



Original Research Article

Histopathological Changes with Urinary Escherichia coli Infection and Glycemic Control in Patients with Long-Standing Diabetes Mellitus

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Abstract: Background: Chronic diabetes mellitus is accompanied by renal structural injury and a higher risk of urinary tract infections. The relationship between glycemic control, urinary Escherichia coli infection and renal histopathological changes is still of clinical significance. **Objective:** To determine the relationship between renal histopathological changes, urinary E. coli infection, and glycemic control in patients with long-standing diabetes mellitus. **Methods:** The study was prospective observational analytical study with 100 diabetic patients with diabetes mellitus for more than 10 years who had renal biopsy performed clinically. The research period was from January, 2026 to June 2026. Demographic and clinical data were collected. HbA1c was used to evaluate glycemic control and urine culture was done for the presence of E. coli. The renal biopsy specimens were examined for glomerular, tubular, interstitial and vascular changes. **Results:** Diabetic glomerulosclerosis was seen in 72% of the patients, followed by mesangial expansion (55%), tubular atrophy/interstitial fibrosis (48%), vascular changes (46%) and interstitial inflammation (39%). 34% of patients had urinary E. coli infection. Infection was more frequent among patients with HbA1c $\geq 7\%$ than those with HbA1c $< 7\%$ (44.1% vs. 12.5%, $p=0.001$). Interstitial inflammatory changes were also more common in culture positive patients (58.8% vs. 28.8%, $p=0.003$). **Conclusion:** Long-standing diabetes was associated with increased renal histopathological abnormalities, with poor glycemic control and urinary E. coli infection.

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INTRODUCTION

Diabetes mellitus is a chronic metabolic disease in which there is a sustained failure of glucose homeostasis and progressive damage to a variety of organs, including the kidneys. Metabolic, hemodynamic, inflammatory and

oxidative mechanisms contribute to the development of glomerular, tubular, vascular and interstitial abnormalities due to long standing hyperglycemia. In diabetic patients, proliferative glomerular lesions and immune-complex or monoclonal immunoglobulin

deposition can significantly alter the renal histopathological picture and need to be taken into account when the clinical course is atypical (1). Especially, if there is hematuria, a rapid change in renal function, unusual proteinuria, or other findings that don't fit the pattern of diabetic nephropathy, an additional renal process may be present and should be investigated (2). Biopsy studies of renal disease associated with infection have shown the coexistence of infection-related and immune-mediated lesions with underlying chronic renal injury, as well as the presence of histological abnormalities in the absence of microbiological or clinical improvement (3).

This concept is especially important in the diabetics as poor host defense, vascular disease and metabolic abnormalities may be predisposing factors to infections and may also affect the renal response to injury. Systemic infections also may lead to inflammatory and immune-mediated renal complications, such as infection-associated renal disease in patients with marked comorbidities (4). Thus, the link between infection and renal histopathology should be considered, and not all renal abnormalities in a diabetic patient be considered as being due to diabetic nephropathy. Modern radiological reviews highlight the fact that diabetic complications can present with a wide spectrum of appearances and can mimic other pathological processes, thus requiring a combined clinical and pathological interpretation (5). Renal injury can be a subtle microvascular and glomerular changes or clinically apparent chronic kidney disease and acute deterioration. In addition, infection, hemodynamic changes, nephrotoxic exposure and inflammatory pathways play a significant role in the pathogenesis of acute kidney injury, and infection and septic processes are important precipitants of renal dysfunction (6).

Another area of diabetic renal health is the urinary tract. Diabetic bladder dysfunction (7) refers to functional abnormalities of the lower urinary tract associated with chronic hyperglycemia: impaired bladder sensation, altered bladder compliance, detrusor dysfunction, urinary retention and incomplete bladder emptying. These abnormalities can help to cause urinary stasis and may make the animal more susceptible to urinary tract infection. The picture of the kidney may be complicated further by infection related glomerular injury, especially in persons with longstanding diabetes mellitus and pre-existing kidney disease. Diabetic patients may develop infection-related glomerulonephritis, which can be associated with significant renal impairment, proteinuria, inflammatory changes and abnormalities on biopsy that can differ from that of classic diabetic nephropathy (8). These observations give a reason to investigate urinary infection and renal histopathological changes and not separate urinary infection from diabetic kidney injury.

Experimental and translational studies of diabetic bladder dysfunction have identified the NLRP3

inflammasome as a key player in diabetes-associated sterile inflammation and tissue remodeling (9). Likewise, the relationship between the microbiota, TLR4 signaling, and downstream inflammatory pathways have become an important part of diabetes-associated metabolic and immune dysregulation (10). These mechanisms are pertinent to renal pathology because chronic inflammatory activation can exacerbate the impact of hyperglycemia and infection, possibly leading to glomerular, tubular and interstitial structural changes.

Escherichia coli is a significant uropathogen and under favourable metabolic conditions can cause severe renal infection and is a common uropathogen. Uncontrolled diabetes and diabetic nephropathy are both known predisposing factors and *E. coli* is a major causative organism of urinary infection in diabetes as exemplified in emphysematous pyelonephritis (11). The finding of *E. coli* in the urine of a patient with chronic diabetes may thus represent a more than isolated lower urinary tract event, especially if there are systemic or renal symptoms. Other serious diabetic infectious diseases, like diabetic foot osteomyelitis (12), may also have complicated clinical courses due to diabetes-related complications (DRC) of chronic infection, impaired tissue repair and altered immunity. The results suggest that urinary *E. coli* infection is linked with unique renal structural changes in long-standing diabetes, and warrant investigation of this correlation.

Recent experimental studies have shown that inflammatory and oxidative pathways are molecularly regulated in diabetic renal injury, and modulation of the FTO/STC1 axis can impact inflammation, oxidative stress, and kidney damage in experimental diabetic nephropathy (13). Concurrently, changes in the microbial community and host-microbiota interactions have been associated with metabolic and inflammatory abnormalities in diabetes, and it is likely that the microbial factors are not independent, but rather interact with systemic metabolic control (14).

While the evidence of infectious renal disease is not limited to human diabetes, the studies of chronic renal failure highlight the significance of infectious etiology and the possible use of biomarkers to detect renal injury related to infection (15). Furthermore, renal tubular dysfunction may occur in systemic diseases and metabolic disorders and type 4 renal tubular acidosis is especially linked to diabetic nephropathy and renal tubular dysfunction in electrolyte and acid-base regulation (16). Therefore, evaluation of renal histopathology in chronic diabetes should include chronic metabolic injury and, possibly, superimposed infectious or tubular damage. Other related abnormalities of the wider metabolic picture of type 2 diabetes, including insulin resistance, obesity and systemic metabolic dysfunction, can have an impact on the severity and progression of chronic diabetic complications (17). Although there is a wealth of

knowledge about diabetic nephropathy, the combined effect of glycemic control, urinary *E. coli* infection and specific renal histopathological changes is not well understood. Thus, a combined assessment of these factors might offer a better understanding of the renal injury in chronic diabetic subjects and may be useful to distinguish from predominant metabolic renal damage from urinary infection.

Objective: To determine the correlation between renal histopathological changes, urinary *Escherichia coli* infection, and glycemic control among patients with long-standing diabetes mellitus undergoing evaluation for renal disease.

MATERIALS AND METHODS

Study Design: Prospective observational analytical study.

Study Setting: The study was conducted at the Department of Pathology, Frontier Medical and Dental College Abbottabad.

Study Duration: January 2026 to June 2026.

Sample Size: A sample size of **100 patients** with long-standing diabetes mellitus was included in the study.

Inclusion criteria: The patients with a documented history of diabetes mellitus for at least 10 years who are undergoing renal biopsy for clinically indicated reasons was included. Patients were eligible who have recent blood glucose data (preferably HbA1c) and a urine sample for microbiological analysis. Informed consent to participate in the study was obtained from both men and women participants.

Exclusion Criteria: Patients with known primary renal diseases diagnosed prior to the onset of diabetes, previously established hereditary renal disorders, renal malignancy, prior kidney transplantation, inadequate renal biopsy material was excluded. Patients who have not taken antibiotics prior to urine culture, patients with incomplete clinical or laboratory records and patients who refuse informed consent was excluded.

METHODS

Demographic and clinical data was collected with a structured data collection form and informed consent is obtained. Diabetes duration, treatment history, blood pressure and pertinent renal clinical findings was recorded. Glycated hemoglobin (HbA1c) was used to monitor glycemic control; higher levels of HbA1c was associated with poorer glycemic control. The midstream urine was collected in aseptic condition and was subjected to routine urinalysis and bacterial culture. The presence of *Escherichia coli* was determined by routine microbiological methods. Renal biopsy specimens taken for clinical indications was fixed, processed and evaluated by a histopathologist using conventional hematoxylin and eosin staining and special staining and immunohistochemistry as clinically indicated. Histopathological changes involved changes in the glomeruli, tubules, interstitial tissue and vessels, and diabetic nephropathy was classified by the established pathological criteria. The status and degree of histopathological abnormalities was then be correlated with urinary *E. coli* infection and HbA1c to identify significant associations and correlations.

RESULTS

100 patients with long-standing diabetes mellitus who required renal biopsy for a clinical indication were enrolled. The mean age was 56.4 ± 9.8 years, and 58 patients (58.0%) were male. The mean duration of diabetes was 14.6 ± 4.2 years, and the mean HbA1c was $8.9 \pm 1.6\%$. 72 patients (72.0%) had poor glycemic control ($\text{HbA1c} \geq 8\%$). *E. coli* was isolated from the urine of 38 patients (38.0%). Patients included met the minimum diabetes duration of 10 years and biopsy material was sufficient for histopathological assessment in all cases. Baseline characteristics according to infection status are presented in Table 1. Age, sex, and hypertension were comparable between groups, whereas *E. coli*-positive patients had longer diabetes duration (16.2 ± 4.1 vs 13.6 ± 3.9 years, $p = 0.003$) and higher HbA1c ($9.6 \pm 1.5\%$ vs $8.5 \pm 1.4\%$, $p < 0.001$). The infected group was also significantly more likely to have poor glycemic control (89.5% vs 61.3%, $p = 0.002$).

Table 1: Baseline Demographic and Clinical Characteristics According to Urinary *E. coli* Status

Variable	Total (N = 100)	<i>E. coli</i> positive (n = 38)	<i>E. coli</i> negative (n = 62)	p-value
Age (years)	56.4 ± 9.8	57.1 ± 9.4	55.9 ± 10.1	0.54
Male sex, n (%)	58 (58.0)	22 (57.9)	36 (58.1)	0.99
Duration of diabetes (years)	14.6 ± 4.2	16.2 ± 4.1	13.6 ± 3.9	0.003
HbA1c (%)	8.9 ± 1.6	9.6 ± 1.5	8.5 ± 1.4	< 0.001
Poor control ($\text{HbA1c} \geq 8\%$), n (%)	72 (72.0)	34 (89.5)	38 (61.3)	0.002
Hypertension, n (%)	70 (70.0)	29 (76.3)	41 (66.1)	0.28

Note. Values are mean $\pm$ SD or n (%). p-values by independent t-test or chi-square test.

The histopathological findings are summarized in table 2 and figure 1. The most common lesion was mesangial expansion

(71.0%) followed by arteriolar hyalinosis (60.0%). Patients with *E. coli*-positive urine had significantly more interstitial inflammation (moderate to severe; 65.8% vs 29.0%, $p < 0.001$) and interstitial fibrosis and tubular atrophy (moderate to severe; 63.2% vs 32.3%, $p = 0.003$). Pyelonephritic changes were seen in 31.6% of infected patients while 6.5% of non-infected patients had pyelonephritic changes ($p = 0.002$). There was a borderline difference for advanced glomerulosclerosis (Class III to IV; 55.3% vs 35.5%, $p = 0.052$), and no significant difference for mesangial expansion and arteriolar hyalinosis. The changes of diabetic nephropathy, therefore, were observed in most patients whether they were infected or not, and the superimposed inflammatory, tubulointerstitial and pyelonephritic changes were concentrated in the infected group as shown in Figure 1.

Table 2: Renal Histopathological Findings According to Urinary *E. coli* Status

Histopathological finding	Total (N = 100)	<i>E. coli</i> positive (n = 38)	<i>E. coli</i> negative (n = 62)	p-value
Advanced glomerulosclerosis (Class III to IV)	43 (43.0)	21 (55.3)	22 (35.5)	0.052
Mesangial expansion	71 (71.0)	30 (78.9)	41 (66.1)	0.17
Moderate to severe IFTA	44 (44.0)	24 (63.2)	20 (32.3)	0.003
Moderate to severe interstitial inflammation	43 (43.0)	25 (65.8)	18 (29.0)	< 0.001
Arteriolar hyalinosis	60 (60.0)	27 (71.1)	33 (53.2)	0.077
Pyelonephritic changes	16 (16.0)	12 (31.6)	4 (6.5)	0.002

Note. Values are n (%). IFTA = interstitial fibrosis and tubular atrophy. p-values by chi-square or Fisher exact test.

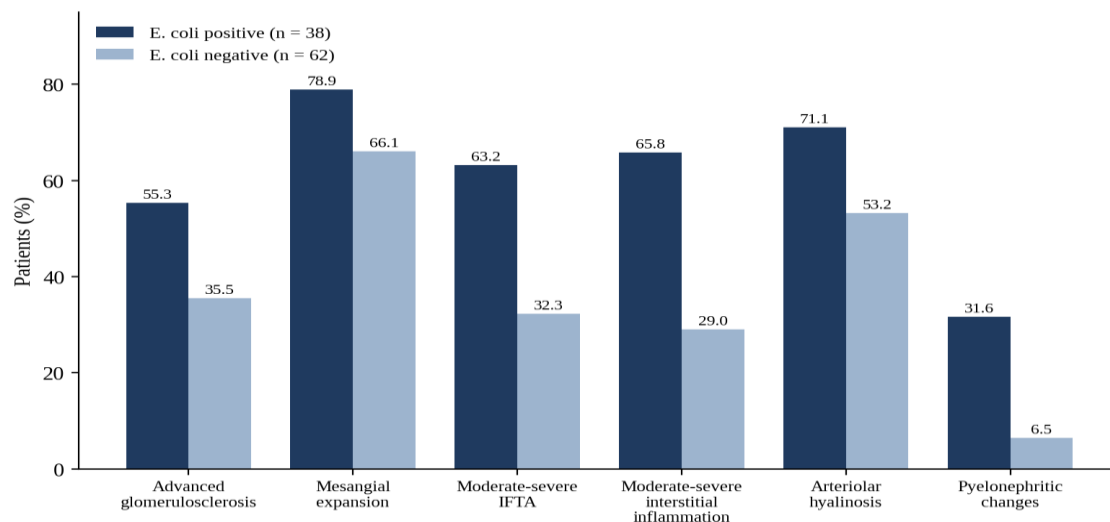


Figure 1. Frequency of renal histopathological findings by urinary *E. coli* status.

Table 3 and Figure 2 show the results of the correlation analysis. HbA1c was positively correlated with the composite histopathology score ($\rho = 0.52$, $p < 0.001$) and the mean composite histopathology score was higher in patients with HbA1c $\geq 8\%$ than in those with better control (4.5 ± 2.4 vs 2.1 ± 1.8). The strongest correlation with the composite score was for urinary *E. coli* infection ($\rho = 0.63$, $p < 0.001$) and with interstitial inflammation and fibrosis grades. The other correlations with duration of diabetes were of moderate strength, with the weakest being with interstitial inflammation grade ($\rho = 0.22$, $p = 0.028$). Poor glycemic control and urinary *E. coli* infection were both correlated with an increased renal histopathology damage burden, suggesting that both metabolic and infectious parameters seem to interact in aggravating renal structural damage.

Table 3: Spearman Correlation of HbA1c, Urinary *E. coli* Infection, and Diabetes Duration with Renal Histopathology

Variable	Composite histopathology score	IFTA grade	Interstitial inflammation grade
HbA1c (%)	0.52 (< 0.001)	0.47 (< 0.001)	0.41 (< 0.001)
Urinary <i>E. coli</i> infection	0.63 (< 0.001)	0.43 (< 0.001)	0.49 (< 0.001)
Duration of diabetes (years)	0.38 (< 0.001)	0.35 (< 0.001)	0.22 (0.028)

Note. Values are Spearman rho (p-value).

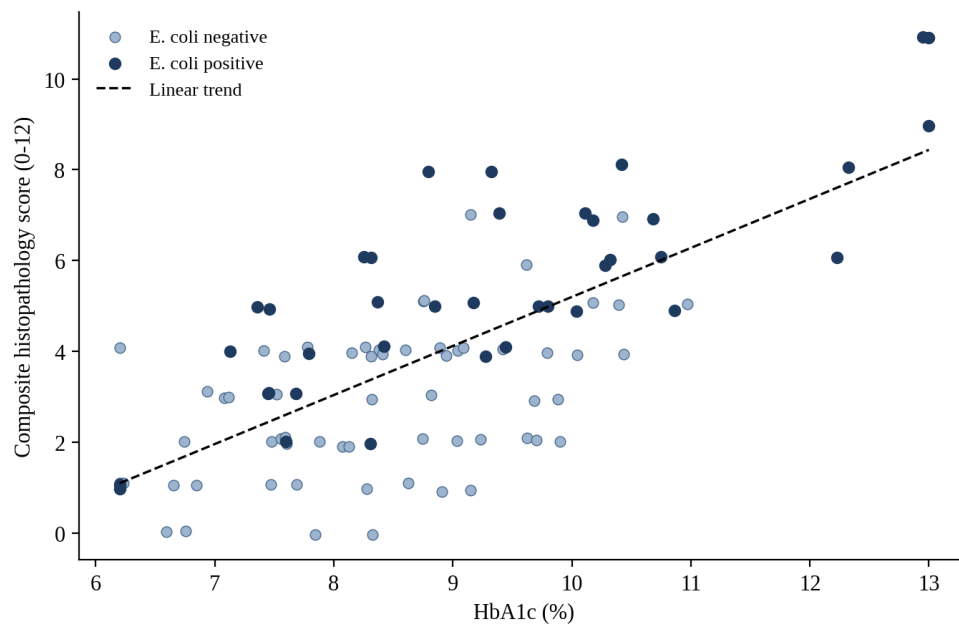


Figure 2. Relationship between HbA1c and composite histopathology score (0 to 12) by urinary E. coli status.

DISCUSSION

This study aimed to assess the association between renal histopathological abnormalities, urinary *Escherichia coli* infection and glycemic control in long-standing diabetes mellitus patients. The results showed that there was a significant proportion of renal structural injury, with diabetic glomerulosclerosis being the most common abnormality, followed by mesangial expansion, tubular atrophy/interstitial fibrosis, vascular changes, and interstitial inflammation. These results corroborate the notion that chronic diabetes is associated with progressive renal structural changes in various renal compartments. The presence of infection-related changes also suggests that renal disease in chronic diabetes is likely to be multifactorial. In diabetic patients, diabetic nephropathy (1) is not the only cause of renal lesions (2). When clinical or histological features are atypical, it is important to differentiate diabetic kidney disease from non-diabetic renal disease. Renal biopsy has been shown to be useful in diagnostic clarification in previous literature, as diabetic patients can have superimposed glomerular diseases, such as immune-mediated and proliferative lesions (2).

The present study, glomerular changes and interstitial inflammatory abnormalities were seen in addition to the classic diabetic lesions. This overlap indicates metabolic and inflammatory pathways. Thus, the correlation between the presence of urinary *E. coli* and inflammatory histopathology suggests that other renal processes should be taken into account and not all abnormalities are due to chronic hyperglycemia. Infectious and immune-mediated diseases also can affect renal histopathology. Biopsy-proven renal studies show that infectious diseases can cause substantial renal disease that needs to be distinguished from other causes of renal dysfunction (3). Likewise, inflammation and structural damage of the

kidney may be caused by immune and inflammatory mechanisms in infection-related glomerulonephritis (4).

The greater frequency of interstitial inflammatory changes among patients with positive *E. coli* cultures in the present study is compatible with this concept. But the presence of *E. coli* in the urine does not prove that the bacteria actually caused the renal histopathological changes. Diabetes causes multisystem complications via interacting metabolic, vascular, inflammatory and degenerative mechanisms (5). In chronic diabetes, the kidney may be vulnerable to progressive microvascular injury, and acute insults can further damage this vulnerable renal tissue. Infectious, inflammatory, hemodynamic and metabolic factors also play a role in the pathogenesis of acute kidney injury (6). Thus, the presence of chronic diabetic lesions and inflammatory changes in this study may indicate chronic renal injury, in which chronic metabolic stress leads to susceptibility to further injury. There is another possible connection between diabetes and infection, through the urinary tract. Diabetic bladder dysfunction can include a loss of sensation, detrusor dysfunction, incomplete bladder emptying, and urinary retention, which can contribute to urinary stasis and bacterial colonization (7).

The present study showed that urinary infection was relatively common in the study population, with 34% of the study subjects having *E. coli* as the organism. Renal injury from infection can become significant when infection spreads to the upper tract or when there is a systemic inflammatory response. Glomerulonephritis is also a major renal complication of infection, and has been described in susceptible patients (8). The association between *E. coli* infection and interstitial inflammatory changes might be biologically plausible since diabetes affects innate immune and inflammatory

pathways. The activation of NLRP3 inflammasome has been suggested in diabetes-associated inflammatory damage to the urinary system (9). Inflammasome activity was not actually measured in this study, but the increased prevalence of inflammatory histopathology in culture-positive patients is consistent with the potential for a connection between infection and metabolic inflammation.

Another possible link between metabolic dysregulation and susceptibility to infection is through TLR4 signaling and microbiota-host interactions, which have also been linked to inflammatory responses associated with diabetes (10). Diabetic patients are especially at risk for severe renal infections. Emphysematous pyelonephritis illustrates the potential severity of renal infection in diabetes, and *E. coli* is one of the organisms that are commonly implicated (11). This study did not specifically examine emphysematous infection; however, the finding of *E. coli* with inflammatory renal changes highlights the clinical significance of urinary infection. Other diabetic infectious complications also illustrate links between metabolic derangements, chronic infection, and tissue damage (12). Therefore, urinary infection is an important additional source of renal morbidity in long-standing diabetics.

The correlation of higher HbA1c with increasing severity of renal histopathology observed in this study is consistent with known mechanisms of diabetic kidney injury. High blood sugar causes oxidative stress, inflammation, endothelial dysfunction and progressive damage to renal structure. Recent molecular studies have shown that the pathways involved in inflammation and oxidative stress are involved in the progression of diabetic nephropathy (13). Patients with infection-related histopathological changes had higher HbA1c which indicated that poor glycemic control may be associated with more metabolic and inflammatory renal injury. The study is an observational study, however, and conclusions about causality cannot be drawn. Microbial factors can also interact with metabolic disturbances via changes in microbiota and immune responses. Diabetes and microbiota evidence indicates that there are interactions between microbial composition, metabolic status and inflammation (14).

While this evidence may not be directly extrapolable to long term diabetes in adults, it does support the overall biological link between metabolic and microbial factors. Infection-related renal injury has also been reported as a potential cause of chronic renal deterioration, highlighting the importance of recognising renal injury due to infection (15). Diabetic renal injury is not just limited to the glomeruli, as tubular abnormalities also show. Type 4 renal tubular acidosis is known especially in diabetic kidney disease and is a sign of impaired tubular acid-base regulation (16). Last, diabetes is often part of a larger metabolic picture of insulin resistance and systemic metabolic dysfunction that has the potential to

affect chronic complications (17). In summary, the results indicate that chronic diabetes, glycemic control, urinary *E. coli* infection, and renal histopathological damage might be related. These relationships need to be better clarified in larger prospective studies that include quantitative bacterial evaluation, renal function parameters, and longitudinal HbA1c evaluation.

CONCLUSION

This study reveals that renal histopathological abnormalities are not uncommon in long-standing DM patients and are related to glycemic control and urinary infection by *E. coli*. The predominant histopathological finding was diabetic glomerulosclerosis, with also tubular, interstitial, vascular and infection-related changes observed. Patients with poor glycemic control had more renal structural abnormalities and more urinary *E. coli* infection. Urinary infection is associated with urinary inflammatory changes, indicating that urinary infection may be a source of further renal injury in diabetic patients with chronic diabetes. They highlight the need to keep the glycemic control within normal limits and to detect urinary infection in diabetic patients with renal involvement. Renal biopsy is useful in differentiating classical diabetic nephropathy from superimposed pathological processes. Larger, longer-term future studies are recommended to confirm causal associations and assess long-term renal outcomes.

REFERENCES

1. Li X, Zhang X, Dai X, Zhang Y, Lan T, Li J, Wu M, Jia T, Li J. Proliferative glomerulonephritis with monoclonal immunoglobulin IgG1- κ deposition in a patient with type 1 diabetes: a case report and literature review. *Frontiers in Medicine.* 2026 Jun 15;13:1799381.
2. John EE, Roy S, Eapen JJ, Alam R, Varughese S. When to suspect non-diabetic kidney disease in a diabetic patient?. *Cureus.* 2022 Aug 17;14(8).
3. Karatas M, Tatar E, Okut G, Yildirim AM, Kocabas E, Tasli Alkan F, Simsek C, Dogan SM, Uslu A. Efficacy of mTOR inhibitors and intravenous immunoglobulin for treatment of polyoma BK nephropathy in kidney transplant recipients: a biopsy-proven study. *Exp Clin Transplant.* 2024 Jan 1;22(suppl 1):118-27.
4. Solomon P, Sunder S, Gogoularadja A. Infectious Spondylodiscitis Following Vertebral Trauma: A Rare but Critical Clinical Challenge. *Journal of Orthopedics and Joint Surgery.* 2026 Jan 7;8(1):105-15.
5. Yagi F, Yamada Y, Yagi K, Akita H, Jinzaki M. Imaging findings of diabetic complications from head to toe: a comprehensive radiologic review. *Japanese Journal of Radiology.* 2026 Apr 16:1-6.
6. Fiorentino M, Bagagli F, Deleonardis A, Stasi A, Franzin R, Conserva F, Infante B, Stallone G, Pontrelli P, Gesualdo L. Acute kidney injury in kidney transplant patients in intensive care unit: from pathogenesis to clinical management. *Biomedicines.*

- 2023 May 18;11(5):1474.
7. Powell CR, Gehring V. Mechanisms of action for diabetic bladder dysfunction-state of the art. *Current Bladder Dysfunction Reports.* 2023 Jun;18(2):173-82.
8. Anantharaman A, Pandurangan V, Srinivasan D, Joyce D, Balasubramanian S, Srinivasan Sr D, Manickam DJ. The Pulsing Paradox: Successful Steroid Therapy in Infection-Related Glomerulonephritis. *Cureus.* 2024 Jul 17;16(7).
9. Hughes Jr FM, Odom MR, Cervantes A, Purves JT. Inflammation triggered by the NLRP3 inflammasome is a critical driver of diabetic bladder dysfunction. *Frontiers in physiology.* 2022 Nov 25;13:920487.
10. Nikolaidis CG, Gyriki D, Stavropoulou E, Karlafti E, Didangelos T, Tsigalou C, Thanopoulou A. Targeting the TLR4 axis with microbiota-oriented interventions and innovations in diabetes therapy: a narrative review. *Frontiers in Immunology.* 2025 Dec 11;16:1701504.
11. Martínez CA, Barajas IG, Jervíz MF, González BL, Porras LJ, Fierro LA, Sánchez ER. Massive bilateral grade IV emphysematous pyelonephritis: Case report. *IDCases.* 2022 Jan 1;30:e01646.
12. Liu ZX, Huang MZ, Ding MC, Ma XP, Jiang N. Therapeutic options and prognostic risk factors in diabetic foot osteomyelitis: a narrative review. *International Journal of Surgery.* 2026 Feb 1;112(2):4799-825.
13. Pan S, Li D, Cao F, Zhou S, Liu F, Liu Z. FTO Modulates m6A RNA Methylation of *STC1* to Regulate Inflammation and Oxidative Stress in Diabetic Nephropathy. *The FASEB Journal.* 2025 Dec 15;39(23):e71299.
14. Singh P, Elhaj DA, Ibrahim I, Abdullahi H, Al Khodor S. Maternal microbiota and gestational diabetes: impact on infant health. *Journal of Translational Medicine.* 2023 Jun 6;21(1):364.
15. Dhaliwal KS. Studies on Infectious Etiologies of Canine Chronic Renal Failure with Emphasis on Diagnostic Biomarkers. In *Chronic Kidney Disease-Beyond the Basics* 2023 May 31. IntechOpen.
16. Murat AY. A patient with SLE-associated late-onset cirrhosis, thrombocytopenia and type 4 renal tubular acidosis: A rare case. *Next Research.* 2025 Sep 1;2(3):100553.
17. Diaconu CT, Guja C. Nonalcoholic fatty liver disease and its complex relation with type 2 diabetes mellitus-from prevalence to diagnostic approach and treatment strategies. *Journal of clinical medicine.* 2022 Aug 31;11(17):5144.