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Prognostic Value of Serum Triglyceride Glucose Index in Acute Heart Failure

Manjunath S, Pankaj Singhai**, Anand Shenoy****

Introduction

Admission with acute heart failure (AHF) carries high morbidity and in-hospital mortality, with nearly half of the patients rehospitalised within 6 months of discharge[1,2]. Clinical data and targeted treatment remain limited compared to chronic stable heart failure, as both represent distinct pathophysiological entities[3]. So far, established adverse prognostic indicators of AHF include blood urea nitrogen (BUN) levels >43 mg/dL, systolic blood pressure (SBP) <115 mmHg, serum creatinine >2.75mg/dL, and elevated cardiac biomarkers like natriuretic peptides and troponins[2,3].

Insulin resistance (IR) and associated reductions in downstream cardio-metabolic signaling are emerging as critical drivers of myocardial dysfunction and structural remodeling[4,5]. Hyperinsulinaemic-euglycaemic glucose clamp (HIEC) is the clinical gold standard for measuring insulin resistance[6], but its complexity and associated costs make it an impractical option. Alternative surrogate measures of insulin resistance include HOMA-IR, HOMA2-IR, QUICKI, and the serum triglyceride-glucose (TyG) index[7,8]. The TyG index is derived by a simple logarithmic formula: ln (Fasting blood glucose in mg/dL x Fasting serum triglycerides in mg/dL/2).

Emerging data suggest that the TyG index is an effective, low-cost surrogate marker for identifying insulin resistance, comparable or even superior to HOMA-IR in tracking insulin resistance-related conditions in diabetic and even in non-diabetic cohorts[12].

Longitudinal studies have indicated that elevated serum TyG levels are independently associated with an increased incidence of chronic heart failure[9,15]. However, its impact and prognostic influence during acute heart failure remain inadequately explored. This study was therefore designed to observe baseline Serum Triglyceride Glucose index (TyG) levels among consecutive patients admitted with AHF and evaluate its short-term, in-hospital prognostic significance alongside conventional clinical and bio-assay markers.

Material and Methods

Study Setting and Design

This study was designed as a single-center, prospective observational study in the Cardiac Care Unit (CCU) of Manipal Hospitals, Bangalore, India. The study period was 12 months following approval from the institutional scientific and ethics committees.

Study Population

Consecutive adult patients presenting with symptoms requiring admission for AHF were screened.

- **Inclusion Criteria:** Adult patients aged more than 18 years presenting with acute-onset breathlessness accompanied by clinical hallmarks of systemic/ pulmonary congestion (e.g., elevated jugular venous pressure, distended neck veins, severe bilateral pedal oedema, and basal crepitations) matching the structural definition of heart failure outlined by the ACC/AHA guidelines.
- **Exclusion Criteria:** AHF secondary to congenital, rheumatic and valvular heart diseases; advanced malignancy or ongoing chemotherapy regimens; active systemic sepsis, acute respiratory distress syndrome (ARDS), pulmonary embolism; liver cirrhosis; pancreatitis; pregnancy; and end-stage renal disease.

Data Collection and Laboratory Analysis

Comprehensive data of clinical presentation, detailed background history, demographic details, and physical examination was documented in a pre-defined proforma. Peripheral venous blood samples were drawn after an overnight fasting window (>10 hours) during the first morning of admission.

Laboratory and other investigation parameters were collected from the institutional electronic database.

**Senior Resident, Department of General Medicine, JSS Medical College, Mysuru - 571 134, Karnataka, **Senior Consultant and Head, Department of Internal Medicine, ***Senior Consultant, Department of Cardiology, Manipal Hospital, Old Airport Road, Bengaluru - 560 017, Karnataka.*

Corresponding Author: Dr Manjunath S, Senior Resident, Department of General Medicine, JSS Medical College, Mysuru - 571 134, Karnataka. Tel: 9738408496, E-mail: sripathymanjunathan561996@gmail.com

Study Outcomes

The primary outcomes tracked were: in-hospital mortality or clinical stability resulting in successful discharge. Secondary clinical endpoints included the specific requirement for and total duration of advanced organ supports, including intravenous inotropes, vasopressors, non-invasive ventilation (NIV), invasive mechanical ventilation (IMV), and intra-aortic balloon pump (IABP) counterpulsation.

Statistical Analysis

Sample size was calculated using Cochran's equation based on an expected historical in-hospital mortality rate of 13% for patients with AHF, yielding a target sample size of 148. Following exclusions and attrition of the sample, a final cohort of 140 patients was analysed. Continuous variables were expressed as Mean ± Standard Deviation (SD) or Median, while categorical variables were summarised as frequencies and relative percentages. Statistical significance was defined at a threshold of alpha = 0.05. Categorical parameters were assessed using Chi-Square and Fisher's Exact tests. Differences in non-parametric data were analysed using the Kruskal-Wallis test. Spearman-rank correlation matrices were constructed to evaluate variables against survival endpoints and explore inter-variable relationships. Receiver Operating Characteristic (ROC) curve analysis evaluated the area under the curve (AUC) and identified optimal biomarker cut-offs via Youden's index. Odds Ratios (OR) with corresponding 95% confidence intervals were derived to evaluate mortality risks. All statistics were computed using standard packages.

Results

Demographic and Clinical Baseline Profiles

A final cohort of 140 consecutive patients was successfully evaluated and consisted of 90 males (64%) and 50 females (36%). The overall mean age of the study group was 67 ± 11.4 years (range: 36 - 90 years), as shown in Fig. 1. Non-survivors were significantly older than survivors (73.6 ± 8.54 years *versus* 66.1 ± 11.45 years; p = 0.009). Patient gender demonstrated no significant prognostic relationship with in-hospital mortality (p = 1.0).

Cardio-metabolic comorbidities were highly prevalent across the cohort: 101 patients (72.1%) had type 2 diabetes mellitus with 33 (32.6%) using insulin therapy, 93 (66.4%) had systemic hypertension, 58 (41.4%) had established CAD, 51 (36.4%) had a history of chronic heart failure, and 23 (16.4%) had hypothyroidism. No statistically significant differences were observed in comorbidity distribution, insulin usage, or daily insulin doses between survivors and non-survivors.

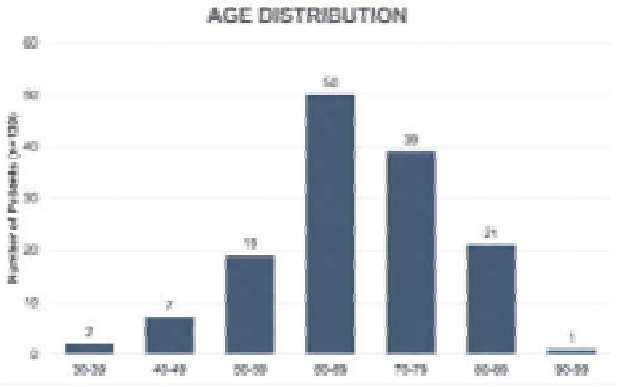


Fig. 1: *Age-wise distribution of the study population.*

Physical Examination, Admission Findings, and Echocardiography

Severe dyspnoea was highly prevalent, with 77 patients (55.0%) classified as NYHA functional class 4 at presentation. Physical examination revealed bilateral basal crackles in 133 patients (95.0%) and peripheral pitting oedema in 89 patients (63.6%). Haemodynamically, 36 patients (25.7%) presented with an SBP <115 mmHg. Mean qSOFA scores were significantly higher in non-survivors than in survivors (1.22 ± 0.43 *versus* 0.93 ± 0.52; p = 0.023).

On electrocardiography at admission, acute ischaemic ST-segment changes were identified in 36 cases (25.7%), predicting significantly increased odds of mortality (OR: 3.5, p = 0.019). Echocardiographic evaluation categorised 89 patients (63.6%) as HFrEF, 26 (18.6%) as HFmrEF, and 25 (17.9%) as HFpEF. The overall mean LVEF was 37% ± 10.4%, which did not differ significantly between survivors and non-survivors (p = 0.844). Left ventricular diastolic dysfunction was present in all 140 patients, with severe grade 3 LVDD identified in 52 individuals (37.1%).

Metabolic Profile Distributions and Biomarker Assay Performance

Difference in mean values of different parameters is shown in Table I and the Box and whisker's plot in Figs. 2 and 3. The mean TyG index for the entire study population was 9.12 ± 0.71. When applying a reference cut-off for insulin resistance (TyG of 8.8 based on previous study), 88 patients (62.9%) were categorised as insulin resistant. In this group, 12 deaths were recorded compared to 6 deaths in the low-TyG cohort, representing a statistically non-significant difference in in-hospital mortality rates (OR: 1.21, p = 0.799). Mean TyG values in subpopulations are shown in Table II. Segmented evaluations across diabetic, non-diabetic, active acute coronary syndrome, and chronic ischemic heart disease cohorts confirmed that a TyG threshold of 8.8 did not significantly alter diagnostic predictive accuracy.

Table I: Mean values of parameters (Survivors *versus* Non-Survivors).

Variables	Died (Mean ± SD)	Survived (Mean ± SD)	Total Pool (Mean ± SD)	p-Value (Two-Tailed)
Ejection Fraction (EF%)	37.2 ± 10.3	36.98 ± 10.4	37.0 ± 10.4	0.844
PASP (mmHg)	44.2 ± 13.8	42.7 ± 13.6	42.9 ± 13.6	0.660
Serum Creatinine (mg/dL)	1.64 ± 0.75	1.40 ± 0.89	1.43 ± 0.88	0.084
Blood Urea Nitrogen (mg/dL)	25.8 ± 14.8	22.3 ± 20.9	22.8 ± 20.2	0.188
Total Cholesterol (mg/dL)	143.6 ± 36.7	144.7 ± 41.7	144.6 ± 40.9	0.869
LDL Cholesterol (mg/dL)	82.7 ± 23.3	87.6 ± 36.6	87.0 ± 35.1	0.968
HDL Cholesterol (mg/dL)	39.5 ± 15.4	37.5 ± 9.8	37.8 ± 10.7	0.784
Fasting Blood Sugar (mg/dL)	192.6 ± 87.0	167.5 ± 83.3	170.7 ± 83.9	0.186
Serum Triglycerides (mg/dL)	132.0 ± 66.0	130.5 ± 62.1	130.7 ± 62.4	0.965
Fasting TyG Index	9.20 ± 0.80	9.10 ± 0.70	9.10 ± 0.70	0.413
HbA1c (%)	8.1±2.1	8.0 ± 2.0	8.0 ± 2.0	0.955
NT-proBNP (pg/mL)	11730 ± 9979	9641 ± 12852	9910 ± 12508	0.117
hs-Troponin I (pg/mL)	5284 ± 10926	3083 ± 15134	3366 ± 14647	**0.009**
AST (U/L)	90.4 ± 132.6	51.3 ± 164.6	56.29 ± 161.0	**0.004**
ALT (U/L)	90.7 ± 160.5	47.8 ± 148.2	53.3 ± 149.9	**0.008**
Serum Uric Acid (mg/dL)	6.8 ± 3.1	5.7 ± 2.3	5.87 ± 2.45	0.281

Note: Evaluated using Kruskal-Wallis non-parametric ANOVA variations.

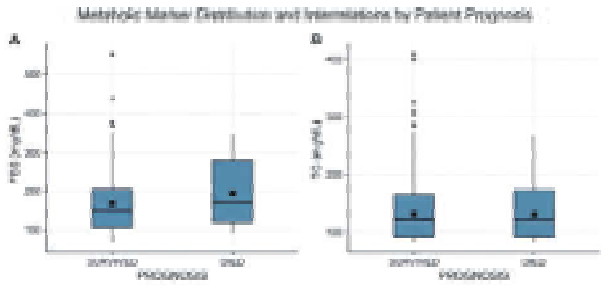


Fig. 2: *Box and whiskers plot showing mean FBS among survivors versus non-survivors.*

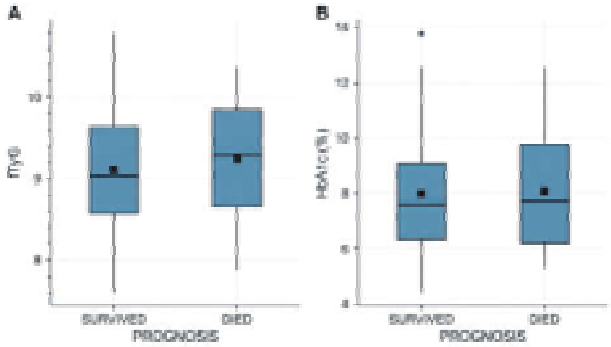


Fig. 3: *Box and whiskers plot showing mean TyG and HbA1c among survivors versus non-survivors.*

Aetiological Diagnoses and Mechanical Support Requirements

The dominant aetiology driving AHF was ischaemic heart disease (IHD), accounting for 113 cases (80.7%), followed by dilated cardiomyopathy (DCM; n = 10, 7.1%). Acute coronary syndromes (ACS) were present in 62 presentations (44.3%), consisting of 24 ST-elevation myocardial infarctions (STEMI), 36 non-ST-elevation myocardial infarctions (NSTEMI), and 2 unstable angina presentations. The presence of an active ACS event significantly increased overall in-hospital mortality (p = 0.046). Concurrently, 77 patients required emergency percutaneous coronary interventions (PCI) during hospital stay.

Duration of hospitalisation and requirements of organ support is shown in Table III. Advanced organ support was common: 83 patients (59.3%) required active intravenous inotropic infusions, 36 (25.7%) required vasopressors, 46 (32.9%) required non-invasive positive pressure ventilation (NIV), and 25 (17.9%) required invasive mechanical ventilation. No statistically significant differences were observed in the total duration or relative incidence of mechanical ventilation, vasopressor use, or inotropic support when partitioning the cohort using the baseline TyG index threshold of 8.8 or the ROC-derived threshold of 9.28.

Table II: Mean TyG values in subpopulations

Sub-Population	Died (Mean ± SD)	Survived (Mean ± SD)	Total (Mean ± SD)	p-Value
TyG in Diabetics	9.42 ± 0.71	9.27 ± 0.70	9.28 ± 0.70	0.438
TyG in Non-Diabetics	8.79 ± 0.82	8.69 ± 0.54	8.70 ± 0.57	0.850
TyG in ACS Patients	9.42 ± 0.83	9.20 ± 0.63	9.24 ± 0.67	0.269
TyG in Non-ACS Patients	8.89 ± 0.57	9.03 ± 0.75	9.02 ± 0.74	0.722
TyG in Ischemic Heart Disease	9.31 ± 0.78	9.16 ± 0.69	9.18 ± 0.70	0.390

Note: Evaluated using Kruskal-Wallis Test.

Table III: Duration of hospitalisation and requirement for organ support.

Component	Minimum (Days)	Maximum (Days)	Mean (Days)	Median (Days)	Standard Deviation
Total Hospitalisation	1	22	5.0	4	2.8
Intravenous Inotrope	0	8	1.2	1	1.4
Vasopressor Support	0	7	0.5	0	1.1
Invasive Mechanical Ventilation (IMV)	0	5	0.4	0	1.0
Non-Invasive Ventilation (NIV)	0	8	0.6	0	1.1
Intra-Aortic Balloon Pump (IABP)	0	4	0.1	0	0.5

Correlation Modeling and Discriminative ROC Curve Analysis

Table IV shows Spearman Rank-Order correlation of key parameters against in-hospital mortality. Only baseline qSOFA scores (rho = 0.193, p = 0.022), hs-Trop-I (rho = 0.221, p = 0.009), AST (rho = 0.245, p = 0.004), and ALT (rho = 0.226, p = 0.007) statistically correlated with in-hospital mortality. The TyG index did not correlate with clinical outcomes (rho = 0.069, p = 0.415).

Table IV: Spearman Rank-order correlation of key parameters against in-hospital mortality.

Parameter	Spearman's Rho (rho)	p-Value
TyG Index	0.069	0.415
NT-proBNP	0.133	0.188
qSOFA Score	0.193	0.022
hs-Trop-I	0.221	0.009
LVEF	0.017	0.845
AST	0.245	0.004
ALT	0.226	0.007
Serum Creatinine	0.147	0.084

Table V shows correlation between various study parameters. Within the study parameters, TyG index correlated with HbA1c (rho = 0.529, p <0.001), but showed no correlation with active markers of myocardial wall stretch (NT-proBNP) or baseline haemodynamic variables (LVEF and LVDD).

Mortality prediction by various parameters was analysed using the Receiver Operating Characteristic curve as shown in Fig. 4. Table VI shows ROC Curve Analysis for Mortality for various parameters. Table VII shows in-hospital mortality odds ratios for ROC-derived cut-off targets. Using the ROC-optimised TyG cut-off of 9.28, the resulting mortality risk calculation (OR: 1.86, p = 0.300) remained non-significant even when evaluated among subgroups. Conversely, risk ratios remained highly significant for elevated transaminases above their respective ROC cut-offs (ALT OR:

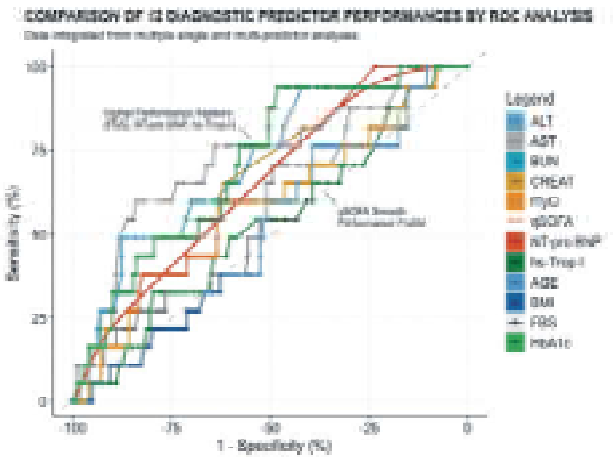


Fig. 4: *Composite ROC analysis comparing different biomarkers and clinical parameters.*

Table V: Comprehensive multi-variable matrix of inter-parameter correlation profiles.

Parameter	Metric	TyG	NT-proBNP	LVEF	AST	ALT	hs-Trop-I
NT-proBNP	rho	-0.072	–	–	–	–	–
	p	0.399	–	–	–	–	–
LVEF	rho	0.121	-0.303	–	–	–	–
	p	0.155	**<0.001**	–	–	–	–
AST	rho	-0.041	0.174	-0.169	–	–	–
	p	0.634	0.039	0.046	–	–	–
ALT	rho	-0.008	0.071	-0.227	0.832	–	–
	p	0.924	0.406	**0.007**	**<0.001**	–	–
hs-Troponin I	rho	0.120	0.330	-0.096	0.190	0.203	–
	p	0.158	**<0.001**	0.261	**0.025**	**0.016**	–
qSOFA Score	rho	-0.002	0.220	-0.095	0.171	0.107	0.165
	p	0.979	**0.009**	0.262	**0.044**	0.208	0.052
Serum Creatinine	rho	0.109	0.374	0.004	0.071	0.028	0.201
	p	0.199	**<0.001**	0.960	0.405	0.739	**0.017**
BUN	rho	0.056	0.401	-0.031	0.101	0.001	0.169
	p	0.509	**<0.001**	0.714	0.234	0.989	**0.045**
LVDD Grade	rho	-0.149	0.118	-0.372	0.014	0.019	-0.082
	p	0.078	0.166	**<0.001**	0.871	0.820	0.333
BMI	rho	-0.137	-0.096	0.057	0.035	-0.028	-0.028
	p	0.107	0.260	0.507	0.677	0.744	0.744
HbA1c	rho	0.529	-0.054	0.012	-0.062	-0.066	0.116
	p	**<0.001**	0.525	0.885	0.468	0.442	0.171
Serum Uric Acid	rho	-0.093	0.093	0.003	-0.070	-0.086	-0.071
	p	0.276	0.274	0.972	0.410	0.313	0.403

Table VI: Receiver operating characteristic curve analysis for mortality evaluation.

Tested Parameter	AUC	Youden Cut-off	Sensitivity (%)	Specificity (%)
AST	0.711	44	61.1	82.8
ALT	0.695	53	50.0	86.9
hs-Troponin I	0.691	74 pg/mL	94.4	43.4
Age	0.691	64 Years	94.4	34.4
qSOFA Score	0.629	2	100.0	17.2
Serum Creatinine	0.626	2.04 mg/dL	33.3	90.2
NT-proBNP	0.615	6440 pg/mL	72.2	55.7
BUN	0.596	29 mg/dL	38.9	82.8
Fasting Blood Sugar	0.563	101 mg/dL	100.0	21.3
TyG Index	0.560	9.28	61.1	59.8
LVDD Grade	0.543	2	100.0	10.7
LVEF (%)	0.514	41%	50.0	68.0
HbA1c (%)	0.504	9.7%	33.3	77.9
BMI (kg/m^2)	0.486	24.5	77.8	35.3

Table VII: In-hospital mortality odds ratios for ROC-derived cut-off targets.

Parameter	Died (n)	Survived (n)	Total	p-Value
TyG Index				0.300 (OR = 1.86)
<9.28	8	73	81	
≥9.28	10	49	59	
NT-proBNP (pg/mL)				0.080 (OR = 2.50)
<6440	6	68	74	
≥6440	12	54	66	
hs-Troponin I (pg/mL)				**0.009** (OR = 6.35)
<74	2	54	56	

≥74	16	68	84	
AST (U/L)				**<0.001** (OR = 6.37)
<44	8	102	110	
≥44	10	20	30	
ALT (U/L)				**<0.001** (OR = 6.60)
<53	9	106	115	
≥53	9	16	25	
Serum Creatinine (mg/dL)				**0.045** (OR = 3.50)
<2.04	13	110	123	
≥2.04	5	12	17	
BUN (mg/dL)				**0.020** (OR = 3.70)
<29	11	104	115	
≥29	7	18	25	
qSOFA Score				0.130 (OR = 2.60)
<2	14	110	124	
≥2	4	12	16	
LVEF (%)				0.590 (OR = 1.46)
<41%	11	85	96	
≥41%	7	37	44	

6.6, p <0.001; AST OR: 6.37, p <0.001) and hs-Trop-I levels exceeding 74 pg/mL (OR: 6.35, p = 0.009). Table VIII shows subgroup mortality signatures for ROC-Optimised TyG Cut-Off. Table IX shows organ support incidences predicted by ROC-Derived TyG Index cut-off. Requirements of organ support remained statistically insignificant even when ROC derived TyG cut-off was used for analysis.

Table VIII: Subgroup mortality signatures tracked by ROC-optimised TyG (9.28) cut-off.

Patient Subpopulation	Died (n)	Survived (n)	Total Subset Size	p-Value
Diabetic	13	88	101	0.560 (OR = 1.60)
– TyG <9.28	5	44	49	
– TyG ≥9.28	8	44	52	
Non-Diabetic	5	34	39	0.200 (OR = 3.90)
– TyG <9.28	3	29	32	
– TyG ≥9.28	2	5	7	
ACS	12	50	62	0.200 (OR = 2.30)
– TyG <9.28	4	27	31	
– TyG ≥9.28	8	23	31	
Non-ACS	6	72	84	1.000 (OR = 0.88)
– TyG <9.28	4	46	50	
– TyG ≥9.28	2	26	28	
Ischaemic Aetiology (IHD) Pool	16	97	113	0.400 (OR = 1.68)
– TyG <9.28	7	55	62	
– TyG ≥9.28	9	42	51	

Note: Evaluated via Fisher's Exact Test.

Table IX: Organ support incidences predicted by ROC-derived TyG index threshold (9.28).

Critical Care Intervention	Support Needed (Yes)	Support Not Needed (No)	p-Value
Intravenous Inotropes	83	57	-0.220 (OR = 0.60)
– TyG <9.28	52	29	
– TyG ≥9.28	31	28	
Vasopressors	36	104	0.300 (OR = 1.50)
– TyG <9.28	18	63	

Discussion

This prospective observational study sought to characterise the presence of metabolic insulin resistance in patients presenting with AHF using the TyG index, comparing its performance with conventional critical care biomarkers. Insulin resistance is known to accelerate coronary atherogenesis, promote macrovascular dysfunction, and induce subclinical myocardial fibrosis[13]. However, our data indicate that these metabolic indicators do not retain significant independent prognostic power during AHF.

Our study cohort had 72.1% prevalence of type 2 diabetes mellitus and an 80.7% prevalence of underlying ischaemic heart disease. These baseline rates are higher than those documented in national heart failure registers, such as the Trivandrum Heart Failure Registry (THFR; DM 55%, IHD 72%) or the National Heart Failure Registry (NHFR; DM 42.5%)[14,1]. This concentration of metabolic risk factors likely stems from our setting as a tertiary referral care facility and our deliberate exclusion of structural congenital or valvular rheumatic heart conditions.

Applying the baseline TyG index threshold of 8.8[12], nearly two-thirds (62.9%) of our AHF cohort showed insulin resistance whereas more than that had diabetes which is a known insulin resistant state. However, this categorisation failed to show a statistically significant correlation with in-hospital mortality or the length and intensity of active mechanical or pharmacological organ supports. Even when refining the index via ROC analysis to an optimized cut-off of 9.28, the resulting trend toward higher mortality remained statistically non-significant (OR: 1.86, p = 0.300).

This lack of acute predictive power contrasts with long-term follow-up registers but aligns with observations made

by other critical care registries. For instance, an evaluation of TyG performance in multi-systemic non-surgical intensive care cohorts found that while elevated TyG indices predicted adverse outcomes in isolated acute coronary syndromes, they showed an inverse relationship in decompensated heart failure presentations[10]. A key confounding factor in our patient cohort was the widespread use of baseline statin therapy (53%), which can alter serum triglyceride fractions independent of underlying peripheral insulin sensitivity. Furthermore, the short-term clinical course of AHF is dominated by immediate hemodynamic shifts, neurohormonal overactivation, and acute multi-organ congestion, which can mask the slower, chronic cellular effects of insulin signaling pathways.

In contrast to metabolic indices, biomarkers reflecting acute congestion, systemic hypoperfusion, and localised cellular stress demonstrated strong prognostic utility. The admission qSOFA score significantly correlated with higher in-hospital mortality, supporting the concept that acute heart failure shares clinical similarities with systemic critical illnesses like severe sepsis or ARDS regarding explosive onset and organ support requirements.

Notably, hepatic aminotransferases (AST and ALT) emerged as the strongest independent predictors of in-hospital mortality within our cohort[11]. Elevated AST (>44 U/L) and ALT (>53 U/L) levels were associated with a 6.3-fold and 6.6-fold increase in in-hospital mortality risk, respectively. This clinical finding highlights the severe impact of acute hepatic congestion and secondary systemic hypoperfusion (shock liver) as major drivers of early mortality in acute heart failure.

Conclusion

Although nearly two-thirds of patients presenting with AHF displayed features of systemic insulin resistance, the TyG index did not serve as a statistically reliable prognostic marker for in-hospital mortality or immediate organ support requirements during the acute phase of decompensation. Traditional markers of systemic strain, acute myocardial injury, and secondary hepatic congestion-specifically qSOFA, hs-Trop-I, AST, and ALT remained superior for early risk stratification. Further multi-center investigations are needed to determine whether surrogate markers of insulin resistance provide better utility for long-term tracking after initial haemodynamic stabilisation.

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