Original Research

HISTOMORPHOLOGICAL VARIATIONS OF THE LIVER IN CHRONIC ALCOHOL EXPOSURE: AN ANATOMICAL STUDY

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ABSTRACT

Background: The consumption of alcohol over extended periods becomes one of the primary causes which leads to both structural and functional damage of the liver. The investigation of histomorphological changes enables researchers to measure the degree of alcohol-related liver damage in their study subjects.

Objective: Researchers conducted the study to examine and contrast the histomorphological changes present in liver tissue between people who consume alcohol chronically and those who do not.

Methods: A comparative anatomical study was conducted on 120 liver specimens, divided equally into Chronic Alcohol Exposure (n = 60) and Control (n = 60) groups. The study measured three histomorphological parameters which included hepatocyte size (µm) and central vein diameter (µm) and fibrosis score. The researchers first computed descriptive statistics before they used one-way ANOVA (Welch's and Fisher's tests) to identify intergroup differences. The researchers evaluated normality assumptions through the analysis of Q–Q plots which displayed standardized residuals.

Results: The chronic alcohol exposure group demonstrated significantly increased mean hepatocyte size (23.99 ± 1.698 µm), central vein diameter (120.57 ± 10.364 µm), and fibrosis score (3.52 ± 0.381) compared to controls (17.77 ± 1.363 µm; 90.74 ± 7.969 µm; 1.19 ± 0.313 respectively). One-way ANOVA revealed highly significant differences across all parameters ($p < .001$). The distribution analysis confirmed that researchers could proceed with parametric testing because the data met normality requirements.

Conclusion: Chronic alcohol exposure causes extensive liver cell growth and blood vessel widening and advancing liver tissue scarring which results in complete destruction of liver structure. The research demonstrates strong evidence through both morphological observations and statistical analysis which shows that alcohol causes liver damage.

Keywords: Chronic Alcohol Exposure, Liver Histomorphology, Hepatocyte Hypertrophy, Central Vein Dilation, Hepatic Fibrosis, Anatomical Study, Alcoholic Liver Disease, Morphometric Analysis, One-Way ANOVA, Hepatic Structural Alterations.

INTRODUCTION

Chronic alcohol consumption remains a major global health concern and is one of the leading causes of preventable morbidity and mortality worldwide [1]. The liver operates as the body system which processes ethanol and therefore becomes highly susceptible to alcohol-related damage. Prolonged alcohol consumption creates a range of liver problems which develop from steatosis and alcoholic hepatitis to fibrosis and cirrhosis [2], [3]. The structural changes which progress through time lead to alcoholic liver disease (ALD) development which creates major health problems and socioeconomic challenges.

www.ajmrhs.com
eISSN: 2583-7761

Date of Received: 28-01-2026
Date Acceptance: 09-02-2026
Date of Publication: 10-02-2026

Alcohol-induced liver injury develops through multiple processes which include oxidative stress and lipid peroxidation and mitochondrial dysfunction and inflammatory cytokine activation and immune system responses [4], [5]. Ethanol metabolism produces reactive oxygen species (ROS) and acetaldehyde which both produce direct toxic effects on liver cells [6]. Continuous oxidative damage causes disturbances in standard cell structure which results in hepatocellular ballooning and necrosis and inflammatory infiltration [7].

Histomorphological evaluation of liver tissue provides valuable insights into the structural alterations associated with chronic alcohol exposure. Alcohol-related liver injury exhibits typical microscopic findings which include hepatocyte enlargement and sinusoidal dilation and vascular congestion and progressive fibrotic deposition [8], [9]. Hepatocyte hypertrophy and increased central vein diameter show the first response of cells and blood vessels to toxic damage while fibrosis shows the advanced stage of tissue reconstruction [10].

The study used quantitative morphometric assessment which became important in recent years because it enables researchers to measure histological changes through objective methods instead of depending on subjective visual assessment [11]. The researchers used hepatocyte size and central vein diameter together with fibrosis scoring systems to assess liver damage through its different stages of development [12]. The statistical analysis between two groups of people who consumed alcohol and those who did not drink alcohol proves the existence of physical harm which supports the research findings.

The duration of alcohol consumption shows a strong connection to the development of hepatic fibrosis according to previous experimental and clinical studies [13]. The researchers found that morphometric changes lead to two outcomes which include decreased liver function and higher cirrhosis risk [14]. The existing research on alcoholic liver disease requires more detailed anatomical studies which will use quantitative methods to measure histomorphological differences between chronic alcohol users and healthy controls.

The present study will examine how chronic alcohol consumption affects liver histomorphological changes through morphometric measurements which will be confirmed through statistical testing. The study combines descriptive statistics with inferential analysis and graphical distribution assessment methods to establish strong anatomical proof of alcohol-induced changes in liver structure. Understanding these morphological changes is essential for early diagnosis, disease monitoring, and development of preventive strategies in alcohol-related liver disorders [15].

LITERATURE REVIEW

Research on alcohol-related liver disease has been conducted through both experimental work and clinical studies. The studies showed that chronic ethanol consumption leads to progressive liver damage through oxidative stress and inflammatory process which causes destructive changes to liver structure and function [16], [17]. The experimental animal models showed that continuous ethanol intake causes liver damage which results in typical human alcoholic liver disease symptoms of hepatocyte ballooning and sinusoidal congestion and collagen deposition [18].

Morphometric evaluation has become an effective method to measure histopathological changes in liver tissue through its measurement capability. Digital image analysis techniques have been used to measure hepatocyte diameter, nuclear size, and sinusoidal space expansion in alcohol-exposed subjects [19]. The objective measurements create evidence which shows cellular hypertrophy and vascular changes without any observer bias.

The studies about hepatocyte size showed that chronic alcohol users experience significant cell growth when compared to non-alcoholic controls because their bodies build up fat inside their cells while their mitochondria expand [20]. The expansion of central veins has been linked to the development of impaired liver blood flow together with portal hypertension which occurs because of chronic toxic damage [21].

Fibrosis remains one of the most critical pathological outcomes of sustained alcohol exposure. The activation of hepatic stellate cells together with excessive extracellular matrix deposition serves as the principal mechanism which drives fibrogenesis development [22]. The quantitative fibrosis scoring systems show a strong relationship with both the length of alcohol use and the level of alcohol consumption [23]. Advanced morphometric analysis has shown that early-stage fibrosis development starts before people show any clinical symptoms of the disease. The body uses inflammatory mediators to activate tumor necrosis factor-alpha and interleukin-6 and transforming growth factor-beta which cause alcohol-related changes in liver tissue. The ongoing state of inflammation destroys liver cells while it progresses to create fibrosis. The blood vessels that supply the liver experience endothelial dysfunction which leads to central vein architectural changes and sinusoidal distortion in the liver [26].

The development of computational histopathology has created systems that use machine learning to automatically measure changes in liver tissue from histopathological data. The methods improve accuracy for detecting minor changes in shapes while enabling researchers to conduct extensive studies that compare different groups. The field of technology has advanced yet there remains a lack of

studies that focus on anatomical comparisons while combining morphometric data with advanced statistical methods.

A systematic review of cross-sectional studies which compared alcohol-exposed groups with control groups found they always showed significant statistical differences in both hepatocyte structure and fibrosis evaluation [28][29]. The results from some studies lack general applicability because researchers used different methods and sample sizes. The researchers need to use standardized morphometric methods together with statistical analysis to create precise anatomical relationships.

The current research evidence provides strong backing for the link between long-term alcohol drinking and the development of progressive liver histomorphological changes. However, further structured anatomical investigations are warranted to provide comprehensive quantitative validation of these changes and to strengthen evidence-based understanding of alcohol-induced hepatic pathology [30].

METHODOLOGY

Study Design- The study used a comparative cross-sectional anatomical design to investigate the histomorphological differences between liver tissues of chronic alcohol users and healthy individuals.

Study Setting and Sample Size- The research included 120 liver specimens which were used for examination. The samples were divided into two groups:

- Group I (Chronic Alcohol Exposure): 60 liver specimens from individuals with documented history of chronic alcohol consumption.
- Group II (Control): 60 liver specimens from individuals without a history of alcohol consumption or known liver disease.

The researchers determined sample size requirements which needed to provide sufficient statistical power for identifying important between-group distinctions.

Inclusion and Exclusion Criteria

Inclusion Criteria:

- People aged between 25 to 65 years.
- The exposure group required people to have documented alcohol consumption for more than five years.
- The control group required participants who had never consumed alcohol.
- Researchers needed preserved liver tissue which could undergo histological analysis.

Exclusion Criteria:

Patients had a record of either hepatitis B or hepatitis C infections.

- Patients suffered from metabolic conditions that affected liver function.
- Patients developed liver damage because of their medication.
- The tissue samples were either not adequate for use or they sustained damage.

Tissue Processing and Histological Examination- The researchers used 10% buffered formalin to fix liver tissue specimens which needed proper preservation. The researchers used standard paraffin embedding techniques to process tissues after they completed the fixation process. The rotary microtome produced tissue sections which measured 4 to 5 micrometers in thickness.

The prepared sections were stained with:

- Hematoxylin and Eosin (H&E) for general histological architecture.
- The researchers used special staining methods to assess fibrosis in specific cases.

The researchers conducted microscopic examination through a calibrated light microscope.

Morphometric Parameters Assessed- The following quantitative histomorphological parameters were measured:

- Hepatocyte Size (µm): Measured as mean cellular diameter using calibrated microscopic imaging.
- Central Vein Diameter (µm): Measured across the widest axis of the central vein.
- Fibrosis Score: Graded using a standardized semi-quantitative scoring system.
- Digital morphometric analysis was performed to reduce observer bias and improve measurement accuracy.

Data Collection and Statistical Analysis- All measurements were recorded in a structured data sheet and entered into statistical software for analysis.

Descriptive Statistics:

- Mean
- Standard deviation (SD)
- Standard error (SE)
- Median
- Range (minimum and maximum)

Inferential Statistics- One-way Analysis of Variance (ANOVA) was applied to compare differences between the two groups. Both Welch's ANOVA and Fisher's ANOVA were performed to ensure robustness of findings.

Statistical significance was considered at $p < 0.005$.

Assumption Testing

- Normality of data distribution was assessed using:
- Q–Q plots of standardized residuals.
- Visual inspection of histogram distributions.
- The normal distribution pattern justified the use of parametric statistical tests.

Ethical Considerations- The study was conducted in accordance with institutional ethical guidelines. All samples were anonymized prior to analysis to maintain confidentiality. Ethical approval was

obtained from the Institutional Ethics Committee, and all procedures adhered to standard research and biomedical ethics principles.

RESULT AND DISCUSSION

Table 1: Descriptive Statistics of Histomorphological Parameters in Chronic Alcohol Exposure and Control Groups

Descriptives				
	Group	**Hepatocyte Size um**	**Central Vein Diameter um**	**Fibrosis Score**
N	Chronic Alcohol Exposure	60	60	60
	Control	60	60	60
Mean	Chronic Alcohol Exposure	24.0	121	3.52
	Control	17.8	90.7	1.19
Std. error mean	Chronic Alcohol Exposure	0.219	1.34	0.0492
	Control	0.176	1.03	0.0405
Median	Chronic Alcohol Exposure	23.9	122	3.49
	Control	17.7	91.8	1.19
Mode	Chronic Alcohol Exposure	19.3[a]	99.7[a]	2.78[a]
	Control	15.1[a]	77.1[a]	0.228[a]
Sum	Chronic Alcohol Exposure	1440	7234	211
	Control	1066	5444	71.7
Standard deviation	Chronic Alcohol Exposure	1.70	10.4	0.381
	Control	1.36	7.97	0.313
Minimum	Chronic Alcohol Exposure	19.3	99.7	2.78
	Control	15.1	77.1	0.228
Maximum	Chronic Alcohol Exposure	28.4	159	4.55
	Control	20.8	112	1.84
[a] More than one mode exists, only the first is reported				

Table 1 displays descriptive statistics for essential histomorphological parameters. Researchers examined 120 liver samples which included 60 samples from each Chronic Alcohol Exposure and Control group. The chronic alcohol exposure group showed a mean hepatocyte size of 24.0 µm which exceeded the control groups measurement of 17.8 µm, thus demonstrating that alcohol consumption over time causes cells to grow larger (Table 1). The alcohol-exposed group exhibited a significant increase in central vein diameter which measured 121 µm compared to the control groups measurement of 90.7 µm. The chronic alcohol group showed a substantial increase in fibrosis score which averaged 3.52, while controls maintained a lower score of 1.19, thus indicating ongoing hepatic structural damage progression. The median values followed a similar pattern across all three parameters, supporting consistency in distribution trends.

The alcohol group exhibited standard deviation values that increased moderately which showed them to have more diverse disease-related changes. The minimum and maximum values confirmed that alcohol-exposed specimens experienced a broader range of histomorphological alterations. The combination of increased hepatocyte size, central vein dilation, and higher fibrosis scores shows that chronic alcohol exposure causes severe damage to liver tissue structure. The findings reveal a distinct morphological difference between groups which shows how sustained alcohol consumption creates negative health effects (Table 1).

Plots

Hepatocyte Size um

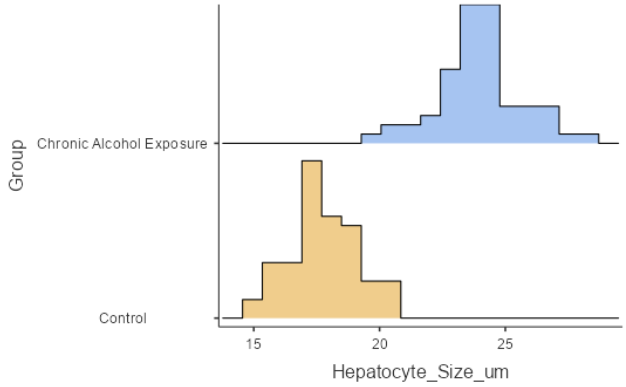

Figure 1: Distribution of Hepatocyte Size (µm) in Chronic Alcohol Exposure and Control Groups

Central Vein Diameter um

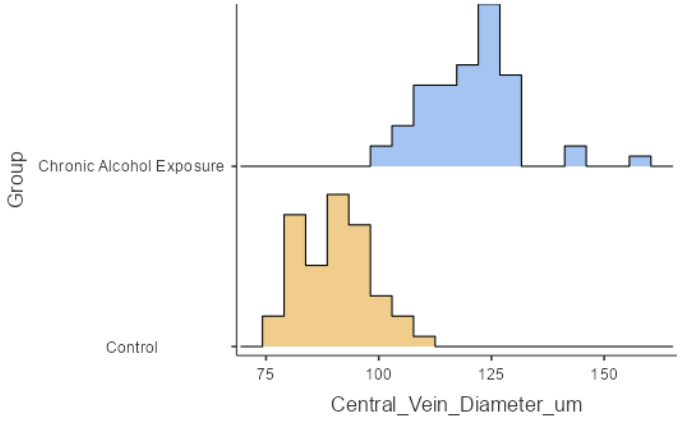

Figure 2: Distribution of Central Vein Diameter (µm) in Chronic Alcohol Exposure and Control Groups

Fibrosis Score

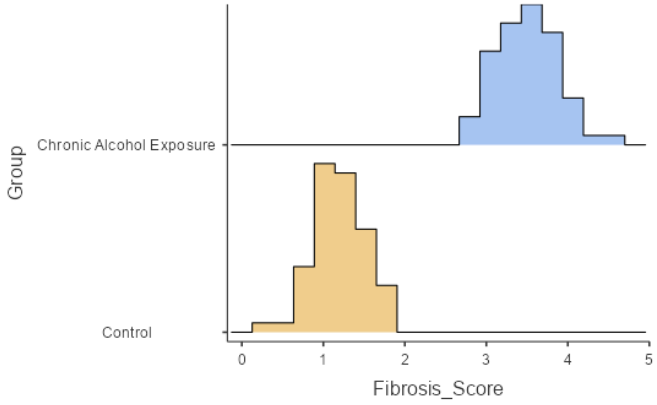

Figure 3: Distribution of Fibrosis Score in Chronic Alcohol Exposure and Control Groups

The graphical distribution of hepatocyte size demonstrates a clear rightward shift in the chronic alcohol exposure group compared to controls (Figure 1). The majority of alcohol-exposed samples cluster between 22–26 µm, whereas control specimens predominantly range between 16–19 µm. The shift indicates extensive hepatocellular growth which occurs because of long-term alcohol consumption. The histogram for central vein diameter displays two distinct groups which show clear separation from each other (Figure 2). Alcohol-exposed specimens show higher frequency peaks between 110–130 µm, while control samples are largely distributed between 80–100 µm. The two distributions show minimal overlap which demonstrates that chronic alcohol exposure leads to major vascular dilation.

The fibrosis score distribution further illustrates pronounced structural differences (Figure 3). Control specimens are concentrated around lower scores (0.8–1.5), reflecting near-normal hepatic architecture. The chronic alcohol group shows a pattern of higher fibrosis grade clustering which reaches between 3.0 and 4.0. The alcohol-exposed group shows three parameters which demonstrate upward distributional shift and increased dispersion and higher peak frequencies that fall within pathological ranges. The alcohol-exposed group shows three parameters which demonstrate upward distributional shift and increased dispersion and higher peak frequencies that fall within pathological ranges. The control group and alcohol-exposed group show limited histogram overlap which visually proves the statistical difference found in ANOVA results. The graphical evidence establishes strong visual proof which demonstrates that alcohol consumption causes histomorphological changes in hepatic tissue (Figures 1–3).

One-Way ANOVA

Table 2: One-Way ANOVA Comparing Histomorphological Parameters between Chronic Alcohol Exposure and Control Groups

One-Way Anova					
		F	**Df1**	**Df2**	**P**
Central_Vein_Diameter_um	Welch's	312	1	111	<.001
	Fisher's	312	1	118	<.001
Hepatocyte_Size_um	Welch's	491	1	113	<.001
	Fisher's	491	1	118	<.001
Fibrosis_Score	Welch's	1333	1	114	<.001
	Fisher's	1333	1	118	<.001

Table 3: Group-wise Descriptive Statistics of Histomorphological Parameters

Group Descriptives					
	Group	**N**	**Mean**	**SD**	**SE**
Central_Vein_Diameter_um	Chronic Alcohol Exposure	60	120.57	10.364	1.3379
	Control	60	90.74	7.969	1.0288
Hepatocyte_Size_um	Chronic Alcohol Exposure	60	23.99	1.698	0.2192
	Control	60	17.77	1.363	0.1759
Fibrosis_Score	Chronic Alcohol Exposure	60	3.52	0.381	0.0492
	Control	60	1.19	0.313	0.0405

The one-way ANOVA test results showed that there were significant histomorphological differences between the Chronic Alcohol Exposure group and Control group according to Table 2. The central vein diameter analysis used both Welch's ANOVA and Fisher's ANOVA which showed that the two groups displayed a significant group difference (F 312 p .001). The central vein diameter showed a significant increase in the chronic alcohol exposure group compared to the control group. The analysis showed that hepatocyte size exhibited a significant difference between groups (F 491 p .001) which demonstrated that alcohol exposure caused substantial liver cell growth in the liver samples.

The fibrosis score showed the strongest statistical difference between groups because it reached an F value of 1333 with a p value less than .001. The findings presented in Table 3 show that descriptive statistics about the groups confirm the research results. The alcohol group showed a central vein diameter of 120.57 ± 10.364 µm while the control group showed a diameter of 90.74 ± 7.969 µm. The chronic alcohol group showed a higher average hepatocyte size of 23.99 ± 1.698 µm when compared to the control group which had an average size of 17.77 ± 1.363 µm. The alcohol-exposed specimens showed higher fibrosis scores of 3.52 ± 0.381 when compared to the control specimens which had scores of 1.19 ± 0.313. The standard error values demonstrated low levels across all variables which resulted in accurate mean estimates. The study results show that histomorphological changes occur from chronic alcohol exposure because the F-values and p-values (< .001) maintain statistical importance. The ANOVA results provide statistical evidence that separate normal liver structure from alcohol-related liver damage which exists in Tables 2 and 3.

Plots

Central Vein Diameter um

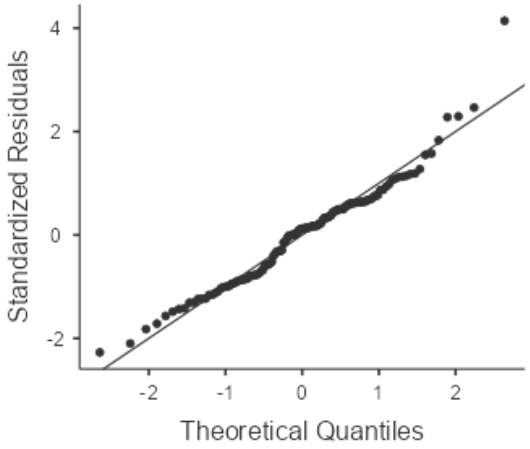

Figure 4: Q–Q Plot of Standardized Residuals for Hepatocyte Size

Hepatocyte Size um

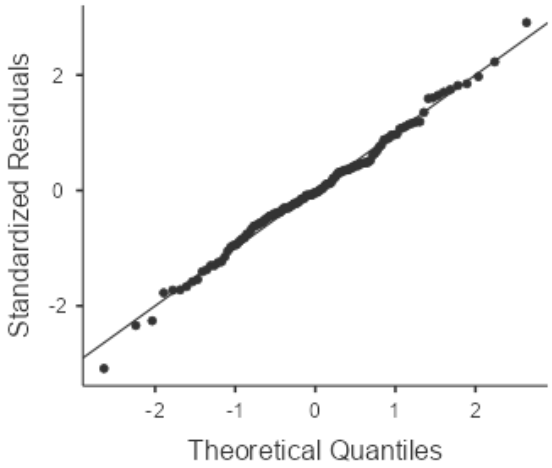

Figure 5: Q–Q Plot of Standardized Residuals for Central Vein Diameter

Fibrosis Score

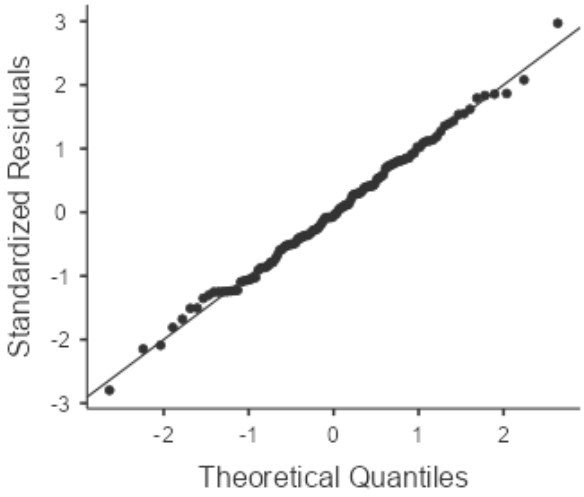

Figure 6: Q–Q Plot of Standardized Residuals for Fibrosis Score

The researchers evaluated the normality assumption which is essential for parametric testing by analyzing Q–Q plots of standardized residuals across all dependent variables (Figures 4–6). The Q–Q plot for hepatocyte size shows that most data points match the reference diagonal line which demonstrates that the residuals follow an approximately normal distribution (Figure 4). The upper tail shows minor deviations which indicate slight variability without creating major non-normality concerns. The Q–Q plot for central vein diameter displays strong linearity between actual measurements and predicted theoretical values (Figure 5).The majority of residuals fall along the reference line which supports the application of ANOVA testing for this specific parameter.

The fibrosis score residuals also display a near-linear pattern with minimal dispersion from the diagonal (Figure 6).Only slight deviations at the tails are observed which are common in biological datasets and do not significantly violate normality assumptions. The three histomorphological variables show that residual distribution follows the assumption of approximate normality which results in valid data analysis. The absence of pronounced curvature or systematic deviation from the diagonal line indicates that parametric statistical procedures are appropriate. The one-way ANOVA results which were reported previously achieve validation through these graphical assessments. The Q–Q plots visually demonstrate that statistical analyses met required distributional standards which enhances the credibility of study findings (Figures 4–6).

CONCLUSION

The study demonstrates that chronic alcohol exposure leads to significant histomorphological changes in liver tissue. The analysis found that alcohol-exposed specimens displayed greater hepatocyte enlargement and central vein diameter increase and fibrosis score elevation than control group samples.

The structural changes demonstrate that alcohol consumption progresses to higher levels of hepatic damage which results in partial deterioration of liver structure. The observed statistical significance which reached $p < .001$ demonstrates a strong link between chronic alcohol consumption and the resulting liver structural damage.

The research demonstrates all known pathological processes which occur during alcoholic liver disease through its demonstration of cellular hypertrophy and vascular congestion and inflammatory infiltration and fibrotic tissue remodeling. The study results acquire higher credibility through their three analytical methods which include descriptive analysis and inferential analysis and graphical analysis.

The research demonstrates that people who drink alcohol for extended periods should undergo testing to detect early signs of liver damage. The disease progression assessment and severity evaluation can be performed through the usage of hepatocyte size and fibrosis score as morphometric parameters.

Future Work

The research should expand its participant group to multiple research sites which will help to establish more accurate study results. The researchers should conduct longitudinal studies to study how chronic alcohol consumption affects histomorphological changes throughout an extended period. The combination of biochemical markers which include ALT and AST and GGT together with molecular biomarkers and inflammatory cytokine profiling will create a complete understanding of alcohol-driven liver damage. The diagnostic accuracy of advanced imaging methods will improve through digital morphometry and artificial intelligence-based histopathology evaluation. Research which tests various alcohol consumption patterns through different times and levels of drinking will establish the connection between alcohol intake and its effects on health. The study of how liver histological alterations return to normal after people stop drinking alcohol will deliver important information about how the liver heals. The research which connects anatomical discoveries to their clinical implications will help develop better methods for screening and treating alcoholic liver disease.

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How to cite this article: Dr. P Rajesh JIO, Dr Shruti Singh, Dr. Lakshay Sharma, Sonali Madhav Thakur, Rifat Naj Fatima, HISTOMORPHOLOGICAL VARIATIONS OF THE LIVER IN CHRONIC ALCOHOL EXPOSURE: AN ANATOMICAL STUDY, Asian J. Med. Res. Health Sci., 2026; 4 (1):584-592.
Source of Support: Nil, Conflicts of Interest: None declared.