Association between lipid metabolism markers and gastric cancer stage and grade: A focus on ApoB

Running Title: Lipid metabolism markers and gastric cancer stage

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Abstract

Background: Gastric cancer (GC), particularly adenocarcinoma, remains a global health burden with high mortality due to late-stage diagnosis and limited reliable biomarkers for disease monitoring. Lipid metabolism plays a crucial role in tumor biology, and serum lipid-related markers, including apolipoproteins, have been suggested as potential non-invasive indicators of tumor progression. This study aimed to evaluate the association between serum lipid profiles and tumor stage and histological grade in patients with gastric adenocarcinoma.

Methods: Fifty patients diagnosed with gastric adenocarcinoma were enrolled. Serum levels of total cholesterol, triglycerides, HDL-C, LDL-C, ApoA1, and ApoB were measured. Patients were categorized into early (stages I–II) and advanced (stages III–IV) tumor stages, as well as into moderately differentiated versus poorly differentiated grades. The Shapiro-Wilk test was used to assess data normality. Parametric and non-parametric tests were applied accordingly. A p-value < 0.05 was considered statistically significant.

Results: ApoB was the only parameter showing a significant association with tumor stage. Patients with advanced-stage GC had significantly lower mean ApoB levels compared to those in early stages (52.4 ± 2.6 vs. 63.4 ± 5.2 mg/dl, $p = 0.042$). No statistically significant differences were observed in ApoA1, HDL-C, total cholesterol, triglycerides, LDL-C, or VLDL for either tumor stage or histological grade.

Conclusion: ApoB levels appear to decline with advancing tumor stage in gastric adenocarcinoma, suggesting a potential role as a marker to evaluate disease burden. While no association was found with tumor grade, further validation in larger prospective studies incorporating metabolic and inflammatory covariates is warranted.

Keywords: Adenocarcinoma, Stomach, Apolipoprotein B, Lipid Metabolism, Neoplasm Staging, Neoplasm Grading

Introduction

Gastric cancer (GC), particularly gastric adenocarcinoma, is a major global health concern and a leading cause of cancer-related deaths, with over 1 million new cases and approximately 769,000 deaths reported in 2020 (1). Traditional prognostic factors, such as tumor stage and histological grade, often require invasive procedures and may not fully reflect the systemic metabolic changes associated with cancer progression. Therefore, there is a growing interest in identifying noninvasive and cost-effective biomarkers that can aid in the early diagnosis, prognosis, and monitoring of gastric cancer. Dysregulation of lipid profiles has been implicated in various malignancies, suggesting that lipid-related parameters may be potential biomarkers for tumor biology, including proliferation, invasion, metastasis, and therapy resistance (2). High-density lipoprotein cholesterol (HDL-C) is known for its cardioprotective effects, mainly through reverse cholesterol transport, anti-inflammatory, and antioxidant properties (3). Interestingly, recent studies have highlighted an inverse relationship between HDL-C levels and GC progression, supporting its role as a negative biomarker (4, 5). A meta-analysis of 156 studies with over 85,000 cancer patients indicated that higher levels of HDL-C, total cholesterol (TC), and ApoA-I were associated with improved overall survival (OS) and disease-free survival (DFS) (6). These findings suggest that dyslipidemia may be associated with the development and progression of GC. The role of low-density lipoprotein cholesterol (LDL-C) in cancer is complex and multifaceted. Elevated LDL-C levels have been associated with increased oxidative stress and inflammation, both of which are beneficial for tumorigenesis. In the context of GC, previous studies reported that higher LDL-C levels were significantly associated with increased risk of GC (5). Additionally, a meta-analytical study conducted by Xu and colleagues demonstrated a weak negative correlation between LDL-C concentrations and GC susceptibility; however, this association was not statistically significant (7). Apolipoproteins, which constitute the protein moiety of lipoproteins, play critical roles in lipid transport and metabolism. Apolipoprotein A-I (ApoA-I), a major component of HDL-C, has anti-inflammatory and antioxidant properties. Lower levels of ApoA-I are implicated in elevating the risk of GC and its progression, suggesting its potential as a protective biomarker (5). Furthermore, Lee and colleagues identified serum ApoA-I as a potential diagnostic and prognostic biomarker for GC and demonstrated that decreased levels were associated with adverse outcomes (8). Conversely, apolipoprotein B (ApoB), the major LDL-C protein, is attributed to the promotion of atherogenesis and possibly tumorigenesis. Elevated ApoB levels may indicate increased lipid availability to support rapid tumor cell proliferation. Notably, the ApoB/ApoA-I ratio has emerged as a potential prognostic marker in GC. A retrospective analysis indicated that a higher preoperative ApoB/ApoA-I ratio predicted poor OS, whereas lower ApoB and ApoB/ApoA-I levels were significantly associated with peritoneal metastasis in advanced GC (9, 10). Despite these insights, the clinical utility of lipid parameters as biomarkers in GC remains unknown due to study heterogeneity, confounding factors, and inconsistent results. Comprehensive analysis of lipid profiles across different stages and grades is necessary to clarify their potential as biomarkers for diagnosis, prognosis, and therapeutic targeting.

Methods

This study was conducted on a group of 50 patients with gastric adenocarcinoma. Plasma samples were obtained from these patients through the Cancer Institute of Tehran University of Medical Sciences. All participants provided written informed consent before entering the study. Ethical approval of the study protocol was obtained from the Ethics Committee of Golestan University of Medical Sciences (Ethics Code: IR.GOUMS.REC.1401.078). Patients were anonymous, and data

confidentiality was strictly maintained throughout the study. To ensure sufficient statistical power and adequate sample size in each subgroup, patients were stratified into broader groups based on tumor stage and histological grade. Tumor staging was determined according to the 8th edition of the American Joint Committee on Cancer (AJCC) tumor, node, and metastasis (TNM) classification system. Tumor stage was divided into two main categories: early stage (including stages 0, I, and II) and advanced stage (including stages III and IV). Histological grading was evaluated by a pathologist based on the degree of glandular differentiation and classified as well-, moderately-, poorly, and undifferentiated adenocarcinoma, according to the World Health Organization (WHO) classification. Similarly, histological grade was stratified into two groups: well- and moderately differentiated tumors, and poorly differentiated tumors. This approach facilitated more reliable statistical comparisons between clinically relevant categories. Serum lipid parameters, including total cholesterol, triglycerides, HDL-C, and LDL-C, were measured using diagnostic reagent kits provided by Delta Darman Audit, while ApoA1 and ApoB levels were determined using Gesan company reagent kits. All assays were performed with the aid of fully automated analyzers, specifically Mindray BS-480 and Biotechnica BT-3500 systems. These procedures were strictly in accordance with the manufacturers' protocols regarding reagent preparation, instrument calibration, quality control, and sample handling to ensure accuracy, reliability, and reproducibility of the measurements. The VLDL level was calculated based on TG/5. The patients had been fasted, and TG levels were less than 400 mg/dl. Due to marked hemolysis, one patient's plasma sample was excluded from the analysis to ensure the reliability of the results.

Statistical analysis

Data were analyzed using SPSS version 22 (IBM Corp, USA). The distribution of lipid and apolipoprotein variables was evaluated using the Shapiro–Wilk test to determine the appropriateness of parametric versus non-parametric statistical analyses. Normality was assessed separately within tumor stage groups (early: stages 0–II; advanced: stages III–IV) and histological grade groups (well/moderately differentiated vs poorly/undifferentiated). Details of the normality tests are provided in Supplementary Table 1. After normality testing, parametric analysis using the independent samples t-test was employed for normally distributed variables (ApoB, ApoA1, HDL, total cholesterol). In contrast, non-normally distributed variables (TG, LDL, VLDL) were analyzed using the nonparametric Mann-Whitney U test. The threshold for statistical significance was set at a p-value less than 0.05.

Result

Comparison by tumor stage

Among the evaluated parameters, Apolipoprotein B (ApoB) emerged as the sole parameter demonstrating statistically significant variation between the early-stage and advanced-stage of disease. Patients with early-stage gastric adenocarcinoma had significantly higher mean ApoB levels (63.4 ± 5.2 mg/dL) compared to those with advanced-stage disease (52.4 ± 2.6 mg/dL), with a p-value of 0.042. Other apolipoprotein-related variables, including ApoA1 and ApoB/ApoA1 ratio, revealed no significant differences between stage groups ($p = 0.409$ and $p = 0.185$, respectively). Similarly, HDL cholesterol showed no meaningful variation between early and advanced stages ($p = 0.419$), and although total cholesterol approached significance ($p = 0.052$), it did not meet the conventional threshold for statistical significance. Regarding variables with non-normal distributions (i.e., TG, LDL, and VLDL), the Mann–Whitney U test was applied. None of these showed statistically significant differences between stage groups. For instance, the median

TG levels were slightly higher in early stages (mean rank = 28.65) compared to advanced stages (mean rank = 23.06), but the difference was not significant (p = 0.193). Likewise, LDL (p = 0.125) and VLDL (p = 0.817) did not show significant variation across stage groups. The median and IQR (Interquartile range) for each of these parameters are: median: 142 and IQR: 111-186 for TG in early stages, median: 135 and IQR: 105-174 for TG in advanced stages, median:76 and IQR: 58-100 for LDL in early stages, median: 66 and IQR: 51-96 for LDL in advanced stages, median: 29 and IQR: 22-37 for VLDL in early stages, median: 28 and 20-33 for VLDL in advanced stages.

Comparison by histological grade

Analysis by histological grade revealed no statistically significant differences in any of the lipid or apolipoprotein variables between well/moderately differentiated and poorly/undifferentiated tumors. ApoB levels were slightly higher in the poorly/undifferentiated group (59.2 ± 4.4 mg/dL) than in the well/moderately differentiated group (53.8 ± 3.02 mg/dL), however, this difference failed to achieve statistical significance (p = 0.310). Similarly, ApoA1 (p = 0.374), total cholesterol (p = 0.317), HDL (p = 0.369), and the ApoB/ApoA1 ratio (p = 0.666) showed no significant differences between grade groups (Figure 1). No statistically significant differences were observed between the two study groups in terms of TG (p = 0.154), LDL (p = 0.331), and VLDL (p = 0.066) levels, all of which were not normally distributed.

In summary, ApoB was the only marker with a significant association with tumor stage, showing lower levels in advanced-stage disease. No lipid or apolipoprotein variable was significantly associated with tumor grade. These findings may indicate a potential stage-related decline in ApoB levels, warranting further investigation. Full statistical details, including descriptive data, test results, and p-values, are provided in Tables 1 and 2.

Discussion

In this cross-sectional study, we evaluated the associations between serum lipid profiles, including apolipoproteins, and clinicopathological features in patients with gastric adenocarcinoma. Our most salient finding was that ApoB levels were significantly lower in patients with advanced-stage disease compared to early-stage cases. This stage-specific decline occurred in the absence of significant correlations between ApoB and tumor grade, suggesting a progressive metabolic alteration tied to tumor burden rather than cellular differentiation. ApoB is the primary structural protein of LDL and VLDL particles, governing lipid transport and delivery to peripheral tissues. In advanced cancer, elevated metabolic demand and systemic inflammation may suppress hepatic lipoprotein production or enhance peripheral uptake of ApoB-containing particles. This mechanism aligns with findings from Zhang et al., which reported lower ApoB in GC patients with peritoneal metastases, illustrating dynamic lipid mobilization during metastatic progression (10). Similarly, Di Zhang et al. observed widespread lipid depletion-including LDL and triglyceride-glucose (TyG) index-in advanced-stage GC patients, reinforcing the notion of metabolic exhaustion in later disease stages (11). Also, Contemporary research indicates that the TyG index serves as a valid surrogate biomarker for insulin resistance and demonstrates significant associations with elevated neoplastic risk, particularly regarding malignancies of the gastrointestinal tract (12). Future studies should consider incorporating such metabolic parameters to further unravel the systemic alterations in cancer progression.

Several serum markers-including total cholesterol, HDL, LDL, ApoA1, and the ApoB/ApoA1 ratio-did not significantly differ across stage or grade. Additionally, non-parametric analyses showed no meaningful differences in TG and VLDL levels between groups. It reflects the relative stability of lipid indices after tumor growth. Also, some factors such as sample size, population

properties, nutrition status, and tumor heterogeneity could be involved. These results suggest that while ApoB is sensitive to tumor progression, most lipid markers remain relatively stable once cancer develops. These observations are supported by Li et al., who found no significant associations between standard lipid markers and tumor stage across a large GC cohort but noted stage-related HDL and LDL variations when comparing cases and controls (13). Moreover, the meta-analysis by Peng et al. reported elevated HDL, total cholesterol, and ApoA1 as favorable prognostic indicators in cancer broadly, without significant roles for ApoB in survival analysis (6). Such heterogeneity across studies likely reflects differences in design, patient demographics, nutritional factors, and tumor heterogeneity. The specific decline in ApoB with advancing disease stage may reflect increased lipoprotein utilization by tumor cells, systemic inflammation, or the impact of cancer-related cachexia. Tumor cells often reprogram lipid metabolism for membrane synthesis and signaling molecule generation-phenomena supported by multi-omics studies like the lipid metabolism-associated genes (LMAG) signature development by others (14). Additionally, dyslipidemia–gastric cancer links may intersect through H. pylori–induced inflammation or statin-related metabolic modulation (15, 16). Clinically, ApoB could be considered a non-invasive serum factor for assessing tumor burden. However, the absence of significant associations with grade suggests limited utility as a marker for cellular differentiation. Integrating ApoB measurements with other lipid and inflammatory indices (e.g., HDL, TyG, hs-CRP) may enhance prognostic models. A key strength of this study is the rigorous statistical approach, utilizing both parametric and non-parametric tests driven by the Shapiro–Wilk normality assessment. The inclusion of multiple lipid-related indicators offers a comprehensive metabolic profile.

Limitations and future research

These findings suggest that ApoB may be a potential stage-related marker in gastric cancer, nevertheless, this study presents several methodological limitations, including an insufficiently powered sample size and the lack of statistical adjustment for potential confounding variables, notably nutritional parameters, liver function markers, and concurrent medical conditions. The cross-sectional design precludes longitudinal assessment of lipid dynamics over treatment courses. Also, assessment of some metabolic parameters, like insulin resistance, could be useful in the evaluation of data. For example, the TyG index, a marker of insulin resistance, is directly correlated with the risk, as well as progression of cancer through induction of visceral adiposity dysfunction, systemic inflammation, and epigenetic modification. So, assessment of insulin resistance and TyG could clarify their importance and correlation with lipid profile and different stages and grades of gastric cancer.

To substantiate our findings, prospective multicenter studies with larger cohorts and longitudinal serum sampling alongside nutritional and inflammatory markers are needed. Investigations should explore mechanisms underlying ApoB decline—such as hepatic synthesis versus tumor uptake and assess whether dynamic changes in ApoB correlate with treatment response or survival. Additionally, integrating lipidomics and transcriptomics may further unravel the metabolic pathways underpinning GC progression (17).

Conclusion

In conclusion, this study demonstrated that among several lipid profile and apolipoprotein markers, only Apolipoprotein B (ApoB) levels were significantly associated with tumor stage in patients with gastric adenocarcinoma, being lower in those with advanced disease. No significant associations were observed for other markers with respect to either tumor stage or histological grade. These findings suggest that the assessment of ApoB may be a good approach in evaluation

of gastric cancer burden or progression. However, due to the observational design of the study and the limited sample size, additional research is necessary to validate and extend these findings. Larger, multicenter studies incorporating nutritional, inflammatory, and metabolic covariates, along with prospective data, are recommended to validate and extend these findings. Understanding the link between lipid metabolism and tumor progression may offer new insights into gastric cancer biology and support the development of metabolism-based diagnostic or prognostic tools.

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Ethical Statement

This study was approved by the Ethics Committee of Golestan University of Medical Sciences (Code: IR.GOUMS.REC.1401.078).

Conflict of Interests

The authors declare no conflict of interest.

Author Contributions

Amir Asghary contributed to the conception and design of the study, biochemical data analysis, interpretation of results, and drafting of the manuscript. Marie Saghaeian Jazi contributed to molecular data acquisition, laboratory experiments, and data validation. Seyed Mostafa Mir contributed to data interpretation. Abbas Doulani contributed to statistical analysis and methodological consultation. Hamid Reza Joshaghani supervised the study, contributed to study design, critically revised the manuscript, and approved the final version to be published.

Data Availability Statment

The datasets generated and analyzed during the current study, including data obtained from the biobank, are available from the corresponding author upon reasonable request. reasonable request.

References

1. Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A, et al. Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA: A Cancer Journal for Clinicians. 2021;71(3):209-49.
2. Beloribi-Djefaflia S, Vasseur S, Guillaumond F. Lipid metabolic reprogramming in cancer cells. Oncogenesis. 2016;5(1):e189-e.
3. von Eckardstein A, Nordestgaard BG, Remaley AT, Catapano AL. High-density lipoprotein revisited: biological functions and clinical relevance. European Heart Journal. 2022;44(16):1394-407.

4. Shen JG, Jin LD, Dong MJ, Wang LB, Zhao WH, Shen J. Low level of serum high-density lipoprotein cholesterol in gastric cancer correlates with cancer progression but not survival. Translational Cancer Research. 2020;9(10):6206.
5. Pih GY, Gong EJ, Choi JY, Kim M-J, Ahn JY, Choe J, et al. Associations of serum lipid level with gastric cancer risk, pathology, and prognosis. Cancer Research and Treatment: Official Journal of Korean Cancer Association. 2021;53(2):445-56.
6. Peng Q, Zhan C, Shen Y, Xu Y, Ren B, Feng Z, et al. Blood lipid metabolic biomarkers are emerging as significant prognostic indicators for survival in cancer patients. BMC Cancer. 2024;24(1):1549.
7. Xu S, Fan Y, Tan Y, Zhang L, Li X. Association between blood lipid levels and risk of gastric cancer: A systematic review and meta-analysis. PLoS One. 2023;18(7):e0288111.
8. Li F, Han M, Gao X, Du X, Jiang C. APOA1 mRNA and serum APOA1 protein as diagnostic and prognostic biomarkers in gastric cancer. Translational Cancer Research. 2024;13(5):2141.
9. Ma M-z, Yuan S-q, Chen Y-m, Zhou Z-w. Preoperative apolipoprotein B/apolipoprotein A1 ratio: a novel prognostic factor for gastric cancer. OncoTargets and Therapy. 2018:2169-76.
10. Zhang H, Liu Y, Feng L, Wang L, Han J, Zhang X, et al. Blood lipid profiles associated with metastatic sites in advanced gastric cancer. BMC Gastroenterology. 2024;24(1):391.
11. Zhang D, Hu R-h, Cui X-m, Jiang X-h, Zhang S. Lipid levels and insulin resistance markers in gastric cancer patients: diagnostic and prognostic significance. BMC Gastroenterology. 2024;24(1):373.
12. Okamura T, Hashimoto Y, Hamaguchi M, Obora A, Kojima T, Fukui M. Triglyceride–glucose index (TyG index) is a predictor of incident colorectal cancer: a population-based longitudinal study. BMC Endocrine Disorders. 2020;20:1-7.
13. Li F, Dou J, Wei L, Li S, Liu J. Biomarkers of lipid metabolism in gastric cancer: a case control study. BMC Cancer. 2024;24(1):455.
14. Pagliari F, Di Franco S, Tirinato L. Lipid alterations in cancer development, resistance and recurrence. Frontiers in Cell and Developmental Biology. 2024;12:1493626.
15. Chen Y, Zhang J, Zhang Y, Zhu L. Effect of statin use on risk and mortality of gastric cancer: a meta-analysis. Anti-Cancer Drugs. 2023;34(8):901-9.
16. Yu Y, Zheng Z, Gao X, Gu Y, Zhang M, Hu B, et al. Plasma metabolomic signatures of H. pylori infection, alcohol drinking, smoking, and risk of gastric cancer. Molecular Carcinogenesis. 2025;64(3):463-74.
17. Dai J, Li Q, Quan J, Webb G, Liu J, Gao K. Construction of a lipid metabolism-related and immune-associated prognostic score for gastric cancer. BMC Medical Genomics. 2023;16(1):93.

Table 1. Comparison of lipid and apolipoprotein levels by tumor stage and histological grades (Parametric tests)

Variable (mg/dl)	Tumor stage			Histological grade		
	Early (Mean ±SEM)	Advanced (Mean ±SEM)	P-value	Well/Moderate (Mean ±SEM)	Poor/Undiff (Mean ± SEM)	P-value
Total cholesterol	134.5 ± 10.7	111.9 ± 6.0	**0.052**	114.7 ± 6.9	125.9 ± 9.0	0.317
HDL	36.7 ± 1.8	34.3 ± 1.9	0.419	34.0 ± 1.8	36.5 ± 2.1	0.369
ApoA1	105.8 ± 4.1	100.4 ± 4.2	0.409	99.8 ± 4.1	105.0 ± 4.7	0.374
ApoB	63.4 ± 5.2	52.4 ± 2.6	**0.042**	53.8 ± 3.02	59.2 ± 4.4	0.310
ApoB/ApoA1	0.59 ± 0.04	0.54 ± 0.02	0.185	0.55 ± 0.03	0.57 ± 0.04	0.666

HDL: High Density Lipoprotein; ApoA1: Apo Lipoprotein A1; ApoB: Apo Lipoprotein B; Early: Early Stages(0,I,II); Advanced: Advanced stages (III,IV); Well/Moderate: Well and Moderate grades; Poor/Undiff: Poor and Undifferentiated grades; SEM: Standard Error of Mean

Table 2. Comparison of non-normally distributed variables by tumor stage and histological grade (Mann–Whitney U test)

Variable (mg/dl)	Tumor stage			Histological grade		
	Early Median [IQR]	Advanced Median [IQR]	P-value	Well/Moderate Median [IQR]	Poor/Undiff Median [IQR]	P-value
TG	142 [111-186]	135 [105-174]	0.193	138 [115-180]	129 [97-172]	0.154
LDL	76 [58-100]	66 [51-96]	0.125	72 [56-96]	64 [48-91]	0.331
VLDL	29 [22-37]	28 [20-33]	0.817	30 [23-35]	26 [19-32]	**0.066**

TG: Triglyceride; LDL: Low Density Lipoprotein; VLDL: Very Low Density Lipoprotein; Early: Early Stages(0,I,II); Advanced: Advanced stages (III,IV); Well/Moderate: Well and Moderate grades; Poor/Undiff: Poor and Undifferentiated grades; IQR: Interquartile Range

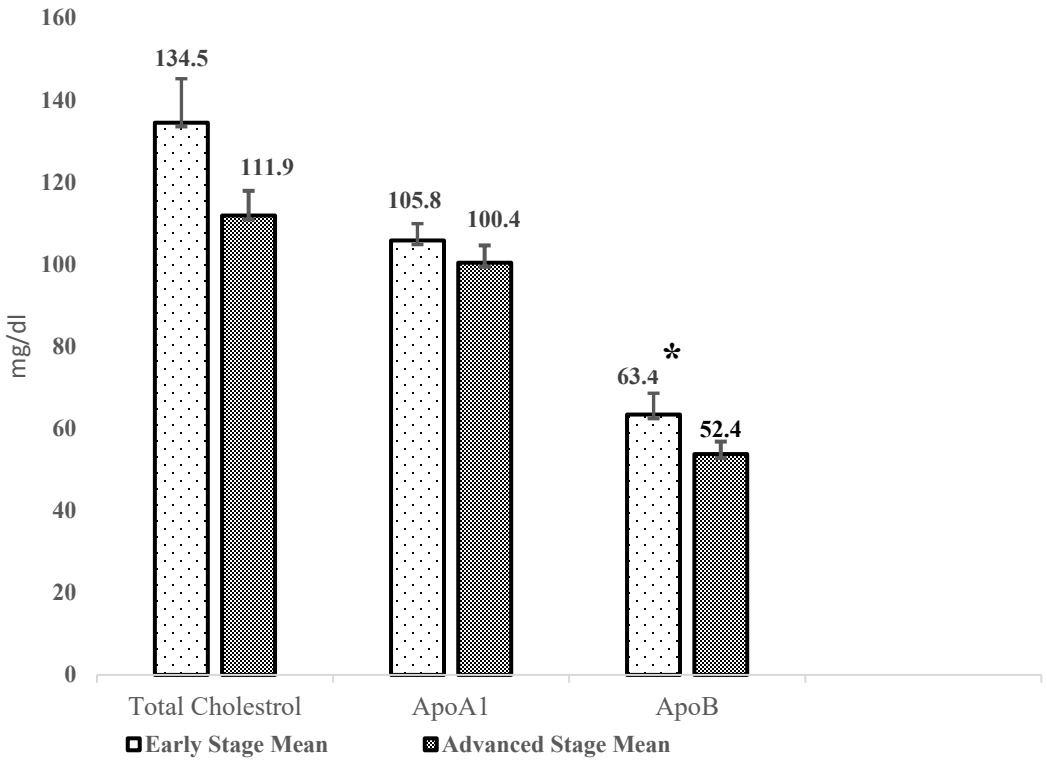

Figure 1. Comparison of selected serum lipid and apolipoprotein levels between early-stage and advanced-stage gastric adenocarcinoma patients**.** Bar plots represent the mean ± standard error (SE) of serum total cholesterol, apolipoprotein A1 (ApoA1), and apolipoprotein B (ApoB) levels stratified by disease stage. To ensure clarity and focus on the most relevant findings, only variables that demonstrated **biologically relevant (notable)** differences between groups were included in the chart. *: p-value< 0.05

Supplementary Table 1. Shapiro-Wilk test of normality in GC patients by stage and grade

Variable	Stage group	W	n	Sig.	Grade group	W	n	Sig.
Total Cholesterol	Early (0, I, II)	0.935	17	0.265	Well/Moderate	0.966	27	0.504
	Advanced (III, IV)	0.958	32	0.237	Low/Undiff.	0.933	22	0.144
TG	Early (0, I, II)	0.792	17	0.002	Well/Moderate	0.96	27	0.373
	Advanced (III, IV)	0.884	32	0.002	Low/Undiff.	0.785	22	0.000
LDL	Early (0, I, II)	0.95	17	0.457	Well/Moderate	0.94	27	0.121
	Advanced (III, IV)	0.921	32	0.022	Low/Undiff.	0.924	22	0.093
HDL	Early (0, I, II)	0.945	17	0.384	Well/Moderate	0.962	27	0.414
	Advanced (III, IV)	0.96	32	0.278	Low/Undiff.	0.939	22	0.188
VLDL	Early (0, I, II)	0.894	17	0.054	Well/Moderate	0.965	27	0.475
	Advanced (III, IV)	0.913	32	0.014	Low/Undiff.	0.81	22	0.001
ApoA1	Early (0, I, II)	0.945	17	0.388	Well/Moderate	0.962	27	0.407
	Advanced (III, IV)	0.96	32	0.271	Low/Undiff.	0.94	22	0.200
ApoB	Early (0, I, II)	0.957	17	0.573	Well/Moderate	0.959	27	0.355
	Advanced (III, IV)	0.948	32	0.123	Low/Undiff.	0.924	22	0.092
ApoB/ApoA1	Early (0, I, II)	0.972	17	0.859	Well/Moderate	0.971	27	0.624
	Advanced (III, IV)	0.979	32	0.770	Low/Undiff.	0.976	22	0.847