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ORIGINAL ARTICLE

Interrelation of Urinary Tract Infections and Renal Failure: Evaluating Pathogen Prevalence and the Diagnostic Utility of FGF23 and CFH Biomarkers

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ABSTRACT

Background: *Urinary tract infections (UTIs) are prevalent bacterial infections, particularly among females, often leading to significant morbidity. Renal failure (RF) compromises immune defenses, and increasing susceptibility to infections. Biomarkers such as Fibroblast Growth Factor 23 (FGF23) and Complement Factor H (CFH) have been implicated in renal pathophysiology.* ***Objective:*** *The aim of this work is to investigate the interplay between UTIs and RF by analyzing pathogen prevalence and assessing FGF23 and CFH levels in affected patients.* ***Methodology:*** *A cross-sectional study was conducted involving patients diagnosed with UTI and/or RF. Midstream urine samples were collected for microbiological analysis using the VITEK® 2 Compact system. Serum levels of FGF23 and CFH were measured using enzyme-linked immunosorbent assay, respectively. Data were analyzed to identify associations between pathogen prevalence, biomarker levels, and patient demographics.* ***Results****. The study revealed a higher incidence of UTIs among younger females. Escherichia coli was identified as the predominant uropathogen. Patients with RF exhibited significantly elevated levels of FGF23 and CFH compared to those without RF. These biomarkers demonstrated high predictive accuracy for renal impairment.* ***Conclusion:*** *The findings highlight the importance of monitoring FGF23 and CFH levels in patients with renal impairment. Early detection of elevated levels can facilitate timely interventions to mitigate progression and manage complications. Understanding demographic predispositions to UTIs can inform targeted prevention strategies, particularly among younger females, to reduce infection rates and associated renal complications.*

INTRODUCTION

Urinary tract infections (UTIs) are among the most prevalent bacterial infections globally, affecting millions annually and imposing significant healthcare burdens[1]. They encompass infections of the urethra, bladder, ureters, and kidneys, with cystitis (bladder infection) being the most common manifestation[2]. The pathogenesis of UTIs is primarily attributed to the ascent of uropathogens from the periurethral area to the bladder and, in severe cases, to the kidneys, leading to pyelonephritis[3].

UTIs exhibit a higher incidence in females than males, a disparity largely due to anatomical differences such as a shorter urethra and its proximity to the anus, facilitating bacterial entry into the urinary tract[4,5]. Approximately 50–60% of women will experience at least one UTI in their lifetime[6]. Risk factors include sexual activity, use of spermicides, a new sexual partner, and a history of previous UTIs[7]. In men, UTIs are less common but can occur secondary to urinary tract obstructions, such as benign prostatic hyperplasia, or catheterization[8]. Other predisposing factors encompass diabetes mellitus, immunosuppression, and structural abnormalities of the urinary tract[9].

The majority of uncomplicated UTIs are caused by uropathogenic *Escherichia coli* (UPEC), accounting for approximately 70–95% of cases[10]. Other notable pathogens include *Klebsiella pneumoniae, Proteus mirabilis, Staphylococcus saprophyticus,* and *Enterococcus faecalis*[11]. These pathogens possess virulence factors such as adhesins, toxins, and biofilm formation capabilities, which facilitate colonization and persistence within the urinary tract[11].

Chronic kidney disease (CKD) and acute kidney injury (AKI) compromise the body's immune defenses, rendering patients more susceptible to infections, including UTIs[12]. Impaired renal function leads to the accumulation of uremic toxins, which can suppress various immune responses[13]. Additionally, patients with renal failure often require interventions such as

catheterization or dialysis, further increasing the risk of infection[14].

Biomarkers play a pivotal role in diagnosing, prognosticating, and monitoring renal diseases[15]. Fibroblast Growth Factor 23 (FGF23) and Complement Factor H (CFH) have emerged as significant biomarkers in renal pathophysiology[16,17]. FGF23 is a hormone predominantly secreted by osteocytes and osteoblasts, regulating phosphate homeostasis and vitamin D metabolism[18]. In CKD, FGF23 levels rise progressively as a compensatory mechanism to maintain phosphate balance[19]. Elevated FGF23 levels have been associated with adverse outcomes, including left ventricular hypertrophy, increased mortality, and progression of renal disease[20].

CFH is a crucial regulator of the alternative complement pathway, preventing uncontrolled complement activation on host tissues[21]. Genetic mutations or deficiencies in CFH can lead to atypical hemolytic uremic syndrome (aHUS), characterized by acute renal failure, hemolytic anemia, and thrombocytopenia[22]. Furthermore, dysregulation of CFH has been implicated in the pathogenesis of other renal conditions, such as membranoproliferative glomerulonephritis[23].

Understanding the interplay between UTIs and renal failure is essential for improving patient outcomes. While UTIs can precipitate renal impairment, existing renal dysfunction can predispose individuals to recurrent and severe UTIs. Investigating the prevalence of specific uropathogens and assessing biomarkers like FGF23 and CFH in patients with renal failure may provide insights into disease mechanisms and potential therapeutic targets.

This study aims to elucidate the relationship between UTIs and renal failure by: Determining the prevalence and distribution of uropathogens in patients with and without renal failure, assessing the levels of FGF23 and CFH in these patient cohorts and exploring potential correlations between these biomarkers, the presence of UTIs, and renal function status

METHODOLOGY

Study Design and Population

This cross-sectional study was conducted at Al-Husseini Teaching Hospital Between September, 2024 to February, 2025 . focusing on patients presenting with urinary tract infections (UTIs) and/or renal failure (RF). The study adhered to ethical standards, with approval from the Institutional Review Board (IRB) of Al-Husseini Teaching Hospital [Ethical No. 3772], and all participants provided informed consent.The research protocol was designed and conducted in accordance with the ethical principles outlined in the Declaration of Helsinki [24].

Inclusion and Exclusion Criteria

Patients aged 18 years and above, diagnosed with UTI and/or RF, were included. Exclusion criteria encompassed individuals with concurrent systemic infections, recent antibiotic therapy (within the past two weeks), or immunocompromised states (e.g., HIV infection, ongoing chemotherapy).

Sample Collection

Midstream urine samples were collected aseptically from all participants for microbiological analysis. Additionally, venous blood samples were obtained to assess immunological markers, specifically Fibroblast Growth Factor 23 (FGF23) and Complement Factor H (CFH)

Microbiological Analysis

Bacterial Identification and Antibiotic Susceptibility Testing

Urine samples were cultured on standard media and incubated at 37°C for 24 hours. Isolated colonies were subjected to identification and antibiotic susceptibility testing using the VITEK® 2 Compact system (bioMérieux), an automated platform that utilizes fluorescence-based technology to detect metabolic changes for microbial identification and susceptibility profiles. The system offers rapid results, typically within 2 to 10 hours for Gram-negative bacilli and 2 to 8 hours for Gram-positive cocci.

Immunological Marker Assessment

Measurement of CFH & FGF23 Levels

Serum CFH & FGF23 concentrations were determined using an enzyme-linked immunosorbent assay (ELISA) kit (BT LAB, China), following the manufacturer's protocol. Samples and standards were added to microplate wells pre-coated with an anti-CFH antibody. After incubation and washing steps, a biotinylated detection antibody specific to CFH & FGF23 was applied, followed by a streptavidin-horseradish peroxidase (HRP) conjugate. The addition of a tetramethylbenzidine (TMB) substrate solution resulted in a colorimetric change proportional to the CFH & FGF23 concentration, measured at an appropriate wavelength using a microplate reader

Data Collection and Statistical Analysis

Demographic and clinical data, including age, sex, and relevant medical history, were collected using standardized forms. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Comparisons between groups were performed using appropriate statistical tests (e.g., t-tests, chi-square tests), with a p-value <0.05 considered statistically significant. All analyses were conducted using SPSS version 25.0 (IBM Corp., Armonk, NY)[25].

RESULTS

In **Table 1.** data reveal variations in the prevalence of UTI and renal failure across different demographic categories. Patients younger than 40 had the highest occurrence of UTI alone (43.33%), whereas the group aged 40–59 exhibited the highest rate of concurrent renal failure and UTI (40%). Patients aged 60 and older had lower isolated renal failure cases (20%) compared to younger age groups. Females showed a higher incidence of UTIs alone (70%) and combined renal failure with UTI (63.33%), in contrast to males who predominantly presented renal failure without UTI (76.67%)**.**

In **Table 2**, the most frequently isolated pathogen was *Escherichia coli*, consistently accounting for half (50%) of infections, irrespective of the presence of renal failure. *Klebsiella pneumoniae* ranked second, accounting for 16.67% overall. Less frequent pathogens included *Staphylococcus aureus*, *Enterococcus faecalis*, and *Pseudomonas aeruginosa*, each contributing with less than 7% individually. Rarer pathogens such as *Proteus mirabilis*, *Staphylococcus haemolyticus, Staphylococcus hominis*, *Staphylococcus saprophyticus*, and *Acinetobacter baumannii* accounted for approximately 5% or lower, reflecting diverse but infrequent pathogenic involvement.

Findings in **Table 3**, demonstrate significant differences in mean values of FGF23 among groups (p = 0.000). Patients with renal failure without UTI exhibited the highest mean (151.958 ± 130.333), while those with renal failure accompanied by UTI showed slightly lower values (115.162 ± 81.140). Both were markedly higher than the UTI-only (33.412 ± 14.633) and control groups (25.185 ± 12.619), indicating a strong correlation between elevated FGF23 mean values and the presence of renal failure.

Table 1: Distribution of Urinary Tract Infections (UTIs) and Renal Failure (RF) among Patients, Categorized by Age Group and Sex[a]

Parameters level		UTI	RF without UTI	RF with UTI	Total
Age Group	< 40	43.33%	40.00%	26.67%	36.67%
	40 - 59	20.00%	40.00%	40.00%	33.33%
	>= 60	36.67%	20.00%	33.33%	30.00%
Sex	Male	30.00%	76.67%	36.67%	47.78%
	Female	70.00%	23.33%	63.33%	52.22%

a; UTI = Urinary Tract Infection; RF = Renal Failure. Percentages indicate the proportion within each category. "RF without UTI" denotes patients having renal failure but no infection, and "RF with UTI" represents those having both renal failure and UTI.

Table 2: Distribution of Pathogenic Microorganisms among Patients with Urinary Tract Infection (UTI), Stratified by Renal Failure (RF)[a]

Types of pathogens	UTI	RF with UTI	Total
Escherichia coli	50.00%	50.00%	50.00%
Klebsiella pneumoniae	20.00%	13.33%	16.67%
Staphylococcus aureus	6.67%	16.67%	11.67%
Enterococcus faecalis	6.67%	3.33%	5.00%
pseudomonas aeruginosa	6.67%	3.33%	5.00%
proteus mirabilis	3.33%	6.67%	5.00%
Staphylococcus haemolyticus	3.33%	0.00%	1.67%
Staphylococcus hominis	3.33%	0.00%	1.67%
staphylococcus saprophyticus	0.00%	3.33%	1.67%
acinetobacter baumannii	0.00%	3.33%	1.67%

a; UTI = Urinary Tract Infection; RF = Renal Failure. Percentages indicate the distribution of pathogens identified within each category.

Table 3: Comparative Analysis of FGF23 Mean Levels across Study Groups (Control, UTI, and Renal Failure (RF))[a]

Groups	Mean	Std. Deviation	P. value
Control	25.185	12.619	0.0003
UTI	33.412	14.633	
RF without UTI	151.958	130.333	
RF with UTI	115.162	81.140	

a; UTI = Urinary Tract Infection; RF = Renal Failure. Values expressed as mean ± standard deviation. Statistical significance indicated at $p < 0.05$.

In **Table 4.** a significant differences were observed in CHF parameter levels among the studied groups (p = 0.0006). Patients with renal failure but without UTI exhibited the highest CHF mean (291.399 ± 199.580), followed by patients having both renal failure and UTI (222.480 ± 189.977). The UTI-only group (59.185 ± 22.716) and control group (42.490 ± 14.971) had substantially lower CHF parameter values.

The presented predictive modeling results in **Table 5.** demonstrate high predictive accuracy of immunological markers (FGF23 and CFH) for renal failure patients. Both markers exhibit low standard error (0.016 for FGF23; 0.018 for CFH) and high asymptotic significance values (>0.93), suggesting their strong predictive capability. Confidence intervals were narrow, indicating high reliability and precision of these markers in discriminating renal failure patients. Sensitivity, specificity, and positive and negative predictive values consistently exceeded 96%, underscoring excellent clinical utility in diagnostic scenarios.

Table 4: Comparisons of CHF Parameter Levels among Patients Categorized by Control, UTI, and Renal Failure (RF) Status[a]

Groups	Mean	Std. Deviation	P. value
Control	42.490	14.971	0.0006
UTI	59.185	22.716	
RF without UTI	291.399	199.580	
RF with UTI	222.480	189.977	

a; CHF = Chronic Heart Failure; UTI = Urinary Tract Infection; RF = Renal Failure. Values are presented as mean ± standard deviation. Statistical significance set at a p-value < 0.05.

Table 5: Model Prediction Metrics of Immunological Markers (FGF23 and CFH) for Renal Failure (RF) Patients[a]

Metrics		RF Patients	
		FGF23	CFH
Std. Error		0.016	0.018
Asymptotic Sig.		0.004	0.005
Asymptotic 95% Confidence Interval	Lower Bound	0.939	0.933
	Upper Bound	1.000	1.000
Cutoff Point		49.507	69.677
Area Under Curve (AUC)		97.000%	96.833%
Sensitivity		90.000%	90.000%
Specificity		96.567%	96.645%
Accuracy		96.755%	96.667%
Positive Predictive Value		96.460%	96.543%
Negative Predictive Value		96.648%	96.722%

a;RF = Renal Failure; FGF23 = Fibroblast Growth Factor 23; CFH = Complement Factor H. Metrics presented include Standard Error, Asymptotic Significance, and Asymptotic 95% Confidence Interval bounds for evaluating predictive accuracy

DISCUSSION

Our study investigated the interplay between urinary tract infections (UTIs) and renal failure (RF), focusing on pathogen prevalence, immunological markers, and demographic influences. The findings revealed a higher incidence of UTIs among younger patients, particularly females, with *Escherichia coli* being the predominant pathogen. Elevated levels of immunological markers, specifically Fibroblast Growth Factor 23 (FGF23) and Complement Factor H (CFH), were observed in patients with renal failure, irrespective of UTI presence. These markers demonstrated high predictive accuracy for renal impairment.

The predominance of *E. coli* in UTIs aligns with existing literature, which identifies it as the leading causative agent in both uncomplicated and complicated UTIs[10]. For instance, a study by GHOSH and Sarwar [26]highlighted that *E. coli* accounts for a significant proportion of UTI cases, emphasizing its role in urinary tract infections. The observed higher incidence of UTIs in younger females is consistent with prior findings attributing this trend to anatomical and behavioral factors that facilitate bacterial entry into the urinary tract[27-29].

The association between elevated FGF23 and CFH levels with renal failure corroborates previous research identifying these markers as significant in renal

pathophysiology. Elevated FGF23 levels have been linked to disrupted phosphate metabolism and cardiovascular complications in chronic kidney disease patients[19,20]. Similarly, CFH plays a crucial role in regulating the complement system, with deficiencies or dysfunctions contributing to renal diseases such as atypical hemolytic uremic syndrome[22].

The increased susceptibility to UTIs in younger females can be attributed to several anatomical and physiological factors. The shorter urethra in females facilitates easier bacterial ascension to the bladder[30]. Additionally, behavioral factors such as certain hygiene practices can introduce bacteria into the urinary tract and sexual activity has also been identified as a risk factor for UTIs in women[7].

Elevated FGF23 levels in renal failure patients are primarily a response to impaired phosphate excretion[31]. As kidney function declines, phosphate accumulates in the blood, stimulating FGF23 secretion to enhance phosphate excretion and suppress vitamin D activation[32]. However, chronic elevation of FGF23 is associated with adverse outcomes, including left ventricular hypertrophy and increased mortality[20].

CFH regulates the alternative pathway of the complement system, preventing uncontrolled complement activation on host tissues[22]. In renal failure, oxidative stress and inflammation can lead to CFH dysfunction or deficiency, resulting in complement-mediated damage to renal tissues[33]. This mechanism contributes to the progression of renal diseases and underscores the importance of CFH in maintaining renal integrity.

Our findings highlight the need for vigilant monitoring of immunological markers such as FGF23 and CFH in patients with renal impairment. Early detection of elevated levels can facilitate timely interventions to mitigate progression and manage complications. Understanding the demographic predispositions to UTIs can inform targeted prevention strategies, particularly among younger females, to reduce infection rates and associated renal complications.

A notable strength of our study is the comprehensive analysis of both microbiological and immunological factors, providing a holistic understanding of the interplay between UTIs and renal failure. However, limitations include a potential selection bias due to the single-center study design and a limited sample size, which may affect the generalizability of our findings. Additionally, the cross-sectional nature of the study precludes establishing causality between observed associations.

Future research should focus on longitudinal studies to establish causal relationships between immunological markers and renal disease progression. Expanding the study population across multiple centers would enhance the generalizability of findings. Investigating the efficacy of interventions targeting FGF23 and CFH pathways could unveil novel therapeutic approaches for managing renal failure. Moreover, exploring the genetic and environmental factors contributing to demographic disparities in UTI prevalence may inform personalized prevention and treatment strategies.

CONCLUSION

The findings emphasize the need to monitor FGF23 and CFH levels in patients with kidney problems. Detecting high levels early can help in taking timely actions to slow disease progression and manage complications. Identifying demographic factors that increase the risk of UTIs can support targeted prevention efforts, especially for young females, to lower infection rates and related kidney issues.

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Declaration

Conflict of Interest: We declare that there are no conflicts of interest associated with this manuscript.

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Authors contribution:

AHK: Conceptualization, methodology, formal analysis, and manuscript writin, Data collection, investigation, and software analysis..

AJA: Supervision, project administration, and final manuscript review

MRR: Literature review, validation, and manuscript editing.

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