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CASE REPORT

A Boy with COVID-19 Associated Severe AKI

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Introduction

COVID-19 is a newly discovered acute infectious disease caused by the SARS-CoV-2 virus, which is mainly manifested as acute respiratory disease characterized by acute interstitial and alveolar pneumonia and can affect multiple organs such as kidneys, heart, digestive tract and blood.[1] The reported incidence of acute kidney injury (AKI) in COVID-19 is found variable from 0.5-23% country to country in adults. Paediatric AKI is less common than adult, accounted for 1% in <10 years and 1.2-4.8% among total pediatric population. Amongst those who develop severe infection and require hospitalization, AKI was reported.[2] This case report presenting a paediatric case of COVID-19 associated AKI.

Case Report

Manik, 12 years old boy hailing from Cox's Bazar, got admitted in the Department of Paediatrics, Chittagong Medical College Hospital on 27 July, 2020 with the complaints of oedema, anuria for last 48 hrs and vomiting for several times for 1 day. Prior to the admission mother gave history of high grade fever for 1 day, not associated with chills and rigor, sweating, cough, respiratory distress, burning micturition, loose motion, altered consciousness and convulsions. No history of fever was found among the family members and close contacts and no history of travelling was found.

On examination, patient was found conscious, oriented, afebrile, having facial puffiness and pitting oedema of both extremities, pulse - 100/min, BP - 100/60 mmHg, RR - 28/min, SpO_2 - 100% in room air. No organomegaly and non-palpable bladder but ascites was present. Respiratory system and other systemic examinations revealed normal findings. Investigation revealed Hb - 11.4 gm/dL, ESR-28 mm, total leucocyte count 14,600/ mm^3, neutrophil - 83%, lymphocyte - 07%, platelet count - 1,50,000/mm^3, PBF - neutrophilic leukocytosis, S. creatinine 5.50 mg/dL, S. electrolytes (Na-130, K - 4.6, Cl-99 mmol/L), CRP - 6 mg/L, S. ALT - 187 U/L, S. albumin - 1.96 gm/dL, S. cholesterol-198 mg/dL, PT - 14.3 sec with INR-1.1, S. PTH - 516.8 pg/mL, CXR-normal, USG of whole abdomen showed acute renal parenchymal disease, moderate ascites, minimal bilateral pleural effusion. Sample for blood C/S was sent and as the patient was anuric, urine sample for routine, microscopic examination and culture could not been sent. Patient was provisionally diagnosed as Rapidly progressive glomerulo-nephritis (RPGN) and treatment was initiated with IV Methylprednisolon and injectable antibiotics (Ceftriaxone and Teicoplanin). Parents were counselled for kidney replacement therapy (KRT).

After 24 hrs, further investigation reports revealed low Hb (9.1 g/dL), neutrophilic leucocytosis with lymphopenia (10%), normal platelet count; raised blood urea (319 mg/dL) and creatinine (9.46 mg/dL), further drop of Na, hyperkalemia, metabolic acidosis. CRP raised to 20.5 mg/L. Raised ferritin - 824 ng/mL,D-dimer >10 mg/L with normal troponin-I, C3, C4, ANA, P-ANCA, C-ANCA and negative HBsAg, Anti-HCV, Anti-HIV were found. Sample for RT-PCR for COVID-19 was sent. Vitals remained

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stable with normal saturation at room air.

Intermittent peritoneal dialysis (IPD) was initiated. IPD was continued for 72 hrs but condition didn't improve rather rising trend of creatinine to12.72 mg/dL. Meanwhile, blood culture report revealed growth of *Acinetobacter baumannii* and RT-PCR for COVID-19 was found positive. Patient was then diagnosed as multisystem Iinflammatory syndrome in childern (MIS-C). Hemodialysis was initiated. Antibiotic was changed to Tygecycline according to culture-sensitivity report, subcutaneous LMWH was initiated. Oral prednisolone was initiated after 5 doses of I/V methylprednisolon. Patient got 3 sessions of hemodialysis by femoral catheter. On 9 August, 2020, patient developed blood vomiting and expired.

Discussion

According to KDIGO severe AKI (Stage 3) defined as when serum creatinine 3 times baseline or increase in S. Cr to ≥4 mg/dl (≥353.6 μmol/l) or initiation of RRT or in patients <18 years, decrease in eGFR to <35ml/min/1.73m^2.[3] So this is a case of severe AKI. Confirmed case of COVID-19 is defined as a person with laboratory confirmation of COVID-19 infection, irrespective of clinical signs and symptoms.[4]

Case definition of MIS-C by CDC[5] is defined as an individual aged <21 years presenting with fever (i), laboratory evidence of inflammation (ii) and evidence of clinically severe illness requiring hospitalization, with multisystem (≥2 organs) involvement (cardiac, renal, respiratory, hematologic, gastrointestinal, dermatologic or neurological) and no alternative plausible diagnoses and positive for current or recent SARS-CoV-2 infection by RT-PCR serology, or antigen test; or COVID-19 exposure within 4 weeks prior to the onset of symptoms.

In this case scenario, patient was 12 years old having history of fever for 24 hrs, laboratory evidence of infection (elevated CRP, d-dimer, ferritin, elevated neutrophils, lymphopenia and low albumin) and clinically severe illness with renal and hematologic involvement are consistent with MIS-C.

According to Morbidity and Mortality Weekly Report (MMWR)[6] 570 MIS-C patients were reported from March to July 2020, 565 patients were positive for SARS-CoV-2 by RT-PCR. Among them, 18.4% had AKI. In Great Ormond Street Hospital for Children NHS Foundation Trust (London, UK), most cases of AKI were found in those admitted to PICU (93%) and those with pediatric multisystem inflammatory syndrome temporarily associated with SIRS-CoV-2 (PIMS-TS, 73%).[7]

Several mechanisms are possible for AKI in COVID-19 patients, including multi-organ dysfunction syndrome, SARS-CoV-2 direct proximal tubular and podocyte injury, imbalanced renin-angiotensin aldosterone system (RAAS), hypovolaemia, hypoxia, acute respiratory distress syndrome (ARDS), infection-related generalized mitochondrial failure, and cytokine storm syndrome.[8]

Renal biopsy was not done. Diffuse proximal tubule injury with the loss of brush border, clusters of coronavirus like particles with distinctive spikes in the tubular epithelium and podocyte could not be demonstrated in this case.[9]

Cytokine storm syndrome (CSS) and MIS-C share some features but CSS typically present later in the course of acute infection (often during the 2nd week of the respiratory illness) with clinical decline, whereas the time frame of the development of MIS-C following COVID-19 exposure is 2-6 weeks, and affected patients are generally well prior to onset of symptoms. GI symptoms (diarrhea) and evidence of myocardial dysfunction tend to be more prominent in MIS-C than in CSS.[10] This patient had no respiratory symptoms and there was no fall of SpO_2. So remdesivir was not given. [8,11]

Continuous kidney replacement therapy (CKRT) specifically continuous venovenous hemo-diafiltration (CVVHDF) was the best choice for this patient for better clearance of cytokines by convection and diffusion. But in resource limited settings, intermittent hemodialysis by right internal jugular vein with increased blood flow than non COVID patient, even PD is a feasible alternative to CKRT.[12] There is sufficient evidence to prove that PD is equally effective as other forms of KRT.[13] As per the best expertise of our institution, peritoneal dialysis followed by hemodialysis was given to this patient.

Conclusion

This is the first paediatric COVID-19 positive severe AKI in our department. Paediatric studies are warranted to determine the cause-effect relationship between COVID-19 and AKI and effective management strategy in resource poor setting.

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