DOI: 10.1111/pde.14699

Pediatric Dermatology WILEY

Henoch-Schönlein purpura: Another COVID-19 complication

Abstract

Whether affecting children or adults, SARS-CoV-2 infection (COVID-19) can have multi-organ involvement mediated by an inflammatory cascade. Immunoglobulin A (IgA) is one of the key components of the inflammatory cascade that can lead to endothelial injury and inflammation. IgA vasculitis or Henoch-Schönlein purpura (HSP) has been rarely reported in the context of COVID-19. In this report, we highlight a case of HSP occurring 2 days after diagnosis of COVID-19 in a 16-year-old boy, who presented with palpable purpura of the lower extremities and buttocks, diffuse abdominal pain, hemoptysis, and hematochezia. He was treated with oral prednisolone with rapid clinical improvement.

1 | INTRODUCTION

Vascular pathology has been linked to COVID-19 pediatric and adult disease manifestations. Endothelial cells are a target for multiple inflammatory mediators including immunoglobulins.[1] As such, multiple cases of different types of COVID-19-associated vasculitis have been reported (1). Among these, Henoch-Schönlein purpura (HSP), an immunoglobulin A (IgA)-mediated self-limiting small vessel vasculitis of childhood,[2] has been rarely described. Herein, we present a case of a 16-year-old boy who developed HSP 2 days after being diagnosed with COVID-19.

2 | CASE PRESENTATION

A previously healthy 16-year-old boy presented with a 1-week history of palpable purpura involving both lower limbs. On the day of presentation, the patient developed severe diffuse abdominal pain and experienced one episode of hemoptysis and hematochezia. Two days before his rash started, the patient had tested positive for COVID-19. He also complained of sore throat and muscle pain.

On physical examination, his blood pressure was normal. There was a non-blanching and purpuric rash over his lower limbs, extending from the soles up to the knees that also involved the buttocks (Figure 1A,B). There were no signs of deep vein thrombosis or superficial thrombophlebitis.

Investigations (Table 1) showed elevated inflammatory markers and serum IgA with hypoalbuminemia and hypogammaglobulinemia. A urinalysis was positive for proteinuria and microscopic hematuria. A subsequent 24-hour urine collection revealed severe proteinuria (3651 mg/24 hours).

The patient was diagnosed with HSP based on the clinical and laboratory findings. He was discharged on prednisolone 30 daily, ramipril 2.5 mg daily for his renal involvement, and trimethoprim/sulfamethoxazole prophylaxis, with recommendations for isolation and close follow-up.

His COVID-19 test became negative 11 days after the initial positivity. Two weeks later, 24-hour urine collection showed less proteinuria (2870 mg/24 hours). Six weeks later, the rash had faded and a 24-hour urine protein had decreased to 780 mg.

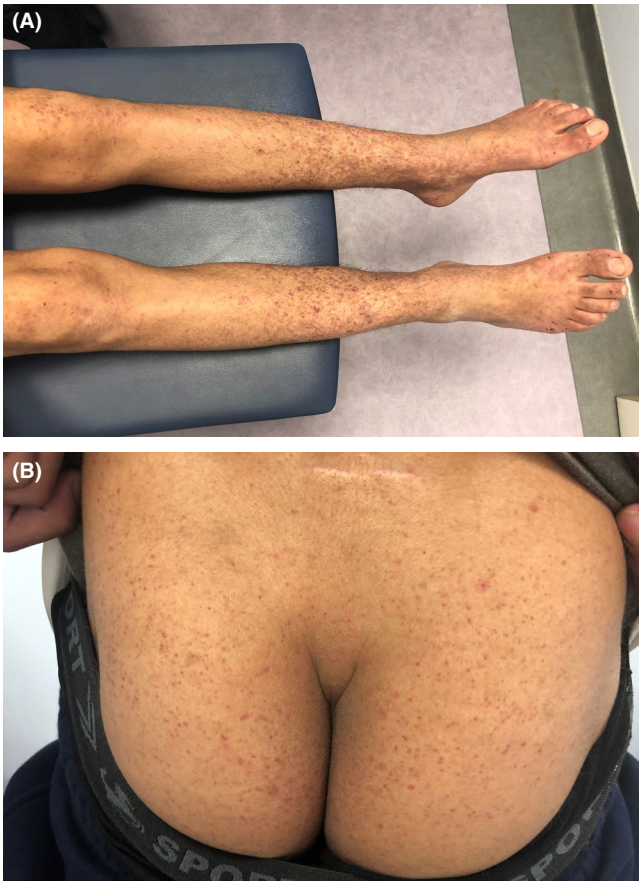

FIGURE 1 Palpable purpura present on the bilateral lower extremities (A) and buttocks (B)

Clinical findings	Laboratory findings
Purpura on bilateral legs and buttocks	ESR 84 mm/hr (0-20 mm/hr)
Severe abdominal pain	CRP 13.8 mg/L (<10 mg/L)
Hematochezia	Serum IgG decreased at 569 mg/dL (>600 mg/dL)
Hemoptysis	Serum IgA increased at 391 mg/dL (80-350 mg/dL)
	D-dimer 405 ng/mL (<250 ng/mL)
	Serum creatinine 0.67 mg/dL (<1.35 mg/dL)
	Urine protein 3651/24 hours (<100 mg/24 hours)

TABLE 1 Clinical and laboratory findings in our case

3 | DISCUSSION

In HSP, a bacterial or viral infectious trigger damages endothelial cells via activation of complement, while IgA contributes to non-granulomatous inflammation and vasculitis.[2] As a result of the enhanced IgA response observed in COVID-19,[3] we hypothesize that IgA could partly explain some common organ injuries that occur in COVID-19.

In our case, aspirin could not be used because of renal involvement. Anticoagulants were not used because of the mild nature of the illness and the absence of multiple comorbidities. Neither a skin nor kidney biopsy was performed because our patient's illness satisfied the EULAR/PRINTO/PRES criteria for the diagnosis of HSP.[4]

4 | CONCLUSION

COVID-19 triggers a severe inflammatory reaction that can lead to endothelial injury by several mechanisms. A key player in such endothelial inflammatory reaction is IgA, which can deposit within the endothelium, activating other cytokines, and rarely leading to HSP.

DATA AVAILABILITY STATEMENT

Research data are not shared.

KEYWORDS

COVID-19, Henoch-Schönlein purpura, immunomodulation, vasculitides

Georges El Hasbani MD[1]
Ali T. Taher MD, PhD[1]
Ali S. M. Jawad MD[2]
Imad Uthman MD, MPH[1]

[1]*Department of Internal Medicine, American University of Beirut Medical Center, Beirut, Lebanon*
[2]*Department of Rheumatology, Royal London Hospital, London, UK*

Correspondence
Georges El Hasbani MD, Department of Internal Medicine, American University of Beirut Medical Center, Beirut, Lebanon.
Email: george.hasbany@lau.edu

ORCID

Georges El Hasbani https://orcid.org/0000-0003-2571-1450

REFERENCES

1. Becker RC. COVID-19-associated vasculitis and vasculopathy. *J Thromb Thrombolysis*. 2020;50(3):499-511.
2. Inamadar AC, Palit A. Childhood cutaneous vasculitis: a comprehensive appraisal. *Indian J Dermatol*. 2009;54(2):110-117.
3. Yu HQ, Sun BQ, Fang ZF, et al. Distinct features of SARS-CoV-2-specific IgA response in COVID-19 patients. *Eur Respir J*. 2020;56(2):2001526.
4. Ozen S, Pistorio A, Iusan SM, et al. EULAR/PRINTO/PRES criteria for Henoch-Schonlein purpura, childhood polyarteritis nodosa, childhood Wegener granulomatosis and childhood Takayasu arteritis: Ankara 2008. Part II: final classification criteria. *Ann Rheum Dis*. 2010;69(5):798-806.