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Clinical and laboratory findings in type 2 diabetes mellitus with COVID-19 infection: a single centre study

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ABSTRACT

Introduction: The coexistence of type 2 diabetes mellitus (T2DM) with coronavirus disease 2019 (COVID-19) poses a major global healthcare concern. Even though both have become significant worldwide health issues, little is still known about how they interact in this region of the world. The purpose of this study is to examine the laboratory markers and clinical features of COVID-19-infected individuals with T2DM.

Methods: The medical records of hospitalized T2DM patients with proven COVID-19 infection at the Prince Mohammed Bin Abdulaziz Hospital (PMAH) in Riyadh, Saudi Arabia, between March 15, 2020, and July 15, 2020, were examined in this single-center retrospective analysis. Laboratory, clinical, and demographic data were collected and examined. The relationships between different demographic traits and T2DM with COVID-19 infection were evaluated using the Fisher Exact test, binary logistic regression, and odds ratio (OR). The quantitative factors between individuals with type 2 diabetes and those without the disease were compared using the Mann-Whitney U test. A P value of less than 0.05 was deemed significant.

Results: About 142 (59.4%) of the 239 patients had type 2 diabetes, and they were substantially older (56 ± 13.1; $P < 0.001$) than those without the disease. Among T2DM patients, cough ($P = 0.02$), tachypnea ($P = 0.001$), and dyspnea ($P = 0.001$) were significant symptoms. Among T2DM patients, hypertension ($P = 0.001$) and hyperlipidemia ($P = 0.001$) were common comorbidities. Patients with type 2 diabetes had substantially higher levels of creatinine ($P = 0.001$), blood urea nitrogen ($P = 0.004$), LDH ($P = 0.033$), CRP ($P = 0.001$), and fibrinogen ($P = 0.001$). Tachypnea (aOR = 3.46 (1.65-7.24) $P = 0.001$) and dyspnea (aOR = 3.25 (1.49-7.05) $P = 0.001$) demonstrated statistical significance on binary logistic regression analysis. In both bivariate analysis and logistic regression, hypertension (aOR = 2.07 (1.03-4.14); $P = 0.040$) and dyslipidemia (aOR = 12.27 (2.63-57.20); $P = 0.001$) maintained statistical significance.

Conclusion: Significantly higher laboratory and inflammatory values are seen in T2DM with COVID-19, underscoring the significance of targeted treatment approaches and resource allocation.

Keywords: clinical, COVID-19, laboratory, markers, T2DM.
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INTRODUCTION

Coronavirus disease 2019 (COVID-19), caused by the severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), has emerged as one of the most significant global public health crises of the 21st century.[1] Since its declaration as a pandemic by the World Health Organization (WHO) in March 2020, COVID-19 has resulted in substantial morbidity and mortality worldwide.[2] Although the clinical spectrum of COVID-19 ranges from asymptomatic infection to severe respiratory failure and multi-organ dysfunction, disease severity is strongly influenced by underlying comorbid conditions.[3]

Among these, type 2 diabetes mellitus (T2DM) has consistently been identified as one of the most important risk factors associated with poor outcomes in COVID-19 patients.[4] The T2DM is characterized by chronic hyperglycemia, insulin resistance, and a persistent low-grade inflammatory state.[5] These pathophysiological alterations contribute to immune system dysfunction, including impaired neutrophil chemotaxis, reduced T-cell response, and abnormal cytokine production. As a result, patients with T2DM are more susceptible to infections and often experience more severe disease progression.[6]

The reciprocal link between COVID-19 and T2DM has been explained by a number of processes. Hyperglycemia may weaken antiviral immune responses and promote viral multiplication.[7] Additionally, SARS-CoV-2 binds to angiotensin-converting

enzyme 2 (ACE2) receptors, which are expressed in multiple tissues, including the lungs, pancreas, kidneys, and vascular endothelium.[8] COVID-19 severity may be further increased by endothelial dysfunction, a pro-thrombotic condition frequently observed in diabetes, and dysregulation of the renin-angiotensin-aldosterone system (RAAS). The exaggerated inflammatory response, often described as a "cytokine storm," is also more pronounced in diabetic individuals, potentially leading to acute respiratory distress syndrome (ARDS), multi-organ failure, and increased mortality.[9]

The Kingdom of Saudi Arabia (KSA) is a crucial location for researching the relationship between diabetes and COVID-19 since it has one of the highest prevalence rates of type 2 diabetes worldwide. Rapid urbanization, sedentary lifestyle patterns, and dietary transitions have contributed to the growing burden of metabolic disorders in the region. Managing patients with numerous comorbidities, such as diabetes and cardiovascular disease, was a major issue for KSA's healthcare systems during the early stages of the COVID-19 epidemic. However, despite the high prevalence of T2DM, limited region-specific data exist describing the clinical characteristics, comorbidity patterns, and laboratory profiles of hospitalized COVID-19 patients with diabetes in Saudi Arabia.[10]

Understanding these characteristics is essential for several reasons. First, early identification of high-risk features can facilitate timely intervention and risk stratification. Second, laboratory markers reflecting inflammation, organ dysfunction, and coagulation abnormalities may help guide clinical decision-making and resource allocation. Third, regional data are crucial, as disease patterns, genetic background, healthcare access, and comorbidity profiles may differ from those reported in Western or Asian populations.

Thus, the purpose of this study was to examine and contrast the clinical presentation, laboratory inflammatory markers, comorbidities, and demographic characteristics of hospitalized COVID-19 patients with and without type 2 diabetes in a tertiary care hospital in Riyadh, Saudi Arabia. By identifying distinctive characteristics associated with T2DM in COVID-19, this study seeks to contribute to improved clinical management strategies and better outcomes for this high-risk population.

METHODS

Design and setting

The medical records of T2DM patients with proven COVID-19 infection who were hospitalized at Prince Mohammed Bin Abdulaziz Hospital (PMAH) in Riyadh, Saudi Arabia, between March 15, 2020, and July 15, 2020, were examined in this retrospective cross-sectional investigation.

Participants

This study comprised the records of 239 hospitalized patients who had a confirmed COVID-19 infection. Children, pregnant women, and those with impaired mental capacity were not included. Because pregnancy is linked to distinct physiological, immunological, and metabolic changes that may independently affect COVID-19 severity and laboratory markers, thereby complicating the relationship between T2DM and COVID-19 results, pregnant patients were excluded. Children were not included since their COVID-19 clinical course, immunological response, and illness presentation differed from those of adults. Because accurate symptom reporting (such as chest discomfort or shortness of breath) and clinical history documentation may be inconsistent in this population, patients with impaired mental ability were excluded. Furthermore, these individuals frequently have complicated co-occurring neurological disorders that might independently affect inflammatory indicators and clinical outcomes, thus creating confounding. SARS-CoV-2 infection was diagnosed using the criteria established by the Saudi Center for Disease Prevention and Control. Anti-diabetic medicine or recorded medical records served as the basis for the study's definition of a diabetic patient. Additionally, a fasting glucose level of at least 7.0 mmol/L upon admission and a HbA1c value of at least 6.5 were regarded as diagnostic of diabetes mellitus. Non-diabetics were defined as those without a history of diabetes, anti-diabetic drug usage, HbA1c < 6.5, and fasting glucose < 7.0 mmol/L.[11]

Data collection

Demographic information, such as gender, age, and nationality, was collected. Shortness of breath (SoB), tachypnea, fever, cough, chest discomfort, blood pressure, heart rate (HR), respiratory rate (RR), oxygen saturation, length of stay, and admission to the intensive care unit (ICU) were among the clinical indicators noted. Along with test results on inflammatory markers, including fibrinogen, D-dimer, and C-reactive protein (CRP), information was also gathered on comorbidities like hypertension, dyslipidemia, congestive heart failure, and ischemic heart disease.

Data analysis

SPSS version 21.0 (IBM Corp., Chicago, IL, USA) was used to analyze the data. While continuous data were first evaluated for normality using the Shapiro-Wilk test and visual examination of histograms, categorical variables were summarized as frequencies and percentages. Non-parametric statistical tests were used since the majority of continuous laboratory data showed a non-normal distribution. Because the Mann-Whitney U test is suitable for skewed data and does not presume a normal distribution, it was utilized to evaluate continuous variables between T2DM and non-diabetic groups. When anticipated cell counts were sufficient, the Chi-square test was used to evaluate correlations between T2DM status and clinical features for categorical variables. However, Fisher's Exact test was employed when anticipated frequencies in contingency tables were less than five in more than 20% of cells, since it offers a more precise estimate for sparse data and small sample sizes. Bivariate logistic regression was used to compute crude odds ratios (COR) with 95% confidence intervals (CI). To calculate adjusted odds ratios (AOR) while accounting for possible confounders, variables that were statistically significant in bivariate analysis were included in a multivariable binary logistic regression model. Statistical significance was defined as a two-tailed P-value of less than 0.05.

RESULTS

Baseline demographic and clinical characteristics

About 142 (59.4%) of the 239 individuals examined had type 2 diabetes (Table 1). Patients with type 2 diabetes were considerably older than those without the disease (56 ± 13.1 vs. 47 ± 15.2 years, P < 0.001). In terms of clinical presentation, T2DM patients had substantially higher rates of cough (P = 0.020), tachypnea (P = 0.001), and shortness of breath (P = 0.001). There were no statistically significant differences between the groups in terms of fever or chest discomfort. In terms of comorbidities, T2DM patients had considerably higher rates of dyslipidemia (P = 0.001) and hypertension (P = 0.001). Furthermore, T2DM patients were more likely than non-diabetic patients to be admitted to the intensive care unit (P = 0.002).

Laboratory and inflammatory markers

Creatinine (P = 0.001), blood urea nitrogen (P = 0.004), lactate dehydrogenase (P = 0.033), C-reactive protein (P = 0.001), and fibrinogen (P = 0.001) were all considerably higher in T2DM patients. The levels of total bilirubin, ALP, ALT, AST, and D-dimer did not differ significantly between the two groups. These results point to increased organ failure and systemic inflammation in COVID-19-affected diabetic individuals (Table 2).

Logistic regression analysis

The crude and adjusted risk ratios for variables linked to type 2 diabetes in hospitalized COVID-19 patients are shown in Table 3. T2DM was shown to be substantially correlated with cough, tachypnea, shortness of breath, hypertension, and dyslipidemia in bivariate analysis. Nevertheless, tachypnea (aOR = 3.46; 95% CI: 1.65–7.24; P = 0.001) and dyspnea (aOR = 3.25; 95% CI: 1.49–7.05; P = 0.003) continued to be independently related with multivariable logistic regression correction. After correction, the comorbidities dyslipidemia (aOR = 12.27; 95% CI: 2.63–57.20; P = 0.001) and hypertension (aOR = 2.07; 95% CI: 1.03–4.14; P = 0.040) continued to be statistically significant.

Table 1. Baseline characteristics of the research subjects

Characteristics	T2DM		P-value
	No	Yes	
Gender			
Male	85	116	0.218
Female	12	26	
Age	47 ± 15.2	56 ± 13.1	<0.001[b]
Nationality			
Non-Saudi	75	104	0.475
Saudi	22	38	
Cough			
Yes	70	120	0.020*
No	27	22	
Fever			
Yes	72	119	0.069
No	25	23	
Tachypnea			
Yes	32	107	0.001*
No	65	35	
Chest pain			
Yes	8	8	0.427
No	89	134	
Shortness of breath			
Yes	54	121	0.001*
No	43	21	
Hypertension			
Yes	22	67	0.001*
No	75	75	
Chronic kidney disease			
Yes	1	5	0.405[a]
No	96	137	
Dyslipidaemia			
Yes	2	45	0.001[a]
No	95	97	
Bronchial asthma			
Yes	12	11	0.234
No	85	131	
Ischemic heart disease			
Yes	3	10	0.250[a]
No	94	132	
Congestive cardiac failure			
Yes	1	6	0.246[a]
No	96	136	
Intensive care unit admission			
Yes	24	63	0.002*
No	73	79	

Note: [a]Fisher's Exact Test; [b]Mann-Whitney U test; *significant (P<0.05)

DISCUSSION

The global COVID-19 pandemic has challenged healthcare systems worldwide, with varying impacts on different populations. Riyadh, Kingdom of Saudi Arabia (KSA), has faced substantial challenges in managing the pandemic due to its unique population demographics and the coexistence of other healthcare burdens, such as T2DM. In line with earlier research that found advanced age to be a risk factor for more severe

Table 2. Laboratory and inflammatory marker results between T2DM and non-diabetic patients

Laboratory and inflammatory marker	Category	Number	Mean rank	U	P value
Creatinine (µmol/l)	Non-diabetic	91	92.08	7818.500	0.001*
	T2DM	132	125.73		
BUN (mmol/l)	Non-diabetic	86	93.80	6940.500	0.004*
	T2DM	131	118.98		
Total Bilirubin (mg/dl)	Non-diabetic	93	121.20	5747.500	0.242
	T2DM	136	110.76		
ALP (U/l)	Non-diabetic	30	29.42	602.500	0.213
	T2DM	34	35.22		
ALT (U/l)	Non-diabetic	91	120.15	5628.500	0.249
	T2DM	136	109.89		
LDH(U/l)	Non-diabetic	75	77.72	4371.000	0.033*
	T2DM	98	94.10		
AST(U/l)	Non-diabetic	84	96.33	5642.000	0.180
	T2DM	121	107.63		
CRP (mg/l)	Non-diabetic	59	56.04	3242.500	0.001*
	T2DM	81	81.03		
D-Dimer (mg/l)	Non-diabetic	74	78.45	4147.500	0.082
	T2DM	97	91.76		
Fibrinogen (mg/l)	Non-diabetic	55	57.55	3214.500	0.001*
	T2DM	88	81.03		

Note: ALP: Alkaline phosphatase; ALT: Alanine transaminase; AST: Aspartate aminotransferase; BUN: Blood urea nitrogen; CRP: C-reactive protein; LDH: Lactate dehydrogenase; *significant Mann-Whitney U test (P<0.05)

outcomes in COVID-19, especially in patients with comorbidities like diabetes, T2DM patients with COVID-19 were considerably older than their non-diabetic counterparts. The severity of COVID-19 in individuals with type 2 diabetes may be influenced by this factor.

Certain clinical signs, such cough, tachypnea, and dyspnea, were much more common in T2DM patients in our research. In this particular patient population, these symptoms might be early markers of the severity of the illness. Our findings align with existing literature, which suggests that individuals with diabetes may present with atypical or more severe symptoms during COVID-19 infection, potentially leading to poor disease prognosis and management.[12–14]

Hypertension and dyslipidemia were commonly observed comorbidities in T2DM patients with COVID-19 in our study. Dyslipidemia is also common among individuals with diabetes, contributing to the elevated cardiovascular risk within this population.[15] These findings are consistent with previous research, emphasizing the importance of recognizing and managing comorbid conditions in T2DM patients with COVID-19. The interplay between diabetes, hypertension, and dyslipidemia may exacerbate the risk of severe outcomes in this group. The findings suggest that the above-mentioned clinical symptoms and comorbidities should be closely monitored in T2DM patients with COVID-19. Early interventions and customized treatments for these patients may help mitigate the risk of severe outcomes.

Laboratory data revealed that several markers, including creatinine, BUN, LDH, CRP, and fibrinogen, were significantly higher in T2DM patients with COVID-19. These markers indicate increased levels of inflammation and organ dysfunction, reinforcing the notion that COVID-19 exacerbates the underlying inflammatory state often seen in diabetes.[16,17] Creatinine and BUN levels are indicators of renal function and can be elevated in patients with severe COVID-19. It is of particular concern in individuals with diabetes, as they are at an increased risk of kidney complications.[18] Elevated LDH levels are associated with tissue damage, particularly lung involvement, and have been identified as a prognostic marker in COVID-19.[19] Our findings suggest that T2DM patients may be more susceptible to lung injury during COVID-19. Similarly, CRP and fibrinogen are well-established markers of inflammation. Their elevation in T2DM patients with COVID-19 highlights the systemic inflammatory response in this group, which may contribute to the severity of the disease.[14] Our findings underscore the significance of monitoring these laboratory markers in T2DM patients with COVID-19. Early detection of elevated levels may prompt more aggressive management strategies, such as anti-inflammatory treatments and closer observation for organ dysfunction.

The higher severity of COVID-19 in individuals with T2DM may be explained

Table 3. Binary logistic analysis of baseline characteristics between T2DM and non-diabetic patients with COVID-19 infection

Characteristics	T2DM		COR (95% CI)	AOR (95% CI)	P value
	No	Yes			
Gender					
Males	85	116	0.62 (0.30-1.31)	0.63 (0.22-1.76)	0.382
Females	12	26			
Nationality					
Non-Saudi	75	104	0.80 (0.43-1.46)	1.07 (0.47-2.41)	0.861
Saudi	22	38			
Cough					
Yes	70	120	2.10 (1.11-3.97)	1.06 (0.46-2.39)	0.506
No	27	22			
Fever					
Yes	72	119	1.79 (0.94-3.39)	1.31 (0.52-2.45)	0.756
No	25	23			
Tachypnea					
Yes	32	107	6.20 (3.51-10.97)	3.46 (1.65-7.24)	0.001*
No	65	35			
Chest pain					
Yes	8	8	0.66 (0.24-1.83)	0.38 (0.94-1.56)	0.181
No	89	134			
Shortness of breath					
Yes	54	121	4.58 (2.48-8.46)	3.25 (1.49-7.05)	0.003*
No	43	21			
Hypertension					
Yes	22	67	3.04 (1.70-5.42)	2.07 (1.03-4.14)	0.040*
No	75	75			
Chronic kidney disease					
Yes	1	5	3.50 (0.40-30.46)	1.03 (0.82-13.05)	0.978
No	96	137			
Dyslipidaemia					
Yes	2	45	22.03 (5.19-93.41)	12.27 (2.63-57.20)	0.001*
No	95	97			
Bronchial asthma					
Yes	12	11	0.59 (0.25-1.40)	0.62 (0.20-1.89)	0.406
No	85	131			
Ischemic heart disease					
Yes	3	10	2.37 (0.63-8.85)	1.16 (0.12-10.51)	0.893
No	94	132			
Congestive heart failure					
Yes	1	6	4.23 (0.50-35.75)	1.63 (0.12-26.07)	0.730
No	96	136			
Intensive care unit admission					
Yes	24	63	2.42 (1.37-4.27)	0.76 (0.35-1.67)	0.50
No	73	79			

by a number of pathophysiological processes, in addition to the clinical relationships shown in our study. Chronic low-grade systemic inflammation, insulin resistance, endothelial dysfunction, and compromised innate and adaptive immunological responses are the hallmarks of type 2 diabetes. A pro-inflammatory and pro-thrombotic milieu is produced by these underlying abnormalities, which may intensify the host's reaction to SARS-CoV-2 infection.[5]

Chronic hyperglycemia reduces the body's capacity to efficiently eliminate viral infections by impairing neutrophil chemotaxis, phagocytosis, and complement activation.[20] Tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and other inflammatory mediators are among the dysregulated cytokines that are linked to type 2 diabetes. This pre-existing inflammatory environment may put diabetic individuals at risk for an excessive immune response, known as a "cytokine storm," when infected with SARS-CoV-2, which can lead to ARDS and multi-organ failure.

Insulin resistance and hyperglycemia may also promote viral replication by increasing glucose availability, which can enhance viral entry and replication within host cells.[21] Furthermore, SARS-CoV-2 utilizes ACE2 receptors for cellular entry. ACE2 expression may be altered in individuals with diabetes, particularly those on RAAS inhibitors, potentially influencing disease severity. Endothelial dysfunction, commonly seen in T2DM, further contributes to vascular inflammation, microvascular injury, and thrombotic complications, which are hallmark features of severe COVID-19.[22]

Another important consideration is the pro-coagulant state associated with T2DM. Elevated fibrinogen and inflammatory markers observed in our study support the presence of heightened coagulation and systemic inflammation in diabetic patients.[23] This hypercoagulable state may increase the risk of thromboembolic events, worsening clinical outcomes. Collectively, these mechanisms suggest that T2DM not only increases susceptibility to infection but also enhances the severity of COVID-19 through immune dysregulation, chronic inflammation, endothelial injury, and metabolic dysfunction. These pathophysiological insights reinforce the importance of early identification, close monitoring, and aggressive management strategies in this high-risk population.[24]

The results of this study have a number of significant ramifications for clinical practice, especially in areas where T2DM is more prevalent. First, when diabetic individuals arrive with COVID-19, early respiratory evaluation should be given first priority. Even in the absence of overt hypoxia at presentation, symptoms like tachypnea and shortness of breath, which were independently linked to T2DM in our trial, should cause early clinical escalation and closer monitoring. Second, routine laboratory surveillance of inflammatory and organ dysfunction markers, including CRP, fibrinogen, LDH, creatinine, and BUN, may assist in identifying high-risk diabetic patients at an early stage. Serial monitoring of these parameters may support timely therapeutic decisions, including the initiation of anti-inflammatory therapy, anticoagulation when indicated, and intensified supportive care. Third, strict glycemic control during hospitalization should be emphasized. Hyperglycemia has been associated with worse outcomes in COVID-19, even in non-diabetic individuals. Therefore, structured inpatient glycemic management protocols may help reduce inflammatory burden and improve immune response in T2DM patients. Finally, integrated multidisciplinary care involving endocrinologists, infectious disease specialists, and critical care teams may improve risk stratification and individualized management strategies for diabetic patients with COVID-19. In healthcare systems with limited resources, identifying T2DM patients as a high-risk group may also guide prioritization of monitoring and resource allocation.

There are a number of limitations to this study that should be noted. First, because the retrospective approach relies on previously recorded medical data, it is intrinsically limited in its capacity to show a causal link and susceptible to information bias. Although hospital records were systematically reviewed, incomplete documentation or variability in clinical recording may have influenced the accuracy of some variables. Second, the results might not be entirely applicable to other parts of Saudi Arabia or to other healthcare environments because this was a single-center research carried out at a tertiary care hospital in Riyadh. Referral patterns and admission procedures unique to a particular hospital may be reflected in the patient population. Third, as the research only included hospitalized individuals, selection bias cannot be ruled out. It could restrict extrapolation to individuals with moderate or asymptomatic illness and overrepresent more severe COVID-19 infections. Furthermore, even after multivariable correction, residual confounding can still exist since several factors—like the length of diabetes, glycemic control prior to admission, medication usage, and socioeconomic determinants—were not thoroughly evaluated.

CONCLUSION

This study demonstrates that hospitalized COVID-19 patients with T2DM exhibit distinct clinical and laboratory profiles associated with greater disease severity. Older age, tachypnea, and shortness of breath were prominent clinical features, while hypertension and dyslipidaemia were significantly more common in this group. Elevated markers of inflammation and organ dysfunction, including creatinine, BUN, LDH, CRP, and fibrinogen, further indicate an amplified inflammatory and metabolic response among diabetic patients. These findings underscore the need for early risk stratification of T2DM patients at hospital admission. Focused respiratory assessment and structured monitoring of inflammatory and renal markers may enable timely clinical intervention. Integrating optimized glycemic management with vigilant surveillance for organ dysfunction could help mitigate complications and improve outcomes in this high-risk population, particularly in regions with a high prevalence of T2DM.

DISCLOSURES

Ethical Clearance

The King Fahad Medical City Institutional Review Board (IRB) approved the study protocol (IRB No. 20-290C).

Conflict of Interest

The author declares no conflict of interest.

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This study did not receive any outside support.

Author Contribution

ASA includes conceptualization, research design, data collection, data interpretation, article drafting, and critical revision. The final document was authorized by the author, who also committed to taking full responsibility for the work.

Generative Artificial Intelligence Statement

To enhance language, grammar, and readability, the author employed artificial intelligence techniques. Following the use of these tools, the authors assumed full responsibility for the published article's content and reviewed and modified it as necessary.

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