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Renal Profile in Covid 19 Infected Patients-A Retrospective Study

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ABSTRACT
COVID-19 infections effected the whole world for last one and half years with lots of casualties. Though SARS-COV-2 is a virus infecting the respiratory system, it also affects other organs of the human body like the heart, kidney, abdomen etc. In this study, we found that there is derangement in the renal profile in COVID-19 infected cases as we have analysed the datas taken from the electronic data record of central clinical laboratory (Biochemistry). High levels of Urea, creatinine and potassium effects the prognosis of the disease caused by SARS-COV-19

KEY WORDS: SARS-COV-2, Renal profile, Respiratory infection

I. INTRODUCTION:

A highly contagious respiratory illness caused by a novel corona virus called SARS-COV-2 (severe acute respiratory syndrome corona virus - 2) started first from Wuhan in the Hubei province of China then spread to more than 150 countries and became a global problem within months. On February 11, 2020 the World Health Organisation (WHO) named the illness Corona virus disease 2019 (COVID 19) and later declared it a pandemic on March 11, 202. (1) The western world became most affected by the illness especially the United States and the European countries by the 1st wave of COVID-19 infection. World meter COVID-19 corona virus pandemic 2021 published online April 10, shows that since the middle of March 2021, the 2nd wave has started and on April 9, 2021, highest number of cases (144,8290) has been identified in India (putting India next to the United States. Classically, patients with COVID 19 present with cough, fever, dyspnoea, fatigue and respiratory failure along with multi organ damage in severe cases.(2,3,4). The disease is highly contagious and spreads in cluster out breaks. Person to person contact via respiratory droplets is the primary mode of transmission. Initial reports from Wuhan suggested the prevalence of acute kidney injuries (AKI) in COVID-19 patients was quite low, ranging from 3-9% however the subsequent analysis showed a relatively high AKI burden of 15%(6). Some cases of COVID-19 pneumonia presented with kidney injury (7) and pathological findings from autopsies also revealed renal damage from the corpses of patients with COVID-19(8) thus SARS-COV-2 may include kidney tropism.

The renin-angiotensin-aldosterone system is involved in electrolyte balance and blood pressure stabilisation. (9) The kidneys convert the precursor (already present in the blood) into renin and secrete directly into the circulation. Plasma renin then carries out the conversion of angiotensinogen released by the liver to angiotensin I(10). Angiotensin I is subsequently converted to angiotensin II by angiotensin converting enzyme(ACE) found on the surface of vascular endothelial cells predominantly those of the lungs(11). Renin angiotensin system is a very complicated system in the body that involves multiple organs- the liver, kidneys, adrenal glands and the lungs that regulate blood pressure, electrolyte levels and other functions. Angiotensin converting enzyme(ACE) converts a hormone called angiotensin I to angiotensin II. The main place of the conversion is the lungs. Angiotensin II increases blood pressure(12) and it stimulates the production of another hormone called aldosterone, a steroid excreted by the adrenal gland. Aldosterone promotes the absorption of water and sodium in the kidney and it promotes the removal of potassium from the body through the kidneys(13).Different factors could result in AKI during SARS COV2 infection. Direct viral damage and/or disturbed haemodynamics of the kidney might account for AKI in Covid 19. Further sepsis associated pathway are probable mechanism for kidney injury.(14) Angiotensin converting enzyme and dipeptidyl peptidase -4 both expressed on renal tubular cells, were identified as binding partners for SARS-COV and MERS-COV respectively .(15,16) Viral RNA has been identified in kidney tissues and urine in both infections. (17,18) Recently Zhong's lab in Guangzhou successfully isolated SARS-COV-2 from the urine sample of an infected patient suggesting the kidney as the target of this novel corona virus.(19) COVID 19 virus attaches to the ACE 2 receptor on the cells in the lungs in order to get into the cells. The virus uses the ACE2 receptor level in the blood to gain entry into the

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cells. Because of the virus taking up all of the receptors and /or the lungs are being destroyed by the infections and the immune responses there is less angiotensin II and less aldosterone. As a result there may be high potassium level in the blood without having kidney failure. The spike protein is a key determinant of the virus tissue tropism and host range SARS-COV-2 competes with angiotensin II for ACE-2 in terms of internalization. The binding however blocks ACE2 activity and thus reduces the enzyme expression in the membrane. (19)

AIMS AND OBJECTIVES:
To see the renal profile (urea, creatinine, sodium, potassium, total protein and albumin) in the COVID-19 positive patients To see the deviation of renal profile in COVID 19 patients from normal healthy individuals.

STUDY DESIGN:
The study is a hospital based retrospective observational study done with the datas collected from Central Clinical laboratory (Biochemistry), Gauhati Medical College.

STUDY POPULATION:
The datas are taken from the central clinical laboratory (Biochemistry) where tests were done in auto analyser Vitros 5600(OCD) at the time of admission of COVID-19 patients (confirmed by RT-PCR test)

SAMPLE SIZE: 111

STUDY DURATION: I(ONE) MONTH

INCLUSION CRITERIA:
- COVID 19 positive patients confirmed by RT-PCR test
- Patients without renal disease
- Patients without co-morbidities like diabetes mellitus, hypertension, heart disease etc
- Patients above 18yrs

EXCLUSION CRITERIA:
- Patients with renal disease
- Patients with comorbidities like diabetes mellitus, hypertension, heart disease etc
- Patients below 18yrs

II. METHODOLOGY:

It is a retrospective observational study. The datas of the renal profile (blood, urea,serum creatinine, sodium, potassium, total protein and albumin) of COVID 19 positive patients were collected from the datas present in the electronic record present in the central clinical laboratory (Biochemistry) of the Gauhati Medical College, Guwahati. The investigations were done at the time of admission of these COVID-19 positive patients confirmed by RAT and RT-PCR. These datas were analysed with the help of Minitab-19 and in M S excel.

ETHICAl PERMISSION was taken from the Institutional Ethical committee

III. RESULTS AND ALALYSIS:

Statistical analysis of the findings will be done after completion of the data collection.

Table 1: Statistics of renal profile in COVID 19 cases

Parameters	Urea	Creatinine	Sodium	Potassium	T. protein	Albumin	BUN
Mean+/-SD	59.67+/-67.67	2.21+/-3.9	136.84+/-7.27	4.24+/-0.99	6.83+/-1.03	3.84+/-0.82	27.57+/-31.62
SE	6.42	0.37	0.69	0.09	0.09	0.08	3.0
MIN-MAx	11-471	0.4-22.3	123-158	1.9-7.4	4.5-9.9	1.8-5.7	5.14-220

Table 2: Percentage variation of parameters

PARAMETERS	RANGE	PERCENTAGE
UREA	0-60mg/dl	72.97%
	>60%	27.02%
CREATININE	0-1.5mg/dl	75.67%
	>1.5mg/dl	24.32%
SODIUM	<135mmol/L	34.23%
	135-145mmol/L	58.55%
	>145mmol/L	7.2%

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POTASSIUM	<3.5mmol/L	3.6%
	3.5-5mmol/L	81.91%
	>5mmol/L	15.31%
TOTAL PROTEIN	<6gm/dl	18.91%
	6-8gm/dl	69.36%
	>8gm/dl	11.17%
ALBUMIN	<3gm/dl	23.42%
	3-5gm/dl	72.07%
	>5gm/dl	4.5%

Figure 1: Gender ratio of COVID 19 patients

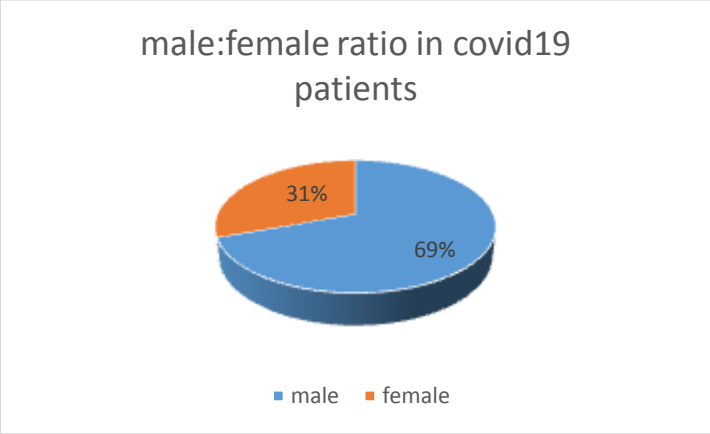

Figure 2: Frequency curve for urea

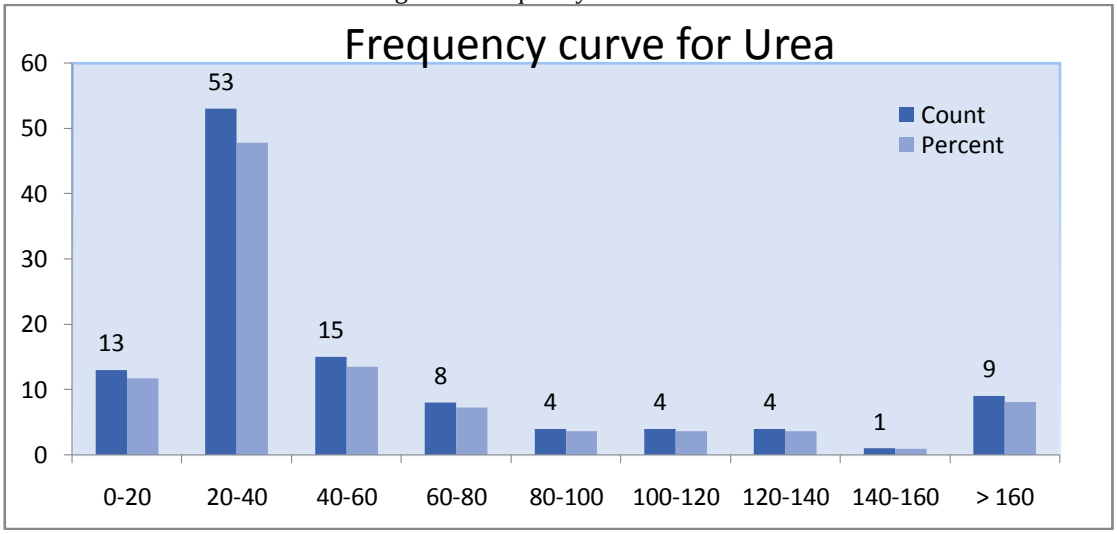

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Figure 3:Frequency curve for creatinine

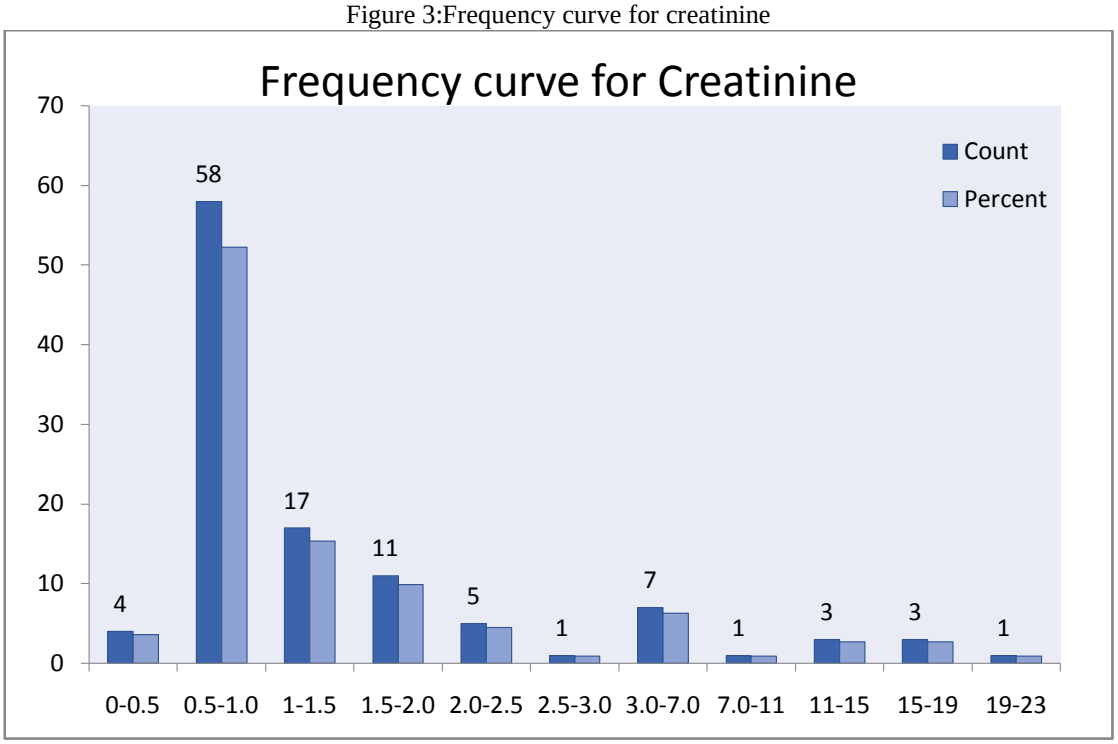

Figure 4: Frequency curve for sodium

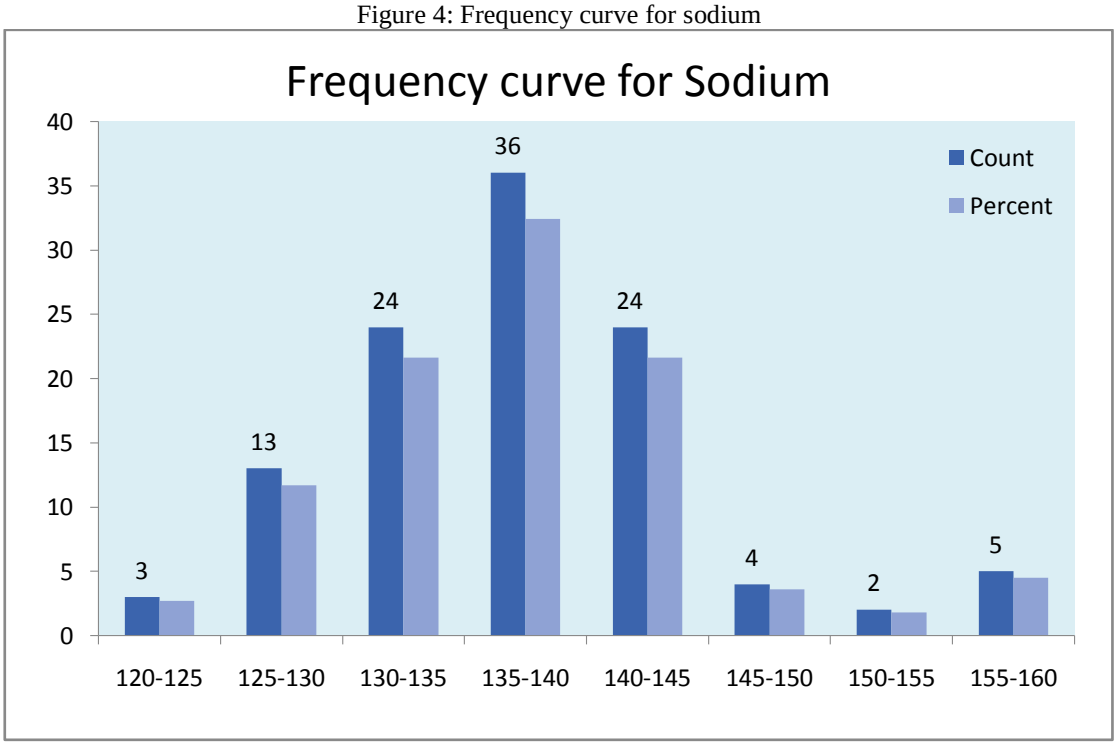

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Figure 5: Frequency curve for potassium

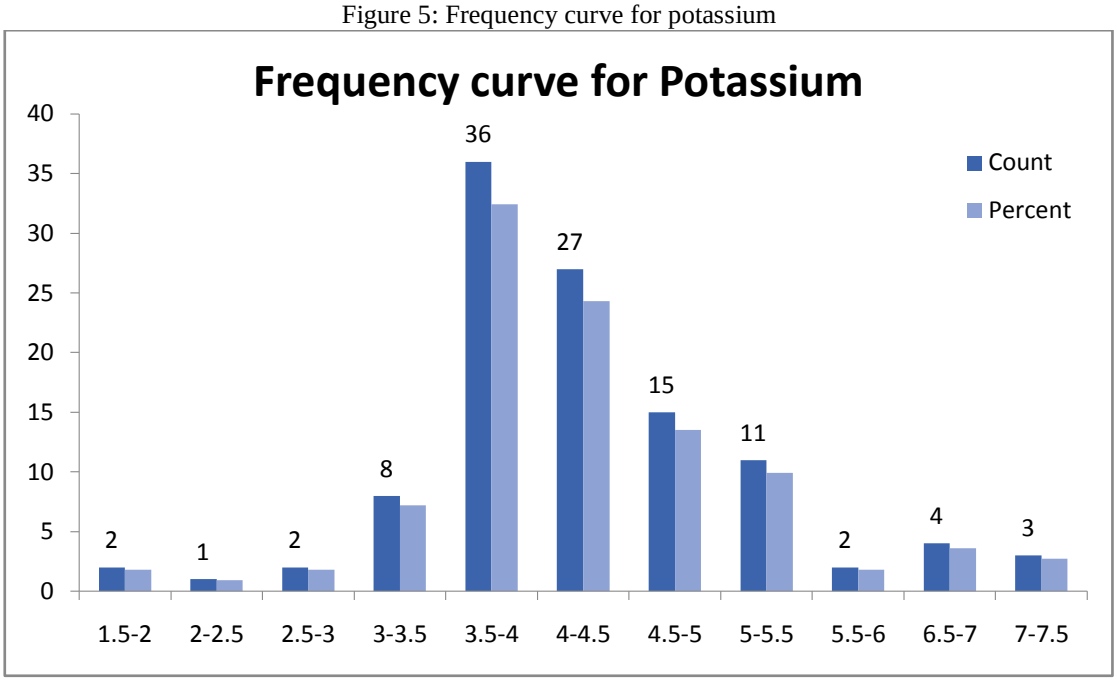

Figure 6: Frequency curve for T. Protein

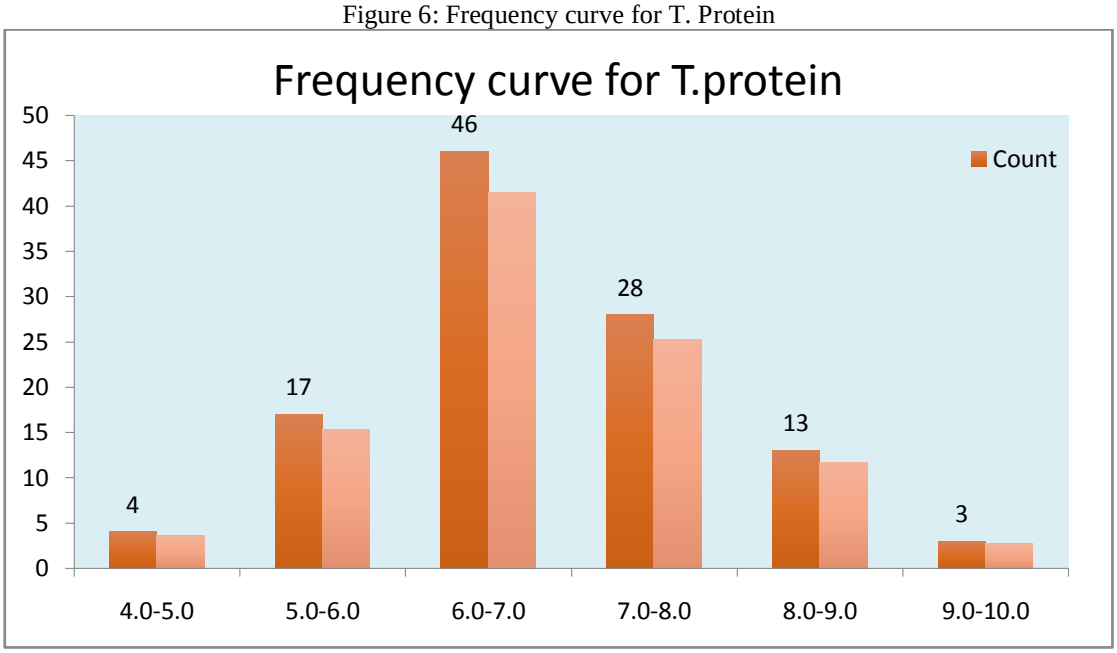

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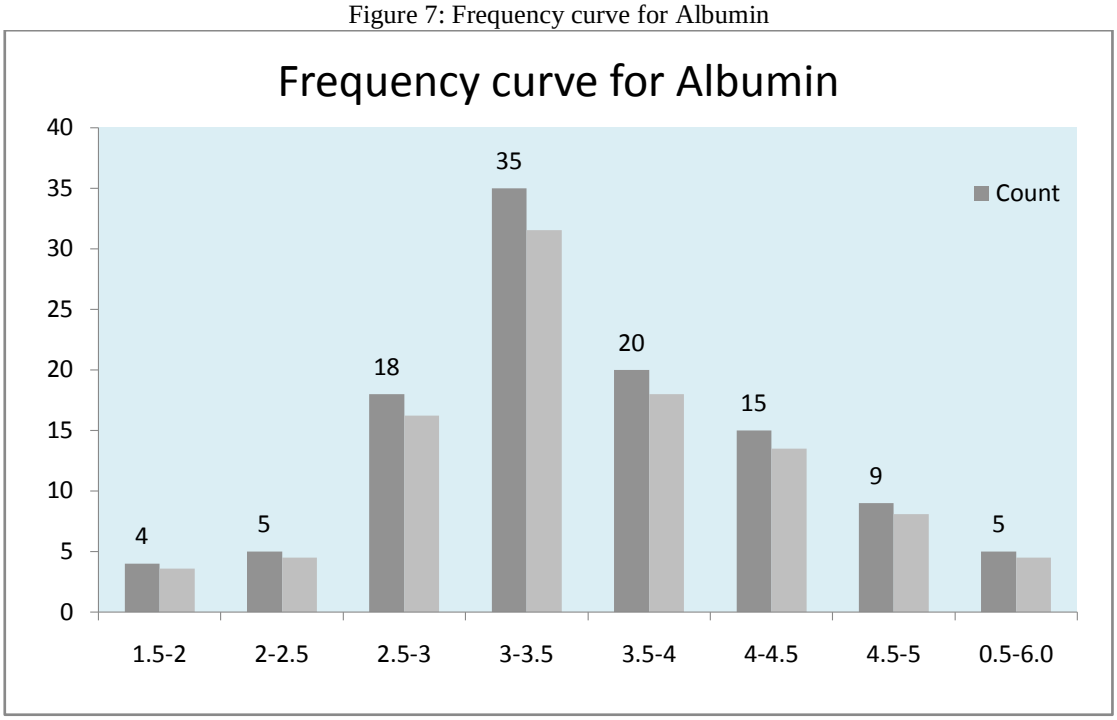
Figure 7: Frequency curve for Albumin



The mean +/- SD of urea is 59.67 +/- 67.67 with a SE of 6.42 and a range between 11-471 as shown in table 1. The frequency distribution of urea is shown in figure 2. The mean +/- SD of creatinine is 2.21+/-3.9 with a SE of 0.37 and a range between0.4 to 22.3 as shown in table 1. The frequency distribution curve is shown in figure 3.The mean +/- SD of potassium and sodium are 136.84+/-7.27 and 4.24+/-0.99 respectively. The sodium level ranges from 123 to 158 and potassium level from 1.9 to 7.4. The frequency distribution of sodium is shown in figure 4 and potassium in figure 5. The mean +/- SDof T.protein and albumin are 6.83 +/- 1.03 and 3.84 +/- 0.82 respectively. The T. protein level ranges between 4.5 to 9.9 and albumin level ranges from 1.8 to 5.7.The frequency distribution curves are shown in figure 6 for T.protein and Figure 7 for albumin. The percentages of normal and abnormal ranges of the parameters are shown in table 2. The sex ratio is shown in figure 1 with 69% male and 31% female.

IV. DISCUSSION:

In this study, the datas were taken from the Central Clinical laboratory electronic record and analysed. It was found out that 27.02% of COVID 19 positive cases have urea level above 60mg/dl as shown in table 2. The mean +/- SD of urea is 59.67 +/67.67 with a SE of 6.42. the mean+/- Sd of BUN is 27.57 +/- 3.62 with a SE of 3.0 as shown in table 1. In this study it was also seen that 24.32% of COVID cases had creatinine level more than 1.5mg/dl as shown in table 2. The mean +/- Sd of creatinine is 2.21 +/- 3.9 with a Se of o.37 and a range between 0.4 to 22.3 as shown in table 1.10.8% of patients without chronic kidney disease showed a mild increase in blood urea nitrogen (BUN) and serum creatinine after infection with SARS-COV-2 (20) and during treatment of pneumonia. This correlates with our findings. However the increase value of serum creatinine were all < 26µmol/L within 48 hours. In addition patients without chronic kidney disease showed traces of 1+albuminurea in urine during treatment of pneumonia (20). In our study 18.91% COVID positive cases showed serum protein level less than 6gm/dl and 23.42% cases showed albumin level below 3gm/dl as shown in table 2. Recent reports have shown higher abnormalities of renal abnormalities. A study of 59 patients developed massive albuminuria on the first day of admission and 63% developed proteinuria during their stay in the hospital. Blood urea nitrogen(BUN) was elevated in 27% overall and in two third patients who died. (21) Cheng et al (1) recently reported that amongst 710 consecutive hospitalised patients with COVID-19, 44% had proteinuria on admission. The prevelance of elevated serum creatinine and BUN was 15.55 and14.1% respectively. This correlates our findings. In this study 34.23% of COVID19 positive patients had sodium level less than 135mmol/L and 7.2% of patients had sodium level above 145mmol/L. At the same time 3.6% of COVID-19 positive patients had

potassium level less than 3.5mmol/L and 15.31% of patients had potassium level more than 5mmol/L. A recent study showed that COVID 19 severity is associated with lower concentration of Potassium (22). Lui S. Zhang L et al (23) in their study found that the risk of death was significantly increased in patients with COVID-19 who has potassium >/= 5.0mmol/L as compared with the 4.0 to < 4.5mmol/L group. Previous studies showed that the prevalence of AKI in severe acute respiratory syndrome (SARS) was 6.7% and mortality rate was upto 91.1% in SARS patients complicated by AKI (24). The reported incidence of AKI in COVID-19 patients were lower in a number of studies (24, 25, 26, 27, 28). The SARS-COV-2 attaches to ACE2 and induces a down regulation of membrane bound ACE 2 that promotes accumulation of angiotensin II by reducing its degradation into angiotensin1-7. Thus COVID-19 mediated angiotensin II accumulationmay promote an imbalanced RAAS activation leading to inflammation, fibrosis, vasoconstriction (29). Moreover ACE 2 usually interacts with ATI receptors forming a complex that prevents internalization and degradation of membrane bound ACE 2 into lysosomes. Accumulation of angiotensin II decreases this interaction and induces ubiquitination and internalization of membrane bound ACE2 into lysosomes (30).

V. CONCLUSION:

In SARS_COV-2 infection, there is derangement of renal parameters to some extend. As the renal parameters were studied were studied at the time of admission of the cases, follow up of the cases are required to see the effect of the derangement in the course of the disease.

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