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Original Research Article

PREVALENCE AND OUTCOME OF CONGESTIVE HEART FAILURE IN CHRONIC LIVER DISEASED PATIENTS: A RETROSPECTIVE STUDY FROM A TERTIARY CARE CENTER

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Abstract

Background: Chronic liver disease (CLD) is a major health problem worldwide, affecting millions of people. The prevalence of congestive heart failure (CHF) in CLD patients is increasing, and there is a need to understand the impact of this condition on patient outcomes. Hence, this study is a retrospective study to evaluate the prevalence and functional outcome by New York Heart Association Score (NYHA) functional Scoring system among adults with age 45 to 65 years with known CLD for 3 years. **Materials and Methods:** The study was retrospective observational research conducted from May 2024 to October 2023 at the Department of General Medicine, Government Medical College, Ongole. The study included patients aged 45-65 years, known CLD patients in the last three years, and hospital stay up to 12 days. **Result:** The study found no mortality rate in CLD patients with class I and II NYHA scores, while mortality was observed in a minor proportion of participants with class III NYHA score. However, mortality was highest in CLD patients with class IV NYHA score. Most participants were males, aged >55 years, and had hypertension, diabetes mellitus, and dyslipidemia. The association of NYHA score with mortality was significant, with values of X2 5.16, 4.92, and 7.22, respectively. **Conclusion:** Comorbidities often lead to CLD, a higher prevalence of CHF, independent of glycemic management, drugs, risk factors, and metabolic syndrome. Future research should clarify molecular processes and predict CHF development.

INTRODUCTION

Globally, both chronic liver disease (CLD) and congestive heart failure (CHF) are major health issues with great morbidity and mortality.[1] These two diseases interact in a complicated way, whereby one could aggravate the other. Considered to range from hepatic steatosis to cirrhosis, CLD has systemic effects, including cardiovascular problems.[2] Subjects of growing clinical interest and investigation are the frequency and effects of congestive heart failure in patients with CLD.[1-3]

For various pathophysiological reasons, patients with CLD are more likely to develop CHF. Portal hypertension, a common outcome of severe liver illness, causes hyperdynamic circulation and higher cardiac output, which over time can lead to cardiac dysfunction.[4] Furthermore, liver disease causes systemic inflammation and metabolic abnormalities, including altered lipid metabolism and insulin resistance, contribute to cardiovascular stress.[5] Further complicating the clinical picture in these patients is the existence of cirrhotic cardiomyopathy, a disorder characterized by a decreased heart response to stress.[6]

The coexistence of CHF and CLD poses special therapeutic and diagnostic problems.[6] Diagnostically, the symptoms of congestive heart failure such as edema and tiredness can coincide greatly with those of liver illness, therefore hindering early identification and suitable treatment.[7] When treating CHF in the context of CLD, it is important to be aware of the liver's decreased ability to work and the possibility that cardiac drugs could be harmful to the liver.[8]

Improving patient outcomes depends on understanding the frequency of CHF in CLD patients. Different studies have found different rates, which are often affected by the cause of the liver disease, like viral hepatitis, alcohol-related liver disease, or non-alcoholic fatty liver disease.[9,10] The typically poor outcomes for these patients are defined by higher rates of hospitalization, lower quality of life, and more deaths compared to individuals with CHF

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alone.[9] Early intervention plans and multidisciplinary care techniques are crucial to meet the complicated needs of this patient population and raise prognostic results. Hence, this study is a retrospective study to evaluate the prevalence and functional outcome by New York Heart Association Score (NYHA) functional Scoring system among adults with age 45 to 65 years with known CLD for 3 years.

MATERIALS AND METHODS

The present study was retrospective observational research spanning eight months (May 2024 – October 2023). The manner of the sampling was intentional Sampling. The site of the present study was Department of General Medicine, Government Medical College, Ongole. The inclusion criteria were age 45 to 65 years, known CLD patients in the last three years, and hospital stay time up to 12 days as IP records. Patients age group below 45 years and over 65 years, patients with hepatorenal syndrome, and patients with cardiorenal syndrome were excluded.

The patients' case files provided the data for collecting information. On day one, the NYHA scale helped one to determine the patients' functional level. For the 12th day, NYHA scale was also used in line with history and test results. In case of discharge, LAMA or death, the NYHA scale was used that specific day.

Statistical analysis: Descriptive statistics on demographic information, risk factors, length of illness were recorded; Student's t-test was conducted for the NYHA scale results of day 1 and day 12. The chi-square test was used to find relationships between demographic information and risk factors and NYHA scale day 1 score.

RESULTS

[Table 1] shows the demographic details and risk factors of the study population. Out of a total of 49 participants, nearly three-quarters (n=47/49, 96%) were males while the rest (n=2/49, 4%) were females. The mean age of study participants was 58.8±7.69 years. More than half (n=35/49, 71%) of our participants were aged > 55 years. With regards to clinical factors, (n=43/49, 88%) of the participants had hypertension while (n=37/49, 76%) had diabetes mellitus. Additionally, (n=36/49, 73%) of the study participants had hyperlipidemia.

Table 1: Demographic details, risk factors of the study population.

Parameters		Number (%)
Gender	Male	47
	Female	2
Age (years)	45 – 55	14
	56 - 65	35
Hypertension	Yes	43
	No	6
Diabetes mellitus	Yes	37
	No	12
Hyperlipidemia	Yes	36
	No	13

Table 2: Association of NYHA with mortality, demographic, and risk factors of the study population.

Parameters		Class I (ordinary physical activity does not cause undue fatigue, palpitations, dyspnea and/or angina) N=6	Class II (ordinary physical activity does cause undue fatigue, palpitations, dyspnea and/or angina) N=12	Class III (Less than ordinary physical activity does cause undue fatigue, palpitations, dyspnea and/or angina) N=26	Class IV (Fatigue, palpitations, dyspnea and/or angina occur at rest) N=5
Mortality	Yes	0	0	2	2
	No	6	12	24	3
Gender	Male	6	12	25	4
	Female	0	0	1	1
Age (years)	45 – 55	6	3	4	1
	56 – 65	0	9	22	4
Hypertension	Yes	6	10	22	5
	No	0	2	4	0
Diabetes mellitus	Yes	1	8	23	5
	No	5	4	3	0
Dyslipidemia	Yes	1	8	22	5
	No	5	4	4	0

Table 3: Liver function tests in the present study population.

Parameters	Day one (n=49)	Day 12 (n=49)	P Value
Aspartate transaminase (AST; U/L)	43.4± 12.6	45.9 ± 14.8	>0.05
Alanine transaminase (ALT; U/L)	56.34 ± 14.5	57.8 ± 24.2	>0.05
Alkaline phosphatase (ALP; IU/L)	124.1± 7.9	126 ± 4.9	>0.05

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Serum bilirubin (mg/dl)	0.7 ± 0.2	0.9 ± 1.1	>0.05

[Table 2] shows the association of NYHA with mortality, demographic, and risk factors of the study population. There was no mortality rate in CLD patients with class I and class II NYHA score (n=6/6 and 12/12, 100%) while mortality was observed in a minor proportion (n=2/26, 8%) of participants with class III NYHA score. In contrast, mortality was observed in the highest proportion (n=2/5,40%) of CLD patients with class IV NYHA score. Most of the participants with class I NYHA score (n=6/6, 100%), class II NYHA score (n=12/12, 100%), class III NYHA score (n=25/26, 96%), and class IV (n=4/5.80%) were males in comparison to females, showing a significant difference statistically ($p < 0.001$). More than half of the participants (n=5/10, 50%) with class II, class III, and class IV NYHA score were aged >55 years while 14 CLD patients were aged ≤55 years, this difference again was statistically significant ($p < 0.05$). A higher proportion of participants with class III NYHA score (n=22/26, 85%) and class IV NYHA score (n=5/5, 100%), belonged to the hypertensive group ($p < 0.05$). Furthermore, there was significant association of NYHA score with diabetes mellitus ($p< 0.05$) and hyperlipidemia ($p< 0.05$).

The association of hypertension, diabetes mellitus, and dyslipidemia with CLD affected with CHF patients in mortality was observed to be significantly associated with the values of X2 5.16; $P<0.05$, X2 4.92; $p < 0.05$ and X2 7.22; $P<0.05$ respectively. On the other hand, we did not find any association between liver enzymes in CLD patients affected with CHF.

DISCUSSION

We desperately need to find the prevalence of CLD in the study population and assess its correlation with CHF. While the extent of the CLD problem in CHF patients remains unknown, recent acknowledgements indicate that CLD significantly contributes to their disease burden.[5,6,11] While further research is necessary to establish the independent role of CLD as a CHF risk factor, it is evident that CLD is associated with CHF in individuals with comorbidities.[7,9] Indeed, the effect of CLD on CHF risk warrants special consideration in light of the implications for screening and surveillance policies in developing countries.

To our knowledge, this is a study with the particular objectives of determining the prevalence of CLD and evaluating the correlation of CLD with CHF. This study reveals that the population has a rather high incidence of CLD, as determined by patient history and distinctive sonographic characteristics. According to ultrasonic analysis, CLD is present in all of our population and is the most common cause of any kind of hepatic disease.

Fascinatingly, this study provides more evidence that a normal serum ALT and AST level has little diagnostic or predictive value when evaluating patients for CLD, since 88% of our CLD patients had normal ALT and AST levels. With CHF, serum ALT and AST levels appear to be insensitive CLD indicators. Indeed, people with normal liver enzymes may exhibit the entire histological spectrum of CLD,[12] making it impossible to consistently rule out the presence of more advanced stages of CLD.

Our study design makes it impossible for us to establish causality in the link between CLD and CHF and ascertain if the greater CHF prevalence among CLD patients influences long-term mortality. Our results suggest that the link between CLD and CHF mostly reflects the general, adverse influence of the metabolic syndrome phenotype, primarily insulin resistance.[6,7] Although our results were adjusted for metabolic syndrome, a condition closely linked to insulin resistance, we did not directly measure insulin resistance (by glucose clamp) in our study cohort, so we cannot be certain that identical results could be obtained after additional adjustment for this CHF risk factor. However, conducting the glucose clamp frequently would not be feasible in a large-scale epidemiological investigation.

Studies have clearly established a strong correlation between CLD and increased CHF prevalence in comorbidities.[6-8] However, in that study, the link between CLD and CHF disappeared after accounting for metabolic syndrome. The increased sample size of this study, which gave more statistical power and allowed more extensive adjustment for possible confounders, helps to mostly explain these apparently inconsistent results. Furthermore, a follow-up study, independent of conventional risk variables, supports our results by linking the presence of metabolic syndrome and CLD to higher CHF incidence in comorbidities.[7,8] Others have cross-sectionally shown that those with somewhat raised blood ALT levels—as surrogate markers of CLD—had a higher CHF risk (as computed by the Framingham risk score).[13] It is unknown right now whether treating CLD will eventually help to prevent CHF development. Interventions that have been proven successful in avoiding CHF in comorbid individuals, such as weight loss and therapy with insulin-sensitizing antidiabetic drugs, may still help CLD.[14,15]

Overall, these findings may have public health and clinical implications. Our findings indicate that the majority of patients with comorbidities suffer from CLD; previous studies showing comorbidity as an independent predictor of advanced liver disease in CLD suggest considering referral to a hepatologist. This will be especially crucial once we develop a suitable therapy and validate better noninvasive approaches for assessing disease severity. Recent studies, which link CLD to higher all-cause death,[16,17] and predict the risk of future CHF

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events,[8,17] generally align with our results. The degree of CLD histology is associated with greater carotid intima-media thickness and plaques.[18] As a result, there is mounting evidence that CLD is not only a CHF marker, but also directly or indirectly involved in its pathophysiology. Although the release of proatherogenic mediators from the liver, including C-reactive protein, in-terleukin-6, and plasminogen activator inhibitor-1,[18,19] has been widely addressed elsewhere, the putative molecular mediators between CLD and CHF have also been thoroughly examined here.

CONCLUSION

Finally, our findings suggest that individuals with comorbidities often have CLD, which is associated with a higher prevalence of CHF. The correlation between CLD and CHF seems to be independent of glycemic management, drugs, conventional risk factors, and the existence of the metabolic syndrome. These findings thereby support the theory that the identification of CLD in comorbidities could assist in CHF risk prediction. Future experimental and follow-up research is required to clarify the probable molecular processes between CLD and CHF and to ascertain whether CLD forecasts the development and progression of CHF.

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