2022-10-06

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By: - Amene Abate (Md, Internal Medicine Resident)

October**, 2022**

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BY: - AMENE ABATE (MD, INTERNAL MEDICINE RESIDENT)

A THESIS REPORTSUBMITTED TO DEPATMENT OF INTERNAL MEDICINE, BAHIR DAR UNIVERSITY COLLEGE OF MEDICINE AND HEALTH SCIENCES INPARTIAL FULFILMENT OF THE REQUIREMENTS FOR CERTIFICATE OF SPECIALITY IN INTERNAL MEDICINE.

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OCTOBER, 2022

BAHIR DAR, ETHIOPIA

Candidate's Declaration Form

This is to certify that the thesis entitled **"Prevalence of acute kidney injury and associated factors among hospitalized covid 19   patients at TibebeGhion Specialized Hospital, Bahir Dar, Ethiopia, 2022"**in partial fulfillment of the requirement for certificate of specialty in internal medicine in College of Medicine and Health Sciences, Department of internal medicine, Bahir Dar University, is a record of original work carried out by me and has never been submitted to this or any other institution to get any other degree or certificates. The assistance and help I received during the course of this investigation have been duly acknowledged.

Name of the student: DrAmene Abate (MD, Internal medicine resident)

Signature _____   Date: _____

Acknowledgements

ABSTRACT

Background:Although diffuse alveolar damage and respiratory failure are the key features of coronavirus disease 2019 (COVID-19), the involvement of other organs such as the kidney has also been reported. The reports of the prevalence of acute kidney injury (AKI) in COVID-19 patients vary widely. However, AKI among hospitalized patients with COVID- 19 in Ethiopia is not well studied.

Objective: To determine the prevalence of AKI and its associated factors in hospitalized medical patients from February 2020 to March 2022 at TibebeGhionspecialized hospital, Bahir Dar.

Methods: A cross sectional study design was used with a chart review for patients admitted to medical wards and all the necessary data was collected from hospitalized medical patients' cards using the pre-developed data collection check list. Data was collected from December 2021 to April 2022. A total of 388patient charts were included in the study. A systematic random sampling method was used to identify the participants chart. The collected data was cleaned, coded and entered and analyzed using IBM SPSS statistics data editor version 27. Multivariable binary logistic regression analysis was conducted. $P < 0.05$ will considered as statically significant.

Results: The overall prevalence of AKI among COVID-19 patients was 25.1% (95% CI 21.1, 29.6).Those withMultiple co morbidity (AOR =4.036%, 95 CI : (1.089,19.739)), Sever covid-19 disease (AOR = 10.21% 95 CI : (1.027,101.644),above 4weeks symptom durations(AOR = 7.317%, 95 CI : (1.516 ,35.306), use cefepim(AOR=3.149%, 95%CI: (1.049,9.488), Lymphopenia(AOR=5.089%, 95% CI:(1.352,19.146), 1 − 2 weeks hospital stay (AOR=3.647%, 95% CI:((1.328,10.01)found to be associated significantly with increased risk for AKI.

Conclusion: Acute kidney injury is a common feature in severely ill patients with COVID-19 pneumonia presenting with acute hypoxic respiratory failure. It is more common in patients with multiple comorbidity, long covid patients, those with lymphopenia as well as using cefepim. These data indicate that patients with COVID-19 should be monitored for the development of AKI while in hospital or after discharge and measures have to be taken to prevent this.

Key words: Acute kidney injury, acute respiratory distress syndrome, Corona virus disease 19.

Lists of Acronyms and Abbreviations

Covid 19------- Corona virus disease 2019

AKI------- Acute kidney injury

ARDS ------ Acute respiratory distress syndrome

DM----------Diabetic mellitus

HTN---Hypertension

ESRD----End stage renal disease

 Cr---------Creatinine

BUN------- Blood urea nitrogen

GFR…..Glomerular filtration rate

SARS COV 2… Sever acute respiratory syndrome corona virus 2

ACE 2 …. Angiotensin converting enzyme 2

ICU…. Intensive care unit

TGSH…. Tibebe Ghion specialized hospital

IL…. Inter leukine

ARF…. Acute renal failure

LDH….. Lactate dehaydrogenious

MERS COV….Middle east respiratory syndrome corona virus

PCR…. Polymerase chain reaction

RT- PCR…. Reverse transcription – polymerase chain reaction

ESKD…End stage kidney disease

MDRD….Modification of diet in renal disease

WHO …….World health organization

CVD--------- Cardio vascular disease

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1. Introduction

1.1 Background

COVID-19, a novel respiratory illness caused by SARS CoV-2, was discovered for the first time in Wuhan in December 2019. COVID-19 symptoms range from a common cold to a high fever and acute respiratory distress syndrome (ARDS), which causes multi-organ failure such as kidney disease (1). In COVID-19 cases, the kidney may be a target for organ injury due to angiotensin converting enzyme-2 (ACE-2), the coupling site for SARS-CoV-2 that is highly expressed in proximal tubule cells and podocytes. AKI has been identified as an extreme COVID-19 obstruction with a higher risk of death in critically ill patients based on several case series and retrospective reports (2),(3). In early studies conducted in China and Italy, the frequency of AKI varied widely from 0.5 to 29%. Data from the USA were restricted ICU patients with a high incidence rate of 37 to40% and an in-hospital mortality rate of 35 to 41% (2).The AKI incidence is generally high in some reports andnegligible in others. For instance, Guan et al. (1) foundthat the AKI frequency was only 0.5% in a multi-center study of 1019 cases. However, Cheng et al. (4) foundthat in a single center study the AKI prevalence was3.2%. Two distinct cohorts revealed a significantly higheroccurrence of AKI. The first cohort had 66% AKI rate inacohort of 193 serious patients, while the second cohort had 50% AKI rate in non-survivors (5)(6)(7).So far, littleinformation on AKI in COVID-19 has been published inEthiopia. The goal of this study is to assess the occurrenceof AKI in hospitalized COVID-19 patients at TGSH as well as factors associated with AKI inCOVID-19 patients.

1.2 Statement of the Problem

Corona virus disease 2019 (COVID-19) imposes a serious public health pandemic affecting thewhole world, as it is spreading exponentially. Besides its high infectivity, SARS-CoV-2 causes multiple serious derangements, where the most prominent is severe acute respiratory syndrome as well as multiple organ dysfunction including heart and kidney injury. While the deleterious Impact of SARS-CoV-2 on pulmonary and cardiac systems have attracted remarkable attention, the adverse effects of this virus on the renal system is still underestimated.

Although the respiratory tract is primarily involved in this disease, kidney affectation is increasinglyreported and has been shown to worsen the prognosis of the disease. Current evidence show that kidney disease is not uncommon in patients with coronavirus infection especially in those with COVID-19 and may arise from a constellation of factors such as hypotension, sepsis, rhabdomyolysis, multi-organ failure, use of nephrotoxic medications as well as direct infection in some cases.

While initial reports fromWuhan, China suggested that the burden of AKIwith COVID-19 was relatively low, ranging from 3%to 9% （9）. Subsequent studies demonstrated incidence rates as high as 15% (8).

AKI was more common in hospitalized mild COVID-19 patients than in home-isolated and ICU COVID-19 patients (15.0% versus 10.8% and 14.2%, respectively). The overall occurrence rate of AKI was significantly higher in COVID-19 patients (n=91, 14%).

1.3 Significance of the Study

Kidney disease was highly prevalent in hospitalized patients with COVID-19, and emphasized the need for early detection and effective intervention. Early identification of AKI, as well as prompt intervention, can improve COVID-19 patient outcomes.

So far, little information on AKI in COVID-19 has been published in Ethiopia. This study is designed to assess the occurrence of AKI in TGSH admitted COVID-19 patients as well as the factors associated with AKI in COVID-19 patients; helps to inform healthcare professional to evaluate and closely monitor hospitalized medical patients for the development of AKI and intervene early.

The study also help leadership of the health system, program managers and policymakers to know the status and consider establishing national guidelines and allocate budget.

1.4 Literature Review

1.4.1 Origin and epidemiology

SARS-CoV-2 is a novel mutant of the coronavirus family that is causing the most recent and ongoing pandemic. The coronavirus is thought to have been transmitted at first instance from bats to humans. A wet wild animal market is likely considered to be the primary focus. The first cases of human infections were then reported in the city of Wuhan, the capital of Hubei Province of China. This was followed by widespread of the pandemic to many countries around the globe.(9).

1.4.2 Pathogenesis

COVID-19 primarily targets the respiratory system, causing a wide clinical spectrum from mild symptoms to adult respiratory distress syndrome. The pathogenesis is mediated in severe cases through the so-called cytokine storm. This involves the secretion of large amounts of pro-inflammatory cytokines and chemokines including IL8, IL 6, IL9, IL10, and many others. Pathogenic mechanisms in the kidneys are not fully elucidated, but the suggested mechanisms are through attachment of the virus to ACE2 receptors. Excess secretion of cytokines leads to multiorgan failure in a percentage of patients, including acute kidney injury (AKI), through tissue hypoxia (10).

1.4.3 AKI as a complication of COVID-19

Acute kidney injury is a common complication of several infections. In the previous SARS-CoV-2 outbreak in 2003, the incidence of AKI was as low as 6%. (10)(11). Nevertheless, AKI case fatality rate was high. (12).

There is heterogeneity among studies regarding the reported incidence of AKI. This may be attributed to inconsistencies in applying AKI definitions or due to genetic variability that merits further studies. Some reports have shown that the incidence of AKI is significant, while others report that the incidence is marginal. Guan et al. have shown, in a large cohort of confirmed COVID-19 cases that the prevalence of AKI was as low as 0.5%. This increased in patients with severe COVID-19 to 2.9% (1).

In other cohorts, the incidence of AKI in confirmed cases of COVID-19 was higher.
In a cohort of 193 patients, the overall incidence of AKI was 28% and the incidence in severe cases was 66% (13). In another cohort of 191 patients, the incidence of AKI in non-survivors was 50% (14). In the study by Hu et al., AKI was present in 17 of all 323 patients (5.3%);

however, the incidence of AKI in patients with critical COVID-19 was 38.5%. Furthermore, in this cohort, most patients who had AKI (14 out of 17) had unfavorable outcomes (15).

In a cohort of electronically collected data from 4,759 hospitalized patients who were tested for COVID-19 between 5 March 2020 and 12 May 2020 in United Kingdom; AKI developed in 304 Patients with COVID-19 (26.2%) and 420 patients without COVID-19 (12.4%) (37).

In a study done on clinical presentation of COVID 19 related deaths in Ethiopia ; the major diagnosis of patients during admission was respiratory condition 39/92 (42.4%) . renal disease contributing for 31/92(33.4%) , from this 17 (18.5 %) has AKI and rest 14 (15.2 %) has CKD (16)

During a previous SARS outbreak in 2003, a study of postmortem kidney biopsies examined usingelectron transmission microcopy, failed to detect any viral particles in kidney tissues. This finding supports the theory that most of the kidney pathogenesis in the earlier SARS outbreak was in the context of multi organ failure. The pathogenesis of AKI may be multifactorial. Suggested mechanisms are direct cytopathic effects on kidney tissues, as denoted by the retrieval of the viral RNA from urine samples (4). The direct cytopathic effect of COVID-19 on kidneys is now more evident, as it has been shown that there is overexpression of both ACE2 receptors and a cleavage spike protein in podocytes and proximal tubular cells (17). This experimental evidence is of paramount importance and can explain proteinuria in patients with COVID-19. Interestingly, the latter experiment reports variable expression of cleaved S protein such that there is low expression in the Chinese race as compared to Caucasians. Important pathological evidence was reported by Diao et al. The pathology team managed to confirm the visualization of the SARS-CoV-2 viralparticles in the renal tubular cells of postmortem kidney biopsies (18), (19). The difference in kidney tropism between SARS-CoV and SARS-CoV-2 may be attributed to the affinity to ACE2 receptors in the kidneys. Tissue hypoxia, in the context of massive cytokine secretion, is a key renal pathogenic mechanism. Rhabdomyolysis and raised creatinine kinase have been observed in a few cases (1). It was also noticed in one cohort that AKI occurred later to acute cardiac injury,
suggesting a temporal relationship between cardiac injury and AKI and the possible occurrence of cardio renal syndrome(14).In a recent single case report, collapsing variant of focal segmental glomerulosclerosis was diagnosed in renal biopsy of African-American woman, who tested positive to COVID-19. The patient presented with confusion and rapidly deteriorating kidney function, she improved markedly with the initiation of dialysis (20).

A study done in Egypt shows; AKI was more common in hospitalized mild COVID-19 patients than in home-isolated and ICU COVID-19 patients (15.0% versus 10.8% and 14.2%, respectively). The overall occurrence rate of AKI was significantly higher in COVID-19 patients (n=91, 14%). Hemodialysis, on the other hand, was required in 76% of the extreme ICU COVID-19 Patientswho developed AKI (22/29). The absolute number of patients with AKI

COVID-19 who requiredhemodialysis was 34 (37%). This accounted for 5.2% of all COVID-19 patients and 37% of those with AKI. The mortality rate in COVID-19 patients with or without AKI was 15.4% and 4.8%, respectively (8).

In a study from two tertiary Centers in South Africa, AKI occurred in 374/1102 (33.9%) patients admitted to the two hospitals (35). A recently released study on acute kidney injury in severe and critically ill patients with COVID-19 in ICU of Eka Kotebe Hospital, Addis Ababa Ethiopia the overall incidence of AKI was 60.7%.(36).

1.4.4 Hematuria and proteinuria

In the largest prospective cohort of kidney diseases in COVID-19, it was found that hematuria occurred in 26% of patients and proteinuria occurred in about 43% (4). Large prevalence of proteinuria could be explained by the finding of the above-mentioned experimental study that showed expression of ACE 2 receptors in podocytes and proximal tubular cells (21). However, quantification of proteinuria, using 24 h urinary collection or protein to creatinine ratio, was notdone within the investigation battery. Kidney biopsy has not been attempted in any patient. In this prospective report, the presence of hematuria or proteinuria signaled poor outcome, as measured by in-hospital mortality

1.4.5 Factors associated with renal impairment in coronavirus infection

Factors shown to be significantly associated with acute renal impairment during the SARS epidemic were age, sex, and presence of acute respiratory distress syndrome, diabetes and heart failure (22). The incidence of acute renal impairment was shown to be higher among the elderly (23) ,(22)and in the presence of co-morbidities such as diabetes, heart failure, respiratory failure, acute respiratory distress syndrome (ARDS) and multiple organ system failure (23),(24).SARS patients who subsequently developed acute renal failure (ARF) also had lower plasma sodium and albumin with elevated alanine aminotransferase and plasma LDH on admission compared with those without ARF (23). As in the case of SARS, older individuals are at a higher independent risk for the development of AKI in MERS and COVID-19 (25), (24),(26).Other factors found to be associated with AKI in COVID-19 patients include male gender, multiple pre-existing comorbidities including hypertension, diabetes and cerebrovascular disease, greater severity of illness, increased infection indicators, lymphopenia, elevated D dimer and impaired heart and liver functions(26).

1.4.6 Effects of AKI on outcomes of patients with covid 19

Coronavirus infection: the development of renal failure has been observed to be an important negative prognostic factor in SARS (11),(22).AKI,together with diabetes and use of continuous renal replacement therapy were identified risk factors for mortality in the retrospective study among MERS-CoV patients (25). Also, in anothercohort of MERS-CoV patients, the presence ofelevated Creatinine> 1.5mg/dl and renal failurewere associated with reduced survival of

patients (24). Elevated serum creatinine, elevatedurea nitrogen, AKI, proteinuria and hematuria were independent risk factors for in-hospital death in COVID-19 after adjusting for age, sex, disease severity, leukocyte count and lymphocyte counts (4). Renal disease is therefore considered an independent risk factor for COVID 19 patients´ in-hospital mortality (4), (26),(27).

1.4.7 Conceptual frame work

Figure 1: Conceptual frame work(22)

PETIENT PRESENTATION
- Disease severity
- Duration of symptoms
- Concomitant infections

Co morbidity
- HTN
- DM
- Asthma
- COPD
- CKD
- Heart failure
- CVD
- Cancer
- HIV

ENVIRONMENTAL FACTORS
- Duration of hospital stay

AKI

TREATMENT GIVEN

DEMOGRAPHIC FACTORS
- Residency
- Occu
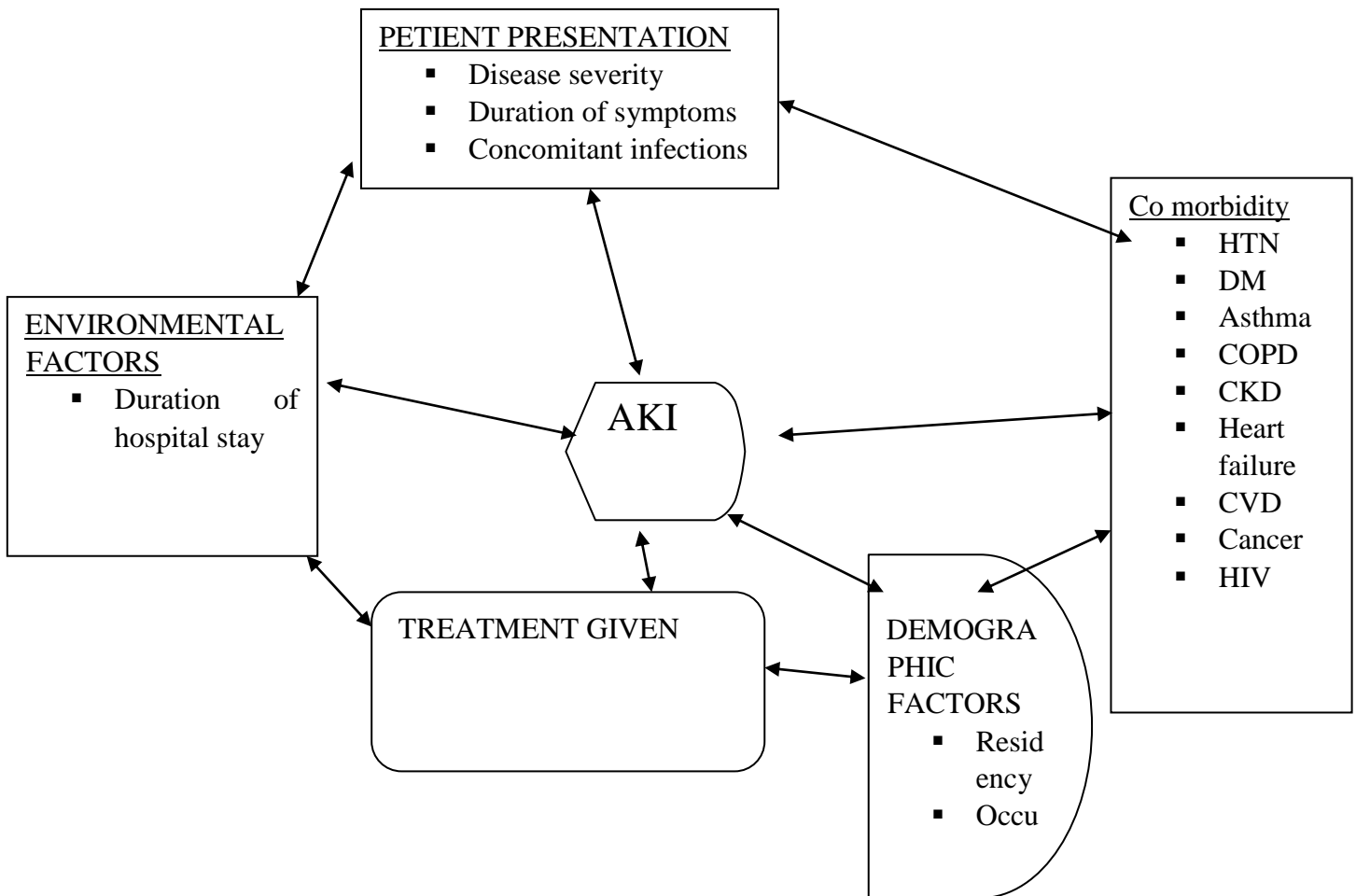
2. Objectives of the Study

2.1 General Objective

To determine the prevalence of in-hospital AKI in COVID-19 patients and to identify factors associated with AKI in hospitalized COVID-19 patientsfromFebruary 2020 to March 2022at TibebeGhion Specialized Hospital, Bahir Dar.

2.2 Specific Objectives

To determine the prevalence of in-hospital AKI in COVID-19 patients.

To identify factors associated with AKI in hospitalized COVID-19 patients.

3. Methods

3.1 Study Design

A cross sectional study was applied based on patient record cards in the year 2020/2021/2022

3.2 Study Area and Period

The study was conducted in TibebeGhion Specialized hospital. The hospital is found in Bahir dar city at 578 km far from Addis Ababa, a capital city of Ethiopia, to the northwest and an elevation of 1,840 meters above sea level. According to Bahidar city administration, Bahir dar city has a population of approximately 221,991 of which 180,174 were urban and 41817 were living at rural kebeles. TibebeGhion specialized hospital is located about 10km south from the city center and about 7 km from the new bus station ('AddisuMeneharia') on the way to Adet District and about 23 km from the Blue Nile Falls (locally called 'Tis abay' (Smoke of Fire).

It is a tertiary university teaching hospital with 450 bed capacity out of which 72 are occupied by medical adult patients. The hospital receives patients who are referred from across the Amhara region and gives outpatient and inpatient services in all major departments. The study was conducted in Tibebe Ghion specialized hospital starting from December 2021 to October 2022.

A total of 1693 covid 19 confirmed patients were admitted at TGSH covid 19 isolation center from February 2020 to March 2022.

3.3.   Source Population

All adult COVID 19 patients admitted to TibebeGhion Specialized Hospital in the year 2020/2021/2022were the source population.

3.4.   Study population

Medical patients who were admitted to TGSH covid isolation centers in the year 2020/202/2022 from February 2020 to March 2022 were the study population. Patients were excluded from the study if they are under 18 yrs, known ESKD prior to admission and those with no Creatinine determination.

3.5. Eligibility Criteria
3.5.1 Inclusion Criteria
Age: > or = 18
Presence of at least 2 serum Creatinine determinations at any point in time.

3.5.2 Exclusion Criteria
Age <18 years old
ESRD on maintenance dialysis
Unavailable or missing data
3.6. Study Variables

3.6.1.Dependent Variables

•Prevalence of AKI in covid 19 patient. (Present, not present or Yes, no)

3.6.2 Independent Variables

Demographic factors
Age
Sex

Comorbidities
Diabetic mellitus.
HTN
Cardiac disease
Asthma
Liver disease
Cancer
Cerebrovascular disease
COPD
HIV

Environmental factors
Duration of hospital stay
Residency
Occupation

Patient presentation

> Disease severity
> Duration of symptoms
> Concomitant infection

Treatment given

> Drugs
> Mechanical ventilator
> Oxygen

3.7 Oprational definition

Coronavirus; This novel human-pathogenic coronavirus, named by the World Health Organization (WHO) "COVID-19", belongs to the Beta coronaviruses (_CoVs) of the coronavirus family (28)that caused the Severe Acute Respiratory Syndrome (SARS-CoV-1) and the Middle East Respiratory Syndrome (MERS-CoV) epidemics in 2003 and 2012, respectively .

AKI ; the primary end point, was defined as KDIGO criteria: a change in the serum creatinine of 0.3 mg/dl over a 48-hour period or 50% increase in baseline creatinine (29). For patients with a previous serum creatinine in the 7–365 days prior to admission, the most recent serum creatinine value was considered the baseline creatinine. For patients without a baseline creatinine in the 7–365 days prior to admission, the admission creatinine was imputed on the basis of a Modification of Diet in Renal Disease (MDRD) eGFR of 75 ml/min per 1.73 m as per the KDIGO AKI guidelines (29). AKI stages were defined using the KDIGO AKI stage creatinine definitions: stage 1 as an increase in serum creatinine of >= 0.3 mg/dl or increase to >= 1.5–1.9 times baseline serum creatinine, stage 2 as an increase to >= 2–2.9 times from baseline serum creatinine, and stage 3 as an increase to more than three times baseline serum creatinine or a peak serum creatinine >= 4.0mg/dl or if the patient received RRT during admission.

Confirmed case of COVID-19; a person with laboratory confirmation of COVID 19, irrespective of clinical sign and symptom (32)

Severe Covid-19- met any of the following criteria

> ✓ Arterial digital oxygen saturation ≤ 93% (at room air),

- The ratio of partial pressure of oxygen to the fraction of inspired oxygen (PaO2/FiO2) ≤300 mmHg (1 mmHg = 0.133 kPa). (36)

Critical Covid-19- met any of the following criteria
- ✓ Respiratory failure requires mechanical ventilation,
- ✓ Shock,
- ✓ Multiple organ failure requires ICU life support
- ✓ The diagnosis of shock was made if patients represented with at least two of the following conditions,
- ✓ The systolic blood pressure being 13.3 kPa (100 mmHg); or lower; (2) the pulse pressure being 4.0 kPa (30 mmHg); or lower; (3) Symptoms consistent with the presentation of shock (i.e. poor peripheral circulation and tachycardia.(36)

Long COVID -signs and symptoms that continue above 4 weeks (34).

Lymphopenia-lymphocyte counts less than one thousand (up to date 2022).

3.8. Sampling Size Estimation

The sample size will be determined by using the following assumptions; proportion of AKI 0.607(from the study done at Severe and Critically ill Patients With COVID-19 in ICU of Eka Kotebe Hospital, Addis Ababa Ethiopia, 2021), 5% margin of error, 95% CI, and 10% for none response rate, yield a sample size of 365 patients, by adding the 10% none response rate the sample size will be 401.

$$ \blacktriangleright \quad n = \frac{Z_{a/2}^{2}\,p(1-q)}{d^{2}} $$

Where   n= sample size

Za½=standard normal distribution corresponding 95% significance= 1.96

P = estimated proportion AKI in covidı9 admitted patient = 60.9% in a study at black lion hospital

d = desired precision =5%

n = (1.96)2 (0.609) (0.391)/ (0.05)^{2}

n = 365

- ○ 10% non-response rate =36
  <u>Sample size = 401(36)</u>

The sample size for the second objective was determined by using double population formula by using epi info version 7 by considering assumption of 95% confidence interval, power 80% and ratio 1:1, OR from previous study.

Table 1: The Sample size calculation by using significant factors

Variables	CI	AOR	Ratio (unexposed/exposed	Power	% of outcome In un exposed	% of out come In exposed	Sample size	Add 10% of NRR
HTN(35)	95%	1.89	1:1	80%	41.77	58.22	312	343
Cancer(30)	95%	5.76	1:1	80%	29.4	70.58	54	58

Therefore, the sample size calculated by using thesingle population proportion is larger than the sample size calculated for second objective. So, that the final sample size of the study was 401.

3.9.   Sampling Technique

Systematic random sampling technique was used. The total number of admitted covid 19 patients in differentisolation centers of TGSH was obtained from the log books. All admitted covid 19 patients in different isolation centers of TGSH were included in the sampling

procedure. The study participants were obtained by calculating K value from the study population with the first participant was selected by using lottery methods.

3.10. Data collection and quality control

Pre-developed data collection chalk list was used to collect data from the medical records.

Socio-demographic data, clinical symptoms, clinical history, comorbidities, laboratory information and management coarse were collected from each cards included in the sample in the work area. Complete blood picture, kidney and liver functions was the lab data. The lab was offer the standard scope of these tests.The chalk listwasfilled by myself using Epi collect5 and checked for completeness.

3.11. Data Processing and Analysis

Data collected by chalk list was coded, entered analyzed using IBM SPSS statistics data editor version 23.Summary statistics such as frequencies and proportions were computed as appropriate.Data was cleaned by running frequencies of all the variables to check for missing value. Descriptive and summary statistics was carried out. Crude and adjusted odd ratio with 95% CI was calculated to determine the strength of association between the dependent and independent variables. Binary and multivariatelogistic regression was used to identify the effect of independent variableand the results was presented by tables, bar graphs, and pie charts. P-value less than 0.05 was considered significant.

3.12 Ethical Considerations

Ethical clearance was obtained from Bahir Dar University Ethics Review Committee. A support letter was sent to TibebeGhion Specialized Hospital. Names wasn't used in collecting the data from the medical files. Confidentiality was maintained by keeping the data collection forms locked in a secure cabinet and the electronic data file was kept securely in a password protected computer. Data obtained in the course of study was only handle by the research team.

4. Results

4.1 Socio demographic factor and baseline Characteristics

A total of 388 cards were selected for the study. Among study participants, 250 (64.4%)were male. Majority of them, 143 (36.9%) were in the age group >60 years. The mean age of the respondents was 53.15 yrs. ± SD of 16.05.

From the total 42.3% (n=164) of patients have at least one comorbidity. The three common singly identified comorbid condition were HTN, DM, and HIV, 8.2%(n=32), 7.7 % (n=30), and 4.1% (n=16), in their respective order of frequency. But about 12.9%(50) has multiple (more than one) comorbidity.
Majority 81.7% (n=317) of the participants come with less than two weeks symptom duration.About52.8 % (n=205) of the participants were mild covid-19 cases, 29.4% (n=144) patients were categorized as having severe disease and the remaining, 17.8 %(n=69) patients as having critical disease in their hospital stay.
Majority; 82.7% (n = 321) of participants had less than one week hospital stay, 13.4% (n= 52) had one week to two weeks hospital stay and the remaining 15 (3.9%) stayed above two weeks.
11.3% (n=44)patients required invasive mechanical ventilation, 14.9%(n=58) took cefepim. (Table2).

Characteristics		Frequency	Percent
SEX	Male	250	64.4%
	Female	138	35.6%
Age	18-20	7	1.8%
	21-30	30	7.7%
	31-40	61	15.7%
	41-50	69	17.8%
	51-60	78	20.1%
	>60	143	36.9%
Pulse rate	<60	1	0.3%
	60-99	276	71.1%
	>=100	111	28.6%
Respiratory rate	<12	0	
	12-24	94	24.2%
	>24	294	75.8%
Temperature	< 36.5	72	18.6%
	36.5-37.5	180	46.4%
	>37.5	136	35.1%
Oxygen saturation	<93	177	45.6%
	So2 93-100	211	54.4%
Fever	FEVER	386	99.5%

Cough	COUGH	385	99.2%
Dysnea	DYSNEA	186	186%
Co morbidity	No commodity	224	57.7%
	HTN	32	8.2%
	DM	30	7.7%
	ASTHMA	11	2.8%
	COPD	5	1.3%
	CKD	2	0.5%
	HIV	16	4.1%
	CANCER	6	1.5%
	Multiple comorbidity	**50**	**12.9%**
	CVD	5	1.3%
	HF	7	1.8 %
Severity	Mild	205	52.8%
	Sever	**114**	**29.4%**
	Critical	69	17.8%
Duration of symptom	Less than 2 weeks	317	81.7%
	2wks-4wks	50	12.9%
	>4 weeks	**21**	**5.4%**
Duration of hospital stay	< 1week	321	82.7%
	1week – 2 weeks	**52**	**13.4%**
	Above 2wks	15	3.9%
Medication used	MV	44	11.3%
	Cefepim	**58**	**14.9%**
	Vancomycin	139	35.8%
	ceftazidim	73	18.8%
	Meropenum	2	0.5%
	PPI	101	26%
	Ceftriaxone	316	26%
	Azithromycin	378	97.4%
	Antivirals	12	3.1%
Instigations	**Lymphopenia**	**295**	**76%**

Table 2: Baseline characteristics of covid 19 patients admitted at TibebeGhion Specialized Hospital, Northwest Ethiopia.

4.2 prevalence of acute kidney injury

In this study, the proportion of covid 19 patients who had acute kidney injury was found to be 25.3 % (n=98). (Figure1)

AKI prevalence

no
yes

98
25.26%

290
74.74%
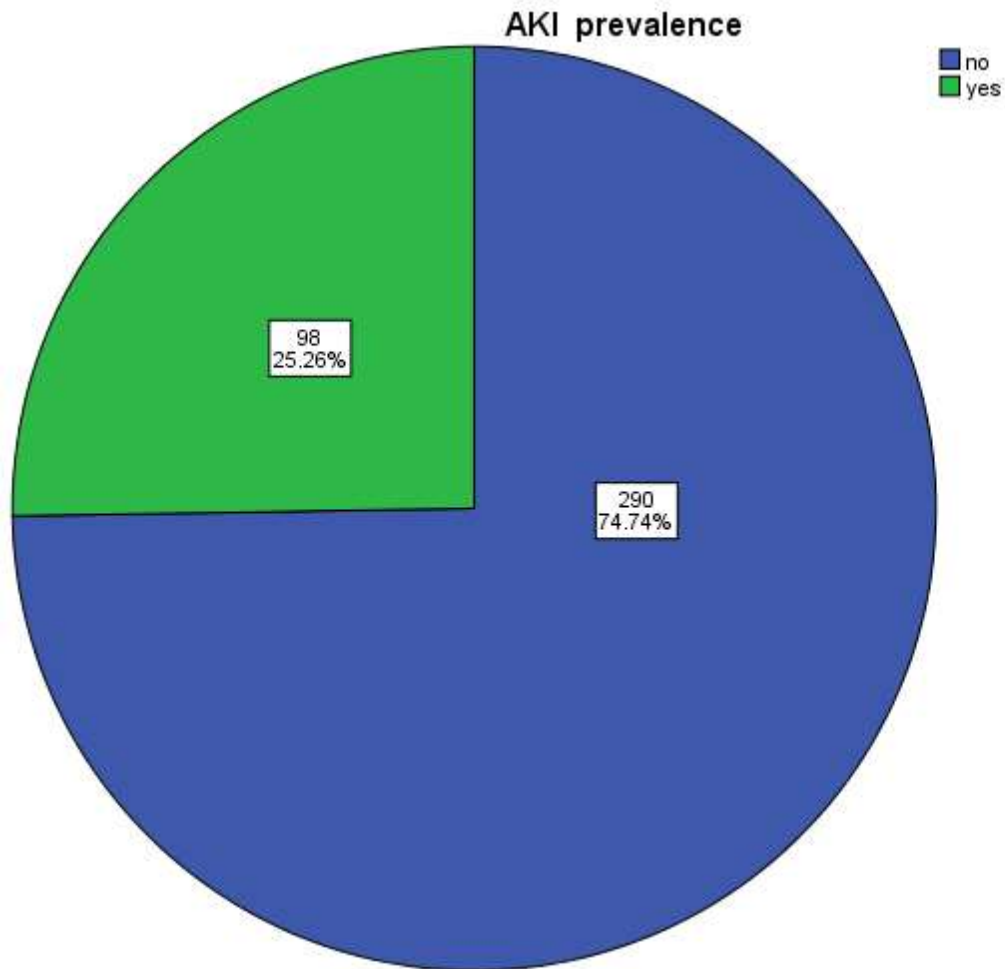
4.3 Factors associated with AKI

In binary logistic regression AKI was significantly associated with thosewho managed with vancomycin,ceftazidime, PPI or antiviral. Both sever and critical disease also associates significantly. From co morbidities those with prior history of DM, Asthma, HTN, COPD or multiple co morbidities associates. Additionally pulse rate above 100, RR above 24, fever anddyspneaassociates significantly with P- value less than 0.25.(Table 2)

In multivariate logistic regression analysis multiple co morbidity, with sever covid 19, more than our weeks symptom duration, cefepim and having Lymphopenia were significantly associated with AKI. (Table 2)

The result of this study showed that those who had multiple comorbidity were 4.036times high likely (AOR=4.036, 95%CI: (1.089,19.739)to have AKI compared to those who had no comorbidity. Compared to mild covid 19 cases those with sever covid 19 pneumonia were 10.217 % high likely (AOR=10.217, 95% CI: (1.027, 101.644) to have AKI, those had more than four weeks symptom duration had 7.313 % (AOR = 7.313, 95%CI :(1.516, 35.306) high likely to have AKI than those who had less than two weeks compliant.

Compared with who didn't use cefepim, those uses cefepim had 3.149% (AOR = 3.149%, 95%CI: (1.045,9.488) high likely and compared with those who have no Lymphopenia those with Lymphopenia were 5.089% times high likely (AOR = 5.089%, 95%CI: (1.352,19.146) to have AKI. patients who stayed in the hospital for one to two weeks had 3.647%   (AOR =3.647, 95%CI:(1.328,10.010) high likely to have AKI.(Table)

Characteristics		AKI	Non AKI	COR 95%CI	AOR 95%CI
PR	< 60	1 (1%)	0 (0%)	1	1
	60-99	60 (61.2%)	216 (74.5%)	3230949731.648	1
	>=100	37 (37.8%)	74 (25.5%)	0.556 (0.341, 0.904)*	0(0.00,0.00)
RR	<12	0	0		
	12-24	13 (13.3%)	81 (27.9%)	1	1
	>24	85 (86.7%)	209 (72.1%)	2.534 (1.339, 4.794)*	0.494 (0.126,1.935)
DYSNEA	yes	85 (86.5)	101 (34.8%)	12.23(6.505, 23.012)*	1.34(0.339, 5.33)
	No	13 (13.3%)	189 (65.2%)	1	1
TO	<36.5	18 (18.4%)	54 (18.6%)	1	1
	36.5-37.5	41 (41.8%)	139(47.9%)	0.829(0.433,1.588)	0.931 (0.308,2.816)
	>37.5	39 (39.8%)	97 (33.4%)	0.734 (0.441,1.221)	0.493 (0.479,4.622)
Saturation	<93	85 (86.7%)	92 (31.7%)	1	1
	93-100	13 (13.3%)	198 (68.3%)	14.072(7.465,26.525)*	0.75 (0.19, 9.6)
CO Morbidity	HTN	13 (13.3%)	19 (6.6%)	0.085(0.01,0.716)*	0.65(0.18,2.38)
	DM	10 (10.2%)	20 (6.9%)	0.538(0.237,1.431)*	2.7(0.49,15.05)
	COPD	3 (3.1%)	2 (0.7%)	0.127(0.064,0.25)*	6.4(0.307,135.78)
	Multiple comorbidity	27 (27.6%)	23 (7.9%)	0.341 (0.060,1.92)*	4.036(1.089,19.739)**

	Asthma	1(1%)	10 3.4%)	0.426 (0.166,1.091)	0.825 (0.058,11.750)
	CKD	2 (2%)	0 (0%)	1.278 (0.196,8.321)	258682659710367456.00
	HIV	6 (6.1%)	10 (3.4%)	0.852 (0.157,4.636)	1.668 (0.257,10.837)
	Cancer	3 (3.1%)	3 (1%)	1376145254.993	0.765(0.035,16.610)
	CVD	2 (2%)	3 (1%)	0.511 (0.161,1.622)	0.336 (0.018,6.215)
	HF	2 (2%)	5 (1.7%)	0.568 (0.087,3.698)	1.482 (0.059,37.325)
	No comorbidity	29	195	1	1
cefepime	Yes	39 (39.8%)	19 (6.6%)	9.428 (5.09,17.4)*	3.149(1.045,9.488)**
	No	59 (60.2%)	271 (93.4%)	1	1
Lymphopenea	Yes	90 (91.8%)	205 (70.7%)	0.214 (0.1, 0.46)*	5.08 (1.35,19.14)**
	NO	8 (8.2%)	85 (29.3%)	1	1
Hospital stay	< 1 week	53 (54.1%)	268 (92.4%)	1	1
	1 – 2 wks	35 (35.7%)	17 (5.9%)	10.411 (5.435,19.94)*	3.6(1.328,10.010)**
	>=2wks	10 (10.2%)	5 (1.7%)	10.1(3.3, 30.78)*	9.574 (1.323, 69.311)**

Severity	mild	7 (7.1%)	198 (68.3%)	1	1
	Sever	53 (54.1%)	61 (21.1%)	24.576 (10.6 ,56.86)*	10.217(1.027,101.644)*
	critical	38 (38.8%)	31 (10.7%)	34.6(14.2 ,84.48)*	6.7 (0.65, 69.37)
Duration of symptom	< 2 wks	57 (58.2%)	260 (89.7%)	1	1
	2 -4 wks	27 (27.6%)	23 (7.9%)	5.355 (2.864,10.011)*	1.558 (0.069,35.248)
	>4wks	14 (14.3%)	7(2.4%)	9.12 (3.5, 23.6)*	7.317(1.516,35.306)**
antiviral	yes	6 (6.1%)	6 (2.1%)	3.087(0.972, 9.805)*	0.81 (0.12, 5.35)
	no	92(93.9%)	284(97.9%)	1	1
PPI	Yes	55 (56.1%)	46 (15.9%)	6.785 (4.081, 11.280)*	1.7 (0.688, 4.45)
	No	43 (43.9%)	244 (84.1%)	1	1
ceftazidim	Yes	38 (38.8%)	35 (12.1%)	4.596(2.682, 7.875)*	1.13(0.8, 1.13)
	No	60 (61.2%)	255 (87.9%)	1	1
vancomycin	Yes	75 (76.5%)	64 (22.1%)	11.515(6.688, 19.827)*	2.25(0.16, 2.25)
	No	23 (23.5%)	226 (77.9%)	1	1
MV use	Yes	24 (24.5%)	20 (6.9%)	4.378(2.293, 8.360)*	0.7 (0.19, 2.75)
	No	74 (75.5%)	270 (93.1%)	1	1

Table 3: Factors associated with AKI in bivariate and multivariate regression analysis among patients with covid-19, at TibebeGhion Specialized Hospital, Northwest Ethiopia

Note: ∗ significant variables at P value <0.25 in the bivariate analysis, ∗∗ statistically significant variables at P value <0.05 in the multivariable analysis, COR: crude odds ratio; AOR, adjusted odds ratio; 1.00 _ reference category; Wks, weeks

5. Discussion

The results of the study revealed that the prevalenceof AKI in COVID-19 patients admitted to TGSH is about 25.3%, 95% CI (21.1, 29.6) and confirms the high prevalence of AKI in severely ill COVID-19 patients, patients with multiple co morbidities, patients with long duration of symptoms, Lymphopenia, use cefepim and those stayed for one to two weeks in the hospital.

Our findings are significantly higher thanthe studies done in SARS-CoV-1in which the prevalence of AKI was as low as 6%(10)(11)). These could be due to difference in tropism to kidney.SARS-CoV-2 appeared to have a higher frequency rate of AKI. The reason could be during a previous SARS outbreak in 2003, a study of postmortem kidney biopsies examined using electron transmission microcopy, failed to detect any viral particles in kidney tissues.(4) This finding supports the theory that most of the kidney pathogenesis in the earlier SARS outbreak was in the context of multi organ failure.But the direct cytopathic effect of COVID-19 on kidneys is now more evident, as it has been shown that there is overexpression of both ACE2 receptors and a cleavage spike protein in podocytes and proximal tubular cells.(17) This experimental evidence is of paramount importance and can explain proteinuria in patients with COVID-19. Interestingly, the latter experiment reports variable expression of cleaved S protein such that there is low expression in the Chinese race as compared to Caucasians. Important pathological evidence was reported by Diao et al. The pathology team managed to confirm the visualization of the SARS-CoV-2 viral particles in the renal tubular cells of postmortem kidney biopsies. (18)

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This result is higher than the studies conducted in Egypt (15%) (30), andstudy done in china (5.3%)(15).As compared to the study done in Egypt; we included only hospitalized covid 19 patients but they included home isolated and hospital admitted cases.Our study primarily focus on AKI prevalence compared to Chinese study whichprimary focus on the outcome of hospitalized Covid 19 patients. Additionally variance between demographic features, study area, study period and sample size of the study's populationsmay partially explained the occurrence of AKI.

Our result is lower than the studies conducted inSouth Africa (33.9%)(35), Eka kotebe (60.7%) (36), Wuhan, china (50%) (14). the possible reason might be the difference in severity of symptoms in the included patients. Our study mainly contains mild cases (52.8%) while the study in Eka kotebe was an ICU based cohort which include only sever and critically ill patients. The study done in South Africa also done in tertiary hospitals which are the main referral hospitals for COVID-19 as well as nephrology services.
Here we had avariety of referred cases from mild to critical cases, especially on the start of COVID 19 pandemic which our hospital was the main isolation centers for COVID 19 patients from different areas. The Chinese study was also on non-survivor.

The result is; however, more or less comparable to a study done ion UK in which AKI developed in 304 (26.2%) (37) COVID-19–positive patients (COVID-19 AKI) and 420 (12.4%) COVID-19–negative patients (AKI controls). The reason may be because of more or less similarity in severity of patient condition included with all mild, sever and critical one are included in both study.

We also confirmed that multiple comorbidities, severillness, usecefepim,longer duration of symptoms, one to two weeks and above two weeks hospital stay and having Lymphopeniawerefactors associated with AKI.

Prescience of prior multiple co morbidity was significantly associated with development of AKI. The result of this study showed that those who had multiple comorbidity were 4.036 times high likely (AOR=4.036, 95%CI: (1.089,19.739) to have AKI compared to those who had no co morbidity. The result is comparable to the study done in Eka kotebe, Addis Ababa (36). In our study the most common combined comorbidities are HTN and DM. Diabetic mellitus is the leading cause of renal failure worldwide. About 45% of patient need renal replacement therapy in USA are due to diabetic nephropathy. Diabetic nephropathy occurs in about 40% of both type one and two DM. It causes glomerular basement membrane thickening and increased GFR which progresses to proteinuria (39).Hypertension by itself causes arteriolar nephrosclerosis which causes progressive kidney damage (39).This finding was consistent with the study performedEka Kotebe(36). So that we have to follow strictly for the development of AKI in such patients.

Those who has above four weeks compliant associated with the development of AKI. This warrants further investigation with the relation of long covid and the kidney. Long term follow up of covid 19 patients should be considered for possible long term impact of Covid 19 on kidney. In a previous single case report, collapsing variant of focal segmental glomerulosclerosis was diagnosed in renal biopsy of African-American woman, who tested positive to COVID-19 (24), considering these we may consider renal biopsy for patients who come with AKI in a long run.

Those with Lymphopenia are at increased risk of AKI. These result is consistent with Wuhan, china study (26) and Eka kotebe study (36). These could be due to Lymphopenia as a marker of severity of inflammation. A study done in Wuhan, China (38) showed that the leukocytes, neutrophils, infection biomarkers [such as C-reactive protein (CRP), procalcitonin (PCT)and ferritin] and the concentrations of cytokines [interleukin (IL)-2R, IL-6,IL-8, IL-10 and tumor necrosis factor (TNF)-α] were significantly increased,while lymphocytes were significantly decreased with increased severity of illness. Upon recovery, lymphocyte counts return to normal in almost all cases. As we will discuss later sever covid infection associate significantly with development of AKI in our study.

In our study Patients with severe covid 19 pneumonia associate significantly with AKI development. These is consistent with the study done in Wuhan, china (26). As severity increase the amount of cytokine release will increase. This involves the secretion of large amounts of pro-inflammatory cytokines and chemokines including IL8, IL 6, IL9, IL10, and many others. Pathogenic mechanisms in the kidneys are not fully elucidated, but the

suggested mechanisms are through attachment of the virus to ACE2 receptors. Excess secretion of cytokines leads to multi organ failure in a percentage of patients, including acute kidney injury (AKI), through tissue hypoxia (10).

In our study critical covid cases which was significantly associate for the study in Eka kotebe, Addis Ababa (36),United Kingdom (35), Wuhan china (26), Egypt (8) didn't associate to AKI development. These could be due to other variable Effect.

Use of cefepim was another variable that associate significantly for the development of AKI.From 58 patients who took cefepim 48 of them had at least one comorbidity and 18 had more than one comorbidity. Cefepime is a fourth-generation cephalosporin active against both gram-positive and gram-negative organisms. Approximately 85% of the drug is excreted unchanged by the kidneys.Renal clearance of cefepime diminishes proportionately with a decrease in the glomerular filtration ratio (GFR). Cephalosporins cause allergic interstitial nephritis(39).The reason cefepim given after trial of first line antibiotics in relatively sever disease could be additional reason.

Those who stayed above two weeks hospital in the hospital significantly associate for the occurrences of AKI. Even patients in the hospital between one and two weeks are significant though its significance is lower than those who stayed above two weeks. So that we can say that the more who stayed in the hospital, the more risky to develop AKI. These could be due to exposure to multiple nephrotoxic medications, superimposed hospital acquired infections ,dehydration and probably the prolonged effect of Covid 19 itself.

6. Conclusions

Acute kidney injury is a common feature in severely ill patients with COVID-19 pneumonia presenting with acute hypoxic respiratory failure. It is more common in patients with multiple co morbidity, long covid patients, those with Lymphopenia as well as using cefepim and stayed more in the hospital. These data indicate that patients with COVID-19 should be monitored for the development of AKI and measures taken to prevent this.

7. Limitations

Our study has several limitations. Firstly, this is confined to patients admitted in a single center that may not be representative of all COVID-19 patients. Secondly, due to the retrospective cross sectional design, some laboratory results were not available for all patients. Third, we did not perform additional renal specific urine biological investigations (urinalysis and urine microscopy) to characterize the AKI, which limits our ability to evaluate the mechanism of renal injury. Lastly true baseline SCr results were not available in most of the patients and we used the SCr results on admission to hospital.

8. Recommendation

- ✓ Clinicians should be cognizant of this higher incidence of AKI in severe covid-19 patients.
- ✓ Efforts should be made to alleviate the risk including, unnecessary use of nephrotoxic drugs and prolonged hospital stay that could contribute significantly to AKI in covid-19 patients.
- ✓ Given the high incidence of AKI in long covid 19 patients, it's worth monitoring the renal status of patients after hospital discharge to further understand the natural course and help in the individual patient's management. We strongly recommend post discharge follow up of those patients.

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Declaration

I, the undersigned, declared that this is my original work, has never been presented in this university, and that all the resources and materials used for the research, have been fully acknowledged.

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Cheek list

Sociodemographic status		
Card number		
Age		
Sex		
male		
Female		
Occupation		
Farmer		
Government employee		
Private organization		
Others (specify)		
Residency		
Urban		
Rural		
Preexisting comorbidities		
No comorbidity	ASTHMA	cardiovascular disease
HTN	COPD	CLD
DM	Cerebrovascular disease	CKD
Cancer	others (specify)	Multiple
Symptoms		
Fever		
Cough		
Dyspnea		
Myalgia		
Fatigue		
Others (specify)		
Sign		
BP		
PR		
RR		
To		
So2		
Body swelling		
Basal crepitation		
Other (specify)		
Investigations		
Leukopenia < 4000 cells/mm3		
Normal WBC (4000-10,000cells/mm3)		
Leukocytosis (>10,000cells/mm3)		
Lymphopenia (<1000cells/mm3)		
Normal lymphocyte (1000-4000cells/mm3)		
Normal LFT		
Deranged LFT		
Normal RFT		

Deranged RFT	
CREATININE	
Initial creatinine	
Progressive creatinine	
Disease severity	
Mild	
Sever	
Critical	
Duration of symptoms	
Less than one weak	
1 to 2 weeks	
2 weeks to one month	
Above 1 month	
Duration of hospital stay	
Less than 1 weak	
1 to 2 weeks	
Above 2 weeks	
Given treatment	
Use of mechanical ventilator	
oxygen	
ceftriaxone	
azithromycin	
vancomycin	
ceftazidime	
Meropenum	
steroid	
heparins	
NSAIDS	
PPIs	
Antiviral	
Opioids	
Antivirals	
Others (specify)	