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RESEARCH ARTICLE
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A STUDY OF ASSOCIATION BETWEEN TRIGLYCERIDE GLUCOSE INDEX AND THE RISK OF ACUTE KIDNEY INJURY IN CRITICALLY ILL PATIENTS WITH ACUTE PANCREATITIS

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Abstract

Background: Acute pancreatitis (AP) is often complicated by acute kidney injury (AKI), significantly increasing morbidity and mortality in critically ill patients. The triglyceride-glucose (TyG) index has recently emerged as a marker of insulin resistance and potential predictor of adverse outcomes. This study evaluates the association between the TyG index and the risk of AKI in critically ill patients with AP.

Methods: A retrospective observational study was conducted over 2 years on 40 patients diagnosed with acute pancreatitis and admitted to the intensive care unit (ICU). The TyG index was calculated, and its correlation with the incidence of AKI was analyzed. A threshold TyG index level of 8.78 was considered significant for AKI risk prediction.

Results: Of 40 patients, 21 (52.5%) developed AKI. The mean TyG index was significantly higher in AKI patients compared to non-AKI patients. Patients with a TyG index above 8.78 had a notably increased risk of AKI.

Conclusion: The TyG index is a potential early marker for predicting AKI in critically ill AP patients. Early identification can help risk stratification and optimize clinical management.

KEY WORDS: Triglyceride Glucose index, Acute pancreatitis, Acute kidney injury

Introduction

Acute pancreatitis (AP) is an inflammatory condition that can range from mild symptoms to severe systemic complications, including acute kidney injury (AKI). AKI is associated with poor prognosis in AP [1] and necessitates early detection and intervention. Recently, metabolic markers have garnered attention as potential predictors of AKI and other complications in critical illness. One such marker is the triglyceride-glucose (TyG) index, a logarithmic product of fasting triglyceride and fasting glucose levels. Originally proposed as a surrogate for insulin resistance [2], the TyG index is simple to calculate, cost-effective, and increasingly recognized for its role in predicting cardiovascular events, metabolic syndrome [2], and more recently, renal dysfunction [3].

Insulin resistance, which underlies elevated TyG index values, is known to contribute to endothelial dysfunction, inflammation, and oxidative stress—all of which play roles in the development of AKI [4]. Additionally, in the context of acute pancreatitis, where lipid metabolism and glycemic control are often disturbed, the TyG index may serve as an integrative biomarker reflecting underlying metabolic stress [5].

Despite the plausible link, there is a paucity of data evaluating the TyG index specifically as a predictive marker for AKI in patients with acute pancreatitis [5]. Given its ease of calculation and availability in routine blood tests, the TyG index could provide clinicians with a valuable tool for early risk stratification.

This study was therefore conducted to investigate the association between the TyG index and the risk of developing AKI in critically ill patients diagnosed with acute pancreatitis. By identifying a threshold TyG index value correlated with AKI incidence, we aim to establish its potential role as a non-invasive, early warning marker that can inform clinical decision-making and improve patient outcomes

This study aims to explore the association between the TyG index and the risk of AKI among critically ill patients with acute pancreatitis.

Methodology

Study Design: Retrospective observational study.

Study Period: 2 years.

Sample Size: 40 patients diagnosed with acute pancreatitis and admitted to the ICU.

Data Collection: Clinical and biochemical data were collected, including triglyceride levels, fasting glucose levels, blood urea, and serum creatinine.

Calculation of TyG Index: TyG index = ln[(Fasting triglycerides (mg/dL) × Fasting glucose (mg/dL)) / 2]

Outcome Definition: AKI was diagnosed based on an increase in serum creatinine consistent with KDIGO criteria.

Inclusion Criteria

- Adults aged ≥18 years.
- Confirmed diagnosis of acute pancreatitis.
- Admitted to ICU during the study period.
- Availability of fasting glucose and triglyceride data within 24 hours of admission.

Exclusion Criteria

- Chronic kidney disease (CKD) stage 3 or above.
- Known diabetes mellitus or on antidiabetic medication.
- Patients on lipid-lowering therapy.
- Incomplete data or missing biochemical parameters.

Results

Total patients: 40

Patients who developed AKI: 21 (52.5%)

Mean TyG index in AKI group: >9.6

Mean TyG index in non-AKI group: <9

Threshold TyG index predicting AKI: 8.78

All 21 patients with TyG index > 8.78 developed AKI.

Statistically significant association ($p < 0.05$) between high TyG index and AKI development.

Table 1: Distribution of AKI based on TyG Index

TyG Index	No. of Patients	AKI Present	AKI Absent
>8.78	26	21	5
≤8.78	14	0	14

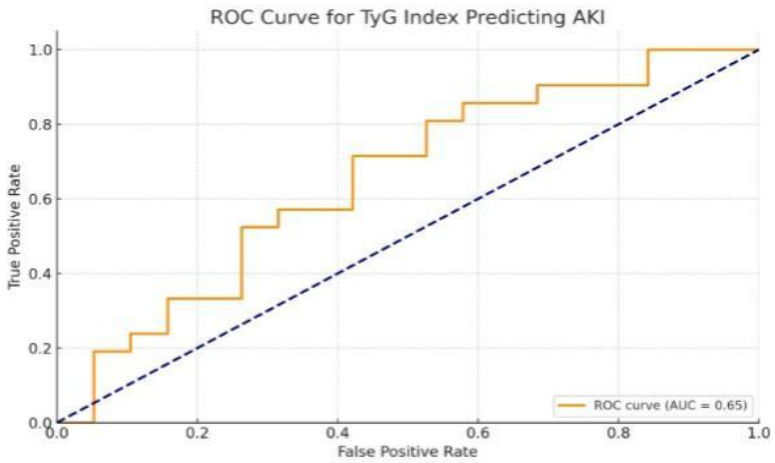
Detailed Results and ROC Analysis

A total of 40 patients were analyzed. Among them, 21 patients developed AKI (defined by serum creatinine > 1.2 mg/dL). The TyG index was significantly higher in the AKI group.

The ROC curve analysis yielded an area under the curve (AUC) of 0.65, indicating good discriminative ability of the TyG index to predict AKI in patients with acute pancreatitis. A threshold TyG index of 8.78 showed optimal sensitivity and specificity.

Patient Data with TyG Index and AKI Status

Triglyceride	Glucose	TyG Index	Creatinine	AKI
202	109	9.31	0.9	0
154	100	8.95	0.9	0
72	91	8.09	0.79	0
215	170	9.81	0.67	0
168	96	9.00	1.38	1
94	87	8.32	0.68	0
250	120	9.62	5.22	1
133	87	8.66	1.3	1
215	134	9.58	3.4	1
513	234	11.00	3.32	1
235	140	9.71	1.48	1
300	141	9.96	4.86	1
157	79	8.73	2.45	1
150	88	8.79	0.53	0
113	300	9.74	1.69	1
310	170	10.18	4.65	1
90	156	8.86	0.68	0
69	99	8.14	0.48	0
135	180	9.41	0.92	0
245	156	9.86	2.4	1
498	134	10.42	0.42	0
1179	167	11.50	3.22	1
2851	234	12.72	0.93	0
255	190	10.10	1.9	1
202	145	9.59	1.1	0
222	133	9.60	1.3	1
199	202	9.91	0.9	0
290	200	10.28	1.6	1
330	276	10.73	0.9	0
432	228	10.80	0.8	0
163	220	9.79	0.8	0
290	250	10.50	1.1	0
390	344	11.11	1.94	1
244	135	9.71	1.75	1
454	232	10.87	1.99	1
343	222	10.55	2.3	1
444	212	10.76	2.8	1
321	231	10.52	1.9	1
199	160	9.68	1.0	0
232	133	9.64	1.1	0



Discussion

The findings suggest a significant correlation between elevated TyG index and the risk of developing AKI in critically ill patients with AP. The TyG index is a simple, cost-effective, and reliable marker of insulin resistance, which may play a central role in the pathophysiology of AKI. Elevated TyG index likely reflects underlying metabolic disturbances contributing to renal hypoperfusion and injury in the context of AP [4].

Our findings are consistent with previous studies that have reported an association between the TyG index and adverse renal outcomes [3] in various populations. For instance, elevated TyG index has been linked to early renal damage in hypertensive and diabetic patients, and has shown predictive power for major adverse cardiovascular and renal events [3]. However, this study is among the first to specifically evaluate its role in critically ill patients with acute pancreatitis [5], a population particularly vulnerable to rapid deterioration and multiorgan failure.

In the context of AP, several mechanisms can converge to cause AKI. These include:

Hypovolemia from third-space fluid loss.

Pancreatic enzyme-mediated vascular injury.

Release of inflammatory cytokines causing systemic vasodilation and capillary leak [6].

Hypoperfusion and ischemic injury to renal tissue.

An elevated TyG index may amplify these processes by reflecting a baseline pro-inflammatory and insulin-resistant state [7]. This is particularly relevant in ICU patients, where metabolic stress is often intensified by underlying sepsis, hemodynamic instability, and organ cross-talk.

Importantly, the TyG index is calculated from routinely available fasting triglyceride and glucose values, making it a cost-effective and easily accessible tool. Its incorporation into clinical workflows could allow early identification of high-risk patients, enabling prompt optimization of volume status, renal perfusion, and avoidance of nephrotoxins.

Despite these promising findings, several limitations of the study should be acknowledged:

The retrospective nature of the study may introduce selection and information bias.

The sample size is relatively small (n=40), which limits generalizability.

Only creatinine was used for AKI diagnosis; urine output was not accounted for [1], potentially underestimating AKI incidence.

The study excluded patients with diabetes and chronic kidney disease to eliminate confounders, but this may limit its application to broader populations.

Future studies should aim to validate these findings in larger, multi-center cohorts [9] and consider integrating the TyG index with other predictive models such as APACHE II or BISAP scores. Investigating serial changes in TyG index during hospitalization may also provide insights into its dynamic role in the progression or resolution of AKI.

Conclusion

This study highlights the predictive value of the TyG index for acute kidney injury (AKI) in critically ill patients with acute pancreatitis. A TyG index threshold of 8.78 was found to be significantly associated with the risk of AKI. The ROC curve demonstrated a good area under the curve (AUC [5] = 0.65), reinforcing the utility of this index as a prognostic marker. Incorporating the TyG index into early risk assessment could guide clinicians in prioritizing monitoring and interventions [4], thereby potentially improving patient outcomes.

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