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Original Research Paper

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COMPARATIVE STUDY OF BIOCHEMICAL ALTERATION IN COVID-19 AND NON COVID 19 PATIENTS: ASSOCIATION WITH KIDNEY FUNCTION TESTS ABNORMALITIES.

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ABSTRACT Abnormal Kidney functions are frequently reported in patients with COVID-19. Renal tests serum parameters abnormalities are common in patients with coronavirus disease 2019 (COVID-19). The aim of the study was the evaluation of kidney function test parameters in patients with COVID-19 and non Covid -19 patients and analysis of the relationships between serum level of urea, uric acid, creatinine, sodium and potassium, C-Reactive protein and ferritin with COVID-19 and non Covid -19 patients. Significantly increased levels of urea, uric acid, C-Reactive protein and ferritin with COVID-19 compare to non Covid -19 patients.

KEYWORDS : Renal function test, COVID-19, Urea, Uric acid , Creatinine, CRP, Ferritin.

INTRODUCTION

Acute kidney injury (AKI) is one of the most frequent complications in patients with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection during the coronavirus disease 2019 (COVID-19) pandemic. Although COVID-19 is characterized by atypical pneumonia and subsequent severe respiratory failure, approximately 10% of inpatients with COVID-19 reportedly suffer from AKI [1], which is significantly associated with poor outcomes [2]. Successive recent publications have reported an even higher global incidence of AKI. Patients with COVID-19 who are being treated in intensive care units (ICUs) are the most susceptible to severe AKI, which requires renal replacement therapy. However, the pathophysiology of COVID-19-associated AKI remains to be determined.1

The pathophysiologic mechanisms of kidney damage and AKI in patients with COVID-19 remain unclear but are known to be multifactorial. Current knowledge implies direct SARS-CoV-2-dependent effects on kidney cells (tubular epithelial cells and podocytes) and indirect mechanisms through the systemic effect of viral infection secondary to the critical pulmonary illness and its management.2 Acute kidney injury (AKI) is strongly associated with poor outcomes in hospitalized patients with coronavirus disease 2019 (COVID-19).3

Various AKI rates and their associated risk factors, lack of AKI recovery in the majority of patients hospitalized with COVID-19, and limited data regarding AKI in patients with COVID-19. 4

Current data show that many hospitalized COVID-19 patients suffer from kidney damage, in the form acute kidney injury (AKI). AKI is especially prevalent among severe and critically ill COVID-19 patients and is a predictor of mortality. The pathophysiology of AKI in COVID-19 is unclear. Early reports of histopathologic examination from autopsied kidney tissue show SARS-CoV-2 viral particles in renal tubular cells and podocytes, suggesting direct viral infection, as well as findings of acute tubular necrosis5

Various biochemical parameters that are determine in serum. The aim of the study was the evaluation of Renal function test parameters in patients with COVID-19 and non Covid -19 patients and analysis of the relationships between serum level of Urea , Uric acid , Creatinine, Sodium, Potassium , CRP and ferritin with COVID-19 and non Covid -19 patients.

MATERIALS AND METHODS

This study was conducted at the department of Biochemistry Clinical Biochemistry Section S.G.M.H., S.S. Medical College Rewa (M.P.) Source of data- The study group compared of 100 patients from 18-60 years of age, covid 19 and non covid 19 patients.

Specimen collection and preparation/ collection of samples-Venous blood was collected from all subjects after 12 hours over night fasting. Fasting venous blood were drawn from all 3 ml of venous blood was collected and stored in a sterile vial. Blood was allowed to clot of room temperature. Clot was rimmed, centrifugation serum was separated by low speed centrifugation and the serum was stored in a sterile vial, hemolyzed and lipemic samples were rejected.

Biochemical analysis-Serum level of Urea, Uric acid, Creatinine, Sodium, Potassium, CRP and ferritin were estimated by fully autoanalyzer BA400. Present work was approved by institutional research and ethical committee. Mean and standard deviation were determined for each variable in all groups. All results were expressed as mean +/- SD. Student "t" test was used to assess statistical significance of the results.

OBSERVATION

Table.1-The level of Serum level of Urea, Uric acid, Creatinin, Sodium, Potassium, CRP, ferritin in the Covid 19 and non Covid 19 patients.

Variables	Covid 19 (100 patients)	Non Covid 19 (100 patients)
Urea (mg/dl)	55.36±26.14	32.11±20.87
Uric acid (mg/dl)	9.10±1.04	4.24±1.61
Creatinine (mg/dl)	1.9±0.94	0.95± 0.61
Sodium (mEq/L)	137.24±6.95	134.70±5.23
Potassium (mEq/L)	3.99±0.63	4.17±0.64
C-Reactive Protein (mg/l)	50.9±6.51	30.12±8.51
Ferritin (ug/l)	595.40± 76.95	340.32±68.74

RESULTS

The present study was done with an aim to screen the subjects18-60 years of age in urban region for renal diseases. The serum parameters level obtained was then correlated with another parameter with determined. descriptive statics of diagnostic parameters presented in Table I. There was a statistically significant Significantly increased levels of urea, uric acid, creatinine, C-Reactive protein and ferritin with COVID-19 compare to non COVID -19 patients.

DISCUSSION

Kidney injury is common in coronavirus disease 2019, and it is associated with poor clinical outcomes.6 Acute kidney injury (AKI) has been reported in up to 25% of critically-ill patients with SARS-CoV-2 infection, especially in those with underlying comorbidities. The pathophysiology of COVID-19 associated AKI could be related to unspecific mechanisms but also to COVID-specific mechanisms such as direct cellular injury resulting from viral entry through the receptor (ACE2) which is highly expressed in the kidney, an imbalanced renin-angotensin-aldosteron system, pro-inflammatory cytokines elicited by the viral infection and thrombotic events.7 Despite initial reports, renal involvement, including acute kidney

injury, has emerged as a serious complication of COVID-19 disease, particularly in critically ill patients.8 Acute kidney injury (AKI) is relatively common in critically ill coronavirus disease 2019 (COVID-19) patients and it increases mortality.9

COVID-19 combined with kidney impairment are associated with a higher risk of mortality than those without comorbidities. The pathological changes of the kidney are mainly due to local SARS-CoV-2 replication or indirectly by pro-inflammatory cytokine response.10 Emerging evidence supports that in patients with SARS-CoV-2 infections the prevalence of kidney injury is high and usually leads to a poor prognosis. Optimal prevention and management of kidney injury will benefit patients with COVID-19. 10

Assess scientific evidence on SARS-CoV-2 Acute Kidney Injury in patients with COVID-19. the studies addressed the mechanism of intracellular infection of SARS-CoV-2, its cytopathic effects on kidney cells and incidence of acute kidney injury in patients infected with SARS-CoV-2. Acute kidney injury is associated with increased mortality and morbidity in these patients.11

Acute kidney injury (AKI) was a relevant finding in patients with COVID-19. There were also significant changes in laboratory tests that indicated kidney injury, such as increased serum creatinine and blood urea nitrogen (BUN), proteinuria, and hematuria. The presence of laboratory abnormalities and AKI were significant in severely ill patients. There was a considerable prevalence of AKI among groups of patients who died of COVID-19. Histopathological analysis of the kidney tissue of patients infected with SARS-CoV-2 suggested that the virus may directly affect the kidneys.12 Although COVID-19 affects mainly the lungs, it can also impact the kidneys. Increased serum creatinine and BUN, proteinuria, and AKI were frequent findings in patients with severe COVID-19 and were related to an increased mortality rate.12

Renal abnormalities occurred in the majority of patients with COVID-19 pneumonia. Although proteinuria, hematuria, and AKI often resolved in such patients within 3 weeks after the onset of symptoms, renal complications in COVID-19 were associated with higher mortality.13 Blood urea nitrogen, a nitrogenous end product of protein metabolism, is commonly used as biomarker for kidney function and hypovolemia. Systemic inflammation is common in severe COVID-19. 14

Studies have shown that COVID-19-related kidney damage may be characterized by increased levels of proteinuria, hematuria, and serum creatinine.3,4 It has been suggested that particularly proximal tubules are affected. Proximal tubule damage increases uric acid excretion, leading to a decrease in serum uric acid levels.5 Given the importance of serum uric acid levels in terms of showing the extent of kidney damage, we believe that they may be associated with the clinical presentation, course and consequences of COVID-19 infection.15

Acute kidney disease and chronic kidney disease are considered conditions that can increase the mortality and severity of COVID-19. However, few studies have investigated the impact of creatinine levels on COVID-19 progression in patients without a history of chronic kidney disease. One study was to assessed the impact of creatinine levels at hospital admission on COVID-19 progression and mortality.16 Acute kidney disease and chronic kidney disease were considered conditions that can increase the mortality and severity of COVID-19. However, few studies have investigated the impact of creatinine levels on COVID-19 progression in patients without a history of chronic kidney disease. 16

COVID-19 is mainly considered a respiratory illness, but since SARS-CoV-2 uses the angiotensin converting enzyme 2 receptor (ACE2) to enter human cells, the kidney is also a target of the viral infection. Acute kidney injury (AKI) is the most alarming condition in COVID-19 patients. Recent studies have confirmed the direct entry of SARS-CoV-2 into the renal cells, namely podocytes and proximal tubular cells, but this is not the only pathomechanism of kidney damage. Hypovolemia, cytokine storm and collapsing glomerulopathy also play an important role. An increasing number of papers suggest a strong association between AKI development and higher mortality in COVID-19 patients. 17

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