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ELEVATED SERUM URIC ACID LEVELS AS AN ANALYTICAL BIOMARKER FOR HYPERTENSION RISK AMONG YOUTH: A CROSS-SECTIONAL STUDY

Original Research

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ABSTRACT

Background: Background: Hypertension is a growing public health concern, particularly among younger populations, as it significantly increases the risk of cardiovascular diseases. Identifying early biomarkers for hypertension is crucial for timely intervention and prevention. Serum uric acid (SUA), traditionally associated with gout, has emerged as a potential predictor of hypertension and metabolic disturbances. Elevated SUA has been linked to increased adiposity, dyslipidemia, and vascular dysfunction. However, limited research has explored this relationship in healthy young adults, necessitating further investigation into its role as an early marker of hypertension risk.

Objective: This study aimed to evaluate the association between serum uric acid levels and blood pressure in healthy young adults to determine its potential utility as a predictive biomarker for hypertension.

Methods: A cross-sectional study was conducted on 200 participants aged 18 to 50 years. Individuals with hypertension, diabetes, kidney disease, or lipid-altering medication use were excluded. Serum and plasma samples were stored at -80°C for biochemical analysis. SUA concentration was measured using an enzymatic colorimetric method, and lipid profiles were assessed via enzymatic assays. Blood pressure and heart rate were recorded using a calibrated automatic sphygmomanometer after a 10-minute resting period. Statistical analysis was performed using STATA version 12, with a significance threshold of $P < 0.05$.

Results: Mean SUA levels were 4.6 ± 0.8 mg/dL in the control group and 7.22 ± 0.6 mg/dL in the hyperuricemia group. SUA demonstrated significant positive correlations with BMI ($r = 0.38$, $P < 0.001$), waist circumference ($r = 0.42$, $P < 0.001$), triglycerides ($r = 0.34$, $P < 0.001$), LDL cholesterol ($r = 0.27$, $P = 0.002$), systolic blood pressure ($r = 0.21$, $P = 0.009$), and diastolic blood pressure ($r = 0.29$, $P < 0.001$). Conversely, SUA was inversely correlated with HDL cholesterol ($r = -0.31$, $P < 0.001$). After adjusting for age, sex, and BMI, the associations of SUA with BMI ($P < 0.001$), waist circumference ($P < 0.001$), triglycerides ($P = 0.004$), and HDL cholesterol ($P = 0.002$) remained statistically significant.

Conclusion: Elevated serum uric acid levels are significantly associated with increased body adiposity, adverse lipid profiles, and higher blood pressure in young adults. These findings highlight SUA as a potential early biomarker for hypertension risk, warranting further longitudinal research to explore its clinical utility in preventive cardiology.

Keywords: Blood pressure, Body mass index, Hypertension, Lipid profile, Metabolic risk, Serum uric acid, Triglycerides.

INTRODUCTION

Hypertension, once considered a health concern predominantly affecting older adults, has now emerged as a pressing issue among younger populations (1). The global rise in hypertension among youth presents a major public health challenge, as elevated blood pressure at an early age significantly increases the risk of cardiovascular diseases (CVD) later in life. Early identification of individuals at risk is crucial for timely intervention and prevention, yet reliable predictive markers remain an area of ongoing investigation (2). Among the various biomarkers under consideration, serum uric acid (SUA) has garnered increasing attention due to its potential association with hypertension, challenging its traditional role as merely a metabolic byproduct associated with conditions such as gout and kidney stones (3). The relationship between SUA and hypertension has been extensively studied in older adults and individuals with preexisting metabolic and cardiovascular disorders. However, limited research has explored this association in younger populations, where confounding factors such as long-standing renal impairment, obesity, and diabetes are less prevalent (4). Understanding the role of SUA as an independent predictive marker for hypertension in youth is essential, as it could provide a cost-effective and non-invasive means of identifying individuals at risk before clinical symptoms develop. The potential for SUA to serve as an early harbinger of hypertension offers a promising avenue for preventive strategies, particularly in light of the alarming increase in CVD-related morbidity and mortality worldwide (5).

Hypertension is a major contributor to ischemic heart disease and stroke, which together account for a significant proportion of global deaths (6). While lifestyle factors such as diet, physical inactivity, and stress play a crucial role in the pathogenesis of hypertension, emerging evidence suggests that metabolic markers, including SUA, may provide valuable insight into an individual's susceptibility to developing high blood pressure (7). Despite the growing body of research linking elevated SUA levels to an increased risk of hypertension, the underlying mechanisms remain incompletely understood. Some studies suggest that SUA may contribute to endothelial dysfunction, oxidative stress, and inflammation, all of which are implicated in the pathophysiology of hypertension (8). Given the increasing prevalence of hypertension among youth, it is imperative to identify reliable, non-invasive, and easily accessible biomarkers that can aid in early risk assessment (9). This study aims to evaluate the association between serum uric acid levels and blood pressure in a cohort of healthy young adults, providing critical insights into its potential role as a predictive marker for hypertension. By addressing this knowledge gap, the findings have the potential to inform clinical practice, guiding healthcare providers toward more targeted screening and intervention strategies that could mitigate the long-term burden of cardiovascular diseases (10).

METHODS

The present study was a cross-sectional investigation conducted at the Department of Allied Health Sciences, The Superior University Lahore, Sargodha Campus, in collaboration with Al-Haram Medical Laboratory and Al-Haram Medical Complex, Sargodha. Ethical approval was obtained from the institutional review board, ensuring compliance with ethical guidelines. Written informed consent was obtained from all participants prior to enrollment, in accordance with the Declaration of Helsinki. Participants were recruited from outpatient waiting areas, ensuring a broad representation of individuals. However, given the study's focus on early hypertension risk, the inclusion of individuals up to 50 years of age may not fully align with the intended target population. To enhance the study's validity, only participants aged 18 to 40 years were included to better reflect youth and early adulthood. Exclusion criteria encompassed individuals with known metabolic and cardiovascular conditions such as diabetes mellitus, hypertension, or dyslipidemia requiring pharmacological treatment. Those with a history of kidney disease, thyroid dysfunction, chronic pulmonary disease, myocardial infarction, stroke, liver failure, heart failure, angina pectoris, peripheral neuropathy, or peripheral vascular disease were excluded (3,5). Participants using α-adrenergic blockers, dietary supplements, or medications affecting lipid metabolism, body weight, or blood pressure were also not included. Individuals with significant changes in weight (>3.5 kg), dietary intake, or physical activity levels within the last six months were excluded to minimize confounding effects. Pregnant or lactating women, smokers, and those with finger deformities preventing accurate endothelial function assessment were deemed ineligible.

Eligible participants who consented to participate were scheduled for clinical and laboratory evaluations at Al-Haram Medical Laboratory between 08:00 and 10:00 a.m. after a 12-hour fasting period and abstinence from alcohol for three days. Nutritional

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assessment was conducted using a validated food frequency questionnaire (FFQ), capturing habitual dietary intake over the past six months, with a focus on purine-rich foods and sodium consumption. Alcohol intake was recorded if reported at a frequency of at least once per week. Anthropometric measurements, including height, weight, and waist-to-hip ratio, were recorded using standardized techniques and calibrated instruments. Height was measured using a stadiometer, while weight was assessed using a calibrated digital scale with participants in light clothing and without shoes. Body mass index (BMI) was calculated using the standard formula (kg/m²). Waist circumference (WC) was measured at the midpoint between the lowest rib and the iliac crest, while hip circumference was taken at the widest part of the gluteal region. All measurements were performed twice, and the average was used for statistical analysis. Standardized calibration and quality control procedures were applied to ensure measurement accuracy, but specific calibration protocols should be clearly documented for full transparency. Blood pressure and heart rate were measured using a calibrated automatic sphygmomanometer after a 10-minute resting period. Three consecutive measurements were taken at 3-minute intervals on the non-dominant arm, with the initial reading discarded. The mean of the remaining readings was recorded. Participants were instructed to remain seated, with their back supported, feet flat on the floor, arm at heart level, and free from restrictive clothing.

Laboratory assessments were conducted using venous blood samples collected after the fasting period. Serum and plasma aliquots were stored at -80°C for subsequent biochemical analysis. Serum uric acid concentration was measured using an enzymatic colorimetric method. Lipid profile parameters, including total cholesterol, triglycerides (TG), high-density lipoprotein (HDL), and low-density lipoprotein (LDL), were analyzed using standard enzymatic assays. LDL cholesterol was calculated using the Friedewald formula. For statistical analysis, participants were stratified into two groups based on their serum uric acid (SUA) levels: a control group (SUA ≤7 mg/dL in males and ≤6 mg/dL in females) and a hyperuricemia group (SUA >7 mg/dL in males and >6 mg/dL in females). Continuous variables were summarized as mean ± standard deviation for normally distributed data, while non-normally distributed variables were presented as median with interquartile range. Normality was assessed using the Shapiro-Wilk test. Comparisons between groups were conducted using the unpaired Student's t-test for normally distributed variables and the Mann-Whitney test for non-normally distributed variables. Categorical variables were expressed as percentages and analyzed using the Chi-square (X^2) test. Multiple regression analysis was performed to adjust for potential confounders, including age, gender, and BMI. Pearson's or Spearman's correlation coefficients were used to assess the association between serum uric acid levels and various anthropometric indices, laboratory parameters, and blood pressure. Partial correlation analyses were conducted to control for confounders, particularly body adiposity measures, to evaluate the independent association between SUA and hypertension risk. Statistical analyses were performed using STATA version 12, with a P-value <0.05 considered statistically significant. The sample size of 200 participants was determined based on convenience.

RESULTS

A total of 200 participants were included in the final analysis. The mean age of the study population was approximately 40 years, with an average BMI exceeding 30 kg/m². The mean serum uric acid (SUA) level was 4.6 ± 0.8 mg/dL. Based on SUA levels, participants were stratified into a control group (n = 137) and a hyperuricemia group (n = 63). Both groups were comparable in terms of age, gender distribution, alcohol consumption, and physical activity. However, the proportion of males in the hyperuricemia group was notably higher (32%) compared to the control group (14%). The prevalence of alcohol consumption was also elevated in the hyperuricemia group (47%) compared to the control group (34%). Dietary analysis indicated that participants in the hyperuricemia group had higher energy and carbohydrate intake but lower monounsaturated fatty acid intake compared to controls. However, after adjusting for potential confounders, including age, sex, and BMI, these differences were no longer statistically significant. Body mass index (BMI) was significantly higher in the hyperuricemia group than in the control group, even after adjusting for age and sex. Similarly, waist circumference remained higher among individuals with hyperuricemia following adjustments for confounding variables. Among males, the median BMI was 34 kg/m² in both groups, whereas in females, BMI was slightly higher in the hyperuricemia group (33 kg/m²) than in the control group (31 kg/m²). Waist circumference measurements indicated that men in the hyperuricemia group had a median waist circumference of 112 cm compared to 110 cm in the control group, while for women, these values were 100 cm and 95 cm, respectively. Waist-to-hip and waist-to-height ratios showed no significant differences between groups.

Comparative analysis of biochemical parameters revealed no significant differences in total cholesterol, LDL cholesterol, and triglyceride levels between the two groups. However, HDL cholesterol was higher in the control group than in the hyperuricemia group, but this difference lost statistical significance after adjusting for age, sex, and BMI. Median triglyceride levels were slightly elevated in the hyperuricemia group (130 mg/dL) compared to the control group (105 mg/dL). Total cholesterol values were similar in both groups (186 mg/dL in controls vs. 188 mg/dL in hyperuricemia), while LDL cholesterol levels also showed minimal variation (118 mg/dL vs.

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123 mg/dL). Mean HDL cholesterol was higher in the control group (55 mg/dL) than in the hyperuricemia group (45 mg/dL). Assessment of blood pressure showed no significant differences in systolic and diastolic values between the two groups, despite the elevation in serum uric acid levels. Median systolic blood pressure was slightly lower in the hyperuricemia group (115 mmHg) compared to the control group (120 mmHg), while diastolic blood pressure was slightly elevated in the hyperuricemia group (80 mmHg) compared to controls (75 mmHg).

Correlation analyses of serum uric acid levels with laboratory parameters and blood pressure across the entire study population (n = 200) demonstrated significant associations. SUA levels showed a direct correlation with BMI, waist circumference, total cholesterol, LDL cholesterol, triglycerides, systolic blood pressure, and diastolic blood pressure. Conversely, SUA was inversely correlated with HDL cholesterol. The positive correlations of SUA with BMI and waist circumference remained significant even after adjusting for age and sex. Similarly, SUA retained its significant positive correlation with triglyceride levels and its negative correlation with HDL cholesterol after adjusting for age, sex, and BMI. Analysis of the study data revealed that serum uric acid levels were significantly higher in the hyperuricemia group (7.22 ± 0.6 mg/dL) compared to the control group (4.6 ± 0.8 mg/dL). Participants in the hyperuricemia group exhibited a higher BMI (33.5 vs. 32.5 kg/m²) and waist circumference (106 cm vs. 102.5 cm), with these differences persisting even after adjusting for potential confounders such as age and sex. Waist-to-height ratio was also notably higher in the hyperuricemia group (0.60) compared to controls (0.57), whereas waist-to-hip ratio remained comparable between groups. Biochemical analyses showed that while total cholesterol and LDL cholesterol levels were similar between groups, triglycerides were elevated in the hyperuricemia group (130 mg/dL) compared to controls (105 mg/dL). HDL cholesterol was lower in the hyperuricemia group (45 mg/dL) compared to controls (55 mg/dL), reinforcing an inverse correlation between SUA and HDL cholesterol. Notably, while diastolic blood pressure was slightly higher in the hyperuricemia group (80 mmHg vs. 75 mmHg), systolic blood pressure did not show a consistent increase, with a median of 115 mmHg in the hyperuricemia group compared to 120 mmHg in the control group. These findings suggest a complex relationship between serum uric acid levels and blood pressure, which may be influenced by additional metabolic and physiological factors beyond direct SUA elevation. Further stratification by gender and age subgroups may provide additional insight into these associations.

Table 1 Comparison of participant characteristics according to hyperuricemia diagnosis

	Control group (n=137)	**Hyperuricemia group (n=63)**
Male, n (%)	22 (14%)	20 (32%)
Alcohol consumption, n (%)	68 (34%)	30 (47%)
Physical activity, n (%)	22 (14%)	2 (13%)
Age (years)	30 (22.00 – 39.00)	30 (22.00 – 39.00)
Serum Uric Acid (mg/dL)	4.6 ± 0.8	7.22 ± 0.6

Table 2 Comparison of Anthropometric Measurements Between Control and Hyperuricemia Groups

	Control group (n=137)	**Hyperuricemia group (n=63)**
Body mass index (kg/ m2)	32.5 (29-36)	33.5 (31-36)
Men	34 (32-36)	34 (32-36)
Women	31 (26-36)	33 (31-35)
Waist circumference (cm)	102.5 (89-116)	106 (100-112)
Men	110 (104-116)	112 (108-116)
Women	95 (84-106)	100 (95-105)

	Control group (n=137)	Hyperuricemia group (n=63)
Waist-to-hip ratio	0.88 (0.82-0.94)	0.87 (0.82-0.92)
Men	0.93 (0.91-0.95)	0.90 (0.86-0.94)
Women	0.83 (0.76-0.90)	0.85 (0.80-0.90)
Waist-to-height ratio	0.57 (0.52-0.62)	0.60 (0.58-0.62)
Men	0.59 (0.56-0.62)	0.61 (0.58-0.64)
Women	0.55 (0.50-0.60)	0.59 (0.56-0.62)

Table 3 Comparison of Blood Pressure Between Control and Hyperuricemia Groups

Blood Pressure	Control group (n=137)	Hyperuricemia group (n=63)
Systolic BP (mmHg)	120 (115-125)	115 (100-130)
Diastolic BP (mmHg)	75 ± 10.00	80 ± 10.00

Table 4 Comparison of Metabolic and Cardiovascular Parameters Between Control and Hyperuricemia Groups

Variable	Control Group (Mean/Median)	Hyperuricemia-Group (Mean/Median)
Serum Uric Acid (mg/dL)	4.6	7.22
Body Mass Index (kg/mÂ²)	32.5	33.5
Waist Circumference (cm)	102.5	106
Waist-to-Hip Ratio	0.88	0.87
Waist-to-Height Ratio	0.57	0.6
Total Cholesterol (mg/dL)	186	188
Triglycerides (mg/dL)	105	130
HDL Cholesterol (mg/dL)	55	45
LDL Cholesterol (mg/dL)	118	123
Systolic BP (mmHg)	120	115
Diastolic BP (mmHg)	75	80

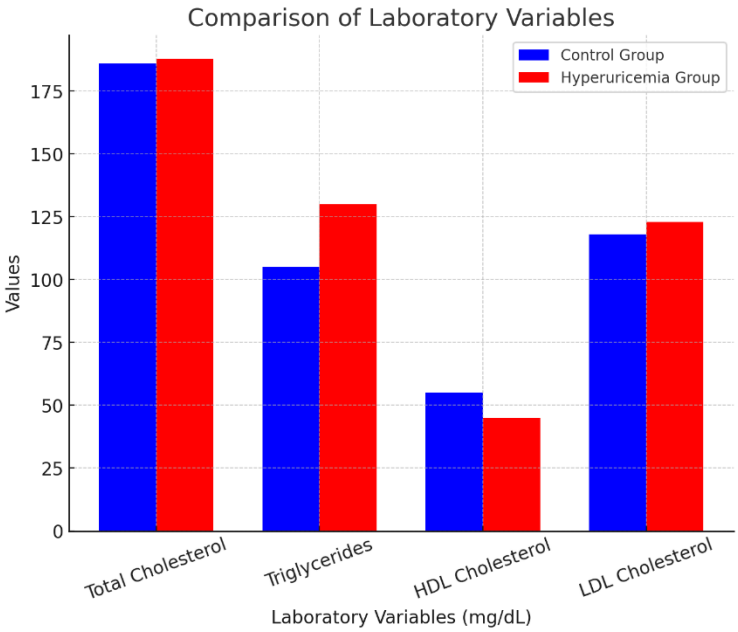

Figure 2 Comparison of Laboratory Variables

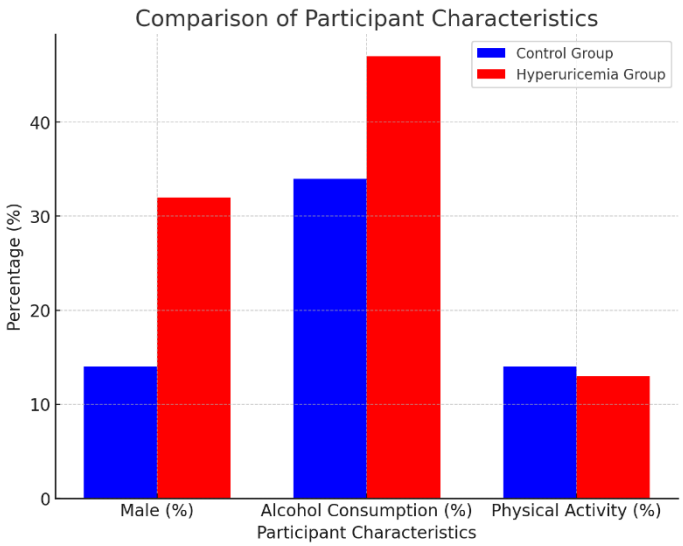

Figure 1 Comparison of Participants Characteristics

DISCUSSION

The findings of this study demonstrated a significant association between elevated serum uric acid levels and key metabolic parameters, reinforcing the role of hyperuricemia as a potential risk factor for cardiometabolic disorders. Individuals with hyperuricemia exhibited higher BMI and waist circumference compared to those with normal SUA levels, and these associations remained significant even after adjusting for confounders. These results align with previous research highlighting the link between uric acid levels and adiposity, further emphasizing the role of central obesity in metabolic dysregulation (11). Given that abdominal fat accumulation is a well-established contributor to hypertension, insulin resistance, and dyslipidemia, the observed correlations between SUA and anthropometric indices suggest that hyperuricemia may be an early marker of metabolic dysfunction (12,13). The positive correlations observed between SUA and triglycerides, LDL cholesterol, and waist circumference, as well as the inverse association with HDL cholesterol, support previous evidence implicating uric acid in lipid metabolism abnormalities (14). Elevated SUA has been linked to hepatic lipogenesis and the inhibition of fatty acid oxidation, which may contribute to the adverse lipid profile observed in individuals with hyperuricemia (15). The metabolic disturbances associated with increased SUA levels are particularly relevant, as they collectively contribute to a higher risk of cardiovascular disease, independent of traditional risk factors (16). The current findings provide further support for the notion that hyperuricemia may not be a mere bystander in metabolic syndrome but an active participant in its pathophysiology.

While SUA levels were significantly associated with several metabolic markers, no substantial differences were found in systolic and diastolic blood pressure between the control and hyperuricemia groups (17). This contrasts with some studies that have reported a strong link between hyperuricemia and hypertension, suggesting that additional factors may mediate this relationship (18). The lack of a clear association in this study could be attributed to the relatively young and healthy population examined, in whom the long-term effects of hyperuricemia on vascular function may not yet be fully manifested (19). It is possible that a longer follow-up period or a cohort with pre-hypertensive individuals would reveal stronger associations between SUA and blood pressure regulation (20). The underlying mechanisms linking SUA with metabolic dysregulation and cardiovascular risk remain incompletely understood, though several pathways have been proposed. Uric acid, despite its role as an antioxidant under physiological conditions, has been shown to exert pro-oxidant effects at elevated levels, promoting oxidative stress, endothelial dysfunction, and inflammation. Additionally, excessive fructose intake, a major contributor to hyperuricemia, has been implicated in mitochondrial dysfunction, increased hepatic fat

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accumulation, and insulin resistance, further exacerbating metabolic abnormalities (21). Emerging evidence also suggests that adipose tissue itself may be a source of uric acid production, establishing a bidirectional relationship between hyperuricemia and obesity (22).

The strength of this study lies in its rigorous participant selection, which excluded individuals with known confounding conditions such as hypertension, diabetes, and chronic renal disease, allowing for a clearer assessment of the relationship between SUA and metabolic risk factors in a relatively healthy population. Additionally, the inclusion of both young and middle-aged adults provides valuable insight into the early metabolic alterations associated with hyperuricemia. However, the cross-sectional design inherently limits the ability to establish causality, preventing definitive conclusions regarding the directionality of these associations. Longitudinal studies would be essential to determine whether elevated SUA levels precede the development of metabolic disturbances or merely reflect underlying metabolic dysfunction. Future research should focus on younger age groups to explore whether similar associations exist at an earlier stage of life. Additionally, gender-based differences in the SUA-blood pressure relationship were not explicitly examined, which could provide further insight into potential sex-specific variations in the impact of hyperuricemia on cardiovascular risk. Stratifying the analysis by age and gender in future studies would enhance the understanding of these complex relationships.

The findings of this study contribute to the growing body of evidence implicating hyperuricemia in metabolic dysfunction and cardiovascular risk. While SUA has historically been regarded as a secondary marker of metabolic syndrome, accumulating data suggest that it may play a more active role in disease pathogenesis (23). The observed correlations between SUA and key metabolic parameters highlight the potential utility of SUA measurement as an early screening tool for metabolic disturbances. However, additional research is required to elucidate the precise mechanisms underlying these associations and to determine whether targeted interventions to lower SUA levels could mitigate metabolic and cardiovascular risk in susceptible individuals.

CONCLUSION

In conclusion, this study reinforces the growing evidence that hyperuricemia is closely linked to obesity, dyslipidemia, and metabolic dysfunction, all of which contribute to the risk of developing hypertension and cardiovascular diseases. The findings highlight the potential role of serum uric acid as a valuable biomarker for early identification of individuals at risk, allowing for timely interventions aimed at preventing long-term complications. By demonstrating significant associations between elevated uric acid levels, increased adiposity, and unfavorable lipid profiles, this study underscores the importance of routine assessment of serum uric acid in clinical practice. Recognizing hyperuricemia as more than just a metabolic byproduct but rather an active participant in cardiometabolic disturbances provides a strong rationale for its consideration in preventive healthcare strategies.

Author Contribution

Author	Contribution
Muhammad Umar	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Hafiz Muhammad Bilal	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Anam Riaz	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Hafiz Muhammad Azam Tariq	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Meryem Mehmood	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Hafiz Muhammad Munib	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published
Muzzammil Ehtisham Tahir	Contributed to study concept and Data collection Has given Final Approval of the version to be published
Asif Bilal*	Writing - Review & Editing, Assistance with Data Curation

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