International Surgery Journal
Sharma PK et al. Int Surg J. 2020 Jul;7(7):2332-2336
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: http://dx.doi.org/10.18203/2349-2902.isj20202845

Original Research Article

Renal profile of COVID-19 infected patients admitted in a tertiary care hospital in Western Rajasthan

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Received: 11 May 2020
Revised: 15 June 2020
Accepted: 16 June 2020

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ABSTRACT

Background: A recent cluster of pneumonia cases all over the world including India, is caused by a novel beta coronavirus, the 2019 novel coronavirus (2019-nCoV). The objective of this study was to observe the effect on renal profiles at admission of these COVID-19 infected patients.
Methods: All the patients were admitted to Dr. S. N. Medical College and Hospital, Jodhpur, a tertiary care hospital of Western Rajasthan and were tested for COVID-19 by real time reverse transcription polymerase chain reaction assay of 2019-nCoVRNA. All data in relation to patients including blood renal profile were collected from data collection form from electronic medical records and history given by COVID-19 infected patients admitted.
Results: Out of total 300 COVID-19 patients, majority of patient were male i.e. 210 (70%) and majority of the patients 70% were below 60 years of age. The underlying diseases were present in 98 (32.67%) patients. Cough was the most common symptom (80%) followed by fever (72%) in out of 180 (60%) symptomatic patients. 64 (21.3%) patients had deranged renal function tests with abnormal blood urea nitrogen and serum creatinine. Overall, 56 patients (18.6%) without chronic kidney disease showed mild increase of BUN or serum creatinine. 35 patients (11.6%) had high blood urea nitrogen and 18 patients (6%) had raised creatinine.
Conclusions: In setting of COVID-19 infected patient's presentation, renal profile remained essentially normal with deranged RFT seen in patients with comorbidities. Nevertheless, the renal function of patients with COVID-19 needs to be monitored regularly, especially in patients with elevated plasma creatinine.

Keywords: Acute kidney injury, Chronic kidney disease, COVID-19, Renal profile

INTRODUCTION

A new respiratory tract infecting agent emerged in Wuhan city of China, known as the coronavirus in December, 2019 which was named COVID-19. 2019-nCoV is a form of beta-coronaviruses indicated from full genome sequencing and phylogenic analysis and it is associated with human severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS).[1] The 2019-nCoV has close similarity to bat coronaviruses, and it has been postulated that bats are the primary source. While the origin of the 2019-nCoV is still being investigated, current evidence suggests spread to humans occurred via transmission from wild animals illegally sold in the Huanan Seafood Wholesale Market.[2] It spread rapidly through China infecting more than 85,000 people and soon it engulfed whole world with massive loss of life. It has infected large number of population in India now which is the second most populous country of the world. As of now more than 30,00,000 people have been infected and 2,00,000 people have succumbed to the illness across the globe.

The WHO declared COVID-19 a global pandemic on 11th March 2020. It is disease in which severity ranges from asymptomatic or mild to severe and with clinically evident infection develop severe disease. True overall mortality rate is uncertain, as the total number of cases (including undiagnosed persons with milder illness) is unknown.[1]

Acute kidney injury (AKI) is a syndrome characterized by a rapid (hours to days) deterioration of kidney function. It is often diagnosed in the context of other acute illnesses. The clinical consequences of AKI include the accumulation of waste products, electrolytes, and fluid, but also less obvious effects, including reduced immunity and dysfunction of non-renal organs (organ cross-talk).[3] The impact and prognosis of AKI vary considerably depending on the severity, clinical setting, comorbid factors, and also geographical location. There is increasing evidence that AKI is associated with serious short- and long-term complications, in particular increased mortality and morbidity, the development of chronic kidney disease (CKD), and high financial healthcare costs. As such, AKI is now recognized as a major public health problem.[4,5]

AKI is defined as a rapid reduction in renal function characterized by progressive azotemia [best measured clinically by serum creatinine and blood urea nitrogen (BUN)], which may or may not be accompanied by oliguria. This abrupt decline in renal function occurs during the course of hours to days and results in the accumulation of byproducts of metabolism and the dysregulation of volume, acid/base, and electrolyte homeostasis. Blood urea nitrogen (BUN) is a laboratory test that measures the amount of urea nitrogen found in blood. BUN is an indication of renal (kidney) health. The normal range is 2.1-7.1 mmol/l or 6-20 mg/dl.[6] The main causes of an increase in BUN are: high protein diet, decrease in glomerular filtration rate (GFR) (suggestive of kidney failure), decrease in blood volume (hypovolemia), congestive heart failure, gastrointestinal hemorrhage, fever, rapid cell destruction from infections, athletic activity, excessive muscle breakdown, and increased catabolism.[1]

The involvement of COVID-19 with lungs is well established but effect on kidney function yet to be explored. 6.7% of patients with SARS had developed acute kidney injury (AKI, and the mortality of those with AKI was 91.7%.[4] Thus, on literature search one study has reported the effect of COVID-19 on kidney.[7] This study was planned to observe the effect on renal profiles at the time of admission of these Covid-19 infected patients at tertiary care teaching hospital.

METHODS

This cross sectional observational study was conducted after approval from institutional ethics committee. All the patients during the study period were enrolled; means consecutive sampling was done. All consecutive patients with confirmed COVID-19 infection admitted to Dr. S. N. Medical College and hospital, Jodhpur from 22 March 2020 to 30 April 2020, were included in the study. Consent was obtained from all the patients. Patients who did not give the consent were excluded out from the study. The medical records of patients were analysed and reviewed. Epidemiological, clinical, laboratory, and radiological characteristics data were obtained with data collection forms from electronic medical records and history given by patients. Information recorded included demographic data, medical history, exposure history, underlying co morbidities like diabetes, hypertension cardiovascular disease, and chronic kidney disease. At the time of admission laboratory blood findings including renal function tests were noted.

Throat swab samples were collected for extracting 2019-nCoV RNA from patients suspected of having 2019-nCoV infection and were placed into a collection tube containing virus transport medium (VTM) for extraction of total RNA and were later centrifuged. The suspension was used for real-time reverse transcription polymerase chain reaction (RT-PCR) assay of 2019-nCoVRNA. This diagnostic criterion was based on the recommendation by the National Institute of Virology (Pune). Acute kidney injury was identified according to the Kidney Disease: Improving Global Outcomes definition.[8]

All data was expressed in number or percentage. Data was analyzed using Microsoft excel.

RESULTS

Total 300 patients were enrolled and identified as having laboratory-confirmed 2019-nCoV infection. The median age for all patients was 41.7 years ranging from 6 to 80 years and the majority 210 (70%) of them were below 60 years of age. 210 (70%) of patients were male while 90 (30%) patients were female (Table 1). Four patients (19.04%) out of 21 were healthcare workers.

The underlying diseases were present in 98 (32.67%) patients, including diabetes 40 (13.3%), hypertension 30 (10%), cardiovascular disease 20 (6.7%), chronic kidney disease 8 (2.7%). Out of 300 patients admitted, majority of patients 260 (86.7%) were from urban area and rest 40 (13.33%) were from rural areas. Out of 260 urban patients 30 patients had travel history while 70 had contact history and while out of 40 rural patients 4 had travel history while 10 had contact history.

Approximately 40% patients were completely asymptomatic and of those who were symptomatic, cough was the most common symptom (80%) followed by fever (72%), myalgia (60%), headache (25%), dyspnea (10%), diarrhea (8.3%) and vomiting (7.7%) (Table 1).

Table 1: Demographic and clinical characteristics of all COVID-19 patients (n=300).

Characteristics	N (%)
Age (years)	41.7 (range 6 to 80)
Male	210 (70)
<60 years	155 (51.67)
>60 years	50 (16.67)
Female	90 (30)
<60 years	55 (18.33)
>60 years	40 (13.33)
Urban	260 (86.7)
Rural	40 (13.33)
Comorbidites	98 (32.67)
Diabetes	40 (13.3)
Hypertension	30 (10)
Cardiovascular diesase	20 (6.7)
Chronic kidney disease	8 (2.7)
Clinical features	
Asymptomatic patients	120 (40)
Symptomatic patients	180 (60)
Cough	144 (80)
Fever	129 (72)
Myalgia	108 (60)
Headache	45 (25)
Dyspnoea	18 (10)
Diarrhoea	15 (8.3)
Vomiting	14 (7.7)

Table 2: Laboratory data of all the COVID-19 patients (n=300).

Investigations	No. (%)
Hemoglobin	
<10 gm%	19 (6.3)
>10 gm%	281 (93.7)
Leucocytosis	40 (13.3)
Neutroopenia	48 (19.04)
Eosinopenia	15 (5)
Lymphopenia	160 (53.3)
Thrombocytopenia	65 (21.6)
Deranged LFT	58 (19.3)
Hypoalbuminemia	24 (8)
Hypertrigylceridemia	42 (14)

19 (6.3%) patients had hemoglobin below 10 gm% while rest all patients had above 10 gm%. 160 patients (53.3%) had lymphopenia. 48 patients (19.04%) had neutropenia and 15 patients (5%) had eosinopenia. 40 patients (13.3%) had leucocytosis. 65 patients (21.6%) presented with thrombocytopenia. 58 (19.3%) patients had derangement in the liver function tests including 24 (8%) patients had hypoalbuminemia. 42 (14%) patients had a deranged lipid profile in the form of hyper-trigylceridemia (Table 2).

64 (21.3%) patients had deranged renal function tests with abnormal blood urea nitrogen (BUN) and creatinine. 35 patients (11.6%) had raised blood urea nitrogen, out of which 15 patients had vomiting and diarrhea and 7 patients were known case of chronic kidney disease. 18 patients (6%) had raised creatinine of which 8 patients were known case of chronic kidney disease and 6 patients were having associated comorbidities like diabetes, hypertension (Table 1). Overall 56 patients (18.6%) without CKD showed mild increase of BUN or Serum creatinine (Table 3).

Table 3: Renal status in all COVID-19 patients (n=300).

Characteristics	N (%)
Patients with normal renal function test	236 (78.6)
Patients with abnormal renal function test	64 (21.3)
Patients with CKD	8 (2.7)
Patients without CKD	56 (18.6)
Increased BUN (>20 mg/dl)	35 (11.6)
Increased creatinine (>1.2 mg/dl)	18 (6)

DISCUSSION

This descriptive study included 300 COVID-19 patients diagnosed by real-time reverse transcription polymerase chain reaction (RT-PCR). Human coronavirus is one of the main pathogens of respiratory infection. The two highly pathogenic viruses, SARS-CoV and MERS-CoV, cause severe respiratory syndrome in humans and four other human coronaviruses (HCoV-OC43, HCoV-229E, HCoV-NL63, HCoV-HKU1) induce mild upper respiratory disease. The major SARS-CoV outbreak occurred during 2002-03 and spread to 29 countries globally.[9,10] MERS-CoV emerged in Middle Eastern countries in 2012.[11,12] The sequence of 2019-nCoV is relatively different from the six other coronavirus subtypes but classified as beta coronavirus.

In present study, the median age was being 41.7 years, which was a decade younger than that reported by Wang et al (56.0 years), Chen et al (55.5 years) and closest to that in Huang et al (49.0 years).[2,13,14] Most of the patients having COVID-19 were male (70%) which was similar to that reported by Huang et al (73%) and Chen et al which showed 73.0% male predominance but higher than that reported by Wang et al (54.3%).[2,13,14] This male predominance may be due to increased foreign travel by males for occupational or educational purposes. The reduced susceptibility of females to viral infections could be attributed to the protection from X chromosome and sex hormones, which play an important role in innate and adaptive immunity.[15]

The underlying diseases were present in 32.67% patients which were in contrast to that reported by Chen et al

where 50% patients had underlying comorbidities.[2] In present study, cough was the most common symptom present in our patients (80%) followed by fever (72%) which was in contrast to that reported in Wang et al and Huang et al where fever was the most common symptom found (91.7%) and Guan et al (87.9%).[13,14,16] 40% patients were asymptomatic at the time of presentation which was in contrast to that reported in Wang et al and Huang et al where fever was the most common symptom found (91.7%).[9,10]

53.3% patients in present study presented with lymphopenia which was lesser than reported by Zhu et al (75.4%).[1] Some patients also presented with thrombocytopenia. This result suggests that 2019-nCoV might mainly act on lymphocytes, especially T lymphocytes, as does SARS-CoV. Virus particles spread through the respiratory mucosa and infect other cells, induce a cytokine storm in the body, generate a series of immune responses, and cause changes in peripheral white blood cells and immune cells such as lymphocytes. 53.3% patients presented with lymphopenia indicating, that occurrence of lymphopenia can be used as a marker of prognosis.

It should be noted that ACE-2 protein has been proved to have an abundant expression in many kinds of cells, such as intestinal epithelial cells, renal tubular epithelial cells, alveolar epithelial cells, heart, artery smooth muscle cells, and gastrointestinal system. Therefore, it is reasonable to speculate that SARS-CoV-2 may invade the lung, upper respiratory tract, ileum, heart, and kidney, which may lead to dyspnea, diarrhea, acute heart injury, and AKI, especially in the case of viremia.[17]

Although 18.6% without CKD showed mild increase of BUN or Serum creatinine after infection with the virus, all these patients did not meet the diagnostic criteria of AKI, this was in contrast to the study of Wang et al where 10% patients without CKD had mild increase of BUN or serum creatinine.[7] The temporary abnormal renal function was probably supposed as secondary injury due to vomiting, diarrhoea or associated comorbidities in these patients.

Limitations

Long term studies are required with follow up of infected patients including urine routine microscopic study, ultrasonography of kidney, ureter and bladder with pelvis, to establish the changes in renal profile of COVID-19 infected patients. The serum of patients was not obtained to evaluate the viremia. The viral load is a potentially useful marker associated with disease severity of coronavirus infection, and this should be determined.

CONCLUSION

This study showed variable range of presentation. Asymptomatic patients during the course of disease despite being COVID-19 positive pose a great epidemiological risk to the society as they can spread the infection unrestrictedly. In setting of Covid-19 infected patient's presentation, renal profile remained essentially normal with deranged RFT seen in patients with comorbidities and acute illness like vomiting and diarrhea. Nevertheless, the renal function of patients with COVID-19 needs to be monitored regularly, especially in patients with elevated plasma creatinine.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Sharma PK, Aggarwal V, Sapariya N, Baweja S, Nagori R, Chabbra MK. Renal profile of COVID-19 infected patients admitted in a tertiary care hospital in Western Rajasthan. Int Surg J 2020;7:2332-6.