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ORIGINAL RESEARCH

Elevated Peripheral White Blood Cell Count and Lower Urinary Tract Symptom Risk: A Population-Based Cross-Sectional Study of Chinese Men

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Objective: To investigate the association between peripheral white blood cell (WBC) count and lower urinary tract symptoms (LUTS) in a male population.

Study Design and Setting: A population-based cross-sectional survey was conducted among males aged 18–88 years in Fangchenggang, Guangxi, China, from July to November 2011. Using multistage cluster sampling, 4,694 participants were enrolled. Multivariable logistic regression models assessed the WBC-LUTS association.

Results: The prevalence of moderate/severe LUTS was 6.67%. Compared to men with none/mild LUTS, those with moderate/severe LUTS had significantly higher mean WBC counts ($P<0.01$). Elevated WBC levels were independently associated with increased risks of overall LUTS (OR=1.93; 95% CI: 1.40–2.66) and voiding symptoms (OR=1.68; 95% CI: 1.19–2.35) after adjusting for confounders. Specifically, higher WBC quartiles (Q3–Q4) showed significantly greater prevalence of intermittency and urgency versus the lowest quartile (Q1). Additional risk factors for LUTS included age, hypertension, lower education, diabetes mellitus, and alcohol consumption.

Conclusion: LUTS severity positively correlates with peripheral WBC counts, suggesting systemic inflammation may contribute to LUTS pathogenesis.

Keywords: lower urinary tract symptoms, systemic inflammation, voiding symptoms, storage symptoms, epidemiology, cross-sectional study

Introduction

Lower urinary tract symptoms (LUTS), encompassing storage, voiding, and post-micturition dysfunction, represent a prevalent clinical syndrome affecting both male and female populations globally.[1–3] These symptoms, typically quantified by the International Prostate Symptom Score (IPSS), arise from heterogeneous etiologies including detrusor overactivity, bladder outlet obstruction, and benign prostatic hyperplasia (BPH).[4] Such pathophysiological complexity contributes to suboptimal treatment efficacy, often resulting in reduced health-related quality of life (QoL) and significant

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psychological sequelae.[5] Consequently, elucidating the underlying mechanisms of LUTS is imperative for developing targeted therapeutic strategies.

Accumulating evidence implicates inflammatory pathways in LUTS pathogenesis. Preclinical studies demonstrate that acute and chronic inflammation induces prostatic stromal proliferation, detrusor hyperexcitability, and bladder fibrosis.[6–8] Epidemiologically, cross-sectional analyses in Western and Asian populations consistently report a positive correlation between elevated serum C-reactive protein (CRP)—a marker of systemic inflammation—and LUTS severity.[9–13] For instance, a 2025 cohort study of 1,167 multiple sclerosis patients revealed that neurogenic inflammation accelerates LUTS onset, with higher baseline disability scores (EDSS) increasing urgency risk (HR=1.27, P<0.001).[14]

Despite established links between CRP and LUTS, the role of peripheral white blood cell count (WBC)—a biomarker of acute inflammation—remains underexplored. Notably, CRP and WBC exhibit non-concordant kinetics in inflammatory responses; WBC elevations may reflect transient infections or tissue injury, while CRP signifies sustained systemic inflammation.[15–17] This divergence was highlighted in a 2025 randomized trial (N=237), where app-based LUTS management significantly reduced IPSS scores but not WBC levels, suggesting distinct inflammatory drivers.[18]

To address this knowledge gap, we leveraged data from the Second Fangchenggang Area Male Health and Examination Survey (FAMHES II). Our objectives were twofold: To determine the association between peripheral WBC count and overall LUTS severity. To evaluate correlations between WBC levels and specific symptom domains (storage vs. voiding) as defined by IPSS. This study provides novel insights into acute inflammatory processes in LUTS, potentially informing early intervention strategies for high-risk subgroups.

Materials and Methods

Overall Design and Study Population

Study Design: The Fangchenggang Area Male Health and Examination Survey (FAMHES) constitutes a large-scale population-based epidemiologic cohort in Guangxi, China. This ongoing study investigates interactions among environmental, genetic, and lifestyle factors in age-related chronic diseases, with biennial follow-ups planned. The first phase (FAMHES I) was conducted from September to December 2009, and its initial study protocol had been published in a peer-reviewed journal.[19]

Participant Recruitment: During the second phase (FAMHES II; July–November 2011), the cohort was expanded from 4,303 to 5,988 eligible males through multistage cluster sampling, with 3,500 newly enrolled participants. Among these, 5,540 individuals (92.3% response rate) completed structured interviews and biomarker collection. The study complied with the Declaration of Helsinki and received ethical approval from the Institutional Review Board of Guangxi Medical University (Approval No: GXMU20110329). All participants provided written informed consent.

Inclusion/Exclusion Criteria: From the FAMHES II cohort, we excluded participants meeting any of the following criteria: Incomplete questionnaire data or missing WBC/biochemical measurements (n=312); Medical history of hematologic disorders, pelvic/prostate malignancies, or rheumatoid arthritis (n= 193); Abnormal WBC levels (>10.0 × 10^9/L or <4.0 × 10^9/L), indicating potential active infection (n= 241); Current use of medications affecting WBC counts or LUTS assessment (NSAIDs, antibiotics, glucocorticoids; n= 108). After exclusions, 4,694 participants comprised the analytical sample for this study.

Data Collection

All participants were enrolled through a comprehensive health examination program at the Medical Examination Centre of Fangchenggang First People's Hospital. Trained physicians conducted face-to-face interviews using standardized questionnaires to systematically collect demographic characteristics, lifestyle factors, medical history, medication use within the preceding 30 days, and detailed urological symptom profiles. Anthropometric measurements including height, weight, and waist circumference were obtained following established protocols with calibrated instruments. Fasting venous blood samples (15 mL) were collected between 08:00 and 10:00 AM for subsequent laboratory analysis.

Assessment of Lower Urinary Tract Symptoms

Lower urinary tract symptoms were evaluated using the validated Chinese version of the International Prostate Symptom Score (IPSS). The IPSS questionnaire comprises seven symptom items (incomplete emptying, frequency, intermittency, urgency, weak stream, straining, and nocturia), each scored from 0 to 5, yielding a total score range of 0–35. An eighth item assesses quality of life (QoL) on a 6-point scale. For analytical purposes, moderate/severe overall LUTS was defined as IPSS ≥8. Voiding symptoms (sum of incomplete emptying, intermittency, weak stream, and straining) were classified as significant at ≥5 points, while storage symptoms (sum of frequency, urgency, and nocturia) used a threshold of ≥4 points.[20] Individual symptoms were considered moderate/severe at scores ≥3, with clinically relevant nocturia defined as ≥2 nightly voids.[9] QoL scores were dichotomized into "satisfied" (0–3) and "dissatisfied" (4–6).

Laboratory Procedures

Fasting blood samples were analyzed at the hospital's clinical laboratory. Biochemical parameters including triglycerides, total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and fasting blood glucose were measured enzymatically using a TOSHIBA TBA-120FR automated analyzer with manufacturer-specified reagents. Dyslipidemia was diagnosed if total cholesterol exceeded 6.22 mmol/L, LDL >4.14 mmol/L, triglycerides >2.26 mmol/L, or HDL <1.04 mmol/L.[21] Diabetes mellitus was defined as fasting glucose ≥7.0 mmol/L or self-reported clinical diagnosis.[22] White blood cell counts were quantified using an ABX Pentra 80 hematology analyzer (HORIBA Jobin Yvon, France) with DiaSys Diagnostic Systems reagents, operating within a detection range of 0.45–124 $\times 10^9$/L and demonstrating an interassay coefficient of variation of 0.2% through daily quality control calibration.

Definition of Covariates

Potential confounding factors were systematically collected and adjusted for in all analyses. Age was stratified into three categories: youth (19–44 years), middle-aged (45–59 years), and older adults (60–78 years). Body mass index (BMI) and waist circumference (WC) were categorized as follows: BMI <24.0 kg/m^2, 24.0–27.9 kg/m^2, or ≥28.0 kg/m^2; WC <90 cm or ≥90 cm. Participants were subsequently classified into three body composition groups: normal weight (BMI <24.0 kg/m^2 and WC <90 cm), overweight (BMI 24.0–27.9 kg/m^2), and obesity (BMI ≥28.0 kg/m^2 or WC ≥90 cm) based on Chinese obesity criteria.[23] Smoking status was defined as never, current (daily smoking >6 months), or former (cessation >6 months); alcohol consumption as never, ≤1 drink/week, or >1 drink/week. Educational attainment was categorized into three levels: primary (0–6 years), junior high (7–9 years), and senior high or above (≥10 years). Hypertension was diagnosed as systolic pressure ≥140 mmHg, diastolic pressure ≥90 mmHg, or self-reported clinical history. Standardized anthropometric measurement protocols have been previously detailed.[9]

Statistical Methods

Continuous variables were compared using Student's t-tests, while categorical variables were analyzed with chi-square (χ^2) tests to evaluate characteristic distributions across LUTS severity strata. Given the physiological range of white blood cell counts (4.0–10.0 $\times 10^9$/L), participants were divided into quartiles based on WBC levels. Multivariable logistic regression models were used to assess the associations between covariates and LUTS risk as well as the relationships between WBC quartiles and overall LUTS severity, individual LUTS symptoms, and voiding and storage symptom domains. Covariate selection employed stepwise regression with bidirectional elimination, retaining variables that achieved statistical significance (P< 0.10) or altered effect estimates by >10%. Results are presented as adjusted odds ratios (ORs) with 95% confidence intervals (CIs). A two-tailed P-value <0.05 defined statistical significance. All analyses were performed using SPSS 17.0 (SPSS Inc., Chicago, IL).

Results

The analytical cohort comprised 4,694 participants, with baseline characteristics stratified by LUTS severity presented in Table 1. The mean white blood cell count was 6.9 $\times 10^9$/L (SD = 1.4 $\times 10^9$/L), with quartile values of 5.9, 6.8, and 7.8 $\times 10^9$/L. Participants with moderate/severe LUTS exhibited significantly higher WBC levels and systolic blood

Table 1 Weighted Characteristics of the 4694 Included Subjects, Stratified by LUTS Severity

Characteristics	Total	LUTS Severity		*P*-value
		None or Mild	Moderate or Severe	
Number of subjects, n	4694	4381	313	
WBC (×10^9/L ± SD)	6.9 ± 1.3	6.8 ± 1.3	7.2 ± 1.3	0.000[a]
Quartile value	5.9, 6.8 and 7.8			
Blood pressure (mmHg ± SD)				
SBP	124.7 ± 16.3	124.1 ± 15.9	132.2 ± 20.7	0.000[a]
DBP	80.6 ± 10.0	80.6 ± 10.0	81.3 ± 10.2	0.188[a]
Age (y), n (%)				
19–44	2846 (60.6)	2748 (62.7)	98 (31.3)	0.000
45 - 59	1287 (27.4)	1201 (27.4)	86 (27.5)	0.894
60 - 78	561 (12.0)	432 (9.9)	129 (41.2)	0.000
BMI (kg/m^2), n (%)				
< 24.0	2152 (45.8)	2012 (45.9)	140 (44.7)	0.599
24.0–27.9	1916 (40.8)	1780 (40.6)	136 (43.5)	0.279
≥ 28.0	626 (13.3)	589 (13.4)	37 (11.8)	0.425
Waist circumference (cm), n (%)				
< 90	3889 (82.9)	3630 (82.9)	259 (82.7)	0.957
≥ 90	805 (17.1)	751 (17.1)	54 (17.3)	0.957
Alcohol drinking (D/W), n (%)				
Never	1172 (25.0)	1108 (25.3)	64 (20.4)	0.055
≤ 1	949 (20.2)	888 (20.3)	61 (19.5)	0.778
> 1	2573 (54.8)	2385 (54.4)	188 (60.1)	0.058
Smoking status, n (%)				
Never	2411 (51.4)	2239 (51.1)	172 (55.0)	0.191
Formal	329 (7.0)	299 (6.8)	30 (9.6)	0.069
Current	1954 (41.6)	1843 (41.1)	111 (35.5)	0.023
Education (y), n (%)				
0 - 6	144 (3.1)	115 (2.6)	29 (9.3)	0.000
7 - 9	656 (14.0)	606 (13.8)	50 (16.0)	0.292
≥ 10	3894 (83.0)	3660 (83.5)	234 (74.8)	0.000
Dyslipidemia, yes, n (%)	1990 (42.4)	1853 (42.3)	137 (43.8)	0.619
Diabetes mellitus, yes, n (%)	254 (5.4)	218 (5.0)	36 (11.5)	0.000

Notes: None or mild and moderate or severe symptoms (IPSS < 8 and IPSS ≥ 8. IPSS, International Prostate Symptom Score); [a] student *t* test and chi square test (χ^2) for others.

Abbreviations: LUTS, lower urinary tract symptoms; BMI, body mass index; WBC, total white blood cell count; SBP, systolic blood pressure; DBP, diastolic blood pressure, D/W, drinks per week; SD, standard deviation.

pressure (SBP) compared to those with none/mild symptoms (Student's *t*-test, P< 0.001). Statistically significant differences in the distribution of LUTS severity (none/mild vs. moderate/severe) were observed in the age groups of 19–44 and 60–78 years, among current smokers, in individuals with education attainment of 0–6 years and ≥10 years, and in those with diabetes mellitus (χ^2-test, P< 0.01), while other variables showed no significant associations.

Tables 2 and 3 present the associations between overall lower urinary tract symptoms (LUTS), individual LUTS symptoms, voiding symptoms, and storage symptoms with white blood cell (WBC) count quartiles. No significant association was observed between storage symptoms and WBC count in univariable analysis (odds ratio [OR] = 1.29, 95% confidence interval [CI] = 0.94–1.77), though this relationship became marginally significant after adjustment for potential confounders (multivariate-adjusted OR = 1.45, 95% CI = 1.04–2.03). In contrast, the risk of overall LUTS was 1.51-fold (95% CI = 1.08–2.11) and 1.93-fold (95% CI = 1.40–2.66) higher in the third and fourth WBC quartiles, respectively, compared with the first quartile. Additionally, a statistically significant positive association was observed between voiding symptoms and WBC count, with participants in the fourth WBC quartile 1.68 times more likely to

Table 2 ORs and 95% CIs for Overall LUTS, Storage Symptoms, and Voiding Symptoms by WBC Quartiles

Dependent Variables	WBC (× 10^9/L)	None or Mild Symptoms n (%)	Moderate or Severe Symptoms n (%)	OR (95% CI) Unadjusted	OR (95% CI) Multivariate Adjusted
Overall LUTS					
	Q1	1083 (94.8)	60 (5.2)	1	1
	Q2	1093 (96.2)	43 (3.8)	0.71 (0.48–1.06)	0.72 (0.48–1.08)
	Q3	1090 (92.3)	91 (7.7)	1.51 (1.08–2.11)*	1.59 (1.12–2.25)*
	Q4	1115 (90.4)	119 (9.6)	1.93 (1.40–2.66)*	2.22 (1.58–3.12)*
Storage symptoms					
	Q1	1072 (93.8)	71 (6.2)	1	1
	Q2	1062 (93.5)	74 (6.5)	1.05 (0.75–1.47)	1.09 (0.77–1.54)
	Q3	1098 (93.0)	83 (7.0)	1.14 (0.82–1.58)	1.21 (0.86–1.69)
	Q4	1137 (92.1)	97 (7.9)	1.29 (0.94–1.77)	1.45 (1.04–2.03)*
Voiding symptoms					
	Q1	1087 (95.1)	56 (4.9)	1	1
	Q2	1094 (96.3)	42 (3.7)	0.75 (0.50–1.12)	0.76 (0.50–1.15)
	Q3	1121 (94.9)	60 (5.1)	1.04 (0.72–1.51)	1.08 (0.74–1.59)
	Q4	1136 (92.1)	98 (7.9)	1.68 (1.19–2.35)*	1.88 (1.31–2.67)*

Notes: None or mild and moderate or severe symptoms (IPSS < 8 and IPSS ≥ 8). Adjustment factors: Models for overall LUTS adjusted for age, smoking status, alcohol consumption, hypertension, education, diabetes mellitus; Models for storage symptoms and voiding symptoms adjusted for age, smoking status, hypertension, education, diabetes mellitus. *$P < 0.05$.

Abbreviations: ORs, odds ratios; CIs, confidence intervals; WBC, Total White Blood Cells Count; Q, WBC quartiles (Q1: 4.0–5.8 × 10^9/L, Q2: 5.9–6.7 × 10^9/L, Q3: 6.8–7.7 × 10^9/L, Q4: 7.8–10.0 × 10^9/L); LUTS, lower urinary tract symptoms.

Table 3 ORs and 95% CIs for Individual Symptoms of LUTS and QoI by WBC Quartiles

Dependent Variables	Quartile of WBCs OR (95% CI) Q1 (n = 1178)	Q2 (n = 1136)	Q3 (n = 1181)	Q4 (n = 1234)
Incomplete emptying				
Unadjusted	1	0.92 (0.58–1.47)	1.21 (0.78–1.86)	1.13 (0.73–1.74)
Multivariate adjusted	1	0.93 (0.58–1.49)	1.20 (0.77–1.87)	1.15 (0.74–1.80)
Frequency				
Unadjusted	1	1.06 (0.67–1.68)	1.21 (0.78–1.88)	1.29 (0.84–1.98)
Multivariate adjusted	1	1.10 (0.70–1.74)	1.30 (0.83–2.04)	1.51 (0.97–2.34)
Intermittency				
Unadjusted	1	0.82 (0.39–1.70)	1.96 (1.07–3.60)*	2.91 (1.65–5.15)*
Multivariate adjusted	1	0.82 (0.39–1.72)	1.95 (1.05–3.61)*	2.96 (1.65–5.30)*
Urgency				
Unadjusted	1	1.07 (0.53–2.18)	1.76 (0.93–3.33)	3.17 (1.77–5.69)*
Multivariate adjusted	1	1.11 (0.54–2.27)	1.87 (0.98–3.56)	3.63 (1.99–6.61)*
Weak stream				
Unadjusted	1	0.65 (0.34–0.84)$^{\Delta}$	0.71 (0.41–1.23)	1.39 (0.87–2.21)
Multivariate adjusted	1	0.69 (0.39–1.12)	0.74 (0.42–1.28)	1.59 (0.98–2.57)
Straining				
Unadjusted	1	0.45 (0.20–1.00)	0.77 (0.40–1.50)	1.30 (0.73–2.33)
Multivariate adjusted	1	0.49 (0.22–1.08)	0.91 (0.46–1.78)	1.74 (0.95–3.19)
Nocturia				
Unadjusted	1	0.91 (0.59–1.41)	0.83 (0.53–1.29)	0.79 (0.51–1.24)
Multivariate adjusted	1	0.93 (0.58–1.49)	0.82 (0.51–1.31)	0.86 (0.53–1.39)

(*Continued*)

Table 3 (Continued).

Dependent Variables	Quartile of WBCs OR (95% CI)			
	Q1 (n = 1178)	Q2 (n = 1136)	Q3 (n = 1181)	Q4 (n = 1234)
QOL				
Unadjusted	1	0.87 (0.64–1.23)	0.81 (0.58–1.13)	0.68 (0.48–0.96)*
Multivariate adjusted	1	0.92 (0.66–1.29)	0.89 (0.63–1.25)	0.77 (0.54–1.25)

Notes: Models Multivariate adjustment factors. Incomplete emptying: alcohol consumption, age, hypertension, education and diabetes mellitus; Frequency: age, smoking status, alcohol consumption and hypertension; Intermittency: age, hypertension, education, dyslipidemia, diabetes mellitus; Urgency: age, hypertension, education, diabetes mellitus, smoking status, waist circumference; Weak stream: age, smoking status, alcohol consumption, hypertension, waist circumference, diabetes mellitus; Strain: age, smoking status, alcohol consumption, hypertension, BMI, diabetes mellitus; Nocturia: age, smoking status, hypertension, diabetes mellitus, education; Quality of life: age, smoking status, hypertension, dyslipidemia. *$P < 0.05$.
Abbreviations: ORs, odds ratios; CIs, confidence intervals; Q, WBC quartiles (Q1: 4.0–5.8 × 10^9/L, Q2: 5.9–6.7 × 10^9/L, Q3: 6.8–7.7 × 10^9/L, Q4: 7.8–10.0 × 10^9/L); QoL, quality of life.

report voiding symptoms than those in the first quartile (Table 2). For individual LUTS symptoms, increasing WBC quartiles were associated with progressively higher risks of intermittency and urgency (OR = 2.91, 95% CI = 1.65–5.15; OR = 3.17, 95% CI = 1.77–5.69, respectively), while no significant associations were found for other individual symptoms (Table 3).

Table 4 summarizes the associations between overall LUTS and major covariates. Advancing age, hypertension, alcohol consumption, and diabetes mellitus were positively associated with LUTS risk, while longer educational attainment demonstrated a significant inverse relationship. All odds ratios and 95% confidence intervals are detailed in Table 4.

Discussion

This population-based study provides novel epidemiological evidence that elevated peripheral white blood cell counts independently predict increased risks of overall lower urinary tract symptoms (LUTS), voiding symptoms, and specific manifestations including intermittency and urgency in Chinese men. The dose-response relationships observed across WBC quartiles—with the highest quartile conferring 1.93-fold (95% CI: 1.40–2.66) and 3.17-fold (95% CI: 1.77–5.69) risks for overall LUTS and urgency, respectively—persisted after rigorous adjustment for confounders such as age, hypertension, and diabetes. These findings collectively implicate acute systemic inflammation, as reflected by leukocyte elevation, in LUTS pathogenesis. Conversely, no significant associations emerged between WBC levels and storage symptoms, quality of life, or other individual symptoms (incomplete emptying, weak stream, straining, frequency, nocturia), suggesting symptom-specific inflammatory mechanisms. Our risk factor analysis further corroborates established contributors: advancing age, hypertension, alcohol consumption, and diabetes mellitus increased LUTS risk, while higher educational attainment demonstrated a protective effect, potentially mediated through enhanced health awareness and early intervention behaviors.

The inflammatory paradigm in LUTS has evolved substantially since Nickel et al's seminal 1994 proposal of prostatic inflammation as a "third component" alongside hormonal and adrenergic pathways in benign prostatic hyperplasia (BPH) development.[24] Histopathological studies consistently identify chronic inflammatory infiltrates in prostate biopsies from BPH patients, with inflammation density correlating positively with both prostate volume and IPSS scores.[6,25,26] More recently, epidemiological investigations have expanded focus toward systemic inflammation markers. While the Olmsted County cohort reported no CRP-LUTS progression linkage,[12] both FAMHES and BACH studies documented significant positive associations.[9,11] Our study extends this framework by establishing WBC—an acute-phase biomarker with distinct physiological regulation from CRP—as an independent predictor of LUTS severity. This divergence is mechanistically plausible: whereas CRP primarily reflects chronic interleukin-6-mediated inflammation, WBC elevations often signify acute tissue-level immune responses, including neutrophil recruitment to urological tissues.

Our results demonstrated both convergence and divergence compared to previously reported relationships between CRP and LUTS.[9,11] These common findings may be attributed to the shared association of both serum CRP and WBCs

Table 4 ORs and 95% CIs for LUTS Stratified by Potential Confounding Factors

Covariates	Moderate or Severe Overall LUTS OR (95% CI)	
	Unadjusted	Multivariate Adjusted
Hypertension		
No	1	1
Yes	2.55 (2.02–3.22)*	1.39 (1.06–1.82)*
Age (y)		
19 - 44	1	1
45 - 59	2.01 (1.49–2.70)*	1.94 (1.42–2.64)*
60 - 78	8.37 (6.32–11.10)*	7.62 (5.47–10.63)*
BMI (kg/m^2)		
< 24.0	1	1
24.0–27.9	1.10 (0.86–1.40)	0.96 (0.73 −1.25)
≥ 28.0	0.90 (0.62–1.31)	0.81 (0.50–1.33)
Waist circumference (cm)		
< 90	1	1
≥ 90	1.01 (0.74–1.37)	0.87 (0.60–1.27)
Alcohol drinking (D/W)		
Never	1	1
≤ 1	1.19 (0.83–1.71)	2.13 (1.45–3.13)*
> 1	1.37 (1.02–1.83)*	2.03 (1.49–2.77)*
Smoking status		
Never	1	1
Formal	1.31 (0.87–1.96)	0.87 (0.57–1.34)
Current	0.78 (0.61–1.01)	0.84 (0.65–1.09)
Education (y)		
0 - 6	1	1
7 - 9	0.33 (0.20–0.54)*	0.58 (0.41–0.91)*
≥ 10	0.25 (0.17–0.39)*	0.67 (0.49–0.95)*
Dyslipidemia		
No	1	1
Yes	1.06 (0.84–1.34)	0.98 (0.76–1.26)
Diabetes mellitus		
No	1	1
Yes	2.48 (1.71–3.60)*	1.81 (1.18–2.92)*

Notes: Multivariate adjustment factors: all others variables in the table *$P < 0.05$.
Abbreviations: ORs, odds ratios; CIs, confidence intervals; LUTS, lower urinary tract symptoms; D/W, drinks per week.

with systemic inflammation, thereby consolidating the evidence that inflammation plays a significant role in LUTS progression. Interestingly, the discrepancy between our current findings and prior studies primarily manifested in voiding symptoms. Clinical studies have shown that inflammatory infiltrates in prostate biopsy sections are associated with increased prostate volume,[6,27] and administration of an immunostimulator to Wistar rats resulted in prostatic epithelial proliferation in an animal model.[28] Furthermore, inflammation can also lead to bladder wall thickening and ultimately impaired bladder function, as prostaglandins mediate bladder cell contractions and contribute to urinary retention.[29] Additionally, reports suggest that the relationship between serum CRP levels and WBC counts is not straightforward, despite both being biomarkers of the acute inflammatory phase.[16,17] As outlined above, this distinction between CRP and WBCs may explain the differential relationship we observed between WBCs and LUTS compared to CRP.

Furthermore, the current study evaluated the risk of LUTS associated with environmental factors, medical history, and physical status. Age, a well-established independent risk factor for LUTS confirmed in numerous reports,[8,30] was likewise associated with a significantly increased risk in our results. Consistent with a previous study,[31] our findings

revealed no significant difference in overall LUTS prevalence between current and non-smokers or between individuals with normal and abnormal BMI. However, a positive association was observed between the frequency of alcohol consumption, a history of hypertension, and LUTS. Diabetes mellitus, previously identified as a significant risk factor for LUTS,[32] was also associated with increased risk in our study. Consistent with previous reports,[33] our data interestingly suggested an inverse association between educational attainment and overall LUTS. This may be explained by individuals with higher education levels potentially paying greater attention to their health and being more sensitive to symptoms; however, these symptoms might not significantly impact their quality of life. This could contribute to the lack of association observed between WBC levels and quality of life (QoL).

To our knowledge, this study represents the first large-scale epidemiological investigation utilizing WBC data to explore the role of systemic inflammation in LUTS progression. The findings not only provide insights into the role of inflammation in LUTS development but also may help identify potential interventions for LUTS prevention. However, several limitations should be acknowledged. Firstly, due to the cross-sectional design, causal relationships cannot be inferred. Secondly, subject characteristics, including lifestyle, medical history, and IPSS scores, were collected via questionnaire, which may introduce recall bias; we attempted to mitigate this by employing specially trained physicians for data collection. Lastly, the cross-sectional design precluded consideration of fluctuations in WBC levels and other biochemical indicators over time; these changes will be monitored in future follow-up studies.

Conclusion

This population-based study establishes elevated peripheral white blood cell count as an independent risk predictor for lower urinary tract symptoms in Chinese men, demonstrating significant dose-dependent associations with overall LUTS severity, voiding symptoms, and specific manifestations including intermittency and urgency. These associations—which remained evident even after full adjustment for metabolic comorbidities—suggest that systemic inflammation may play a role in LUTS pathogenesis.

Ethics Approval and Informed Consent

The study complied with the Declaration of Helsinki and received ethical approval from the Institutional Review Board of Guangxi Medical University (Approval No: GXMU20110329).

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Disclosure

The authors report no conflicts of interest in this work.

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