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Estimation of Serum Urea, Creatinine, And Electrolytes in Corona Virus Disease 2019 Covid-19 Positive Patients

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KEYWORDS

Severe acute respiratory syndrome coronavirus-2(SARS-C0V-2), Coronavirus disease 2019, Renal dysfunction, Coronavirus disease 2019 (COVID-19).

ABSTRACT:

Introduction: China saw the outbreak of Coronavirus Disease 2019 (COVID-19), an emerging infectious disease with an unknown etiology, during the winter of 2019. This unidentified virus was later identified and given the name severe acute respiratory syndrome. second coronavirus (SARS-CoV-2). More data point to the association between renal dysfunction and coronavirus disease 2019 (COVID-19). Uncertainty surrounds the relationship between renal dysfunction brought on by the SARS-CoV-2 virus and prognosis.

Objectives: The objectives center around recognizing the impact of COVID-19 on renal function, advocating for continuous monitoring, and proposing specific blood markers as indicators for predicting patient outcomes.

Methods: 160 hospitalised COVID-19-positive patients were enrolled in the current study, of which 80 were assigned to Group I — COVID-19 patients who were critically sick (severe cases), and 80 to Group II — COVID-19 patients who were not critically ill (mild and moderate cases). Using the Architect system (201837-110) to measure serum urea and creatinine levels, data were reported as mg/dL

Results: In the current investigation, group I samples demonstrated a highly significant increase in serum urea, creatinine, and Sodium compared to group II samples When comparing critically sick COVID-19 patients (severe cases) to non-critically ill COVID-19 patients (mild and moderate cases), there was no discernible difference in the mean difference of serum potassium.

Conclusions: Impaired kidney function, hyponatremia, and developing kidney damage should notify clinicians during COVID-19 patient care. In clinical practice, serum electrolytes, as well as serum urea and creatinine, can be utilized to predict Covid-19 patient survival.

1. Introduction

Due to a new coronavirus discovered in Wuhan, China, pneumonia incidence will rise by the end of 2019. An epidemic was brought on by its rapid spread, speedy progression, and lack of a specific treatment strategy (minerals and renal markers in COVID-19 patients who have tested positive for the disease. For determining the patient's state and prognosis, determining the best course of treatment, and even assessing the effectiveness of the cure, blood biochemical changes are essential.

(1).On January 30, 2020, the World Health Organisation (WHO) declared the 2019-nCoV epidemic a public health emergency of international concern (PHEIC). The WHO categorized the sickness as coronavirus disease 2019 (COVID-19) on February 11, 2020. The coronavirus family of non-diverging positive-sense RNA viruses,

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which harms both humans and other animals (3), comprises coronaviruses. The severe acute respiratory syndrome 2 coronavirus (SARS-C0V-2) belongs to the seventh class of human diseases. The most common symptoms of COVID-199 (4) are fever, a dry cough, fatigue, muscular aches, and other symptoms like shortness of breath, headache, diarrhea, and indigestion.

Clinical features and patient fatality rates, particularly for very ill patients, have been of concern in addition to incidence and transmission qualities. The clinical symptoms and blood chemistry features of COVID-19 patients have been extensively documented in research (5). However, there were significant inconsistencies in the results due to the diverse study designs and low sample sizes. Declining renal function as a result of the virus's potential appears to be a novel trait of those who test positive for COVID-19 (5).

The most prevalent signs and symptoms of COVID-19 patients, according to prior studies, are fever, diarrhoea, dry cough, lymphocyte depletion, and radiographic signs of pneumonia (6). Recent studies have demonstrated that SARS-CoV-2, in addition to causing severe acute respiratory syndrome, also produced significant organ damage, including heart destruction, lymphocyte depletion, and even liver failure (7).

The clinical characteristics of renal impairment caused by SARS-CoV-2 are rarely described, nevertheless. Furthermore, it is yet unknown what clinical significance renal impairment brought on by SARS-CoV-2 has and what the prognosis is. To draw more firm conclusions on the biochemical characteristics of individuals with COVID-19, we plan to perform a study on the levels of minerals and renal markers in COVID-19 patients who have tested positive for the disease. For determining the patient's state and prognosis, determining the best course of treatment, and even assessing the effectiveness of the cure, blood biochemical changes are essential.

2. Objectives

1. Correlating Renal Function with Disease Severity: The primary objective is to establish a correlation between the degree of COVID-19 disease severity and renal function. This involves analyzing data to support the notion that the extent of COVID-19 illness is proportional to the impact on kidney function.

2. Highlighting the Importance of Kidney Function Monitoring: The statement emphasizes the importance of not overlooking kidney function and damage markers in COVID-19 patients. The objective is to underscore the need for continuous and systematic monitoring of renal parameters to assess the evolving impact of the disease on the kidneys.

3. Advocating for Serial Monitoring: The study aims to explore the feasibility and benefits of serial monitoring of kidney function in COVID-19 patients. Serial monitoring involves repeated assessments over time to track changes in renal markers and identify trends that may inform clinical decisions.

4. Early Recognition of Impaired Kidney Function: The study aims to raise awareness among clinicians about the significance of impaired kidney function as an indicator of COVID-19 severity. The objective is to ensure that clinicians are vigilant in recognizing signs of renal impairment and damage during patient care.

5. Utilizing Serum Electrolytes, Urea, and Creatinine for Predicting Survival: The objective is to propose the use of specific blood markers, including serum electrolytes, urea, and creatinine, as predictive indicators for COVID-19 patient survival. This involves suggesting these parameters as potential tools in clinical practice to assess the likelihood of patient outcomes.

6. Informing Clinical Practice: Overall, the study seeks to provide practical insights for clinicians in managing COVID-19 patients. This includes integrating renal function assessment into routine care and utilizing readily available blood markers for predicting patient survival.

In summary, the objectives center around recognizing the impact of COVID-19 on renal function, advocating for continuous monitoring, and proposing specific blood markers as indicators for predicting patient outcomes. These efforts aim to improve the management and care of COVID-19 patients, particularly by emphasizing the importance of renal function assessment in clinical practice

3. Methods

The proposed research was conducted at the Bharati Vidyapeeth (Deemed to be University) Medical College & Hospital in Sangli, Department of Biochemistry. The Institute of Ethical Committee has given the study its

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approval. The study subjects provided informed consent before participating. 160 hospitalized COVID-19-positive patients were enrolled in the current trial, of which 80 were assigned to Group I — COVID-19 patients who were critically sick (severe cases), and 80 to Group II — COVID-19 patients who were not critically ill (mild and moderate cases). Patient histories and digital medical records were used to collect information on epidemiological, clinical, laboratory, and radiological factors. Information on the patient's medical history, exposure history, and underlying morbidities such as diabetes, hypertension, cardiovascular disease, asthma, and chronic renal disease were all documented.

The diagnosis of COVID-19 on the nasopharyngeal and oropharyngeal swab samples was confirmed in our molecular diagnostic laboratory using real-time reverse-transcription polymerase chain reaction (RT-PCR). We performed the RT-PCR by the directions provided by the manufacturer. Using the most recent revision of our national COVID-19 standards, suspected COVID-19 cases were identified (8).

On the day of admission, all patients underwent standard blood workups, coagulation profiles, and serum biochemistry assays. Standard-grade chemicals and reagents were used for the examination of biochemical experiments. Using the Architect system (201837-110) to measure serum urea and creatinine levels, data were reported as mg/dL (9, 10). The ST-200 Plus Electrolyte Analyzer used the Ion ion-selective electrode Method to measure the serum potassium and sodium levels and values were expressed as mEq/L(11)

Every newly enrolled COVID-19-positive patient met the inclusion criteria. The study did not include patients who demonstrated or reported negative medication effects. According to guidelines established by the Indian government, doctors diagnose and categorize their patients (12). Cases classified as critically ill were those who tested positive for COVID-19 and had specific CT and lab results. patients with severe instances of COVID-19. They will be divided as,

- Group I - Critically ill COVID-19 patients (Severe cases)
- Group II – Noncritical illCOVID-19 patients(Mild and moderate cases)

Table 1: The mean values of biochemical parameters in Group I - critically ill COVID-19 patients (severe cases) and Group II-non-critically ill COVID-19 patients (Mild and moderate cases)

Biochemical Parameters	Group	Mean ± Std. Deviation	Std. Error Mean	unpaired t	p-value	Significance
Serum Urea mg/dl	Group I (Severe cases) N= 80	51.15 ± 17.99***	2.01	8.233	0.000	Highly significance
	Group II (Mild and moderate cases) N= 80	32.56 ± 9.18***	1.03			
Serum Creatinine mg/dl	Group I (Severe cases) N= 80	1.49 ± 1.14***	0.13	3.332	0.001	Highly significance
	Group II (Mild and moderate cases) N= 80	1.05 ± 0.31***	0.03			
Serum Sodium (Na^+) mEq/L	Group I (Severe cases) N= 80	139.67 ± 3.51***	0.40	14.263	0.000	Highly significance
	Group II (Mild and moderate cases) N=80	131.13 ± 4.03***	0.45			
Serum	Group I (Severe cases) N=80	4.51 ± 0.79*	0.09	1.588	0.114	Not significant

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Potassium (K^+) mEq/L	Group II (Mild and moderate cases) N=80	4.32 ± 0.69*	0.08			

*P >0.05 – Not Significant

***P < 0.001 – Highly significant

Statistical Analysis:

With spreadsheet software (Excel, Microsoft), all statistical comparisons were carried out. Version 22 of SPSS software was used to conduct the statistical analysis. The unpaired "t" test was used to determine if critically sick COVID-19 patients (severe cases) and non-critically ill COVID-19 patients (mild and moderate cases) had different serum urea, creatinine, sodium, and potassium levels. The "p" value of 0.05 was taken into consideration to be statistically significant for all data, which were calculated as Mean ± SD.

4.Results

Analysis was done on the relationship between COVID-19 severity renal failure and electrolyte imbalance. We examined the serum renal function indices of serum urea, creatinine, creatinine, and electrolytes. As shown in Table 1, the concentrations of serum urea, creatinine, sodium, and potassium in critically ill COVID-

19 patients (severe cases) and non-critically ill COVID-19 patients (mild and moderate cases) are statistically compared in Table 1.

In the current investigation, group I samples demonstrated a highly significant increase in serum urea, creatinine, and Sodium compared to group II samples (p = 0.000). When comparing critically sick COVID-19 patients (severe cases) to non-critically ill COVID-19 patients (mild and moderate cases), there was no discernible difference in the mean difference of serum potassium (P=0.114).

5.Discussion

A new problem for the entire planet emerged at the turn of the decade. This time, the adversary was evasive—a tiny protein- and nucleic acid-containing particle. The third significant epidemic of the coronavirus began in December 2019 (12). It spread to other nations in just a few months after beginning in the Chinese province of Hubei's Wuhan. The World Health Organisation (WHO) first identified this condition as coronavirus disease 2019 (COVID-19) and later classified it as a pandemic (13). Increasing data suggest that SARS-CoV-2 injection caused many organ abnormalities, including liver dysfunction, cardiac injury, kidney impairment, and even respiratory failure (14,15).

Recent research suggests that Acute Kidney Injury (AKI) is associated with greater morbidity and death in COVID-19 patients. It is also regarded as a sign of disease severity and a risk factor for survival. The incidence of renal function impairment in COVID-19 patients was studied prospectively in this research study. Furthermore, we looked at renal routine biomarkers to see if there was any clear evidence of COVID-19-related acute kidney injury. We evaluated renal biomarkers in our study to acquire definite evidence of COVID-19-associated acute kidney injury. We discovered that COVID-19 patients in Group I (severe cases) had significantly higher levels of serum urea and creatinine than patients in Group II (mild and moderate).

The involvement of the kidneys during COVID-19 is now recognized as a regular occurrence. Reduced density in the kidneys, which is associated with inflammation and edema, has been characterized as typical CT scan findings of COVID-19 involvement (16). Our findings show that renal dysfunction is associated with the severity of COVID-19 patients on admission. More and more reports have revealed that SARS-CoV-2 plays a pathogenetic role in COVID-19 patients by attaching to the receptor of angiotensin-converting enzyme (ACE)2 Several studies have shown that ACE2 can be found in the renal tubular epithelium (18). As a result, SARS-CoV-2 may cause direct kidney tissue injury via binding to the ACE2 receptor (17-19).

According to the National Centre for Biotechnology Information's (NCBI) gene database, the kidney is the fourth most ACE2-expressing organ in the human body, following the small intestine, duodenum, and gall bladder (20,21). The brush border of proximal tubular cells is the

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principal source of ACE2 extent in the kidney. However, multiple cases have already shown the co-occurrence of AKI with COVID-19 indicating that SARS-CoV-2 may have a kidney tropism (22). In this context, a recent study by Diao et al. (23) is quite remarkable. The kidney tissue was discovered to be one of SARS-CoV-2 particular targets. The authors discovered a distinct SARS-CoV-2 nucleocapsid protein in kidney specimens and identified viral antigens accumulated in kidney tubules using postmortem tissue analysis. They concluded that SARS-CoV-2 infects human kidney tubules directly, which might be causing acute tubular injury. According to Diao et al, in addition to direct cytotoxicity, it also initiates macrophage and complement-mediated tubular pathogenesis as a result of accumulating viral antigens.

A potential link between electrolyte disturbance and coronavirus disease 2019 (COVID-19) in patients visiting the emergency department (ED) has not been thoroughly investigated. Hyponatremia and hypokalemia were more common in patients infected with COVID-19 than in controls. In this study, we observed that hyponatremia is independently linked with COVID-19 in people visiting the emergency department. Hyponatremia was also linked to COVID-19 and the most severe form of the disease, requiring ICU admission (24). Hyponatremia in COVID-19 adults may be associated with increased antidiuretic hormone (ADH) release in response to volume depletion caused by gastrointestinal fluid losses. Carvalho et.al. demonstrated that symptoms such as diarrhea and vomiting are common in COVID-19 patients and can cause increased ADH release due to extracellular dehydration. A syndrome of antidiuresis (SIAD) can develop in response to COVID-19 consequences such as pneumonia or acute respiratory distress syndrome (24). Thus, hyponatremia could serve as a surrogate indicator for the emergency physician in suspected COVID-19 patients to promptly evaluate at-risk patients. In contrast to the previous investigation, we did not detect a significant relationship between COVID-19 severity and serum potassium level in our study (p=0.114).

These data support the notion that COVID-19 hurts renal function that is proportionate to the degree of the disease. In this sense, kidney function and damage markers in COVID-19 patients should not be overlooked, and serial monitoring should be explored. Impaired kidney function and developing kidney damage should notify clinicians during COVID-19 patient care. In clinical practice, serum electrolytes, as well as serum urea and creatinine, can be utilized to predict Covid-19 patient survival.

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