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ORIGINAL PAPER

Impact of diabetes on dengue – a comparative study of clinical and inflammatory variables in patients with and without diabetes

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ABSTRACT

Introduction and aim. Dengue fever is a mosquito-borne viral disease and its severity may be influenced by comorbid conditions such as diabetes mellitus, which can alter the inflammatory and clinical response.
This study aimed to evaluate and compare clinical characteristics, laboratory findings, and inflammatory markers between patients with and without diabetes who were diagnosed with dengue infection.

Material and methods. The retrospective observational study included 100 patients with confirmed dengue infection, divided equally into 50 with diabetes and 50 without. It examined the distribution of age, HbA1C levels, clinical symptoms, bleeding events, liver enzymes, and inflammatory markers. Correlation analyzes were conducted to assess the relationship between HbA1C levels and inflammatory markers within each group. In addition, inflammatory markers were compared in different age categories (<50 years and ≥50 years) and by diabetic status.

Results. Laboratory findings, including liver enzymes and inflammatory markers, were markedly elevated in the diabetic cohort ($p<0.001$). The correlation analysis revealed a strong positive relationship between HbA1c and inflammatory markers in the diabetic group ($r>0.8$, $p<0.001$), while weaker correlations were observed in the non-diabetic group ($r=0.4–0.6$, $p<0.001$). Inflammatory markers were significantly higher in diabetic patients, particularly those 50 and older.

Conclusion. Diabetes may contribute to a more intense inflammatory response in dengue, highlighting it as an independent risk factor for severe clinical outcomes in dengue infection.

Keywords. C-reactive protein, dengue and diabetes, dengue fever, ferritin, interleukin 6, triglyceride

Introduction

Dengue fever is one of the most common arthropod-borne viral infections in the world; it is caused by four different serotypes of the dengue virus (DENV). DENV is a positive-stranded RNA virus of the Flaviviridae family that primarily infects humans through the bite of infected Aedes mosquitoes. When infected mosquitoes feed on human blood, they inject saliva containing infectious virus particles into the skin. According to current estimates, more than 390 million DENV infections occur yearly.[1,2] Among the known aedes-borne viruses, DENV is the most common, deadly and widespread virus.[3] Currently, there are no targeted or specific therapies available for diseases caused

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by Aedes-borne viruses. However, medical treatment is available for symptoms such as headache, seizure, and fever. Several techniques, such as those targeting human hosts, human-vector interactions, and vectors specifically, can be utilized to prevent the spread of Aedes-borne illnesses.[4] Vector control techniques are mostly used because they provide direct or biological reduction/elimination of vectors while causing no major impact on human hosts. The Wolbachia-based control method is one of the biological vector control methods that involves replacing wild mosquito populations with mosquitoes infected with Wolbachia. This bacterium inhibits viral replication in the mosquito's midgut, thereby reducing the mosquito's ability to transmit viruses.[5,6] Hyperglycemia on a molecular level increases the severity of DENV infection by potentially inducing the poly A binding protein (PABP) to facilitate viral translation. Hyperglycemia affects the structural and functional integrity of the endothelium, resulting in a persistent inflammatory condition caused by the activation of T-lymphocytes and the secretion of pro-inflammatory cytokines. Furthermore, hyperglycemia adversely impacts the immune system by reducing chemotaxis, leukocyte adhesion, and the phagocytosis process.[7] Microvascular and macro-vascular damage, which compromises circulation, is a histopathologic hallmark of patients with diabetes.[8]

Dengue viruses can cause a diverse array of clinical manifestations, ranging from asymptomatic infections to mild febrile illnesses, and extending to severe dengue, characterized by heightened capillary permeability and the potential for shock.[9] Increased levels of inflammatory markers are pathologically associated with chronic inflammatory conditions. Therefore, people with underlying inflammatory conditions, such as diabetes, may exhibit a heightened vulnerability to mortality associated with dengue. This association, which is related to immunological and metabolic dysregulation, is frequently cited as a significant predictor of the clinical progression of dengue infection in diabetes.[10]

Diabetic patients with dengue are at increased risk of developing severe complications, highlighting the role of comorbidities and poor glycemic control in the worsening of disease outcomes.[11] Compromised immune systems and fragile blood vessels, as well as increased vulnerability to bleeding, can contribute to a worsening of dengue symptoms in these patients.

Aim

Consequently, this study aims to improve the understanding of the relationship between inflammatory mediators and the clinical manifestations observed in both diabetic and nondiabetic patients suffering from dengue infection.

Material and methods

Study design and setting

This retrospective observational study was conducted at SUM Ultimate Medicare, Bhubaneswar. Due to the retrospective nature of the study, the requirement of informed consent was waived. Ethical approval was obtained from the Institutional Ethics Committee Clinical Research & Studies, SUM Ultimate Medicare, Bhubaneswar (Registration No. ECR/1604/Inst/OD/2021).

Participants

A total of 100 patients with confirmed dengue infection were included in the analysis, comprising 50 individuals with diabetes and 50 without diabetes. The sample size was determined based on a power analysis assuming a median value of medium to large effect size (Cohen's d=0.65), 80% power and a significance level of 0.05 required at least 39 patients in each of the two groups. The sample size was calculated using software power analysis and sample size version 3.1.9.3.

Inclusion and exclusion criteria

Participants aged between 15 and 70 years with a confirmed diagnosis of dengue infection were eligible for inclusion. In the diabetic group, a documented history of diabetes mellitus was required for a minimum duration of five years. Exclusion criteria included individuals with chronic kidney disease, autoimmune disorders, chronic obstructive airway disease, chronic steroid use, hemoglobinopathies, pregnancy, and other chronic diseases such as tuberculosis

Data collection

Data were collected for each patient to ensure a complete clinical and laboratory profile. Demographic information included age and sex. A detailed medical history was documented, with particular emphasis on the presence of diabetes mellitus, its duration, and any additional comorbidities. The clinical features evaluated at the time of presentation included fever, arthralgia, myalgia, abdominal pain, diarrhea, bleeding manifestations such as petechiae, melena, or epistaxis, and signs of serositis. Laboratory investigations included complete blood count (CBC) and liver function tests, specifically serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT). Inflammatory markers measured included interleukin-6 (IL-6), ferritin, triglycerides (TG), and C-reactive protein (CRP). Glycemic control was evaluated using the level of glycated hemoglobin (HbA1c).

Dengue diagnosis

Dengue infection was confirmed using both NS1 antigen testing and IgM antibody detection. NS1 antigen testing was performed using a rapid immunochromato-

graphic test, while IgM antibodies were detected using an enzyme-linked immunosorbent assay (ELISA). A positive result in either test, along with compatible clinical characteristics, was considered a diagnostic of dengue infection. Dengue virus-specific IgM antibodies were detected using the PanbioTM Dengue IgM Capture ELISA kit (Catalog No. 01PE20, Abbott Laboratories, Brisbane, Australia). This qualitative enzyme-linked immunosorbent assay (ELISA) is intended to support the diagnosis of dengue infection in patients presenting clinically consistent symptoms. According to the manufacturer's data, the assay has a serological sensitivity of 94.7% for primary dengue infections and 55.7% for secondary infections, with a serological specificity of 100% in negative samples. Although the assay does not report a numerical detection limit, it is optimized for identifying IgM antibodies during the acute phase of infection. The reported intraassay variability is <10%, and the interassay variability is <15%.

Diabetes classification

Patients were classified as having diabetes according to their medical history and current use of antidiabetic medications. Furthermore, an HbA1c level ≥6.5% was used to confirm the diagnosis in patients with a known history of diabetes.

Blood sample collection and analysis

Blood samples were taken from all patients within 24 hours of hospital admission. The samples were processed according to standard laboratory protocols. CBC was performed using an automated hematology analyzer. Liver function tests and inflammatory markers were measured using standard biochemical assays. HbA1c was measured using high performance liquid chromatography (HPLC). Blood samples were collected in EDTA anticoagulant tubes and analyzed within 24 hours. HbA1c was separated from other hemoglobin fractions on charge differences using a cation-exchange HPLC column. The percentage of HbA1c was determined by calculating the ratio of glycated hemoglobin to total hemoglobin. This method is certified by the National Glycohemoglobin Standardization Program (NGSP) and is standardized according to the reference of the Diabetes Control and Complications Trial (DCCT), ensuring high accuracy and reproducibility.

Outcome measures

Primary outcome measures included the levels of inflammatory markers (CRP, ferritin, triglycerides, and IL-6) and the severity of clinical manifestations in diabetic versus nondiabetic patients with dengue infection. Secondary outcomes involved evaluating the correlation between HbA1c levels and levels of inflammatory markers.

Statistical analysis

The normality of continuous variables was assessed using Z scores, with values falling within±3.29 considered normally distributed. Continuous variables are presented as mean±standard deviation, while categorical variables are reported as frequencies and percentages. Independent sample t tests were used to compare the mean between the two groups. Associations between categorical variables or comparisons of proportions were analyzed using Chi-square tests or Fisher's exact tests, as appropriate. Pearson's correlation coefficients were calculated to evaluate the linear relationships between continuous variables. Generalized linear regression models were used to investigate the associations between continuous outcome variables and mixed-type independent variables. All statistical analyzes were conducted using IBM SPSS Statistics version 26 (IBM Corp., Armonk, NY, USA). A p-value of <0.05 was considered statistically significant for all analyzes.

Results

A total of 100 patients with confirmed dengue infection were included in the study, comprising 50 patients with diabetes and 50 patients without diabetes. Diabetic patients had significantly higher mean age and HbA1c levels compared to non-diabetic patients. While the proportions of men and individuals with hypertension were higher in the diabetic group, these differences were not statistically significant. The clinical characteristics and manifestations of bleeding were also compared between the two groups, with no significant differences observed. However, laboratory parameters, including liver enzymes (SGPT, SGOT) and inflammatory markers (CRP, ferritin, triglycerides and IL-6) were significantly elevated in diabetic patients compared to nondiabetic patients ($p<0.001$). The baseline characteristics, clinical characteristics, bleeding manifestations, and laboratory findings are summarized in Table 1.

Correlation analysis

A correlation analysis was performed to assess the relationship between HbA1c levels and inflammatory markers within each group. Among diabetic patients, all inflammatory markers demonstrated a strong positive correlation with HbA1c levels ($r>0.8$, $p<0.001$). On the contrary, the non-diabetic group showed moderate correlations ($r=0.4–0.6$, $p<0.001$). All results were statistically significant, indicating a significant association between HbA1c and inflammatory markers. Detailed findings are presented in Table 2.

Subgroup analysis

The association of inflammatory variables was assessed with age groups (<50 years and ≥50 years) and diabetic status (diabetic and non-diabetic) was evaluated. There

was a significant difference in each of the inflammatory variables between the two age groups in diabetic patients, while in non-diabetic patients, except in TG values, the rest inflammatory variables were also significantly different between the two age groups. For TG, there were no significant differences between diabetic and nondiabetic patients for the age group of <50 years, while the differences were significant in the age group of ≥50 years. In other inflammatory variables, significant differences were found between diabetic and non-diabetic patients. In the above results, the values of the inflammatory variables were higher in older patients compared to younger patients. A similar result was also found, where the values of the inflammatory variables were higher in diabetic patients compared to nondiabetic patients. These results suggest that the association between diabetes, which is usually common in older age, elevated inflammatory markers in dengue infection, supporting the hypothesis that diabetes is an independent risk factor for a more severe inflammatory response in dengue infection. The results are summarized in Table 3.

Table 1. Baseline characteristics and clinical characteristics of study patients (n=100), data are presented as mean±standard deviation and compared using independent samples t-tests, categorical variables are expressed as number (%) and compared using Chi-square test

Demographic characteristic	Diabetic (n=50)	Non-diabetic (n=50)	p
Age (years), mean±SD	56.48±8.48	39.28±13.228	<0.001
Male n (%)	36 (72%)	33 (66%)	0.523
Female n (%)	14 (28%)	17 (34%)	
Hypertension, n (%)	9 (18%)	5 (10%)	0.249
HbA1c (%), mean±SD	8.38±1.16	4.44±0.619	<0.001
Clinical feature			
Fever, n (%)	39 (78%)	41 (82%)	0.617
Arthralgia, n (%)	37 (74%)	35 (70%)	0.658
Myalgia, n (%)	21 (42%)	18 (36%)	0.539
Abdominal pain, n (%)	41 (82%)	38 (76%)	0.461
Diarrhoea, n (%)	9 (18%)	7 (14%)	0.585
Bleeding manifestations			
Petechiae, n (%)	19 (38%)	17 (34%)	0.675
Melena, n (%)	6 (12%)	4 (8%)	0.505
Epistaxis, n (%)	5 (10%)	7 (14%)	0.538
Polyserositis, n (%)	34 (68%)	27 (54%)	0.151
Platelet transfusion requirement, n (%)	24 (48%)	21 (42%)	0.548
Laboratory parameters			
SGPT (units/L)	296.89±119.36	154.49±112.37	<0.0001
SGOT (units/L)	250.41±108.97	118.16±100.19	<0.0001
CRP (mg/dL)	21.045±5.28	8.96±3.98	<0.0001
Ferritin (ng/mL)	9141.311±2860.75	2264.29±2686.413	<0.0001
Triglycerides (mg/dL)	328.96±86.44	233.26±82.354	<0.0001
IL-6 (pg/mL)	12.27±2.83	5.324±2.49	<0.0001

Table 2. Correlation between HbA1c and inflammatory markers in diabetic and nondiabetic patients (n=100), Pearson correlation coefficients were used to assess the relationships

Inflammatory marker	Diabetic Group (n=50)		Diabetic Group (n=50)	
	r value	p	r value	p
HbA1c	--	--	--	--
CRP	0.856	<0.001	0.529	<0.001
IL-6	0.893	<0.001	0.568	<0.001
Ferritin	0.869	<0.001	0.291	<0.001
Triglycerides	0.877	<0.001	0.466	<0.001

Table 3. Association between age groups and diabetes status with inflammatory markers (n=100), data are expressed as mean±standard deviation, comparisons were performed using independent samples t test: between age groups within diabetic patients (#), within non-diabetic patients (##) and between diabetic and non-diabetic patients within the same age group ($)

Variable's	Age group	Diabetic (11/39)	p #	Non-diabetic (43/7)	p ##	p $
CRP (mg/dL)	<50 years (n=54)	17.8±3.43	0.019	8.36±3.45	0.06	<0.001
	≥50 years (n=46)	21.96±5.38		12.72±5.21		<0.001
IL-6 (pg/mL)	<50 years (n=54)	10.07±1.07	0.02	4.98±1.88	0.013	<0.002
	≥50 years (n=46)	12.9±2.87		7.46±4.48		<0.002
Ferritin (ng/mL)	<50 years (n=54)	7241.45±1193.01	0.011	1406.22±908.96	<0.001	<0.003
	≥50 years (n=46)	9677.17±2973.02		7535.29±3932.34		0.018
TG (mg/dL)	<50 years (n=54)	253.85±58.46	0.001	228.63±73.72	0.329	0.298
	≥50 years (n=46)	350.15±81.51		261.73±127.48		0.02

Table 4. Association of sex, age, and diabetes status with inflammatory markers (n=100), a generalized linear regression analysis was performed to assess the relationship between inflammatory markers and sex, age, and diabetes status

Dependent variables		Regression coefficient (β)		
		β	95% confidence interval	p
CRP	Sex	-0.082	(-3.27–0.57)	0.166
	Age	0.231	(0.05–0.21)	0.003
	Diabetes	-0.646	(-12.07– -7.59)	<0.001
IL-6	Sex	-0.017	(-1.28–0.95)	0.774
	Age	0.221	(0.02–0.12)	0.004
	Diabetes	-0.659	(-7.06– -4.45)	<0.001
Ferritin	Sex	-0.136	(-2348.47– -231.21)	0.017
	Age	0.329	(59.24–148.08)	<0.001
	Diabetes	-0.570	(-6251.64– -3781.63)	<0.001
TG	Sex	0.099	(-11.74–53.09)	0.209
	Age	0.521	(2.23–4.95)	<0.001
	Diabetes	-0.183	(-72.99–2.64)	0.068

Multivariate analysis

Given the association between inflammatory markers and HbA1c, further analysis was conducted to assess individual relationships of these inflammatory markers with sex, age and diabetes status using a generalized linear regression model. The results revealed that age and diabetes status were significant independent predictors of CRP, IL-6, and triglyceride levels. On the contrary, ferritin levels were significantly influenced by sex, age, and diabetes status. A summary of these findings is presented in Table 4.

Discussion

Research on the role of increased inflammatory markers in diabetic patients with dengue remains limited. Growing research indicates a connection between endothelial dysfunction and insulin resistance, suggesting a close relationship between the endothelium and insulin action.[12] In this study, patients with diabetes had more complications and a higher percentage of elevated inflammatory markers, suggesting that diabetes may be a risk factor for severe dengue infection. The clinical presentation of dengue can vary widely from mild symptoms to severe forms such as hemorrhagic fever, shock syndrome, and even death. This spectrum of severity is often influenced by underlying chronic diseases such as diabetes, which can alter immune responses.[13,14] Although several studies have demonstrated that diabetes mellitus can result in immunological and endothelial dysfunction, the exact mechanism by which diabetes causes severe dengue manifestation remains unclear.[15] Recent meta-analyses that examine diabetes mellitus as a risk factor for severe dengue reported that hyperglycemia in patients with diabetes may impair immune function by altering cytokine production. These changes can potentially improve dengue virus replication and contribute to greater disease severity and mortality.[16,17] Therefore, diabetic patients with dengue are at increased risk of developing severe complications, highlighting the role of comorbidities and poor glycemic control in worsening disease outcomes.[11]

Multiple studies have highlighted the increased severity of dengue in the context of poor glycemic control. For example, Lee et al. found that diabetic patients with well-managed blood glucose levels and no additional comorbidities were less likely to develop severe dengue compared to those with poor glycemic control, regardless of the presence of other health conditions. Similarly, Raj et al. concluded that hyperglycemia, independent of other clinical symptoms, is associated with worse outcomes in diabetic dengue patients. Marques-Vidal et al. also observed that elevated inflammatory markers are related to poor glycemic control and increased insulin resistance.[18] Understanding the interaction between inflammatory markers and demographic factors such as age, sex, and diabetes status can provide deeper insight into how these variables influence disease progression and severity

Elimam et al. established that CRP and IL-6 serve as important inflammatory markers, with elevated levels often associated with insulin resistance and poor glycemic control in individuals with diabetes.[19] These markers are produced by adipose tissue and the liver during systemic inflammation and demonstrate a relationship with insulin resistance. Hotamisligil noted in his research that increased concentrations of CRP and IL-6 can interfere with insulin signaling and glucose metabolism.[20] Furthermore, Masenga indicated that although triglycerides are primarily classified as a lipid marker, they are also associated with inflammatory processes and have been associated with increased oxidative stress and insulin resistance in diabetic patients.[21] Similarly, Zheng et al. reported a strong association between elevated triglyceride levels and both insulin resistance and poor glycemic control.[22]

Ferritin, a protein that stores iron, has been increasingly recognized as a marker of chronic inflammation. Increased levels of ferritin are frequently found in individuals with diabetes, and it has been proposed that ferritin may play a role in the promotion of oxidative stress and insulin resistance.[23] Ferritin concentrations can increase as part of the acute phase response, which occurs in reaction to systemic inflammation, and such elevations can be observed in both diabetic and non-diabetic individuals experiencing inflammatory states.

The relationship between CRP and HbA1c has been extensively studied, revealing a positive association, particularly among individuals diagnosed with diabetes. CRP, an acute-phase protein, tends to increase during episodes of systemic inflammation. According to Pradhan et al., elevated CRP levels in diabetic patients correlate with inadequate glycemic control, suggesting that inflammation plays a role in the disruption of glucose metabolism.[24,25] A recent study demonstrated that patients with diabetes and dengue had significantly higher CRP levels compared to patients without diabetes. Additionally, older patients with dengue, particularly those with diabetes, were more likely to show a marked rise in peak CRP levels during hospitalization and had an increased risk of progressing to severe dengue, findings consistent with our study.[26]

Research shows that both age and gender significantly influence inflammatory responses. For example, Marcos-Pérez et al. found that older adults tend to exhibit elevated levels of CRP, IL-6, and ferritin, which may contribute to an increased risk of type 2 diabetes with advancing age.[27] Gender-related differences in inflammation have also been observed, reporting that women often display higher levels of IL-6 and CRP than men, likely due to hormonal factors. Furthermore, diabetes status is

a critical factor, as individuals with diabetes frequently show elevated inflammatory markers compared to non-diabetic individuals, underscoring the role of inflammation in the pathophysiology of the disease. In line with these findings, our study identified a significant correlation between inflammatory markers and glycemic levels. This observation is further supported by evidence from a separate investigation, which revealed a strong association between serum inflammatory cytokine levels and both disease severity and clinical outcomes in dengue patients.[28] These insights highlight the importance of closely monitoring glycemic control in diabetic individuals to reduce the risk of severe dengue outcomes.

Latt et al. found that people with diabetes had a higher probability of experiencing severe manifestations of dengue, indicating that diabetes is an important risk factor for poor clinical outcomes.[29] Similarly, a study conducted by Figueiredo MAA and colleagues in Brazil found a strong association between dengue severity and diabetes mellitus, further supporting our findings.[30] Diabetes is known to nearly double the risk of a wide range of vascular complications, independent of other conventional risk factors.[31] The clinical presentation of dengue can vary widely from mild symptoms to severe forms such as hemorrhagic fever, shock syndrome, and even death. This spectrum of severity is often influenced by underlying chronic conditions like diabetes, which can impair immune responses. Recent meta-analyses have identified diabetes as a significant contributor to an increased risk of developing severe dengue and mortality associated with dengue.

This study compared the levels of the inflammatory mediators CRP, TG, ferritin, and IL-6 between dengue patients with and without diabetes. The observed association between these inflammatory markers and HbA1c levels is consistent with the findings reported by Singh et al.[32] These results confirm that individuals with diabetes who contract dengue tend to exhibit higher levels of inflammatory variables and experience more severe disease outcomes compared to non-diabetic patients. These findings highlight the importance of routine HbA1c testing in all diabetic patients diagnosed with dengue. Effective management of blood glucose levels is crucial to improving clinical outcomes. In addition, more extensive public health efforts, including comprehensive national dengue control and elimination campaigns, are essential to reduce the burden of the disease.

This study presents a novel comparative analysis of inflammatory and clinical profiles in patients with and without diabetes with dengue, offering a unique perspective on the immunometabolic interactions that may exacerbate the severity of the disease. Unlike previous studies that often focus on dengue or diabetes in isolation, this research bridges the gap by investigating how chronic hyperglycemia and long-standing diabetes impact the inflammatory environment during dengue infection. By integrating biochemical data (CRP, IL-6, ferritin, triglycerides) with glycemic control and age-related differences, the study uncovers a compelling pattern of amplified inflammatory responses in diabetics, particularly those aged ≥50 years. Furthermore, the robust correlation between HbA1c and pro-inflammatory markers provides new evidence supporting the role of poor glycemic control as a modifiable risk factor for adverse outcomes in dengue. This nuanced stratification of patients not only contributes to the limited body of literature on dengue-diabetes interactions, but also underscores the importance of metabolic comorbidities in shaping infectious disease trajectories. To our knowledge, this is one of the few studies in the country that quantitatively evaluate and contrast inflammatory markers in dengue patients stratified by diabetes status and age, making a significant contribution to tropical medicine and chronic disease research.

This study has several limitations that should be taken into account. Firstly, its retrospective design limits the ability to establish baseline equivalence between the diabetic and non-diabetic groups, introducing the possibility of selection bias. A significant proportion of diabetic participants were older than 50 years, with a mean age of 56 years, and severe dengue cases were predominantly observed in this older age group. This introduces age as a possible confounding factor in the association between diabetes and dengue severity. Additionally, the relatively small sample size may reduce statistical power and limit the generalizability of the findings to larger or more diverse populations. Another important limitation is the lack of serotype-specific data, which is critical, as different dengue virus serotypes can have varying impacts on disease progression and outcomes. In addition, the study did not distinguish between primary and secondary dengue infections, which could further increase the immune response. The scope of the inflammatory markers evaluated was also limited due to the unavailability and high cost of advanced immunological assays. As a result, key immune mediators such as interferons, interleukins, and other cytokines could not be evaluated.

Future research should address these limitations by including larger and more heterogeneous study populations, incorporating serotype-specific analysis, and expanding the range of inflammatory and immunological markers. Such studies would be instrumental in deepening our understanding of the immunopathogenesis of dengue, particularly in patients with diabetes, and in clarifying the mechanistic role of inflammatory mediators in shaping disease severity and outcomes.

Conclusion

Diabetes is an independent risk factor for progressing to severe dengue infection. Inflammatory marker levels

were significantly higher in dengue patients with diabetes compared to those without the disease, suggesting their potential role in the progression of severe disease and their utility as a possible predictor of disease severity. Consequently, dengue infections in people with diabetes were linked to increased inflammation and a greater probability of experiencing severe manifestations of dengue.

Declarations

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Author contributions
Conceptualization, A.D.J., S.K.D., S.M., A.J.S., A.D., K.R. and A.V.; Methodology, A.D.J. and S.K.D.; Software, A.D.J., S.K.D., S.M., A.J.S., A.D., K.R. and A.V.; Validation, A.D.J., S.K.D., S.M., A.J.S., A.D., K.R. and A.V.; Formal Analysis, A.D.J., S.K.D., S.M., A.J.S., A.D., K.R. and A.V.; Investigation, A.D.J., S.K.D., S.M., A.J.S. and A.D.; Resources, A.D.J., S.K.D., S.M., A.J.S., A.D., K.R. and A.V.; Data Curation, A.D.J. and S.K.D..; Writing – Original Draft Preparation, A.D.J.; Writing – Review & Editing, A.D.J., S.K.D., S.M., A.J.S., A.D., K.R. and A.V.; Visualization, A.D.J., S.K.D., S.M., A.J.S., A.D., K.R. and A.V.; Supervision, A.D.J. and S.K.D.; Project Administration, A.D.J. and S.K.D..

Conflicts of interest
The author(s) declared that they have no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Data availability
All data generated or analyzed during this study are included in this published article.

Ethics approval
This study was approved by the Institutional Ethics Committee Clinical Research & Studies, SUM Ultimate Medicare, Bhubaneswar (Registration No. ECR/1604/Inst/OD/2021).

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