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Correlation between Triglycerides Glucose Index and Left Ventricular Global Longitudinal Strain in Prediction of Heart Failure in Cases with NSTE-ACS

Mahmoud S. Abd Elmoneam, Osama S. Arafa, Esraa I. Mohammed, Amr M. Abd Elaziz

Department of Cardiology, Faculty of Medicine Benha University, Egypt.

Corresponding to: Esraa I. Mohammed, Department of Cardiology, Faculty of Medicine Benha University, Egypt.

Email: esraaismail213@gmail.com

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Abstract

Background: Insulin resistance (IR) is a central contributor to the development of both atherosclerotic disease and myocardial dysfunction. Triglyceride-glucose (TyG) index has gained prominence due to its simplicity and reliability. Concurrently, global longitudinal strain (GLS) has emerged as a highly sensitive parameter for the early diagnosis of subclinical left ventricular (LV) systolic impairment. Within the clinical landscape of non-ST elevation acute coronary syndrome (NSTE-ACS), early identification of cases predisposed to heart failure (HF) remains a significant diagnostic hurdle. This investigation explored the association between the TyG index and LV GLS in cases diagnosed with NSTE-ACS, utilizing speckle-tracking echocardiography as the primary assessment modality. **Methods:** This prospective cohort study was done at Benha University Hospital, including 315 cases admitted with NSTE-ACS between November 2023 and November 2024. Cases were divided into two groups: Group I (TyG index < 8.47) and Group II (TyG index ≥ 8.47).**Results:** The TyG index showed a strong inverse correlation with GLS (r = -0.673, P<0.001), indicating that elevated TyG values are associated with reduced myocardial deformation.. Multivariate logistic regression exhibited that the TyG index independently predicted in-hospital major adverse cardiovascular events (OR = 12.861, 95% CI = 4.03–41.043, P<0.001), even after adjusting for confounders. **Conclusion:** In NSTE-ACS, an elevted TyG index is associated with greater impairment of LV function as measured by speckle-tracking echocardiography. Routine assessment of the TyG index may aid in identifying cases at increased risk of adverse cardiac outcomes.

Keywords: TyG index, Global longitudinal strain, Speckle-tracking echocardiography, Heart failure, NSTE-ACS.

Introduction

Heart failure (HF) continues to represent a widespread and enduring complication leading to hospitalization among cases diagnosed with coronary heart disease (CHD). Importantly, this risk persists even after successful revascularization procedures, as cases with CHD frequently demonstrate a sustained vulnerability to progressive left ventricular dysfunction, reflected by reduced ejection fraction (LVEF), which predisposes them to the development of HF over time [(1).]

Extensive prior investigations have consistently identified diabetes mellitus (DM), hypertension (HTN), obesity, and multi-vessel coronary artery disease (CAD) as independent predictors of chronic-phase cardiac insufficiency in CHD cohorts. More recently, clinical data have underscored the contributory role of hypertriglyceridemia, implicating disordered lipid metabolism in recurrent cardiovascular events (CVEs). Elevated triglycerides (TG) in CHD cases are now recognized as a potential aggravating factor in the progression of cardiovascular disease (CVD), suggesting a more multifactorial metabolic involvement than previously appreciated [(2).]

Despite substantial progress in preventive cardiology, diagnostic methodologies, and therapeutic strategies over recent decades, atherosclerosis continues to represent the principal pathological foundation underlying cardiovascular disease (CVD). Its clinical manifestations, including coronary artery disease (CAD) and cerebrovascular accidents (CVAs), remain the fundamental causes of mortality worldwide. Disruptions in metabolic homeostasis, most notably chronic hyperglycemia and dyslipidemia, are well-recognized as fundamental etio-pathogenic contributors to the development and progression of CVD [(3)]

The triglyceride-glucose (TyG) index, first introduced in 2008, has gained traction as a simple yet robust surrogate for insulin resistance (IR), with demonstrated correlations to gold-standard measures such as the hyperinsulinemia-euglycemic clamp and the homeostatic model assessment for IR (HOMA-IR), supporting its clinical and investigational relevance [(4).]

Conventional transthoracic echocardiography (TTE) continues to be a cornerstone in noninvasive cardiac imaging, facilitating evaluation of both structural and functional cardiac parameters, even in populations with chronic kidney disease (CKD). However, multiple investigations have shown that assessment of LV mechanics via global longitudinal strain (GLS), a sensitive parameter reflecting myocardial deformation during the cardiac cycle, yields superior diagnostic and prognostic insights compared to LVEF alone [(5).]

Two-dimensional speckle-tracking echocardiography (2D-STE) has emerged as an advanced echocardiographic technique enabling quantification of regional and global LV function, as well as left atrial (LA) dynamics. By tracing intrinsic acoustic reflections or “speckles” across sequential

frames of the cardiac cycle, 2D-STE provides reproducible measurements of myocardial strain and velocity. Validation investigations using sonomicrometry and cardiac magnetic resonance imaging (MRI) tagging have confirmed its accuracy and clinical utility [6].

The TyG index has also been associated with both the occurrence and prognostic trajectory of CVD. Elevated TyG levels correlate with increased CAD incidence, more severe angiographic lesion burden, and poorer long-term outcomes; even among cases initially free of obstructive CAD [7].

Furthermore, increased TyG values have demonstrated a direct relationship with structural LV remodeling, enhanced myocardial fibrosis, and elevated risk for HF progression [8].

This investigation assessed the relationship between TyG index and GLS, utilizing 2D-STE to evaluate subclinical LV dysfunction in cases presenting with CHD and HF in the clinical context of non-ST elevation ACS (NSTE-ACS).

Patients and methods

Patients: This prospective, single-center cohort study enrolled 315 cases diagnosed with NSTE-ACS who were admitted to the coronary care unit at Benha University Hospital between November 2023 and November 2024. Prior to inclusion, informed written consent was obtained from all cases following a comprehensive explanation of the study's aims and procedures. To ensure confidentiality, each subject was assigned a unique anonymized identification code. The study protocol was reviewed and approved by the Institutional Ethics Committee (MS 9-11-2023) of the Faculty of Medicine, Benha University.

Eligibility Criteria

Inclusion criteria comprised adult cases above 18 years of age, of either sex, presenting with a confirmed or suspected diagnosis of ACS. Diagnosis was based on typical ischemic chest discomfort, ECG findings, imaging indicators of myocardial ischemia, or angiographic evidence of coronary thrombus. The research specifically focused on cases with NSTE-ACS, encompassing NSTEMI and UA. As per the 2023 ESC guidelines, NSTEMI was identified by elevated hs-cTn in the absence of persistent ST-segment elevation, in conjunction with clinical signs of ischemia. UA was diagnosed in the presence of ischemic symptoms and normal hs-cTn values, with or without accompanying ECG changes.

Exclusion criteria included chronic infectious illness, hepatic, or renal insufficiency with creatinine clearance <15 mL/min, and any malignant hematologic disorder. Other criteria for exclusion were severe valvular abnormalities, prior diagnosis of HF, extreme obesity (BMI ≥45 kg/m²), neuropsychiatric illness, suspected familial hypertriglyceridemia (TG ≥500 mg/dL), or absence of baseline TyG index data.

Grouping

The enrolled population was stratified into two subgroups according to the median TyG value. Group I (n = 147) included cases with a TyG index <8.47, while Group II (n = 168) consisted of cases with TyG ≥8.47.

Clinical and Laboratory Evaluation

All cases underwent standardized evaluations, including detailed history and clinical examination. Demographic data included name, age, sex, and BMI. Cardiovascular history emphasized typical HF-related symptoms (dyspnea, orthopnea, paroxysmal nocturnal dyspnea, and exertional intolerance). Documentation of comorbidities such as HTN, DM, dyslipidemia, and CHF was also completed. Physical examination involved general inspection for congestion, along with assessment of pulse, HR, SBP, and DBP.

Laboratory workup included CBC, RBS, kidney and liver function panels, CK-MB, and troponin. Lipid profile analysis covered TGs, LDL, HDL, VLDL, and TC.

The TyG index was derived using a logarithmic transformation of the product of fasting TG and fasting plasma glucose concentrations, standardized to account for variations in reporting units. When biochemical parameters were expressed in mg/dL, the index was calculated as follows:

$$TyG = Ln[\text{fasting TG (mg}/dL) \times fasting\ glucose\ (\text{mg}/dL)]/2$$

In cases where analytes were reported in mmol/L, the following unit-adjusted formula was applied to ensure equivalency:

$$TyG = Ln[\text{fasting TG (mmol}/L) \times 88.57 \times fasting\ glucose\ (\text{mmol}/L) \times 18]/2$$

ECG Analysis

A standard 12-lead ECG was performed for all cases to validate diagnosis, identify rhythm disturbances, and assess ST segment deviations, T wave abnormalities or chamber enlargement.

Echocardiographic Protocol

All TTE assessments were carried out utilizing a high-definition imaging platform (EPIQ 7C; Philips, Amsterdam, The Netherlands) featuring a broadband S5-1 transducer. Imaging modalities, including 2D, M-mode, and Doppler, were acquired following the protocol outlined by the American Society of Echocardiography (ASE), with data securely stored on magneto-optical media and the EchoPAC archive system (Image Vault 5.0; GE Healthcare, Horten, Norway). Heart rate (HR) measurements were recorded while cases were in supine position during echocardiographic capture. Immediately after the TTE, systolic (SBP) and diastolic blood pressures (DBP) were obtained in the same posture.

Standardized cardiac measurements were obtained from the parasternal long-axis view and included LVIDd, SWTd, PWTd, and LADs. These parameters were selected to assess key structural aspects of LV and atrial dimensions. RWT was calculated using the formula (SWTd + PWTd) / LVIDd, while MWT was defined as the average thickness of the septal and inferior LV walls. LVEF

was determined using the modified Simpson's biplane method, which offers a reliable estimate of systolic performance.

LVM was calculated from M-mode images in the parasternal long-axis view using the Devereux equation, in line with ASE guidelines. To standardize body size, LVM was indexed to BSA to yield LVMI. LVH was defined as LVMI >95 g/m² in females and >115 g/m² in males, based on ASE criteria.

All quantitative values represented the mean of three consecutive cardiac cycles to ensure consistency. Myocardial strain imaging was performed using 2D-STE by two independent cardiologists blinded to clinical and laboratory information. Offline analysis was conducted using specialized software, and data were included only when image quality allowed for complete and accurate speckle tracking throughout the cardiac cycle.

GLS was measured from apical four-, two-, and three-chamber views using 2D grayscale imaging, following EACVI and ASE recommendations. Strain curves were automatically generated and then reviewed for reliability by both observers.

Pulsed-Wave Doppler Assessment

Pulsed-wave (PW) Doppler assessment of mitral inflow dynamics was performed by placing a 2 mm sample volume at the tips of the mitral valve leaflets, visualized from the apical four-chamber view. To ensure measurement accuracy and account for beat-to-beat variability, data were collected over ten consecutive cardiac cycles and averaged.

Several key parameters of diastolic function were evaluated, including the peak early diastolic velocity (E wave), which reflects passive ventricular filling during early diastole, and the peak late diastolic velocity (A wave), which corresponds to atrial contraction-mediated filling. Transmitral deceleration time (E-DT) was also assessed and defined as the interval from the peak of the E wave to its return to baseline, with a normal physiological range of 160 to 260 milliseconds. Additionally, isovolumic relaxation time (IVRT), representing the time between aortic valve closure and mitral valve opening, was measured by positioning the PW Doppler sample volume near the LV outflow tract, adjacent to the anterior mitral leaflet. The normal range for IVRT is between 60 and 100 milliseconds [(9).]

Ethical Approval: MS 9-11-2023

Results

A comparative analysis indicated that cases with a TyG index ≥8.47 had a significantly elevated prevalence of DM than those with TyG <8.47 (80.4% vs. 15.6%, $P<0.001$). Other clinical variables were comparable between the two groups, including age ($P=0.578$), gender ($P=0.782$), BMI ($P=0.665$), clinical presentation ($P=0.158$), SBP ($P=0.651$), DBP ($P=0.509$), HR ($P=0.437$), RR ($P=0.933$), SpO_2 ($P=0.609$), temperature ($P=0.707$), smoking status ($P=0.268$), HTN ($P=0.439$), prior stroke ($P=0.086$), and other comorbidities ($P=0.231$). **(Table 1)**

The TyG index showed significant associations with several echocardiographic

parameters. It had a strong negative correlation with global longitudinal strain (GLS) (r = -0.673, P < 0.001), indicating that higher TyG levels were linked to worse myocardial deformation. It also showed a moderate negative correlation with three-dimensional ejection fraction (STE 3D-EF) (r = -0.445, P < 0.001) and a similar inverse relationship with conventional ejection fraction (EF%) (r = -0.401, P < 0.001). On the other hand, the TyG index was positively correlated with end-diastolic volume (EDV) (r = 0.297, P < 0.001) and end-systolic volume (ESV) (r = 0.319, P < 0.001), suggesting a link to increased cardiac chamber size. **Table 2, Figure 1**

Patients with a TyG index ≥ 8.47 had significantly lower left ventricular function compared to those with a TyG index < 8.47. Their conventional ejection fraction (EF%) was reduced (51 ± 11% vs. 57 ± 8%, P < 0.001), as was their three-dimensional ejection fraction (STE 3D-EF) (45 ± 12% vs. 52 ± 9%, P < 0.001). They also had significantly larger end-diastolic volume (EDV) (5.17 ± 0.82 vs. 4.9 ± 0.68, P = 0.001) and higher end-systolic volume (ESV) (3.9 ± 0.83 vs. 3.57 ± 0.73, P < 0.001). In addition, global longitudinal strain (GLS) was more impaired in this group (-14.6 ± 4.1 vs. -18.3 ± 2.4, P < 0.001). Patients with a TyG index ≥ 8.47 experienced significantly higher rates of in-hospital major adverse cardiovascular events (MACE) compared to those with a TyG index < 8.47 (22.6% vs. 10.2%, P = 0.003). Similarly, the rate of composite MACE during follow-up was also higher in the elevated TyG group (14.9% vs. 7.5%, P = 0.04). Heart failure (HF) was significantly more common in patients with a higher TyG index, both during hospitalization (17.3% vs. 7.5%, P = 0.009) and at follow-up (7.1% vs. 1.4%, P = 0.013). **Table 3, Figure 2**

ROC analysis for predicting in-hospital HF using TyG yielded an AUC of 0.694 (95% CI: 0.600–0.788, P<0.001). A cutoff value >9.07 provided 47.5% sensitivity, 84.73% specificity, 31.1% PPV, and 91.7% NPV. For predicting in-hospital composite MACE, the AUC was 0.699 (95% CI: 0.616–0.783, P<0.001), with the optimal threshold >8.98 yielding 49.06% sensitivity, 82.44% specificity, 36.1% PPV, and 88.9% NPV. **(Table 4)**

Logistic regression confirmed TyG as an independent predictor of in-hospital composite MACE after adjustment for age, gender, BMI, smoking, HTN, DM, and other comorbidities (OR = 12.861, 95% CI: 4.03–41.043, P<0.001). HTN was inversely associated with risk (OR = 0.437, 95% CI: 0.225–0.849, P=0.015). **(Table 5)**

In a multivariate linear regression model for GLS, TyG remained a significant predictor. Each one-unit increase in TyG corresponded to a 7.16-unit decline in GLS (B = -7.161, 95% CI: -8.206 to -6.116, P<0.001). Female gender (B = 1.515, 95% CI: 0.475–2.554, P=0.004), HTN (B = 0.642, 95% CI: 0.012–1.271, P=0.046), and DM (B = 1.11, 95% CI: 0.233–1.987, P=0.013) were also independently associated with GLS. **(Table 6)**

Table 1: General characteristics according to TyG index.

	TyG Index		P
	< 8.47 (n=147)	**≥ 8.47 (n=168)**	
Age (years)	60 ±11	61 ±11	0.578
Gender			
Male	87 (59.2)	102 (60.7)	0.782
Female	60 (40.8)	66 (39.3)	
BMI	30.46 ±5.16	30.2 ±5.28	0.665
Presentation			
Unstable Angina	52 (35.4)	47 (28)	0.158
NSTEMI	95 (64.6)	121 (72)	
Systolic Blood Pressure	131 ±22	130 ±23	0.651
Diastolic Blood Pressure	84 ±14	83 ±14	0.509
Heart Rate	86 ±10	87 ±11	0.437
Heart Rate	17 ±4	17 ±4	0.933
S (%)	96 ±2	96 ±3	0.609
Temperature	36.8 ±0.3	36.8 ±0.3	0.707
Smoking	87 (59.2)	89 (53)	0.268
Hypertension	86 (58.5)	91 (54.2)	0.439
Diabetes mellitus	23 (15.6)	135 (80.4)	**<0.001***
Stroke	2 (1.4)	8 (4.8)	0.086
Other comorbidities	13 (8.8)	22 (13.1)	0.231

Table 2: Correlations between TyG index and other parameters.

	TyG Index	
	r	**P**
Age (years)	0.032	0.571
Body Mass Index	-0.003	0.962
Systolic Blood Pressure	-0.003	0.957
Diastolic Blood Pressure	-0.012	0.835
Heart Rate	0.085	0.132
Respiratory Rate	0.076	0.177
Peripheral Oxygen Saturation (%)	-0.069	0.221
Temperature	0.036	0.529
Hemoglobin	-0.07	0.218
Total Leukocyte Count	0.102	0.071
Platelets	0.031	0.584
Urea	0.107	0.059
Creatinine	0.01	0.855
Alanine Aminotransferase	-0.012	0.831
Aspartate Aminotransferase	0.061	0.279
Ejection Fraction%	-0.401	**<0.001***
End-Diastolic Volume	0.297	**<0.001***
End-Systolic Volume	0.319	**<0.001***
Speckle Tracking Echocardiography 3D Ejection Fraction	-0.445	**<0.001***
Global Longitudinal Strain	-0.673	**<0.001***

TyG Index: Triglyceride-Glucose Index. *P<0.05 is considered statistically significant.

Table 3: Echocardiographic findings and endpoints according to TyG index.

	TyG Index		P
	< 8.47 (n=147)	**≥ 8.47 (n=168)**	
Ejection Fraction%	57 ±8	51 ±11	**<0.001***
Regional Wall Motion Abnormality	112 (76.2)	137 (81.5)	0.244
Mitral Regurgitation			
No	51 (34.7)	48 (28.6)	0.629
Mild	68 (46.3)	81 (48.2)	
Moderate	19 (12.9)	25 (14.9)	
Severe	9 (6.1)	14 (8.3)	
End-Diastolic Volume	4.9 ±0.68	5.17 ±0.82	**0.001***
End-Systolic Volume	3.57 ±0.73	3.9 ±0.83	**<0.001***
STE 3D-EF	52 ±9	45 ±12	**<0.001***
Global Longitudinal Strain	-18.3 ±2.4	-14.6 ±4.1	**<0.001***
Composite MACE - In hospital	15 (10.2)	38 (22.6)	**0.003***
Arrhythmia	1 (0.7)	2 (1.2)	1
Heart Failure	11 (7.5)	29 (17.3)	**0.009***
Cardiogenic shock	0 (0)	5 (3)	0.063
Mortality	0 (0)	4 (2.4)	0.126
Stroke	1 (0.7)	1 (0.6)	1
Hemorrhage	1 (0.7)	0 (0)	0.467
Reinfarction	1 (0.7)	3 (1.8)	0.626
Composite MACE - Follow-up	11 (7.5)	25 (14.9)	**0.04***
Non-fatal Myocardial infarction	8 (5.4)	11 (6.5)	0.681
Stroke	1 (0.7)	3 (1.8)	0.626
Heart Failure	2 (1.4)	12 (7.1)	**0.013***
Mortality	0 (0)	2 (1.2)	0.501

Table 4: ROC analysis for TyG index to predict in-hospital heart failure and predict composite MACE - in hospital.

ROC characteristics	TyG index
AUC	0.694
95% CI	0.600 - 0.788
Best cutoff point	>9.07
Sensitivity	47.50 %
Specificity	84.73 %
PPV	31.1 %
NPV	91.7 %
P	**<0.001***
ROC characteristics	**TyG index**
AUC	0.699
95% CI	0.616 - 0.783
Best cutoff point	>8.98
Sensitivity	49.06 %
Specificity	82.44 %
PPV	36.1 %
NPV	88.9 %
P	**<0.001***

Table 5: Multivariate logistic regression analysis to predict composite MACE - in hospital.

	OR (95% CI)	P
Age (years)	1.025 (0.994 - 1.056)	0.111
Gender	0.969 (0.344 - 2.727)	0.952
Body Mass Index	1.022 (0.959 - 1.088)	0.502
Smoking	0.665 (0.24 - 1.849)	0.435
Hypertension	0.437 (0.225 - 0.849)	**0.015***
Diabetes mellitus	0.486 (0.185 - 1.272)	0.141
Other comorbidities	0.29 (0.077 - 1.087)	0.066
TyG index	12.861 (4.03 - 41.043)	**<0.001***

Table 6: Multivariate linear regression analysis to predict GLS.

	B (95% CI)	P
Age (years)	-0.01 (-0.04 - 0.02)	0.5
Female gender	1.515 (0.475 - 2.554)	**0.004***
Body Mass Index	-0.049 (-0.109 - 0.012)	0.114
Smoking	0.297 (-0.733 - 1.327)	0.571
Hypertension	0.642 (0.012 - 1.271)	**0.046***
Diabetes mellitus	1.11 (0.233 - 1.987)	**0.013***
Other comorbidities	0.665 (-0.338 - 1.667)	0.193
TyG index	-7.161 (-8.206 - -6.116)	**<0.001***

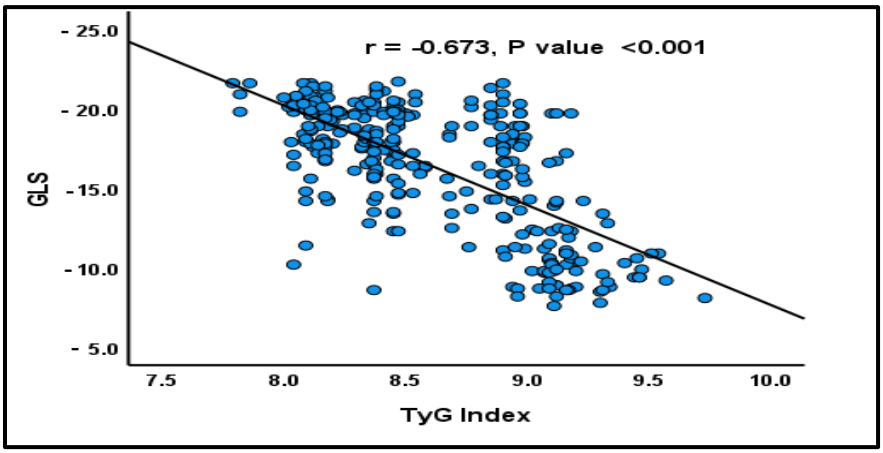


Figure 1: Correlation between TyG index and GLS.

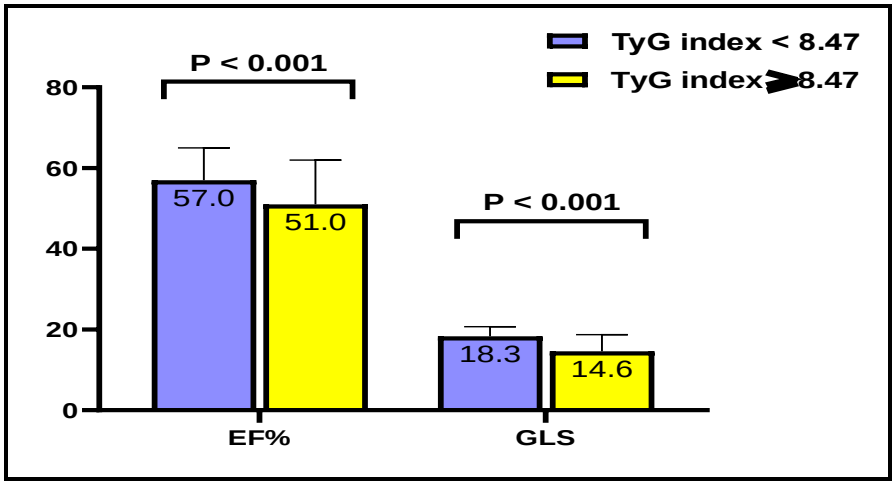
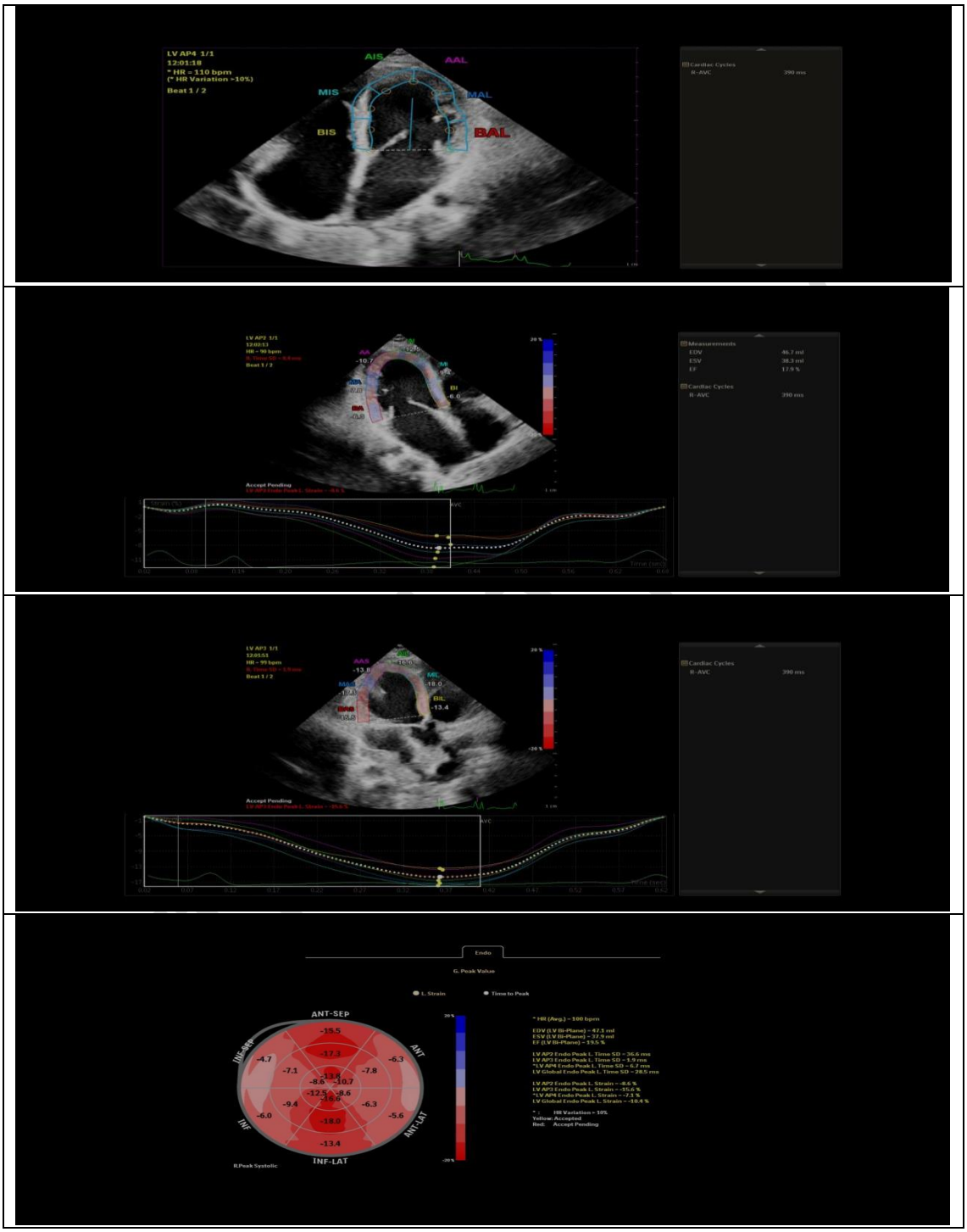
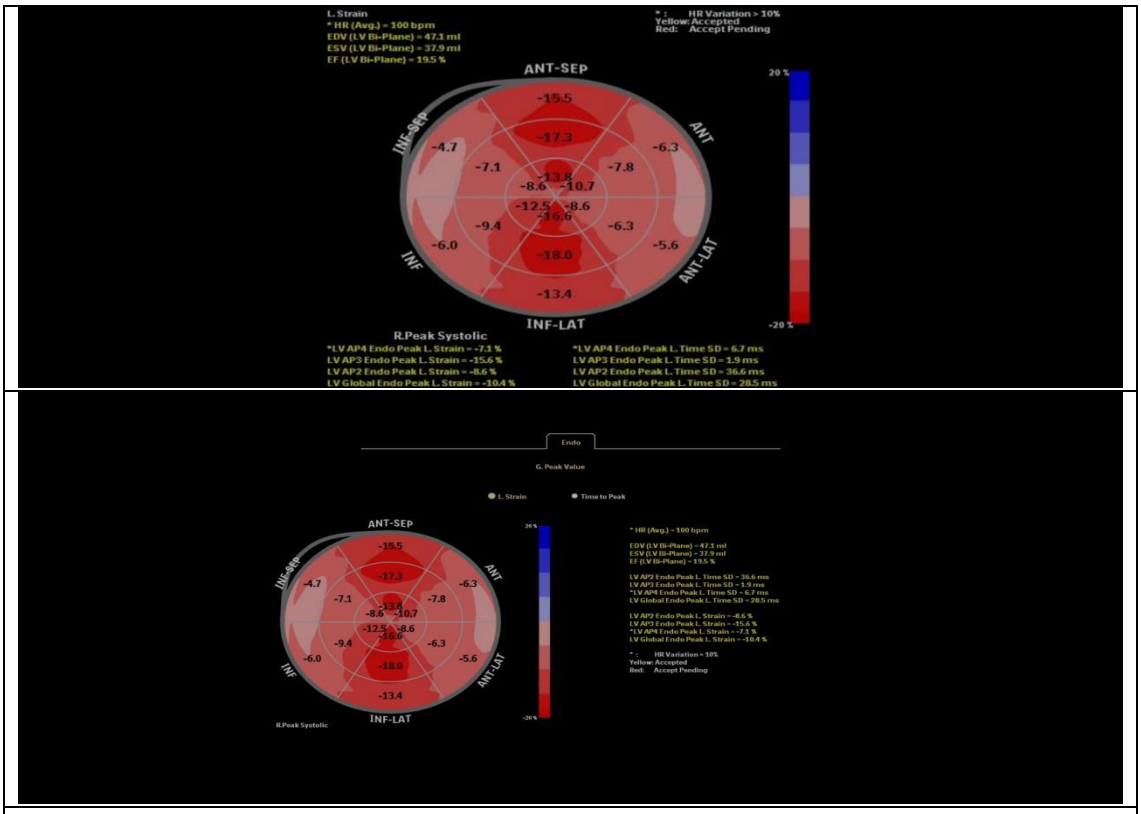


Figure 2: EF and GLS according to TyG index.

Case 1: There was significant reduction in GLS in this patient who had high TyG index. (echo)





Case1: Global Longitudinal Strain (GLS) Estimation

Discussion

In this investigation, age did not differ significantly between the TyG-defined groups (61 ± 11 years, P = 0.571). These findings are comparable to those reported by [10], who observed no significant variation in age across TyG quartiles (53.4 ± 13.8 years, P = 0.692). In contrast, [11] noted that cases with elevated cumulative TyG exposure tended to be older (49.42 ± 11.93 years, P<0.01), indicating a possible age-related component to chronic metabolic stress.

A significantly greater proportion of TyG ≥8.47 cases had DM (80.4%) compared to those with lower indices (15.6%, P<0.001). This aligns with data from [12], who also demonstrated increased DM prevalence among cases with TyG ≥9.5 (P<0.001).

LVEF was markedly reduced in TyG ≥8.47 cases (51 ± 11%) versus the lower group (57 ± 8%, P<0.001), mirroring the results from [13], who reported a negative association between TyG and systolic performance (P<0.001). However, [12] observed a modest increase in LVEF with elevated TyG (coefficient = 0.457%, P = 0.021), while [10] found no significant difference across quartiles (P > 0.05).

EDV was significantly higher in TyG ≥8.47 cases (5.17 ± 0.82 vs. 4.9 ± 0.68, P = 0.001), consistent with [12], who found a positive

association with LVEDV (coefficient = 0.134%, $P<0.001$). In contrast, [14] reported no significant difference in EDV (P = 0.308).

ESV was also greater among cases with elevated TyG (3.9 ± 0.83 vs. 3.57 ± 0.73, $P<0.001$), although [15] and [14] found no significant differences across TyG strata.

GLS was significantly impaired in the TyG ≥8.47 group (-14.6 ± 4.1 vs. -18.3 ± 2.4, $P<0.001$), in agreement with [12], who reported a strong inverse relationship (coefficient = 0.315%, $P<0.001$). Similarly, [10] showed GLS deterioration across TyG quartiles (P = 0.001), and [14] also found worse GLS values in the higher TyG group ($P<0.05$).

Multivariate analysis confirmed TyG as an independent predictor of in-hospital MACE (OR = 12.861, 95% CI: 4.03–41.043, $P<0.001$). HTN was also independently associated, but with a protective effect (OR = 0.437, 95% CI: 0.225–0.849, P = 0.015). These findings aligned with [16], who reported elevated TyG linked to increased CV and all-cause mortality (RR = 2.71 and 2.35, respectively). Similarly, [17] found higher TyG associated with mortality risk in HF and T2DM cases (RR = 4.42, 95% CI: 1.49–13.15). Supporting these results, [18] reported IR (via HOMA-IR) as predictive of CAD-related mortality (HR = 1.69, 95% CI: 1.15–2.48). In contrast, [14] observed no significant association between TyG and CV mortality (HR = 1.10, 95% CI: 0.82–1.47), highlighting notable heterogeneity across investigations (I^2 = 76%).

Several limitations should be acknowledged. The limited sample size may have reduced power to detect differences in subgroups. Lack of long-term follow-up restricted assessment of TyG impact on GLS and outcomes. Lastly, although echocardiographic evaluation was appropriately conducted, the use of additional imaging such as MRI could have enhanced the investigation.

Conclusion

The present findings suggest that elevated TyG index levels in cases with NSTE-ACS are associated with significantly impaired LV function as assessed via speckle-tracking echocardiography. TyG demonstrated independent predictive value for reduced GLS and increased incidence of in-hospital MACE. These results underscore the clinical utility of TyG as a prognostic biomarker, highlighting its potential role in guiding early intervention strategies and risk stratification in this vulnerable population.

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