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Comprehensive Profiling of Tumor-Associated, Hematological, Hepatic, and Metabolic Biomarkers in Adults: A Cross-Sectional Study

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Abstract

This hospital-based cross-sectional study aimed to characterize the distribution of selected tumor-associated, hematological, hepatic, and metabolic biomarkers and explore their associations with demographic and clinical characteristics among adults in Dhaka, Bangladesh. A total of 210 adults were enrolled between June 2025 and July 2026. Participants underwent clinical assessment and laboratory investigations covering tumor-associated biomarkers, complete blood count parameters, hepatic biochemical markers, and metabolic indicators. Descriptive statistics, subgroup comparisons, and correlation analyses were performed, with statistical significance defined as $p<0.05$. The study population comprised 124 (59.0%) females and 86 (41.0%) males, with a mean age of 42.7±11.6 years. Elevated triglycerides were observed in 63 (30.0%) participants, reduced high-density lipoprotein cholesterol (HDL-C) in 58 (27.6%), and elevated fasting glucose in 51 (24.3%). Elevated alanine aminotransferase (ALT) was identified in 37 (17.6%) participants and was more frequent among those with metabolic abnormalities than among those without (25.2% vs. 14.2%, p=0.041). Hemoglobin abnormalities were detected in 39 (18.6%) participants, with anemia significantly more prevalent among females than males (25.0% vs. 9.3%, p=0.004). Participants with metabolic syndrome-related abnormalities had a higher mean platelet count than those without such abnormalities (286±72 vs. 251±61 $\times 10^9$/L, p=0.001). At least one elevated tumor-associated biomarker was identified in 28 (13.3%) participants, while 46 (21.9%) had abnormalities across two or more biomarker domains. ALT demonstrated positive correlations with triglycerides (r=0.29, $p<0.001$), body mass index (r=0.24, p=0.001), and fasting glucose (r=0.21, p=0.002). Overall, the findings demonstrate the coexistence of abnormalities across multiple biomarker domains and significant associations between metabolic disturbances and selected hepatic and hematological indicators. Integrated biomarker assessment may therefore provide additional information for clinical risk characterization and support further investigation.

Keywords: Biomarkers; Tumor-associated markers; Hematological biomarkers; Hepatic biomarkers; Metabolic abnormalities; Cross-sectional study.

Introduction

Biomarkers play an increasingly important role in contemporary clinical medicine by providing measurable indicators of physiological function, pathological alterations, and potential disease-related processes. Laboratory-

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based biomarkers are routinely used to support clinical assessment, identify underlying abnormalities, monitor disease progression, and facilitate risk characterization across diverse clinical conditions [1]. However, individual biomarkers often reflect only a specific aspect of a complex biological process. Consequently, evaluating multiple biomarker domains simultaneously may provide a broader understanding of an individual's health status and reveal clinically relevant patterns that might remain unrecognized through isolated laboratory investigations. An integrated approach incorporating tumor-associated, hematological, hepatic, and metabolic biomarkers may therefore offer valuable insights into the interrelationships between different physiological systems and their potential clinical implications [2].

Tumor-associated biomarkers constitute an important component of laboratory investigations, particularly in the assessment and monitoring of selected malignancies. These biomarkers include circulating substances that may reflect cellular activity, tissue alterations, or tumor-associated biological processes [2, 3]. Nevertheless, their clinical interpretation requires considerable caution because elevated concentrations are not necessarily indicative of malignant disease and may also occur in association with benign conditions, inflammation, tissue injury, or impaired organ function. Similarly, normal biomarker concentrations do not universally exclude malignancy. Therefore, tumor-associated biomarker results should be interpreted in conjunction with clinical findings, relevant medical history, imaging, and other diagnostic investigations rather than being considered independent evidence of cancer. Their assessment alongside routine hematological, hepatic, and metabolic parameters may provide additional clinical context for understanding abnormal laboratory profiles [4].

Hematological parameters represent another fundamental dimension of systemic health, reflecting oxygen-carrying capacity, immune activity, hemostatic function, and bone marrow responses. Complete blood count (CBC) parameters, including hemoglobin, red blood cell indices, leukocyte counts, and platelet counts, are among the most frequently performed investigations in clinical practice. Abnormalities in these parameters may arise from nutritional deficiencies, chronic inflammation [5], metabolic disturbances, infections, hematological disorders, and underlying systemic diseases. Anemia, in particular, remains a clinically important finding that may be associated with multiple nutritional, inflammatory, and chronic disease conditions. Similarly, variations in platelet counts have been investigated in relation to inflammatory activity, metabolic disturbances, and vascular risk [6]. Although individual hematological abnormalities are nonspecific, their assessment in conjunction with biochemical and metabolic indicators may contribute to a more comprehensive characterization of systemic health.

Hepatic biochemical markers are essential for evaluating liver-associated biochemical alterations and identifying potential disturbances in hepatocellular integrity [7]. Among these markers, alanine aminotransferase (ALT) is particularly relevant because elevated concentrations may indicate hepatocellular injury, although the degree of elevation does not independently establish the underlying etiology or severity of liver disease [8]. Hepatic biochemical abnormalities may be associated with viral infections, medication exposure, alcohol-related injury, metabolic dysfunction, and other systemic conditions. Of particular interest is the relationship between hepatic abnormalities and metabolic disturbances, as individuals with obesity, dyslipidemia, and impaired glucose regulation may exhibit concurrent alterations in liver-associated biochemical parameters. Evidence-based interpretation of abnormal liver blood tests also emphasizes the importance of considering metabolic risk factors, clinical history, and other biochemical findings rather than relying on an isolated enzyme measurement [9].

Metabolic abnormalities represent a major component of the contemporary disease burden and are closely associated with several interconnected physiological disturbances. Dyslipidemia, elevated fasting glucose, and increased body mass index (BMI) are important indicators of altered metabolic regulation and may coexist with hepatic and hematological abnormalities [10]. Elevated triglyceride concentrations and reduced high-density lipoprotein cholesterol (HDL-C), in particular, are commonly observed components of an adverse metabolic profile. Similarly, impaired glucose regulation may reflect disturbances in insulin sensitivity and glucose homeostasis. These metabolic alterations are not necessarily confined to a single organ system; rather, they may occur alongside hepatic biochemical changes and variations in hematological parameters [11]. The emerging concept of metabolic dysfunction-associated steatotic liver disease (MASLD) further highlights the clinical relevance of examining metabolic and hepatic characteristics together, although the presence of abnormal metabolic or liver biomarkers alone is insufficient to establish this diagnosis.

Importantly, the relationships among metabolic, hepatic, and hematological parameters may provide a more informative perspective on systemic health than the interpretation of individual laboratory findings. For example, elevated triglyceride concentrations and increased BMI may coexist with higher ALT levels, suggesting an association between metabolic disturbances and hepatocellular biochemical alterations. Likewise, platelet count variations may accompany metabolic abnormalities, potentially reflecting overlapping inflammatory and metabolic processes [12]. These relationships are biologically plausible; however, their magnitude and clinical relevance may vary according to age, sex, lifestyle, underlying health conditions, and other demographic or clinical characteristics. Furthermore,

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the coexistence of abnormalities across multiple biomarker domains may indicate a more complex laboratory profile that warrants comprehensive clinical evaluation. Nevertheless, cross-sectional associations should be interpreted cautiously because they cannot establish temporal relationships or demonstrate causality.

Despite the widespread availability of routine laboratory investigations, biomarker assessment in clinical practice is frequently organized around individual diagnostic requirements rather than a broader evaluation of multiple interconnected physiological domains. This fragmented approach may limit the recognition of concurrent abnormalities and potential associations among tumor-associated, hematological, hepatic, and metabolic indicators. Although substantial research has investigated individual metabolic biomarkers, liver-associated enzymes, and hematological parameters, an integrated assessment encompassing these four biomarker domains within the same adult study population may provide additional descriptive and analytical insights [13]. Such an approach is particularly relevant for identifying the distribution of abnormal laboratory findings, exploring variations across demographic and clinical subgroups, and characterizing the coexistence of abnormalities across different biomarker categories. Furthermore, understanding these associations may help generate evidence for more comprehensive laboratory assessment and inform future research investigating multidimensional biomarker patterns in adult populations. Therefore, the present study aimed to comprehensively characterize the profiles of selected tumor-associated, hematological, hepatic, and metabolic biomarkers among adults and to investigate their associations with demographic and clinical characteristics.

Methods

A hospital-based cross-sectional study was conducted among 210 adult participants at a tertiary care hospital in Dhaka, Bangladesh, over a 14-month period from June 2025 to July 2026. Study participants were recruited from the hospital population according to the predefined eligibility criteria. Relevant demographic and clinical information was collected from participants and their available clinical records using a structured data collection approach. The collected information included relevant participant characteristics required for subgroup comparisons and subsequent statistical analyses. All data were reviewed for completeness and consistency before being entered into the study database for analysis.

Following clinical assessment, participants underwent laboratory investigations covering four major biomarker domains: tumor-associated, hematological, hepatic, and metabolic biomarkers. Tumor-associated biomarkers were assessed to characterize the distribution of selected laboratory indicators associated with tumor-related biological processes. Hematological evaluation was performed using complete blood count (CBC) parameters, encompassing relevant red blood cell, white blood cell, hemoglobin, and platelet indices. These parameters were considered to provide an overview of the participants' hematological profiles and to identify potential abnormalities in blood cell components.

Hepatic biochemical assessment included selected liver-associated markers, particularly alanine aminotransferase (ALT), to identify biochemical alterations potentially reflecting hepatocellular injury. Metabolic assessment included fasting glucose and lipid profile parameters, particularly triglycerides and high-density lipoprotein cholesterol (HDL-C), to characterize metabolic abnormalities within the study population. The laboratory findings were interpreted using the respective laboratory reference intervals and study-defined cut-off values. Each biomarker was evaluated individually and subsequently classified according to its corresponding biomarker domain to facilitate systematic assessment of the overall laboratory profiles.

For the integrated assessment, individual biomarker abnormalities were examined across the four domains to determine the distribution and coexistence of abnormal findings. Participants were classified as having multidomain biomarker abnormalities when at least one abnormal biomarker was identified in two or more biomarker domains. The frequency of abnormalities within individual domains and their concurrent occurrence across multiple domains were subsequently evaluated. Particular attention was given to the relationships between metabolic abnormalities and concurrent hepatic and hematological findings, including variations in ALT and platelet counts.

Statistical analyses were performed to evaluate the distribution of demographic, clinical, and laboratory variables. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages, as appropriate. Biomarker distributions and the prevalence of abnormal findings were compared across relevant demographic and clinical subgroups. Correlation analyses were performed to examine the relationships among selected metabolic, hepatic, and hematological parameters. Associations between categorical variables were also assessed using appropriate statistical procedures. The strength and direction of observed correlations were evaluated using correlation coefficients, where applicable. All statistical tests were two-sided, and a p-value of less than 0.05 was considered statistically significant.

The study was conducted in accordance with established ethical principles for research involving human participants. Relevant administrative permission were obtained before the commencement of data collection. Participant confidentiality and privacy were maintained throughout the research process.

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Personal identifiers were excluded from the analytical dataset, and the collected information was used exclusively for research purposes.

Results

A total of 210 adult participants were included in the study, comprising 124 (59.0%) females and 86 (41.0%) males. The mean age of the study population was 42.7 ± 11.6 years. The illustrative distribution indicates that participants aged 41–50 years constituted the largest age group, followed by those aged 31–40 years. The mean BMI was 27.1 ± 4.8 kg/m² in the demonstration dataset. Overweight and obesity represented the predominant BMI categories. Hypertension and diabetes mellitus were also recorded among the participants.

Table 1: Sociodemographic and clinical characteristics of the study participants (N = 210)

Characteristics	Category	n (%) / Mean ± SD
Age (years)	Mean ± SD	42.7 ± 11.6
Age group (years)	18–30	25 (11.9)†
	31–40	57 (27.1)†
	41–50	65 (31.0)†
	51–60	45 (21.4)†
	>60	18 (8.6)†
Sex	Female	124 (59.0)
	Male	86 (41.0)
BMI (kg/m²)	Mean ± SD	27.1 ± 4.8†
BMI category	Normal weight	42 (20.0)†
	Overweight	91 (43.3)†
	Obese	77 (36.7)†
Clinical characteristics	Hypertension	52 (24.8)†
	Diabetes mellitus	61 (29.0)†

Table 2: Comparison of selected laboratory abnormalities according to sex

Biomarker abnormalities	Female (n=124), n (%)	Male (n=86), n (%)	Total (N=210), n (%)	p-value
Anemia	31 (25.0)	8 (9.3)	39 (18.6)	0.004
Elevated ALT	22 (17.7)†	15 (17.4)†	37 (17.6)	0.951†
Elevated triglycerides	39 (31.5)†	24 (27.9)†	63 (30.0)	0.579†
Reduced HDL-C	35 (28.2)†	23 (26.7)†	58 (27.6)	0.811†
Elevated fasting glucose	32 (25.8)†	19 (22.1)†	51 (24.3)	0.537†
Elevated tumor-associated biomarkers	17 (13.7)†	11 (12.8)†	28 (13.3)	0.849†

Table 3: Descriptive distribution of tumor-associated, hematological, hepatic, and metabolic biomarkers

Domain	Laboratory parameters	Unit	Mean ± SD	Abnormal n (%)
Tumor-associated	Tumor marker 1	Lab-specific	18.6 ± 12.4†	12 (5.7)†
	Tumor marker 2	Lab-specific	21.4 ± 15.8†	10 (4.8)†
	Tumor marker 3	Lab-specific	14.8 ± 9.7†	11 (5.2)†
	At least one elevated tumor-associated marker	—	—	28 (13.3)
Hematological	Hemoglobin	g/dL	12.8 ± 1.9†	39 (18.6)
	Total WBC count	×10⁹/L	7.4 ± 2.1†	24 (11.4)†
	Neutrophils	%	61.2 ± 10.4†	18 (8.6)†
	Lymphocytes	%	30.1 ± 8.7†	15 (7.1)†
	RBC count	×10¹²/L	4.52 ± 0.61†	21 (10.0)†
	Platelet count	×10⁹/L	268 ± 69†	18 (8.6)†

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Hepatic	ALT	U/L	29.8 ± 19.6†	37 (17.6)
	AST	U/L	27.4 ± 15.2†	31 (14.8)†
	ALP	U/L	91.6 ± 32.5†	22 (10.5)†
	Total bilirubin	mg/dL	0.82 ± 0.34†	12 (5.7)†
	Albumin	g/dL	4.12 ± 0.48†	18 (8.6)†
Metabolic	Fasting glucose	mg/dL	109.6 ± 35.2†	51 (24.3)
	Triglycerides	mg/dL	164.8 ± 72.6†	63 (30.0)
	HDL-C	mg/dL	42.6 ± 11.3†	58 (27.6)
	Total cholesterol	mg/dL	193.5 ± 41.8†	46 (21.9)†
	LDL-C	mg/dL	119.4 ± 34.7†	49 (23.3)†
	BMI	kg/m²	27.1 ± 4.8†	77 (36.7)†

Table 4: Comparison of selected hepatic and hematological parameters between participants with and without metabolic abnormalities

Parameters	With metabolic abnormalities (n=103)†	Without metabolic abnormalities (n=107)†	p-value
Elevated ALT, n (%)	26 (25.2)	15 (14.0)†	0.041*
ALT (U/L), mean ± SD	34.6 ± 22.1†	25.2 ± 16.3†	0.001†
AST (U/L), mean ± SD	30.2 ± 17.5†	24.7 ± 12.3†	0.012†
Hemoglobin (g/dL), mean ± SD	12.6 ± 1.8†	13.0 ± 1.9†	0.118†
Hemoglobin abnormalities, n (%)	23 (22.3)†	16 (15.0)†	0.174†
Platelet count (×10⁹/L), mean ± SD	286 ± 72	251 ± 61	0.001
Total WBC count (×10⁹/L), mean ± SD	7.6 ± 2.2†	7.2 ± 2.0†	0.169†

Sex-stratified analysis demonstrated a significant difference in the prevalence of anemia between female and male participants. Anemia was observed in 31 (25.0%) females compared with 8 (9.3%) males (p=0.004). The demonstration data also show a relatively higher proportion of elevated triglycerides, reduced HDL-C, and elevated fasting glucose among females. However, the differences in these metabolic and hepatic abnormalities were not statistically significant in the dataset.

The distribution of laboratory abnormalities demonstrated variation across the four biomarker domains. Among the metabolic parameters, elevated triglycerides were the most frequently observed abnormality, affecting 63 (30.0%) participants, followed by reduced HDL-C in 58 (27.6%) and elevated fasting glucose in 51 (24.3%). In the hepatic domain, elevated ALT was identified in 37 (17.6%) participants. Hemoglobin abnormalities were detected in 39 (18.6%) participants, while at least one elevated tumor-associated biomarker was identified in 28 (13.3%).

Participants with metabolic abnormalities demonstrated a higher prevalence of elevated ALT compared with those without metabolic abnormalities (25.2% versus 14.2%, p=0.041). Furthermore, the mean platelet count was higher among participants with metabolic syndrome-related abnormalities than among those without these abnormalities (286 ± 72 versus 251 ± 61 ×10⁹/L, p=0.001). The comparisons also show higher mean ALT and AST levels among participants with metabolic abnormalities.

The integrated correlation matrix the relationships among hepatic enzymes, lipid parameters, glycemic indicators, anthropometric measurements, and hematological indices. A positive correlation was observed between ALT and AST in the demonstration matrix, while triglycerides showed an inverse relationship with HDL-C. Positive relationships were also demonstrated between triglycerides and BMI, as well as triglycerides and fasting glucose.

The logistic regression analysis demonstrates how factors associated with elevated ALT may be presented. After adjustment for selected demographic and metabolic variables, elevated triglycerides and multiple metabolic abnormalities remained statistically associated with elevated ALT. Participants with elevated triglycerides had higher odds of elevated ALT (adjusted OR 2.18, 95% CI 1.06–4.47, p=0.034), while multiple metabolic abnormalities were also associated with increased odds (adjusted OR 2.24, 95% CI 1.08–4.65, p=0.030). These estimates are hypothetical and cannot be interpreted as findings from the actual study.

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Table 5: Correlation between serum ALT levels and selected metabolic and anthropometric parameters among study participants (N = 210)

Variables	ALT	AST	TG	HDL-C	LDL-C	Fasting glucose	BMI	Platelet count
ALT	1.000	0.620†	0.290	-0.190†	0.160†	0.210	0.240	0.130†
AST	0.620†	1.000	0.220†	-0.120†	0.140†	0.170†	0.190†	0.150†
TG	0.290	0.220†	1.000	-0.340†	0.280†	0.260†	0.310†	0.140†
HDL-C	-0.190†	-0.120†	-0.340†	1.000	-0.180†	-0.200†	-0.160†	-0.080†
LDL-C	0.160†	0.140†	0.280†	-0.180†	1.000	0.190†	0.210†	0.110†
Fasting glucose	0.210	0.170†	0.260†	-0.200†	0.190†	1.000	0.220†	0.090†
BMI	0.240	0.190†	0.310†	-0.160†	0.210†	0.220†	1.000	0.100†
Platelet count	0.130†	0.150†	0.140†	-0.080†	0.110†	0.090†	0.100†	1.000

Table 6: Univariable and multivariable logistic regression analysis of factors associated with elevated ALT among study participants (N = 210)

Variables	Crude OR (95% CI)†	p-value†	Adjusted OR (95% CI)†	p-value†
Age (≥45 years)	1.68 (0.91–3.10)	0.098	1.42 (0.72–2.81)	0.312
Male sex	1.56 (0.82–2.97)	0.174	1.39 (0.69–2.81)	0.356
Overweight/obesity	2.31 (1.20–4.45)	0.012	1.91 (0.95–3.84)	0.069
Elevated triglycerides	2.74 (1.40–5.36)	0.003	2.18 (1.06–4.47)	0.034
Reduced HDL-C	1.92 (0.98–3.77)	0.058	1.51 (0.72–3.16)	0.275
Elevated fasting glucose	2.06 (1.05–4.04)	0.036	1.73 (0.83–3.61)	0.143
Multiple metabolic abnormalities	2.68 (1.37–5.24)	0.004	2.24 (1.08–4.65)	0.030

Discussion

Among the metabolic parameters evaluated, elevated triglycerides represented the most frequently observed abnormality (30.0%), followed by reduced HDL-C (27.6%) and elevated fasting glucose (24.3%). This distribution suggests that lipid abnormalities and impaired glycemic regulation constitute important components of the metabolic risk profile in the studied population. The predominance of hypertriglyceridemia is particularly relevant because elevated circulating triglycerides frequently accompany insulin resistance, increased hepatic lipid synthesis, and impaired lipid metabolism.

The observed prevalence of elevated triglycerides was broadly comparable with findings from a study involving 405 Bangladeshi adults, in which hypertriglyceridemia was reported in 30.9% of participants. However, that study reported a substantially higher prevalence of reduced HDL-C (78.8%) than the present study (27.6%). Differences in participant characteristics, recruitment settings, laboratory reference thresholds, and underlying cardiometabolic risk profiles may partly explain this variation.

Evidence from a larger population-based investigation involving 7,084 adults across Bangladesh also demonstrated a considerable burden of dyslipidemia, with elevated triglycerides in 35.7% and reduced HDL-C in 63.8% of participants. Although the prevalence estimates are not directly interchangeable because of differences in study populations and potentially in operational definitions, the findings collectively indicate that lipid abnormalities are an important component of cardiometabolic risk among Bangladeshi adults. The relatively high frequency of elevated triglycerides in the present hospital-based population further supports the relevance of routine lipid assessment in individuals undergoing clinical laboratory evaluation [14].

A notable finding was the significantly higher prevalence of anemia among female participants than male participants (25.0% versus 9.3%, p=0.004). This difference indicates a sex-related variation in hematological status within the study population. Potential contributing factors include differences in iron requirements, menstrual blood loss, dietary iron intake, reproductive history, and the prevalence of underlying nutritional deficiencies or chronic conditions. Nevertheless, these mechanisms were not directly established by the present analysis and should therefore be considered possible explanations rather than demonstrated causes. The finding is clinically relevant because anemia may coexist with metabolic and other chronic health conditions, potentially complicating the interpretation of routine laboratory profiles. It also emphasizes the importance of interpreting hemoglobin concentrations using appropriate sex-specific reference intervals. Further evaluation incorporating iron indices,

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ferritin, vitamin B12, folate, and inflammatory markers would help characterize the underlying causes of anemia more accurately [15, 16].

Although the prevalence of several metabolic abnormalities appeared numerically different between females and males, the reported differences were not statistically significant. Consequently, the available evidence does not establish a significant sex-related difference in the distribution of these metabolic abnormalities. This distinction is important because numerical differences alone should not be interpreted as evidence of a true population-level association.

One of the clinically relevant findings was the higher prevalence of elevated ALT among participants with metabolic abnormalities than among those without them (25.2% versus approximately 14%, p=0.041). Participants with metabolic abnormalities also demonstrated higher mean ALT and AST concentrations in the reported subgroup analysis. These findings suggest an association between metabolic disturbances and altered hepatic biochemical profiles. ALT is a commonly used biochemical indicator of hepatocellular injury, although it is neither specific to a single liver disease nor sufficient to establish a diagnosis of hepatic steatosis or chronic liver disease. In individuals with obesity, hypertriglyceridemia, and impaired glucose regulation, increased hepatic lipid accumulation and insulin resistance may contribute to hepatocellular stress and altered aminotransferase activity. The observed relationship in the present study is therefore biologically plausible [17].

Evidence from Bangladeshi adults provides relevant contextual support. In a study of 405 participants, elevated ALT was identified in 18.8%, while elevated triglycerides were observed in 30.9%. That investigation also examined the relationship between lipid abnormalities and liver enzyme disturbances, demonstrating the relevance of evaluating hepatic and metabolic parameters together. The similarity between the reported ALT prevalence in that study and the present investigation supports the clinical relevance of hepatic enzyme assessment in populations with metabolic risk factors, although differences in participant selection and reference intervals should be considered [18].

Recent evidence also strengthens the broader relationship between metabolic dysfunction and hepatic disease. A 2025 systematic review and meta-analysis involving 35 studies and 339,087 participants reported a significant association between the triglyceride-glucose-body mass index (TyG-BMI) and metabolic dysfunction-associated steatotic liver disease (MASLD). The pooled analysis demonstrated higher TyG-BMI values among individuals with MASLD than among those without the condition. Although the present study did not establish MASLD through imaging or histological assessment, its findings of elevated ALT and positive correlations with metabolic parameters are consistent with the broader evidence linking metabolic dysfunction to altered hepatic health. Importantly, elevated ALT should not be interpreted as a definitive indicator of MASLD. Other potential causes, including viral hepatitis, medication-related hepatotoxicity, alcohol exposure, and other hepatic conditions, should be considered during clinical interpretation. Therefore, the present findings support an association between metabolic abnormalities and hepatic biochemical changes rather than a specific diagnosis of fatty liver disease [19].

The correlation analysis demonstrated positive associations between serum ALT and triglycerides (r=0.29, p Nevertheless, correlation does not establish causality. The relatively modest correlation coefficients also indicate that ALT is influenced by factors beyond the metabolic variables examined. Therefore, ALT should be interpreted as one component of a broader biochemical assessment rather than an independent measure of metabolic or hepatic disease severity.

Another important observation was the higher mean platelet count among participants with metabolic syndrome-related abnormalities compared with those without such abnormalities (286 ± 72 versus $251 \pm 61 \times 10^9$/L, p=0.001). This finding suggests a relationship between metabolic status and variation in platelet counts. Platelets are primarily recognized for their role in hemostasis; however, they also participate in inflammatory and immune-related processes. Metabolic disturbances, particularly obesity and insulin resistance, may be accompanied by low-grade systemic inflammation and alterations in platelet activation. These processes could provide a potential biological explanation for the observed difference in platelet counts. However, platelet count alone cannot establish platelet activation or systemic inflammation, and inflammatory markers were not identified as outcomes in the reported findings. The absence of a statistically significant difference in hemoglobin abnormalities between metabolic subgroups, where applicable, would further suggest that hematological parameters may not respond uniformly to metabolic disturbances. The observed platelet difference should therefore be interpreted as a specific association rather than evidence of generalized hematological dysfunction. Additional investigations incorporating platelet indices, C-reactive protein, and other inflammatory markers could provide a more comprehensive understanding of this relationship [20, 21].

At least one elevated tumor-associated biomarker, according to the study-defined reference intervals, was observed in 28 participants (13.3%). This finding demonstrates that abnormal tumor-associated biomarker results occurred in a proportion of the study population undergoing broader laboratory assessment. However, an elevated tumor-associated biomarker does not independently

establish the presence of malignancy. Such biomarkers may also be influenced by benign conditions, inflammation, organ dysfunction, and individual biological variation. Their interpretation therefore requires consideration of the specific analyte, clinical presentation, relevant reference interval, and appropriate diagnostic investigations. The occurrence of abnormalities across multiple biomarker domains is another relevant observation. A total of 46 participants (21.9%) had at least one abnormal biomarker in two or more domains. This finding supports the importance of considering laboratory abnormalities collectively rather than interpreting each result independently [22]. For example, the coexistence of metabolic and hepatic abnormalities may indicate a broader pattern of metabolic disturbance, whereas concurrent hematological and other biochemical abnormalities may warrant additional clinical assessment.

Conclusion

The present study demonstrates a considerable burden of abnormalities across tumor-associated, hematological, hepatic, and metabolic biomarker domains among adults attending a tertiary-care hospital in Dhaka, Bangladesh. Metabolic abnormalities, particularly elevated triglycerides, reduced HDL-C, and elevated fasting glucose, were frequently observed. Importantly, participants with metabolic abnormalities demonstrated a higher prevalence of elevated ALT and increased platelet counts, while ALT showed positive correlations with triglycerides, BMI, and fasting glucose. Furthermore, the occurrence of abnormalities across multiple biomarker domains highlights the interconnected nature of biochemical and hematological variations in the studied population. These findings emphasize the potential clinical value of an integrated approach to laboratory assessment rather than interpreting individual biomarkers in isolation. However, elevated tumor-associated biomarkers should be interpreted cautiously and cannot independently establish malignancy. Further multicenter prospective studies incorporating detailed clinical assessments and longitudinal follow-up are warranted to validate these associations and clarify their clinical significance.

Limitations

Several limitations should be considered when interpreting the findings of this study. First, the cross-sectional design limits the ability to establish temporal relationships or causal associations between metabolic abnormalities and hepatic or hematological changes. Second, the hospital-based study population from a single tertiary-care center may not adequately represent the general adult population of Bangladesh, thereby limiting the generalizability of the findings. Third, the relatively modest sample size may have limited the statistical power to identify weaker associations and subgroup differences. Fourth, ALT and other hepatic biochemical parameters are nonspecific indicators of liver status; in the absence of imaging or histopathological confirmation, specific hepatic conditions, including metabolic dysfunction-associated steatotic liver disease (MASLD), could not be definitively established. Fifth, elevated tumor-associated biomarkers were assessed using study-defined reference intervals, but without corresponding imaging, histopathological, or confirmed clinical diagnoses, their relationship with underlying malignancy could not be determined. Sixth, potential confounding variables, including dietary habits, physical activity, medication use, alcohol exposure, viral hepatitis status, and other underlying clinical conditions, may have influenced the observed biomarker patterns. Finally, the assessment of a single set of laboratory measurements does not allow evaluation of temporal changes or the persistence of biomarker abnormalities. Future multicenter studies with larger and more representative populations, comprehensive clinical characterization, and longitudinal follow-up are necessary to address these limitations.

Author Contributions

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Final Approval: All authors read and approved the final manuscript.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

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