

Case Report

COVID-19 triggered systemic lupus erythematosus in a child: a case report

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ABSTRACT

The COVID-19 pandemic has continued to wreak havoc globally during the second wave. Even though it tends to be asymptomatic or cause only a trivial illness in children, it is reported to be associated with a delayed hyper-inflammatory response syndrome resulting in multi-organ dysfunction in children. It is possible that through unknown mechanisms, it could also result in triggering of other auto-immune disorders. We report a case of pediatric systemic lupus erythematosus (SLE) with lupus nephritis suspected to be triggered by SARS-CoV-2 virus which is not reported in the literature so far.

Keywords: COVID-19, Systemic lupus erythematosus, Auto-immunity

INTRODUCTION

COVID-19 is caused by a newly emerged Corona virus, SARS-CoV-2(1). In adults, it causes respiratory illness and cytokine storm which could result in multi-organ dysfunction and death.² However, in children, COVID-19 infection results in an asymptomatic or mildly symptomatic disease.³ Some children developed late onset hyper-inflammatory syndrome, labeled as multisystem inflammatory syndrome in children (MIS-C), characterized by multi organ dysfunction induced by inflammation secondary to a previous SARS-COV-2 infection.⁴ We still do not know whether this virus has the capability of triggering or precipitating any auto-immune illnesses. We report a case of SLE in a 12-year-old male child thought to be triggered by COVID-19 infection.

CASE REPORT

A 12-year-old male child, previously fit and well, was admitted to our hospital with a history of intermittent fever for 8 days and difficulty in breathing of one day

duration. Other family members of the child were healthy, and the child had been up to date with recommended vaccinations.

On initial assessment, he was hypoxic (SPO₂ of 75% in room air) tachypneic, tachycardic and normotensive. He was afebrile and there was no rash, edema or lymphadenopathy. He was screened positive for COVID-19 using RT-PCR test. His chest x-ray showed bilateral lung parenchymal infiltrates. A CT Chest demonstrated evidence of bilateral ground glass opacities consistent with a CORADS score of 5 (Figure 1). Initial bloods showed anemia (Hb of 6.5 gm/dl) and lymphocytosis and elevated inflammatory and evidence of cytokine storm. He had a CRP of 4.8 mg/L (<0.6 mg/L), IL-6 of 86 pg/ml (<15 pg/ml), lactate dehydrogenase 896 U/L (<170 U/L) and D-dimers were 2770 ng/mL (<250 ng/mL). He had no evidence of hemolysis on peripheral smear and had normal serum ferritin, vitamin B12 and serum folate levels and anemia were thought to be secondary to a chronic inflammatory process. He was started on IV methylprednisolone (10 mg/kg/dose twice daily) for 5 days and subcutaneous low molecular weight heparin

(Enoxaparin) as per our COVID-19 management protocol. He was started on non-invasive ventilatory support (Bi-level positive airway pressure) and was given broad spectrum antibiotic cover (Meropenem, vancomycin) after sending appropriate cultures. In view of clinical deterioration, intra-venous Remdesivir was given for 5 days as per unit protocol. He showed signs of recovery and had been self-ventilating in room air by day-4 of admission. He was discharged after 7 days of hospital admission on tapering doses of steroids.

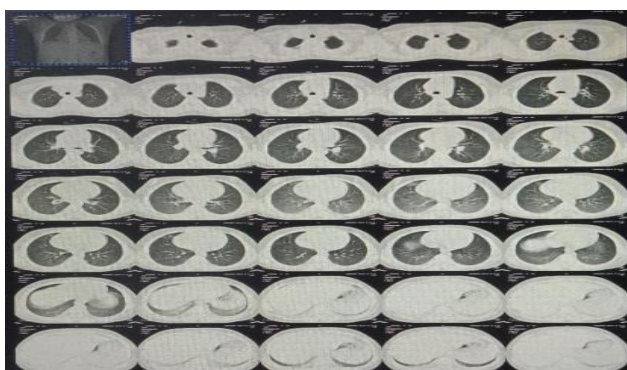


Figure 1: CT chest of the child of bilateral ground glass opacities (CORAD-5) consistent with COVID-19 lung.

However, one week following discharge, he developed itchy, vesiculo-bullous skin eruption over the trunk (Figure 2) and developed facial puffiness and pedal oedema. There was no history of hematuria. He had normal urine output. On assessment, his blood pressure was within normal limits (110/60 mmHg). Further work-up revealed gross urinary proteinuria (3+), no hematuria and an elevated urine protein creatinine ratio of 10.4 mg/mg (<0.2). He had low serum albumin level (2.2 g/dL) and a high cholesterol level (250 mg/dL), normal ASO titers, and low C3 and C4 levels. He also had a strongly positive anti-nuclear antibodies (3+, 1 in 80 dilution) and anti-ds DNA antibodies (4+, 1 in 10 dilution) which raised the suspicion of SLE. A biopsy of the skin lesions demonstrated sub-epidermal bullae with eosinophilic infiltrate consistent with a differential diagnosis of Bullous pemphigoid or bullous SLE.



Figure 2: Vesiculo-bullous skin lesions on the trunk.

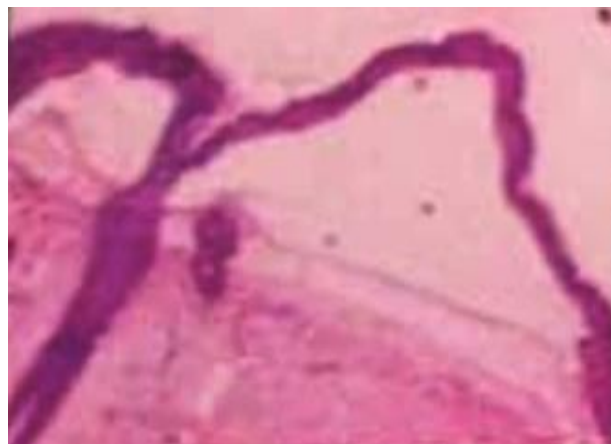


Figure 3: Biopsy of skin lesions demonstrating sub-epidermal bullae with eosinophilic infiltrate.

He was started on oral steroids along with diuretics as part of initial management plan. However, due to poor response to diuretics and steroids, he underwent a renal biopsy a week later which was consistent with class V lupus nephritis with no tubular atrophy. Following inputs from a multi-disciplinary team (general pediatricians, pediatric intensivists, pediatric nephrologist, rheumatologist and dermatologist) he was started on IV cyclophosphamide therapy at a dose of 750 mg/m² once in 2 weeks. His skin lesions showed remarkable improvement (Figure 4A and B) after 2 cycles of IV cyclophosphamide along with signs of gradual resolution of proteinuria and edema. He continued to be in remission during follow-up.

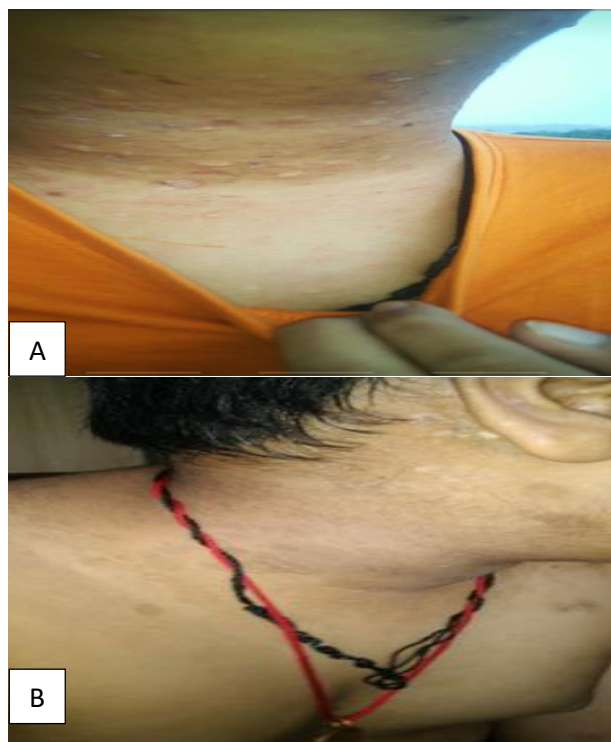


Figure 4 (A and B): Skin lesions before and after IV cyclophosphamide treatment.

DISCUSSION

COVID-19 induced hyperinflammatory syndrome is well described in the literature. However, whether COVID-19 could trigger other autoimmune diseases remains unknown. So, far there is only one reported case of COVID-19 triggered SLE in a 85 years old female in adult literature and we failed to find any similar case reports in pediatric age group.⁵ In this child, we speculate that SARS-CoV-2 infection could be the most likely triggering agent for SLE though, we failed to explain the association between symptomatology and underlying pathogenesis. It is unclear whether triggering of autoimmunity is the result of direct viral attack or secondary to stimulation of complex autoimmune mechanisms. There is an urgent need to investigate the underlying pathogenetic mechanisms triggering autoimmune pathology in SARS-COV2 infected patients.

CONCLUSION

COVID-19 is associated with a spectrum of illness in children ranging from trivial febrile illness to a potentially life-threatening multi-system inflammatory syndrome. It could also act as a trigger of other autoimmune diseases (like SLE in our case) in children. The mechanisms underlying COVID-19 triggered autoimmune diseases are poorly understood and should be an area of active research in future.

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