Original Article

IMPACT OF DIABETES MELLITUS, INSULIN RESISTANCE AND OBESITY IN ALCOHOLIC LIVER CIRRHOSIS

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Abstract

Background: A leading cause of chronic liver disease and liver failure, with increased morbidity and mortality, diabetes mellitus, obesity and insulin resistance are ubiquitous in Alcoholic liverdisease patients and increase the risk of liver-related frailty. The prevalence of risk factorslike diabetes mellitus, insulin resistance and obesity in alcoholic liver cirrhosis and to analyse the clinical profile and severity of alcoholic liver cirrhosis within these risk factorseither alone or in combinations were the objective of our study.

Methods: An observational study with 107 patients and onset of liver disease, anthropometric examination, clinical examination and laboratory data were recorded. Diabetes mellitus was diagnosed by WHO criteria of fasting, postprandial blood glucose and HbA1C levels.Obesity was diagnosed in patients with BMI >25 kg/m^2. IR diagnosis is as per HOMA-IR score.

Results: The male predominace was higher in alcoholic liver cirrhosis. Mean age was 48.3 ± 10.03years; age group between 40-49 years had 40.19% of study population. Most common symptom was abdomen distension (57.94%) followed by leg swelling (55.14%) andjaundice (54.21%). The mean alcohol duration was 18.69 years. The mean alcohol consumption was 130.7 ± 53.62 gm/day. Relationship between Alcoholic liver cirrhosis and diabetes mellitus, IR and Obesity when compared with CTP score had P value 0.743, 0.170 and 0.500 respectively, MELD score had P-value 0.967, 0.649 and 0.324 respectively.

Conclusion: Alcohol duration and consumption increases the severity of alcohol liver cirrhosis. Impactof Diabetes Mellitus, IR and Obesity in alcohol liver cirrhosis was no significant.

Keywords: Diabetes Mellitus, Insulin Resistance, Obesity, Alcoholic Liver Cirrhosis

Introduction

Cirrhosis is a chronic parenchymal liver disease, defined histologically as diffuse hepatic process which is characterised by fibrosis and the transformation of normal liver architecture into structurally aberrant lesions.(1)Alcohol is the third leading cause of diseaseburden in the world. Each year, 2.5 million people die as a

result of alcohol consumption.

[2] The World Health Organization (WHO) estimates that 140 million people worldwide are addicted to alcohol, causing harm to their lives and economies.

According to an India Spend analysis of 2013 National Crime Records Bureau (NCRB) data, 15 people die every day or one every 96 minutes as a result of the effects of drinkingalcohol in India. In 2016, India's alcohol consumption was estimated to be around 5.4 billion litres, with the figure expected to rise to around 6.5 billion litres by 2020. In India, per capita alcohol consumption increased by 38%.[2]Alcoholic liver disease (ALD) continues to be a leading cause of chronic liver disease and liver failure, with increased morbidity and mortality. Individuals who have used alcohol for a long time are more likelyto develop advanced liver disease such as alcoholic steatohepatitis (ASH), cirrhosis, and hepatocellular carcinoma (HCC) due to a variety of risk factors.[3] The majority of ALD patients seek medical attention after developing jaundice or cirrhosis as complications.

[4] To date, the most effective therapy for slowing the progression of ALD and even reversing liver damage is abstinence from alcohol for an extended period of time.[5,6] Diabetes Mellitus, Obesity and Insulin Resistance (IR) are ubiquitous in ALD patients and increase the risk of liver-related frailty. The burden of diabetes, insulin resistance and obesity is higher in patients of cirrhosis.

The aim of our study is to study the prevalence of risk factors like diabetes mellitus, insulinresistance and obesity in alcoholic liver cirrhosis and to analyse the clinical profile, liver functions, imaging and severity of alcoholic liver cirrhosis within these risk factors eitheralone or in combinations and compare with controls.

Material and Methods

A total of 107 consecutive patients admitted in General Medicine and Gastroenterology wards/ HDU of Pradyumna Bal Memorial Hospital, KIMS, Bhubaneswar with diagnosisof alcoholic cirrhosis with concern. It was a prospective, observational study. All consecutive patients with diagnosis of alcoholic liver cirrhosis and age more than 18 years were in inclusion criteria. Non alcoholic causes of liver cirrhosis like viral hepatitis, autoimmune hepatitis, drugs induced, NAFLD, Age less than 18 years,thyroid disease, cardiac disease like DCMP/CAD, chronic obstructive pulmonary disease, chronic kidney disease, pregnancy, Heptocellular carcinoma and other malignancies were the exclusion criteria of this study.

The severity of cirrhosis was calculated and classified according to the Child–Pugh, Modelfor End-Stage Liver Disease (MELD) score and MELD-Na score. The diagnosis of alcoholic liver cirrhosis was based upon history of excess alcohol consumption, clinical, laboratory and imaging studies. Diabetes mellitus was diagnosed by WHO criteria of fasting, postprandial blood glucose and HbA1C levels. Obesity was diagnosed in patientswith BMI >25 kg/m^2. BMI in Cirrhotic patients with ascites or edema was calculated separately, 5% of weight subtracted from measured weight with mild ascites, 10% in moderate ascites and 15% in severe ascites, 5% with edema. Insulin resistance (IR) diagnosis is as per calculations of HOMA-IR score = fasting plasma glucose (mmol/L) X fasting serum insulin (mU/L) divided by 22.5. HOMA-IR score ≥ 2.5 indicates IR.

Study Approval

Approval of the Institutional Research and Ethics committee (IEC-122/2019), KIMS, KIIT university, Bhubaneswar, was taken prior to the commencement of the study.

Statistical analysis

Descriptive data was analysed using the Statistical Package for Social Sciences (SPSS ® Inc., Chicago, IL), version 21, and Microsoft Office Excel Professional 2013 ®. All the quantitative data were expressed in mean ±

SD. Spearman's Rank correlation coefficient was used to summarise the strength and direction (negative or positive) of a relationship between two variables. ANOVA (Analysis of variance),

Independent t-test was used for variance and significance testing. Fisher's exact test used for statistical significance and in the analysis of contingency tables. ROC (Receiver Operating Characteristic) curve was used to demonstrate connection between clinical sensitivity and specificity and to choose the most appropriate cut off for an assessment tool.$p < 0.05$ was considered significant.

Results

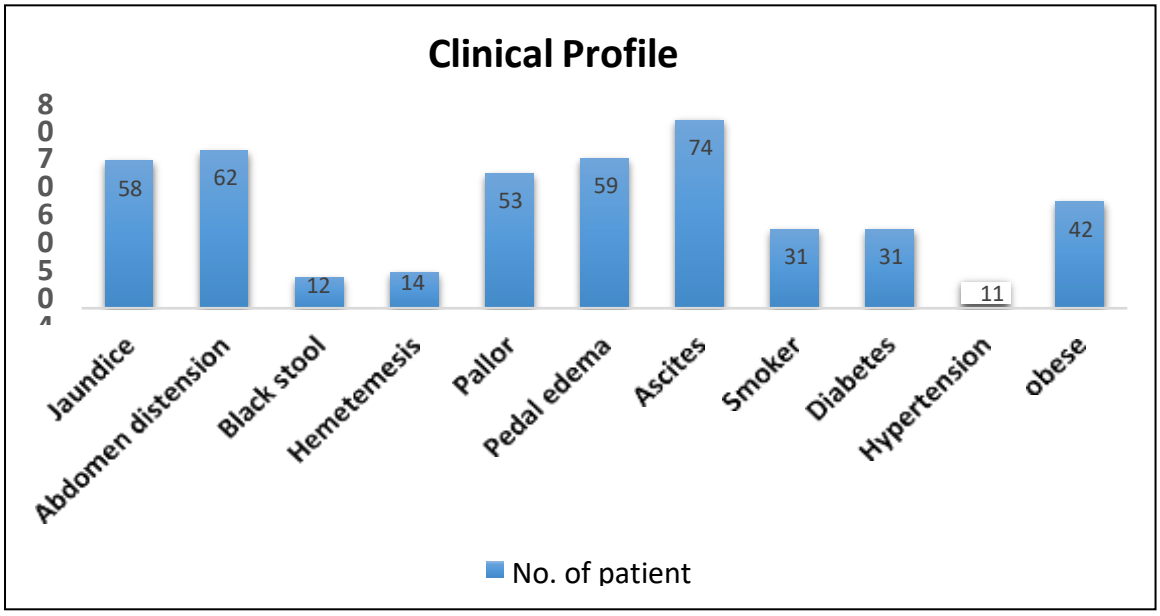
The study enrolled 107 individuals who had been diagnosed with Alcoholic liver cirrhosisbased on clinical characteristics, lab and radiologic findings. [2] There were 102 (95.33%)males and 5 (4.67%) females, with M: F ratio of 4.9:1 .There was only one subject in theage group <30 years, 18 (16.82%) in the age group 30-39. There were43 (40.19%) in the

age group 40-49 years, 27 (25.23%) in age group 50-59 years. 16 (14.95%) in age group 60-69 years and one in the age group 70-79 years and 80-89 years group.

Clinical profile ofstudy population

Clinical characteristics of the studied sample were 58 (54.21%) had jaundice, 62 (57.94%) had abdominal distension, 12(11.21%) had melena, 14 (13%) had hematemesis, 31 (28.97%) had history of smoking cigarettes. 31(28.97%) diabetes, 11(10.28%) had historyof hypertension. 42 (39.25%) obese. On clinical examination, 53(49.53%) had pallor withsignificant p value 0.0001, 58(54.21%) had icterus with significant p value 0.0001,59(55.14%) had pedal edema with significant p value 0.0001 and those with ascites were 74 (69.16%) with significant p value 0.0001 shown in Fig 1.

Figure 1. Clinical profile of study population.



Alcohol spectrum in study population.

The mean alcohol duration in this study population was 18.69 years, in which inimum duration is 9 years and maximum duration is 40 years. The mean alcohol consumption was 130.7 ± 53.62 gm/day, minimum individual

consumption is 56 gm/day and maximumindividual consumption is 233 gm/day.

Relationship between alcohol duration and CTP score

Average alcohol duration in this study population is 18.69 years. Alcohol duration when compared with CTP score was significant with P value 0.001.

Relationship between ascites and CTP score and MELD score.

Among 107 patients , 77 had ascites in which 42 had mild ascites (mean 8.79 ± 2.07), 25 had moderate ascites (mean 10.32 ± 1.88) and 10 had severe ascites (mean 10 ± 1.53). relationship between ascites and severity of alcoholic liver cirrhosis based on CTP score was significant with p value 0.0001 and meld score with p value 0.037, in Table 1.

Table 1. Relationship between ascites and CTP and MELD score.

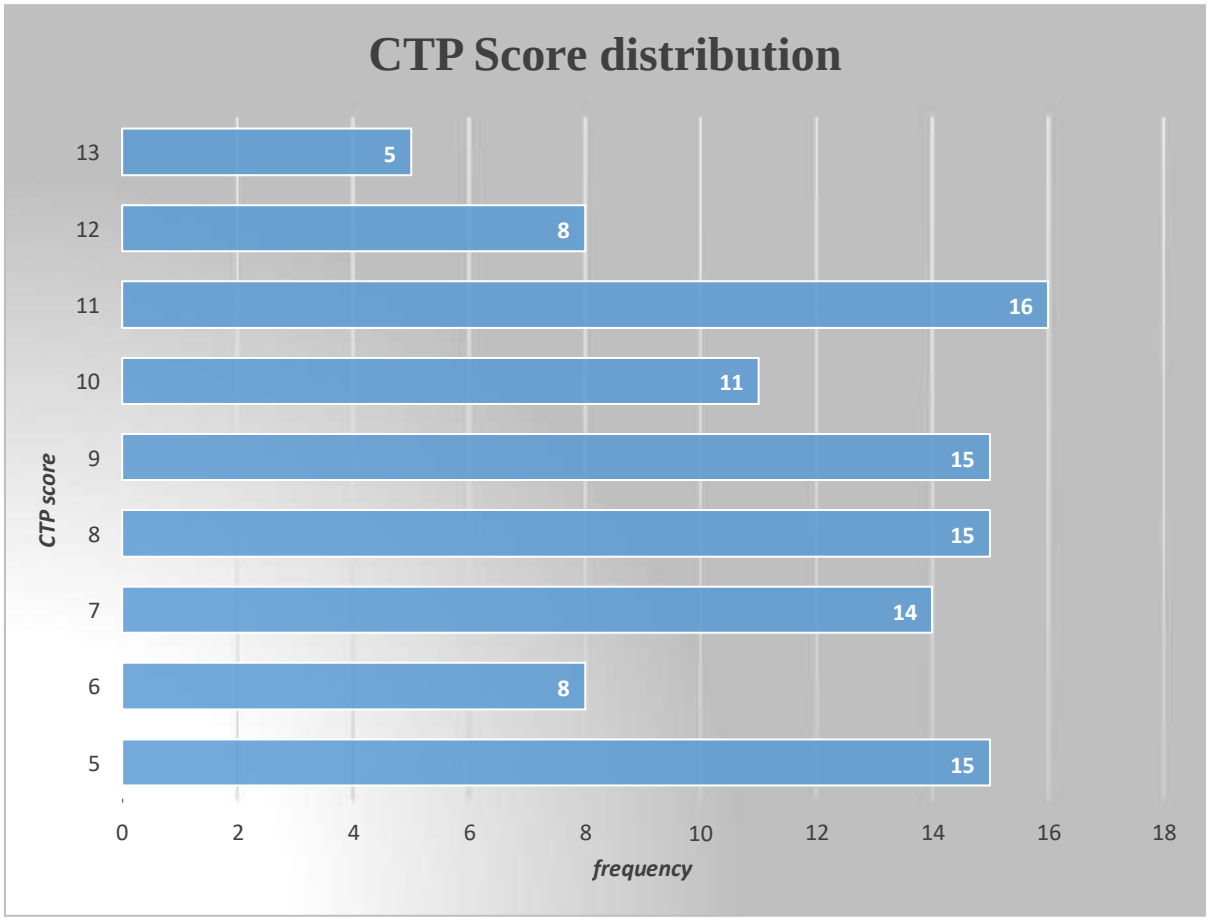
	Ascites	Freq	Mean	Std. Deviation	95% Confidence Interval for Mean		p-value
					Lower Bound	Upper Bound	
CTP Score	No	30	6.53	1.737	5.88	7.18	0.000
	MILD	42	8.79	2.078	8.14	9.43	
	Moderate	25	10.32	1.887	9.54	11.10	
	Severe	10	10.00	1.563	8.88	11.12	
	Total	107	8.63	2.369	8.17	9.08	
MELD Score	No	30	17.60	5.184	15.66	19.54	0.037
	MILD	42	22.12	7.435	19.80	24.44	
	Moderate	25	22.08	8.190	18.70	25.46	
	Severe	10	20.80	5.574	16.81	24.79	
	Total	107	20.72	7.108	19.36	22.08	

CTP Scores in the study population

Child Pugh Score was calculated in the study population. Most of the patients, sixteen (14.9%) of 107 had score of 11 out of 15, lying in Class C . This was followed by a scoreof 9, 8 and 5, with fifteen (14.01%) patients respectively. Eleven (10.2%) patients had a score of 10. Eight (7.47%) patients had a score of 6 and 12 respectively. Five (4.67%) patients had a CTP score of 13/15.As per the CTP score, patients were categorised as

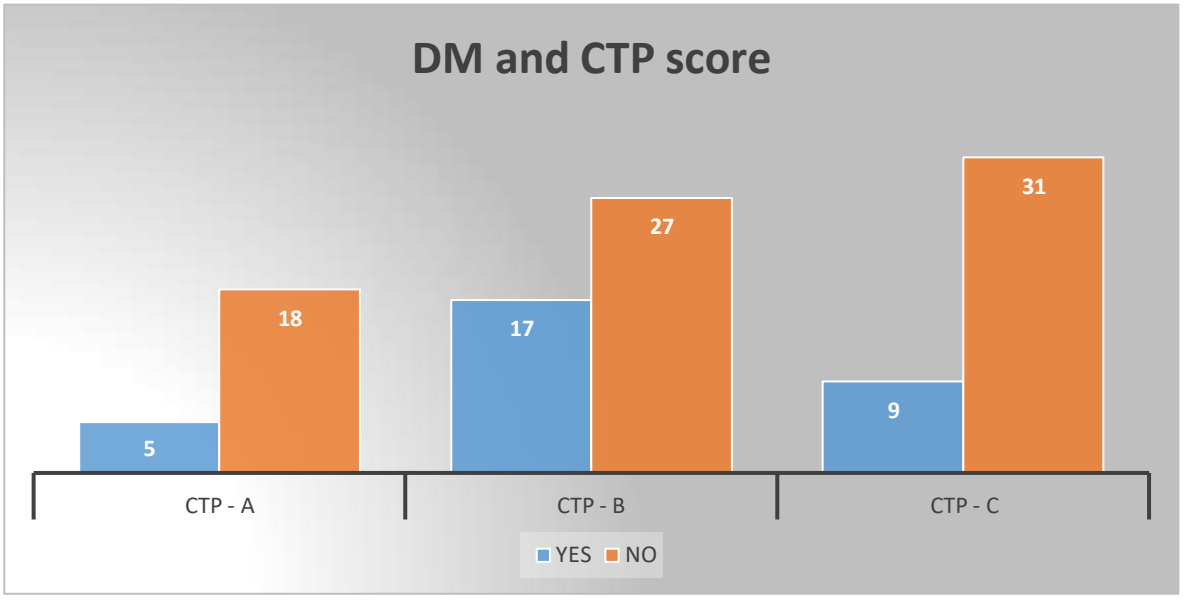
ClassA, B and C shown in Fig 2 . Out of the total 107 patients; 22(20.56%) patients were underclass A, 41(38.31%) were under class B and 37(34.57%) were under class C.

Figure 2. CTP score distribution of study population. Relationship between Diabetes Mellitus and CTP score



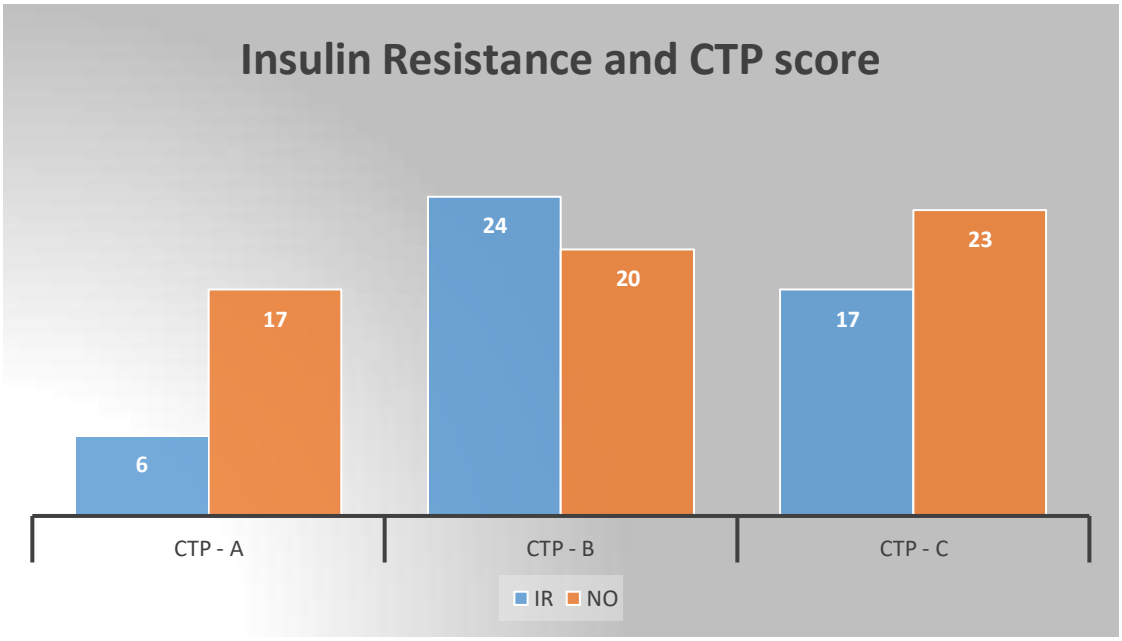
Among 107 patients in this study population diabetes were 31 (29%). The mean FBS was 113.42 ± 46.40 mg/dl and mean PPBS was 153.45 ± 57.06 mg/dl. The mean HbA1c was 6.11 ± 1.04. Relationship between diabetes mellitus and CTP score was not significant asP value 0.743 (figure 14) and with meld score (P value 0.967) was not significant in Fig 3.

Figure 3. Relationship between diabetes and CTP score.Relationship between Insulin Resistance and CTP score



Among 107 patients in this study population, 47 (43.92%) had insulin resistance. The Mean serum insulin was 10.25 ± 8.40 µU/ml. The mean HOMA IR score was 3.38 ± 4.63.Risk of insulin resistance in alcohol liver cirrhosis was not significant when compared withCTP score (p value 0.170) figure 15 and MELD score (p value 0.649) shown in Figure 4.

Figure 4 . Relationship between insulin resistance and CTP score.



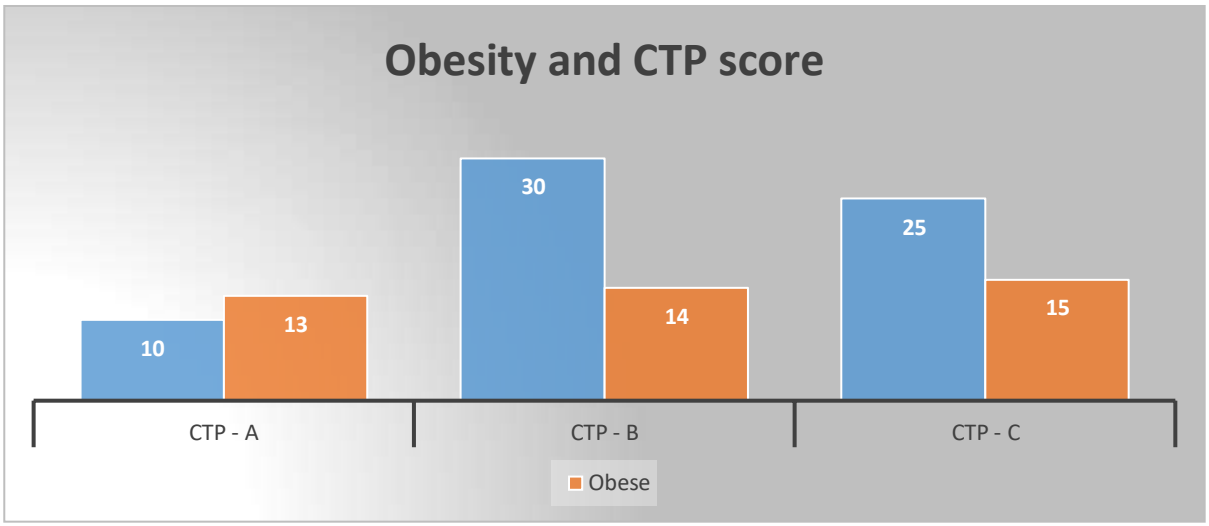
Relationship between obesity and CTP score

Among 107 patients in this study population, 43 (40.18%) had obesity. The mean weight was 62.11 ± 12.10 kg. The mean height was 1.66 ± 0.08 meter. The mean BMI value was

24.06 ±3.95 kg/m^2. Relationship between obesity and alcholic liver cirrhosis was insignificant compared with

CTP score with P value 0.500 and with MELD score had p value 0.324, shown in figure 5.

Figure 5. Relationship between obesity and CTP score.



Discussion

In this study group, 107 patients were included with diagnosed alcoholic liver cirrhosis. All these patients were then assessed with clinical parameters, history, alcohol use, comorbidities associated, and lab parameters like CBC, LFT, RFT, PT/INR, serum insulinetc. and were then radiological parameters using USG-Abdomen, CT abdomen and UGI Endoscopy done.

The major bulk of the patients in our research study, around 69.16%, had ascites, followed by abdominal distension (57.9%), Pedal oedema (55.14%), jaundice (54.21%), black stool,and hematemesis were other typical symptoms. These symptoms were consistent for both male and female group.

Singh et al.[7] in a study from eastern India reported that 50% of the patients with alcoholicliver disease started drinking before the legal age of drinking.In Nand N et al,[8] studied 149 patients (74%) had been drinking for ≤20 yrs, which further consolidates the fact that Indians develop liver disease in lesser duration than Western population.

In a study of 1604 alcoholic patients, Naveau et al,[9] 48 found that being overweight wasassociated with a greater prevalence of alcoholic hepatitis and cirrhosis (60% vs 35% in lean patients). In our study total number obesity found was 42 (39.25%). The mechanismswhereby obesity and insulin resistance may exacerbate liver injury in alcoholic liver disease are still debated.

The prevalence of the metabolic syndrome, as defined by the 2001 ATP III criteria, was evaluated in the third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1994). In our study compare the relationship between obesity and severity of alcoholic liver cirrhosis is insignificant comparing with CTP score p value 0.500 and MELD score p value 0.324.

In our study, 47 (43.92%) had insulin resistance. The Mean serum insulin was 10.25 ±

8.40 µU/ml. The mean HOMA IR score was 3.38 ± 4.63. when compared HOMA IR between obese and normal adolescents, the mean HOMA-IR was 6.04 and 6.57 respectively, which is insignificant p value 0.543. In Lee J at el study,[10] a significant difference among the HOMA-IR values of normal weight, overweight, and obese adolescents was observed. High values for HOMA-IR in obese subjects compared to normal adolescents (Mean HOMA-IR: 4.93 (95% CI 4.56-5.35) vs. 2.30 (2.21-2.39), respectively), has also been validated in a large study

among adolescents from USA.

An association between diabetes mellitus and liver cirrhosis was first described byBohan[11] and named as hepatogenous diabetes by Megyesi et al,[12] in which 57% of cirrhotic patients showed increased insulin resistance. In our study 47 (43.92%) patient showed insulin resistance.

In Goswami A at el study,[13]HOMA-IR value was raised in CTP score >9 (P value 0.0004)and model of end stage liver disease (MELD) score >15 (P value 0.02). The mean FBS level was 84.7±20.2 mg/dL, and the mean fasting insulin was 11.8±6.7 μU/mL. The overallIR in the alcholic cirrhotic patients was 68.5%.The mean FBS level was113.42 ± 46.40 mg/dL, and the mean fasting insulin was 10.25 ± 8.40 μU/mL. The mean levels of HOMA-IR were 2.54±1.71.In our study, HOMA IR value was raised in CTP class B and C (p value0.170) in meld score > 20 (0.649). Total IR in our study was 43.92 %.

In Hsieh et al study,[14] Alcoholic cirrhotic men had a significantly higher risk of type 2 diabetes (aHR 1.182, CI: 1.046-1.335) compared with non-cirrhotic individuals. Increasedrisks were seen in men (aHR 1.690; CI: 1.455-1.963) and women (aHR 1.715; CI: 1.113- 2.645) with alcoholic cirrhosis compared to those with cirrhosis without alcohol. In our study Among 107 patient in this study population diabetes were 31 (29%). The mean FBSwas 113.42 ± 46.40 mg/dl and mean PPBS was 153.45 ± 57.06 mg/dl. The mean HbA1c was 6.11 ± 1.04. Relationship between diabetes mellitus and severity of alcoholic liver cirrhosis was not significant comparing with CTP score P value 0.743 and with meld score(P value 0.967) was insignificant.

Conclusion

In our study, the male predominance was higher in alcoholic liver cirrhosis.The mean ageof the studied population was 48.3 ± 10.03 years.The age group between 40-49 years had40.19% of study population.Most common symptom was abdomen distension (57.94%)followed by leg swelling (55.14%) and jaundice (54.21%).The mean alcohol duration in this study population was 18.69 years.The mean alcohol consumption was

130.7 ± 53.62 gm/day.The most common type of alcohol intake in this study population is Whiskey (44.85%) followed by Rum (30.84%). Alcohol duration and consumption increases the severity of alcohol liver cirrhosis. Impact of Diabetes Mellitus in alcohol livercirrhosis was not significant.Impact of Insulin Resistance in alcohol liver cirrhosis was not significant.Impact of obesity in alcholic liver cirrhosis was insignificant.

Additional Information Disclosures

Human subjects: Consent was obtained or waived by all participants in this study. Kalinga Institute of Medical Sciences (KIMS), KIIT, Bhubaneswar, and Clinical Trials Registry of India issued approval #KIMS/KIIT/ IEC-122/2019

Animal subjects: All authors have confirmed that this study did not involve animal subjects or tissue.

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authorsdeclare the following:

Payment/services info: All authors have declared that no financial support was received from any organization for the submitted work.

Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that mighthave an interest in the submitted work.

Other relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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