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**Original Research Article**

# Renal involvement and outcome in children with COVID-19 infection

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## ABSTRACT

**Background:** COVID-19 infection is known for multi-organ involvement of which Kidney injury predominates. AKI is associated with raised mortality in intensive care setting worldwide. Due to scarcity of data of renal involvement in pediatric COVID-19 population, we aimed to study its incidence in pediatric population requiring admission for COVID 19.
**Methods:** This was a single centred observational study where all the indoored COVID-19 infected children were looked for any form of renal involvement. We divided the study population into subgroups of those having any underlying renal ailment and the other without any underlying illness.
**Results:** 342 patients with COVID-19 infection were enrolled. Renal involvement was seen in 38 (11%) subjects. Of them 31 subjects developed AKI. Maximum number of population had severe/ stage III AKI (as per KDIGO criteria). Among the AKI subgroup (N=31), 41.9% (n= 13) required acute RRT of which 6 subjects were successfully discharged. 20/31 (64.5%) subjects of AKI subgroup were discharged with complete renal recovery and 11/31 (35.4%) had expired. Among the 7 subjects who did not develop AKI, all were discharged successfully.
**Conclusions:** AKI occurred in significant proportion of study population with SARS-Co-V2 infection. AKI subgroup reports higher mortality rate which is proportional to the stage of AKI.

**Keywords:** COVID-19, Pediatric, Renal involvement, Acute kidney injury

## INTRODUCTION

In December 2019, Wuhan Hubei Province, reported cases with pneumonia which was later identified as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). This illness caused by the novel coronavirus disease 2019 (COVID-19) was declared as a pandemic by the WHO on March 11, 2020. The disease had presentations from self-limiting flu like illness, acute pneumonia to sepsis leading to life-threatening complications, including acute respiratory distress syndrome (ARDS), acute cardiac injury, acute kidney injury (AKI), and septic shock.[1] As compared to adult counterparts, the incidence of COVID-19 in the pediatric cases is from 0.8 to 2.7% with 0.58-9.7% of patients requiring intensive care.[2] With the widening of research, data from worldwide showed significant involvement of renal functions in patients admitted with COVID-19.

Various studies from the foreign have showed the incidence of renal dysfunction from 0.5 to 27% among hospitalized patients.[1,3] AKI is more common among patients with more severe disease and is considered a negative prognostic factor for survival.[4] There is scarcity of data in the Indian scenario, that provides the incidence of renal involvement in pediatric COVID-19 subjects.

The aim of the study was to determine the incidence of renal involvement in pediatric COVID-19 subjects at our centre, a dedicated COVID-19 care centre.

## METHODS

This study was a single centred study done at a Bai Jerbai Wadia Hospital in Mumbai, India which is a pediatric dedicated tertiary care centre in Mumbai, India. It was a retrospective observational study done over a period of 12

months from August 2020 to July 2021. This study was approved by the Institutional Ethics Committee of the centre.

***Recruitment procedure***

*Inclusion criteria*

All pediatric patients with SARS CoV-2 positive RT-PCR, serology, or antigen test admitted to Bai Jerbai Wadia Hospital, Mumbai were included. Pediatric patients with underlying comorbid kidney disease (e.g. nephrotic syndrome, chronic kidney disease, tubular disorder or urological disorder) and who tested SARS CoV-2 by positive RT-PCR, serology, or antigen test were included in the study.

*Exclusion criteria*

Patients who had history of COVID-19 exposure but were negative by RT PCR, serology or Antigen test or children with radiological evidence (by HRCT or Chest X ray) suggestive of COVID-19 but negative by RT PCR, serology or antigen test were excluded of the study to reduce the bias. Patients who were being treated as per COVID treatment protocol but were negative by RT PCR, serology or antigen test were excluded. Patients who were diagnosed cases of CKD but having COVID-19 infection were excluded.

***Methodology***

Retrospective medical record review of all children admitted to the hospital were accessed. All children with RT-PCR positive for SARS-CoV-2, positive serology (IgG or IgM antibodies by enzyme chemiluminescence immunoassay method) or antigen test (by rapid chromatographic immunoassay) were included in the study. As per institutional protocol, every child requiring admission was tested by RT-PCR for SARS-CoV-2 from an Indian Council of Medical Research (ICMR) recognized laboratory.

Detailed review of previous history and pre-existing co morbidities were noted. The children were grouped into 2 - as no underlying renal comorbidity and another as children with underlying renal comorbidity. Details of age, sex, height, weight, date of admission and date of discharge/death, their clinical summary, presenting symptoms, onset of symptoms, examination findings such as- weight, height, Body surface area, Blood pressure, hydration status, contact history with COVID case, any underlying co-morbidity or drug history was obtained from the medical records during their hospital admission.

Records of children who already had underlying renal disorder and were eligible as per the inclusion criteria were reviewed in detail to obtain information about the exact underlying renal illness, any drugs history specific to their condition, any nephrotoxic agent exposure. The COVID-19 positivity was defined by either reverse transcriptase-polymerase chain reaction (RT-PCR) positive for SARS-CoV-2, positive serology (IgG/ IgM antibodies by enzyme chemiluminescence immunoassay method) or antigen test (by rapid chromatographic immunoassay)

Records of routine blood investigations were noted. Parameters related to renal involvement variable such as blood urea nitrogen, serum creatinine, serum electrolytes, s. albumin, serum cholesterol, serum complement 3 levels (in whichever case applicable), serum lactate dehydrogenase, serum uric acid, urine routine examination and microscopy, ultrasonography of the kidneys and urinary tract were noted. Treatment details and in cases where renal replacement therapy was administered, mode of RRT, number of sessions that were required and details of RRT were noted. Outcome was defined in terms of death or discharge. COVID specific inflammatory markers were recorded. Stages of AKI were defined as per KDIGO criteria. Baseline creatinine was defined as the last creatinine within the previous 6 months prior to the admission. For those patients admitted for the first time, an average GFR according to age, sex, and height of the child was calculated.

**Table 1: Definitions.**

| Stage of AKI | Defined as |
|---|---|
| 1 | Increase ≥0.3 mg/dl within 48 h or ≥1.5- to 2-fold from baseline |
| 2 | 2.0-2.9 times from baseline |
| 3 | 3.0 times from baseline or increase in serum creatinine to ≥4.0 mg/dl or initiation of renal replacement therapy or, in patients <18 years, decrease in eGFR to <35 ml/min per 1.73 $m^2$ |

## RESULTS

Study population included 342 patients who were admitted in hospital and diagnosed with COVID infection. Of the study population, maximum children (56%) were in the age group of 1-5 years followed by 23% who were in between 5 to 10 years of age.

Renal involvement was seen in 11% (N=38) of the total population. 81.5% (n=31) of the 38 subjects had renal involvement in form of AKI defined as the criteria mentioned earlier. Among them, the Male: Female Ratio was 1.38:1. Of the subjects that developed AKI, 22 (70.95%) were positive for COVID antibodies classifying them in Multisystem Inflammatory Syndrome in children (MIS-C) while 9 subjects (29%) were positive for COVID-19 infection by RTPCR method (acute COVID).

23 subjects (74.19%) had stage 3 AKI as per the KDIGO criteria while 6 (19%) and 2 (7%) had developed stage 2

and stage 1 AKI respectively. The mean duration of hospital stay for the cases that had AKI (n=31) was 23 days. 17 subjects who had AKI (54.83%) had edema, 20 (64.51%) had oliguria, 11 subjects (35.4%) had hypertension on presentation. 14 subjects (45.16%), 8 subjects (25.8%) and 9 subjects (29.03%) had nephrotic range proteinuria, sub- nephrotic range proteinuria and no proteinuria respectively. 12 cases (38.7%) of these had transient proteinuria. 8 children (25.8%) had microscopic hematuria while 4 (12.9%) had gross hematuria on presentation. Raised LDH was seen in 29 (93.5%) subjects, while hyperferritinemia, raised IL 6 levels, raised D-Dimer, hyperuricemia were seen in 28 (90.3%), 26 (83.87%), 30 (96.77%) and 19 subjects (61.23%) respectively. Of the subjects who developed AKI (n=31), 13 (41.9%) cases required intravenous immunoglobulin (IVIg), 18 (58.06%) required steroids, 5 (16.12%) required anti-coagulation and 19 subjects (61.2%) required vasoactive drugs for treatment of shock. 20 cases of AKI subgroup (64.51%) were discharged and sent home while 11 (35.4%) of these expired. The mortality rate was higher in patients with severe stages of AKI.

Renal recovery was seen in all (n=20, 100%) of the cases who were discharged from the hospital in the AKI subgroup. Renal replacement therapy (RRT) was instituted in 13 subjects of AKI subgroup (41.93%). 2 subjects required conventional hemodialysis, while Sustained low efficiency dialysis (SLED) was initiated in 2 subjects. 9 of 13 subjects were managed with acute peritoneal dialysis. Of these 13 subjects, 6 (46.15%) children were discharged while 7 children (53.84%) had expired during the study period. 20 of 31 subjects (64.51%) of AKI subgroup were discharged and had complete renal recovery on discharge measured with adequate urine output and return of renal markers to baseline. Of the 31 patients who had AKI, 80.64% (N=25) did not have any other underlying renal disease or any previous history of nephrotoxic drugs exposure or any other ailment. Of the remaining 6 cases, 3 were already diagnosed with steroid sensitive nephrotic syndrome in remission. Renal biopsy was performed in 2 of these cases that showed focal segmental glomerulosclerosis (Figure 1). 1 of the child who was biopsied manifested steroid resistance in subsequent course.

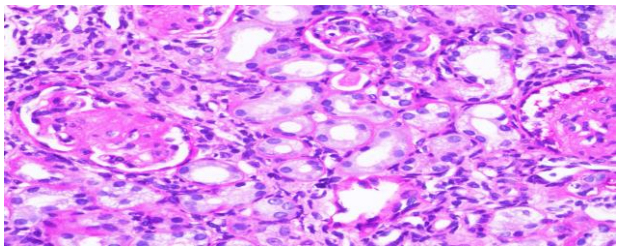

**Figure 1: Light microscopy image picture showing mesangial hypercellularity and focal glomerular sclerosis in 2 glomeruli in biopsy of a subject.**

The remaining 7 subjects did not have AKI but manifested renal involvement in form of nephrotic range proteinuria (42.85%) or sub-nephrotic range proteinuria (28%) or transient proteinuria (14%). Microscopic hematuria was seen in 2/7 subjects (28.5%). Of these 7 subjects, 71.4% (N= 5) were COVID PCR positive (acute COVID) while remaining 2 had MIS-C. 3/7 subjects were already diagnosed cases of steroid sensitive nephrotic syndrome in remission and had relapses with COVID infection. Raised LDH, hyperferritinemia, raised IL-6, raised D-dimer levels were seen in 7 (100%), 6/7 (85.7%), 4/7 (66.6%), 5 (71.4%) subjects respectively. The mean levels of LDH, ferritin, IL-6, D-dimer was lower as compared to AKI subgroup. None of them required inotropic drugs. Steroid was used in 6 of them while 1 required IVIg. 3 were started on anticoagulation with Low molecular weight heparin owing to raised d-dimer levels. The mean duration of stay in these 7 subjects was 18 days and all 7 were discharged (100%) successfully. Of the 38 enrolled subjects who had renal involvement, 27 (71%) were discharged from hospital and death occurred in 11 (29%) subjects. Among the AKI group, 20 (64.51%) were discharged successfully with complete renal recovery while mortality occurred in 11 subjects (35.45%). All of the 7 patients (100%) of the no AKI group were discharged from the hospital.

**Table 2: Clinical, epidemiological, biochemical, treatment and outcome of the subjects of the AKI and no AKI sub groups.**

| Clinical and biochemical features | Subjects with AKI | | No AKI | |
|---|---|---|---|---|
| | n=31 | % | n=7 | % |
| **No underlying renal comorbidity** | **25** | 80.64 | **3** | 42.8 |
| **Underlying renal comorbidity** | NS (3)<br>Misc 2<br>HUS 1 | | NS 3<br>Misc 1 | |
| Gender | | | | |
| Males | 18 | | 5 | |
| Females | 13 | | 2 | |
| M: F ratio | 1.38:1 | | 2.5 : 1 | |
| **COVID PCR** | 9 | 29.03 | 5 | 71.4 |
| **COVID antibodies RAT** | 22 | 70.95 | 2 | 28.57 |
| **Duration of stay (mean)** | 23 days | | 18 days | |

Continued.

| Clinical and biochemical features | Subjects with AKI | | No AKI | |
|---|---|---|---|---|
| | n=31 | % | n=7 | % |
| **Outcome** | | | | |
| **Discharged** | 20 | 64.5 | 7 | 100 |
| **Death** | 11 | 35.48 | | |
| **Stage of AKI** | | | | |
| Stage 1 | 2 | 6.4 | | |
| Stage 2 | 6 | 19.3 | | |
| Stage 3 | 23 | 74.19 | | |
| **Edema** | 17 | 54.83 | 6 | 85.7 |
| **Oliguria** | 20 | 64.51 | 5 | 71.4 |
| **Hypertension** | 11 | 35.4 | 4 | 57.6 |
| **Anemia** | 18 | 58 | 2 | 28.57 |
| **Thrombocytopenia** | 9 | 29.03 | 1 | 14.28 |
| **Proteinuria** | | | | |
| No proteinuria | 9 | 29.03 | 1 | 14.28 |
| Subnephrotic | 8 | 25.80 | 2 | 28.5 |
| Nephrotic | 14 | 45.16 | 3 | 42.85 |
| Transient | 12 | 38.7 | 1 | 14.2 |
| **Hematuria** | | | | |
| Microscopic | 8 | 25.8 | 2 | 28.5 |
| Gross | 4 | 12.9 | 0 | |
| **Dyselectrolytemia** | | | | |
| **Hyponatremia** | 12 | 38.7 | 1 | 14.28 |
| **Hypernatremia** | 3 | 9.6 | 0 | |
| **Hypokalemia** | 3 | 9.6 | 2 | 28.5 |
| **Hyperkalemia** | 5 | 16.12 | 0 | |
| **Acidosis** | 22 | 70.9 | 2 | 28.5 |
| **Raised LDH** | 29 | 93.5 | 7 | 100 |
| | Mean=642 | | Mean=353 | |
| **Hyperferritinemia** | 28 | 90.3 | 6 | 85.7 |
| | Mean=520 | | Mean=445 | |
| **Hyper IL-6** | | | | |
| Raised | 26 | 83.87 | 4 | 66.6 |
| Not tested | 5 | 16.12 | 1 | 14.2 |
| | Mean=78 | | Mean=35 | |
| **Raised D-dimer** | 30<br>Mean=2.9 | 96.77 | 5<br>Mean=1.2 | 71.4 |
| **Requirement of IvIg** | 13 | 41.9 | 1 | 14.28 |
| **Steroid use** | 18 | 58.06 | 6 | 85.71 |
| **Use of anticoagulation** | 5 | 16.12 | 3 | 42.85 |
| **Use of inotropic agents** | 19 | 61.2 | 0 | 0 |

**Table 3: Details of the subjects that were initiated on RRT in the AKI sub-group.**

| Variables | Patients with AKI (N=31) | % |
|---|---|---|
| **RRT** | **13** | **41.9** |
| HD | 2 | |
| SLED | 2 | |
| PD | 9 | |
| **On RRT (13)** | | **n=13** |
| Death | 7 | 53.84 |
| Discharged | 6 | 46.15 |
| Renal recovery | 20 on discharge | 64.51 (n=31) |

## DISCUSSION

The emergence of COVID infection and its variants have shown various involvements and multi organ targets. The rise in the number of AKI cases secondary to COVID infection has been a concern amongst the nephrologists worldwide. In our study the incidence of renal involvement COVID infected patients was 11%. Incidence of AKI in our study population was 9%. Renal Involvement is seen in upto 27% of children admitted with COVID-19 infection. AKI is the most common form of renal involvement followed by proteinuria, isolated hematuria. The incidence of AKI ranges from 1.2-70% in the literature (Table 4). The incidence of AKI is higher in patients with MIS-C compared to children with acute COVID-19 infection. The wide range of distribution is likely due to the heterogenesity of the studied population and different markers used to describe AKI. COVID-19 related kidney injury is more severe in adults in presence of co-existing morbidities and presence of acute lung injury or requirement of mechanical ventilation.[5] The various mechanism of injury caused by COVID-19 virus include the cytokine storm production leading to inflammation, altered permeability, hypovolemic injury and circulatory shock to the renal tissue. Expression of angiotensin converting enzyme 2 and organ cross talk may promote inflammation. Emerging evidences have also demonstrated direct cytopathic action by release of inflammatory cytokines (CD 8+, interleukins) in the kidney tissue.[1]

**Table 4: Incidence of AKI in various published studies across the world involving the pediatric population.**

| Author | Country | Study population (number) | Incidence of AKI (%) | Incidence of AKI (%) | | Incidence of stage 3 AKI (%) |
|---|---|---|---|---|---|---|
| | | | | Acute COVID | MIS-C | |
| **Kari et al[6]** | Saudi Arabia | 89 | 21 | 1.5 | 15 | 10.5 |
| **Basalely et al[7]** | NY, USA | 152 | 11.8 | 8.2 | 80 | 40<br>RRT (n=2) |
| **Deep et al[14]** | UK | 116 | 41.4 | All included subjects had MIS-C | | 27.6 |
| **Wang et al[8]** | China | 238 | 1.2 | NA | | |
| **Whittaker et al[9]** | UK | 58 | 22 | | | |
| **Godfred et al[10]** | USA | 570 | 18.4 | | | |
| **Gonzales et al[11]** | Columbia | 17 | 18 | | | |
| **Capone et al[12]** | NY | 33 | 70 | | | |
| **Derespina et al[13]** | NY | 70 | 12.9 | | | |

Stage III AKI was seen in 74% of the AKI subgroup in our study. Various studies by Deep et al, Stewart et al and Kari et al reported that children have a higher rate and faster progression to severe stages of AKI in presence of COVID infection. This progress is faster in presence of antibodies as the multi organ involvement is upscaled to many folds AKI in the presence of antibodies could be a part of the multisystem inflammatory syndrome that is precipitated by immune-complex deposition.[6,14,15]

Various studies reports presence of presence of proteinuria and hematuria as a presenting feature of patients with AKI in accordance with our study.[15-17] Raised inflammatory markers is associated with higher mortality in the pediatric population developing AKI secondary to COVID infection.[7,11] Higher mean values of D-dimer, ferritin has been reported in children with severe (stage II/III) AKI.[14] COVID-19 infection in severe cases has been shown to have increased coagulation activity, resulting in consumption of coagulation factors and disseminated microvascular thrombosis. Mortality is associated with higher and a rising levels of D- dimers levels.[18] In our study wherein children who developed AKI had higher requirement of IvIg and vasoactive drugs compared to those who did not have AKI or CKD cases. This may be due to the higher incidence of AKI in children who were admitted of MIS-C. The kidney injury predominates in MIS-C and is one of the leading cause of mortality. IVIg is known to reduce COVID-19 induced inflammatory response by blocking FcR activation on monocytes and its use is associated with higher recovery rate and lower 28 day mortality rate.[19] Requirement of vasoactive drugs, IvIg has been reported higher in children with AKI and acute COVID or MIS-C compared to those with no AKI.[7,20] In a study by Capone et al the incidence of AKI with COVID-19 was 70% and all of them required IvIg. Enoxaparin was used in 42% patients.[12]

Biopsy findings in our study showed focal segmental glomerulosclerosis (FSGS). Magoon et al in their study reported collapsing FSGS variant in their 2 adult subjects infected with COVID-19 infection.[23] RRT was used in 41.9% of our AKI population. Pediatric population with COVID-19 related AKI has been managed conservatively in various studies and do not require RRT. Stewart et al, States et al, Bjornstad et al and Kari et al have reported 28.8%, 1.9%, 47 % and 21.3% of AKI related to COVID-19 infection respectively.[6,10,15,21]

RRT was not required in any of these studied population. In certain studies, use of RRT and ECMO has been advocated.[7,13] Early initiation of RRT is associated with

better outcome in critically ill subjects irrespective of state of AKI. Removal of cytokines and restoration of defective complement regulation helps in preventing further inflammatory damage to the renal tissue.[8] In comparison to the pediatric population, the studies including adult subjects report a higher incidence in AKI as well as higher incidence of requirement of RRT and mortality. A Brazilian data reports the incidence of AKI as 50% of which 49% required an acute modality of RRT.[1,17]

Renal recovery was seen in 64.5% of patients who had AKI in our study. These included 6 discharged subjects who required RRT. Kari et al reported that 9% of AKI subgroup had residual renal impairment at the time of discharge and was attributed to hypotension, hypoxic injury, higher incidence of sepsis in these patients.[6]

In accordance to our study, a higher mortality rate was reported among the confirmed COVID cases and AKI compared to that of confirmed COVID cases and no AKI.[6,21] The mortality rate is significantly higher in severe stages of AKI.[22] This explains the raised mortality in our AKI patients as most of these patients were in stage 3 of AKI in their duration of admission.

Adults also report higher mortality rates in COVID related kidney injury compared to the pediatric population. 35% mortality in the adult population with AKI and COVID-19 was reported by Hirsch.[5] Advanced age is considered as the main risk factor for mortality due to COVID-19 infection.[4]

## CONCLUSION

Pediatric population although less significantly affected than adult cohorts may have worse outcomes in crtitically ill children. MIS-C has multi organ involvement and kidney injury predominates after the Lung injury. AKI in severe stages has been associated with higher mortality and poor renal recovery. Early initiation of RRT irrespective of the stage of AKI may reverse the cytokine induced injury and may improve the outcome.

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