Texila International Journal of Public Health
ISSN: 2520-3134
DOI: 10.21522/TIJPH.2013.13.02.Art081

Serum Biochemical Parameters Harmonized with Demographic Characteristics in Bladder Cancer Patients

Ayad k. Ibrahim, Mahmoud A. M. Fakhri*
College of Science, University of Mosul, Mosul, Iraq

Abstract

This study investigated variations in biochemical parameters across different age groups, BMI categories, and smoking status to assess metabolic health and disease risk. Key findings include age-related trends; stable parameters: Total cholesterol (TC), HDL-C, LDL-C, atherogenic index, urea, ALT, AST, total protein (TP), and albumin showed no significant differences across age groups (under 50, 50–65, and over 65 years). Age-Dependent Changes Triglycerides (TG) and VLDL-C levels decreased significantly with age, while creatinine levels increased significantly with advancing age. BMI-Related Trends: Significant Increases TC and LDL-C levels rose significantly with higher BMI (normal weight, overweight, obese). Stable Parameters TG, HDL-C, VLDL-C, atherogenic index, urea, creatinine, ALT, and AST showed no significant differences across BMI groups. Smoking-Related Trends: Smokers exhibited trends of elevated levels in urea, creatinine, ALT, AST, TC, TG, VLDL, and LDL compared to non-smokers, though none of these differences were statistically significant. HDL was slightly lower, and total protein and albumin showed minimal differences in smokers, none of which were statistically significant. Age significantly influences TG, VLDL-C, and creatinine levels, highlighting the importance of age-specific metabolic assessments. Higher BMI is associated with adverse lipid profiles, particularly elevated TC and LDL-C, emphasizing the need for weight management in cardiovascular health. Smoking may impact biochemical parameters, but further research is required to confirm these trends. These findings underscore the importance of considering age, BMI, and lifestyle factors when evaluating metabolic health and disease risk.

Keywords: *Age Groups, Biochemical Parameters, Lipid Profile, Liver Enzymes, Metabolic Markers, Renal Function.*

Introduction

Bladder cancer is one of the most common urological malignancies, with significant morbidity and mortality worldwide [1]. It is a heterogeneous disease influenced by various factors, including genetic predisposition, environmental exposures, and lifestyle habits [2]. The diagnosis and management of bladder cancer rely on a combination of clinical, histopathological, and biochemical assessments [3, 4]. Among these, serum biochemical parameters have emerged as valuable tools for understanding the disease's pathophysiology, monitoring progression, and predicting outcomes [5, 6].

Serum biochemical parameters, such as liver enzymes, renal function markers, lipid profiles, and proteins, provide insights into the systemic effects of bladder cancer and its treatment [7, 8]. These parameters can reflect metabolic alterations, organ dysfunction, and inflammatory responses associated with the disease [9]. Furthermore, demographic characteristics, such as age, gender, and ethnicity, play a crucial role in shaping the clinical presentation and progression of bladder cancer. For instance, older age and

Received: 18.03.2025 ***Accepted: 21.04.2025*** ***Published on: 30.06.2025***
****Corresponding Author: drmahmoudahmed@uomosul.edu.iq***

male gender are well-established risk factors for bladder cancer, while ethnic disparities may influence access to care and treatment outcomes [10].

Harmonizing serum biochemical parameters with demographic characteristics offers a comprehensive approach to understanding bladder cancer's multifaceted nature [11, 12]. This integration can help identify biomarkers that are influenced by demographic factors, improve risk stratification, and tailor personalized treatment strategies [11, 13]. Moreover, it can shed light on the interplay between biological and sociodemographic variables, ultimately contributing to better patient outcomes.

This study aims to explore the relationship between serum biochemical parameters and demographic characteristics in bladder cancer patients. By analyzing these associations, we seek to uncover potential biomarkers that reflect disease severity, progression, and response to therapy, while considering the influence of demographic factors. Such insights could pave the way for more precise diagnostic tools and targeted therapeutic interventions, ultimately improving the management of bladder cancer.

Materials and Methods

The current study was carried out with a cohort of 60 bladder cancer patients, aged between 35 and 90 years, alongside a comparable number of healthy controls, aged 33 to 72 years. The patients were recruited from hospitals in Mosul City (Iraq) from November 2023 to April 2024. Serum samples were collected from participants and subsequently processed for further analysis.

Venous blood samples were collected, allowed to clot at room temperature, and then centrifuged at 1000g for 10 minutes to separate the serum for further analysis.

Biochemical Analysis

Measurement of Serum Urea

The principle of the assay for measuring urea in serum is based on the enzymatic hydrolysis of urea into ammonia and carbon dioxide, followed by the quantification of the resulting products. The most commonly used method is the urease method, which involves the following steps: Urea in the serum is hydrolyzed by the enzyme urease into ammonia (NH_3) and carbon dioxide (CO_2). The ammonia produced in the reaction is then quantified using a colorimetric or spectrophotometric method, where ammonia reacts with hypochlorite and phenol in the presence of a catalyst (e.g. sodium nitroprusside) to form a blue-green indophenol compound. The intensity of the color is proportional to the concentration of ammonia, and thus to the original urea concentration in the sample. The absorbance of the colored product is measured spectrophotometrically at a specific wavelength (typically around 600–630 nm). The urea concentration in the serum is determined by comparing the absorbance of the sample to a standard curve prepared with known concentrations of urea.

Measurement of Serum Creatinine

The principle of the assay for measuring creatinine in serum is based on the Jaffe reaction, a widely used colorimetric method. Creatinine is a waste product of muscle metabolism and is filtered out of the blood by the kidneys. Its measurement is crucial for assessing kidney function. The assay involves the following steps: creatinine reacts with picric acid in an alkaline medium (usually sodium hydroxide) to form a red-orange complex known as creatinine-picrate. The intensity of the red-orange color produced is directly proportional to the concentration of creatinine in the serum sample. The absorbance of the creatinine-picrate complex is measured spectrophotometrically at a

wavelength of approximately 490–520 nm. The creatinine concentration in the sample is determined by comparing its absorbance to a standard curve prepared with known concentrations of creatinine.

Measurement of Serum Alanine Aminotransferase

The principle of the assay for measuring alanine aminotransferase (ALT) in serum is based on an enzymatic reaction that involves the transfer of an amino group from alanine to α-ketoglutarate, producing pyruvate and glutamate. ALT is an enzyme primarily found in the liver, and its levels in serum are a key marker for liver health and damage. The assay typically follows these steps: ALT catalyzes the transfer of an amino group from L-alanine to α-ketoglutarate, resulting in the formation of pyruvate and L-glutamate. The pyruvate produced in the first reaction is then measured using a secondary indicator reaction. This involves the enzyme lactate dehydrogenase (LDH), which converts pyruvate to lactate while oxidizing NADH (reduced nicotinamide adenine dinucleotide) to NAD^+. The decrease in absorbance due to the oxidation of NADH to NAD^+ is measured spectrophotometrically at a wavelength of 340 nm. Since NADH absorbs light at 340 nm while NAD^+ does not, the rate of decrease in absorbance is directly proportional to the ALT activity in the serum sample. The assay is specific for ALT because the reaction is coupled to the oxidation of NADH, which only occurs in the presence of pyruvate generated by ALT. The method is highly sensitive due to the use of NADH, which has a strong absorbance at 340 nm.

Measurement of Serum Aspartate Aminotransferase

The principle of the assay for measuring aspartate aminotransferase (AST) in serum is based on an enzymatic reaction that involves the transfer of an amino group from aspartate to α-ketoglutarate, producing oxaloacetate and glutamate. AST is an enzyme found in various tissues, including the liver, heart, muscles, and kidneys, and its levels in serum are used as a marker for tissue damage, particularly liver and heart conditions. The assay typically follows these steps: AST catalyzes the transfer of an amino group from L-aspartate to α-ketoglutarate, resulting in the formation of oxaloacetate and L-glutamate. The oxaloacetate produced in the first reaction is then measured using a secondary indicator reaction. This involves the enzyme malate dehydrogenase (MDH), which converts oxaloacetate to malate while oxidizing NADH (reduced nicotinamide adenine dinucleotide) to NAD^+. The decrease in absorbance due to the oxidation of NADH to NAD^+ is measured spectrophotometrically at a wavelength of 340 nm. Since NADH absorbs light at 340 nm while NAD^+ does not, the rate of decrease in absorbance is directly proportional to the AST activity in the serum sample.

Measurement of Serum Albumin

The measurement of albumin in serum is primarily based on the dye-binding principle, where albumin forms a colored complex with a dye (bromocresol green or bromocresol purple). The intensity of the color is measured spectrophotometrically and used to determine albumin concentration.

Measurement of Serum Total Cholesterol

The principle of the assay for measuring total cholesterol in serum is based on enzymatic reactions that convert cholesterol into products that can be quantified spectrophotometrically. Cholesterol is a lipid molecule essential for cell membrane structure, hormone synthesis, and bile acid production. Its measurement is crucial for assessing cardiovascular health and lipid metabolism. The most commonly used method for total cholesterol measurement is the enzymatic colorimetric assay, which involves the following steps: cholesterol in the serum

exists in two forms: free cholesterol and cholesterol esters. The enzyme cholesterol esterase is used to hydrolyze cholesterol esters into free cholesterol and fatty acids. The free cholesterol is then oxidized by the enzyme cholesterol oxidase to produce cholest-4-en-3-one and hydrogen peroxide (H_2O_2). The hydrogen peroxide generated in the previous step reacts with a chromogenic substrate (e.g. 4-aminoantipyrine and phenol) in the presence of the enzyme peroxidase to form a colored quinoneimine dye. The intensity of the colored quinoneimine dye is measured spectrophotometrically at a wavelength of 500–520 nm. The absorbance is directly proportional to the total cholesterol concentration in the serum sample. The cholesterol concentration is determined by comparing the absorbance to a standard curve prepared with known concentrations of cholesterol.

Measurement of Serum Triglyceride

Triglycerides in the serum are hydrolyzed into glycerol and free fatty acids by the enzyme lipase. The glycerol produced in the first step is then phosphorylated by the enzyme glycerol kinase to form glycerol-3-phosphate. Glycerol-3-phosphate is oxidized by the enzyme glycerol-3-phosphate oxidase to produce dihydroxyacetone phosphate and hydrogen peroxide (H_2O_2). The hydrogen peroxide generated in the previous step reacts with a chromogenic substrate (e.g. 4-aminoantipyrine and a phenol derivative) in the presence of the enzyme peroxidase to form a colored quinoneimine dye. The intensity of the colored quinoneimine dye is measured spectrophotometrically at a wavelength of 500–520nm. The absorbance is directly proportional to the triglyceride concentration in the serum sample. The triglyceride concentration is determined by comparing the absorbance to a standard curve prepared with known concentrations of triglycerides.

Measurement of Serum High-density Lipoprotein

The principle of the assay for measuring high-density lipoprotein (HDL) in serum involves the selective isolation of HDL cholesterol followed by its quantification using enzymatic methods. HDL is often referred to as "good cholesterol" because it helps remove excess cholesterol from tissues and transport it to the liver for excretion. Measuring HDL cholesterol is important for assessing cardiovascular risk and lipid metabolism. The most commonly used method for HDL cholesterol measurement is the precipitation-based enzymatic assay, which involves the following steps: To isolate HDL cholesterol, non-HDL lipoproteins (e.g. very low-density lipoprotein (VLDL) and low-density lipoprotein (LDL)) are precipitated using a precipitating agent, such as polyethylene glycol (PEG), dextran sulfate, or phosphotungstic acid. These agents selectively bind to non-HDL lipoproteins, leaving HDL in the supernatant. After precipitation, the mixture is centrifuged to separate the precipitate (non-HDL lipoproteins) from the supernatant, which contains HDL cholesterol. The HDL cholesterol in the supernatant is quantified using the same enzymatic method used for total cholesterol measurement. This involves the following reactions: Cholesterol esters in HDL are hydrolyzed to free cholesterol by the enzyme cholesterol esterase. Free cholesterol is oxidized by the enzyme cholesterol oxidase to produce cholest-4-en-3-one and hydrogen peroxide (H_2O_2). The hydrogen peroxide reacts with a chromogenic substrate (e.g. 4-aminoantipyrine and phenol) in the presence of the enzyme peroxidase to form a colored quinoneimine dye. The intensity of the colored quinoneimine dye is measured spectrophotometrically at a wavelength of 500–520 nm. The absorbance is directly proportional to the HDL cholesterol concentration in the serum sample. The HDL cholesterol concentration is determined by

comparing the absorbance to a standard curve prepared with known concentrations of cholesterol.

Measurement of Serum Total Protein

The principle of the assay for measuring total protein in serum is based on the chemical or colorimetric detection of proteins. Total protein measurement is an important diagnostic tool for assessing nutritional status, liver function, kidney function, and various other conditions. The most commonly used methods for total protein measurement are the Biuret method and the dye-binding method (e.g. Bradford assay). Below is an explanation of the Biuret method, which is widely used in clinical laboratories. Proteins in the serum react with alkaline copper sulfate (Biuret reagent) to form a violet-colored complex. This reaction occurs because peptide bonds in proteins (which link amino acids together) react with copper ions (Cu^{2+}) in an alkaline medium. The intensity of the violet color produced is directly proportional to the number of peptide bonds present, and thus to the total protein concentration in the serum sample. The absorbance of the violet-colored complex is measured spectrophotometrically at a wavelength of 540–550 nm. The total protein concentration is determined by comparing the absorbance of the sample to a standard curve prepared with known concentrations of protein (e.g. bovine serum albumin).

Results

The table outlines various biochemical parameters analyzed across three age groups: individuals under 50 years, those aged 50-65 years, and those over 65 years. The results reveal several key trends. Total cholesterol (TC) levels remained consistent across all age groups, showing no significant differences. Similarly, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and the atherogenic index did not exhibit significant variations with age. Urea levels also remained stable across the different age categories.

However, certain parameters demonstrated significant changes with age. Triglycerides (TG) and very low-density lipoprotein cholesterol (VLDL-C) both showed a notable decrease as age increased. In contrast, creatinine levels significantly rose with advancing age. Other parameters, such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), total protein (TP), and albumin, did not show any significant differences across the age groups.

In summary, while most biochemical parameters remained stable across the age groups, triglycerides (TG), VLDL-C, and creatinine displayed significant age-related changes. These findings highlight specific age-dependent variations in certain metabolic markers (Table 1).

Table 1. The Levels of Biochemical Parameters in Different Age Groups

	Age groups (years)			**p value**
	<50	50-65	>65	
TC, mg/dl	179.35±10.23	178.04±6.50	168.72±8.12	P>0.05
TG, mg/dl	133.57±9.96	127.78±7.46	108.95±7.52	P≤0.01
HDL-C, mg/dl	41.92±0.65	42.77±0.81	42.44±0.74	P>0.05
LDL-C, mg/dl	111.20±9.34	113.28±9.45	104.13±7.88	P>0.05
VLDL-C, mg/dl	26.71±1.99	24.96±1.51	21.79±1.50	P≤0.01
Atherogenic	4.23±0.23	4.19±0.20	3.95±0.23	P>0.05
Urea, mmol/L	5.93±0.90	5.69±0.65	6.19±0.79	P>0.05
Creatinine, μmol/L	94.35±12.60	95.08±7.58	100.90±10.01	P≤0.01
ALT, U/l	18.00±2.70	17.08±1.84	25.36±4.58	P>0.05

AST, U/l	10.85±2.5	9.43±1.96	8.68±1.55	P>0.05
TP, g/dl	5.55±0.2	5,48±0.37	5.41±0.31	P>0.05
Albumin, g/dl	3.94±0.24	3.74±0,28	3.65±0.26	P>0.05

The table outlines various biochemical parameters analyzed across different BMI categories: normal weight (<25), overweight (24.9-30), and obese (>30). The results reveal several notable trends. Total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) both showed significant increases as BMI rose, indicating a clear association between higher BMI and elevated levels of these lipids. In contrast, triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and very low-density lipoprotein cholesterol (VLDL-C) did not exhibit significant differences across the BMI groups, suggesting that these parameters remain relatively stable regardless of weight category.

Similarly, other markers such as the atherogenic index, urea, creatinine, alanine aminotransferase (ALT), and aspartate aminotransferase (AST) did not show significant variations across the BMI groups. This implies that these biochemical measures are not strongly influenced by changes in BMI within the studied population.

In summary, while total cholesterol (TC) and LDL-C levels increased significantly with higher BMI, the other parameters remained consistent across the different weight categories. These findings highlight specific lipid-related changes associated with increasing BMI, while other metabolic markers appear unaffected by weight status (Table 2).

Table 2. The Levels of Biochemical Parameters in Different BMI Groups

	BMI groups (kg/m^2)			**p-value**
	<25	24.9-30	>30	
TC, mg/dl	164.46±8.15	172.09±5029	197.45±13.91	P≤0.05
TG, mg/dl	110.0±8.51	123.63±6.15	134.18±13,91	P>0.05
HDL-C, mg/dl	41.66±0.76	42.69±0.58	42.72±1.28	P>0.05
LDL-C, mg/dl	100.78±8.48	104.86±4.73	134.62±12.95	P≤0.05
VLDL-C, mg/dl	22.00±1.70	24.72±1.23	25.59±2.76	P>0.05
Atherogenic Index	3.93±0.25	4.01±0.13	4.66±0.14	P>0.05
Urea, mmol/L	4.86±0.28	5.79±0.56	7.81±1.46	P>0.05
Creatinine, µmol/L	82.66±4046	96.12±7.30	119.63±18.01	P>0.05
ALT, U/l	17.46±2.21	22.51±2.95	18.00±5.23	P>0.05
AST, U/l	9.93±2.24	22.51±1.38	9.09±3.06	P>0.05

The table compares the levels of various biochemical parameters between smokers and non-smokers. Overall, the results indicate that smokers tend to have higher levels of most biochemical markers compared to non-smokers, although none of these differences reached statistical significance.

For instance, urea levels were higher in smokers (6.3106 mmol/L) than in non-smokers (4.4667 mmol/L), but the difference was not statistically significant (P=0.091). Similarly, creatinine levels were elevated in smokers (101.5957 µmol/L) compared to non-smokers (79.4167 µmol/L), though this difference also lacked statistical significance (P=0.107). Liver enzymes, such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST), were also higher in smokers (21.5319 U/l and 9.9362 U/l, respectively) than in non-smokers (15.9167 U/l and 7.7500 U/l, respectively), but these differences were not statistically significant (P=0.261 and P=0.434, respectively).

In terms of lipid profiles, total cholesterol (Cho) was higher in smokers (178.9787 mg/dl) compared to non-smokers (158.8333 mg/dl), but the difference was not statistically significant (P=0.078). Similarly, triglycerides (Tri), very low-density lipoprotein cholesterol (VLDL), and low-density lipoprotein cholesterol (LDL) were all higher in smokers, though none of these differences reached statistical significance (P=0.416, P=0.476, and P=0.073, respectively). On the other hand, high-density lipoprotein cholesterol (HDL) was slightly lower in smokers (42.3617 mg/dl) compared to non-smokers (42.7500 mg/dl), but this difference was also not statistically significant (P=0.729).

Other parameters, such as total protein and albumin, showed minimal differences between smokers and non-smokers, with total protein being slightly higher in smokers (5.488 g/dl vs. 5.457 g/dl) and albumin slightly lower in smokers (3.759 g/dl vs. 3.814 g/dl). However, these differences were not statistically significant (P=0.83 and P=0.69, respectively) (Table 3).

Table 3. The Levels of Biochemical Parameters Based on Smoking Status

	Smoking	**Mean**	**Std. Error**	**P-value**
Urea (mmol/L)	Yes	6.3106	0.529	0.091
	No	4.4667	0.392	
Cr (µmol/L)	Yes	101.5957	6.651	0.107
	No	79.4167	50870	
ALT (U/l)	Yes	21.5319	2.428	0.261
	No	15.9167	2.097	
AST (U/l)	Yes	9.9362	1.338	0.434
	No	7.7500	1.557	
Cho (mg/dl)	Yes	178.9787	5.363	0.078
	No	158.8333	6.898	
Tri (mg/dl)	Yes	124.1277	5.459	0.416
	No	114.3333	10.087	
HDL (mg/dl)	Yes	42.3617	0.482	0.729
	No	42.7500	0.678	
VLDL (mg/dl)	Yes	24.5340	1.094	0.476
	No	22.8667	2.013	
LDL (mg/dl)	Yes	113.3553	5.190	0.073
	No	93.8000	5.604	
Total protein	Yes	5.488	0.331	0.83
	No	5.457	0.305	
Albumin	Yes	3.759	0.302	0.69
	No	3.814	0.313	

Discussion

The current study, along with previous research, underscores the significance of the prolidase enzyme in cancer biology. Its elevated activity in bladder, breast, and prostate cancers suggests a potential role as a biomarker and a therapeutic target. Furthermore, the enzyme's involvement in ECM remodelling and integrin-mediated interactions highlights its contribution to tumour progression and invasion. Future research should focus on elucidating the molecular mechanisms underlying prolidase enzyme activity and exploring its therapeutic potential in cancer treatment.

The current study demonstrates that the effectiveness of the prolidase enzyme is significantly higher in the serum of bladder cancer patients compared to the healthy group**.** This finding is consistent with previous studies, which have reported higher prolidase enzyme activity in bladder cancer patients compared to healthy individuals [14. 15]. These studies suggest that prolidase enzyme activity may serve as a potential biomarker for bladder cancer.

A critical factor in this process is collagen hydrolysis, mediated by genetically regulated collagenases, which play a vital role in the development of the metastatic phenotype. These enzymes break down the extracellular barriers of cells, enabling cancer to spread through tissues. While exogenous collagenases initiate this degradation, the final step is driven by the enzyme prolidase [16]. Research has shown a significant reduction in collagen levels within the tissues of cancer patients, accompanied by a corresponding increase in prolidase activity. This reflects the extensive tissue remodelling associated with the invasive behaviour of cancer cells. The interaction between collagen hydrolysis and elevated prolidase activity promotes the lysis of the stromal matrix, creating pathways for cancer cells to spread [17].

In general, urinary tract cancers, such as bladder and prostate cancer, are strongly linked to aging. Oxidative damage, which is associated with the aging process, has been identified as a contributing factor [18]. Age is a significant risk factor for bladder cancer, with 90% of cases in the United States diagnosed in individuals over the age of 55, and 80% occurring in those aged 65 and older. The average age at diagnosis for bladder cancer in the U.S. is 73 years [19], indicating that the majority of bladder cancer patients are over the age of 50 years, a finding consistent with other previously published studies [20, 21]. Additionally, Messing (2008) emphasized that age is a highly indicative and predominant risk factor for cancer [22]. The results in the table below demonstrate the influence of age on certain biochemical variables studied in both bladder cancer patients and healthy individuals.

The results of this study also revealed significant differences in several key parameters, including creatinine, body mass index (BMI), triglycerides, and very low-density lipoprotein (VLDL). These findings align with previous research, which highlighted similar trends [23]. Specifically, the variation in creatinine levels from the normal range is influenced by factors such as age, muscle mass, weight, and sex. Additionally, a new study emphasized that elevated triglyceride levels in bladder cancer patients, particularly with advancing age, contribute to alterations in the physiological hormonal balance [24]. These changes may ultimately increase the risk of cancer development. Together, these findings underscore the complex interplay between metabolic factors, aging, and cancer risk, as reflected in the observed differences in biochemical parameters.

The study examined the effect of body mass index (BMI) on various biochemical variables in bladder cancer patients. The patients were categorized into three groups based on their BMI: less than 24.9 kg/m^2, 24.9-29.9 kg/m^2, and 30 kg/m^2 or more. Notably, approximately 81.3% of bladder cancer patients were overweight or obese, with a BMI exceeding 25 kg/m^2. This finding is significant, as weight gain during youth and its persistence into older age has been independently linked to an increased risk of developing cancer later in life [25].

The study further revealed a significant rise in total cholesterol and low-density lipoprotein (LDL) levels as BMI increased, with these changes reaching a probability level of $p<0.05$ in bladder cancer patients. This aligns with a previously demonstrated study that obesity is closely associated with dyslipidemia, a

condition characterized by abnormal lipid levels, including elevated LDL cholesterol, which is often linked to weight gain [26]. Additionally, it has been found that prolidase activity, an enzyme involved in collagen metabolism, is positively correlated with BMI, further underscoring the metabolic changes associated with increased body weight [15].

The study highlights the significant impact of BMI on biochemical variables in bladder cancer patients, particularly the rise in cholesterol and LDL levels with increasing BMI. These findings are consistent with previous research linking obesity, dyslipidemia, and cancer risk, emphasizing the importance of weight management in reducing the likelihood of developing cancer and its associated metabolic complications.

Smoking is recognized as one of the strongest risk factors for bladder cancer, accounting for approximately 50-65% of all cases [27]. The pattern of smoking, including the number of cigarettes smoked per day and the duration of smoking, plays a significant role in increasing the risk of bladder cancer [28]. Furthermore, smoking is associated with a higher frequency of bladder cancer and a reduction in the effectiveness of intra-bladder treatments [29].

The carcinogenic effects of smoking are primarily attributed to the presence of aromatic amines and polycyclic aromatic hydrocarbons in cigarette smoke. These harmful substances can damage the DNA in the epithelium of the urinary tract, leading to changes in the urine environment. Additionally, tobacco carcinogens can transform into active compounds capable of interacting with DNA, further promoting carcinogenic processes. It has been emphasized that cigarette smoke possesses significant potential to induce cancer, underscoring the direct link between smoking and bladder cancer development [30, 31].

Preliminary data from the study reveal that 80% of bladder cancer patients were smokers. This high percentage strongly suggests that smoking has a substantial impact on the development of bladder cancer. The findings align with existing research, reinforcing the critical role of smoking as a major contributor to bladder cancer risk. These insights highlight the importance of smoking cessation programs and public health interventions to reduce the prevalence of bladder cancer and improve treatment outcomes.

Conclusion

In conclusion, this study highlights significant variations in biochemical parameters across different age groups, BMI categories, and smoking status, providing valuable insights into metabolic health and disease risk. Age was found to significantly influence triglyceride (TG), VLDL-C, and creatinine levels, underscoring the necessity of age-specific metabolic assessments. Higher BMI was associated with adverse lipid profiles, particularly elevated total cholesterol (TC) and LDL-C, reinforcing the critical role of weight management in maintaining cardiovascular health. While smoking showed trends of elevated levels in several biochemical parameters, including urea, creatinine, ALT, AST, TC, TG, VLDL, and LDL, these differences were not statistically significant, indicating a need for further research to confirm these observations. Overall, the findings emphasize the importance of considering age, BMI, and lifestyle factors when evaluating metabolic health and disease risk, providing a foundation for targeted interventions and personalized healthcare strategies.

Acknowledgements

The authors are very grateful to the University of Mosul, College of Science, for the provision of their facilities, which helped to improve the quality of this study.

Conflict of Interest

The authors declare no conflict of interest.

References

[1]. Nautiyal, M., Ganapathy, D., Ameya, K. P., Sekar, D., 2024, Analysis of Carboplatin and STAT3 in the Breast Cancer MCF7 Cell Line. *Texila International Journal of Public Health*, 12(2):1-10, doi:10.21522/TIJPH.2013.12.02. Art026.

[2]. Ravikumar, L., Velmurugan, R., Hashvitha, P., Subhashini, K., Sheshan, A., Subburam, R., Gnanaprakash K., 2024, Flavonoid Nanoparticles: Revolutionizing Cancer Treatment Strategies. *Texila International Journal of Public Health*, 12(04):1-9, doi:10.21522/TIJPH.2013.12.04. Art051.

[3]. Thoke, G. M., Usman P, P., Ganapathy, D., Sekar, D., 2024, Analysis of TGF-β Gene Expression in Carboplatin Treated Lung Cancer Cells. *Texila International Journal of Public Health*,12(04):1-10, doi:10.21522/TIJPH.2013.12.04. Art094.

[4]. Abed, W. T., Alabbas, E. A. K., Hawass, A. A., 2025, Estimating of the Levels of Some Biochemical Variables, Adipokines and Cytokines in Women with Polycystic Ovarian Syndrome. *J Nat Sc Biol Med*, 16(1):90–7. doi: 10.4103/jnsbm.JNSBM_16_1_10.

[5]. Rani, V., Amudha, P., Vidya, R., Jayalakshmi, M., Kalpana, C. S., 2024, Molecular Docking Analysis of 9-Octadecene, 9,12,15-Octadecatrienoic acid, Methyl Ester, Phytol, 9,12-Octadecadienoic Acid and 9-Octadecenoic Acid with Anticancer Target Enzyme Caspase 3 (PDB: 1CP3). *Texila International Journal of Public Health*, 12(04):1-14. doi:10.21522/TIJPH.2013.12.04. Art097.

[6]. Moulazadeh, A., Ranjbar, R., Kouhpayeh, S. A., Najafipour, S., 2024, The Effect of Different Polar Solvents on the Extraction of Bioactive Compounds in Ferula assafoetida and Subsequent Cytotoxic Effects. *Physiology and Pharmacology*, 28(4), 476-485. doi:10.61186/phypha.28.4.476.

[7]. Digala, P., Muthu, S., Subramani, N., Duraisamy, N., Sundararaj, D., 2024, Understand the Fatty Acid Metabolic Reprogramming of Immune Cells in Colorectal Cancer. *Texila International Journal of Public Health*, 12(3): 1-8, doi:10.21522/TIJPH.2013.12.03. Art017.

[8]. Mohammed, M. H., Mohammad, J. A., IZBA, S. H., 2025, Serum Resistin, Vitamin D, and Vitamin E levels in Patients with Mild and Moderate Psoriasis. *J Nat Sc Biol Med*, 16(1):75–81. doi: 10.4103/jnsbm.JNSBM_16_1_8.

[9]. Anbu, P., Vadhana, T. I. M., Saravanabhavan, S. S., Kumar, S. T., 2024, Nano-materials in Drug Delivery Systems for Oral Cancer Treatment. *Texila International Journal of Public Health*, 12(3):1-6, doi:10.21522/TIJPH.2013.12.03. Art052.

[10]. Kumar, R. S., Amudha, P., Vidya, R., Kalpana, C. S., Sudhashini, S., 2024, A Review on Anticancer Properties of Chebulagic Acid from Terminalia chebula. *Texila International Journal of Public Health*, 12(3): 1-12, doi:10.21522/TIJPH.2013.12.03. Art047.

[11]. Zbaar, S., Kamal, I., Alchalabi, A., 2024, Association between serum levels of adipokines in patients with prostate cancer. *Georgian medical news*, (357), 173-177.

[12]. Alsarraf, Z., Nori, A. Y., Oraibi, A. I., Al_hussaniy, H. A., Jabbar, A. A., 2024, BIBR1591 induces apoptosis in breast cancer cell line and increases expression of DAPK1, AND NR4A3. *Cancer*, 9, 11.

[13]. Matsumoto, Y., Hayashi, T., Amano, Y., Abiko, K., Konishi, I., 2024, Development of Endosalpingiosis in Patients with a History of Breast Cancer. *Georgian medical news*, (355), 72-76.

[14]. Gecit, I., Aslan, M., Gunes, M., Pirincci, N., Esen, R., Demir, H., Ceylan, K., 2012, Serum prolidase activity, oxidative stress, and nitric oxide levels in patients with bladder cancer. *Journal of cancer research and clinical oncology*, 138, 739-743. doi:10.1007/s00432-011-1136-4.

[15]. Aslan, M., Duzenli, U., Esen, R., Soyoral, Y. U., 2017, Serum prolidase enzyme activity in obese subjects and its relationship with oxidative stress markers. *Clinica Chimica Acta*, 473, 186-190. doi: 10.1016/j.cca.2017.08.039.

[16]. Kaba, M., Pirincci, N., Demir, H., Verep, S., 2024, Serum prolidase activity, oxidative stress,

and antioxidant enzyme levels in patients with prostate cancer. *In Urologic Oncology: Seminars and Original Investigations*, 42(4): 116-e9, doi: 10.1016/j.urolonc.2024.01.007.

[17]. Belli, S., Demir, H., Ozdemir, B., Demir, C., 2021, Prolidase, Paraoxonase-1, Arylesterase Activity In Oral Squamous Cell Carcinoma. *Eastern Journal of Medicine*, 26(1), doi:10.5505/ejm.2021.39358.

[18]. Kilic-Toprak, E., Toprak, I., Caliskan, S., Ozdemir, Y., Demirtas, O., Altintas, F., Kucukatay, V. 2019, Oxidative stress and genotoxicity in pterygium: a systemic investigation. *Eye & Contact Lens*, 45(6), 399-404. doi:10.1097/ICL.0000000000000620.

[19]. Siegel, R. L., Miller, K. D., Jemal, A., 2019, Cancer statistics, 2019. *CA: a cancer journal for clinicians*, 69(1), 7-34. doi:10.3322/caac.21551.

[20]. Richters, A., Aben, K. K., Kiemeney, L. A., 2020, The global burden of urinary bladder cancer: an update. *World journal of urology*, 38(8), 1895-1904. doi:10.1007/s00345-019-02984-4.

[21]. Plouvier, S. D., Bonnal, J. L., Machuron, F., Colin, P., Vankemmel, O., Leroy, X., Pasquier, D., 2020, Impact of age on bladder cancer management practices: a general population study. *Acta Oncologica*, 59(4), 462-466. doi:10.1080/0284186X.2020.1723164.

[22]. Messing, E., Gee, J. R., Saltzstein, D. R., Kim, K., diSant'Agnese, A., Kolesar, J., Bailey, H., 2012, A phase 2 cancer chemoprevention biomarker trial of isoflavone G-2535 (genistein) in presurgical bladder cancer patients. *Cancer prevention research*, 5(4), 621-630. doi: 10.1158/1940-6207.CAPR-11-0455.

[23]. Kamińska, J., Dymicka-Piekarska, V., Tomaszewska, J., Matowicka-Karna, J., Koper-Lenkiewicz, O. M., 2020, Diagnostic utility of protein to creatinine ratio (P/C ratio) in spot urine sample within routine clinical practice. *Critical reviews in clinical laboratory sciences*, 57(5), 345-364. doi:10.1080/10408363.2020.1723487.

[24]. Feng, H., Deng, Z., Huang, Y., Liu, Z., Ruan, Y., Wang, T., Liu, J., 2023, A novel cuproptosis pattern and tumor immune microenvironment characterization in urothelial carcinoma of the bladder. *Frontiers in Immunology*, 14, 1219209. doi:10.3389/fimmu.2023.1219209.

[25]. Han, X., Stevens, J., Truesdale, K. P., Bradshaw, P. T., Kucharska-Newton, A., Prizment, A. E., Joshu, C. E., 2014, Body mass index at early adulthood, subsequent weight change and cancer incidence and mortality. *International journal of cancer*, 135(12), 2900-2909. doi:10.1002/ijc.28930.

[26]. Khanna, D., Khanna, S., Khanna, P., Kahar, P., Patel, B. M., 2022, Obesity: a chronic low-grade inflammation and its markers. *Cureus*, 14(2). doi:10.7759/cureus.22711.

[27]. Barsouk, A., Padala, S. A., Vakiti, A., Mohammed, A., Saginala, K., Thandra, K. C., Barsouk, A., 2020, Epidemiology, staging and management of prostate cancer. *Medical sciences*, 8(3), 28. doi:10.3390/medsci8030028.

[28]. Suyanto, J., Sianipar, B. K., Sofais, D. A. R., Panduragan, S. L., 2024, Structured Training Program About Breast Self-Examination Toward Awareness and Behavior Among Women in Bengkulu City, Indonesia. *Public Health of Indonesia*, 10(3), 383-390. doi:10.36685/phi. v10i3.833.

[29]. Soria, J. C., Ohe, Y., Vansteenkiste, J., Reungwetwattana, T., Chewaskulyong, B., Lee, K. H., Ramalingam, S. S., 2018, Osimertinib in untreated EGFR-mutated advanced non–small-cell lung cancer. *New England journal of medicine*, 378(2), 113-125. doi: 10.1056/NEJMoa1713137.

[30]. Ma, W., Zhang, W., Shen, L., Liu, J., Yang, F., Maskey, N., Yao, X., 2021, Can smoking cause differences in urine microbiome in male patients with bladder cancer? A retrospective study. *Frontiers in oncology*, 11, 677605. doi:10.3389/fonc.2021.677605.

[31]. Skulberg, K. R., Hamid, S., Vaktskjold, A., 2019, Smoking among adolescent males at Pulau Weh, Indonesia. *Public Health of Indonesia*, 5(3):62–70.
http://stikbar.org/ycabpublisher/index.php/PHI/index.