


Post BNT162b2 mRNA COVID-19 vaccination Henoch-Schönlein Pupura

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INTRODUCTION

Administration of BNT162b2 mRNA COVID-19 vaccine on a large scale was performed to combat the COVID-19 pandemic. Leading to the identification of various rare but significant vaccine-associated side effects one of which is myocarditis and pericarditis post vaccination.¹ There have been reports of COVID-19 vaccine-associated vasculitis, both as a new onset IgA-positive leukocytoclastic vasculitis in an otherwise healthy adult man and also a leukocytoclastic vasculitis flare in an adult woman with autoimmune conditions.^{2,3}

CASE REPORT

Our case is a 15-year-old Indian girl presenting with bilateral lower limb rash 2 hours after second dose of BNT162b2 mRNA COVID-19 vaccination, and 1 week after her first dose. The rash started at the right medial knee before spreading to bilateral lower limbs. The maculopapular rash was purpuric, palpable and non-blanching. She also had a fever and a tender right ankle swelling ([figure 1](#)). No angioedema, no wheeze or dyspnoea was noted. The patient has a history of atopy including asthma and allergic rhinitis which was well managed without medications.

Given the ankle arthritis, fever and a vasculitic rash, clinical suspicion was for small vessel vasculitis, clinically Henoch-Schönlein Pupura

(HSP), likely triggered by the second vaccine. A series of investigations were performed ([table 1](#)). Biochemically, the most significant results were the elevated C3 1.74 g/L (0.80–1.60 g/L), erythrocyte sedimentation rate 48 mm/hour (3–15 mm/hour), C reactive protein 10.7 mg/L (0–5 mg/L). Haematologically, there was also a mild leucocytosis $10.2 \times 10^9/L$ high ($4-9.6 \times 10^9/L$) and a mild neutrophilia $7.42 \times 10^9/L$ ($1.90-6.6 \times 10^9/L$) with no disturbance in the coagulation. Renally, there was no evidence of renal impairment, however, proteinuria and a high leukocyturia was noted. Subsequent biopsy of the rashes showed evidence of a leukocytoclastic vasculitis with negative direct immunofluorescence.

After being started empirically on 90 mg erto-coxib and topical betamethasone valerate 0.1% cream, patient improved symptomatically. On review in clinic in about 2 weeks after the initial flare of the rash, the rashes have since resolved. The clinical history with the physical examination, laboratory findings and clinical response to treatment is highly suggestive of a BNT162b2 mRNA vaccine-triggered HSP.

DISCUSSION

This is a rare case of BNT162b2 vaccine-triggered HSP which has been noted in paediatric cases.⁴ Though the BNT162b2 vaccine is generally safe, there have been reports of

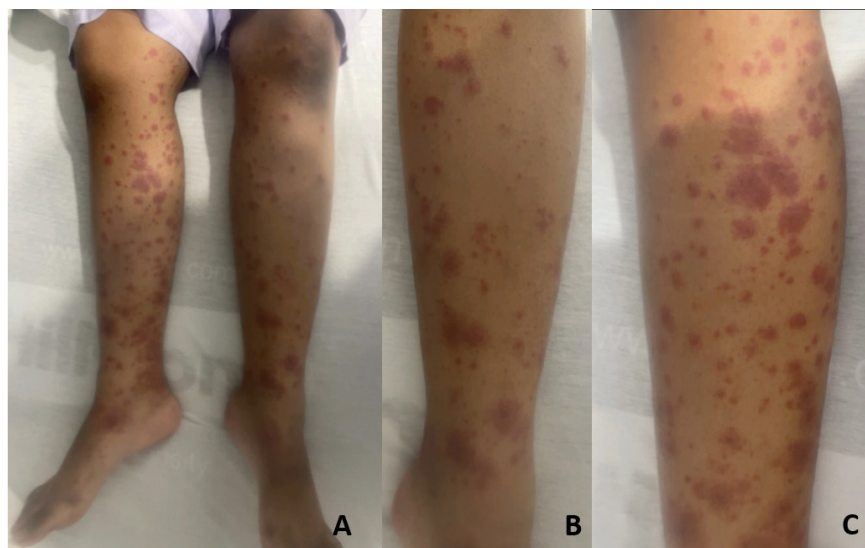


Figure 1 Maculopapular purpuric palpable and non-blanching rash in keeping with a vasculitic rash. Part a shows both legs, part B is a close up of the left leg while part C is a close up of the right leg.



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Table 1 Summary of patient's investigations

Inflammatory and autoimmune markers	
ANCA	Negative
ANA-immunofluorescence	<80
Anti-ds-DNA	<25 (0–25 IU/mL)
Cryoglobulin	Negative
Complement 3 (C3)	1.74 elevated than reference range (0.80–1.60 g/L)
Complement 4 (C4)	0.41 (0.17–0.60 g/L)
Anti-HBc, Total	Non-reactive
HBsAg	Non-reactive
Anti-HCV	Non-reactive
Erythrocyte sedimentation rate (ESR)	48 elevated (3–15 mm/hour)
C reactive protein (CRP)	10.7 elevated (0–5 mg/L)
Full blood count	
White blood cell	10.2 high (4–9.6×10 ⁹ /L)
Haemoglobin	122 (118–146 g/L)
Platelets	338 (150–360×10 ⁹ /L)
Absolute neutrophils	7.42 elevated (1.90–6.6×10 ⁹ /L)
Absolute lymphocytes	2.04 (1.10–3.10×10 ⁹ /L)
Absolute monocytes	0.56 (0.20–0.70×10 ⁹ /L)
Absolute eosinophils	0.05 (0.00–0.60×10 ⁹ /L)
Absolute basophils	0.08 (0.00–0.10×10 ⁹ /L)
Coagulation panel	
APTT	31.4s
PT	13.9s
INR	1.1
Urine protein and creatinine ratio	
Total protein on admission	0.3 U
Creatinine admission	18.1 U
Prot:crea ratio on admission	14
Urine microscopy	
WBC, urine on admission	>225 (0–6 cells/μL)
RBC, urine on admission	18 (0–13 cells/μL)
Histopathology	
Punch biopsy of skin to the subcutis. The stratum corneum is basketweave. The epidermis is unremarkable. There is a superficial and deep perivascular infiltrate of lymphocytes with many neutrophils. There is nuclear dust and red cell extravasation. Fibrin exudation is seen around some upper dermal blood vessels. This is in keeping with the diagnosis of leukocytoclastic vasculitis.	
Direct immunofluorescence	
IgM	Negative
IgG	Negative
C3	Negative
Fibrinogen	Negative

autoimmune/inflammatory complications.¹ The mechanisms underlying the pathogenesis of vaccination-associated vasculitis remain unclear, although innate immunity-mediated response to viral agents or vaccine excipients via molecular mimicry, epitope spreading, bystander activation and polyclonal activation have been proposed.⁵ For the vasculitis to

be suggested to be vaccine linked, it should be of a relatively short, self-limited course which may occur after a latent period of about 1–2 weeks, showing a wide geographic distribution and with no alternative explanation that can be identified and in some instances shows response to immunosuppressive therapy.⁶ These were features seen in our case.

This report hopes to highlight to clinicians some of the potential rare complications associated with BNT162b2 vaccination. Hopefully encouraging further studies to identify the relationship between vasculitides and vaccination. Future studies can be performed to further improve the already considerable vaccine safety profile, hopefully reducing vaccine hesitancy and increasing future vaccine uptake.

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