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Alterations of basic blood parameters in Iraqi COVID-19 patients with hyperglycaemia and acute kidney injury

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ABSTRACT

Emerging evidence indicates that coronavirus disease 2019 (COVID-19) can exert direct effects on the kidneys, leading to acute kidney injury (AKI). This is particularly concerning for individuals with diabetic nephropathy, who may already exhibit compromised renal function. The pathophysiology of COVID-19-related AKI may involve mechanisms such as a direct viral invasion of renal cells, systemic inflammation, and microvascular damage. Our study has included a total of 250 patients with confirmed COVID-19 that were treated between February and April 2022 in designated COVID-19 centres throughout the Babil Province (Iraq). Clinical and laboratory data were retrospectively obtained from the patients' medical records. Across all COVID-19 severity categories, our study has observed notable elevations in blood markers, including urea, creatinine, D-dimer, and blood glucose levels. Moreover, male patients exhibited significantly higher levels of urea, creatinine, D-dimer, C-reactive protein, and blood glucose compared to female patients (p<0.05). Our study's findings suggest that diabetes mellitus and AKI can both be associated with heightened systemic inflammation and greater disease severity, and could be key predictors of adverse COVID-19 outcomes.

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1. Introduction

Coronavirus disease (COVID-19), caused by the novel RNA virus SARS-CoV-2, was first identified in late 2019 in China. Clinical presentations range from asymptomatic infection to severe respiratory

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Table 1. Selected blood variables were measured in 250 COVID-19 patients. One-way ANOVA was used in order to assess significant differences across patient groups based on disease severity (mild to moderate vs. severe) and gender.

	Mild to moderate COVID-19 patients		**Severe COVID-19 patients**		
Parameters	**Male** (N=70)	**Female** (N=55)	**Male** (N=60)	**Female** (N=65)	***p*-value**
Glucose (mmol/L)	10.50 ± 0.64	9.00 ± 0.40	21.00 ± 0.57	19.75 ± 0.85	<0.001
Serum creatinine (Umol/L)	79.50 ± 2.10	77.75 ± 0.85	168.75 ± 3.72	155.00 ± 6.45	<0.001
Blood urea (mmol/L)	8.00 ± 0.40	6.50 ± 0.28	18.00 ± 0.40	17.00 ± 0.44	<0.0001
D-dimer (ng/mL)	562.50 ± 55.43	512.50 ± 31.45	7,550.00 ± 212.00	4,750.00 ± 220.02	<0.05
C-reactive protein (mg\L)	8.50 ± 0.64	9.80 ± 0.50	14.00 ± 0.57	12.75 ± 0.85	<0.001

illness, including acute respiratory distress syndrome (ARDS). Recurrent respiratory infections are a common clinical complaint and can progress to pneumonia[1]. While the majority of individuals experience mild to moderate symptoms, a subset may rapidly deteriorate, developing life-threatening complications such as ARDS, severe respiratory failure, metabolic acidosis, coagulopathies, and septic shock. Reported rates of intensive care unit (ICU) admission range from 5% to 16%, depending on the demographic and regional characteristics of the studied population.

The widespread transmission of SARS-CoV-2 overwhelmed public health infrastructure in numerous countries, with ICUs becoming particularly strained[2]. Although respiratory symptoms predominate, SARS-CoV-2 has systemic effects. Among the hospitalised patients, 60%–70% present with hypercoagulability, thrombocytopenia, venous thrombosis, or disseminated intravascular coagulation (DIC). Elevated D-dimer levels are now used not only in order to diagnose deep vein thrombosis, but also to anticipate recurrence and signal early DIC. Bode *et al.*[3] have reported that 36% of the COVID-19 patients exhibit tachycardia, with an average D-dimer level of 0.9 mg/L.

Diabetes mellitus and hyperglycaemia are critical prognostic factors in hospitalised patients with acute medical conditions, including COVID-19. Recent data confirm that diabetes is one of the most prevalent comorbidities among individuals infected with SARS-CoV-2. Initial studies have identified diabetes in 7.4% to 19% of COVID-19 cases, with notably higher prevalence (16.2% to 26.9%) among patients with severe disease[2].

Up to 25% of patients in retrospective cohorts have shown evidence of acute kidney injury (AKI); a known contributor to mortality in individuals with ARDS. The hallmark features of AKI include endothelial dysfunction, microangiopathy, and tubular injury, frequently associated with elevated inflammatory markers. The present study aimed at identifying urinary parameters predictive of AKI development in hospitalised COVID-19 patients.

2. Methodology

Clinical and demographic data were obtained from medical records at hospitals across the Babil Province, Iraq. Data collection included patient symptoms, medical history, comorbid conditions, chest computed tomography (CT) findings, and laboratory results from February 20 to April 25, 2022. Samples were extracted from confirmed COVID-19 cases treated in designated COVID-19 centres throughout the Babil Province.

Blood samples were collected on the same day from hospitalised individuals aged 20–80 years (mean age: 36 years). The cohort included 250 participants. Informed consent was obtained from all individuals, and the study was conducted under ethical approval (number: A-0028) granted by the Institutional Ethics Committee for Human Research at the Babylon Health Directorate (Babil, Iraq) in January 2021. The study complies with the principles of the Declaration of Helsinki, and all participants were

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Iraqi nationals.

Serum levels of D-dimer, C-reactive protein (CRP), urea, creatinine, and blood glucose were measured. Statistical analyses were conducted using the SPSS software. Results were deemed as statistically significant at $p<0.05$.

3. Results and Discussion

Our study has found that male patients exhibited significantly higher levels of urea, creatinine, D-dimer, CRP, and blood glucose compared to female patients ($p<0.05$).

Due to their low cost, automated processing, and non-invasive sampling, urinary biochemical markers are frequently employed to detect urinary tract infections, monitor kidney disease, and assess treatment efficacy. However, evidence remains limited regarding their prognostic value in critically ill COVID-19 patients. A marked difference in the frequency of haematuria was observed between healthy controls and individuals with COVID-19. Prior research has demonstrated that urine sediment analysis may aid in predicting in-hospital mortality among admitted COVID-19 patients[5], particularly in those presenting with elevated serum creatinine and urea levels.

The majority of COVID-19 patients in this cohort exhibited increased blood urea and serum creatinine concentrations, with statistically significant findings detailed in Table 1. These results are consistent with observations reported by Bonetti *et al.*[5], who have noted similar elevations in renal biomarkers among patients with a SARS-CoV-2 infection. The subgroup with the highest frequency of urea and creatinine elevation comprised male patients. Multiple mechanisms may contribute to renal impairment in patients with SARS-CoV-2 pneumonia, leading to elevated levels of these markers[6].

Hyperglycaemia was also prevalent, affecting between 42.4% and 51.1% of patients whose blood glucose exceeded 7.77 mmol/L[6]. Discrepancies in disease severity across studies – some focusing on moderate cases, others on severe – may account for the variability observed in the reported rates. Individuals with chronic kidney disease appear more vulnerable to SARS-CoV-2–associated pneumonia, potentially due to inflammation-induced immunosuppression affecting both innate and adaptive immunity. This immunocompromised state is linked to increased risk of upper respiratory infections and pneumonia-related mortality[7].

Research from Indonesia, Japan, Greece, and Wuhan (China), has consistently associated elevated D-dimer levels with severe SARS-CoV-2 infection. Thrombocytopenia in affected patients may exacerbate this pattern, with elevated D-dimer contributing to delayed coagulation and a heightened risk of DIC; a potentially fatal complication[8].

Further analysis revealed that creatinine and CRP were robust predictors of mortality in COVID-19 patients with diabetes. Chronic hyperglycaemia and systemic inflammation impair immune function, and diabetes itself may accelerate progression to severe or fatal COVID-19 outcomes. The present findings highlight an increased susceptibility in diabetic individuals, particularly those with poor glycaemic control. These insights underscore the need to prioritize high-risk patients for access to safe and effective COVID-19 vaccination[9].

4. Conclusion

Our study's findings suggest that diabetes mellitus and AKI can both be associated with heightened systemic inflammation and greater disease severity, and could be key predictors of adverse COVID-19 outcomes.

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Conflicts of interest

None exist.

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