e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2023; 15(4); 1112-1123

Original Research Article

Biochemical Insights into Chronic Alcoholism: A Prospective Observational Study on Liver Function and Oxidative Stress Markers

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Received: 26-03-2023/ Revised: 10-04-2023/ Accepted: 19-04-2023

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Conflict of interest: Nil

Abstract

Chronic alcoholism, defined by the World Health Organization as habitual consumption of around 75ml of pure ethanol or 200ml or more of 40-60% alcohol, is a pressing public health issue worldwide. Biochemical tests have been instrumental in uncovering the complexities of chronic alcoholism, providing valuable prognostic insights. In this study, we investigated the levels of total bilirubin, direct bilirubin, indirect bilirubin, liver enzymes (SGPT, SGOT, ALP, GGT), serum calcium, serum magnesium, and malondialdehyde (MDA) in chronic alcoholics compared to healthy controls.

A prospective observational case-control study was conducted in the Department of Biochemistry with a total of 100 male subjects, 50 of whom were chronic alcoholics (Group 1), and 50 age and sex-matched healthy controls (Group 2). Our findings indicated that chronic alcoholics had a mean age of 45.06± 8.83 years, while the mean age of controls was 42.44 ± 8.91 years.

Our analysis revealed that chronic alcoholics had significantly higher levels of total bilirubin and direct bilirubin compared to healthy controls. Indirect bilirubin levels were also higher in the chronic alcoholics, but the difference was not statistically significant. Furthermore, chronic alcoholics had significantly higher levels of SGPT, SGOT, ALP, and GGT enzymes compared to healthy controls. Conversely, the mean value of serum calcium was significantly lower in the chronic alcoholics. Finally, our study demonstrated that malondialdehyde (MDA) levels were significantly higher in the chronic alcoholics compared to the healthy controls.

In conclusion, our study provides compelling evidence of the detrimental effects of chronic alcoholism on liver function and oxidative stress markers. These findings underscore the importance of early diagnosis and intervention in the management of chronic alcoholism, which is critical for improving patient outcomes and reducing the burden of this debilitating disease.

Keywords: Serum Calcium, Serum Magnesium, SGPT, SGOT, MDA, Alcoholism.

Introduction

Alcoholism is a grave health concern in India and affects people of all socioeconomic and ethnic backgrounds. The World Health Organization defines chronic alcoholism as the habitual consumption of approximately 75ml of pure ethanol or 200ml or more of 40-60% alcohol, such as whisky, brandy, rum, gin, or country liquor, daily for at least five years [1]. Alcohol ranks third among the leading preventable causes of death in India, following smoking and obesity. It is responsible for a significant number of fatalities, including homicides, suicides, and motor vehicle accidents [2]. Chronic alcoholism is linked to the risk of multi-organ failure due to its ability to cause micronutrient deficiency and toxicity in all body tissues [3].

Alcohol is a central nervous system depressant, and the extent of depression is directly proportional to the amount consumed. Its effects include euphoria, loss of social skills, ataxia, drowsiness, coma, deep coma, and even death. Since alcohol can permeate all tissues in the body, it can cause harm to various organs such as the nervous system, liver, pancreas, gastrointestinal tract, heart, endocrine gland, hematopoietic system, and bones. Furthermore, alcohol is taken up unchanged from the stomach into the bloodstream, making it a rapid energy source. Chronic alcoholism disrupts the essential balance among carbohydrates, proteins, and fats, leading to malnutrition and harmful consequences. Excessive alcohol intake increases the need for essential nutrients, worsening any existing deficiency [1-3].

In the human body, the liver is the primary organ responsible for the oxidation of alcohol. Chronic alcoholism can lead to fatty liver and toxicity due to the accumulation of acetaldehyde dehydrogenase. Electrolyte disturbances may also occur, leading to a higher incidence of arrhythmias [4]. Ethanol can cause disturbances in acid-base homeostasis, as well as monovalent and divalent cation metabolism [5]. Chronic alcohol abuse can lead to various liver injuries ranging from mild fatty infiltration to cirrhosis and hepatocellular carcinoma [6].

Alcohol is primarily metabolized in the liver, making it vulnerable to oxidative stress-induced injury due to the generation of reactive oxygen metabolites [7]. During the detoxification process of xenobiotics like alcohol, free radicals are generated through the Microsomal Ethanol Oxidizing System and by leakage from the mitochondrial Electron Transport Chain (ETC) [8]. Malondialdehyde (MDA), a product and marker of oxidative stress-mediated lipid peroxidation, is generated during this process [9].

Biochemical tests have aided in better understanding chronic alcoholism, providing essential prognostic information. This study aims to estimate the levels of various substances, including total bilirubin, direct bilirubin, indirect bilirubin, liver enzymes (SGPT, SGOT, ALP, GGT), serum calcium, serum magnesium, and malondialdehyde (MDA), in cases of chronic alcoholism and compare them with those of healthy controls of the same age and sex.

Objectives of the Study

1. To estimate the levels of total bilirubin and direct bilirubin in cases of chronic alcoholism and to compare them with that of healthy controls of same age and sex.
2. To estimate the levels of serum glutamate pyruvate transaminase (SGPT), serum glutamate oxaloacetate transaminase (SGOT), alkaline phosphatase (ALP), and gamma-glutamyl transferase (GGT) in cases of chronic alcoholism and to compare them with that of healthy controls of same age and sex.

3. To estimate the levels of serum Calcium in cases of chronic alcoholism and to compare them with that of healthy controls of same age and sex.
4. To estimate the levels of serum Magnesium in cases of chronic alcoholism and to compare them with that of healthy controls of same age and sex.
5. To estimate the levels of malondialdehyde (MDA), a product of lipid Peroxidation as an index of oxidative stress in cases of chronic alcoholism and to compare them with that of healthy controls of same age and sex.

Materials and Methods

Study Design:

This was a prospective observational case-control study conducted in the Department of Biochemistry at Andhra Medical College, Visakhapatnam, Andhra Pradesh, India. The study was approved by the institutional ethics committee and was carried out in accordance with the Declaration of Helsinki.

Study Participants:

One hundred male subjects participated in the study, comprising 50 chronic alcoholics (Group 1) and 50 age and sex-matched healthy controls (Group 2). All participants provided written informed consent before participating in the study.

Inclusion Criteria:

Participants in Group 1 were included if they had a history of chronic alcoholism, defined by the World Health Organization as habitual consumption of around 75ml of pure ethanol or 200ml or more of 40-60% alcohol. Participants in Group 2 were included if they had no history of alcohol abuse or dependence and were free of any chronic illness.

Exclusion Criteria:

Participants in both groups were excluded if they had a history of liver disease, renal impairment, diabetes, hypertension, or any other chronic illness. Additionally, participants were excluded if they were taking any medications that could interfere with the study parameters.

Data Collection:

After obtaining informed consent, detailed medical histories were obtained from all participants. Anthropometric measurements, including height, weight, and body mass index (BMI), were recorded. Blood samples were collected from all participants after an overnight fast of at least 10 hours. Serum levels of total bilirubin, direct bilirubin, indirect bilirubin, SGPT, SGOT, ALP, GGT, serum calcium, serum magnesium, and malondialdehyde (MDA) were measured using standard laboratory methods.

Statistical Analysis

All statistical analyses were performed using SPSS version 20.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize the demographic and clinical characteristics of the study participants.

Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables were expressed as frequencies and percentages. Student's t-test was used to compare continuous variables between the two groups, and a p-value of less than 0.05 was considered statistically significant.

Results & Discussion

Table 1: Age distribution between Cases & Controls

			Age			Total
			31-40 years	41-50 years	51-60 years	
Groups	Cases	n	20	14	16	50
		%	40.0%	28.0%	32.0%	100.0%
	Controls	n	28	10	12	50
		%	56.0%	20.0%	24.0%	100.0%
Total		n	48	24	28	100
		%	48.0%	24.0%	28.0%	100.0%

Parameter	Group	No	Mean ± SD	t-value	p- value*
Age	Cases	50	45.06 ± 8.83	1.476	0.143
	Controls	50	42.44 ± 8.91		

Table 2: Comparison of levels of Total Bilirubin, Direct Bilirubin, Indirect Bilirubin Between Cases & Controls

Parameter	Group	No	Mean ± SD	t-value	p- value*
Total Bilirubin	Cases	50	1.05 ± 0.60	2.203	0.03
	Controls	50	0.83 ± 0.31		
Direct Bilirubin	Cases	50	0.27 ± 0.28	3.195	0.002
	Controls	50	0.13 ± 0.06		
In-Direct Bilirubin	Cases	50	0.77 ± 0.43	1.054	0.294
	Controls	50	0.70 ± 0.27		

The mean value of Total Bilirubin and Direct Bilirubin significantly increased in cases when compared to controls.

The mean value of indirect bilirubin increased in cases when compared to controls but which is statistically insignificant.

Table 3: Comparison of levels of Liver Enzymes (SGPT, SGOT, ALP and GGT) Between Cases & Controls

Parameter	Group	No	Mean ± SD	t-value	p- value*
SGPT	Cases	50	51.96 ± 52.30	2.044	0.044
	Controls	50	35.97 ± 17.98		
SGOT	Cases	50	53.99 ± 45.12	4.273	0.005
	Controls	50	26.18 ± 9.03		
ALP	Cases	50	100.42 ± 19.34	2.962	0.004
	Controls	50	89.14 ± 18.73		
GGT	Cases	50	82.20 ± 84.00	4.501	0.001
	Controls	50	28.08 ± 13.13		

The mean value of SGPT, SGOT, ALP and GGT significantly increased in cases when compared to controls.

Table 4: Comparison of levels of Serum Calcium Between Cases & Controls

Parameter	Group	No	Mean ± SD	t-value	p- value*
Serum Calcium	Cases	50	8.49 ± 0.43	-7.08	0.300
	Controls	50	9.10 ± 0.42		

The mean value of serum calcium significantly decreased in cases when compared to controls

Table 5: Comparison of levels of Serum Magnesium Between Cases & Control

Parameter	Group	No	Mean ± SD	t-value	p- value*
Serum Magnesium	Cases	50	1.72 ± 0.13	-4.057	0.000
	Controls	50	1.86 ± 0.20		

The mean value of serum magnesium significantly decreased in cases when compared to controls

Table 6: Comparison of levels of Malondialdehyde (MDA) Between Cases & Controls

Parameter	Group	No	Mean ± SD	t-value	p- value*
MDA	Cases	50	5.14 ± 2.43	2.95	0.004
	Controls	50	4.01 ± 1.18		

The mean value of malondialdehyde (MDA) significantly increased in cases when compared to controls.

Total bilirubin, direct bilirubin and indirect bilirubin in chronic alcoholism: In this study, there was a significant increase in total bilirubin levels with a mean value (1.05 ± 0.60mg/dL) in chronic alcoholics as compare to controls (0.83 ± 0.31 mg/dl).

In this study, there was a significant increase in direct bilirubin levels with a mean value (0.27± 0.28mg/dL) in chronic alcoholics as compare to controls (0.13 ± 0.06mg/dL).

Our findings were in accordance with the studies conducted by Torkadi PP *et al* [11], Singh M *et al* [12], and De Marchi S *et al* [5].

Table 7: Comparison of levels of Total Bilirubin Between Cases & Controls in Present versus Previous Studies

Study	Controls	Cases	P-value
Torkadi PP *et al*.,[11]	1.14 ± 0.219	3.94 ± 2.88	<0.01
Singh M *et al*.,[12]	0.54 ± 0.31	2.40 ± 0.58	<0.001
De Marchi S *et al*.[5]	0.5 ± 0.3	1.3 ± 0.7	<0.001
Present study	0.83 ± 0.31	1.05 ± 0.60	<0.03

Table 8: Comparison of levels of Direct Bilirubin Between Cases & Controls in Present versus Previous Studies

Study	Controls	Cases	P-value
Torkadi PP *et al*.,[11]	0.506 ± 0.17	2.01 ± 1.86	<0.01
Singh M *et al*. [12]	0.11 ± 0.13	0.90 ± 0.49	<0.001
Present study	0.13 ± 0.06	0.27 ± 0.28	<0.002

Biochemical elevations of total bilirubin and direct bilirubin can be attributed to severe hepatic damage seen in chronic alcoholics as compared to controls.

In this study, there was an increase in indirect bilirubin levels with a mean value (0.77 ± 0.43 mg/dL) in chronic alcoholics as compare to controls (0.70 ± 0.27 mg/dL) but the p value was not significant. Our findings did not correlate with any study for indirect bilirubin.

Table 9: Comparison of levels of Indirect Bilirubin Between Cases & Controls in Present versus Previous Studies

Study	Controls	Cases	P-value
Torkadi PP *et al*.,[11]	1.09 ± 0.05	1.93 ± 1.02	<0.01
Singh M *et al*.,[12]	]0.43 ± 0.18	1.50 ± 0.09	<0.001
Present study	0.70 ± 0.27	0.77 ± 0.27	<0.294

Liver enzymes in chronic alcoholism: SGPT & SGOT

In this study, there was a significant increase in SGPT levels with a mean value (51.96 ± 52.30 U/L) in chronic alcoholics as compare to controls (35.97 ± 17.98 U/L).

In this study, there was a significant increase in SGOT levels with a mean value (53.99 ± 45.12 U/L) in chronic alcoholics as compare to controls (26.18± 9.03U/L). Our findings are in accordance with the study conducted by Gill GK *et al*.[13], De Marchi S *et al* [5] and Torkadi PP *et al*.,[11]

Table 10: Comparison of levels of SGPT Between Cases & Controls in Present versus Previous Studies

Study	Controls	Cases	P-value
Gill GK *et al*.[13]	29.30 ± 8.45	61.96± 15.47	<0.003
De Marchi S *et al*.[5]	23± 12	33± 21	<0.02
Torkadi PP *et al*.[11]	24.22 ±7.31	49.32±20.99	<0.01
Present study	35.97±17.98	51.96±52.30	<0.044

Table 11: Comparison of levels of SGOT Between Cases & Controls in Present versus Previous Studies

Study	Controls	Cases	P-value
Gill GK *et al*.[13]	31.69± 8.24	52.57± 15.73	<0.002
De Marchi S *et al* [5]	24± 12	44± 35	<0.02
Torkadi PP *et al*.,[11]	23.78 ± 5.56	125.58± 57	<0.01
Present study	26.18±9.03	53.99±45.12	<0.05

The elevated SGPT and SGOT levels are the predictors of underlying liver injury. The liver parenchymal cells are found to be sensitive to alcohol. Acetaldehyde which results from the catalytic activity of alcohol dehydrogenase is highly reactive and is found capable of making adducts with proteins and nucleic acids. This adduct formation with membrane protein can be considered as the first step in liver cell injury. In addition to the leakage of these enzymes into the circulation due to necrosis of hepatocytes, these enzymes (SGOT in particular) are also induced by the presence of alcohol [14].

Alkaline phosphatase (ALP) & Gamma glutamyl transferase (GGT)

In this study, there was a significant increase in ALP levels with a mean value (100.42 ± 19.34 U/L) in chronic alcoholics as compare to controls (89.14 ± 18.73U/L). Our findings

are in accordance with the study conducted by Torkadi PP *et al*.,[11] and Singh M *et al*.[12]

In this study, there was a significant increase in GGT levels with a mean value (82.20 ± 84.00 U/L) in chronic alcoholics as compare to controls (28.08 ± 13.13 U/L). Our findings are in accordance with the study conducted by Torkadi PP *et al*.,[11] , Singh M *et al*.,[12] and De Marchi S *et al*.[5]

Table 12: Comparison of levels of Alkaline Phosphatase Between Cases & Controls in Present versus Previous Studies

Study	Controls	Cases	P-value
Torkadi PP *et al*.,[11]	49.68± 13.48	117.52± 43.93	<0.01
Singh M *et al*.,[12]	4.90 ± 2.05	30.25 ± 3.10	<0.001
Present study	89.14 ± 18.73	100.42 ± 19.34	<0.004

Table 13: Comparison of levels of Gamma Glutamyl Transferase Between Cases & Controls in Present versus Previous Studies

Study	Controls	Cases	P-value
Torkadi PP *et al*.,[11]	24± 7.6	55.12 ± 17.02	<0.01
Singh M *et al*.,[12]	10.2 ± 3.5	82.54±16.42	<0.001
De Marchi S *et al*.[5]	29±6	150±182	<0.001
Present study	28.08 ± 13.13	82.20 ± 84.00	<0.05

High ALP & GGT values may indicate enzyme induction by alcohol and mild cholestasis, thus reflecting on the severe damage in chronic alcoholics. Rise in serum ALP is definitely a better indicator of cholestasis than GGT [15]. Elevated GGT in association with increased serum ALP shows that ALP in serum is hepatic in origin, as GGT is not raised in bone diseases. The increase in serum GGT in chronic alcoholics may be due hepatic microsomal enzyme induction by alcohol [16-19]

Serum calcium in chronic alcoholism:

In this study, there was a significant decrease in serum calcium levels with a mean value (8.49± 0.43 mg / dl)in chronic alcoholics as compare to controls (9.10 ±0.42 mg/ dl). Our findings are in accordance with the study conducted by Gill GK *et al*.[13], De Marchi S *et al*.[5], Asha Lata *et al*. [3], Abbott *et al* [20].

Table 14: Comparison of levels of Serum Calcium Between Cases & Controls in Present versus Previous Studies

Study	Controls	Cases	P - value
Gill GK *et al*. [13]	9.41 ± 095	8.11±0.93	<0.001
AshaLata *et al*. [3]	9±0.23	7.9 ±0.31	<0.0001
Present study	9.10 ± 0.42	8.49 ± 0.43	<0.05

In this study, serum calcium levels are significantly decreased in chronic alcoholics compared with control groups and this can be attributed to reduced intake or inadequate absorption due to vitamin D deficiency as it is essential for adequate absorption of calcium from the gut. Malabsorption, a condition seen commonly in chronic alcoholics has resulted in poor absorption of vitamin D and other fat-soluble vitamins. The unabsorbed fats in the intestine form insoluble soaps with calcium which lead to

further decrease in the amount of available calcium [21]. The calcium depletion in chronic alcoholics may be associated with alcohol induced hypoparathyroidism and parathyroid hormone resistance of skeletal muscle as well as with the decrease of serum osteocalcin [22]. In addition, low serum magnesium levels in alcoholics diminish the sensitivity of parathyroid gland to low serum calcium levels as a result of which the gland fails to secrete hormones in sufficient amount when the serum calcium levels drop [21].

Magnesium in chronic alcoholism:

In this study, there was a significant decrease in serum magnesium levels with a mean value (1.72± 0.13 mg/dL) in chronic alcoholics as compare to controls (1.86± 0.20 mg/dl). Our findings are in accordance with the study conducted by Gill GK *et al*.[13], Randall *et al*. [22], Asha lata *et al*., [3] De Marchi S *et al*.[5] Gill GK *et al*.[13] in their study, observed serum magnesium is significantly decreased in chronic alcoholics when compared to controls and also found the levels to be further diminished in progression with age.

Table 15: Comparison of levels of Serum Magnesium Between Cases & Controls in Present versus Previous Studies

Study	Controls	Cases	P - value
Gill GK *et al*. [13]	2.33±0.37	1.64±0.30	<0.001
De Marchi S *et al*.[5]	1.80±0.38	1.40± 0.15	<0.002
Asha lata *et al*.[3]	2.2± 0.21	1.5±0.09	<0.001
Present study	1.86 ± 0.20	1.72 ± 0.13	<0.05

In this study, serum magnesium levels are significantly decreased in chronic alcoholics compared with controls as reduced secretion of parathyroid hormone in them causes the enhanced fractional excretion of magnesium[23]. It has been observed that hypomagnesemia in chronic alcoholics could be due to the net effect of various factors such as dietary deficiency, reduced intestinal absorption, and enhanced sensitivity to alcohol related toxicity [24,25].

Alcohol, owing to its action as a diuretic, increases the magnesium excretion with increase in alcohol ingestion, which might be due to the decreased renal sensitivity of vasopressin.

Hypomagnesemia causes cardiac arrhythmias, mental and emotional changes and hence needs proper evaluation, appropriate correction during the management of chronic alcoholics in IC Units, etc.[26]

Malondialdehyde in chronic alcoholism:

In this study, there was a significant increase in MDA levels with a mean value (5.14± 2.43 nmol/ ml) in chronic alcoholics as compared to controls (4.01 ± 1.18nmol/ml). Our findings are in accordance with the study conducted by Singh M *et al*.,12, Subhani TF *et al*. 27, and Zhang X *et al* [28]

Table 16: Comparison of levels of Malondialdehyde (MDA) Between Cases & Controls in Present versus Previous Studies

Study	Controls	Cases	P - value
Singh M *et al*.,[12]	3.61±0.63	9.02± 1.21	<0.001
Subhani TF *et al*.[27]	5.04±0.95	6.08±0.71	<0.001
Present study	4.01 ± 1.18	5.14 ± 2.43	<0.004

The rise in serum MDA indicated that any oxidative stress incurred sufficiently could cause free radical mediated peroxidation of lipid component in cell membrane, thus MDA is a good indicator for oxidative stress in chronic alcoholics.

Conclusion

among carbohydrates, proteins, and fats, thereby leading to detrimental outcomes. Alcohol consumption can impede the absorption and retention of specific vitamins essential for healthy bodily functions. Moreover, in cases of chronic alcoholism, serum glutamic-pyruvic transaminase (SGPT) and serum glutamic-oxaloacetic transaminase (SGOT) levels tend to rise as a result of liver injury and leakage of these enzymes into the bloodstream. Elevated alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT) levels often indicate mild cholestasis and enzyme induction.

Hypocalcemia, which is prevalent in chronic alcoholism, may be attributable to several factors such as vitamin D deficiency, alcohol-induced hypoparathyroidism, and parathyroid hormone resistance of skeletal muscle, along with a decrease in serum osteocalcin levels. Moreover, hypomagnesemia caused by chronic alcoholism can lead to cardiac arrhythmias, mental and emotional disturbances, necessitating appropriate assessment and treatment.

In addition to the aforementioned physiological effects, chronic alcoholism can also lead to oxidative stress, as indicated by elevated levels of malondialdehyde. Therefore, it is crucial to recognize and manage these complications to prevent further deterioration of health.

It is worth noting that several studies have documented the adverse effects of chronic alcoholism on various organs and systems of the body, including the cardiovascular, digestive, and nervous systems. In fact, alcoholism is associated with an increased risk of developing several chronic diseases, such as liver cirrhosis, pancreatitis, and certain cancers. Furthermore, alcoholism can have significant social and economic consequences, such as impaired work productivity, increased healthcare costs, and family disruption. Thus, it is imperative to raise awareness about the harmful effects of alcoholism and promote responsible alcohol consumption.

Future Perspectives

Future perspectives for the findings related to chronic alcoholism and its effects on the body are essential to improving our understanding and management of this condition. One potential avenue of research is to investigate novel therapeutic strategies for treating alcohol-related liver disease, as liver injury is a common and serious consequence of chronic alcoholism. For instance, recent studies have suggested that targeting specific molecules or pathways involved in liver injury, such as toll-like receptors or the inflammasome, could be promising therapeutic targets for preventing or reversing alcohol-induced liver injury.

Another important area of investigation is the identification of biomarkers that can be used to diagnose and monitor the progression of chronic alcoholism and its associated complications. For example, studies have shown that measuring serum levels of certain proteins or metabolites, such as fibroblast growth factor 21 or acetaldehyde-modified proteins, could be useful biomarkers for detecting alcohol abuse or predicting the risk of alcohol-related liver disease.

Furthermore, the development of personalized medicine approaches could also be beneficial for managing chronic

alcoholism. By identifying genetic or epigenetic factors that contribute to an individual's susceptibility to alcohol abuse or its complications, it may be possible to develop tailored treatment strategies that are more effective and less prone to adverse effects.

Finally, public health campaigns and policies aimed at reducing alcohol consumption and preventing alcohol abuse could also have a significant impact on the burden of chronic alcoholism and its associated health and social consequences. Such initiatives could include increasing alcohol taxes, implementing stricter regulations on alcohol advertising and sales, and promoting educational programs on responsible alcohol consumption.

In summary, future perspectives for chronic alcoholism research encompass a wide range of approaches, including therapeutic interventions, biomarker identification, personalized medicine, and public health policies. By advancing our understanding of this complex condition and its effects on the body, we can develop more effective strategies for managing and preventing alcohol-related complications, ultimately improving the health and well-being of individuals affected by chronic alcoholism.

Acknowledgements

The authors would like to express their heartfelt gratitude to several individuals and organizations for their invaluable support during the course of this study. First and foremost, we are deeply indebted to Professor Dr. Gera Prem Kumar and Professor Dr. Vellanki Lakshmi Narasamma, the Heads of the Department of Biochemistry at Government Medical Colleges in Vizianagaram and Kadapa, respectively, for their guidance and mentorship throughout this project.

We would also like to extend our sincere appreciation to Dr. Madaka Bharathi, a postgraduate student at the Department of Biochemistry, Andhra Medical College, Visakhapatnam, for her contributions to this study. Additionally, we would like to acknowledge Saripalli Sandhya, a Civil Assistant Surgeon at King George Hospital, Visakhapatnam, for her support in facilitating data collection and analysis.

Finally, we would like to thank the participants of this study for their cooperation and willingness to contribute to our understanding of chronic alcoholism and its effects on the body. Without their support and participation, this study would not have been possible.

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