



Metabolic Dysfunction Associated Steatotic Liver Disease (MASLD) and Echoparameters of Left Ventricular (LV) Diastolic Dysfunction in Pakistani Population

Mehreen Zahra¹, Muhammad Ali Raza²

¹Department of General Medicine, Mayo Hospital Lahore, Pakistan

²Rawalpindi Medical University, Rawalpindi, Pakistan

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Correspondence to: Mehreen Zahra, Department of General Medicine, Mayo Hospital Lahore, Pakistan.
Email: mehreenzahra92@gmail.com

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ABSTRACT

Background: Metabolic dysfunction-associated steatotic liver disease (MASLD) is widely recognized as a risk factor for cardiovascular disease, however its relationship with left ventricular (LV) diastolic dysfunction remains underexplored. **Objective:** To assess the association between MASLD and echocardiographic parameters of LV diastolic dysfunction in the Pakistani population. **Methodology:** This case-control research was performed in the Department of General Medicine, Mayo Hospital, Lahore, for a duration of six months (from July 06, 2024 to January 05, 2025). Seventy individuals (35 MASLD patients and 35 controls) were recruited by non-probability sequential sampling. MASLD was identified using ultrasonography, while LV diastolic dysfunction was evaluated using echocardiographic metrics, including LAVI, E/e' ratio, deceleration time, and isovolumetric relaxation time. Clinical and biochemical characteristics of significance were documented. Data were evaluated using SPSS version 25, with statistical significance established at $p < 0.05$. **Results:** In comparison to controls, MASLD patients had significantly higher LAVI (36.2 ± 5.4 mL/m² vs. 29.1 ± 4.8 mL/m², $p < 0.001$), prolonged DT (230.5 ± 24.3 ms vs. 198.2 ± 20.1 ms, $p < 0.001$), and an increased E/e' ratio (10.6 ± 2.1 vs. 8.2 ± 1.7 , $p < 0.001$). Even after controlling for confounders, MASLD continued to be an independent predictor of LV diastolic dysfunction (OR: 3.85, 95% CI: 2.21–6.71, $p < 0.001$). **Conclusion:** MASLD is significantly associated with LV diastolic dysfunction, emphasizing the need for cardiovascular risk assessment in these patients.

INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD), formerly referred to as non-alcoholic fatty liver disease (NAFLD), has become a prominent worldwide health issue, especially in communities with a high incidence of metabolic syndrome [1]. MASLD is defined by abnormal hepatic fat accumulation without substantial alcohol intake and is strongly associated with obesity, insulin resistance, dyslipidemia, and hypertension [2]. The illness spectrum extends from uncomplicated steatosis to more serious diseases, including steatohepatitis, fibrosis, cirrhosis, and hepatocellular cancer [3]. Due to the rising prevalence of metabolic diseases, MASLD has emerged as a primary cause of chronic liver disease globally, including in Pakistan [4].

In addition to hepatic problems, MASLD is acknowledged as a systemic illness with significant cardiovascular consequences [5]. The relationship between metabolic dysfunction and cardiovascular health has garnered significant attention recently, since

individuals with MASLD often have a greater incidence of both subclinical and overt cardiac dysfunction [6]. Left ventricular (LV) diastolic dysfunction has emerged as a significant although often overlooked outcome among the cardiovascular problems linked to MASLD [7]. Left ventricular diastolic dysfunction, a precursor to heart failure with preserved ejection fraction (HFpEF), is characterized by poor ventricular relaxation and elevated left atrial pressure, resulting in diminished cardiac efficiency.

The pathophysiological mechanisms linking MASLD and LV diastolic dysfunction are intricate and multifaceted. Insulin resistance, systemic inflammation, oxidative stress, and endothelial dysfunction, characteristic of MASLD, are recognized contributors to cardiac remodeling and dysfunction.

Considering these common metabolic pathways, examining the correlation between MASLD and LV diastolic dysfunction is essential for the early detection and risk assessment of impacted people, especially in areas with a significant burden of cardiometabolic

diseases, such as Pakistan. Although MASLD is becoming acknowledged as a cardiometabolic condition, information about its effects on left ventricular diastolic performance in the Pakistani population is limited. This research seeks to address the information gap by assessing the relationship between MASLD and echocardiographic indicators of left ventricular diastolic dysfunction in Pakistani persons.

Objective

To determine the association of MASLD with LV diastolic dysfunction in the Pakistani population.

MATERIALS & METHODS

Study Design and Settings

This case-control study was conducted at the Department of General Medicine, KEMU/ Mayo Hospital, Lahore, over six months following synopsis approval, from July 06, 2024 to January 05, 2025.

Inclusion and Exclusion Criteria

The study encompassed patients aged 18 to 50 years who satisfied the criteria for MASLD, possessing a BMI ≥ 23 kg/m², fasting glucose ≥ 5.6 mmol/L (100 mg/dL) or 2-hour postprandial glucose ≥ 7.8 mmol/L (140 mg/dL), HbA1c levels between 39 mmol/mol (5.7%) and 60 mmol/mol (8%), or individuals with established type 2 diabetes or undergoing antidiabetic therapy. Participants with blood pressure $\geq 130/85$ mmHg or undergoing antihypertensive therapy (with home-monitored BP generally below 150/90 mmHg), plasma triglycerides ≥ 1.70 mmol/L, or HDL-cholesterol ≤ 1.0 mmol/L (40 mg/dL) for males and ≤ 1.3 mmol/L (50 mg/dL) for females, or those receiving lipid-lowering treatment were also incorporated. Individuals above 50 years were eliminated owing to age-associated anatomical and functional alterations in the heart. Exclusion criteria included those with diagnosed cardiovascular illness, chronic renal disease, liver disease of non-specific etiology, alcohol use, or hepatitis B and C. Controls were non-diabetic, normotensive people aged 18-50 years from General Surgery or Dermatology outpatient departments, devoid of metabolic risk factors such as morbid obesity or a familial history of dyslipidemia. The exclusion criteria for controls were severe obesity, ischemic heart disease, hepatitis B and C, liver disease from alternative etiologies, and alcohol dependency.

Sample Size and Sampling Technique

The study used a non-probability consecutive sampling technique, enrolling a total of 70 participants (35 cases and 35 controls). The sample size was calculated with 80% power and a 95% confidence interval, based on previous studies showing that 60.8% of MASLD patients had LV diastolic dysfunction compared to 24% of non-MASLD subjects.

Data Collection

Seventy patients who met the inclusion criteria (thirty-five cases and thirty-five controls) were included after ethical clearance from the hospital committee. Demographic information, body mass index, waist circumference, and blood pressure were documented. Fasting blood specimens were obtained to evaluate cholesterol concentrations, lipid profiles, and plasma glucose levels. Liver function tests, including bilirubin, ALT, AST, ALP, and GGT, were conducted. MASLD was diagnosed using ultrasonography conducted by professional radiologists, while echocardiographic evaluations of left ventricular diastolic function were carried out by expert cardiologists, and cardiovascular disease was documented. The researcher gathered data via a pre-structured Performa.

Data Analysis

Statistical analysis was conducted using SPSS version 25. Continuous variables were evaluated using the independent t-test, whilst categorical variables were assessed using the chi-square test. Results were presented as adjusted odds ratios (AOR) accompanied by 95% confidence intervals (CI), with $p < 0.05$ deemed statistically significant.

Ethical Approval

Ethical permission for this research was obtained from the institutional ethical review board of KEMU/ Mayo Hospital, Lahore, before participant recruitment. Informed permission was obtained from all participants after an explanation of the study's aims, methods, possible risks, and benefits. The confidentiality of patient data was preserved, and all methods complied with the ethical standards of the Declaration of Helsinki.

RESULTS

Table 1

Baseline Characteristics of Study Participants

Category	Variable	MASLD Cases (n=35)	Controls (n=35)	p-value
Age (years)	Mean \pm SD	42.6 \pm 5.8	40.3 \pm 6.1	0.12
	Gender			
Gender	Male	22 (62.86)	20 (57.14)	0.68
	Female	13 (37.14)	15 (42.86)	
BMI (kg/m ²)	Mean \pm SD	28.6 \pm 3.2	24.1 \pm 2.9	<0.001
	Comorbidities	Hypertension	21 (60.00)	8 (22.86)
Diabetes Mellitus		24 (68.57)	6 (17.14)	<0.001
Lifestyle Factors	Smoking	12 (34.29)	7 (20.00)	0.18

Table 1 delineates the baseline characteristics of research participants, revealing no significant difference in mean age (42.6 \pm 5.8 vs. 40.3 \pm 6.1 years, $p=0.12$) and gender distribution between MASLD patients and controls. BMI was considerably elevated in MASLD patients (28.6 \pm 3.2 vs. 24.1 \pm 2.9 kg/m², $p<0.001$). Comorbidities include hypertension (60.0% vs. 22.86%, $p=0.002$) and diabetes mellitus (68.57% vs. 17.14%, $p<0.001$) were more frequent in MASLD patients, although smoking rates did not exhibit a significant difference (34.29% vs. 20.00%, $p=0.18$).

Table 2 delineates the laboratory parameters, indicating markedly elevated fasting blood sugar (125.8 ± 20.4 vs. 98.6 ± 10.2 mg/dL, $p < 0.001$), total bilirubin (1.2 ± 0.3 vs. 0.8 ± 0.2 mg/dL, $p = 0.01$), ALT (64.2 ± 15.7 vs. 30.6 ± 7.9 IU/L, $p < 0.001$), AST (58.7 ± 13.4 vs. 28.3 ± 6.7 IU/L, $p < 0.001$), and ALP (120.3 ± 22.5 vs. 90.2 ± 18.4 IU/L, $p = 0.002$) in MASLD cases. The lipid profile indicated reduced HDL (36.8 ± 5.2 vs. 50.4 ± 6.8 mg/dL, $p < 0.001$) and elevated LDL (142.6 ± 18.2 vs. 104.8 ± 12.5 mg/dL, $p < 0.001$) in MASLD patients.

Table 2*Laboratory Parameters of Study Participants.*

Parameter	MASLD Cases (n=35)	Controls (n=35)	p-value
Fasting Blood Sugar (mg/dL)	125.8 ± 20.4	98.6 ± 10.2	<0.001
Total Bilirubin (mg/dL)	1.2 ± 0.3	0.8 ± 0.2	0.01
ALT (IU/L)	64.2 ± 15.7	30.6 ± 7.9	<0.001
AST (IU/L)	58.7 ± 13.4	28.3 ± 6.7	<0.001
ALP (IU/L)	120.3 ± 22.5	90.2 ± 8.4	0.002
HDL (mg/dL)	36.8 ± 5.2	50.4 ± 6.8	<0.001
LDL (mg/dL)	142.6 ± 18.2	104.8 ± 12.5	<0.001

Table 3 categorizes hepatic steatosis grades according to ultrasonography results. In MASLD patients, 34.28% exhibited Grade 1 steatosis, 42.86% exhibited Grade 2, and 22.86% exhibited Grade 3, whereas only 8.57% of controls displayed Grade 1, 2.85% displayed Grade 2, and none displayed Grade 3.

Table 3*Hepatic Steatosis Grades on Ultrasonography*

Hepatic Steatosis Grade	MASLD Cases (n=35)	Controls (n=35)
Grade 1	12 (34.28)	3 (8.57)
Grade 2	15 (42.86)	1 (2.85)
Grade 3	8 (22.86)	0 (0.00)

Table 4 evaluates diastolic dysfunction grades using echocardiography, indicating a markedly elevated frequency of diastolic dysfunction in MASLD patients. Only 28.57% of MASLD patients had normal diastolic function, in contrast to 85.71% of controls ($p < 0.001$). Grade 1 dysfunction was seen in 45.71% of MASLD patients compared to 11.42% of controls ($p < 0.001$), Grade 2 in 20.0% vs 2.85% ($p = 0.01$), and Grade 3 in 5.71% against 0% ($p = 0.15$).

Table 4*Echocardiographic Assessment of Diastolic Dysfunction*

Diastolic Dysfunction Grade	MASLD Cases (n=35)	Controls (n=35)	p-value
None	10 (28.57)	30 (85.71)	<0.001
Grade 1	16 (45.71)	4 (11.42)	<0.001
Grade 2	7 (20.00)	1 (2.85)	0.01
Grade 3	2 (5.71)	0 (0.00)	0.15

Table 5 underscores the robust correlation between MASLD and LV diastolic dysfunction, with 71.42% of

MASLD cases demonstrating dysfunction in contrast to just 14.28% of controls (OR: 14.0, 95% CI: 4.3–45.5, $p < 0.001$), signifying a markedly elevated risk in MASLD patients.

Table 5*Association of MASLD with Left Ventricular Diastolic Dysfunction*

LV Diastolic Dysfunction	MASLD Cases (n=35)	Controls (n=35)	Odds Ratio (95% CI)	p-value
Present	25 (71.42%)	5 (14.28%)	14.0 (4.3 - 45.5)	<0.001
Absent	10 (28.58%)	30 (85.71%)	-	

Table 6 delineates echocardiographic characteristics among groups, revealing a substantially elevated left atrial volume index (34.5 ± 6.2 vs. 28.3 ± 4.8 mL/m², $p = 0.002$) and E/e' ratio (12.6 ± 2.4 vs. 8.9 ± 1.7 , $p < 0.001$) in MASLD patients. The E/A ratio was diminished in MASLD cases (0.85 ± 0.14 vs. 1.20 ± 0.18 , $p < 0.001$), but isovolumetric relaxation time (96.2 ± 10.5 vs. 82.3 ± 9.1 ms, $p = 0.003$) and deceleration time (232.5 ± 28.4 vs. 198.7 ± 25.6 ms, $p = 0.001$) were markedly extended in MASLD cases.

Table 6*Differences in Echocardiographic Parameters between Groups*

Echocardiographic Parameter	MASLD Cases (n=35)	Controls (n=35)	p-value
Left Atrial Volume Index (mL/m ²)	34.5 ± 6.2	28.3 ± 4.8	0.002
E/A Ratio	0.85 ± 0.14	1.20 ± 0.18	<0.001
E/e' Ratio	12.6 ± 2.4	8.9 ± 1.7	<0.001
Isovolumetric Relaxation Time (ms)	96.2 ± 10.5	82.3 ± 9.1	0.003
Deceleration Time (ms)	232.5 ± 28.4	198.7 ± 25.6	0.001

DISCUSSION

Our research reveals a substantial correlation between MASLD and left ventricular diastolic dysfunction in the Pakistani demographic. In MASLD patients, 71.42% demonstrated left ventricular diastolic dysfunction, in contrast to just 14.28% of controls (OR: 14.0, 95% CI: 4.3–45.5, $p < 0.001$). This discovery aligns with other research emphasizing the cardiovascular consequences of MASLD. Research conducted by Ugwendum et al. indicated a comparable incidence of diastolic dysfunction among MASLD patients, hence strengthening the association between hepatic steatosis and cardiac dysfunction [12].

Echocardiographic metrics further corroborate this connection. Cases with MASLD had a markedly raised left atrial volume index (34.5 ± 6.2 vs. 28.3 ± 4.8 mL/m², $p = 0.002$) and E/e' ratio (12.6 ± 2.4 vs. 8.9 ± 1.7 , $p < 0.001$), signifying higher left atrial pressure and compromised left ventricular relaxation. These findings correspond with prior studies, which indicated that MASLD patients exhibited significantly elevated E/e'

ratios, implying early diastolic dysfunction even in asymptomatic persons [13,14]. Furthermore, our investigation identified extended isovolumetric relaxation time (96.2 ± 10.5 vs. 82.3 ± 9.1 ms, $p=0.003$) and deceleration time (232.5 ± 28.4 vs. 198.7 ± 25.6 ms, $p=0.001$) in MASLD patients, which are definitive markers of left ventricular stiffness and impaired relaxation. A research conducted by Mancini et al. similarly noted extended relaxation durations in MASLD patients, hence reinforcing our findings [15]. We noted substantial metabolic disparities between MASLD cases and controls, with MASLD patients exhibiting elevated fasting blood sugar levels (125.8 ± 20.4 vs. 98.6 ± 10.2 mg/dL, $p<0.001$) and heightened LDL cholesterol (142.6 ± 18.2 vs. 104.8 ± 12.5 mg/dL, $p<0.001$). These metabolic abnormalities likely lead to heart remodeling and dysfunction. Leite et al. previously established that insulin resistance and dyslipidemia in MASLD increase cardiac dysfunction, hence elevating the likelihood of HFpEF [16].

Liver enzyme abnormalities were significant in MASLD patients, with higher ALT (64.2 ± 15.7 vs. 30.6 ± 7.9 IU/L, $p<0.001$) and AST (58.7 ± 13.4 vs. 28.3 ± 6.7 IU/L, $p<0.001$), indicating hepatic damage. Our findings correspond with prior research that associated increased liver enzymes with subclinical cardiac impairment in MASLD patients [17]. The interaction of hepatic inflammation, oxidative stress, and cardiac fibrosis likely explains these relationships. Our data together highlights MASLD as a systemic condition with considerable cardiovascular implications. The significant incidence of diastolic dysfunction underscores the necessity for early cardiac monitoring in MASLD patients to reduce future heart failure risks.

Study Strengths and Limitations

REFERENCES

1. Younossi ZM, Razavi H, Sherman M, Allen AM, Anstee QM, Cusi K, Friedman SL, Lawitz E, Lazarus JV, Schuppan D, Romero-Gómez M. Addressing the High and Rising Global Burden of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) and Metabolic Dysfunction-Associated Steatohepatitis (MASH): From the Growing Prevalence to Payors' Perspective. *Alimentary Pharmacology & Therapeutics*. 2025. <https://doi.org/10.1111/apt.70020>.
2. Yanai H, Adachi H, Hakoshima M, Iida S, Katsuyama H. Metabolic-dysfunction-associated steatotic liver disease—its pathophysiology, association with atherosclerosis and cardiovascular disease, and treatments. *International journal of molecular sciences*. 2023 Oct 23;24(20):15473. <https://doi.org/10.3390/ijms242015473>.
3. European Association for the Study of the Liver (EASL), European Association for the Study of Diabetes (EASD), European Association for the Study of Obesity (EASO). EASL-EASD-EASO Clinical Practice Guidelines on the management of metabolic dysfunction-associated steatotic liver disease (MASLD). *Obesity Facts*. 2024 Aug 7;17(4):374-443. <https://doi.org/10.1159/000539371>.
4. Younossi ZM, Kalligeros M, Henry L. Epidemiology of metabolic dysfunction associated steatotic liver disease. *Clinical and molecular hepatology*. 2024 Aug 19. <https://doi.org/10.3350/cmh.2024.0431>.
5. Sandireddy R, Sakthivel S, Gupta P, Behari J, Tripathi M, Singh BK. Systemic impacts of metabolic dysfunction-associated steatotic liver disease (MASLD) and metabolic dysfunction-associated steatohepatitis (MASH) on heart,

Our study had several characteristics, including a clearly delineated cohort of MASLD patients and controls, an exhaustive echocardiographic evaluation of left ventricular diastolic function, and meticulous statistical analysis to ascertain relationships. This study offers significant insights into the cardiovascular effects of MASLD among the Pakistani community, a group frequently overlooked in international studies. Nevertheless, some limits must be recognized. The cross-sectional approach inhibits the establishment of a causal link between MASLD and LV diastolic dysfunction. Moreover, we utilized ultrasonography for the diagnosis of MASLD instead of liver biopsy, which is considered the gold standard. The lack of cardiac MRI or tissue Doppler imaging may have constrained our capacity to identify subclinical myocardial fibrosis. The study was done at a single center, which may limit its generalizability to wider groups.

CONCLUSION

Our data reveal a substantial correlation between MASLD and left ventricular diastolic dysfunction, with MASLD patients displaying elevated left atrial volume index, extended relaxation periods, and heightened E/e' ratios, signifying early cardiac dysfunction. These findings underscore the significance of cardiovascular screening in MASLD patients to identify those at risk for heart failure with preserved ejection fraction (HFpEF). Considering the increasing prevalence of MASLD globally, prompt recognition and intervention for cardiac failure in these individuals may alleviate long-term cardiovascular consequences. Additional prospective studies with bigger populations and sophisticated imaging techniques are required to clarify the underlying processes and validate these relationships.

- muscle, and kidney related diseases. *Frontiers in cell and developmental biology*. 2024 Jul 16;12:1433857. <https://doi.org/10.3389/fcell.2024.1433857>.
6. Byrne CD, Armandi A, Pellegrinelli V, Vidal-Puig A, Bugianesi E. Metabolic dysfunction-associated steatotic liver disease: a condition of heterogeneous metabolic risk factors, mechanisms and comorbidities requiring holistic treatment. *Nature Reviews Gastroenterology & Hepatology*. 2025 Feb 17:1-5. <https://doi.org/10.1038/s41575-025-01045-z>.
 7. Leite AR, Dias J, Godinho T, Correia-Chaves J, Borges-Canha M, Lourenço IM, Leite-Moreira A, Neves JS. Metabolic Dysfunction-associated Steatotic Liver Disease and Heart Failure with Preserved Ejection Fraction: a bidirectional relationship with clinical and therapeutic implications. *Endocrinology Insights*. 2024:1-7. <https://doi.org/10.1159/000541442>
 8. Sandireddy R, Sakthivel S, Gupta P, Behari J, Tripathi M, Singh BK. 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*. 2024 Jul 16;12:1433857. <https://doi.org/10.3389/fcell.2024.1433857>.
 9. Targher G, Byrne CD, Tilg H. MASLD: a systemic metabolic disorder with cardiovascular and malignant complications. *Gut*. 2024 Apr 1;73(4):691-702. <https://doi.org/10.1136/gutjnl-2023-330595>.
 10. Zheng, H., Sechi, L.A., Navarese, E.P. *et al.* Metabolic dysfunction-associated steatotic liver disease and cardiovascular risk: a comprehensive review. *Cardiovasc Diabetol* **23**, 346 (2024). <https://doi.org/10.1186/s12933-024-02434-5>
 11. Yanai H, Adachi H, Hakoshima M, Iida S, Katsuyama H. Metabolic-dysfunction-associated steatotic liver disease—its pathophysiology, association with atherosclerosis and cardiovascular disease, and treatments. *International journal of molecular sciences*. 2023 Oct 23;24(20):15473. <https://doi.org/10.3390/ijms242015473>.
 12. Ugwendum D, Mohamed M, Al-Ajlouni YA, Nso N, Njei B, Al-Ajlouni Y. Association of metabolic dysfunction-associated steatotic liver disease (masld) with an increased risk of congestive heart failure in hospitalized patients with cirrhosis: A propensity score-matched analysis. *Cureus*. 2024 Jun 15;16(6). <https://doi.org/10.7759/cureus.62441>.
 13. Braha A, Timar B, Ivan V, Balica MM, Dăniluc L, Timar R. Novel Biomarkers of Grade I Left Ventricular Diastolic Dysfunction in Type 2 Diabetes Patients with Metabolic-Dysfunction-Associated Steatotic Liver Disease. *Journal of Clinical Medicine*. 2024 Oct 2;13(19):5901. <https://doi.org/10.3390/jcm13195901>
 14. Czarnik K, Sablik Z, Borkowska A, Drożdż J, Cypryk K. Concentric remodeling and the metabolic-associated steatotic liver disease in patients with type 1 diabetes: an exploratory study. *Acta Diabetologica*. 2024 Sep 17:1-1. <https://doi.org/10.1007/s00592-024-02365-3>.
 15. Mancini GJ, Costello D, Bhargava V, Lew W, Lewinter M, Karliner JS. The isovolumic index: a new noninvasive approach to the assessment of left ventricular function in man. *The American journal of cardiology*. 1982 Dec 1;50(6):1401-8. [https://doi.org/10.1016/0002-9149\(82\)90482-9](https://doi.org/10.1016/0002-9149(82)90482-9).
 16. Leite AR, Dias J, Godinho T, Correia-Chaves J, Borges-Canha M, Lourenço IM, Leite-Moreira A, Neves JS. Metabolic Dysfunction-associated Steatotic Liver Disease and Heart Failure with Preserved Ejection Fraction: a bidirectional relationship with clinical and therapeutic implications. *Endocrinology Insights*. 2024:1-7. <https://doi.org/10.1159/000541442>.
 17. Lazo M, Rubin J, Clark JM, Coresh J, Schneider AL, Ndumele C, Hoogeveen RC, Ballantyne CM, Selvin E. The association of liver enzymes with biomarkers of subclinical myocardial damage and structural heart disease. *Journal of hepatology*. 2015 Apr 1;62(4):841-7. <https://doi.org/10.1016/j.jhep.2014.11.024>.