

**Abstract Submission No. : 2294**

**Coronavirus (COVID-19) induced oxidative stress and proinflammatory cytokines leads to Acute Kidney Injury (AKI).**

**Dr Rajiv Nehra**<sup>1</sup>, Dr Manju Nehra<sup>2</sup>

<sup>1</sup>Department of Biochemistry, Rajkiya Medical College, Jalaun (Orai), U.P, India

<sup>2</sup>Department of Dental, Community Health Center, Siyana, Bulandshehar, U.P, India

**Objectives:** Novel coronavirus disease (COVID-19) is a newly discovered acute infectious disease caused by the SARS-CoV-2 virus, characterized by acute respiratory diseases and can affect multiple organs such as the kidneys, the heart, the digestive tract, and blood. Acute Kidney Injury (AKI), biochemically characterized as abnormality in kidney function test which causes accumulation of creatinine and blood urea and functionally by a rapid decline in the glomerular filtration rate (GFR). Inflammatory cytokines and various growth factors in renal cells modulate the local response are responsible for AKI.

**Methods:** 10 ml of fasting venous blood was collected from the antecubital vein in a plain, fluoride and EDTA vacutainers. The blood sample was centrifuged and stored at 4<sup>o</sup> C for biochemical and immunological investigations. The study group consisted of n=25 healthy individuals (Group I), n=25 COVID-19 (Group II) of either sex aged between 40-65 years Serum levels of inflammatory markers (IL-6 & CRP), antioxidants (Glutathione) were estimated.

**Results:** Concentration of inflammatory molecules such as IL-6  $9.24 \pm 1.20$  and  $36.76 \pm 11.56$ ; hs-CRP  $0.90 \pm 1.10$  and  $2.18 \pm 0.90$  was significantly elevated in Group II. GSH were significantly lower in the group II without AKI when compared to controls.  $7.10 \pm 0.58$  and  $5.80 \pm 0.80$ . Mean value for Urea  $25.34 \pm 6.29$  and  $116.57 \pm 39.07$ ; S.Creatinine  $1.04 \pm 0.27$  and  $12.12 \pm 3.79$  were significantly elevated in Group II.

**Conclusions:** Results of the present study indicates that inflammatory markers and oxidative stress are increased with decreased antioxidant defense levels in patients will cause AKI. AKI as a complication of COVID-19 could not only increase the mortality rate of COVID-19 but also, increase the risk of developing chronic kidney disease. It is therefore important to pay more attention to finding AKI in a timely manner and treat the patients properly when we are fighting COVID-19.

Biochemical Parameters and inflammatory response in COVID-19

**TABLE I- BIOCHEMICAL PARAMETERS AND INFLAMMATORY RESPONSE IN NORMAL AND COVID-19 POSITIVE PATIENTS.**

INDIVIDUALS	Urea (mg/dl)	Creatinine (mg/dl)	GSH	IL-6	Hs-CRP
Normal n=25 (Group I)	25.34 ± 6.29	1.04 ± 0.27	7.10 ± 0.58	9.24±1.20	0.90±1.10
COVID-19 Positive n=25 (Group II)	116.57 ± 39.07**	12.12± 0.27**	5.80 ± 0.80**	36.76±11.56**	2.18±0.90**

The

data were expressed as mean ± SD. The data was analyzed using the student's t- test. \*indicates p<0.05 and statistically significant, \*\*indicates p<0.001 and statistically highly significant

**TABLE-I**

With respect to biochemical parameters, Mean value of Urea 25.34±6.29 and 116.57±39.07; S.Creatinine 1.04±0.27 and 12.12±3.79 were significantly elevated in Group II.

Concentration of cytokine IL-6 9.24±1.20 and 36.76±11.56; acute phase protein hs-CRP 0.90±1.10 and 2.18±0.90 was significantly elevated in Group II as compared to Group I. GSH were significantly lower in the group II without AKI when compared to controls. 7.10±0.58 and 5.80±0.80.