International Journal of Contemporary Pediatrics
Pereira RA et al. Int J Contemp Pediatr. 2023 Aug;10(8):1342-1344
http://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20232258

Case Report

Purpura fulminans-a rare complication post COVID-19 infection in children

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Received: 22 May 2023
Revised: 15 June 2023
Accepted: 20 June 2023

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ABSTRACT

Coronavirus disease 2019 (COVID-19) was officially declared as a pandemic in March 2020. COVID-19 infection and the post infectious sequalae has been widely researched since then. Post infectious inflammatory sequalae and its associated complications like toxic shock syndrome, Kawasaki like disease, macrophage activation syndrome, rather than the infection per se was found to be more of a concern in children, leading to more morbidity and mortality. Purpura fulminans was not a presentation reported post COVID-19 infection. Herein, we aim to describe a post COVID-19 infection patient who presented with purpura fulminans as a rare complication.

Keywords: Children, Purpura fulminans, Post COVID-19 infection

INTRODUCTION

Severe acute respiratory syndrome coronavirus 2 is a viral infection that has affected large number of people all over the globe. COViD-19 was declared a Pandemic in March 2020 and has been the center of research be it with respect to its course, clinical manifestations, complications, treatment, prevention and post infectious sequalae.[1-3]

COVID-19 infection has a different course in different age groups. In children, COVID-19 infection as such was not studied to have major adverse outcomes. However, the post infectious inflammatory sequalae has caused significant morbidity and mortality. The common manifestations being toxic shock syndrome, Kawasaki like disease, macrophage activation syndrome.[4]

Herein we bring forth a case manifesting a rare complication of purpura fulminans following COVID-19 infection in children.

CASE REPORT

A 2-year-old, female child was brought to our hospital on 26/12/2021 with history of high-grade fever, irritability and bluish-black reticular pattern of discoloration of skin over both upper and lower extremities and trunk noticed 2 days prior to admission and had progressively increased. The mother did not give history of the presence of any other systemic symptoms in the child at admission. She however gave a history of fever and respiratory infection 3 weeks prior to admission in the child and family members.

On physical examination, the child was toxic looking and pale. She had a GCS of 15/15, tachycardia (Heart rate of 118/min), hypotension (BP-70/40 mmHg), capillary refill time of more than 3 seconds and was febrile (Temperature-101F). Saturation was 86% at room air by pulse oximetry. Both the upper and lower limbs were noticed to have bluish-black ecchymotic patches with a reticular pattern, the digits being the most affected

(Figure 1). Systemic examination revealed hepatosplenomegaly.

The Table 1 summarizes the laboratory parameters of the patient. Due to COVID-19 outbreak, samples of pharynx and nasopharynx of the patient were taken and PCR for COVID-19 infection was done which were negative. Due to the presence of high inflammatory markers, sterile blood culture and negative serology reports for other infections, COVID-19 antibodies (IgM antibodies) were done which were significantly positive (Titre >200). She also underwent the other work-up for Multisystem inflammatory syndrome in children (MIS-C) like serum ferritin which was elevated (900 ng/ml), however D-Dimer, ECHO which were normal.

The child was admitted to the paediatric ICU and a possible diagnosis of infection induced purpura fulminans was made. She was started on empirical broad-spectrum antibiotics, inotropic support, oxygen therapy and other symptomatic treatment. The child responded to the given treatment, inotropic support and oxygen supplementation was gradually tapered and stopped after 48 hours of admission, fever subsided by day 3 of admission with no further worsening of the escharic patches. On day 5 of admission the eschars were well demarcated and healing had begun. By day 10 of admission most of the lesions had healed, with digital gangrenous changes of few finger and toe tips. The lesions were dressed with SSG dressing to aid in healing.

The child was shifted to the paediatric ward on day 4 of admission and remained haemodynamically stable and afebrile during the rest of hospital stay. She was given a course of 21 days of intravenous antibiotics. There was serial improvement in the inflammatory markers and the general condition of the child. She was discharged after 21 days of hospitalisation (Figure 2).

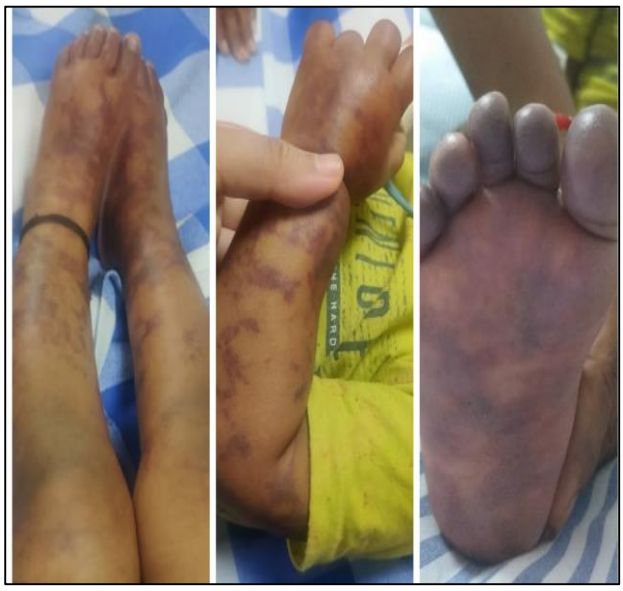

Figure 1: Clinical picture at admission.

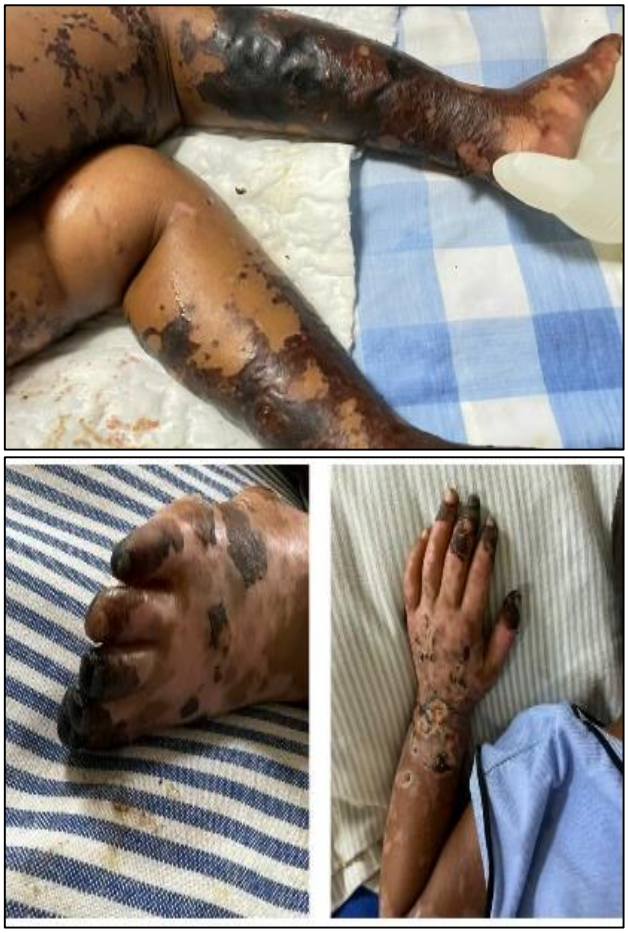

Figure 2: Clinical picture at discharge.

Table 1: Investigation reports of the child.

Investigation	At admission	At discharge
WBC (mm^3)	26900	19900
Hb (gm/dL)	7.8	10.2
Platelet (mm^3)	83000	718000
PT (sec)	11.9	
INR (index)	1.08	
PTT (sec)	25.2	
Cr (mg/dL)	0.34	0.41
Na (mEq/L)	129	134
K (mEq/L)	3.2	4
AST (IU/L)	39	
ALT (IU/L)	18	
CRP (mg/L)	137.08	41.68
ESR (mm/h)	23	10
Albumin (g/dL)	2.6	3.4
Ferritin (ng/ml)	900	35
Blood culture	No growth	

DISCUSSION

Purpura fulminans is a life-threatening condition of acute onset characterized by cutaneous haemorrhage and necrosis caused by disseminated intravascular coagulation and dermal vascular thrombosis.

Three types are distinctly identified: inherited or acquired abnormalities of the protein C or other coagulation systems, acute infectious purpura fulminans and idiopathic.[5] The idiopathic type was recognized first as an entity in 1964 and described as a disorder of childhood, which generally follows a benign illness.[6] Inherited and acquired abnormalities of the protein C and protein S anticoagulant pathway were found to be responsible for the majority of cases of purpura fulminans.

Gram-negative bacteria like *Neisseria meningitidis* are the most common cause of the acute infectious variety. Varicella virus infection, *Pneumococcal* sepsis and measles have also been found to be associated with the infectious variety of purpura fulminans.

In the neonatal period group B *Streptococcus* is the major cause but *Staphylococcus*, *Escherichia coli*, *Enterobacter* and others have also been described.[7]

Shah et al explained a rare association of purpura fulminans with West Nile virus infection.[8] Clinical patterns associated with post COVID-19 infection in children is associated with elevated D-dimer reports, however disseminated intravascular haemolysis has rarely been found. This patient presented to us in January 2022. To the best of our knowledge, no case has yet been published in literature of purpura fulminans as a complication post COVID-19 infection in children and this case appears to be the first.

CONCLUSION

The post infectious inflammatory sequelae post COVID-19 infection is associated with intense inflammation, leading to high rates of thrombotic complications. Purpura fulminans being a life-threatening condition that needs early intervention and timely treatment. Paediatricians should hence identify the condition early, start treatment, provide early wound care and surgical management, preventing severe necrosis.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Pereira RA, Mundkur SC, Neminathan A, Moras KJ, George DM. Purpura fulminans-a rare complication post COVID-19 infection in children. Int J Contemp Pediatr 2023;10:1342-4.