Residência RP Pediátrica
Publicação Oficial da Sociedade Brasileira de Pediatria
ISSN-Online: 2236-6814

Submitted on: 06/28/2020
Approved on: 06/29/2020

CASE REPORT

SARS-CoV-2 infection in a patient with rare chronic glomerulopathy: a case report

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Keywords:
Coronavirus Infections, Glomerulonephritis, Pediatrics.

Abstract

The pandemic by the coronavirus of severe acute respiratory syndrome 2 triggered, in late 2019, a great scientific mobilization in order to better understand the disease and unveil therapeutic strategies. This case report aims to describe a case of COVID-19 in an adolescent with rare chronic glomerulopathy. A 15-year-old patient, diagnosed with C3 glomerulonephritis in 2018. On May 15, 2020, after 2 months of abandoning treatment for maintenance of glomerulopathy, the patient was admitted with edema, oliguria, hypertension and worsening renal function. On May 18, 2020, already hospitalized and undergoing hemodialysis, he had low fever without a well-defined focus. The next day, he developed a sudden drop in oxygen saturation, around 79% in room air, high fever, mild dyspnea and hypotension. Collected exams for sepsis and PCR for COVID-19, the diagnosis of staphylococcy and COVID-19 was confirmed. Chest tomography revealed a nodular ground-glass image with bilateral pleural effusion. After removing the dialysis catheter and instituting antibiotic therapy, the patient evolved well clinically, with no new repercussions after 5 weeks of the onset of the condition. It is known that chronic kidney disease is an independent risk factor for the development of pneumonia and, although there is evidence that it is associated with an increased risk of developing the severe form of COVID-19 in adults, studies in pediatric populations show, up to the moment, that children with kidney diseases did not have an unfavorable evolution in relation to healthy children of the same age group.

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INTRODUCTION

The pandemic of severe acute respiratory syndrome caused by the coronavirus 2 (SARS-CoV-2) triggered, at the end of 2019, a great scientific mobilization in order to better understand the disease and find therapeutic possibilities. Despite the scarcity of data in the literature in the pediatric age group, there are records that children with chronic diseases are at risk for severe forms of infection by the new coronavirus (COVID-19)[1,2]. There are several case reports published about children with underlying diseases who developed the severe forms of COVID-19[1]. This report aims to describe a case of COVID-19 in an adolescent with a rare chronic glomerulopathy, C3 glomerulopathy.

CLINICAL CASE

A.J.P.F., 15 years old, presented, in October 2018, a rapidly progressive glomerulonephritis, diagnosed with C3 glomerulopathy. He underwent pulse therapy with methylprednisolone for 3 days, followed by 6 months of monthly doses of cyclophosphamide and, finally, maintenance treatment with mycophenolate mofetil (MMF) and oral corticosteroids. He lost clinical follow-up in December 2019, having suspended MMF in March 2020. On May 15, 2020, he was hospitalized with edema, oliguria, high blood pressure and significant worsening of kidney function, requiring hemodialysis therapy on May 16. 2020. On this occasion, the patient had no fever or any infectious component. On May 18, 2020, he developed a low fever without a clear site of infection. The next day, however, he developed a sudden drop in oxygen saturation, around 79% in room air, high fever, mild dyspnea and hypotension. On this occasion, a dialysis catheter was removed, oxygen support was provided with a non-reinhalant mask, all samples to test for sepsis and PCR for COVID-19 were collected, volume expansion and antibiotic therapy were performed, central venous access and a new dialysis catheter were provided, and he was transferred to intensive care unit. The patient did not require orotracheal intubation and was transferred on the following day to an infirmary bed for COVID-19 due to clinical stability and good saturation with a nasal oxygen catheter. Cultures of peripheral and central blood collected on the date of decompensation were positive for S. aureus. PCR result for COVID-19 collected on the third day of fever was positive. In order to expand the investigation regarding the repercussion of the infection by the new coronavirus, a chest tomography was performed, which revealed a nodular image in ground glass with the appearance of bronchopneumonia, with minimal bilateral pleural effusion and laminar atelectasis, lactate dehydrogenase of 289 IU/L, 260ng/Ml ferritin and 0.94ng/dL D-dimer. Fever and hypotension, justified by staphylococci, resolved with the institution of antibiotic therapy. The sharp drop in oxygen saturation, without significant dyspnea, in turn, is consistent with the diagnosis of pneumonia by COVID-19 and improved with non-invasive oxygen therapy. There was no specific treatment for SARS-CoV-2 infection, evolving without new clinical repercussions of COVID-19 pneumonia after 5 weeks of onset. The presence of mild bilateral pleural effusion was explained by the decompensation by C3 Glomerulopathy, for which immunosuppressive treatment was not performed because of infections. Despite the clinical stability achieved, it was necessary to maintain hemodialysis due to non-recovery of renal function.

COMMENTS

Chronic kidney disease (CKD) is an independent risk factor for the development of pneumonia, increasing the likelihood of a chronic kidney patient developing it by 97%. This was evidenced in a cohort study of more than one million medical records from 1996 to 2010, obtained from the Longitudinal Health Insurance Database of the National Institute of Health Research in Taiwan. The incidence rate of hospital and outpatient pneumonia was retrospectively compared in 15,562 patients with CKD and 62,109 without CKD. Other comorbidities present in patients, such as diabetes, cardiovascular disease, asthma and chronic obstructive pulmonary disease were independently associated with an increased risk of pneumonia[3].

Although there is evidence that chronic kidney disease is associated with an increased risk of developing the severe form of COVID-19 in adults[4], studies in pediatric populations have revealed that, to date, children with kidney disease have not had an unfavorable evolution in relation to healthy children of the same age group[2].

In a Spanish study[2] involving 16 children with chronic kidney disease and who had positive RT-PCR for SARS-CoV-2, the basic kidney diseases were: renal dysplasia (5 patients), nephrotic syndrome (5 patients), uropathies (2 patients), IgA nephropathy (1 patient), renal scarring (1 patient), vasculitis (1 patient) and cortical necrosis (1 patient). In this series of cases, therefore, of all children with renal pathologies who tested positive for the new coronavirus, 37.5% had the prevalence of glomerulopathies. In addition, of the total patients studied, 56% were on some immunosuppression regimen. No child in this study needed to be transferred to the intensive care unit or died[2]. In another study[4], however, 1,391 symptomatic and asymptomatic children with a history of contact with suspected or confirmed cases of SARS-CoV-2 infection were tested, and 171 showed positive RT-PCR for SARS-CoV-2, three required mechanical invasive ventilation. Of these patients, however, all had some adjacent clinical condition, such as hydronephrosis, leukemia under chemotherapy and intussusception. The child with a history of intussusception died[5].

In this report, we describe the case of a patient with C3 Glomerulopathy, who had suspended maintenance immunosuppression 2 months before admission, was hospitalized with worsening kidney function, requiring hemodialysis. At the same hospitalization, he was diagnosed with acute SARS-CoV-2 infection through RT-PCR, which was collected because he had severe hypoxemia, without dyspnea, in the presence of a bacteremia. Despite the radiological changes suggestive of COVID-19, the patient evolved with rapid clinical improvement after removal of the dialysis catheter and institution of antibiotic therapy. We can conclude that S. aureus bacteremia led to the clinical instability with hypotension and fever, in addition to SARS-CoV-2 pneumonia through the manifestation of severe oxygen hyposaturation without major dyspnea. On the other hand, we cannot affirm the influence of the virus in the decompensation of glomerulopathy and in the worsening of renal function, since immunosuppressive treatment was suspended 2 months before admission. Finally, much remains to be known about the SARS-CoV-2 infection in pediatric patients with kidney disease.

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