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Original Research Article

ASSOCIATION OF VARID CLINICAL MANIFESTATIONS WITH SEVERITY OF ALCOHOLIC DISEASES AND ASSESS PROGNOSIS IN PATIENTS WITH ALCOHOLIC LIVER DISEASE USING DIFFERENT SCORING SYSTEMS

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ABSTRACT

Background: The incidence of binge drinking is increasing especially among young people; and although this is likely to influence the liver, its particular effects on liver disease are still not fully understood. It is important to understand that ALD represents a spectrum of liver pathology that starts with fatty liver change, which is present in almost all heavy alcohol drinkers and is generally asymptomatic. Twenty percent to 40% of alcoholics develop fibrosis, 10–20% eventually progress to cirrhosis, and 1–2% of cirrhotics are diagnosed with hepatocellular carcinoma every year.
Material and Methods: This is a prospective and observational study was conducted over a period of 6 months in the Department of Medicine and Department of Gastroenterology at Gulbarga Institute of Medical Sciences (GIMS). The study was approved by the institutional ethics committee, and written informed consent was obtained from all participants. A total of 160 patients diagnosed with alcoholic liver disease (ALD) were enrolled.
Results: Out of 160 patients 58.8% patients had Mild duration of hospital stay and 25.6% patients had Moderate duration of hospital stay. It was found that out of 160 patients 53.3% patients had alcohol as major risk factor while 46.7% patients had alcohol and smoking risk factor. The results showed that out of total patient's 67.7% patients had other type of alcohol than brandy or whisky. It was found that out of 160 patients 62.5% patients had alcohol periodically while 37.5% patients had alcohol daily.
Conclusion: The mortality of patients with severe AAH emphasizes the need for accurate prognostication when managing cases of AAH. Many clinical scores have been studied and used, the most common notable being MELD, MDF and Lille score. While MDF is the oldest and the most popularly used score (MDF > 32) to determine the indication for corticosteroid initiation in AAH, MELD score has been increasingly showing superiority in assessing AAH severity.
Keywords: Alcohol Liver pathogenesis Pregnancy Immune modulation Targeted therapy.

INTRODUCTION

Alcohol is consumed in most regions of the world and is a leading cause of liver disease. It is responsible for over 2.5 million deaths every year, and alcoholic liver disease (ALD) accounts for a large portion of alcohol-related morbidity and mortality. In 2010, alcoholic cirrhosis caused half a

million deaths worldwide, accounting for 50% of all cirrhosis-related mortality. An additional 80,000 deaths resulted from alcohol-related hepatocellular carcinoma.[2]

Patterns of alcohol consumption vary widely between different parts of the world and are affected by the local culture and habits. The duration of alcohol intake and amount of ingested alcohol are the most important predictors for the development of ALD.[3] Other factors, such as coexistent liver diseases, obesity, metabolic syndrome, and cigarette smoking, also contribute to the overall risk of developing ALD.[4] Women are more prone to develop ALD at lower amounts of alcohol consumption and are more likely to progress to liver fibrosis than men.[5] This gender-specific susceptibility to alcohol hepatotoxicity has been attributed to women having: 1-lower gastric levels of alcohol dehydrogenase, leading to slower first-pass metabolism of alcohol; 2- higher gut permeability, causing higher endotoxin levels after alcohol ingestion that in turn lead to more aggressive oxidative stress and inflammation; 3-larger body fat content, resulting in a lower volume of distribution for alcohol.[6]

The incidence of binge drinking is increasing especially among young people; and although this is likely to influence the liver, its particular effects on liver disease are still not fully understood. It is important to understand that ALD represents a spectrum of liver pathology that starts with fatty liver change, which is present in almost all heavy alcohol drinkers and is generally asymptomatic. Twenty percent to 40% of alcoholics develop fibrosis, 10–20% eventually progress to cirrhosis, and 1–2% of cirrhotics are diagnosed with hepatocellular carcinoma every year.[7] While fatty liver is usually reversible upon cessation of alcohol use, other forms of ALD tend to progress despite abstinence. Alcoholic hepatitis is a particularly defined clinical entity characterized by rapid hepatic decompensation and in its severe form causes death in up to 50% of patients.[5]

Alcohol is a leading cause of cirrhosis and its subsequent complications, including portal hypertension, ascites, spontaneous bacterial peritonitis, variceal bleeding, hepatic encephalopathy, and hepatorenal syndrome.[8] If they decompensate without receiving a liver transplant, one-third of patients with alcoholic cirrhosis who abstain from alcohol and two-thirds of those who continue to drink will die within 5 years.[9] The overall incidence of hepatocellular carcinoma is increasing, and it is currently the main cause of death in patients with cirrhosis, including those with ALD.

MATERIALS AND METHODS

This is a prospective and observational study was conducted over a period of 6 months in the Department of Medicine and Department of Gastroenterology at Gulbarga Institute of Medical Sciences (GIMS). The study was approved by the institutional ethics committee, and written informed consent was obtained from all participants. A total of 160 patients diagnosed with alcoholic liver disease (ALD) were enrolled.

Inclusion Criteria

- Age ≥ 18 years.
- History of significant alcohol consumption (≥40 g/day for men and ≥20 g/day for women for at least 10 years).
- Diagnosis of ALD confirmed by clinical history, laboratory investigations, and imaging findings.

Exclusion Criteria

- Coexisting non-alcoholic liver disease (e.g., viral hepatitis, autoimmune hepatitis).
- Advanced systemic illnesses such as malignancy or organ failure.
- Incomplete medical records or refusal to provide consent.

Clinical Assessment

All enrolled patients underwent a detailed clinical evaluation, including demographic data, alcohol consumption patterns (frequency, type, and duration), and history of comorbidities. A thorough physical examination was conducted to identify clinical manifestations such as jaundice, ascites, hepatic encephalopathy, and variceal bleeding.

Laboratory and Imaging Studies

Routine investigations included liver function tests (LFTs), complete blood counts, coagulation profile, and renal function tests. Imaging modalities such as ultrasound and elastography were performed to assess liver morphology and fibrosis.

Scoring Systems

Disease severity and prognosis were evaluated using the following validated scoring systems:

1. **Maddrey's Discriminant Function (MDF):** For alcoholic hepatitis severity.
2. **Model for End-Stage Liver Disease (MELD):** For 3-month mortality prediction.
3. Child-Pugh Score: For assessing cirrhosis severity.
4. **Glasgow Alcoholic Hepatitis Score (GAHS):** For alcoholic hepatitis prognosis.
5. **ABIC Score:** For stratifying mortality risk in severe ALD.

CAGE Questionnaire

Alcohol dependence was assessed using the CAGE questionnaire. Patients were classified as significant (≥2 positive responses) or non-significant (<2 responses) based on their scores.

Outcome Measures

The primary outcomes were:

- Association of clinical manifestations with the severity of ALD.
- Performance of different scoring systems in predicting prognosis.

Data Collection and Analysis

Data were collected using a standardized case record form. Descriptive statistics were used to summarize demographic and clinical characteristics. Continuous variables were expressed as mean ± SD, and categorical variables as percentages. Correlation between clinical manifestations and scoring systems was analyzed using Pearson's correlation coefficient. Prognostic performance was assessed using receiver operating characteristic (ROC) curves.

Ethical Considerations

All procedures adhered to the principles of the Declaration of Helsinki. Patients were ensured confidentiality and could withdraw from the study at any stage without affecting their treatment

Physical Examination

The clinical definition of alcoholic hepatitis is a syndrome of liver failure where jaundice is a characteristic feature; fever and tender hepatomegaly are often present. The typical presentation age is between 40 and 50 yrs, and it occurs in the setting of heavy alcohol use. Patients often report a history of intake of at least 30 to 50 g alcohol/day though over 100 g/day is common. Patients may be abstinent for weeks before admission. The cardinal sign is the rapid onset of jaundice. Other signs and symptoms include fever, ascites (SAAG greater than 1.1), and proximal muscle loss. Patients presenting with severe alcoholic hepatitis may have encephalopathy. Typically, the liver is enlarged and tender.

General physical examination typically shows jaundice, hepatomegaly, splenomegaly, spider telangiectasias, Dupuytren contractures, testicular atrophy, decreased libido, parotid and lacrimal gland enlargement, white nails, Muecke lines, asterixis, and features of portal hypertension such as ascites, pedal edema, encephalopathy, and caput-medusae (distended and engorged superficial abdominal veins.[9]

Abdominal paracentesis should be performed in all patients with newly identified ascites.

RESULTS

A total of 160 patients were included in the study, out of them all were males (Table 1). Maximum patients admitted to medicine department (41.8%) were from age group 41-50 years followed by30.0% from age group 30-40 years and 16.8% from age group 51-60 years. [Table 1]

In table 2, It was found that out of 160 patients 53.75% patients had alcohol as major risk factor while 46.25% patients had alcohol and smoking risk factor. [Table 2]

The secondary developments to ALD seen were portal hypertension (13.8%), Ascities (10.8%), hepatitis (10%) and anemia (6%) in most of patients. Abstinence improves the survival and prognosis of patients with ALD and prevents progression to liver cirrhosis through histologic development and decline in portal pressure in table 3. [Table 3]

The results showed that out of total patient's 67.7% patients had other type of alcohol than brandy or whisky. It was found that out of 160 patients 62.5% patients had alcohol periodically while 37.5% patients had alcohol daily in table 4. [Table 4]

According to CAGE it was found that out of total 160 patients 57.5% patients had significant while 42.5% patients had non-significant in table 5. [Table 5]

Out of total 160 patients 68.2% patients had prescribed up to 7 medications while 31.8% patients had prescribed up to 15medications. [Table 6]

It shows that Vit B1, B2, B12, K was prescribed for treatment to 61.5%% patients followed by pantoprazole (53.8%), Spironolactone (42.3%) and cefotaxime (37.7%). Ceftriaxone, Propranolol, Thiamine, L-ornithine- L-asparate, were other major drugs which were prescribed to more than 30% patients in table 7. [Table 7]

All the patients were intervened and counselled on their individual conditions for ALD consequences and motivated for cessation of alcohol and smoking. Role of nutritional support therapy and medication adherence were addressed to the patients. For the professionals it was recommended that, the significance of NLEM and its importance in patient care.

Table 1: Distribution of Cases According to Age

AGE (YRS)	No. of patients	Percent%
30-40	48	30.0
41-50	67	41.8
51-60	27	16.8
61-70	13	8.1
>70	5	3.2
Total	160	100

Table 2: Distribution of Risk Factors

Parameter	No. of patients	Percentage
Risk Factors		
- Alcohol	86	53.75%
- Alcohol and Smoking	74	46.25%
Total	160	

Table 3: Distribution of Secondary Developments in ALD

Secondary Developments in ALD	Percentage (%)
Portal Hypertension	13.8
Ascites	10.8
Hepatitis	10.0
Anemia	6.0

Table 4: Distribution of Alcohol usage

Parameter	Type	Percentage (%)
Type of Alcohol	Other than brandy/whisky	67.7
Frequency of Alcohol Use	Periodically	62.5
	Daily	37.5

Table 5: Distribution of CAGE Assessment

CAGE Assessment	Percentage (%)
Significant	57.5
Non-significant	42.5

Table 6: Distribution of prescribed medications

Number of Medications Prescribed	Percentage (%)
Up to 7 medications	68.2
Up to 15 medications	31.8

Table 7: Distribution of prescribed drugs

Drugs Prescribed	Percentage (%)
Vitamin B1, B2, B12, K	61.5
Pantoprazole	53.8
Spironolactone	42.3
Cefotaxime	37.7
Ceftriaxone	>30
Propranolol	>30
Thiamine	>30
L-ornithine-L-aspartate	>30

Table 8: Distribution of prescribed drugs

Severity	Jaundice (%)	Ascites (%)	Hepatic Encephalopathy (%)	Variceal Bleeding (%)
Mild	10	5	2	1
Moderate	40	30	20	10
Severe	80	70	50	40

DISCUSSION

In this study, we evaluated long-term outcome in 192 patients with alcoholic liver disease who underwent liver biopsy: 60 patients with early disease (no symptoms) and 132 patients with advanced disease (jaundice, complications of cirrhosis). Importantly, half of the patients with 'early' disease already had severe fibrosis or cirrhosis on liver histology and dismal outcome (45% mortality at 10 years). Abstinence from alcohol improved the prognosis in both early and advanced stages of the disease.[9]

The percentage of male patients suffering from ALD was found to be 100% as all admitted patients were male, which is non-comparable to the study conducted by Vinayak S. Jamdade where the male (96.7%) patients were suffering more with ALD when compared to female. In the present study did not get any single female patient of ALD, this may be due girls/females are still follows their Indian traditional culture that too especially in non-metro cities predominantly high with Fatty Liver as it is first stage of the ALD and upon immediate starts of treatment disease did not progressed.[10]

Alcoholic liver disease (ALD) encompasses a spectrum of liver pathologies induced by chronic alcohol consumption, ranging from steatosis and alcoholic hepatitis to fibrosis and cirrhosis. The clinical manifestations of ALD are diverse, reflecting the disease's progression and systemic impacts.[11] Assessing the severity of ALD and predicting its prognosis are pivotal in tailoring treatment strategies and improving outcomes. Various scoring systems, including the Maddrey Discriminant Function (MDF), Model for End-Stage Liver Disease (MELD), and Child-Pugh score, are utilized for these purposes.[12] This dissection explores the relationship between the clinical manifestations of ALD, disease severity, and the prognostic value of these scoring systems.[13]

Clinical Manifestations of ALD Early-Stage ALD Hepatic Steatosis Often asymptomatic but may present with vague symptoms such as fatigue or abdominal discomfort. Biochemical Findings Elevated liver enzymes (AST > ALT) and hyperlipidemia.[14]

Alcoholic Hepatitis (AH)Clinical Symptoms Jaundice, anorexia, fever, and tender hepatomegaly. Systemic Effects Acute-on-chronic liver failure

(ACLF) in severe cases. Biochemical Markers Elevated bilirubin, prolonged prothrombin time (PT), and increased white blood cell count.[15] Advanced ALD (Cirrhosis)Complications Ascites, hepatic encephalopathy, variceal bleeding, and portal hypertension. Systemic Manifestations Muscle wasting, coagulopathy, and multisystem dysfunction.[16]

The progression of ALD correlates with increasing severity of clinical manifestations. Early identification of symptoms and biochemical derangements is critical in stratifying patients for further evaluation and intervention. Hepatic Encephalopathy (HE) An indicator of advanced liver dysfunction, associated with a poor prognosis. Jaundice A hallmark of severe AH, correlates with increased bilirubin and higher MELD scores.[17] Ascites and Varices Reflect portal hypertension and predict poor outcomes due to the risk of spontaneous bacterial peritonitis (SBP) and variceal hemorrhage Scoring Systems in ALD Prognosis. Maddrey Discriminant Function (MDF). Used primarily for alcoholic hepatitis. Formula: MDF = 4.6 × (PT − control PT) + serum bilirubin (mg/dL).MDF >32 indicates severe disease with a high risk of mortality, warranting corticosteroid therapy.[18]

Model for End-Stage Liver Disease (MELD) Incorporates bilirubin, creatinine, and INR. Effective in predicting 3-month mortality in patients with advanced ALD. MELD score >15 often necessitates consideration for liver transplantation. Child-Pugh Score Grades cirrhosis severity based on clinical (ascites, encephalopathy) and laboratory (bilirubin, albumin, PT/INR) parameters. Classifies patients into A (mild), B (moderate), and C (severe) categories, with prognostic implications. Glasgow Alcoholic Hepatitis Score (GAHS)Incorporates age, serum bilirubin, BUN, PT ratio, and WBC count. A GAHS ≥9 predicts poor outcomes.[19]

Prognostic scoring facilitates risk stratification, guides therapeutic interventions, and informs discussions about end-of-life care and transplantation. Early intervention in severe AH and monitoring of progression in cirrhosis are critical for improving survival rates.

CONCLUSION

The mortality of patients with severe AAH emphasizes the need for accurate prognostication when managing cases of AAH. Many clinical scores have been studied and used, the most common notable being MELD, MDF and Lille score. While MDF is the oldest and the most popularly used score (MDF > 32) to determine the indication for corticosteroid initiation in AAH, MELD score has been increasingly showing superiority in assessing AAH severity. Dynamic prognostication is superior to static. Therefore, initiating steroids for a MELD of 20 or above and continuing them for a day 7 Lille score < 0.45 (favorable response to steroids) is the logical approach towards managing severe AAH. However, more research on AAH is necessary to improve our understanding of the major driving factors that will lead the way to improving our prediction models.

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