Development of anti-NXP2 dermatomyositis following Comirnaty COVID-19 vaccination

Adrian Y S Lee,1,2,3 Caroline Lee,1 David A Brown,1,2,3 Dan Suan1,3

A 53-year-old Asian man, previously well with no family history of autoimmunity, received his second Comirnaty (Pfizer) COVID-19 vaccine in 2021. Two weeks after, he developed bilateral proximal myalgias and a crustated eruption across his anterior chest, shoulders, deltoids and around the frontal hairline (figure 1A–C). He was referred to hospital where his plasma creatine kinase (CK) returned elevated at 14 659 IU/L (<200).

He was afebrile and had proximal muscle weakness with 4/5 power in the upper and lower limbs. Blood tests demonstrated a mildly raised C reactive protein (5 mg/L, <4), normal troponin, normal renal function and raised aspartate aminotransferase (457 U/L, 10–35) and alanine aminotransferase (206 U/L, 10–50). Autoimmune serology revealed the presence of antinuclear matrix protein 2 (anti-NXP2) antibodies.

A pan-CT scan did not reveal evidence for solid organ malignancies or interstitial lung disease. A transthoracic echocardiogram was normal. An electromyography of the vastus lateralis confirmed the presence of myopathy, and his thigh MRI revealed symmetrical, hyperintense T2 signal in his quadriceps muscles (figure 1D). A vastus lateralis biopsy confirmed a non-necrotising myositis. Wound swabs of his skin lesions confirmed heavy growth of Staphylococcus aureus.

The patient was treated with pulse intravenous methylprednisolone, followed by 1 mg/kg prednisolone and initiation of mycophenolate. His CK fell to 7236 IU/L 3 days after treatment commenced, associated with improved myalgias. He was commenced on oral antibiotics with rapid resolution of his skin infection and was discharged.

One week later, he noticed increasing myalgias, dysphagia and was readmitted. His CK was 10 387 IU/mL. He was treated again with methylprednisolone, intravenous immunoglobulin (2 g/kg loading dose), followed by rituximab (1 g, fortnight apart), with good improvement in his symptoms and CK. He remains stable on mycophenolate, tapering oral prednisolone and monthly intravenous immunoglobulin infusions.

Anti-NXP2 dermatomyositis is a subset of idopathic inflammatory myopathies (IIM) that can present in both children and adults and may be associated with malignancies.3 Patients tend to have a relapsing and remitting course and may remain refractory to first-line immunomodulatory agents as was seen in our patient. Despite its distribution, the phenotype of his skin lesions were not in keeping with dermatomyositis. In retrospect, we cannot help but wonder if the ecchyma was in fact a mild dermatomyositis cutaneous eruption with superimposed bacterial infection.

COVID-19 vaccinations have been reported to stimulate several immunological disorders. Post ChAdOx1 (AstraZeneca) and Ad26.COV2.S (Janssen) COVID-19 vaccine inflammatory myositis, without defining autoantibodies, have been reported in a South Asian cohort that responded well to courses of prednisolone.2 Another case reported a woman who developed isolated deltoid myositis following injection with a COVID-19 mRNA vaccine.3

Figure 1 Skin lesions and MRI of the thigh. Crusted skin lesions were present on the patient’s scalp (A), chest (B) and upper back (C). (D) A T2-weighted MRI of his thighs were performed demonstrating hyperintense signals (arrows) in his vastus lateralis.
Other vaccines have been documented as inducing myositis and/or myocarditis including the influenza and hepatitis B vaccines. However, IIMs have rarely been reported. To our knowledge, this is the first report of a Comirnaty (Pfizer) vaccine-induced, autoantibody-positive IIM. Future case reports on this rare sequela will provide greater insight into the natural history of these disorders and how they compare with de novo IIM.

Acknowledgements The patient is thanked for his informed consent for the publication of the case and associated images. The authors are grateful to the medical and nursing staff involved in the care of this patient.

Contributors AYSL contributed to concept design, clinical care of the patient and drafted the manuscript. CL contributed to clinical care of the patient. DAB contributed to clinical care of the patient and supervision. DS contributed to concept design, clinical care of the patient and supervision. All authors have reviewed and revised manuscript critically for important intellectual content.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Consent obtained directly from patient(s).