European Journal of Clinical Pharmacy
Print ISSN: 2385-409X, **Online ISSN**: 3105-0409
Website: https://farmclin.com/

Impact of Alcoholic Liver Disease on Cardiac Structure and Function

Article History:

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Received: 02-11-2025
Revised: 10-11-2025
Accepted: 06-11-2025
Published: 19-11-2025

Abstract: Background: Alcoholic liver disease (ALD) is among the greatest health issues globally particularly in the developing world because of over-consumption and long-term use of alcohol. It has serious systemic effects, especially on the cardiovascular system, although it predominantly impacts the liver. With a chronic alcoholic use, structural and functional cardiac abnormalities such as alcoholic cardiomyopathy, ventricular dysfunction and poor cardiac output can occur. Such changes in the heart might be long-standing subclinical and lead to a higher morbidity and mortality in the affected individuals.**Place and Duration of Study**This research was carried out in the Department of Medicine amd Cardiology of Shaikh Zayed Hospital, Lahore from January 2025 to June 2025.**Objective:**To ascertain how alcoholic liver disease affects cardiac structure and function and to establish the relationship between the severity of liver disease and cardiac abnormalities.**METHODOLOGY:**This is a cross-sectional study that involved 150 patients who were diagnosed with alcoholic liver disease in a tertiary care hospital. The WHO sample size calculation formula was used to calculate the sample size:n=(Z^2·p·(1-p))/d^2 Demographic information such as age, gender, length of alcohol use and clinical history were collected in a structured proforma. Clinical examination and laboratory studies of patients were performed in detail. The cardiac examination was conducted under the echocardiography method and used the following parameters left ventricular ejection fraction (LVEF), left ventricular end-diastolic diameter (LVEDD), wall thickness, and diastolic functioning. To abscond confounding factors, those patients who already had a cardiac disease, hypertension, diabetes mellitus, or other chronic systemic diseases were excluded. All data obtained was inputted and analysed with statistical software to find the correlation between alcoholic liver disease and cardiac structural and functional alterations.**RESULTS:**It was conducted on 150 patients, most of which were men and had a history of chronic alcohol use. In the research, it was found that a considerable percentage of the patients exhibited cardiac abnormalities. Many patients experienced decreased left ventricular ejection fraction, which is a sign of systolic dysfunction. Also, many patients presented with diastolic dysfunction and larger ventricular dimensions. In patients with severe alcoholic liver disease, cardiac changes were more pronounced in patients than in patients with mild cases. Worsening heart function was closely linked with the time and amount of alcohol consumption. These results show that structural and functional heart impairment is in close relation with alcoholic liver disease.**CONCLUSION**:The cardiac structure and functioning of alcoholic liver disease are severely affected. Most patients have subclinical cardiac abnormalities that can develop when they are not early identified. Echocardiography is suggested as a routine cardiac assessment in patients with alcoholic liver disease to detect and control the disease at an early stage. Complications can be minimized and patient outcomes can be enhanced through preventive measures, alcohol consumption reduction and early medical intervention.

Keywords: Alcoholic liver disease, cardiomyopathy, echocardiography, cardiac function, alcohol, ventricular dysfunction, liver disease

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INTRODUCTION

Alcoholic liver disease (ALD) is a widely recognized cause of chronic liver disease all over the world and is still a significant health issue of concern to the general population, especially developing countries where alcoholism is on the rise [1]. It includes a range of liver diseases that include the simple steatosis (fatty liver) to alcoholic hepatitis, fibrosis, and finally cirrhosis [2]. Long-term and excessive alcohol use causes a progressive liver damage, impacting not only the hepatic functioning but also systemic effects, including several organ systems, particularly the cardiovascular one [3].

The connection of long-term alcohol intake with heart failure has become well-known [4]. The chronic alcohol consumption has a direct influence on myocardial cells, which results in structural and functional changes in the heart [5]. Alcoholic cardiomyopathy is one of the most significant cardiac complications of chronic alcohol consumption; it is a condition, which is characterized by the dilation of the heart chambers, decreased myocardial contractility and cardiac output [6]. These can initially be asymptomatic but of course with time they can develop to open heart failure unless they are detected and taken care of early enough [7].

The impact of alcoholic liver disease on cardiac performance is normally multivariate among patients [8]. The hemodynamic changes that are caused by liver dysfunction include hyperdynamic circulation, increased cardiac output, reduced systemic vascular resistance. These compensatory mechanisms may eventually become maladaptive resulting in cardiac dysfunction. Moreover, there is also alcohol and alcohol metabolite toxicity, malnutrition, as well as electrolyte disturbances which also contribute to myocardial and cardiac performance impairment [9].

Cirrhotic cardiomyopathy is an important but under-recognized condition in patients with advanced liver disease. It is a condition that is marked by a reduction in cardiac contractility, diastolic dysfunction, and electrophysiological abnormalities without the presence of any known heart disease. The patients can be normal at rest, but they exhibit a poor response to stress or physiological demand [10]. This subcriticality renders it difficult to diagnose at an early stage and most of them go unnoticed until they start to complicate.

Echocardiography is instrumental in cardiac structure and cardiac functioning evaluation in alcoholic liver disease patients. It is an affordable, non invasive and reliable device, which could give rich details of the size of cardiac chambers, ventricular activity, wall thickness and diastolic measurements. Abnormalities can also be detected early with the help of echocardiographic examination, and this can be used to intervene and prevent further deterioration.

Although there is an increasing body of evidence to suggest the presence of cardiac involvement in alcoholic liver disease, this is rarely considered in clinical practice. Most of the research involves hepatic complications, but minimal attention is paid to cardiovascular variations, especially in local populations [11]. Hence, the scope and character of the cardiac involvement in alcoholic liver disease patients need to be better known [12].

This paper seeks to assess alcoholic liver disease on cardiac structure and cardiac function through echocardiographic parameters and the relationship between the intensity of liver disease and cardiac abnormalities, as well [13]. These changes can be detected early and allow to enhance the management of patients, minimize complications, and improve the overall clinical outcomes.

OBJECTIVE

To assess the effects of alcoholic liver disease on cardiac structure and cardiac function using echocardiographic parameters in patients with alcoholic liver disease. The research will also seek to establish the relationship between severity and duration of alcohol intake and the severity of cardiac abnormalities, to aid in early identification and better clinical treatment.

METHODOLOGY

This cross-sectional study enrolled 150 patients with an alcoholic liver disease diagnosis in Medicine and Cardiology Department of Shaikh Zayed Hospital, Lahore from January 2025 to June 2025.

Participants were recruited using non-probability consecutive sampling technique. They were included among patients of age 20-70 years with an established chronic history of alcohol intake and clinical or laboratory signs of alcoholic liver disease. Patients with established cardiovascular diseases,

hypertension, diabetes mellitus, congenital heart disease or other chronic systemic illnesses were excluded to reduce confounding factor.

Demographic data such as age, gender, duration and number of alcohol intake and appropriate clinical history were collected using a structured and pre-tested proforma. Clinical examination of all the patients was done in detail.

Lab tests comprised liver tests and normal haematological parameters. Transthoracic echocardiography was performed by a trained cardiologist. The parameters measured were the left ventricular ejection fraction (LVEF), left ventricular end-diastolic diameter (LVEDD), interventricular septal thickness, and the index of diastolic function. The institutional review committee approved the study, and informed consent was obtained before all the participants became part of the study. All the data were collected and analysed with the help of SPSS version ___. Mean plus standard deviation statistics were used to represent quantitative variables and behaviours as frequencies and percentages of qualitative variables. Associations were found using statistical tests with a p-value of less than 0.05 as statistically significant.

INCLUSION AND EXCLUSION CRITERIA
INCLUSION CRITERIA
The age group of 20 to 70 years old with the confirmed diagnosis of alcoholic liver disease (according to clinical, laboratory, or imaging data) were included. Only those participants who had chronic drinking history were picked. Male and female patients with informed consent and who were clinically stable during assessment were both eligible to participate.

EXCLUSION CRITERIA
Patients who had pre-existing cardiovascular diseases and hypertension, diabetes mellitus, congenital heart disease, or chronic kidney disease were excluded. Those who experienced acute medical crises or were in a critical condition were excluded. The study also excluded patients with non-alcoholic liver disease and those who did not want to give informed consent.

DATA COLLECTION
A structured and pre-tested proforma was used to collect data specifically created to carry out this study. Informed consent was obtained and all eligible patients were enrolled and their demographic information, such as age, gender, and duration and quantity of alcohol use were recorded. Full clinical history was taken, including the symptoms of liver disease such as jaundice, abdominal distension and fatigue and any complaints of cardiac problems.
All the patients were carefully assessed physically to identify general conditions and clinical symptoms of alcoholic liver disease. Anthropometric measurement like weight and body mass index (BMI) were collected where feasible.

Lab tests were done in normal aseptic conditions. To find out the liver functioning tests, blood samples were taken and they consisted of serum bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), and serum albumin. Haematological parameters of frequency were also taken.

An expert cardiologist performed the transthoracic echocardiography. Left ventricular ejection fraction (LVEF), left ventricular end-diastolic diameter (LVEDD) and interventricular septal thickness and diastolic cardiac function were taken to ascertain cardiac anatomy and physiology. Data collected was appropriately recorded and verified as full and correct. These were then entered into a statistical software package to further process the data to ascertain the association between alcoholic liver disease and cardiac abnormalities.

RESULTS
The study involved 150 alcoholic liver disease patients. The average age of the individuals was 46.2108 years and mostly the males. Most patients had a history of chronic alcohol use of over 10 years. Echocardiographic assessment showed severe cardiac defects in a considerable percentage of patients. Systolic dysfunction was evident in one out of every three patients, with a reduced left ventricular ejection fraction (LVEF <55). Forty percent had a diagnosis of diastolic dysfunction and 32 percent of the patients had left ventricular dilatation. Thickening of the walls was detected in a quarter of the participants. It was found that there was a strong correlation between the time of alcohol use and cardiac dysfunction severity ($p < 0.05$). Alderley alcoholic liver disease patients exhibited greater structural and functional changes in the heart as compared to the early stages. These results indicate a great role that alcoholic liver disease plays in cardiac health.

Table 1: Demographic Characteristics of Study Participants (n = 150)

Variable	Category	Frequency (n)	Percentage (%)
Age Group	20–30 years	30	20%
	31–50 years	75	50%
	51–70 years	45	30%
Gender	Male	120	80%
	Female	30	20%

Duration of Alcohol Use	<5 years	25	16.7%
	5–10 years	50	33.3%
	>10 years	75	50%
Severity of ALD	Mild	40	26.7%
	Moderate	60	40%
	Severe	50	33.3%

Table 1 presents the demographic characteristics of the 150 patients who participated in the study. The age range of most patients was 31-50 years old with most of them being males. The proportion of those with a history of alcohol consumption over 10 years was large. The severity of the disease shows that majority of patients reported moderate and severe alcoholic liver disease.

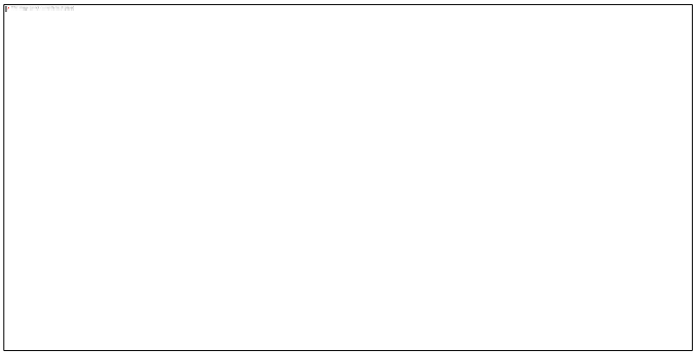


Table 2: Echocardiographic Findings in Patients with Alcoholic Liver Disease (n = 150)

Variable	Category	Frequency (n)	Percentage (%)
Left Ventricular Ejection Fraction (LVEF)	Normal (≥55%)	96	64%
	Reduced (<55%)	54	36%
Diastolic Function	Normal	90	60%
	Diastolic Dysfunction	60	40%
Left Ventricular Dilatation	Present	48	32%
	Absent	102	68%
Wall Thickness	Increased	36	24%
	Normal	114	76%

Table 2 reveals echocardiographic results of patients who have alcoholic liver disease. The proportion of the patients with the reduced ejection fraction and diastolic dysfunction was rather high, and it is an indicator of the systolic and diastolic dysfunction. Left ventricular dilatation and thickening of the walls were also seen in some patients. These findings show drastic structural and functional changes of the heart associated with alcoholic liver disease.

Table 3: Comparison of Cardiac Parameters between Normal and Abnormal Findings (n = 150)

Parameter	Normal (Mean ± SD)	Abnormal (Mean ± SD)
LVEF (%)	60.8 ± 4.5	48.6 ± 5.2
LVEDD (mm)	48.2 ± 3.6	56.4 ± 4.1
Interventricular Septal Thickness (mm)	9.2 ± 1.1	11.6 ± 1.4
E/A Ratio (Diastolic Function)	1.2 ± 0.3	0.8 ± 0.2

Table 3 of the article compares the echocardiographic parameters of patients with normal and abnormal cardiac findings. Aboriginal patients who have abnormal results demonstrate a much lower left ventricular ejection fraction (LVEF), which represents a defective systolic activity. Ventricular dilatation is indicated by increased left ventricular end-diastolic diameter (LVEDD) and structural remodelling is indicated by increased septal thickness. The decreased E/A ratio shows that there is diastolic dysfunction. These disparities underscore the negative effect of alcoholic liver disease on the cardiac structure and functioning.

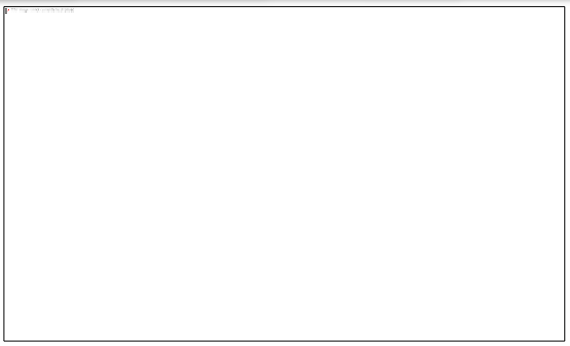


Table 4: Association of Severity of Alcoholic Liver Disease with Cardiac Dysfunction (n = 150)

Severity of ALD	Cardiac Dysfunction Present (n)	Cardiac Dysfunction Absent (n)	Total (n)
Mild	10	30	40
Moderate	28	32	60
Severe	42	8	50
Total	80	70	150

Table 4 presents the correlation between severity of alcoholic liver disease and cardiac dysfunction. Clearly, cardiac dysfunction is much more prevalent among patients with severe alcoholic liver disease than in mild and moderate cases. Conversely, normal cardiac function is demonstrated in most patients with mild disease. This shows a close correlation between deteriorating liver disease and the growing abnormalities in the heart.

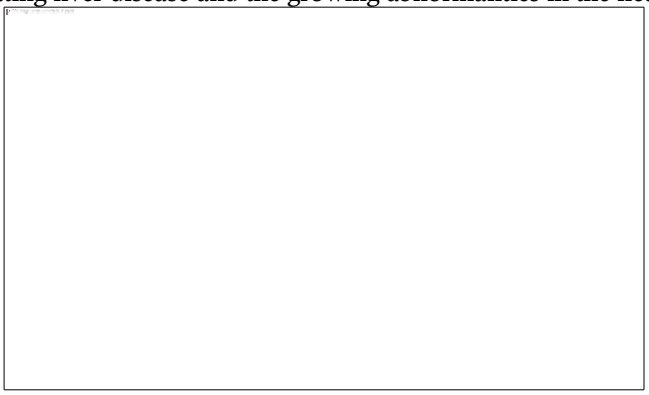
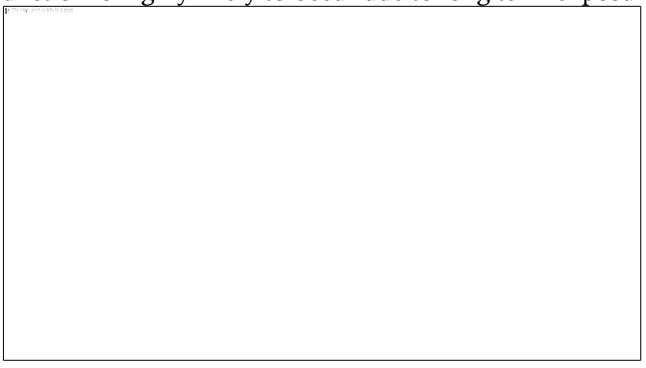


Table 5: Duration of Alcohol Consumption and Cardiac Dysfunction (n = 150)

Duration of Alcohol Use	Cardiac Dysfunction Present (n)	Cardiac Dysfunction Absent (n)	Total (n)
<5 years	8	17	25
5–10 years	22	28	50
>10 years	50	25	75
Total	80	70	150

Table 5 presents the correlation between time of alcohol use and cardiac dysfunction. The most cardiac abnormalities are in patients with longer duration (>10 years). Conversely, patients with less time depict fewer cardiac problems. This implies that cardiac dysfunction is highly likely to occur due to long term exposure to alcohol.



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DISCUSSION

The current article points out the profound influence of alcoholic liver disease (ALD) on cardiac structure and functioning and indorses its importance as a noteworthy but neglected systemic complication of chronic alcohol use. The results indicate that a significant percentage of patients with ALD present with structural and functional cardiac abnormalities

despite the lack of heart disease diagnosis before [14]. This coincides with the increasing amount of evidence that chronic alcohol consumption does not only impact the liver, but the cardiovascular system as well [15].

Among the major discoveries of this research is the very high prevalence of left ventricular dysfunction in ALD patients [16]. The decreased left ventricular ejection fraction (LVEF) which is seen in a significant proportion of patients signifies systolic dysfunction . This could be owed to the direct toxicity of alcohol and its metabolites on the myocardial cells causing degeneration, fibrosis, and decreased contractility. Other researchers have found similar results in their research and have termed alcoholic cardiomyopathy as one of the significant effects of chronic alcoholism [17].

Besides systolic dysfunction, diastolic dysfunction was prevalent in this study. Early myocardial involvement indicated by impaired ventricular relaxation and filling abnormalities may be a precursor to overt systolic failure. Diastolic dysfunction is quite significant because it is not always accompanied by symptoms and can be identified solely with the help of echocardiography. The presence of systolic and diastolic impairment suggests that there is a wide spectrum of cardiac involvement in ALD patients [18].

Another worthy observation is that there is a correlation between the severity of the liver disease and the level of cardiac abnormalities [19]. Ventricular dilatation, decreased ejection fraction and abnormal diastolic parameters were more common in patients with severe ALD than mild or moderate disease [20]. This finding suggests that progressive liver disease contributes to the worsening of heart performance, and it can be mediated by many processes, such as chronic inflammation, neurohormonal imbalance, and hemodynamic alterations.

The duration of alcohol consumption was also greatly associated with cardiac dysfunction. The patient who had a longer history of alcohol consumption, that is, more than 10 years of alcohol consumption had more cardiac changes as compared to the patients who had less history of alcohol consumption [21]. This contributes to the notion that cumulative exposure to alcohol is a critical factor in the pathogenesis of myocardial damage. The long-term effects of alcoholism lead to chronic oxidative stress, mitochondrion dysfunction and impaired protein synthesis in cardiac cells that ultimately leads to structural remodelling and functional deficiency.

This observation of the present study is consistent with the concept of cirrhotic cardiomyopathy, a condition, which is characterized by failure to respond to stress, diastolic dysfunction, and electrophysiological abnormalities in the heart of the patients with a progressive liver disease. Although the majority of the patients could be clinically stable during rest, the amount of cardiac reserve is typically impaired, and they are at great risk of complications during stressful events such as infections, surgery, or liver transplantation.

The clinical implications of the findings are that the heart should be checked frequently in patients who have alcoholic liver disease. [22]. Echocardiography being a non-invasive approach to diagnosis and being readily available could play a significant role in early diagnosis of subclinical cardiac issues. The early detection can lead to early intervention, lifestyle change and proper medical treatment and prevent the development to overt heart failure [23].

All in all, this paper has emphasized the significance of a joint approach as far as treating alcoholic liver disease patients are concerned. In addition to the management of liver related complications, cardiovascular evaluation and risk mitigation must be a priority to healthcare providers. The public health strategies that will be used to reduce the consumption of alcohol and raise awareness about the systemic effects of alcohol will be required to decrease the burden of liver and cardiac diseases.

CONCLUSION

Lastly, alcoholic liver disease can have a significant impact on cardiac anatomy and cardiac performance, and a significant proportion of patients exhibits systolic and diastolic dysfunctions. The findings of the research indicate that the severity and duration of alcohol use are seriously correlated with cardiac dysfunction. Most of these changes are subclinical and in fact, they might not be detected unless the relevant evaluation is performed. Another good and reliable test that could be utilized in detecting early cardiac

defects in alcoholic liver disease was Echocardiography. The timely diagnosis and management of these changes may help prevent the further progression of an open-heart failure and reduce morbidity and mortality. Thus, regular cardiac examination can be regarded as a crucial component of clinical examination of alcoholic liver disease patients. Avoidance measures such as decreasing alcohol consumption, early medical care, and enhanced awareness are essential to enhancing patient outcomes and quality of life.

BIBLIOGRAPHY

1. Cai, J., Zhang, X.-J., Ji, Y.-X., Zhang, P., She, Z.-G., & Li, H. (2020). Nonalcoholic fatty liver disease pandemic fuels the upsurge in cardiovascular diseases. Circulation research, 126(5), 679-704.
2. Chiu, L. S., Pedley, A., Massaro, J. M., Benjamin, E. J., Mitchell, G. F., McManus, D. D., Aragam, J., Vasan, R. S., Cheng, S., & Long, M. T. (2020). The association of non-alcoholic fatty liver disease and cardiac structure and function—Framingham Heart Study. Liver International, 40(10), 2445-2454.
3. Della Torre, S. (2020). Non-alcoholic fatty liver disease as a canonical example of metabolic inflammatory-based liver disease showing a sex-specific prevalence: relevance of estrogen signaling. Frontiers in Endocrinology, 11, 572490.
4. Ferro, D., Baratta, F., Pastori, D., Cocomello, N., Colantoni, A., Angelico, F., & Del Ben, M. (2020). New insights into the pathogenesis of non-alcoholic fatty liver disease: gut-derived lipopolysaccharides and oxidative stress. Nutrients, 12(9), 2762.
5. Ghodsian, N., Abner, E., Emdin, C. A., Gobeil, É., Taba, N., Haas, M. E., Perrot, N., Manikpurage, H. D., Gagnon, E., & Bourgault, J. (2021). Electronic health record-based genome-wide meta-analysis provides insights on the genetic architecture of non-alcoholic fatty liver disease. Cell Reports Medicine, 2(11).
6. Gonzalez, A., Huerta-Salgado, C., Orozco-Aguilar, J., Aguirre, F., Tacchi, F., Simon, F., & Cabello-Verrugio, C. (2020). Role of oxidative stress in hepatic and extrahepatic dysfunctions during nonalcoholic fatty liver disease (NAFLD). Oxidative medicine and cellular longevity, 2020(1), 1617805.
7. Kasper, P., Martin, A., Lang, S., Kuetting, F., Goeser, T., Demir, M., & Steffen, H.-M. (2021). NAFLD and cardiovascular diseases: a clinical review. Clinical research in cardiology, 110(7), 921-937.
8. Loomba, R., Friedman, S. L., & Shulman, G. I. (2021). Mechanisms and disease consequences of nonalcoholic fatty liver disease. Cell, 184(10), 2537-2564.
9. Mantovani, A., Csermely, A., Petracca, G., Beatrice, G., Corey, K. E., Simon, T. G., Byrne, C. D., & Targher, G. (2021). Non-alcoholic fatty liver disease and risk of fatal and non-fatal cardiovascular events: an updated systematic review and meta-analysis. The lancet Gastroenterology & hepatology, 6(11), 903-913.
10. Mantovani, A., Petracca, G., Csermely, A., Beatrice, G., Bonapace, S., Rossi, A., Tilg, H., Byrne, C. D., & Targher, G. (2023). Non-alcoholic fatty liver disease and risk of new-onset heart failure: an updated meta-analysis of about 11 million individuals. Gut, 72(2), 372-380.
11. Matyas, C., Haskó, G., Liaudet, L., Trojnar, E., & Pacher, P. (2021). Interplay of cardiovascular mediators, oxidative stress and inflammation in liver disease and its complications. Nature Reviews Cardiology, 18(2), 117-135.
12. Nanchal, R., Subramanian, R., Karvellas, C. J., Hollenberg, S. M., Peppard, W. J., Singbartl, K., Truwit, J., Al-Khafaji, A. H., Killian, A. J., & Alquraini, M. (2020). Guidelines for the management of adult acute and acute-on-chronic liver failure in the ICU: cardiovascular, endocrine, hematologic, pulmonary, and renal considerations. Critical care medicine, 48(3), e173-e191.
13. Ndrepepa, G. (2021). Aspartate aminotransferase and cardiovascular disease—a narrative review. Journal of Laboratory and Precision Medicine, 6.
14. Powell, E. E., Wong, V. W.-S., & Rinella, M. (2021). Non-alcoholic fatty liver disease. The Lancet, 397(10290), 2212-2224.
15. Ramos, M. J., Bandiera, L., Menolascina, F., & Fallowfield, J. A. (2022). In vitro models for non-alcoholic fatty liver disease: Emerging platforms and their applications. Iscience, 25(1).
16. Rinaldi, L., Pafundi, P. C., Galiero, R., Caturano, A., Morone, M. V., Silvestri, C., Giordano, M., Salvatore, T., & Sasso, F. C. (2021). Mechanisms of non-alcoholic fatty liver disease in the metabolic syndrome. A narrative review. Antioxidants, 10(2), 270.
17. Sandireddy, R., Sakthivel, S., Gupta, P., Behari, J., Tripathi, M., & Singh, B. K. (2024). Systemic impacts of metabolic dysfunction-associated steatotic liver disease (MASLD) and metabolic dysfunction-associated steatohepatitis (MASH) on heart, muscle, and kidney related diseases. Frontiers in cell and developmental biology, 12, 1433857.
18. Targher, G., Byrne, C. D., & Tilg, H. (2024). MASLD: a systemic metabolic disorder with cardiovascular and malignant complications. Gut, 73(4), 691-702.
19. Targher, G., Tilg, H., & Byrne, C. D. (2021). Non-alcoholic fatty liver disease: a multisystem

disease requiring a multidisciplinary and holistic approach. The lancet Gastroenterology & hepatology, 6(7), 578-588.

20. Tilg, H., Adolph, T. E., & Trauner, M. (2022). Gut-liver axis: Pathophysiological concepts and clinical implications. Cell metabolism, 34(11), 1700-1718.
21. VanWagner, L. B., Wilcox, J. E., Ning, H., Lewis, C. E., Carr, J. J., Rinella, M. E., Shah, S. J., Lima, J. A., & Lloyd-Jones, D. M. (2020). Longitudinal association of non-alcoholic fatty liver disease with changes in myocardial structure and function: the CARDIA study. Journal of the American Heart Association, 9(4), e014279.
22. Xiao, H., Li, M., Li, A., Amaerjiang, N., Zunong, J., Vermund, S. H., Perez-Escamilla, R., Song, M., Hu, Y., & Jiang, G. (2022). Passive smoking exposure modifies cardiovascular structure and function: Beijing child growth and health cohort (PROC) study. Environmental Science & Technology, 56(20), 14585-14593.
23. Yan, C., Hu, W., Tu, J., Li, J., Liang, Q., & Han, S. (2023). Pathogenic mechanisms and regulatory factors involved in alcoholic liver disease. Journal of translational medicine, 21(1), 300.

