İstanbul Med J 2026; 27(2): 149-54
**DOI:** 10.4274/imj.galenos.2026.03161

# The Relationship Between Fibrosis-4 Score and Major Cardiovascular Events and Mortality in Patients Under 40 Years of Age with Acute Myocarditis

Emine Altuntaş, Kadir Sadıkoğlu, Kübra Balçın, Gizemnur Coşkun, Hasan Şahin, Abidin Emre Tırnaksız, Mehmet Ertürk

University of Health Sciences Türkiye, Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, Clinic of Cardiology, İstanbul, Türkiye

## ABSTRACT

**Introduction:** Fibrosis-4 index (Fib-4i) is a useful and practical indicator of fibrosis risk in chronic liver disease. The study aimed to explore the relationship between Fib-4i and major adverse cardiovascular events (MACEs) within one year in cases of inflammatory myopericardial syndrome (IMPS).

**Methods:** Between January 2018 and December 2023, 152 patients diagnosed with IMPS were stratified into two groups based on the presence of late gadolinium enhancement (LGE) on cardiac magnetic resonance imaging. The Fib-4i was calculated at admission, and MACEs due to IMPS were recorded at 12 months.

**Results:** Group 1 consisted of 36 LGE-negative patients, while group 2 consisted of 116 LGE-positive patients. The Fib-4i was higher in group 2 than in group 1 (p=0.014). Furthermore, it was found that aspartate aminotransferase, high-sensitivity (hs) troponin T, N-terminal pro-brain natriuretic peptide, and C-reactive protein (CRP) were higher in group 2 than in group 1 (respectively, p=0.03, p=0.002, p=0.008, p=0.014). No difference in MACEs between groups was observed at one month, six months, and one year (p=0.581, p=0.558, p=0.665, respectively). However, a positive correlation was observed between the Fib-4i and both hs-troponin T and CRP.

**Conclusion:** Fib-4i was higher in patients with LGE than in those without LGE. However, its predictive power for MACEs could not be demonstrated.

**Keywords:** Fibrosis-4 index, inflammatory myopericardial syndrome, cardiac magnetic resonance, major cardiovascular events

## Introduction

Inflammatory myopericardial syndrome (IMPS) is a newly described syndrome. This encompasses myocarditis and pericarditis. This description was first defined in the 2025 European Society of Cardiology (ESC) guideline on myocarditis and pericarditis. The clinical spectrum of IMPS encompasses isolated myocarditis and pericarditis, as well as combined forms, including myopericarditis and perimyocarditis. Early-onset coronary artery disease manifests in individuals under 40 and can be diagnostically challenging, particularly when it presents with symptoms similar to those of myocarditis. The aetiology of this clinical scenario is multifaceted, with potential causes including exposure to toxic substances, infectious agents, or inflammatory conditions. This diagnosis indicates a serious, potentially underdiagnosed condition that affects individuals of all ages. The clinical manifestations are heterogeneous and could overlap significantly with other acute cardiac conditions. That causes complications in the diagnostic process (1-3).

In previous consensus statements, the diagnosis of myocarditis was based on the finding on endomyocardial biopsy (EMB). EMB is an effective diagnostic tool for identifying the histological type of the disease, determining specific aetiologies, and differentiating between IMPS and other conditions. However, multimodality imaging has become a cornerstone for diagnosis, with cardiovascular magnetic resonance imaging (CMRI) playing a crucial role (4,5).

Fibrosis-4 index (Fib-4i), a non-invasive marker of hepatic fibrosis in patients with a viral infection, is calculated using three biochemical values and age (6,7). In recent years, the validity of this score has been demonstrated in liver diseases. However, other studies have

**Address for Correspondence:** Assoc. Prof. Emine Altuntaş, MD, University of Health Sciences Türkiye, Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, Clinic of Cardiology, İstanbul, Türkiye
**E-mail:** emine_altuntas@hotmail.com **ORCID ID:** orcid.org/0000-0001-5887-5422

**Cite this article as:** Altuntaş E, Sadıkoğlu K, Balçın K, Coşkun G, Şahin H, Tırnaksız AE, et al. The relationship between Fibrosis-4 score and major cardiovascular events and mortality in patients under 40 years of age with acute myocarditis. İstanbul Med J. 2026; 27(2): 149-54

**Received:** 08.12.2025
**Accepted:** 17.04.2026
**Publication Date:** 12.05.2026

suggested that, in patients with non-hepatic primary diseases, this index is significantly associated with poor clinical outcomes. It has been demonstrated by several recent studies that Fib-4i is a valuable predictor of prognosis in patients suffering from coronary artery disease, heart failure (HF), or atrial fibrillation (6,8,9). Nevertheless, correlation between Fib-4i, and its clinical implications in IMPS patients remains to be elucidated.

The aim of this study was to ascertain whether the Fib-4i increased in IMPS patients undergoing CMRI and to determine whether there was a relationship between the Fib-4i score and prognosis in the clinical follow-up of these patients.

## Methods

### Study Subjects and Protocol

The study was designed as a retrospective cross-sectional investigation. Between January 2018 and December 2023, patients hospitalized with a provisional diagnosis of acute coronary syndrome who were under the age of 40 were selected. Coronary angiography or coronary computed tomography was performed to exclude coronary artery disease in these patients. Patients who satisfied the diagnostic criteria outlined in 2025 ESC guidelines for myocarditis and pericarditis were evaluated as having IMPS (2). Patients who had undergone CMRI were included in the study. During follow-up, death, rehospitalization due to HF or arrhythmia, and recurrent IMPS were recorded at the 1-month, 6-month, and 1-year outpatient clinic visits. The major cardiovascular events (MACEs) in this study constituted a composite endpoint. Participants were excluded from the study if they were under the age of 18, pregnant, had a history of acute or chronic hepatitis B or C, or had liver cirrhosis due to any cause. Also, patients who did not complete follow-up or who had missing data were excluded from the study. Medical records indicated that all patients had experienced an acute infection before the establishment of the IMPS clinic. The present study comprised 152 patients. Patients were divided into two groups based on CMRI involvement: group 1, without CMRI involvement (n=36), and group 2, with CMRI involvement (n=116). Patient data were retrieved from hospital medical records.

All patients were treated from admission with ibuprofen (600-800 mg, 3 x 1), proton pump inhibitors (PPIs), and colchicine (0.5 mg, 2 x 1). Ibuprofen and PPI treatments were given for 2 weeks, while colchicine treatment was given for at least 3 months. For patients with recurrent IMPS, the duration of colchicine treatment has been extended to a maximum of six months.

The study protocol adhered to the ethical guidelines of the Declaration of Helsinki and was approved by the Ethics Committee of the University of Health Sciences Türkiye, Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital (approval number: 2025.01-04, date: 14.01.2025). Because of the retrospective design of the study, individual informed consent was waived.

### Laboratory Test and Fib-4i Calculation

On admission, venous blood samples were drawn from each patient and analyzed. N-terminal pro B-type natriuretic peptide (NT-proBNP) and high-sensitivity (hs) troponin-T were measured on admission. An automated biochemical analyzer (Roche COBAS 6000) was used to measure the concentrations of total cholesterol (TC), fasting blood glucose (FBG), aspartate aminotransferase (AST), alanine aminotransferase (ALT), and serum creatinine, using an enzymatic assay. A complete blood count was performed using the Mindray BC-6000 autoanalyzer.

The Fib-4i at admission was calculated by the formula: age (years) × AST(U/L)/(platelets count [$10^9$/μL] × $\sqrt{\text{ALT}}$ (U/L) (6).

### Trans Thoracic Echocardiography

Echocardiographic assessments were performed at baseline by two independent, experienced physicians who were blinded to the results. All parameters were assessed following the current guidelines of the American Society of Echocardiography and the ESC (10,11).

### CMRI Acquisition Protocol

CMRI was performed using a 1.5-Tesla MR scanner (Magnetom Aera; Siemens Healthcare, Erlangen, Germany). Cine imaging was obtained with a breath-hold balanced steady-state free precession sequence in standard long-axis planes (two-, three-, and four-chamber views) and contiguous short-axis orientations (slice thickness of 6–8 mm, without interslice gap; 10–15 slices). Myocardial edema was assessed using T2-weighted short tau inversion recovery sequences in combination with T2 mapping.

Native T1 mapping was acquired using an electrocardiography-triggered modified Look-Locker inversion recovery sequence with a 3(3)3(3)5 scheme at three short-axis levels of the left ventricle (basal, mid-ventricular, and apical). After intravenous injection of gadobutrol at a dose of 0.15 mmol/kg, late gadolinium enhancement (LGE) images were obtained using a two-dimensional breath-hold phase-sensitive inversion-recovery gradient-echo sequence in matching imaging planes. The inversion time was individually adjusted to suppress the signal from normal myocardium. Post-contrast T1 mapping was performed following LGE imaging.

### Statistical Analysis

Statistical analysis was implemented using SPSS 22.0 (USA, Amonk, NY, IBM Corporation) from gathered data analysis, continuous factors (CFs) are stated as mean ± standard deviation, and categorical factors are stated as a percentage of group total %. The Kolmogorov–Smirnov test was utilized to determine whether the factors demonstrated a normal distribution. CFs with a normal distribution were evaluated using Student's t-test. The chi-square test was used for categorical variables. Correlation analyses were performed using Pearson or Spearman correlation coefficients as appropriate. A p value $<0.05$ was considered significant.

## Results

Patients were divided into two groups based on the presence of LGE on CMRI: group 1 (n=36) included patients without LGE, and group 2 (n=116) included patients with LGE. Group 1 had a mean age of 24.5 years and consisted of 24 men (66.7%), while group 2 had a mean age of 21 years and consisted of 89 men (76.7%). The groups were similar in terms of age and gender (p=0.051 and p=0.161, respectively). The patients

had no chronic illnesses. One patient in group 1 and eight patients in group 2 were smokers (p=0.325). Furthermore, groups were compared with respect to symptoms presenting to the emergency department; no statistically significant difference was detected (p>0.05). The groups were also compared with respect to laboratory tests. AST, C-reactive protein (CRP), hs-troponin T, NT-proBNP, and Fib-4i were higher in group 2 than in group 1, and these differences were statistically significant (p=0.03, p=0.044, p=0.002, p=0.008, p=0.014, respectively). The laboratory test results were summarised in Table 1.

The left ventricular ejection fraction (LVEF) in patients was assessed using both transthoracic echocardiography and CMRI, and the results were compared. The groups showed similar LVEF levels (p=0.771 and p=0.851). Furthermore, a comparative analysis of MACE between the two groups was conducted. Recurrent episodes of myocarditis were observed as MACE in the groups. No further events were observed. Again, no statistically significant difference was observed between the groups with respect to MACE at 1, 6, and 12 months (p=0.581, p=0.558, and p=0.665, respectively). The results are summarized in Table 2.

A correlation analysis was performed among the Fib-4i, NT-proBNP, hs-troponin T, TC, and FBG. The present study found a positive correlation between the FIB-4 index and hs-troponin T and CRP. The results of the correlation analysis were presented in Table 3, Figures 1 and 2.

**Table 1. Comparison between two groups in terms of demographical, clinical, and laboratory tests results**

| Variables | Total (n=152) | Group 1 (n=36) | Group 2 (n=116) | p |
|---|---|---|---|---|
| Gender (male, %) | 113 (74.3%) | 24 (66.7%) | 89 (76.7%) | 0.161* |
| Age (years) | 31 (25.5-34.5) | 24.5 (19.7-33.25) | 21 (19-29) | 0.051** |
| Smoking | 9 (5.9%) | 1 (2.8%) | 8 (6.9%) | 0.325* |
| Admission complaint | | | | |
| Chest pain | 143 (93.6%) | 32 (88.9%) | 111 (94.8%) | 0.207* |
| Dyspnea | 4 (2.6%) | 2 (5.6%) | 2 (1.7%) | |
| Syncope | 3 (2%) | 1 (2.8%) | 2 (1.7%) | |
| Palpitation | 2 (1.3%) | 1 (2.8%) | 1 (0.9%) | |
| Creatinine (mg/dL) | 0.73 (0.66-0.81) | 0.8 (0.74-0.9) | 0.79 (0.63-0.86) | 0.082** |
| Hb (g/dL) | 11.9 (11.0-12.9) | 14.65(12.8-15.4) | 14.2 (13.3-15) | 0.713** |
| WBC ($10^9$/L) | 10 (9.73-10.85) | 9.3 (7-10.8) | 8.13 (6.5-10) | 0.319** |
| PLT ($10^9$/L) | 214 (195-217) | 238.5 (210.25-267) | 218 (196.75-266) | 0.152** |
| AST (U/L) | 33 (23-60) | 23.5 (17-38.5) | 39 (25-63) | 0.03** |
| ALT (U/L) | 19 (13-27) | 19,5 (12.25-28.75) | 19 (13-27) | 0.804** |
| Fasting glucose (mg/dL) | 100.5 (89-107) | 98.5 (85-110) | 101 (89-107) | 0.822** |
| Total cholesterol (mg/dL) | 150 (141-150.5) | 154 (97-185) | 138 (113-152) | 0.322** |
| hs-Troponin T (ng/L) | 147.2 (92.1-673.6) | 109 (29.25-384.75) | 408 (111.25-860) | 0.002** |
| CRP (mg/L) | 10 (5.1-20.5) | 5.5 (2-38.6) | 28.12 (8-46) | 0.044** |
| NT-BNP (pg/mL) | 193 (103.5-320) | 122.5 (97.5-233.5) | 207 (109-361) | 0.008** |
| Fib-4 index | 0.9 (0.5-1.26) | 0.53 (0.4-0.96) | 0.97 (0.62-1.34) | 0.014** |

*: Chi-square test, **: Mann-Whitney U test, Hb: Hemoglobin, WBC: White blood cell, PLT: Platelet, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, hs-troponin T: High sensitivity Troponin T, CRP: C-reactive protein, NT-BNP: N-terminal pro b-type natriuretic peptide, Fib-4 index: Fibrosis-4 index, dL: Deciliter, mg: Milligram, mL: Milliliter, L: Liter, ng: Nanogram, pg: Picogram

**Table 2. Comparison between two groups in terms of trans thoracic echocardiographic, cardiac magnetic resonance imaging test results, and major cardiac events**

| Variables | Total (n=152) | Group 1 (n=36) | Group 2 (n=116) | p |
|---|---|---|---|---|
| EF on admission (%) | 60 (60-60) | 60 (58.7-60) | 60 (60-60) | 0.771* |
| EF measured by cardiac MRI (%) | 61 (59-64) | 60 (60-63) | 61 (59-65) | 0.865* |
| First month MACE | 2 (1.3%) | 0 | 2 (1.7%) | 0.581** |
| Sixth month MACE | 3 (2%) | 1 (2.8%) | 2 (1.77%) | 0.558** |
| First year MACE | 4 (2.6%) | 1 (2.8%) | 3 (2.6%) | 0.665** |

*: Mann-Whitney U test, **: Chi-square test. EF: Ejection fraction, MACE: Major cardiovascular events, mortality, heart failure, rehospitalization due to decompensated heart failure, and arrhythmia, recurrent myocarditis attack, MRI: Magnetic resonance imaging

**Table 3. Spearman's correlation analysis among Fib-4 index and some laboratory tests results**

| Variables | Fib-4 index | NT-pro BNP | hs-troponin T | Total cholesterol | Fasting glucose level | CRP |
|---|---|---|---|---|---|---|
| Fib-4 | 1 | p= 0.843<br>r= 0.020 | p= <0.001<br>r= 0.683 | p= 0.147<br>r= 0.195 | p= 0.090<br>r= 0.176 | p= <0.001<br>r= 0.397 |
| NT-proBNP (pg/mL) | | 1 | p= 0.854<br>r= 0.017 | p= 0.067<br>r= 0.238 | p= 0.489<br>r= 0.070 | p= 0.798<br>r= 0.025 |
| hs-troponin T (ng/L) | | | 1 | p= 0.483<br>r= 0.093 | p= 0.073<br>r= 0.181 | p= <0.001<br>r= 0.464 |
| Total cholesterol (mg/dL) | | | | 1 | p= 0.532<br>r= 0.082 | p= 0.250<br>r= 0.155 |
| Fasting glucose level (mg/dL) | | | | | 1 | p= 0.759<br>r= 0.032 |
| CRP (mg/L) | | | | | | 1 |

Fib-4: Fibrosis-4, NT-proBNP: N-terminal pro b-type natriuretic peptide, hs-troponin T: High sensitivity troponin T, CRP: C- reactive protein, dL: deciliter, L: liter, mg: Miligram, mL: Milliliter, ng: Nanograms, pg: picogram, r: Correlation coefficient

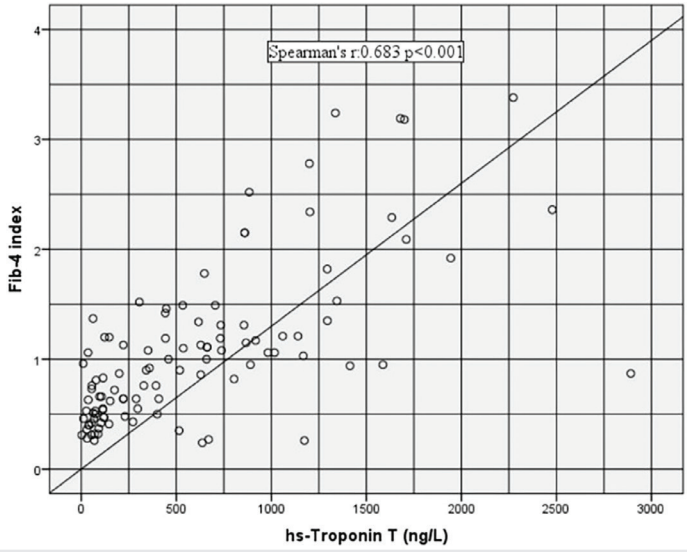


**Figure 1.** Spearman's correlation analysis between Fib-4 index and hs-troponin T. Fib-4: Fibrosis-4, hs-troponin T: High sensitivity troponin T

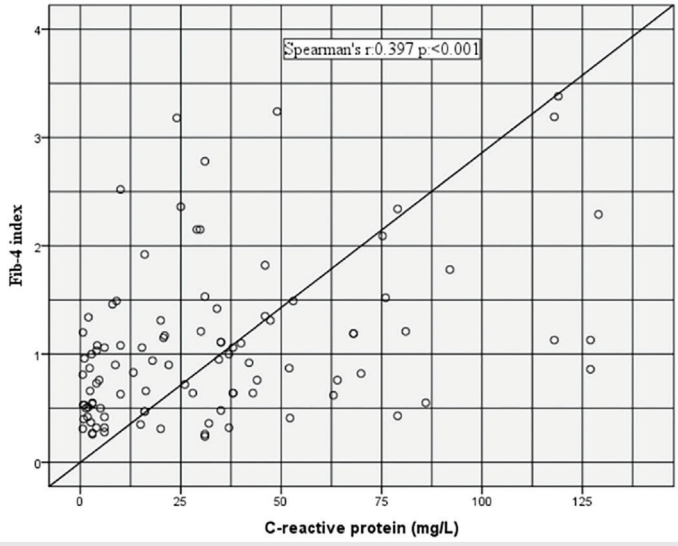


**Figure 2.** Spearman's correlation analysis between Fib-4 index and CRP. Fib-4: Fibrosis-4, CRP: C- reactive protein

## Discussion

This study investigated the relationship between Fib-4i and 1-year MACEs in IMPS patients. Fib-4i was higher in LGE-positive patients. Despite the absence of a demonstrable relationship between this elevation and MACEs, a positive correlation was identified between the Fib-4i and hs-troponin T.

Fib-4i has been demonstrated to be a significant predictor in various clinical scenarios. The increased index is an independent risk factor for diabetic neuropathy and chronic kidney disease in patients with type 2 diabetes, hepatocellular carcinoma in patients with chronic hepatitis B, in patients with atrial fibrillation-related stroke, and hospitalized acute HF (12-16). Despite the general prognostic ability of Fib-4i, it did not emerge as an independent prognostic factor in outcome of this study. The absence of HF, arrhythmia, and pericardial effusion in patients may have led to this outcome. In a multicenter study involving 1,162 patients hospitalized due to acute HF, it was demonstrated that Fib-4i calculated at admission was an important predictor of both all-cause mortality, and rehospitalization (16).

In another study, 704 HF patients were followed for 5 years. The patients were divided into three groups: HF with preserved EF, HF with mildly reduced EF, and HF with reduced EF. The primary outcome was a composite of total cardiovascular events (CVEs). Cardiovascular-related death; hospitalization for HF decompensation; non-fatal MI; unstable angina pectoris; coronary revascularization for a new diagnosis of angina or for in-stent restenosis after percutaneous coronary intervention; and non-fatal ischemic stroke were defined as total CVEs. In study, it was found that, Fib-4i was a substantial predictor for total CVEs in HF population (6). In both patient groups, systemic venous congestion increases neurohormonal activation. This activation leads to HF progression and may contribute to more severe multiple organ failure, resulting in a poor prognosis. HF can involve congestion and reduced arterial flow, resulting in hypoxic hepatopathy. Hypoxia can cause centrilobular necrosis of the liver, leading to elevated transaminase levels. Increased central venous pressure also causes hepatocyte atrophy and perisinusoidal oedema. Evidence suggests a link between liver congestion and liver stiffness. This can lead to fibrosis. This can result in a poor prognosis (6,17-19). IMPS represents a clinical spectrum. It can range from a silent clinical course to fulminant hepatitis or constrictive pericarditis. If the clinical presentation leads to non-ischaemic dilated HF or right HF secondary to constrictive pericarditis, fibrosis is triggered by the aforementioned pathophysiology. However, hepatomegaly, splenomegaly, or both may be present in patients, and reduced platelet counts may also occur. Consequently, the FIB-4 index may be elevated in these patients (2,4,6,16-18).

Our study established positive correlations between Fib-4i and hs-troponin T and between Fib-4i and CRP. In a retrospective analysis, the CMRI data and laboratory parameters of 244 patients with clinical suspicion of acute myocarditis were assessed. Analysis demonstrated that hs-troponin T levels ≤18 pg/mL corresponded to a very low-risk of acute myocarditis, suggesting that CMRI may not be necessary for exclusion (19). Another study assessed the correlation between hs-troponin I levels and myocardial damage on CMRI, represented by LGE percentage, in patients diagnosed with myocarditis. The study consisted of 101 patients. They found a linear association between LGE percentage and maximal hs troponin I value with r: 0.49 ($p<0.001$) (20). In light of this information, two hypotheses can be established: 1) In patients with negative hs-troponin T values and no other clinical conditions, the Fib-4i may also remain low. However, a cut-off value must be determined for this purpose. 2) A positive correlation between hs-troponin T and the Fib-4i may also exist between LGE percentage and Fib-4i. However, additional studies are required to substantiate these findings.

### Study Limitations

The study had several limitations: 1) Sample size was relatively small. A more extensive study is required to determine whether the Fib-4i is an effective predictor of MACE. 2) The study was conducted at a single center and was observational. The inclusion of a single population and a single center in the study limits the generalisability of the results and suggests that further research is required to assess predictive value of Fib-4i in other populations. 3) Sample population comprised young patients with uncomplicated IMPS. A more homogeneous population was required to enable a more accurate assessment. 4) Another limitation is the lack of quantitative assessment of the LGE ratio on CMRI.

## Conclusion

This study demonstrated that the FIB-4 index was higher in IMPS patients with LGE detected on CMRI, and there was a positive correlation between troponin and the FIB-4 index. Therefore, Fib-4i could be useful for assessing clinical risk in this patient group.

### Ethics

**Ethics Committee Approval:** The study was approved by the Ethics Committee of the University of Health Sciences Türkiye, Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital (approval number: 2025.01-04, date: 14.01.2025).

**Informed Consent:** Because of the retrospective design of the study, individual informed consent was waived.

### Footnotes

**Authorship Contributions:** Surgical and Medical Practices - E.A., K.S., K.B., G.C., H.Ş., A.E.T.; Concept - E.A., M.E.; Design - E.A., M.E.; Data Collection or Processing - K.S., K.B., G.C., H.Ş., A.E.T.; Analysis or Interpretation - E.A., M.E.; Literature Search - E.A., K.S., K.B., G.C., H.Ş., A.E.T.; Writing - E.A.

**Conflict of Interest:** No conflict of interest was declared by the authors.

**Financial Disclosure:** The authors declared that this study received no financial support.

## References

1. Lampejo T, Durkin SM, Bhatt N, Guttmann O. Acute myocarditis: aetiology, diagnosis and management. Clin Med (Lond). 2021; 21: e505-10.
2. Schulz-Menger J, Collini V, Gröschel J, Adler Y, Brucato A, Christian V, et al. 2025 ESC Guidelines for the management of myocarditis and pericarditis. Eur Heart J. 2025; 46: 3952-4041.

3. Usalp S, Altuntaş E, Bağırtan B, Karabay KÖ. Comparison of serum lipoprotein(a) levels in young and middle-aged patients presenting for the first time with ST-elevation myocardial infarction: a single-centre study. Cardiovasc J Afr. 2023; 34: 1-5.

4. Writing Committee; Drazner MH, Bozkurt B, Cooper LT, Aggarwal NR, Basso C, Bhave NM, et al. 2024 ACC Expert Consensus decision pathway on strategies and criteria for the diagnosis and management of myocarditis: a report of the American College of Cardiology Solution Set Oversight Committee. J Am Coll Cardiol. 2025; 85: 391-431.

5. Caforio AL, Pankuweit S, Arbustini E, Basso C, Gimeno-Blanes J, Felix SB, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J. 2013; 34: 2636-48, 2648a-2648d.

6. Takae M, Fujisue K, Yamamoto E, Egashira K, Komorita T, Oike F, et al. Prognostic significance of liver stiffness assessed by fibrosis-4 index in patients with heart failure. ESC Heart Fail. 2021; 8: 3809-21.

7. Raposeiras-Roubín S, Parada Barcia JA, Lizancos Castro A, Noriega Caro V, Ledo Piñeiro A, González Bermúdez I, et al. Liver fibrosis and outcomes of atrial fibrillation: the FIB-4 index. Clin Res Cardiol. 2024; 113: 313-23.

8. McNally BB, Rangan P, Wijarnpreecha K, Fallon MB. Fibrosis-4 index score predicts concomitant coronary artery diseases across the spectrum of fatty liver disease. Dig Dis Sci. 2023; 68: 3765-73.

9. Sudo M, Shamekhi J, Sedaghat A, Aksoy A, Zietzer A, Tanaka T, et al. Predictive value of the Fibrosis-4 index in patients with severe aortic stenosis undergoing transcatheter aortic valve replacement. Clin Res Cardiol. 2022; 111: 1367-76.

10. Wiegers SE, Ryan T, Arrighi JA, Brown SM, Canaday B, Damp JB, et al. 2019 ACC/AHA/ASE advanced training statement on echocardiography (Revision of the 2003 ACC/AHA clinical competence statement on echocardiography): a report of the ACC Competency Management Committee. J Am Coll Cardiol. 2019; 74: 377-402.

11. Galderisi M, Cosyns B, Edvardsen T, Cardim N, Delgado V, Di Salvo G, et al. Standardization of adult transthoracic echocardiography reporting in agreement with recent chamber quantification, diastolic function, and heart valve disease recommendations: an expert consensus document of the European Association of Cardiovascular Imaging. Eur Heart J Cardiovasc Imaging. 2017; 18: 1301-10.

12. Kim K, Oh TJ, Cho HC, Lee YK, Ahn CH, Koo BK, et al. Liver fibrosis indices are related to diabetic peripheral neuropathy in individuals with type 2 diabetes. Sci Rep. 2021; 11: 24372.

13. Seko Y, Yano K, Takahashi A, Okishio S, Kataoka S, Okuda K, et al. FIB-4 index and diabetes mellitus are associated with chronic kidney disease in Japanese patients with non-alcoholic fatty liver disease. Int J Mol Sci. 2019; 21: 171.

14. Suzuki T, Matsuura K, Nagura Y, Iio E, Ogawa S, Fujiwara K, et al. Development of hepatocellular carcinoma from various phases of chronic hepatitis B virus infection. PLoS One. 2021; 16: e0261878.

15. Kim TH, Kim SY, Jung YK, Yim HJ, Jung JM, Seo WK. FIB-4 index and liver fibrosis are risk factors for long-term outcomes in atrial fibrillation-related stroke. Clin Neurol Neurosurg. 2022; 217: 107235.

16. Shibata N, Kondo T, Kazama S, Kimura Y, Oishi H, Arao Y, et al. Impact of predictive value of Fibrosis-4 index in patients hospitalized for acute heart failure. Int J Cardiol. 2021; 324: 90-95.

17. Jalal Z, Iriart X, De Lédinghen V, Barnetche T, Hiriart JB, Vergniol J, et al. Liver stiffness measurements for evaluation of central venous pressure in congenital heart diseases. Heart. 2015; 101: 1499-504.

18. Mohamed BA, Schnelle M, Khadjeh S, Lbik D, Herwig M, Linke WA, et al. Molecular and structural transition mechanisms in long-term volume overload. Eur J Heart Fail. 2016; 18: 362-71.

19. Puetz A, Berger M, Lebherz C, Bauermann K, Hartmann NU, Kappel BA, et al. High-sensitivity troponin T as a rule-out marker for myocardial inflammation detectable by CMR imaging. Open Heart. 2025; 12: e003508.

20. Marcusohn E, Barbara A, Epstein D, Massalha S, Zukermann R. Correlations between high sensitive troponin I and acute myocarditis extent in cardiac magnetic resonance imaging. J Cardiovasc Med (Hagerstown). 2023; 24: 334-9.