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Research Article

The role of the triglyceride-glucose index (TyG Index) in predicting disease severity, ICU admission, and total hospital stay in patients with myocarditis

Miyokarditli hastalarda hastalık şiddetini, YBÜ'ye yatışı ve toplam hastanede kalış süresini tahmin etmede trigliserit-glikoz indeksinin (TyG indeksi) rolü

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Abstract

Aim: Myocarditis is characterized by myocardial inflammation, with varying etiologies, including infectious and autoimmune causes, and presents with a broad range of clinical severity. Identifying prognostic markers is essential to tailor treatment and optimize outcomes. This study aims to evaluate the Triglyceride-Glucose (TyG) Index as a potential marker for disease severity, ICU admission, and hospital length of stay in patients with acute myocarditis.

Material and Methods: In this retrospective study, 326 patients diagnosed with acute myocarditis between January 2015 and December 2023 were analyzed. Clinical and laboratory data, including demographics, disease severity markers, and TyG Index values, were collected. Statistical analyses evaluated associations between TyG Index and key clinical outcomes, such as ICU admission and total hospital stay.

Results: Patients with higher TyG Index values had significantly increased ICU admission rates, prolonged hospital stays, and higher levels of inflammatory markers, including CRP and ferritin. The TyG Index also correlated with markers of myocardial injury, such as elevated troponin and D-dimer levels, and was notably higher in patients with comorbidities like hypertension, diabetes, and hyperlipidemia.

Conclusion: The TyG Index appears to be a valuable biomarker for assessing myocarditis severity and predicting clinical outcomes. Given its accessibility, the TyG Index could be a practical tool for risk stratification in clinical settings. Prospective studies are needed to confirm these findings and further clarify its role in the pathophysiology of myocarditis.

Keywords: Myocarditis, TyG Index, ICU admission, Disease severity

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Öz

Amaç: Myokardit, enfeksiyöz ve otoimmün nedenler dahil olmak üzere çeşitli etiyolojilere sahip miyokardiyal inflamasyon ile karakterizedir ve geniş bir klinik şiddet aralığında seyredebilir. Prognostik belirteçlerin belirlenmesi, tedavinin bireyselleştirilmesi ve klinik sonuçların iyileştirilmesi açısından önemlidir. Bu çalışmada, Trigliserid-Glukoz (TyG) İndeksi'nin akut myokarditli hastalarda hastalık şiddeti, yoğun bakım ihtiyacı ve hastanede yatış süresi ile ilişkisini değerlendirmek amaçlanmıştır.

Gereç ve Yöntemler: Bu retrospektif çalışmada, Ocak 2015 - Aralık 2023 tarihleri arasında akut myokardit tanısı alan 326 hasta analiz edilmiştir. Demografik veriler, hastalık şiddeti belirteçleri ve TyG İndeksi değerleri dahil olmak üzere klinik ve laboratuvar verileri toplanmıştır. İstatistiksel analizlerle TyG İndeksi'nin yoğun bakım yatışı ve toplam hastanede kalış süresi gibi klinik sonuçlarla ilişkisi değerlendirilmiştir.

Bulgular: Yüksek TyG İndeksi değerine sahip hastalarda yoğun bakım yatış oranları belirgin şekilde artmış, hastanede kalış süresi uzamış ve CRP, ferritin gibi inflamatuar belirteçler daha yüksek bulunmuştur. Ayrıca, TyG İndeksi miyokard hasarı belirteçleri olan troponin ve D-dimer seviyeleri ile de korelasyon göstermiştir. Hipertansiyon, diyabet ve hiperlipidemi gibi ek hastalıkları olan bireylerde TyG İndeksi daha yüksek bulunmuştur.

Sonuç: TyG İndeksi, myokardit şiddetini değerlendirmede ve klinik sonuçları öngörmede değerli bir biyobelirteç olabilir. Kolay erişilebilirliği sayesinde, klinik pratiğe yönelik risk sınıflandırmasında pratik bir araç olarak kullanılabilir. Bulguların doğrulanması ve myokardit patofizyolojisindeki rolünün daha iyi anlaşılması için ileriye dönük çalışmalar gereklidir.

Anahtar Kelimeler: Myokardit, TyG İndeksi, Yoğun bakım yatışı, Hastalık şiddeti

Introduction

Myocarditis is a condition characterized by inflammation of the myocardium. Various etiologies, including viral or bacterial infections and autoimmune reactions, can contribute to this clinical presentation (1). The prevalence of myocarditis varies depending on the population and diagnostic criteria used, but it is estimated to affect 10 to 22 individuals per 100,000 persons annually (2). Common symptoms include chest pain, which may mimic myocardial infarction, as well as fatigue, dyspnea, and palpitations (3,4). Diagnostic methods for myocarditis encompass electrocardiography (ECG), echocardiography, cardiac magnetic resonance imaging (MRI), and endomyocardial biopsy (4,5). These tools are essential for identifying inflammation and structural changes in the heart, which are critical for confirming the diagnosis.

The length of hospital stay for patients with myocarditis can vary widely depending on numerous factors (6). These factors include the patient's clinical condition, treatment response, presence of complications, and post-discharge care needs (7). For instance, patients exhibiting symptoms of heart failure or arrhythmias may require prolonged observation in the hospital. Conversely, patients with mild symptoms and no complications may be suitable for outpatient management with short-term follow-up (8).

Treatment for myocarditis largely depends on the severity of the disease, which can range from a self-limiting condition to a life-threatening state (9). Therapeutic strategies vary accordingly and may include supportive care, immunosuppressive therapy, and, in severe cases, mechanical circulatory support or heart transplantation (10,11). Identifying the factors that determine disease severity is crucial for tailoring treatment plans and improving patient outcomes. Traditional biomarkers, such as troponins and natriuretic peptides, have been used to assess myocardial injury and stress, yet there remains a need for additional biomarkers that could offer more comprehensive insights into disease activity and prognosis (12).

Elevated triglyceride-glucose (TyG) index values are associated with insulin resistance and an increased risk of metabolic syndrome (13). Recent studies have shown that the TyG index is an independent predictor of prognosis, demonstrating potential clinical utility in predicting cardiovascular risk among both diabetic and non-diabetic patients with cardiovascular disease (14). Inflammatory diseases like myocarditis involve a complex interplay between metabolic and inflammatory

processes. Therefore, further investigation is warranted to elucidate the role of the TyG index in myocarditis and its relationship with triglycerides and glucose. Understanding the role of the TyG index in myocardial pathophysiology may provide insights into disease mechanisms and contribute to the development of improved therapeutic approaches.

Material and Methods

Compliance with Ethical Standards

The study was reviewed and approved by the institutional research ethics board in accordance with the principles of the Declaration of Helsinki. Artificial intelligence-supported technologies were not used in the study. We received ethics committee approval from the Konya Necmettin Erbakan University Ethics Board. The ethics committee approval of the study was obtained by the decision of the university board meeting dated 17/05/2024 and numbered 2024/4975.

Study Design

This retrospective study was conducted to assess the use of the Triglyceride-Glucose (TyG) Index as a biomarker for determining disease severity and predicting ICU admission and total hospital stay duration in patients with acute myocarditis. Patients diagnosed with acute myocarditis at our institution between January 2015 and December 2023 were included in the study. A total of 326 patients were enrolled, comprising 177 males and 149 females. Patients presenting with resistant chest pain, hypotension, pericardial effusion, ejection fraction (EF) reduction, and those unresponsive to medical and symptomatic treatment were classified as having severe disease. Diagnosis was based on clinical assessment, cardiac biomarkers, and cardiac imaging, with coronary angiography or coronary CT angiography performed to exclude atherosclerotic heart disease. Although cardiac MRI is considered the gold standard for noninvasive myocarditis diagnosis, it was not routinely performed in our study due to its unavailability at our institution. Triglyceride-Glucose index was calculated with the formula TyG = ln(fasting triglyceride mg/dL × fasting glucose mg/dL/2).

Data on demographic information, clinical presentation, laboratory findings, imaging results, treatment, and outcomes were collected from medical records. Patients were eligible for inclusion if they were aged 18 years or older and had a confirmed diagnosis of acute myocarditis. Exclusion criteria were as follows: chronic kidney disease, liver disease, malignancy, any types of diabetes mellitus, pancreatic diseases, patients with incomplete medical records; and those who had received glucose- or triglyceride-altering therapies prior to myocarditis diagnosis. This approach was implemented to ensure homogeneity within the study population and to prevent confounding of TyG Index measurements by underlying conditions.

Statistical Analysis

Statistical analyses were performed using SPSS 21.0 (IBM Inc, Chicago, IL, USA) program in the study. Kolmogrov-Smirnov test, histogram analyses, skewness/kurtosis data and Q-Q plots were used to assess the conformity of numerical variables to normal distribution. Descriptive statistics of numerical and categorical data obtained in the study were analyzed and expressed as IQR (median [minimum-maximum]) since quantitative parameters did not exhibit a normal distribution pattern. Relationships between the two groups were examined with Mann-Whitney U test. Correlation relationships between quantitative parameters were performed with Spearman correlation analysis. Type-I error rate was taken as 5% ($\alpha = 0.05$) throughout the study and $p<0.05$ level was accepted as the significant limit.

Results

Table 1 summarizes the distribution of quantitative parameters in patients with myocarditis. Patient ages ranged from 18 to 74 years, with a median age of 40 years. White blood cell (WBC) counts demonstrated a broad range, with a median of 10.24 $\times 10^3$/μL (IQR: 8.01–23.82), while neutrophil counts had a median of 7.48 $\times 10^3$/μL (IQR: 3.62–19.93). Troponin levels varied significantly, ranging from 48 to 50,000 ng/L, with a median of 456 ng/L. Other noteworthy parameters included C-reactive protein (CRP) levels with a median of 33 mg/L (IQR: 10–348) and D-dimer levels with a median of 560.5 ng/mL (IQR: 365–987).

Table 2 compares parameters between male and female myocarditis patients. The median age for males was 46 years (IQR: 38–59), while for females, it was 34 years (IQR: 29–41), with a significantly higher age in males ($p = 0.023$). Hemoglobin levels were significantly higher in males (mean: 15.1 ± 1.2 g/dL) compared to females (mean: 13.4 ± 1.1 g/dL, $p < 0.001$). WBC and neutrophil counts were significantly lower in males,

with median values of 9.92 $\times 10^3$/µL (IQR: 8.08–21.26) and 7.2 $\times 10^3$/µL (IQR: 3.62–18.28), respectively. Females had a higher mean platelet count (253.8 ± 49.6 $\times 10^3$/µL). CRP levels were significantly higher in females (median: 38 mg/L, IQR: 16–284) compared to males (median: 29 mg/L, IQR: 12–174, p = 0.001). Triglyceride levels were also significantly higher in females (median: 127 mg/dL, IQR: 94–186) than in males (median: 124 mg/dL, IQR: 91–173, p = 0.028).

Table 3 presents the assessment of the TyG index according to specific clinical conditions. Patients with pericardial effusion, those requiring inotropic support, IV steroids, and IV immunoglobulin (IVIG) had significantly higher TyG indices. Additionally, the TyG index was notably elevated in patients with hypertension (HT), diabetes mellitus (DM), hyperlipidemia (HPL), a family history of atherosclerotic coronary artery disease, prior tonsillitis, and recent gastroenteritis within the past 3–4 weeks. These findings suggest that a higher TyG index is associated with indicators of more severe disease, such as pericardial effusion and the need for inotropic support, IV steroids, and IVIG.

Table 4 shows that patients with a longer hospital stay (>2 days) had higher mean WBC, neutrophil, CRP, troponin, and ferritin levels compared to those with shorter stays. Importantly, patients with a hospital stay longer than two days had a significantly higher TyG index (p < 0.001).

Table 5 presents correlations between the TyG index and other clinical parameters, revealing significant associations with ICU stay duration, total hospital stay, and various hematological and biochemical markers. These findings suggest that the TyG index could serve as a useful indicator of disease severity and hospital outcomes in myocarditis patients. Strong positive correlations were observed between total hospital stay and elevated troponin, D-dimer, and fibrinogen levels. Additionally, there was a strong positive correlation between uric acid and the TyG index. A strong negative correlation was found between age and levels of ASO and CRP.

Table 1. Summary of the general distribution of quantitative parameters in myocarditis patients.

Parameters	Unit	Minimum	Maximum	Distribution †
Age	years	18	74	40 (18 – 74)
WBC	103/mL	8.01	23.82	10.24 (8.01 – 23.82)
Neutrophil	103/mL	3.62	19.93	7.48 (3.62 – 19.93)
Monocyte	%	0.04	1.82	0.67 (0.04 – 1.82)
Hemoglobin	g/dL	10.50	17.70	14.3±1.4
Lymphocyte	103/mL	0.40	4.78	2.15±0.69
Platelet	103/mL	146.0	366.0	245.4±49.1
RDW	%	11.2	17.7	13.4±1.1
Albumin	g/L	32.0	50.6	42.6±3
LDL	mg/dL	53.0	198.0	135.4±23.7
HDL	mg/dL	23.0	65.0	44.0±6.6
TRG	ng/dL	72.0	307.0	126 (72 – 307)
ASO	IU/mL	111.0	387.0	180 (111 – 387)
EF	%	30.0	65.0	60 (30 – 65)
Troponin	ng/L	48	50000	456 (48 – 50000)
CRP	mg/L	10.0	348.0	33 (10 – 348)
D-dimer	ng/mL	365.0	987.0	560.5 (365 – 987)
Ferritin	ng/mL	19.0	165.0	76 (19 – 165)
Fibrinogen	ng/dL	2.56	4.16	3.41 (2.56 – 4.16)
Uric Acid	mg/dL	3.60	6.20	4.4 (3.6 – 6.2)
Glucose	mg/dL	77.0	167.0	97 (77 – 167)
TyG index		8.17	9.92	8.70 (8.17 – 9.92)
ICU Time	Day	1	10	1 (1 – 10)
Total Hospitalization Time	Day	1	14	3 (1 – 14)

† Parameters are expressed as IQR (Interquartile Range) [median, min and max] or mean±SD.
WBC: White Blood Cell; RDW: Red Cell Distribution Width; ASO: Antistreptolysin O; EF: Ejection Fraction; CRP: C Reactive Protein; LDL: Low Density Lipoprotein; HDL: High Density Lipoprotein; TRG: Triglyceride; TyG Index: Triglyceride-Glucose Index; ICU: Intensive Care Unit

Table 2. Comparison of parameters according to gender in myocarditis patients

		Sex		p
		Male (n=177, %54,3)	Female (n=149, %45,7)	
Parameters	Unit	Distribution*		
Age	years	46 (18 – 74)	34 (18 – 65)	0.023
WBC	103/mL	9.92 (8.08 – 21.26)	11.29 (8.01 – 23.82)	<0.001
Neutrophil	103/mL	7.2 (3.62 – 18.28)	8.36 (4.66 – 19.93)	<0.001
Monocyte	%	0.66 (0.04 – 1.82)	0.68 (0.28 – 1.8)	0.106
Hemoglobin	g/dL	15.1±1.2	13.4±1.1	<0.001
Lymphocyte	103/mL	2.2±0.73	2.1±0.64	0.188
Platelet	103/mL	238.3±47.7	253.8±49.6	0.005
RDW	%	13.6±1	13.2±1.2	0.005
Albumin	g/L	43.3±2.6	41.7±3.3	<0.001
LDL	mg/dL	136.2±25	134.5±22	0.522
HDL	mg/dL	41.8±6.1	46.7±6.3	<0.001
TRG	ng/dL	124 (72 – 298)	127 (78 – 307)	0.028
ASO	IU/mL	182 (111 – 387)	178 (113 – 386)	0.364
EF	%	60 (30 – 65)	60 (45 – 65)	<0.001
Troponin	ng/L	560 (51 – 50000)	455 (48 – 38990)	0.406
CRP	mg/L	29 (10 – 348)	38 (11 – 256)	0.001
D-dimer	ng/mL	577 (367 – 987)	550 (365 – 790)	0.026
Ferritin	ng/mL	67 (19 – 165)	78 (24 – 144)	0.001
Fibrinogen	ng/dL	3.38 (2.56 – 4.16)	3.41 (2.66 – 4.01)	0.801
Uric Acid	mg/dL	4.4 (3.6 – 6.2)	4.4 (3.6 – 6.1)	0.597
Glucose	mg/dL	96 (78 – 132)	99 (77 – 167)	0.649
TyG index		8.68 (8.17 – 9.82)	8.73 (8.35 – 9.92)	0.110
ICU Time	Day	1 (1 – 10)	1 (1 – 7)	0.818
Total Hospitalization Time	Day	2 (1 – 14)	3 (2 – 14)	0.112

† Parameters are expressed as IQR (Interquartile Range) [median, min and max] or mean±SD.
WBC: White Blood Cell; RDW: Red Cell Distribution Width; ASO: Antistreptolysin O; EF: Ejection Fraction; CRP: C Reactive Protein; LDL: Low Density Lipoprotein; HDL: High Density Lipoprotein; TRG: Triglyceride; TyG Index: Triglyceride-Glucose Index; ICU: Intensive Care Unit

Discussion

This study aimed to evaluate the potential use of the TyG Index as a marker for assessing disease severity and predicting ICU and total hospital stay durations in patients with acute myocarditis. Previous studies have demonstrated that, in cases of acute myocarditis, persistent chest pain, increased pericardial effusion, cardiac tamponade, hypotension, reduced ejection fraction, low cardiac output, and sustained ventricular arrhythmias indicate a more severe, refractory, and rapidly progressing disease course (15). The Lombardy registry, a multicenter Italian study involving 443 hospitalized patients with confirmed myocarditis, identified severe hemodynamic compromise at admission as the highest risk factor for cardiac mortality (16-18). Similarly, another study from 16 tertiary care centers, including 220 patients with biopsy-confirmed myocarditis, found that hemodynamic compromise at presentation was the primary determinant of both short- and long-term prognosis (19-21). In fulminant myocarditis, symptoms typically appear rapidly, within 2 days to 2 weeks, leading to marked hemodynamic dysfunction and circulatory failure, often necessitating aggressive blood pressure management and vasopressors, with mechanical circulatory support devices required in advanced stages (22-24).

Our findings indicate that higher TyG Index values are significantly associated with increased severity of myocarditis, correlating with longer ICU and total hospital stays, greater need for inotropic support, IV steroids, IVIG, and elevated levels of inflammatory markers such as CRP and ferritin, as well as elevated troponin, fibrinogen, and D-dimer levels. These results align with prior research suggesting that oxidative stress and inflammation play central roles in the pathophysiology of myocarditis.

Table 3. Comparison of TyG index values according to the presence of specific conditions

Parameters		TyG index Medyan (min-max)	P*
Pericardial effusion	No (n=224)	8.62 (8.17-9.53)	<0.001
	Yes (n=102)	9.33 (9.04-9.92)	
Beta blocker use	No (n=191)	8.69 (8.17-9.79)	0.181
	Yes (n=135)	8.74 (8.19-9.92)	
ACEi use	No (n=191)	8.69 (8.17-9.79)	0.181
	Yes (n=135)	8.74 (8.19-9.92)	
Inotropic support	No (n=312)	8.69 (8.17-9.92)	<0.001
	Yes (n=14)	9.39 (9.14-9.68)	
IV steroid intake	No (n=308)	8.69 (8.17-9.92)	<0.001
	Yes (n=18)	9.39 (9.14-9.68)	
IVIG	No (n=318)	8.7 (8.17-9.92)	0.005
	Yes (n=8)	9.25 (9.14-9.44)	
Coronary angiography	No (n=85)	8.66 (8.17-9.4)	0.043
	Yes (n=241)	8.71 (8.19-9.92)	
Hypertension	No (n=190)	8.69 (8.17-9.71)	0.123
	Yes (n=136)	8.74 (8.19-9.92)	
Hyperlipidemia	No (n=247)	8.65 (8.17-9.68)	<0.001
	Yes (n=79)	8.87 (8.19-9.92)	
Diabetes Mellitus	No (n=269)	8.67 (8.17-9.71)	<0.001
	Yes (n=57)	8.84 (8.46-9.92)	
Smoking	No (n=147)	8.71 (8.19-9.79)	0.996
	Yes (n=179)	8.7 (8.17-9.92)	
Family History	No (n=244)	8.68 (8.17-9.79)	0.003
	Yes (n=82)	8.82 (8.19-9.92)	
Obesity	No (n=135)	8.71 (8.17-9.68)	0.603
	Yes (n=191)	8.7 (8.19-9.92)	
Flu in 4 weeks	No (n=121)	8.69 (8.17-9.54)	0.09
	Yes (n=205)	8.74 (8.19-9.92)	
Tonsillitis	No (n=200)	8.73 (8.19-9.92)	0.123
	Yes (n=126)	8.67 (8.17-9.71)	
Gastroenteritis within 4 weeks	No (n=263)	8.69 (8.17-9.92)	0.002
	Yes (n=63)	8.81 (8.33-9.82)	
Coronary CT Angiography	No (n=238)	8.71 (8.19-9.92)	0.055
	Yes (n=88)	8.68 (8.17-9.4)	

† Parameters are expressed as IQR (Interquartile Range) [median, min and max].
*Mann-Whitney U test.
IV: intravenous; IV: Intravenous Immunoglobulin; ECG: electrocardiography; CT: Computed Tomography; ACEI: ACE inhibitor

The relationship between TyG Index and disease severity, prognosis, ICU stay, and overall hospital stay in myocarditis is a complex issue requiring further detailed investigation. The Tehran Lipid and Glucose Study found a significant association between the TyG Index and increased cardiovascular and coronary heart disease risk in 7,521 Iranian men (25). The Kailuan Study reported that high initial and long-term TyG Index levels are associated with an increased risk of myocardial infarction (26). Research conducted in Korea demonstrated that an elevated TyG Index is a significant indicator of ischemic heart disease in a non-diabetic population (27). Additionally, the Atherosclerosis Risk in Communities (ARIC) Study identified a higher TyG Index as independently associated with an increased risk of peripheral artery disease (28). The cumulative exposure, variability, and progression of the TyG Index have been linked to higher cardiovascular disease rates over time (29-32).

Table 4. Comparison of quantitative parameters with total hospital stay groups

		Total Hospitalization Time		
		1-2 day (n=159, %48,8)	>2 day (n=167, %51,2)	P**
Parameters	Unit	Distribution*		
Age	year	43 (19 – 68)	32 (18 – 74)	0.011
WBC	103/mL	9.78 (8.01 – 23.82)	11.95 (8.23 – 22.71)	<0.001
Neutrophil	103/mL	6.72 (3.62 – 19.93)	8.66 (3.67 – 18.47)	<0.001
Monocyte	%	0.66 (0.04 – 1.37)	0.69 (0.2 – 1.82)	0.013
Hemoglobin	g/dL	14.5±1.4	14.1±1.5	0.030
Lynphocyte	103/mL	2.22±0.77	2.08±0.6	0.064
Platelet	103/mL	246.5±51	244.3±47.4	0.687
RDW	%	13.2±1.1	13.7±1	<0.001
Albumin	g/L	42.8±3.0	42.3±3.0	0.132
LDL	mg/dL	130.7±22.8	139.9±23.7	<0.001
HDL	mg/dL	44.6±6.9	43.5±6.4	0.131
TRG	ng/dL	118 (72 – 290)	132 (77 – 307)	<0.001
ASO	IU/mL	172 (111 – 387)	189 (121 – 386)	<0.001
EF	%	60 (45 – 65)	60 (30 – 65)	<0.001
Troponin	ng/L	228 (48 – 18511)	4246 (51 – 50000)	<0.001
CRP	mg/L	23 (10 – 203)	43 (10 – 348)	<0.001
D-dimer	ng/mL	506 (365 – 722)	598 (367 – 987)	<0.001
Ferritin	ng/mL	67 (19 – 165)	78 (22 – 165)	0.001
Fibrinogen	ng/dL	3.12 (2.56 – 3.87)	3.61 (2.88 – 4.16)	<0.001
Uric Acid	mg/dL	4.3 (3.6 – 6.1)	4.7 (3.6 – 6.2)	<0.001
Glucose	mg/dL	93 (78 – 167)	101 (77 – 145)	<0.001
TyG index		8.62 (8.19 – 9.72)	8.83 (8.17 – 9.92)	<0.001

† Parameters are expressed as IQR (Interquartile Range) [median, min and max] or mean±SD.
**Mann-Whitney U test.
WBC: White Blood Cell; RDW: Red Cell Distribution Width; ASO: Antistreptolysin O; EF: Ejection Fraction; CRP: C Reactive Protein; LDL: Low Density Lipoprotein; HDL: High Density Lipoprotein; TRG: Triglyceride; TyG Index: Triglyceride-Glucose Index.

Emerging evidence suggests that the TyG Index reflects underlying metabolic dysregulation, which may contribute to the pathophysiology of myocarditis through mechanisms involving oxidative stress, endothelial dysfunction, and systemic inflammation. Insulin resistance, a key component of TyG Index elevation, has been associated with increased pro-inflammatory cytokine release, mitochondrial dysfunction, and impaired myocardial energy metabolism, all of which may exacerbate myocardial injury in acute myocarditis. Additionally, hypertriglyceridemia promotes lipotoxicity and enhances the production of reactive oxygen species, leading to cardiomyocyte apoptosis and fibrosis. These processes may amplify the inflammatory cascade and perpetuate myocardial damage. Given the established role of oxidative stress and metabolic disturbances in myocarditis, the strong association between a high TyG Index and severe disease manifestations in our cohort supports the hypothesis that metabolic and inflammatory pathways are intertwined in myocardial injury.

In our study, patients with pericardial effusion, those requiring inotropic support, IV steroid, and IVIG treatments had significantly higher TyG Index values. These findings suggest that the TyG Index may be influenced by both the clinical presentation of myocarditis and the therapeutic interventions employed. A high TyG Index was also observed in patients with a history of tonsillitis or recent gastroenteritis (3–4 weeks prior to diagnosis), which may reflect infectious etiologies. Additionally, the association between an elevated TyG Index and comorbidities such as HT, DM, HPL, and a family history of atherosclerotic risk factors suggests that these comorbidities may enhance the inflammatory response.

Methodologically, the TyG Index can be easily calculated based on routine blood biochemistry tests, making it accessible for

Table 5. Examining the correlation relationships of quantitative parameters with age, ICU length of stay, total hospital length of stay and TyG index

		Age	ICU Time	Hospitalization Time	TyG index
Age	rho	–	-0,204	-0,155	0,114
	P	–	<0,001	0,005	0,039
ICU Time	rho	-0,204	–	0,475	0,331
	P	<0,001	–	<0,001	<0,001
Hospitalization Time	rho	-0,155	0,475	–	0,41
	P	0,005	<0,001	–	<0,001
TyG index	rho	0,114	0,331	0,41	–
	P	0,039	<0,001	<0,001	–
WBC	rho	-0,305	0,304	0,424	0,476
	P	<0,001	<0,001	<0,001	<0,001
Neutrophil	rho	-0,271	0,311	0,414	0,435
	P	<0,001	<0,001	<0,001	<0,001
Monocyte	rho	-0,29	0,224	0,244	0,073
	P	<0,001	<0,001	<0,001	0,186
Hemoglobin	rho	0,092	-0,209	-0,182	-0,356
	P	0,096	<0,001	0,001	<0,001
Lymphocyte	rho	0,003	-0,073	-0,052	0,175
	P	0,952	0,186	0,347	0,001
Platelet	rho	0,096	-0,071	-0,017	0,186
	P	0,084	0,204	0,754	0,001
RDW	rho	0,207	0,293	0,314	0,555
	P	<0,001	<0,001	<0,001	<0,001
Albumin	rho	0,068	-0,269	-0,186	-0,321
	P	0,219	<0,001	0,001	<0,001
LDL	rho	0,36	0,161	0,276	0,528
	P	<0,001	0,004	<0,001	<0,001
HDL	rho	-0,479	-0,101	-0,051	-0,273
	P	<0,001	0,068	0,36	<0,001
TRG	rho	-0,029	0,329	0,412	–
	P	0,606	<0,001	<0,001	–
ASO	rho	-0,696	0,106	0,143	-0,025
	P	<0,001	0,056	0,01	0,656
EF (%)	rho	-0,454	-0,121	-0,223	-0,22
	P	<0,001	0,029	<0,001	<0,001
Troponin	rho	-0,371	0,287	0,678	0,131
	P	<0,001	<0,001	<0,001	0,018
CRP	rho	-0,609	0,34	0,438	0,167
	P	<0,001	<0,001	<0,001	0,002
D-dimer	rho	-0,153	0,358	0,618	0,274
	P	0,006	<0,001	<0,001	<0,001
Ferritin	rho	-0,587	0,344	0,248	0,074
	P	<0,001	<0,001	<0,001	0,184
Fibrinogen	rho	-0,1	0,362	0,652	0,323
	P	0,07	<0,001	<0,001	<0,001
Uric Acid	rho	-0,11	0,351	0,353	0,673
	P	0,047	<0,001	<0,001	<0,001
Glucose	rho	0,191	0,255	0,298	–
		0,001	<0,001	<0,001	–

*Spearman correlation analysis.
WBC: White Blood Cell; RDW: Red Cell Distribution Width; ASO: Antistreptolysin O; EF: Ejection Fraction; CRP: C Reactive Protein; LDL: Low Density Lipoprotein; HDL: High Density Lipoprotein; TRG: Triglyceride; TyG Index: Triglyceride-Glucose Index.

clinical applications worldwide. This straightforward approach may provide valuable information regarding disease severity, prognosis, ICU, and total hospital stay durations in myocarditis patients. However, further studies are necessary to validate the clinical applicability of these parameters. Future research should also consider the potential influence of factors such as ethnicity, comorbidities, and follow-up duration on outcomes.

Limitations

This study has several limitations that should be acknowledged. First, the diagnosis of myocarditis in our cohort was based on clinical presentation and non-invasive tests rather than on endomyocardial biopsy or cardiac MRI, which are considered gold standards for definitive diagnosis. The absence of these diagnostic methods may have led to misclassification or underestimation of disease presence and severity. Second, we did not measure B-type natriuretic peptide (BNP) levels, a well-established biomarker in heart failure and myocarditis. BNP could have provided additional insights into the severity of myocardial dysfunction and patients' hemodynamic status. The lack of BNP data may limit a comprehensive assessment of cardiac function in our study population.

Additionally, this study's retrospective nature introduces the potential for selection and recall bias. Reliance on medical records for data collection may lead to missing or inaccurate information, potentially affecting study outcomes. Prospective studies are needed to validate our findings and provide more robust evidence. Another limitation involves potential confounding factors not accounted for in our analysis. Underlying comorbidities, medications, and lifestyle factors (e.g., diet, smoking) could influence glucose and triglyceride levels, thereby affecting the TyG Index. A more comprehensive analysis controlling for these variables would strengthen the validity of our results.

Conclusion

In conclusion, our study provides evidence supporting the use of TyG Index as a new marker for assessing disease severity, ICU and total hospital length of stay in patients with acute myocarditis. By integrating markers of oxidative stress and nutritional/inflammatory status, TyG Index provides a comprehensive tool for risk stratification and management in clinical practice. Future studies should aim to confirm these findings in larger, prospective cohorts and explore the mechanistic pathways linking TyG Index to myocarditis severity.

Ethics Committee Approval

This study complies with all relevant national regulations, institutional policies, and the principles of the Declaration of Helsinki and has been approved by the Ethics Committee of Konya Necmettin Erbakan University Faculty of Medicine (approval number: 2024/4975).

Informed Consent

All rights of the participants were protected and written informed consent was obtained before the procedures in accordance with the Declaration of Helsinki.

Author Contributions

Concept - A.E.; Design - A.T.S.; Supervision - H.E.; Resources - A.E.; Materials - A.E., H.E.; Data Collection and/or Processing - A.E.; Analysis and/or Interpretation - A.T.S.; Literature Review - A.E.; Writing the Article - H.E.; Critical Review - A.E., A.T.S.

Conflict of Interest

The authors have no conflict of interest to declare.

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