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Urinary Tract Infection as an Independent Predictor of Myocardial Injury in Patients with Diabetes Mellitus A Laboratory-Based Cardiovascular Study

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Abstract

Background: Diabetes mellitus makes people more vulnerable to infections and subclinical cardiac damage, however it is yet unknown whether UTIs may cause myocardial damage on their own in diabetic individuals.

Objective: To evaluate urinary tract infection as an independent predictor of myocardial injury in patients with diabetes mellitus through laboratory-based cardiovascular assessment.

Methodology: This prospective observational analytical study was conducted from January 2023 to June 2024. A total of 210 adult patients with confirmed diabetes mellitus were enrolled and categorized into those with UTI (n = 108; 51.43%) and without UTI (n = 102; 48.57%). Demographic and clinical data were collected, and laboratory investigations included urine culture, fasting blood glucose, HbA1c, high-sensitivity cardiac troponin (hs-Tn), and B-type natriuretic peptide (BNP). Laboratory-defined myocardial injury was considered present if hs-Tn was elevated above the reference limit. Comparative analysis

was performed using t-tests and chi-square tests, and multivariate logistic regression was applied to identify independent predictors of myocardial injury.
Results: Mean age was comparable between groups (57.2 ± 9.6 vs 55.6 ± 10.0 years; p = 0.214), while females were more frequent in the UTI group (60/108; 55.56% vs 38/102; 37.25%; p = 0.009). Patients with UTI had higher HbA1c (8.8 ± 1.3 vs 8.0 ± 1.1; p < 0.001) and hypertension prevalence (72/108; 66.67% vs 52/102; 50.98%; p = 0.021). Elevated hs-Tn and BNP were more common in the UTI group (hs-Tn: 39/108, 36.11% vs 14/102, 13.73%; BNP: 34/108, 31.48% vs 16/102, 15.69%). Laboratory-defined myocardial injury occurred in 53/210 patients (25.23%), more frequently in the UTI group (38/108; 35.19%) compared to the non-UTI group (15/102; 14.71%; p < 0.001). Multivariate analysis confirmed UTI (adjusted OR = 2.84; 95% CI: 1.45–5.57; p = 0.002) and HbA1c ≥8% (adjusted OR = 2.31; 95% CI: 1.14–4.68; p = 0.019) as independent predictors of myocardial injury.
Conclusion: Urinary tract infection is an independent predictor of laboratory-defined myocardial injury in patients with diabetes mellitus.

Introduction

Defects in insulin production, action, or both may cause persistent hyperglycemia, which is a hallmark of diabetes mellitus (DM), a chronic metabolic disease [1]. It is a powerful and well-established risk factor for cardiovascular disease (CVD), which accounts for a large portion of morbidity and death worldwide [2]. Patients with diabetes often have subclinical myocardial damage in addition to overt ischemia episodes. This is caused by oxidative stress brought on by chronic hyperglycemia, endothelial dysfunction, microvascular disease, and low-grade systemic inflammation. The diabetic myocardium is more vulnerable to other physiological stresses as a result of these processes [3,4].

One of the most prevalent bacterial infections in people with diabetes is urinary tract infection (UTI), which is more frequent, more severe, and more likely to recur than in those without the disease [5]. Diabetic individuals are at risk for complex UTIs due to a combination of impaired immunological response, autonomic bladder dysfunction, and glycosuria [6]. Although UTIs have historically been thought of as localized infections, there is growing evidence that, even in the absence of underlying obstructive coronary artery disease, acute systemic inflammatory responses linked to infections may cause myocardial stress, ischemia, and damage [7,8].

Microvascular dysfunction, endothelial activation, increased myocardial oxygen demand, and the release of inflammatory cytokines are all brought on by systemic infections [9]. cardiac damage may be disproportionately caused by these infection-related inflammatory surges in individuals with diabetes, who already have baseline endothelial impairment and metabolic cardiac susceptibility [10]. Crucially, only sensitive laboratory indicators may be able to identify such damage, which may remain clinically silent [11].

Even in non-ischemic conditions, cardiac biomarkers such as B-type natriuretic peptide (BNP) and high-sensitivity cardiac troponins have become trustworthy markers of myocardial stress and subclinical myocardial damage [12]. Adverse cardiovascular outcomes have been associated with elevated levels of these markers during systemic infections. However, there is still a lack of research on the precise function of urinary tract infection as an independent predictor of myocardial damage in diabetic patients, especially in low- and middle-income environments where infections and diabetes are both quite common [13].

Finding UTIs to be a separate and perhaps controllable cause of cardiac damage may have significant effects on clinical surveillance, early cardiovascular risk assessment, and diabetic patient prevention measures. In order to ascertain whether UTI

independently predicts myocardial damage after controlling for pertinent variables, this research intends to assess the relationship between UTI and laboratory-defined myocardial injury in individuals with diabetes mellitus.

Research Objective

To evaluate the role of urinary tract infection as an independent predictor of myocardial injury in patients with diabetes mellitus through laboratory-based cardiovascular assessment

Methodology

Study Design and Setting

This prospective observational analytical study was conducted at the Al-Noor Hospital Sialkot, over an 18-month period from January 2023 to June 2024. The purpose of the research was to compare diabetes individuals with and without UTIs in order to ascertain if, using laboratory indicators, UTIs may independently predict cardiac damage.

Study Population

Participants were adults (18 years of age and older) having a verified diagnosis of type 1 or type 2 diabetes mellitus. In order to compare myocardial damage indicators between the two groups, participants were divided into diabetes individuals with a confirmed UTI and diabetic patients without any indication of a UTI.

Inclusion and Exclusion Criteria

The research comprised diabetes mellitus patients who gave their informed permission. Urinary symptoms accompanied by positive urine cultures and/or urinalysis results were considered indicative of a urinary tract infection. To prevent confounding from known cardiac pathology, patients with pre-existing cardiovascular illness, including past myocardial infarction or heart failure, were excluded. Pregnancy, immunosuppressive medication usage, active systemic infections other than UTIs, and chronic renal disease stage 3 or higher were further exclusions. To separate the independent impact of UTI on cardiac damage, these parameters were used.

Sample Size and Sampling Technique

During the research period, a convenience sample strategy was used to enroll 210 patients in total. The sample size was deemed sufficient to do multivariate regression analysis and identify clinically significant variations in myocardial damage biomarkers, even with the non-probability sampling technique.

Data Collection and Laboratory Assessment

A systematic proforma was used to record demographic and clinical information, such as age, gender, length of diabetes, glycemic control measured by HbA1c, and concomitant diseases including hypertension. B-type natriuretic peptide (BNP), fasting blood glucose, HbA1c, high-sensitivity cardiac troponin, and urine routine examination and culture were among the laboratory tests. In the absence of clinical or electrocardiographic indications of acute coronary syndrome, myocardial damage was operationally defined as elevation of cardiac troponin over the laboratory-specific upper reference limit, with or without concurrent BNP increase.

Statistical Analysis

SPSS version 25 was used to analyze the data. Whereas categorical data were shown as frequencies and percentages, continuous variables were represented as mean ± standard deviation. The independent t-test for continuous variables and the chi-square test for categorical variables were used to compare diabetes individuals with and without UTIs. Multivariate logistic regression analysis was performed after controlling for possible confounders such as age, gender, duration of diabetes, HbA1c levels, hypertension, and renal function in order to prove UTI as an independent predictor of myocardial damage. A p-value of less than 0.05 was deemed statistically significant, and adjusted odds ratios with 95% CIs were computed.

Ethical Considerations

Ethical approval for this study was obtained from the Ethics Review Committee of the Department of Allied Health Sciences, University of Sialkot. The research was subsequently conducted at Al-Noor Hospital, Sialkot, in accordance with the ethical principles governing research involving human subjects. Prior to participant enrollment, written informed consent was obtained from all participants.

Results

Among 210 diabetic patients, 108 (51.43%) had UTI and 102 (48.57%) did not (Table 1). Mean age was comparable between groups (57.2 ± 9.6 vs 55.6 ± 10.0 years; p = 0.214). Females were significantly more common in the UTI group (n=60; 55.56%) compared to the non-UTI group (n=38; 37.25%; p = 0.009). Patients with UTI had significantly higher HbA1c levels (8.8 ± 1.3 vs 8.0 ± 1.1; p < 0.001) and a higher prevalence of hypertension (66.67% vs 50.98%; p = 0.021), while the duration and type of diabetes were similar between groups.

Table 1. Baseline Demographic and Clinical Characteristics of the Study Population (n = 210)

Variable	UTI Present (n=108)	No UTI (n=102)	Overall (n=210)	p-value
Age (years), mean ± SD	57.2 ± 9.6	55.6 ± 10.0	56.4 ± 9.8	0.214
Male, n (%)	48 (44.44)	64 (62.75)	112 (53.33)	0.009
Female, n (%)	60 (55.56)	38 (37.25)	98 (46.67)	0.009
Duration of diabetes (years), mean ± SD	9.6 ± 4.5	8.6 ± 4.1	9.1 ± 4.3	0.118
HbA1c (%), mean ± SD	8.8 ± 1.3	8.0 ± 1.1	8.4 ± 1.2	<0.001
Hypertension, n (%)	72 (66.67)	52 (50.98)	124 (59.05)	0.021
Type 2 diabetes, n (%)	98 (90.74)	90 (88.24)	188 (89.52)	0.548

Diabetic patients with UTI demonstrated significantly higher cardiac biomarker abnormalities than those without UTI (Table 2). Elevated hs-troponin was observed in 36.11% of patients with UTI compared to 13.73% of those without UTI (p < 0.001), while elevated BNP levels were noted in 31.48% versus 15.69%, respectively (p = 0.006). Mean hs-troponin levels (29.6 ± 11.4 vs 18.2 ± 8.9 ng/L) and mean BNP levels (186.5 ± 72.1 vs 122.4 ± 55.6 pg/mL) were also significantly higher in the UTI group (p < 0.001).

Table 2. Laboratory Findings and Cardiac Biomarkers in Diabetic Patients with and Without UTI

Parameter	UTI Present (n=108)	No UTI (n=102)	p-value
Positive urine culture, n (%)	92 (85.19)	0 (0.00)	<0.001
hs-Cardiac troponin elevated, n (%)	39 (36.11)	14 (13.73)	<0.001
BNP elevated, n (%)	34 (31.48)	16 (15.69)	0.006
Mean hs-troponin (ng/L), mean ± SD	29.6 ± 11.4	18.2 ± 8.9	<0.001
Mean BNP (pg/mL), mean ± SD	186.5 ± 72.1	122.4 ± 55.6	<0.001

Overall, laboratory-defined myocardial injury was present in 25.23% of the study population (Table 3). The prevalence was significantly higher among diabetic patients with UTI (n=38; 35.19%) compared to those without UTI (n=15; 14.71%), representing more than a twofold increase in myocardial injury in the presence of UTI ($p < 0.001$).

Table 3. Prevalence of Laboratory-Defined Myocardial Injury in the Study Population

Myocardial Injury Status	UTI Present (n=108)	No UTI (n=102)	p-value
Myocardial injury present, n (%)	38 (35.19)	15 (14.71)	<0.001
Myocardial injury absent, n (%)	70 (64.81)	87 (85.29)	—

Univariate analysis demonstrated that myocardial injury was significantly associated with the presence of UTI (71.70% vs 44.59%; $p < 0.001$), age >60 years (54.72% vs 35.67%; $p = 0.015$), poor glycemic control defined as HbA1c ≥8% (77.36% vs 52.23%; $p = 0.002$), and hypertension (69.81% vs 55.41%; $p = 0.047$). Male gender was not significantly associated with myocardial injury (45.28% vs 56.05%; $p = 0.186$), as shown in Table 4.

Table 4. Univariate Analysis of Factors Associated with Myocardial Injury

Variable	Myocardial Injury Present (n=53)	Myocardial Injury Absent (n=157)	p-value
UTI present, n (%)	38 (71.70)	70 (44.59)	<0.001
Age >60 years, n (%)	29 (54.72)	56 (35.67)	0.015
HbA1c ≥8%, n (%)	41 (77.36)	82 (52.23)	0.002
Hypertension, n (%)	37 (69.81)	87 (55.41)	0.047
Male gender, n (%)	24 (45.28)	88 (56.05)	0.186

On multivariate logistic regression analysis, urinary tract infection remained an independent predictor of myocardial injury (adjusted OR = 2.84; 95% CI: 1.45–5.57; $p = 0.002$), as shown in Table 5. Poor glycemic control (HbA1c ≥8%) was also independently associated with myocardial injury (adjusted OR = 2.31; 95% CI: 1.14–4.68; $p = 0.019$). Age >60 years, hypertension, and male gender were not statistically significant predictors after adjustment.

Table 5 Multivariate Logistic Regression Analysis for Predictors of Myocardial Injury

Variable	Adjusted OR	95% CI	p-value
Urinary tract infection	2.84	1.45 – 5.57	0.002
Age >60 years	1.67	0.88 – 3.18	0.114
HbA1c ≥8%	2.31	1.14 – 4.68	0.019
Hypertension	1.42	0.74 – 2.71	0.289
Male gender	0.91	0.47 – 1.78	0.792

Discussion

Urinary tract infection occurred in 108/210 (51.43%) of our diabetic cohort and was associated with worse metabolic and hemodynamic profiles (HbA1c 8.8 ± 1.3 vs 8.0 ± 1.1; hypertension 66.67% vs 50.98%). These baseline differences mirror large clinical series that report higher UTI prevalence and greater metabolic dysregulation among people with diabetes—particularly women—where poor glycemic control is a consistent risk factor for infection. Our observed female predominance in the UTI group (55.56% vs 37.25%; p = 0.009) is concordant with prior hospital-based diabetes-UTI surveys that identify anatomical and glycosuric drivers for this sex disparity [14].

Cardiac biomarker abnormalities were markedly more frequent in patients with UTI: elevated hs-troponin in 39/108 (36.11%) vs 14/102 (13.73%), and elevated BNP in 34/108 (31.48%) vs 16/102 (15.69%) (both p ≤ 0.006). Such infection-associated troponin and natriuretic peptide rises have been described in sepsis and other systemic infections, where inflammatory cytokine release and myocardial strain produce subclinical myocardial injury. Our mean hs-troponin values (29.6 ± 11.4 vs 18.2 ± 8.9 ng/L) and BNP (186.5 ± 72.1 vs 122.4 ± 55.6 pg/mL) are within ranges reported in cohorts of non-ACS infectious illness, supporting the biological plausibility that UTIs—though often localized—can trigger measurable myocardial stress [15].

Overall laboratory-defined myocardial injury affected 53/210 (25.23%); prevalence was higher with UTI (38/108; 35.19%) than without (15/102; 14.71%; p < 0.001). This two-fold difference aligns with epidemiologic data linking acute infections to short-term increases in major cardiovascular events, including findings from a large self-controlled case series showing elevated MI/stroke incidence in the first week after microbiologically confirmed UTI (IRR ≈2.3–2.5). While population studies evaluate hard events, our biomarker-based approach demonstrates a likely mechanistic intermediate—myocardial injury—that may underlie those event signals [16].

Univariate associations in our cohort (UTI present in 71.70% of those with myocardial injury vs 44.59% without; p < 0.001) reinforce infection as a correlate of myocardial damage. Prior literature likewise documents high proportions of troponin elevation among patients hospitalized with noncardiac infections and sepsis; one review suggested infections are the commonest non-ACS cause of troponin rise in medical inpatients. Our findings extend these observations specifically to diabetic patients with UTI, a subgroup at heightened baseline cardiovascular vulnerability [17].

Multivariate modeling demonstrated UTI remained an independent predictor of myocardial injury (adjusted OR = 2.84; 95% CI, 1.45–5.57; p = 0.002) after adjustment for age, sex, hypertension and glycemic control; HbA1c ≥8% was also independently associated (adjusted OR = 2.31; 95% CI, 1.14–4.68; p = 0.019). These adjusted effect sizes are consistent with pathophysiologic and cohort data indicating that infection-related myocardial injury is not fully explained by traditional risk factors and that poor glycemic control amplifies susceptibility to both infection and cardiac injury [18].

Mechanistically, our results dovetail with experimental and translational work linking systemic inflammatory mediators, endothelial activation and microvascular

dysfunction to troponin release and natriuretic peptide secretion during infection. Urine-based troponin/peptide measures and studies in diabetic populations (e.g., urinary hs-TnI predicting cardiovascular events) provide additional supportive evidence that laboratory biomarkers capture subclinical myocardial injury relevant to long-term risk stratification in diabetes. Together, these data situate our findings within a growing literature that frames acute infection including UTI as a biologically plausible trigger of myocardial injury in vulnerable patients [19].

Strengths and limitations

The prospective design of this research, the inclusion of a well-defined diabetic population free of cardiovascular illness, and the use of objective, high-sensitivity laboratory biomarkers (hs-troponin and BNP) to identify subclinical myocardial damage are some of its strong points. After controlling for important confounders including age, glycemic management, and hypertension, the comparison of diabetic patients with and without UTI using multivariate logistic regression allowed for a strong evaluation of UTI as an independent predictor. However, it is vital to recognize significant limits. Generalizability may be restricted by the convenience sampling and single-center design. Although sufficient for regression analysis, the sample size limits subgroup analyses. Long-term cardiovascular outcomes and structural or functional cardiac alterations cannot be correlated with the lack of cardiac imaging and longitudinal follow-up. Furthermore, there was no measurement of inflammatory markers, which would have bolstered the mechanistic conclusions connecting infection to cardiac damage.

Conclusion

Urinary tract infections were significantly linked to subclinical myocardial damage in individuals with diabetes mellitus in this laboratory-based cardiovascular investigation. When compared to individuals without UTI, diabetic patients with UTI had more than twice as much laboratory-defined myocardial damage and far higher rates of raised hs-troponin and BNP. Along with poor glycemic management, UTI continued to be an independent predictor of myocardial damage even after controlling for known cardiovascular risk factors. These results emphasize the need of early infection detection and cardiovascular risk assessment in this high-risk group by highlighting urinary tract infections as a clinically significant and possibly preventable cause of cardiac damage in diabetes individuals.

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