

Original Research Paper

General Medicine

A STUDY TO ASSESS THE INCIDENCE OF ACUTE KIDNEY INJURY IN COVID-19 PATIENTS AND ITS CORELATION WITH IL-6 AND OTHER INFLAMMATORY MARKERS SUGGESTING CYTOKINE STORM

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Patients with SARS-CoV-2 are at risk of multiorgan complications, including but not limited to acute respiratory distress syndrome (ARDS), sepsis, infarcts, gastrointestinal (GI) manifestations and acute kidney injury (AKI). One of the major systems affected by this virus is the renal system. Acute Kidney Injury (AKI) is a frequent complication of severe SARS-CoV-2 infection but data are scarce in ICUs. Recent studies point to the possibility of direct viral infection of kidney cells via angiotensin-converting enzyme-2 (ACE2). Elevated production of proinflammatory cytokines/chemokines or even hypercytokinemia, also known as cytokine storm, may develop in SARS.CoV.2 infections and contributes to acute lung injury and development of ARDS. There is lack of sufficient data determining the role of cytokine storm in acute kidney injury in covid-19 patients. Identification of the role and effect of cytokine storm and other inflammatory markers in AKI will help in better clinical management hence reducing Covid-19 associated mortality and morbidity.

KEYWORDS: Covid-19, Cytokine Storm, Acute Kidney Injury, Inflammatory markers

INTRODUCTION

There is a related relationship between Covid-19 & Acute kidney Injury (AKI). Although the lungs are considered as the major targeted organ by SARS.CoV.2 with diffuse alveolar damage, SARS.CoV.2 may also cause significant damage to other organs such as the heart, liver and kidneys.[1] COVID-19associated acute kidney injury (AKI) has been noted in several reports, and the incidence varies depending on criteria for hospitalization in different locales. Acute Kidney Injury (AKI) is a frequent complication of severe SARS-CoV-2 infection but data are scarce in ICUs.[2] AKI has been previously reported with an average incidence of 11% (8-17%) overall, with highest ranges in the critically ill (23%; 14-35%).[3] Recent studies point to the possibility of direct viral infection to kidney cells via angiotensin-converting enzyme-2 (ACE2).[4] The kidneys have one of the highest content of ACE2, which SARS.CoV.2 spike protein directly recognizes and binds to. [5] It is not known whether COVID-19 causes progressive kidney damage, in the proximal tubules in SARS-CoV-2, by direct viral infection or proinflammatory cytokine-mediated mechanisms. In view of same, the present study was planned to find out incidence of Acute kidney injury in Covid-19 Patients and also to assess co-relationship of acute kidney injury in Covid-19 positive patient with IL-6 levels and other inflammatory markers I.e. CRP and Ferratin. We thus sought to describe the prevalence, risk factors and prognostic impact of AKI during COVID-19 in the ICU.

AIMS AND OBJECTIVES:

- To find out incidence of Acute kidney injury in Covid-19
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- To assess co-relationship of acute kidney injury in Covid-19 positive patient with IL-6 levels.

 To assess co-relationship of Acute kidney injury in Covid-19 positive patient with other inflammatory markers I.e. CRP and Ferritin

MATERIAL AND METHODS:

The present study was a Cross sectional Study planned and carried out in Department of Medicine, MGM college & MY hospital, Indore for a period of 12 months. This study included 313 consecutive patients of 18-80 years age group with documented and laboratory confirmed COVID-19 disease, at the time of enrollment who were admitted or availing treatment for COVID-19 in wards of Medicine, ICU and COVID special wards of M.Y. Hospital.

METHODOLOGY:

313 patients who were newly diagnosed covid positive and were fulfilling the inclusion criteria were included in the study. After taking pre-Informed written consent from the patients, a pre-structured proforma was used to collect the desired baseline data. Detailed clinical examination and biochemical tests were done on all patients as per the protocol. All the patients diagnosed with COVID-19 were thoroughly investigated for AKI manifestations. All the relevant personal history and medical history was obtained. The obtained data was coded and entered into Microsoft excel 2010 (Microsoft corp.), and was analyzed using excel 2010 and SPSS 20.0 for Windows (SPSS inc). Prevalence of an outcome variable along with 95% confidence limits was calculated and descriptive analysis of the population was carried out.

OBSERVATIONS AND RESULTS:

In present study, out of total 313 covid-19 positive patients, majority were males (61%) with 24.6% of age group 56-65

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years 50.5% patients age group 41-60 years. Majority participants (70.3%) were of Severe and the lower proportion (29.7%) were Very Severe clinically. Higher proportion (54.3%) had Raised IL-6 the lower proportion (45.7%) was for Normal Grade respectively. Higher proportion (57.5%) had Raised CRP Level and the lower proportion (42.5%) had Normal CRP Level. USG Abdomen showed that a higher proportion of Covid 19 patients i.e., 168 (53.7%) had Raised renal echotexture as compared to 145 (46.3%) which had no significant anomaly. A statistically significant correlation (P<0.05) was found between Mean Serum Urea value & Mean Serum Creatinine value among Covid 19 severity Group. The mean Serum Urea value in the Very Severe group was higher i.e., 72.00 ± 42.840 as compared to mean Serum Urea in severe group i.e., 68.89 ± 47.489. among Covid 19 severity Group. The mean Serum Creatinine value in the Very Severe group was higher i.e., 2.2370 \pm 1.33429 as compared to mean Serum Urea in severe group i.e., 2.1483 ± 0.88379 .

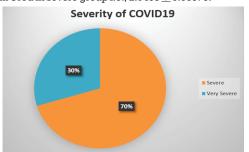


Figure 1: Distribution of Study Participants according to Disease Severity

Source: M. Y. Hospital, Indore, Madhya Pradesh, India

Table-1 Distribution of Study Participants according to gender

OUTCOME	Frequency (n)	Percentage (%)
Male	192	61.3%
Female	121	38.7%
Total	313	100%

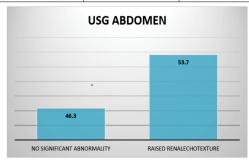


Figure 2: Distribution of USG findings observed among the Study Participants

CONCLUSION:

In conclusion, approximately one in six patients with COVID.19 eventually develops AKI. Kidney injury on admission, development of cytokine storm, and ARDS during the course of the disease are influence factors of AKI development and could be used to stratify COVID.19 patients to mitigate AKI risk. Cytokine storm and secondary bacterial infections may be responsible for AKI development in COVID.19 patients. Our study suggests a tremendously high incidence of AKI in our cohort of critically ill COVID-19 patients, along with an independent association between AKI and outcome.

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