Clinical Profile of Alcoholic Liver Disease in Saveetha Medical College and Hospital and Its Association with Type, Amount and duration of Alcohol Consumption

S. Aswathi¹ and V. Vikranth²*

¹ Saveetha Medical College and Hospital, Saveetha Nagar, Thandalam, Chennai 602105, India.
² Department of General Medicine, Saveetha Medical College and Hospital, Saveetha Nagar, Thandalam, Chennai 602105, India.

Authors’ contributions

This work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.

Article Information

DOI: 10.9734/JPRI/2021/v33i48B33270

Editor(s):
(1) Sawadogo Wamtinga Richard, Ministry of Higher Education, Scientific Research and Innovation, Burkina Faso.

Reviewer(s):
(1) Chike John Okeke, Lagos University Teaching Hospital, Nigeria, (2) Moses Darpolor, Stillman College, USA.

Complete Peer review History: https://www.sdiarticle4.com/review-history/74894

Received 24 August 2021
Accepted 21 October 2021
Published 08 November 2021

ABSTRACT

Introduction: Alcoholic liver disease is caused as a result of overconsuming alcohol that damages the liver, leading to inflammation, and scarring. It is often fatal with mortality and morbidity worldwide. Many studies in various countries show contradictory results about the role of amount, type and duration of alcohol exposure in determining the risk to develop ALD. This study aims to evaluate clinical profile of ALD in south Indian population and to correlation of disease severity with alcohol intake.

Material and Methods: A total of 50 patients of ALD were evaluated to correlate their clinical findings, biochemical parameters, prognostic markers (Discriminant function [DF] score, Model for end-stage liver disease [MELD] score and Child-Pugh score) and with their alcohol intake data in form of type, amount and duration of alcohol intake.

Results: Hepatic encephalopathy, neutrophil to lymphocyte ratio (NLR) and all three prognostic scores showed a dose-dependent relation with the amount of alcohol intake (p <0.05). The results

*Corresponding author: E-mail: vikranth@gmail.com;
showed that the duration of alcohol had a positive impact on the results. NLR correlates well with all prognostic markers (p <0.05 for NLR's Spearman correlation with DF score and Child-Pugh Score), more so with MELD score (p <0.0001); and complications like hepatic encephalopathy and hepatorenal syndrome.

**Conclusion:** In this study we conclude that there is significant dose dependent relation of ALD along with its complications, prognostic markers and NLR with the amount, type and duration of alcohol consumption. Although the type of alcohol consumption didn’t have much influence on the results, amount of intake had a correlation with NLR.

**Keywords:** Alcoholic liver disease; Hepatic encephalopathy; neutrophil to lymphocyte ratio (NLR).

### 1. INTRODUCTION

Alcoholic liver disease (ALD) is a disease which affects the population worldwide [1]. But however not everyone who consumes alcohol gets affected by the disease [1]. There is a lot of underlying factors like amount, duration and type of the alcohol consumed, nutritional status, race, genetic factors, sex which determine the risk of getting affected by this disease [1].

Various studies have given a wide variety result regarding the role of drinking pattern including amount, time duration and type of alcohol in the pathogenesis of disease. In those studies, only a few showed a major dose-dependent effect on the risk of developing Alcoholic liver disease, while others showing a threshold effect. The results of various studies should that South Asian race and female sex are more prone to develop liver disease with lesser alcohol consumption and in shorter duration of time than their counterparts [1], illicitly brewed liquor has been found to be more toxic than licit drinks despite low level of alcohol [2] and an important role is also played by the extent of protein calorie malnutrition determining the outcome of Alcoholic liver disease patients [3].

The main purpose of this study is to check the results on Indian population. As we know the Indian population has a wide variety ethnic difference when compared to the western population. Therefore, this study is mainly planned to get a clinical profile of Alcoholic Liver Disease in South Indian population and its Correlation with Type, Amount and Duration of alcohol consumption.

### 2. MATERIALS AND METHODS

This study is a prospective study in which a total of fifty alcoholic adult patients with chronic liver disease were chosen for the study for a study duration of six months. Inclusion criteria for the study were patients with Alcohol Use Identification Test (AUDIT-C) score ≥4. The exclusion criteria were patients with HIV, hepatitis B or C positive and hemodynamically unstable patients.

The study included a detailed history of alcoholism including type, duration (years), amount (units per day) and frequency (days per month), a clinical history and a complete physical examination. This study also includes biochemical findings of patients.

The alcoholic history:

- **Amount of alcohol:** This was arbitrarily divided the intake as light [alcohol intake was <360 units per month], moderate [360-719 units per month] and heavy [≥720 units per month] [4].
- **Alcoholic concentration:** A unit of alcohol was defined as amounts of liquor equivalent to 10 grams of pure alcohol. This amount equates roughly to 30 ml of spirits (concentration 40% by volume, e.g., whisky, vodka, gin), 100 ml of wine or 250 ml of beer. Alcohol concentration in local country-made spirits have no fixed standards, ranging from 25% to 50% by volume. Hence in present study, a unit of country liquor was also taken as 30 ml, equivalent to branded spirits.
- **Alcoholic consumption duration:** short (it was up to 10 years), moderate (11-20 years) and long (more than 20 years) [4].
- **Average amount per month was calculated by multiplying daily amount with frequency.**

With all these information the patients were further divided according to their type (country, whiskey and variable drinkers), amount and duration. Based upon these clinical and biochemical data, various prognostic markers (Maddrey's Discriminant function score [DF] score, Model for end-stage liver disease [MELD]...
score and Child-Pugh score) were calculated for each participant. The complications in this were made on the clinical examination, ultrasonography findings and CT scan.

After obtaining the data, it was then compared with the prognostic scores and analysed to obtain the results.

### 2.1 Statistical Analysis

All the statistical comparisons were done with GraphPad Instat Version 3.0 software and p values were obtained. Categorical data were compared with Fisher's Exact test, Chi-square test and Odds ratio with 95% confidence interval was calculated. Quantitative data were compared with analysis of variance (ANOVA) test.

---

**Fig. 1. Age of the study group**

**Fig. 2. Common presentations of the patients**

**Fig. 3. Type of liquor consumed**
Fig. 4. Amount of consumption

Biochemical findings of majority of the study population

Anemia, hyperbilirubinemia, leukocytosis

Fig. 5. Biochemical findings of majority of the study population

Table 1. Comparison of patients consuming different alcoholic beverages with the complication rates, prognostic markers and the P value

| Complication | Country (N=40) | Whiskey (N=4) | Variable (N=6) | P value |
|--------------|----------------|---------------|----------------|---------|
| Encephalopathy | 58.5% | 53.8% | 64.3% | 0.785 |
| Ascites | 72.9% | 61.5% | 71.4% | 0.676 |
| HRS | 35.2% | 38.5% | 32.1% | 0.917 |
| GI bleed | 59.7% | 46.2% | 60.7% | 0.62 |
| N/L ratio | 5.74 | 4.62 | 4.60 | 0.168 |

**PROGNOSIS AND OUTCOME**

| DF score | 55.4 | 50.22 | 52.08 | 0.86 |
| MELD score | 20.83 | 18.61 | 19.77 | 0.66 |
| Child-Pugh | 10.9 | 10.38 | 11.14 | 0.64 |
Table 2. Comparison of the effect of amount of alcoholic exposure with the complication rates, prognostic markers and P value

| UNIT/MONTH COMPLICATION | <360 (N=14) | 360-719 (N=19) | >720 (N=17) | P value |
|-------------------------|-------------|----------------|-------------|---------|
| Encephalopathy          | 50.9%       | 52%            | 71.83%      | 0.020   |
| Ascites                 | 67.27%      | 74.67%         | 73.24%      | 0.628   |
| HRS                     | 30.9%       | 34.67%         | 38%         | 0.707   |
| GI bleed                | 65.45%      | 52%            | 62%         | 0.256   |
| N/L ratio               | 4.64        | 5.44           | 6.21        | 0.035   |

**PROGNOSIS AND OUTCOME**

| UNIT/MONTH COMPLICATION | <360 (N=14) | 360-719 (N=19) | >720 (N=17) | P value |
|-------------------------|-------------|----------------|-------------|---------|
| Encephalopathy          | 50.9%       | 52%            | 71.83%      | 0.020   |
| Ascites                 | 67.27%      | 74.67%         | 73.24%      | 0.628   |
| HRS                     | 30.9%       | 34.67%         | 38%         | 0.707   |
| GI bleed                | 65.45%      | 52%            | 62%         | 0.256   |
| N/L ratio               | 4.64        | 5.44           | 6.21        | 0.035   |

Table 3. Comparison of the effect of duration of alcoholic exposure and the complication rate, prognostic markers and P value

| Duration | Upto 10 Years (N=13) | 11-20 Years (N=24) | >20 Years (N=13) | P value |
|----------|----------------------|--------------------|------------------|---------|
| Encephalopathy | 47.1%               | 61.2%              | 65.4%            | 0.13    |
| Ascites   | 76.5%                | 74.5%              | 63.5%            | 0.26    |
| HRS       | 33.3%                | 36.7%              | 32.7%            | 0.856   |
| GI bleed  | 64.7%                | 58.2%              | 55.8%            | 0.63    |
| N/L ratio | 5.59                 | 5.15               | 6.02             | 0.32    |

**PROGNOSIS AND OUTCOME**

| UNIT/MONTH COMPLICATION | Upto 10 Years (N=13) | 11-20 Years (N=24) | >20 Years (N=13) | P value |
|-------------------------|----------------------|--------------------|------------------|---------|
| Encephalopathy          | 47.1%                | 61.2%              | 65.4%            | 0.13    |
| Ascites                 | 76.5%                | 74.5%              | 63.5%            | 0.26    |
| HRS                     | 33.3%                | 36.7%              | 32.7%            | 0.856   |
| GI bleed                | 64.7%                | 58.2%              | 55.8%            | 0.63    |
| N/L ratio               | 5.59                 | 5.15               | 6.02             | 0.32    |

Table 4. Correlation of N/L ratio with complications and prognostic markers

| Complication | N/L<2.5 | N/L 2.5-4.5 | N/L>4.5 | P VALUE |
|--------------|---------|-------------|---------|---------|
| Encephalopathy | 44.8% | 60.7% | 61.4% | 0.25   |
| Ascites      | 82.75% | 69% | 71.6% | 0.36   |
| HRS          | 13.8% | 33.33% | 43.2% | 0.01   |
| GI bleed     | 62.07% | 61.9% | 55.6% | 0.66   |

**PROGNOSIS**

| Complication | N/L<2.5 | N/L 2.5-4.5 | N/L>4.5 | P VALUE |
|--------------|---------|-------------|---------|---------|
| DF score     | 41.12 | 50.5 | 63.3 | 0.03   |
| MELD score   | 16.09 | 19.08 | 23.22 | 0.004  |
| Child Pugh   | 10.11 | 10.57 | 11.23 | 0.043  |

3. RESULTS

Most of the patients in this study were in the age group of 45-55 years as shown in Fig. 1, consumed country made liquor as shown in Fig. 3 consumed more than the other type of alcohol consumers shown in Fig. 4 and came the most common presentation as ascites as showed in Fig. 2. The major biochemical findings in this study were anaemia, hyperbilirubinemia, leucocytosis shown in in Fig. 5 and elevated transaminase with the mean AST/ALT ratio 2.10 ± 0.8.

The biochemical findings showed that majority of the study population had anemia (82%), hyperbilirubinemia (81%), elevated transaminase with the mean AST/ALT ratio 2.10 ± 0.8 and leucocytosis (30%). The common complications seen in this study patients was mainly ascites (75%), HRS (hepatorenal syndrome) (32%), hepatic encephalopathy (60%) and gastrointestinal bleeding (55%). Among all these complications only hepatic encephalopathy correlated well with the amount (Table 2) of alcohol consumption.

NLR correlated well with all the three prognostic scores with DF score p<0.05, with MELD score [best correlation] p<0.001 and Child Pugh score p<0.05 and also NLR correlated well with amount of alcohol consumption (Table2).
complications hepatorenal syndrome was found to have a correlation with NLR and also hepatic encephalopathy also had a small correlation with NLR [Table 4].

4. DISCUSSION

Any pathological condition of the liver as a result of chronic and excessive alcohol consumption leading to a spectrum of conditions ranging from asymptomatic fatty liver to alcoholic hepatitis to end stage liver failure with clinical presentations jaundice, encephalopathy or ascites is defined as alcoholic liver disease. In the 2010 Global Burden of Disease (GDB) study, more than one million deaths (1,030,800 deaths representing 2.0% of all deaths, 1.4% of all deaths of women and 2.4% of all deaths of men) and 31,027,000 Disability Adjusted Life Years (DALYs) (1.2% of all DALYs, 0.8% of all DALYs for women, 1.6% of all DALYs for men) were due to liver cirrhosis [5].

Chronic excessive alcohol consumption is the most common cause of chronic liver diseases such as cirrhosis and hepatocellular carcinoma worldwide [5]. Alcoholic liver disease has a broad spectrum of complications [5]. The first study establishing the correlation of alcohol with cirrhosis was done by Mathew Baillie in 1793 [6]. Quantity and duration of alcohol consumption is known to play a play factor in the development of alcohol liver disease [1]. Even though till now many studies are taken up on this topic still the amount or the ideal safe drinking amount is not clear and vary from study to study [1,2].

In this study a majority consumed country made liquor but however this didn’t show any difference in the disease severity, complications or prognostic markers with the other types of liquor consumed. In relationship to the duration of alcohol consumption Child Pugh score was lower in patients with exposure less than 10 years. In this study majority of the patients 72% consumed alcohol for more than or equal to 20 years. Comparing this study with the study of Narawane et al. we can conclude that Indians tend to develop ALD in a shorter span of exposure when in comparison with the western population [7]. This fact may be considered due to the genetic polymorphism of genes like ADH, ALDH, CYP2E1, TNF alpha and IL10 [8,9].

Hepatic encephalopathy was more common in people who consumed more alcohol than people with lesser consumption. A significant correlation of the amount of alcohol consumption with the 3 prognostic scores was seen to be higher in heavy consumers of alcohol. Narawane et al. [7] also had a similar comparison and found that ALD patients consumed more alcohol than non-liver disease people. An important finding in this study is that NLR had a positive correlation with the 3 prognostic markers, with hepatic encephalopathy and also with the amount of alcohol consumption.

5. CONCLUSION

Alcoholic liver disease is a disease with a high morbidity. The main intervention should be early detection and alcohol abstinence in the initial stage. In this study we conclude that there is significant dose dependent relation of ALD along with its complications, prognostic markers and NLR with the amount, type and duration of alcohol consumption. Although the type of alcohol consumption didn't have much influence on the results, amount of intake had a correlation with NLR. And also, the duration of alcohol consumption had a positive correlation mainly with Child Pugh score. Further studies on a larger population with an even wider variety of alcohol consumption may throw more light on this topic.

DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

CONSENT

A pre-informed written consent was obtained from each participant (or attendant if patient was unconscious).

ETHICAL APPROVAL

This study was approved by the ethics committee.

COMPETING INTERESTS

Authors have declared that no competing interests exist.
REFERENCES

1. Hozo I, Mirić D, Ljutić D, Giunio L, Andelinović S, Bojić L. Relation between the quantity, type and duration of alcohol drinking and the development of alcoholic liver cirrhosis. Med Arh. 1995;49:5-8.

2. Becker U, Deis A, Sorensen TI, Gronbaek M, Borch-Johnsen K, Muller CF, et al. Prediction of Risk of Liver Disease by Alcohol intake, Sex, and Age: A prospective population study. Hepatology. 1996;23:1025-9.

3. Mendenhall C, Roselle GA, Gartside P, Moritz T. Relationship of protein calorie malnutrition to alcoholic liver disease: a reexamination of data from two Veterans Administration Cooperative Studies. Alcohol Clin Exp Res. 1995;19:635-41.

4. Nand N, Malhotra P, Dhoot DK. Clinical profile of alcoholic liver disease in a tertiary care centre and its correlation with type, amount and duration of alcohol consumption. The Journal of the Association of Physicians of India. 2015;63(6):14-20.

5. Rehm J, Samokhvalov AV, Shield KD. Global burden of alcoholic liver diseases. Journal of hepatology. 2013;59(1):160-8.

6. Bailie M. Alcohol and the liver. Gut. 1971;12(3):222.

7. Narawane NM, Bhatia S, Abraham P, Sanghani S, Sawant SS. Consumption of 'country liquor' and its relation to alcoholic liver disease in Mumbai. J Assoc Phys Ind 1998;46:510-13.

8. Vaswani M, Prasad P, Kapur S. Association of ADH1B and ALDH2 gene polymorphisms with alcohol dependence: a pilot study from India. Hum Genomics 2009;3:213-20.

9. Bhaskar LVKS, Thangaraj K, Osier M, Reddy AG, Rao AP, Singh L et al. Single nucleotide polymorphism of the ALDH2 gene in six Indian populations. Ann Hum Biol 2007;34:607-19.

© 2021 Aswathi and Vikrannth; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle4.com/review-history/74894