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Inflammatory Parameters in UTIs Infection Caused by *E. coli* in Diabetic Patients

Shahrazad Ahmed Khalaf

Diyala University, College of Science, Department of forensic sciences, Diyala city Iraq.

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*Corresponding Authors: Shahrazad Ahmed Khalaf: Diyala University, College of Science, Department of forensic sciences, Diyala city Iraq. *Tel*: +96-477-29709217, *E-mail:* shahrazadah.kh@gmail.com.

ABSTRACT

Background: Urinary tract infections (UTIs) are a public health concern, mainly for diabetic infections, who are more susceptible due to weakened immunity and high blood sugar. Understanding the role of immune parameters can help improved diagnose and attendant more effective treatment for diabetic mellitus patients with UTIs. This study aims to estimate the serum levels of AGEs, SAA, PCT, CD23 and CD25 in UTIs caused by *E. coli* in Diabetic Patients.

Methods: This study had been included 300 samples (blood for Inflammatory parameters and urine for bacterial diagnosis) obtained from Diabetic patients found in Al-Betool Teaching Hospital. And Baquba Teaching Hospital between June 2023 and August 2024. Urine and blood samples were collected from 300 individuals with diabetes mellitus with both type who were suspected of having UTIs. Serum was isolated from the blood, blood glucose and HBA1c test was completed to check the presence of the diabetic illnesses. The serum samples were then stored for used in measuring immunological parameters that includes AGEs, SSA, PCT, CD23 and CD25 that done later by ELISA. Urine samples were culture on the blood agar and MacConkey agar, and then Bacteria diagnoses was done by using an automatic VITEK 2 system.

Results: The current study showed that 220 samples urinary tract infections, 180 of which were bacterial. Of the 180 samples, 116 have E. coli bacterial isolates and 46 have other bacterial species. The study also shown an increase in the concentration of AGEs, SAA, PCT, CD23 and CD25 in the serum of Diabetic Mellitus patients have UTIs compared with the healthy.

Conclusion: These inflammatory markers not only serve as diagnostic tools but also help in understanding the complex immune dysfunction present in diabetic patients with UTIs caused by *E. coli*.

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Introduction

Urinary Tract Infections (UTIs) are common bacterial infections, mainly between individuals with diabetic mellitus diseases. Escherichia coli (*E. coli*) is the common source of UTIs among the numerous microbes that cause them, accounting up to 80% of all cases (1). Diabetes is characterized by anatomical abnormalities in the urinary tract, chronic hyperglycemia, and impaired immunological response there for people with diabetic mellitus diseases are more susceptible to UTIs (2) and these features contribute to severe and public nature of UTIs in these individuals in adding to increase susceptibility to infection (3).

The inflammatory response has important role in body fight against UTIs, such as the pro-inflammatory cytokines (e.g., IL-6) that are released as part of the immune system's response to infection (4). These inflammatory responses are higher through infection and can contribution as indicators of both infection severity and response to treatment (5). Hyperglycemia is a strong inflammatory stimulus and the treating infections in diabetic patients more difficult by their higher levels of inflammatory indicators (6). Virulence of bacteria that causes UTIs can improve in hyperglycemia and lead to increase the inflammatory cytokine response, potentially prolonging the infection and promoting relapse (7).

Harmful substances known as Advanced Glycation End Products (AGEs) are formed after proteins or lipids bind with sugars in a non-enzymatic method. Particularly in diabetics, whose high blood glucose levels race their synthesis, these chemicals build up in the body over time. AGEs are significant in many pathophysiological processes, such as the spread of diseases including UTIs (8). Accumulation of the AGEs in the body can affects on the ability of immune system to remove the infections by impairing the immune cells such as neutrophils and macrophages in diabetes patients and reducing their capacity to fight the infections like *E. coli*, the primary cause of UTIs (9). Previous study indicates that accumulation AGEs can disrupt the recruitment of immune cells and the released of cytokines (9). The accumulation of AGEs in tissues of the body including the urinary tract can weakens its defense against infections and rises susceptibility to UTIs. Previous study suggests that accumulation of AGEs is involved in endothelial disfunction and decreased tissue repair in diabetic patients (10).

One type of acute phase protein called serum amyloid A(SAA) is important for the body's inflammatory response. Increased SAA levels can provide information about the severity of an illness and are commonly seen through infections especially UTIs. The immune response and the removal of microbes from the urinary system have both been related to SAA (11). SAA is higher during inflammation and infections such as UTIs, SAA plays a central role in attracting immune cells to the site of infection and helping in the clearance of pathogens. High levels of SAA correlated with the strength of inflammation in UTIs and can assist as a valuable biomarker for infection severity (11). In diabetic illness, high levels of SAA may indicate a significant inflammatory response that may lead for more aggressive treatment and can lead to foreknowing complications (12).

Procalcitonin (PCT) is a protein precursor to calcitonin formed in response to bacterial infections. It is observed as a significant biomarker for determining whether infections, such as UTIs, are present and whether they are caused by bacteria (13). PCT is important for both diagnosis and therapy monitoring because of its great sensitivity to bacterial infections (13).

CD23 is a receptor expressed on cell surface of numerous immune cells such as B cells and dendritic cells. It has an important role in immune responses mainly in allergic reactions and infections. Previous studies shown that it has important role in the immune response to UTIs, in modulating the immune responses through infections, plays a significant role in activating B cells and regulating the production of

immunoglobulin (14). Higher levels of CD23 in UTIs may show an active immune response and help in protection against bacterial invasion (14).

CD25 is one of the important receptors found on T cells and has a role in their activation and differentiation, especially regulatory T cells (Tregs), which help maintain immune balance. The immune system to UTIs mainly E. coli infections, is regulated by CD25 expression on Tregs cells (15).

This study aims to examine the role of inflammatory indicators in the susceptibility and outcomes of UTIs caused by *E. coli* in diabetic patients. By examining the levels of key inflammatory mediators in these individuals that includes AGEs, SSA, PCT, CD23 and CD25, the research seeks to enhance our understanding of the pathophysiological mechanisms underlying UTIs and their clinical implications for managing infections in diabetic patients.

Materials and Methods

This study was conducted on type I and type II diabetic patients with bacterial UTIs to measure some immune markers. Samples were collected from Al-Betool Teaching Hospital. And Baquba Teaching Hospital between June 2023 and August 2024. The study included 300 blood and urine samples from both sexes. All urine samples were examined with a microscope, infections were diagnosed only by culturing on blood agar and MacConkey agar plates, and the bacterial species was diagnosed using a Vitek2 compact device. Blood samples (5 ml) were collected in a clean plain tube with gel, and left to coagulate for 30 min at 37°C before centrifugation. The tubes were centrifuged for 5 minutes at 6000 rpm, then serum samples were collected, and levels of AGEs, SSA, PCT, CD23, and CD25 were measured using an ELISA according to the instructions included in the kit from CUSABio company. GraphPad Prism 10 has been used to examine the data statistically.

Results

In this study, 300 specimens (blood and urine) isolated from Baquba Teaching Hospital in Diyala. After culturing the specimens on special media, the results showed that 220 specimens contained growth and 80 did not show any growth. After diagnosis, it was found that 180 specimens contained bacterial growth and 40 did not show any bacterial growth. Then it was noted that 116 samples were due to *E. coli* and 44 for other type of bacteria. As shown in table 1.

Table 2 and figure 1 shows that UTIs infection in DM I and DM II patients were compared to the control group, they had higher levels AGEs, SAA, PCT, CD23 and CD25.The findings demonstrated a significantly significant difference ($p<0.0001$) between the patients and the healthy control group. In comparison to male patients, female patients had higher levels of AGEs, SAA, PCT, CD23 and CD25 in UTIs diabetic Type II patients with significant difference in p-value for PCT only ($p<0.0001$). The female patients had lower levels of SAA, PCT and CD23 compare with male but have higher levels for AGEs and CD25 as shown in table 3 and figure 2.

Figure 2 shows the ROC curves for immune parameters studied in this research, these parameters are estimated for their sensitivity and specificity in distinctive between DM I and DM II. These indicators show variable degrees of sensitivity and specificity, with high sensitivity representing their efficiency in identifying those infected with the disease, while high specificity indicates the ability to accurately identify those who are not infected with the disease.

Table 1. Distribution of study groups.

Total of samples / 300			
Growth			No Growth
220			80
Bacterial Growth		No. Bacterial Growth	
180		40	
E. coli	Other Types		
116	44		

Table 2. Serum level mean of AGEs, SAA, PCT, CD23 and CD25 and healthy control groups.

Parameter	**AGEs (µg/mL)**	**SAA (ng/mL)**	**PCT (pg/mL)**	**CD23 (ng/mL)**	**CD25 (pg/mL)**
Control (Mean ± S.E)	1.627±0.07	229.5±2.255	8.877±0.104	1.696±0.082	92.4±0.267
Patients DM I (Mean ± S.E)	5.087±0.150	263.5±1.898	11.65±0.138	3.740±0.114	96.09±0.157
Patients DM II (Mean ± S.E)	7.20±0.09	271.8±1.358	12.04±0.106	6.187±0.148	96.8±0.222
P. value	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

Table 3. Relationship between AGEs, SAA, PCT, CD23 and CD25 and sex in studied groups.

Parameter	**Patients DM I (Mean ± S.E)**		**Patients DM II (Mean ± S.E)**	
	Male	Female	Male	Female
AGEs	5.187±0.246	7.267±0.156	5.349±0.169	7.22±0.133
P. value	0.6117		0.8219	
SAA	262.1±3.33	265±1.88	273.3±2.122	270.3±1.678
P. value	0.4495		0.2769	
PCT	11.15±0.162	12.16±0.129	12.18±0.161	11.9±0.132
P. value	<0.0001		0.1914	
CD23	3.693±0.183	3.787±0.143	6.353±0.209	6.020±0.208
P. value	0.6916		0.2694	
CD25	95.89±0.164	96.28±0.263	96.43±0.345	97.17±0.257
P. value	0.2243		0.0968	

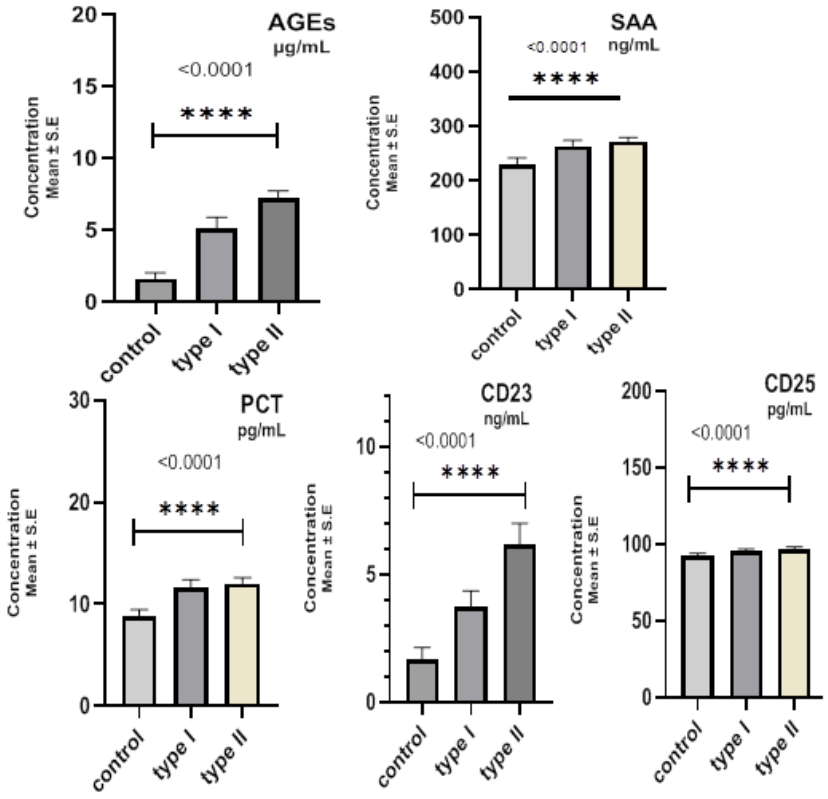

Fig 1. Serum level mean of AGEs, SAA, PCT, CD23 and CD25 in patients and healthy control groups.

Table 4. Correlation Matrix of Various Biomarkers and Their Subtypes.

	AGEs control	**AGEs type I**	**AGEs type II**	**SAA control**	**SAA type I**	**SAA type II**	**PCT control**	**PCT type I**	**PCT type II**	**CD23 control**	**CD23 type I**	**CD23 type II**	**CD25 control**	**CD25 type I**	**CD25 type II**
AGEs control	1	-0.03	-0.54	0.17	-0.39	-0.26	-0.22	-0.51	0.26	0.26	0.05	0.19	-0.46	-0.49	-0.50
AGEs type I	-0.03	1	-0.12	0.40	0.17	0.019	0.019	0.52	0.001	0.33	0.09	0.42	0.57	0.12	0.26
AGEs type II	-0.54	-0.12	1	-0.06	0.42	0.23	0.31	0.39	0.209	-0.05	0.07	0.01	0.26	0.41	0.12
SAA control	0.17	0.40	-0.06	1	0.20	-0.02	-0.10	0.23	0.394	0.16	0.18	0.37	0.23	-0.05	0.18
SAA type I	-0.39	0.17	0.42	0.20	1	0.62	0.38	0.37	0.227	0.10	0.57	0.31	0.42	0.47	0.04
SAA type II	-0.26	0.01	0.23	-0.02	0.62	1	0.49	-0.06	0.211	0.23	0.52	0.24	0.27	0.29	-0.23
PCT control	-0.22	0.01	0.31	-0.10	0.38	0.49	1	0.18	0.384	-0.01	0.47	0.13	0.35	0.08	-0.11
PCT type I	-0.51	0.52	0.39	0.23	0.37	-0.06	0.18	1	-0.078	0.16	0.06	0.13	0.71	0.39	0.41

PCT type II	0.26	0.001	0.20	0.39	0.22	0.21	0.38	-0.07	1	0.12	0.29	0.57	-0.06	-0.20	-0.32
CD23 control	0.26	0.33	-0.05	0.16	0.10	0.23	-0.01	0.16	0.12	1	0.15	0.25	0.11	0.12	-0.31
CD23 type I	0.05	0.09	0.07	0.18	0.57	0.52	0.47	0.06	0.29	0.15	1	0.34	0.41	0.20	-0.39
CD23 type II	0.19	0.42	0.01	0.37	0.31	0.24	0.13	0.13	0.57	0.25	0.34	1	0.04	0.04	-0.07
CD25 control	-0.46	0.57	0.26	0.23	0.42	0.27	0.35	0.71	-0.06	0.11	0.41	0.04	1	0.41	0.08
CD25 type I	-0.49	0.12	0.414	-0.05	0.47	0.29	0.08	0.39	-0.20	0.12	0.20	0.04	0.41	1	-0.04
CD25 type II	-0.50	0.26	0.122	0.18	0.04	-0.23	-0.11	0.41	-0.32	-0.31	-0.39	-0.07	0.08	-0.04	1

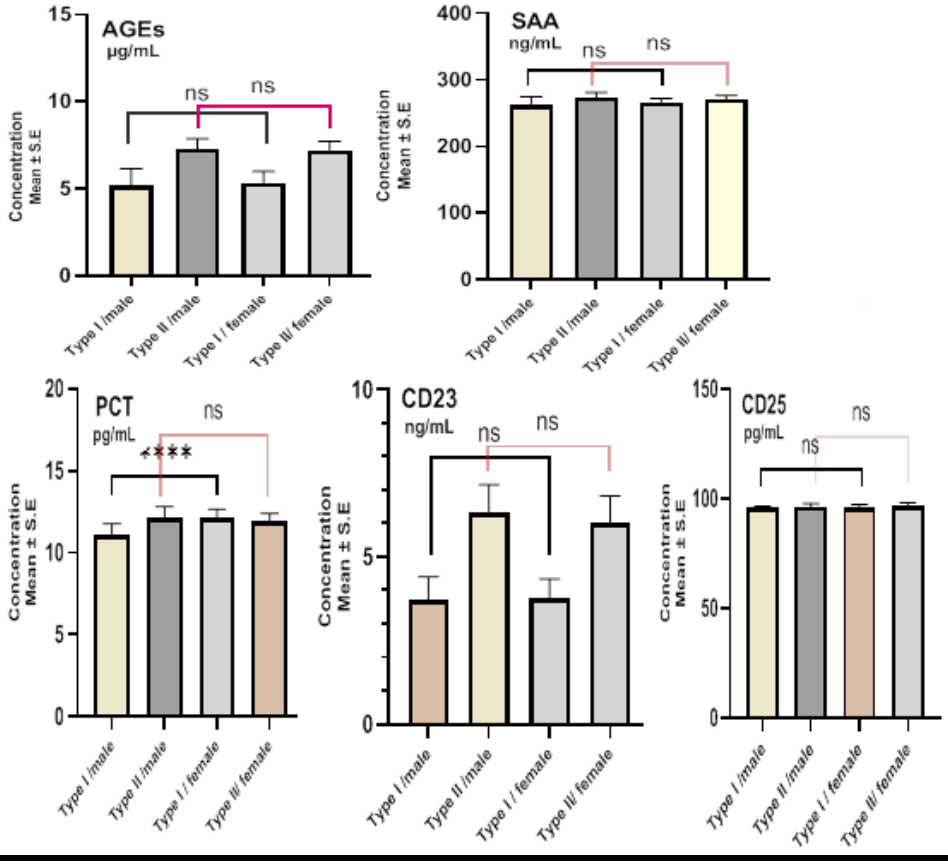

Fig 2. Relationship between AGEs, SAA, PCT, CD23 and CD25 and sex in studied groups.

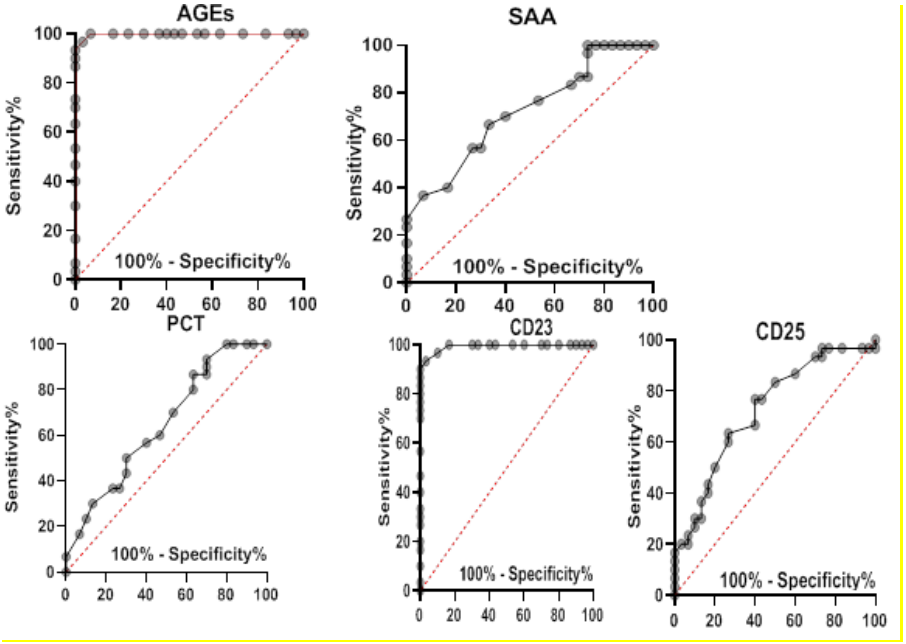

Fig 3. ROC analysis of AGEs, SAA, PCT, CD23 and CD25 in TDI and TDII patients.

Table 4 shows the Pearson correlation coefficient for the relationships between the various indicators studied AGEs, SAA, PCT, CD23 and CD25 in UTIs infections with TDI and TDII patients. The control groups for AGEs showed negative associations with several parameters. This study displays a strong negative correlation with DM II AGEs (- 0.54), representing that as levels of control AGEs increase, the levels of DM II AGEs tend to decrease. SAA type I displayed a strong positive correlation with SAA type II (0.62), suggesting that both parameters incline to increase together, suggesting that they may be influenced by similar factors. PCT type I showed the highest positive correlation with CD25 than the control group (0.71), suggesting a strong relationship between there two parameters in this condition.

Discussion

UTIs caused by *E. coli* bacteria are common between diabetics. This is due to an imbalance in the immune response and weak defense mechanisms linked with diabetes mellitus diseases (16).

Chronic inflammation can happen as a result of AGEs binding to its receptor (RAGE) and a number of pro-inflammatory cytokines are activated upon this binding. These cytokines are frequently observed in diabetic mellitus disease, where bad glucose management often leads to high levels of AGEs and It can also lead to immune dysfunction, this inflammation is one of the main reasons that aggravate diabetes complications (18). AGEs weaken the function of blood vessel endothelium and white blood cells, reducing the capacity of the immune system to fight infection and making diabetic mellitus more susceptible to infections such as UTIs (19). Accumulation of AGEs may increase the probability and severity of UTIs in diabetic mellitus patients, exacerbating inflammation and weakening immune function and this accumulation can be reduced by monitoring blood sugar levels (20). The

accumulation of AGEs impairs immune response and thus can increases the risk of infection in individuals with diabetic mellitus (21).

One of the immune parameters in the body is SAA, which is an essential parameter of the presence of inflammation and its high levels indicate an immune response to bacterial infections, including UTIs specially those caused by *E. coli* (22). Diabetic mellitus is correlated with a dysfunctional immune response, which weakens the immune system capacity to fight diseases such as UTIs. SAA levels may persist higher in diabetic mellitus with a history of recurrent UTIs, representing persistent inflammation and an increased risk of future infections (23).

Procalcitonin is widely used a diagnostic marker for bacterial infections. In Diabetic mellitus patients, PCT levels rise in response to UTIs caused by *E. coli*. Because *E. coli* is the most public pathogen causing UTIs, measure PCT levels can assistance distinguish between bacterial infections and non-bacterial infections, especially in diabetic mellitus patients who may have a weakened immune response (24). Higher PCT levels in patients with diabetic mellitus are associated with more severe bacterial infections including UTIs (25). Levels of PCT have important role to help clinicians detect whether a UTI is bacterial type such as those caused by *E. coli* and this is important in diabetic mellitus patients who may have other illness such as cystitis or interstitial nephritis, that present with like symptoms but need unlike treatment lines (26). CD23 plays an important role in regulating the immune response through B cell activation and differentiation. The immune response to *E. coli* infection may be affected by changes in CD23 expression that occur during bacterial infections, such as UTIs. In diabetic mellitus patients, as a result of a malfunction of the immune system, CD23 regulation may occur, which may affect their capacity to create a successful immune response against *E. coli* infection (27). CD23 protein levels may rise through infection, as previous studies have shown that CD23 protein contributes to the inflammatory response (28). Previous studies have shown that the genetic expression of the CD23 gene may change in diabetic mellites diseases, which may affect the production of antibodies and the function of the immune cells. Because of this immune defect, diabetics may be more susceptible to severe and recurrent UTIs (29). Diabetic patients normally suffer from recurrent and chronic UTIs. CD23 protein has been shown to regulate immune memory and may contribute to the etiology of recurrent UTIs (30). CD25 is a critical component of the interleukin-2 (IL-2) receptor, which is involved in T cell activation and immune responses during infections. In UTIs caused by *E. coli*, CD25 plays an essential role in modulating the immune response. Diabetic patients, who often have impaired immune function, may have altered CD25 expression, which can affect T cell responses and their ability to control bacterial infections such as UTIs (31). CD25 is involved in the regulation of inflammation, particularly in autoimmune and inflammatory responses. In diabetic patients, who are prone to chronic low-grade inflammation, CD25 expression can be upregulated during infections such as UTIs. Increased CD25 expression may reflect a heightened inflammatory response in diabetic patients with *E. coli* infections, which can contribute to tissue damage and complications such as pyelonephritis (32). Diabetic patients often exhibit immune dysregulation, characterized by an impaired ability to respond to infections effectively. CD25, through its role in T cell activation and immune tolerance, is involved in modulating this dysregulation. In UTIs caused by *E. coli*, the immune system's inability to effectively activate CD25-expressing T cells may result in an ineffective response to the infection, leading to more severe or recurrent infections (33). The role of CD25 in predicting the outcome of UTIs caused by *E. coli* in diabetic patients is important, especially considering that diabetes can

delay the immune response to infections. Studies suggest that monitoring CD25 levels could help predict the severity and prognosis of UTIs in these patients, providing insights into whether the infection is likely to resolve or progress to more severe conditions, such as pyelonephritis or sepsis (34).

Conclusion

Inflammatory parameters play a key role in the pathogenesis of UTIs caused by *E. coli* in diabetic patients. Diabetes exacerbates the inflammatory response, increasing susceptibility to infections and complicating treatment. Monitoring these markers can help assess infection severity and guide therapeutic interventions, emphasizing the need for targeted approaches in diabetic individuals with UTIs.

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Ethics approval and consent to participate

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Conflict of interest

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