult to determine.[7] The profile of cirrhosis may vary with different age and ethnic groups, geographical, social and etiological factors. So we undertook this study to see the clinical profiles of patients with cirrhosis of liver in tertiary care hospital in AP.

II. Materials And Methods

A prospective observational study was carried out on 300 consecutively diagnosed patients of cirrhosis attending the outpatient department of Gastroenterology at Kurnool Medical College and Govt General Hospital from June 2016-May 2017. After informed consent, detailed history and clinical examination was done.

Relevant biochemistry including Complete blood count, liver function tests, renal function tests, serum electrolytes, fasting and post prandial blood sugar, serum ammonia etc were done. Abdominal ultrasound for liver and spleen size, parenchymal echogenicity, portal vein diameter, and ascites and other concomitant findings and CT/ MRI abdomen in suspected cases of liver cancer was done. Upper GI endoscopy was carried out in all eligible cases. Prognosis was measured by Child Turcot Pugh scores, MELD and APRI scores.

Patients were followed up during their hospitalisation and up to 1 month to determine survival. Variables were recorded in a predesigned proforma, analysed and compared with other studies.

Clinical cirrhosis was defined as a patient having at least one sign of hepatocellular failure, one of portal hypertension along with at least three ultrasound findings suggestive of cirrhosis of liver and or liver biopsy evidence of cirrhosis in permissible cases. Socioeconomic status was based on modified Kuppuswamy Socioeconomic scale. Nutritional assessment was done based on anthropometric measurements: body mass index (BMI): weight/height, Mid arm circumference and Subjective Global assessment (SGA) by Detsky et al. Patients were graded as well nourished; SGA grade A, moderately malnourished; grade B and severely malnourished; grade C. The diagnosis of alcoholic cirrhosis was made on the basis of history of alcohol consumption >80g/dl in men and > 40g/dl in women for 10yrs. Viral hepatitis B and C related cirrhosis was based on serological evidence of HBsAg, HBV DNA estimation, anti-HCV and HCV RNA estimation in patients with cirrhosis. Autoimmune Hepatitis was diagnosed based on the International Diagnostic Criteria for the diagnosis of Autoimmune Hepatitis. NASH related cirrhosis was diagnosed based on presence of cirrhosis in patients with evidence of BMI >28kg/m2, diabetes, negative viral studies, alcohol less than 20gm/day in men and <10gm/day in females and histological features like lobular or portal inflammation , ballooned hepatocytes with Mallory Denk bodes and fibrosis in a pericentral vein or zone 3 distribution. In absence of liver biopsy, even with probable NASH patients were categorised as cryptogenic cirrhosis. Diagnosis of cryptogenic cirrhosis was made on the basis of exclusion of all known causes of cirrhosis. [5]

Hepatic encephalopathy was diagnosed on basis of history, West Haven‟s criteria and number connection test A and B, Ascites, clinically and ultrasound examination. HRS was diagnosed in cirrhotics with ascites, with serum creatinine >1.5mg/dl, no improvement of ascites after at least 2 days of diuretic withdrawal and plasma expansion, absence of shock and other parenchymal kidney diseases. SBP was diagnosed on the basis of clinical suspicion and presence of PMN>250 cells / mm3 in the ascitic fluid. Gastroesophageal varices were detected and graded by endoscopy. HCC was diagnosed by radiology and or the presence of high alpha foetoprotein (>200 ng/ml) in the setting of a mass in a cirrhotic liver.

Statistical Analysis:

Data were managed on Microsoft excel spread sheet. Continuous variables were summarized by means and standard deviations. All statistical analysis was carried out by SPSS 16.0 version. Chi-square test for discrete variables (sex, Childs grade, symptoms etc), Independent t test or non parametric test for continuous variables and ANOVA test have been performed (wherever required) and p value of <0.05 has been considered as significant.

III. Results

PARAMETER	TOTAL	MALE	FEMALE
Total bilirubin	4.23±7.76	4.38±8.12	3.13±4.57
Cong. bilirubin	1.63±3.60	1.71±3.67	1.33±3.25
AST(U/L)	117.5±126.8	120.2±133.5	82.90±60.5
ALT(U/L)	64±86.78	65.23±91.0	53.56±44.6
Alk Phos (IU/ml)	219.7±173.69	220.2±170.5	212.65±183
Total protein (g/dl)	7.22±0.95	7.26±0.956	6.94±0.93
Albumin (g/dl)	0.64±0.33	0.64±0.34	0.62±0.23
Globulin (g/dl)	4.52±0.80	4.50±0.78	4.32±0.62
Albumin : Globulin	0.63±0.32	0.64±0.34	0.63±0.23
PT (sec)	19.12±7.67	19.23±7.60	18.45±14.79
INR	1.70±0.67	1.75±0.57	1.67±0.42
GGT (U/L)	176.50±333.6	188.76±356.5	91.34±98.2
Serum ammonia (mg/dl)	70.1±54.7	71.66±55.0	41.30±35.7
TLC per cub mm	7640.6±7352.5	7865.25±6409.5	6004.13±3390
Haemoglobin (mg/dl)	8.65±2.50	8.70±2.24	8.10±4.03
RBC (per cub mm)	3.37±0.76	3.24±0.70	3.20±0.87
Platelet count	1.28±0.42	1.30±0.53	1.24±0.36
Serum Sodium (meq/l)	134.57±8.5	134.6±9.14	135.3±5.76
Serum potassium (mg/dl)	4.15±6	4.23±5.45	3.71±0.80
Serum Urea (mg/dl)	35.32±29.13	36.12±30.17	30.07±16.5
Serum creatinine (mg/dl)	1.35±2.15	1.40±2.13	1.01±0.53
FBS (mg/dl)	110.21±54.12	110.13±56.67	107.23±45.13
PPBS (mg/dl)	139.54±66.80	140.02±65.02	136.50±60.15

Serum calcium (mg/dl	7.80±0.50	7.83±0.50	7.82±0.40
Magnesium (mg/dl)	1.70±0.5	1.72±0.36	1.52±0.27

Table 1: Showing the biochemistry findings in all cases

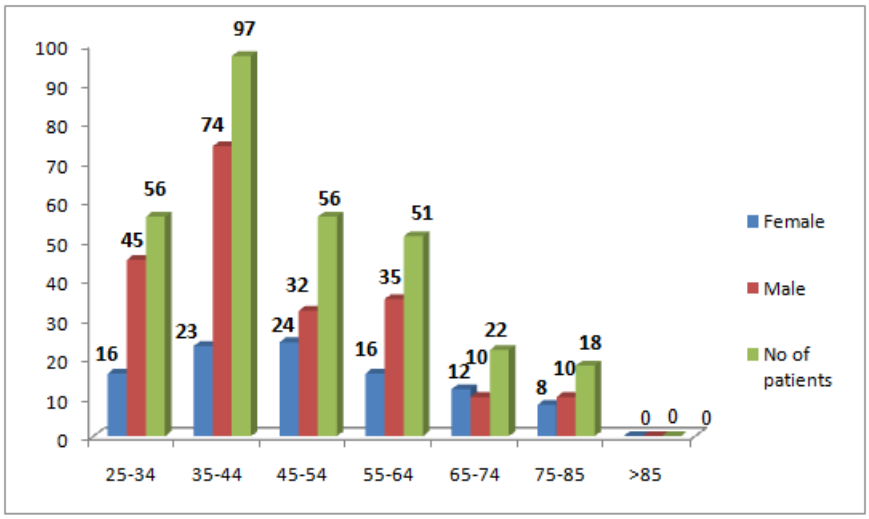

Figure 1: Showing age distribution of the cases

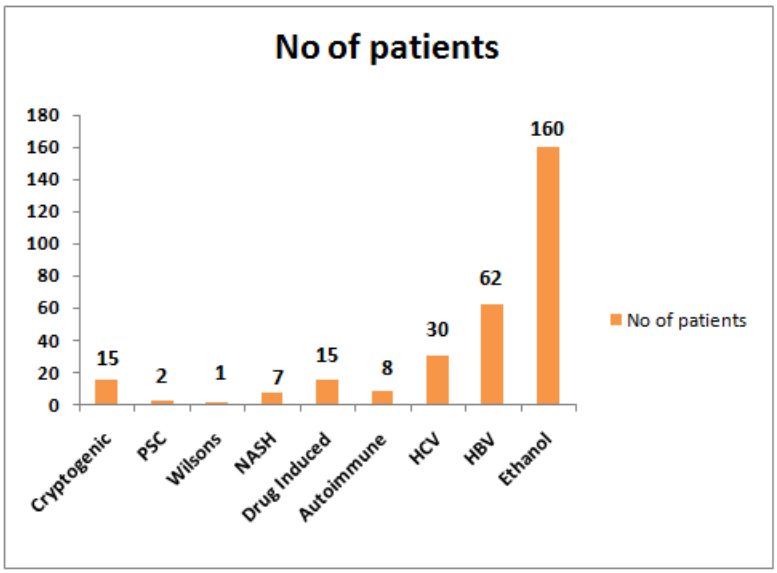

Figure 2: Showing etiological distribution of all cases

IV. Discussion

Studies have found an association between low socioeconomic status and increased cirrhosis incidence. In our study however, majority were from middle class or lower middle class background 72.7% and 23.4% respectively. This could be because health care facilities at government hospitals are mostly availed by middle socioeconomic group. Similar findings were observed by Goel A et al, 2013 where majority belonged either to middle class (n=329; 70%) or lower class. Protein Energy Malnutrition (PEM) is often observed in liver cirrhosis. Western studies have documented malnutrition rates from 20% in compensated liver cirrhosis to 60% in decompensated disease. Increased sepsis, reduced life span have been observed in cirrhotics with poor nutrition status compared to those without. In a study in Malaysian patients (Tai et al 2010), 66.7% had SGA GradeB and 33.3% ,Grade C nutritional status. In our study, 41% had moderate malnutrition and 37.1% had severe malnutrition and was similar to findings in other studies.

Most patients present late with advanced disease. Ascites (74.3%), UGI bleeding (43.4%), jaundice (36.3%) and altered behaviour (20.3%) were the commonest presentation in our study. Ascites and upper GI bleeding was the commonest complications in other studies too; Maskey R et al 2011[24] (ascites 84.4% and

Upper GI bleeding in 35.5%) and Md Shahid Aziz et al 2009; (Ascites 53.8% and upper GI bleed 25.1%) too had similar findings.

V. Conclusion

Cirrhosis of liver is a major health problem in South India and affects males in the most productive years. Alcohol abuse is the major cause of cirrhosis in south India that is entirely preventable through proper education and legislation. Malnutrition, widely prevalent among cirrhotics is associated with high mortality. Patients present in advanced stage of disease with complications. However, limitation of the study was that patients were not followed up for long and the effect of the various treatment options were not recorded.

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