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Original Article

Evaluation of Triglyceride Glucose Index and Systemic Inflammatory Biomarkers in Non-Diabetic Patients with Adhesive Capsulitis: A Cross-Sectional Clinical Study

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ABSTRACT

Objective: Adhesive capsulitis (AC) is a painful shoulder condition characterized by progressive restriction of active and passive range of motion. This study aimed to compare the triglyceride–glucose (TyG) index and haematology-derived inflammatory indices between patients with AC and healthy controls and to evaluate their associations with the presence of AC.

Methods: In this study, the demographic data, clinical findings and laboratory results of non-diabetic patients with AC were evaluated. Fasting plasma glucose (FPG), lipid profile [triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and total cholesterol], C-reactive protein (CRP), vitamins D and B12, and complete blood count, obtained within the preceding 3 months, were recorded. The TyG index, neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII) were calculated.

Results: Seventy-one participants were included (38 controls and 33 AC). The AC group was older and had higher body mass index (both p≤0.001). FPG (p=0.010), LDL (p=0.001), total cholesterol (p=0.049), and TyG index (p=0.020) were higher in AC, whereas NLR, PLR, SII, CRP, HDL, vitamin D, and B12 were comparable between groups (all p>0.05). In adjusted models, age was a consistent predictor of AC [adjusted odds ratio (aOR) 1.094, 95% confidence interval (CI) 1.036–1.155; p=0.001], and LDL was independently associated with AC (aOR 1.019, 95% CI 1.001–1.037; p=0.044).

Conclusion: Non-diabetic AC was associated with metabolic dysregulation, while haematology-derived inflammatory indices remained comparable to controls. Beyond symptomatic management, clinicians should consider screening patients with AC for metabolic dysregulation, as this may aid risk stratification and care.

Keywords: Adhesive capsulitis, blood glucose level, bloog triglycerides level, inflammation

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INTRODUCTION

Adhesive capsulitis (AC) is a painful shoulder disorder characterised by restricted movement.[1] It affects approximately 2–5% of the general population and occurs more frequently among individuals with metabolic and endocrine comorbidities.[2] While AC has traditionally been viewed as a local mechanical and inflammatory disorder, growing evidence suggests that chronic low-grade inflammation and metabolic dysfunction may also contribute to its pathogenesis.[3] The risk of AC is particularly increased in individuals with diabetes mellitus (DM).[4] More broadly, metabolic and endocrine abnormalities have been linked to AC; inflammatory and other metabolic profiles may influence disease development and clinical course.[5]

Studies evaluating glucose-related parameters in AC have yielded inconsistent findings, with some reporting associations with symptom severity and others showing no differences in blood glucose compared with controls.[6-10] In contrast, adverse lipid profiles—particularly higher total cholesterol and triglyceride levels—have been associated

with AC.[11,12] Thyroid dysfunction may also contribute to pro-inflammatory and fibrotic pathways relevant to AC.[13] Overall, these observations support further investigation of immunometabolic markers in AC.

The triglyceride–glucose (TyG) index is a practical surrogate marker of insulin resistance, calculated from fasting triglyceride and glucose values, and is readily available in clinical practice.[14,15] It has also been used as an indicator of metabolic syndrome.[16] Haematology-derived inflammatory indices, obtained from routine complete blood count parameters, are increasingly used as accessible markers of systemic inflammation. The neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) are widely used indices,[17] and the systemic immune-inflammation index (SII), combining neutrophil, lymphocyte and platelet parameters, has also been proposed as a useful marker of systemic inflammation.[18]

It is increasingly emphasised that AC may involve systemic inflammation and metabolic disturbances beyond localised pathology.[2,3] Given that insulin resistance has been linked to increased pain and restricted movement in AC,[2] further studies focusing on the immunometabolic profile are warranted. Accordingly, we compared the TyG index and haematology-based inflammatory indices between non-diabetic patients with AC and age- and sex-matched healthy controls and evaluated their associations with the presence of AC. We hypothesised that non-diabetic patients with AC would have a higher TyG index and a more pro-inflammatory haematological profile than controls, and that higher TyG index and inflammatory markers would be independently associated with AC.

METHODS

This was an observational, cross-sectional, case-control study involving prospective recruitment. It compared non-diabetic patients with AC to age- and sex-matched healthy controls. Approval was obtained from the University of Health Sciences Türkiye, Prof. Dr. Cemil Taşcıoğlu Ethics Committee (approval number: 63, date: 24.02.2025), and written informed consent was obtained from all participants. The principles of the Declaration of Helsinki were adhered to throughout the research process.

Participants

Patients who had attended the Physical Medicine and Rehabilitation outpatient clinics for at least three months between March and November 2025, who presented with complaints of shoulder pain and limited movement, who were clinically diagnosed with AC, and who met the inclusion/exclusion criteria were invited to participate in the study.

Inclusion criteria were: age between 18–70 years; shoulder pain lasting at least 3 months with limited shoulder movement; magnetic resonance imaging of the affected shoulder performed within the last three months; complete blood count, C-reactive protein (CRP), fasting plasma glucose (FPG), and fasting lipid panel [triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL), total cholesterol]; and 25(OH)D and vitamin B12 levels measured within the last three months. Exclusion criteria were: a history of shoulder surgery or shoulder trauma; diagnosis of DM or pre-DM; use of triglyceride-lowering medication (statins, fibrates, omega-3, thiazolidinediones); thyroid disease (thyroidectomy, autoimmune thyroiditis, or thyroid dysfunction); pregnancy; acute inflammation; history of malignancy; alcohol use; chronic kidney disease or active infectious disease; viral hepatitis; and liver cirrhosis.

A total of 192 AC patients were evaluated during the study period. Of these, 159 were excluded from the study because they met exclusion criteria or had missing data (shoulder trauma: 15; DM: 83; use of lipid-lowering medication: 20; lack of current laboratory tests: 35; chronic kidney disease: 3; thyroid dysfunction: 17). The control group was recruited sequentially from the same outpatient clinics and consisted of individuals without shoulder-related symptoms who did not meet the exclusion criteria. Demographic, clinical, and laboratory data were recorded for all cases included in the study.

Calculation of Indices

The TyG index was calculated using the following formula:

TyG index = natural log of
((fasting triglycerides [mg/dL] × FPG [mg/dL]) / 2)

The following complete blood count parameters were used for the assessment of systemic inflammation:

NLR = neutrophil count/lymphocyte count

PLR = platelet count/lymphocyte count

SII = platelet count × neutrophil count/lymphocyte count

Statistical Analysis

Descriptive statistics were expressed as the mean ± standard deviation for continuous variables, and as the number of observations (n) for categorical variables. The normality of the data distribution was tested using the Shapiro–Wilk test. Continuous variables that were normally distributed were compared using the independent-samples t-test, whereas non-normally distributed variables were compared using the Mann–Whitney U test. Categorical variables were compared using the chi-squared test or Fisher's exact test.

To evaluate factors associated with the presence of AC, binary logistic regression analyses were conducted, and the results were reported as adjusted odds ratios (aORs) with 95% confidence intervals (CIs). Given the modest sample size, the number of covariates entered into each multivariable model was restricted, and covariates were selected a priori based on clinical relevance to reduce the risk of overfitting. A series of hierarchical models was constructed: Model 0 included age and BMI; Model 1 included LDL; Model 2 included FPG; and Model 3 included the TyG index. To minimise potential multicollinearity, collinearity diagnostics were assessed (e.g., tolerance and variance inflation factors). B ecause the TyG index is derived from FPG and triglycerides, closely related metabolic variables were not entered simultaneously the same model when appropriate. Model fit was evaluated using the Hosmer–Lemeshow goodness-of-fit test. A two-sided p value <0.05 was considered statistically significant. Analyses were performed using IBM SPSS Statistics for Windows, version 23.0 (IBM Corp., Armonk, NY, USA).

RESULTS

A total of 71 participants were included in the study (38 healthy, 33 AC). The mean age and BMI of the patient group were higher than those of the control group (p<0.001 and p=0.001, respectively). The healthy group had a higher level of education, whereas a higher proportion of the patient group were retired or housewives (p=0.001 and p=0.047, respectively). No significant differences in gender or smoking habits were found between the groups (p=0.967 and p=0.908, respectively). As the variables relating to hand dominance and affected side were only present in the patient group, no intergroup comparisons were made for these variables (see Table 1).

Compared with the healthy group, the patient group had higher FPG, LDL, and total cholesterol levels (p=0.010, p=0.001, and p=0.049, respectively). Additionally, the TyG index was significantly higher in patients (p=0.020). However, no significant differences were observed between the groups with respect to neutrophil, lymphocyte, platelet, NLR, PLR, SII, HDL, vitamin D, vitamin B12, and CRP values (all p>0.05). Triglyceride levels tended to be higher in the patient group, although the difference did not reach statistical significance (p=0.071; see Table 2).

Multivariate logistic regression analyses were performed to identify factors that predict the presence of AC. In Model 0, which included age and body mass index (BMI), age was an independent predictor of illness (aOR =1.094; 95% CI 1.036–1.155; p=0.001), whereas BMI was not significant (p=0.111). In Model 1, which included LDL, age remained significant (aOR =1.085; 95% CI 1.025–1.148; p=0.005), and LDL level was independently associated with the presence of AC (aOR =1.019; 95% CI 1.001–1.037; p=0.044). In Model 2, when FPG was added to LDL, FPG did not show an independent association (p=0.705), but LDL retained its significance (p=0.042). In Model 3, when the TyG index was added alongside LDL, the TyG index did not demonstrate an independent association (p=0.299), while the association of LDL declined to borderline significance (aOR =1.017; 95% CI 0.999–1.036; p=0.064). Age remained a consistent independent predictor of the presence of AC in all models (p≤0.013) (Table 3).

DISCUSSION

This study examined whether the TyG index, lipid profile, and haematology-derived inflammatory indices differ between non-diabetic patients with AC and healthy controls, and whether these markers are associated with AC. Overall, our findings indicate that non-diabetic AC have a more adverse metabolic profile, whereas haematology-derived inflammatory indices were comparable to controls. In regression analyses, age remained associated with AC across adjusted models. Although LDL showed a significant association with AC in the age-adjusted analysis, this association was no longer statistically significant after adjustment for the TyG index, indicating that the LDL–AC association may overlap with insulin resistance-related metabolic factors rather than representing a stable independent risk factor.

DM, thyroid disorders, and dyslipidaemia have been reported as risk factors for AC.[19,20] However, because dyslipidaemia commonly coexists with diabetes and thyroid disease, its independent contribution is difficult to isolate.[19,21] Evidence is also mixed: Zhang et al.[21] (including Mendelian randomisation analyses) found no significant association between circulating lipid levels and AC, whereas case-control data suggest that higher lipid levels may be related to AC, although triglycerides may not differ consistently from controls.[7,22] In our cohort, LDL and total cholesterol were higher in patients with AC, whereas triglyceride levels were comparable between groups. Moreover, the LDL–AC association was evident only in age-adjusted analyses and was attenuated after accounting for the TyG index, underscoring the importance of insulin resistance–related metabolic context when interpreting lipid associations. Differences across studies may reflect design- and cohort-related factors, including medication use and residual confounding.

Hyperglycaemia may promote connective tissue changes via glycation and may be linked to heightened oxidative and inflammatory signalling.[23,24] Several studies have associated higher FPG with AC risk,[25,26] although much of this evidence derives from diabetic cohorts. In non-diabetic populations, higher FPG within the normoglycaemic range has also been

Table 1. Comparison of demographic and clinical characteristics between the healthy and patient groups						
		Healthy (38)	**Patient (33)**	**p value**	**ES**	**CI**
Age (years) (mean ± SD)		42.08±12.43	56.73±11.11	<0.001[M]	0.520	0.33–0.67
Sex (female), n		24	21	0.967[χ]	0.005	-0.228–0.238
BMI (kg/m²) (mean ± SD)		25.42±3.42	28.33±4.20	0.001[M]	0.410	0.19–0.59
Education	**Primary and middle school, n**	14	25	0.001[χ]	0.39	0.17–0.57
	High school, n	24	8			
Occupation	**Retired/household, n**	12	18	0.047[χ]	0.29	-
	Office work, n	20	8			
	Manual labor, n	6	7			
Hand dominans (right), n		-	32	-	-	-
Affected side	**Dominant, n**	-	21	-	-	-
	Non-dominant, n	-	12			
Smoking status (current), n		12	10	0.908	0.014	-0.23–0.23
VAS activity (mean ± SD)		-	5.73±2.44	-	-	-
VAS rest (mean ± SD)		-	1.39±1.46	-	-	-
VAS night (mean ± SD)		-	4.91±2.71	-	-	-

Data are presented as mean ± SD for continuous variables and as n for categorical variables [M]Mann–Whitney U test; [χ]chi-square test were used

BMI: Body mass index, VAS: Visual analog scale, n: Number, CI: Confidence interval, ES: Effect size, SD: Standard deviation Variables related to hand dominance and the affected side were available only in the patient group; therefore. no between-group statistical comparisons were performed for these variables (—)

Table 2. Comparison of laboratory parameters between the healthy and patient groups					
	Healthy (38)	**Patient (33)**	**p value**	**ES**	**95% CI**
Neutrophil (Neu) (×10⁹/L)	4.16±1.09	4.39±1.30	0.408[t]	0.196	-0.658–0.267
Lymphocyte (Lym) (×10⁹/L)	2.39±0.80	2.42±0.98	0.845[M]	0.023	-0.211–0.255
Platelets (Plt) (×10⁹/L)	254.84±75.94	278.09±85.88	0.156[M]	0.168	-0.068–0.386
Neu/Lym (NLR)	1.91±0.74	2.16±1.75	0.854[M]	0.022	-0.213–0.254
Plt/Lym (PLR)	117.05±50.08	125.17±42.70	0.203[M]	0.150	-0.370–0.080
SII (SIII)	488.36±245.09	559.18±336.19	0.170[M]	0.163	-0.073–0.382
Fasting plasma glucose (mg/dL)	90.55±11.38	98.82±16.66	0.010[M]	0.305	0.078–0.503
Triglycerides (mg/dL)	110.37±48.60	138.76±64.69	0.071[M]	0.214	-0.020–0.426
HDL (mg/dL)	57.71±17.14	53.45±13.15	0.250[t]	0.273	-0.191–0.736
LDL (mg/dL)	111.61±27.88	146.67±47.90	**0.001**[t]	0.901	-1.383–(-0.413)
Total cholesterol (mg/dL)	188.42±34.71	211.67±57.79	**0.049**[t]	0.491	-0.957–(-0.021)
Vitamin D (µg/L)	19.65±7.44	19.94±9.82	0.822[M]	0.027	-0.208–0.258
Vitamin B12 (ng/L)	346.89±109.31	298.76±138.21	0.109[t]	0.385	-0.085–0.852
TyG index	8.31±0.79	8.72±0.52	0.020[M]	0.276	0.046–0.479
CRP (mg/L)	2.31±2.14	3.32±2.74	0.071[M]	0.214	-0.020–0.426

Data are presented as mean ± SD. p values were obtained using Mann–Whitney U test ([m]) or independent-samples t-test ([t]), as appropriate. ES indicates ES (r for Mann–Whitney U; |Hedges' g| for t-test). 95% CI denotes the 95% CI of the ES

NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, SII: Systemic immune-inflammation index, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TyG: Triglyceride–glucose index, CRP: C-reactive protein, SD: Standard deviation, ES: Effect size, CI: Confidence interval

associated with shoulder pathology and AC.[27,28] Consistent with these reports, FPG levels were higher in our non-diabetic AC group than in controls.

The TyG index, a practical surrogate marker of insulin resistance derived from fasting glucose and triglyceride levels, is feasible for routine clinical use and epidemiological research.[14] To our knowledge, this is the first study to assess TyG in patients with AC, and we observed higher TyG values in the AC group than in controls. Limited prior evidence supports a link between insulin resistance and AC severity, with higher homeostasis model assessment of insulin resistance reported to be associated with greater pain, disability, and reduced shoulder mobility.[10] In our regression analyses, the lack of an independent TyG association may reflect limited statistical power and/or shared variance with correlated metabolic measures, underscoring the need for cautious, model-aware interpretation. Overall, these findings suggest that insulin resistance–related metabolic alterations may be relevant in AC even when diabetes is excluded

Inflammation is implicated in AC pathogenesis, with cytokine-mediated processes contributing to fibrosis and abnormal tissue repair.[29-31] Haematology-derived indices such as NLR, PLR and SII are widely used as accessible, non-specific markers of systemic inflammation,[17] but data in AC are limited. Prior studies have reported differences in these indices across AC subtypes or related shoulder conditions.[32,33] In our study, haematology-based inflammatory markers did not differ between patients with AC and controls, suggesting that any inflammatory signal in AC may not be reliably detected by peripheral blood indices. Given the measures' non-specificity and susceptibility to residual variability, limited power may also have contributed to null findings. Future studies incorporating cytokine-based biomarkers and/or tissue-level assessments may better characterise the inflammatory profile of AC.

This study has limitations. First, FPG was based on a single measurement, and repeated assessments would better account for within-person variability. Second, the single-centre design and modest sample size limited the number of covariates that could be included in multivariable models, thereby reducing the precision of estimates. Therefore, we used a streamlined, clinically informed modelling strategy and interpreted results as model-specific. Finally, laboratory values obtained within the preceding three months may have introduced variability due to timing differences relative to clinical evaluation.

Table 3. Factors predicting the presence of adhesive capsulitis: multivariable logistic regression models

Variable	Model 0: age + BMI	Model 1: age + BMI + LDL	Model 2: age + BMI + LDL + FPG	Model 3: age + BMI + LDL + TyG
Age (years) aOR (95% CI) p	1.094 (1.036–1.155) **0.001**	1.085 (1.025–1.148) **0.005**	1.080 (1.016–1.147) **0.013**	1.079 (1.020–1.141) **0.008**
BMI (kg/m²) aOR (95% CI) p	1.129 (0.973–1.310) 0.111	1.101 (0.947–1.281) 0.211	1.100 (0.945–1.280) 0.217	1.091 (0.936–1.271) 0.264
LDL (mg/dL) aOR (95% CI) p	—	1.019 (1.001–1.037) **0.044**	1.019 (1.001–1.037) **0.042**	1.017 (0.999–1.036) 0.064
FPG (mg/dL) aOR (95% CI) p	—	—	1.008 (0.967–1.051) 0.705	—
TyG index aOR (95% CI) p	—	—	—	1.836 (0.583–5.782) 0.299

Dependent variable: patient =1, healthy =0. aOR: Adjusted odds ratio, CI: Confidence interval. BMI: Body mass index, LDL: Low-density lipoprotein, FPG: Fasting plasma glucose, TyG: Triglyceride–glucose index.

CONCLUSION

These findings support consideration of a broader immunometabolic perspective in AC. In clinical practice, evaluating and addressing metabolic risk, particularly insulin resistance–related profiles, may be relevant even in non-diabetic patients and could inform holistic management beyond symptomatic treatment. Further research is needed larger longitudinal studies incorporating direct measures of insulin resistance and more specific inflammatory biomarkers to confirm these associations and clarify the underlying mechanisms.

Ethics

Ethics Committee Approval: Approval was obtained from the Univeristy of Health Sciences Türkiye, Prof. Dr. Cemil Taşcıoğlu Ethics Committee (approval number: 63, date: 24.02.2025).

Informed Consent: Written informed consent was obtained from all participants.

Footnotes

Authorship Contributions

Surgical and Medical Practices: D.E.Z., Z.E.E., S.A., E.D., M.Z., Concept: D.E.Z., S.A., Ö.K., M.Z., Design: D.E.Z., S.A., Ö.K., Data Collection or Processing: D.E.Z., Z.E.E., S.A., E.D., M.Z., Analysis or Interpretation: D.E.Z., Z.E.E., S.A., E.D., Ö.K., M.Z., Literature Search: D.E.Z., Z.E.E., S.A., E.D., M.Z., Writing: D.E.Z., E.D., Ö.K., M.Z.

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