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

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Abstract

Background: Gastric cancer (GC), particularly adenocarcinoma, remains a major global health burden with high mortality, largely because of late-stage diagnosis and the limited availability of reliable biomarkers for disease monitoring. Lipid metabolism plays a crucial role in tumor biology, and serum lipid-related markers, including apolipoproteins, have been proposed 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 than 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 with respect to 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 for evaluating disease burden. Although no association was found with tumor grade, further validation in larger prospective studies incorporating metabolic and inflammatory covariates is warranted.

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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, increasing attention has been directed toward identifying noninvasive and cost-effective biomarkers that may 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 serve as potential biomarkers of 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 and 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 involving 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 may promote tumorigenesis. In the context of GC, previous studies reported that higher LDL-C levels were significantly associated with an 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 ApoA-I levels have been implicated in an increased 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, has been linked 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 uncertain because of study heterogeneity, confounding factors, and inconsistent results. A comprehensive analysis of lipid profiles across different stages and grades is therefore necessary to clarify their potential as biomarkers for diagnosis, prognosis, and therapeutic targeting.

Methods

This study included 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 enrollment. Ethical approval for the study protocol was obtained from the Ethics Committee of Golestan University of Medical Sciences (Ethics Code: IR.GOUMS.REC.1401.078). Patient data were anonymized, and confidentiality was strictly maintained throughout the study. To ensure sufficient statistical power and an 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, or 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 or undifferentiated 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 using fully automated analyzers, specifically the Mindray BS-480 and Biotechnica BT-3500 systems. These procedures were performed strictly in accordance with the manufacturers' protocols for reagent preparation, instrument calibration, quality control, and sample handling to ensure measurement accuracy, reliability, and reproducibility. VLDL levels were calculated as TG/5. Patients had 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, and total cholesterol). In contrast, non-normally distributed variables (TG, LDL, and 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.

Results

Comparison by tumor stage

Among the evaluated parameters, Apolipoprotein B (ApoB) emerged as the only parameter demonstrating statistically significant variation between early-stage and advanced-stage 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 the ApoB/ApoA1 ratio, showed 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. For variables with non-normal distributions (i.e., TG, LDL, and VLDL), the Mann-Whitney U test was applied. None of these variables showed statistically significant differences between stage groups. For example, 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) values for each parameter were as follows: 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; and median: 28 and IQR: 20-33 for VLDL in advanced stages.

Comparison by histological grade

Analysis by histological grade revealed no statistically significant differences in any 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 did not reach 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), or VLDL (p = 0.066) levels, all of which were not normally distributed.

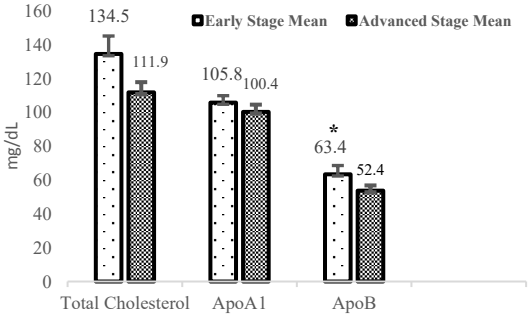

Figure 1. Comparison of selected serum lipid and apolipoprotein levels between early-stage and advanced-stage gastric adenocarcinoma patients. Bar plots represent the mean ± standard error (SE) of serum total cholesterol, apolipoprotein A1 (ApoA1), and apolipoprotein B (ApoB) levels stratified by disease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

In summary, ApoB was the only marker significantly associated 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 and warrant further investigation. Full statistical details, including descriptive data, test results, and p-values, are provided in Tables 1 and 2.

Table 1. Comparison of lipid and apolipoprotein levels by tumor stage and histological grade (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: Apolipoprotein A1; ApoB: Apolipoprotein 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 the 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.

Discussion

In this cross-sectional study, we evaluated the associations between serum lipid profiles, including apolipoproteins, and clinicopathological features in patients with gastric adenocarcinoma. The 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 associated with tumor burden rather than cellular differentiation. ApoB is the primary structural protein of LDL and VLDL particles and regulates 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., who reported lower ApoB levels 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 the triglyceride-glucose (TyG) index, in advanced-stage GC patients, reinforcing the concept of metabolic exhaustion in later disease stages (11). Contemporary research also indicates that the TyG index serves as a valid surrogate biomarker for insulin resistance and demonstrates significant associations with elevated neoplastic risk, particularly for malignancies of the gastrointestinal tract (12). Future studies should consider incorporating such metabolic parameters to further elucidate 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. These findings may reflect the relative stability of lipid indices after tumor growth. Factors such as sample size, population characteristics, nutritional status, and tumor heterogeneity may also 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 a significant role 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, a phenomenon supported by multi-omics studies such as the development of lipid metabolism-associated gene (LMAG) signatures (14). Additionally, links between dyslipidemia and gastric cancer 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 of cellular differentiation. Integrating ApoB measurements with other lipid and inflammatory indices (e.g., HDL, TyG, and hs-CRP) may enhance prognostic models. A key strength of this study is its rigorous statistical approach, utilizing both parametric and non-parametric tests guided by Shapiro-Wilk normality assessment. The inclusion of multiple lipid-related indicators also provides 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 has 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 the course of treatment.

In addition, assessing metabolic parameters, such as insulin resistance, could be useful in data evaluation. For example, the TyG index, a marker of insulin resistance, is directly correlated with the risk and progression of cancer through the induction of visceral adiposity dysfunction, systemic inflammation, and epigenetic modification. Therefore, assessment of insulin resistance and TyG could clarify their importance and correlation with lipid profiles across 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 elucidate 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, with lower levels observed 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 assessing ApoB may be useful for evaluating 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 confirm these results. 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).

Conflicts of Interest

The authors declare no conflicts of interest.

Author Contributions

Amir Asghary contributed to the conception and design of the study, biochemical data analysis, interpretation of the 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 for publication.

Data Availability Statement

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

Use of Artificial Intelligence

No artificial intelligence, including large language models or generative tools, was used in the research, data analysis, or preparation of this manuscript.

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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	Poor/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	Poor/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	Poor/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	Poor/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	Poor/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	Poor/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	Poor/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	Poor/Undiff.	0.976	22	0.847