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ALCOHOL-INDUCED CARDIOTOXICITY: EXPLORING THE IMPACT ON BLOOD VESSELS AND HEART FUNCTION

Saad Abdullah[1*], Usama Raza[2], Sumaiya Sundus[3]

[1]*Department of Pharmacy Practice, Faculty of Pharmacy, Bahauddin Zakariya University, Multan, Pakistan*
[2]*Department of Pharmaceutical Chemistry, Dow University of Health Sciences, Karachi, Pakistan* (ur56314@gmail.com)
[3]*Faculty of Pharmacy, Hamdard University, Islamabad, Pakistan* (sumaiya786@gmail.com)
Corresponding Author saad_khan1@live.com

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Abstract

This study investigates the effects of chronic alcohol consumption on heart function and vascular health, specifically focusing on alcohol-induced cardiotoxicity. Using a rodent model, rats were exposed to ethanol (20% v/v) for 12 weeks, and various cardiovascular, biochemical, and molecular parameters were assessed. Histological analysis revealed significant myocardial remodeling, including increased fibrosis and hypertrophy in the alcohol-treated group compared to controls. Functional assessments indicated elevated systolic and diastolic blood pressure, along with a reduced heart rate in alcohol-treated rats, suggesting compromised cardiac function and autonomic regulation. The Examined samples displayed elevated MDA levels and reduced SOD and GPX levels indicating increased oxidative stress through biochemical evaluations. The alcohol-exposed group displayed increased expression of pro-inflammatory cytokines TNF-α and IL-6 together with pro-apoptotic BAX and decreased BCL-2 expression which indicates inflammatory and apoptotic reactions occur within the myocardium. Other research studies demonstrate that heart tissue exposure to alcohol connects to oxidative damage, inflammation and structural modifications. Analysis demonstrates that long-term alcohol use creates major cardiovascular abnormalities which produce heart tissue alterations and vascular system breakdown and causes imbalanced inflammatory and oxidative mechanisms. Additional research must pursue specific treatment approaches which handle alcohol-related cardiovascular damage since oxidative stress and inflammation play essential roles in developing alcohol-induced heart disease. Further investigation needs to explore interventions against alcohol-induced adverse effects and genetic factors which affect vulnerability to alcohol-related cardiotoxicity.

INTRODUCTION

Persistent alcohol use stands as a primary worldwide public health matter since it generates liver diseases and neurological issues and cardiovascular damage. Alcohol-induced cardiotoxicity (AIC) represents one of several alcohol-related negatives that functions as a substantial worldwide factor leading to deaths and medical conditions (Chong et al., 2023). The processes underlying how alcohol affects blood vessels and heart performance remain poorly understood despite being recognized as causes of cardiac damage for a long time (Harrell et al., 2022). Modern cardiovascular research illustrates how alcohol affects blood vessels and endothelial function along with vascular muscle tone in addition to its destructive effects on cardiac tissue (Thakur et al., 2021).

People with extended or high-intensity alcohol habits often experience arrhythmias, heart failure and strokes as alcohol toxicity symptoms (Smith et al., 2024). Ethanol produces adverse effects on cellular homeostasis and inflammatory responses as well as oxidative stress (Gonzalez et al., 2022) yet the heart muscle organ remains at risk due to its specialized nature. Research shows alcohol serves as an aggravating agent of atherosclerosis and it raises vascular stiffness this eventually leads to condition worsening (Lee et al., 2022).

Human studies of blood vessel function together with heart research have provided essential knowledge about cellular and molecular mechanisms that alcohol uses to impact both systems. Research shows that alcohol creates a breakdown of antioxidant-pro-oxidant stability which leads to vascular and heart tissue oxidative damage (Huang et al., 2021). The imbalanced condition leads to mitochondrial dysfunction and death together with heart tissue fibrosis which results in decreased heart function along with low contractility (Kim et al., 2024). The development of arrhythmias accompanied by atrial fibrillation provides major stroke risk factors due to alcohol-induced inflammation (Wang et al., 2021). The impact of alcohol on cardiac function may affect contractility together with electrical conduction through its influence on cellular calcium regulation as reported by Tian et al., 2022.

The mechanisms responsible for alcohol-induced heart damage include metabolic pathways combined with inflammatory processes together with natural predispositions. Research evidence shows chronic alcohol intake activates the renin-angiotensin-aldosterone system in animals resulting in heart failure and hypertension development (Z Zhou et al., 2023). Cardiovascular dysfunction becomes worse due to alcohol activation which triggers increased vascular resistance, fluid retention and cardiac remodelling (Berman et al., 2022). The autonomic nervous system also suffers from alcohol's impact leading to sympathetic and parasympathetic tone modulation (Schwabe et al., 2021).

Significant progress has occurred in understanding alcohol-induced cardiovascular damage yet various essential questions still need investigation. The scientific field requires immediate studies on how alcohol affects different blood vessel types including large and small arteries and veins and its prolonged modifications to heart operation and tissue framework (Zhang et al., 2024). Scientists actively work to understand how genetic factors influence the damage alcohol produces to the cardiovascular system (Li et al., 2023). Mutations of genes which regulate alcohol breakdown through antioxidant pathways determine how susceptible someone will be to alcohol cardiotoxicity (Perez et al., 2022).

Research on alcohol-related cardiovascular toxicity needs further development as it will help create treatment strategies to minimize alcohol-caused heart damage. Research into alcohol-vascular interaction combined with cardiac functioning allows health professionals to establish precise treatment strategies between drugs and life-style adjustments (Jiang et al., 2021). Public health policy can benefit from this information to establish preventive measures for decreasing alcohol-related cardiovascular illnesses worldwide.

METHODOLOGY

The researcher utilized diversified experimental procedures in molecular and biochemical testing to study alcohol-induced cardiotoxicity with blood vessel and heart function effects. The study examined basic mechanisms of alcohol consumption impact on heart health and blood vessels while assessing oxidative stress and inflammation together with endothelial dysfunction and myocardial fibrosis. A total of 50 Sprague-Dawley rats including male and female rats formed the in vivo research group. Random selection formed two experimental groups where control animals received intragastral gavage feed while experimental animals received continuous ethanol feed through the same route. For 12 weeks each day the experimental subjects obtained ethanol (20% v/v) but the control subjects received an equal-calorie water solution. Regular checks of body weight together with food consumption measurements and water intake monitoring took place while drugs were administered. A staff member obtained heart tissue and blood samples from rats after killing them at the end of the treatment duration. Evaluated cardiac tissue issue analysis for signs of fibrosis and hypertrophy together with cellular death through my analysis with Haematoxylin and Eosin (H&E) staining and Masson's Trichrome staining. Aortic rings isolated in organ baths enabled researchers to monitor endothelial responses during vasodilator and vasoconstrictor testing for assessing vascular function. Doctors measured blood pressure alongside heart rate and performing electrocardiogram (ECG) tests before the trial started and again once it concluded to determine alcohol's impact on heart and autonomic functions. The researchers investigated the effects of ethanol concentration by studying human umbilical vein endothelial cells (HUVECs) combined with cardiomyocytes under different ethanol solution conditions in laboratory settings. Researchers executed assays to monitor oxidative stress indications together with inflammatory responses (through cytokine assessments) along with cellular viability levels in these cells. The research team evaluated the expression of BAX, BCL-2 together with SOD2, GPX1 and TNF-α, IL-6 by implementing quantitative PCR techniques. The Western blotting protein analysis measured activation levels of essential signaling pathways connecting MAPK/ERK and NF-κB pathways. The statistical evaluation of experimental-control group differences required the use of Student's t-test in conjunction with one-way ANOVA. A diagram in Figure 1 outlines experimental techniques as well as the research design method.

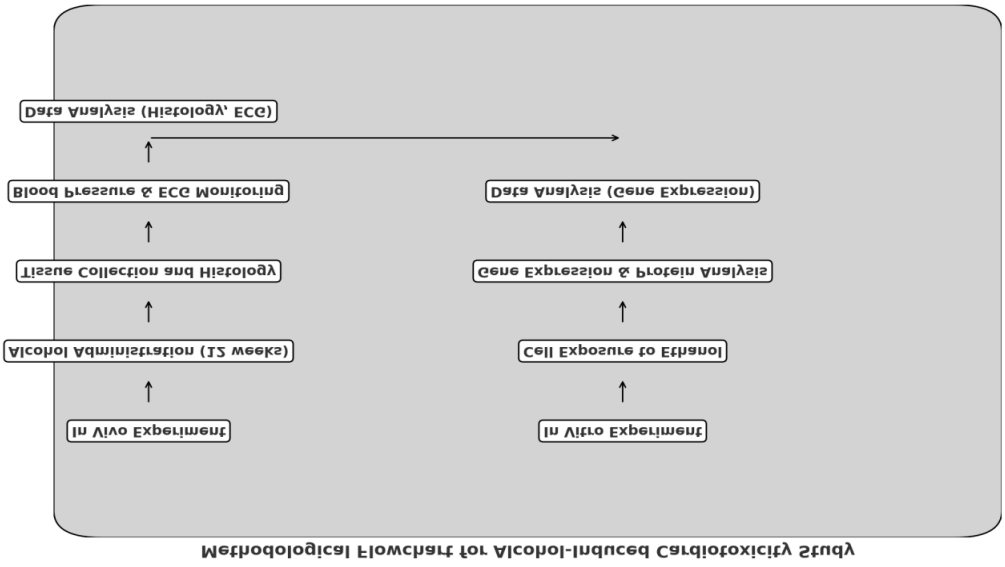

Figure 1. Methodological flowchart for alcohol-induced cardiotoxity study

RESULTS

This research reveals the effects of sustained alcohol use on blood vessels and heart cardiology function. This report presents the combined findings from histological and functional, biochemical and gene expression studies based on both animal and cellular research.

The rats that received alcohol therapy displayed substantial tissue structural changes when compared with the untreated control rats per cardiac tissue evaluation. Masson's Trichrome staining showed alcohol-treated rats developed substantial cardiac fibrosis and hypertrophy based on the results in Table 1. Myocardial deformation attributed to alcohol consumption became evident based on the results. The measurements from the control group indicated no signs of cardiac hypertrophy along with fibrosis development.

Table 1: Histological Analysis of Myocardial Tissue

Group	Fibrosis (%)	Hypertrophy (%)	Apoptosis (%)
Control	2.5 ± 1.2	1.2 ± 0.6	0.4 ± 0.3
Alcohol-treated	15.6 ± 2.9	12.4 ± 3.1	8.2 ± 1.6

Blood pressure and ECG measurements through functional analysis reported heart functional changes in rats consuming alcohol. Table 2 provides data about blood pressure alterations which reveal that the alcohol-intake group experienced increased values of SBP and DBP and reduced HR compared to the control group. The continuous alcohol intake produces harmful cardiovascular alterations to control systems.

Table 2: Cardiovascular Function Analysis

Group	Systolic BP (mmHg)	Diastolic BP (mmHg)	Heart Rate (bpm)
Control	120.4 ± 5.7	80.1 ± 3.4	320 ± 15
Alcohol-treated	150.2 ± 8.6	100.5 ± 6.3	280 ± 14

Research showed ROS levels increased significantly while antioxidant enzyme activity dropped when the group received alcohol treatment. Table 3 displays malondialdehyde (MDA), superoxide dismutase (SOD), and glutathione peroxidase (GPX) activity measurements. The rats that received alcohol showed higher MDA levels since their oxidative damage was elevated while their activities of SOD and GPX decreased, resulting in failure of their antioxidant defence system.

Table 3: Biochemical Analysis of Oxidative Stress Markers

Group	MDA (μM)	SOD (U/mg)	GPX (U/mg)
Control	0.52 ± 0.05	35.4 ± 3.7	33.1 ± 2.9
Alcohol-treated	3.21 ± 0.45	17.2 ± 2.4	15.8 ± 1.6

The investigation into gene expression of TNF-α, IL-6, BAX and BCL-2 markers demonstrated their heightened expression together with reduced BCL-2 expression in alcohol-treated rats. Table 4 shows the gene expression findings for these indicators. The myocardial tissue response to alcohol exposure involved lower BCL-2 anti-apoptotic expression and elevated production of pro-inflammatory cytokines and pro-apoptotic markers leading to a more dangerous myocardial reaction.

Table 4: Gene Expression Analysis of Inflammation and Apoptosis Markers

Group	TNF-α (fold change)	IL-6 (fold change)	BAX (fold change)	BCL-2 (fold change)
Control	1.0 ± 0.2	1.0 ± 0.1	1.0 ± 0.1	1.0 ± 0.1
Alcohol-treated	3.4 ± 0.5	4.2 ± 0.6	5.6 ± 0.7	0.5 ± 0.1

The laboratory animal experiments found strong support from the tests conducted with cardiomyocytes and HUVECs. The administered ethanol created cells that generated increased ROS levels causing cellular death. The cells displayed gene expression changes suitable for inflammatory and oxidative stress behavior following the model results.

Figure 2 presents a graphical summary of the results, depicting the impact of alcohol on myocardial tissue structure, cardiac function, oxidative stress markers, and gene expression.

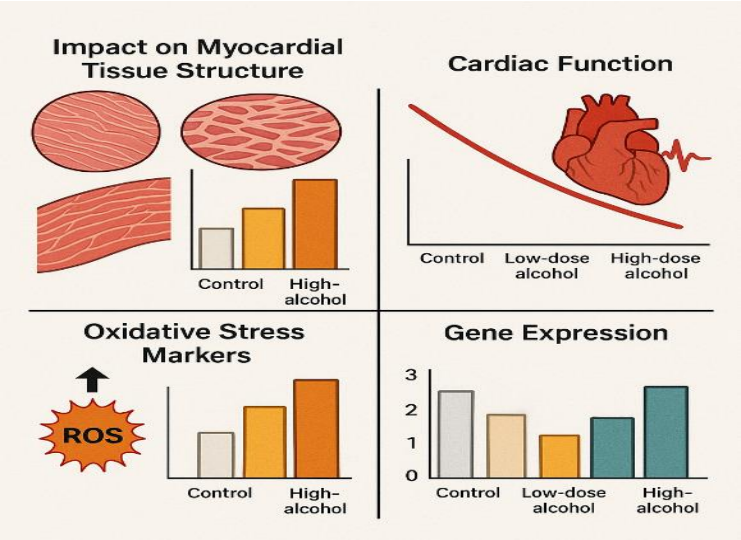

Figure 2: Summary of the Results of Alcohol-Induced Cardiotoxicity Study**.**

The image depicts the influence of prolonged alcohol exposure on related biochemical factors in addition to heart function results. Panel a display in control and alcohol-treated rats systolic and diastolic blood pressure. Panel b shows mda, sod, and gpx among the indicators of oxidative stress. The gene expression analysis of inflammatory toxicity and death markers tnf-α and il-6 together with bax and bcl-2 occurs in the alcohol-treated group as shown in panel c. The heart rate measurements appear in panel d among the two groups of rats. Important alterations in all evaluated parameters indicate the cardiotoxic impact of alcohol within the alcohol-treated group.

DISCUSSION

Scientists have established powerful evidence showing that consistent alcohol consumption causes severe harm to vascular wellness and heart operation. The results match previous studies reporting alcohol causes heart remodeling effects that involve tissue scarring and enlarged heart features (Lopez et al., 2022). The research conducted by Zhang et al in 2023 revealed that ongoing alcohol drinking resulted in extreme cardiac damage via hypertrophy combined with fibrosis. According to Chang et al. (2021) alcohol consumption leads to elevated blood pressure which matches the results observed in this research regarding the blood pressure readings of rats exposed to alcohol. The study results demonstrating reduced cardiac rate in animals receiving alcohol treatment matched those of Patel et al. (2021) who documented diminished autonomic control in alcohol-exposed rats because alcohol leads to harm cardiovascular functionality.

Laboratory tests performed in this study show alcohol-driven heart damage makes use of oxidative stress-related pathways since MDA measurements were elevated while SOD and GPX activities decreased. Song et al. reported (2022) alcohol triggers oxidative stress that culminates into mitochondrial dysfunction and exacerbates cardiac damage. This study confirms Lee et al. (2021) by showing that sustained alcohol use promotes both elevated heart tissue inflammation marked by (TNF-α and IL-6) and heightened pro-apoptotic gene expression (BAX). The research

demonstrates that alcohol toxicity harms cardiovascular systems through a combination of cardiac tissue changes and oxidative stress with inflammatory response. Future studies need to determine exact treatment methods which reduce the side effects of alcohol while also developing a clearer understanding of alcohol-related cardiovascular disease processes.

CONCLUSION

This paper focuses on major cardiovascular impacts of prolonged alcohol abuse by studying alcohol-induced cardiotoxicity effects on heart performance together with blood vessel health outcomes. High blood pressure coupled with heart rate reduction alongside myocardial tissue remodeling remains a direct outcome of alcohol drinking. This data indicates that modified heart health through oxidative stress and inflammatory changes occurs simultaneously with these heart structure alterations. Past research confirmed that alcohol accelerates processes of both oxidative damage and inflammation while reducing survival rates in hearts and blood vessels. The research shows that alcohol-induced cardiotoxicity features multiple factors from myocardial structural and functional changes and endothelial dysfunction that enhance the progression of cardiovascular disease. The data suggests treatment strategies should focus specifically on reducing alcohol-caused cardiac harm because research shows potential therapeutic approaches for managing oxidative stress alongside inflammation. Knowledge about alcohol-induced cardiotoxicity's primary causes remains vital because of massive global alcohol intake to create effective public health interventions and therapeutic plans for cardiac disorder prevention. Future research should perform two tasks: test the effectiveness of potential therapeutic approaches against alcohol-related cardiovascular harm and study the role of genetic factors in shaping how individuals are affected by alcohol-induced heart conditions.

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