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Cutaneous vasculitis following COVID-19 vaccination

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Vaccines against SARS-CoV-2 represent a pivotal and effective countermeasure to contain the COVID-19 pandemic. Four vaccines are approved by the European Medicines Agency: two messenger RNA-based vaccines encoding the spike protein of SARS-CoV-2 (BNT162b2, Pfizer–BioNTech; mRNA-1273, Moderna) and two adenoviral vector-basedvaccines encoding the spike protein (ChAdOx1 nCoV-19, AstraZeneca; Ad.26.COV2.S, Janssen).[1]

As of Sept 23, 2021, more than 83 million vaccine doses were administered in Italy, with approximately a fifth of recipients receiving ChAdOx1 nCoV-19 vaccine.[2] Here, we report three cases of cutaneous vasculitis developing in previously healthy individuals shortly after vaccination with ChAdOx1 nCoV-19.

The clinical features of the patients are summarised in the appendix (p 1). Briefly, patient 1 was a 57-year-old man with a history of hypertension but no previous personal or family history of autoimmunity. Purpura developed 14 days following the first vaccine dose, initially affecting the lower limbs and rapidly spreading to the abdomen, torso, and head (figure). He received treatment with 1 mg/kg prednisone, which led to progressive resolution of skin lesions over 3 weeks. Patient 2 was a 58-year-old man, whose previous medical history was also unremarkable with no history of autoimmunity. Purpura developed 7 days following the second dose of vaccine, spreading from the lower limbs to the abdomen and trunk (appendix p 2). He received 0·5 mg/kg prednisone, to no clinical benefit, and then 1 mg/kg prednisone, with progressive resolution of skin lesions over 10 days. Patient 3 was a 53-year-old woman with no underlying health conditions or history of autoimmunity. Purpura developed 6 days following the first dose, affecting the lower and upper limbs. She received treatment with 1 mg/kg prednisone, which led to a progressive resolution of skin lesions over 2 weeks.

All cases were investigated for laboratory abnormalities or organ involvements that are typically associated with small-vessel vasculitis. However, laboratory tests showed only non-specific increases in erythrocyte sedimentation rate and C-reactive protein (CRP); anti-neutrophil cytoplasmic antibodies, cytoplasmic anti-neutrophil cytoplasmic antibodies, perinuclear anti-neutrophil cytoplasmic antibodies, rheumatoid factor, cryoglobulins, antinuclear antibodies, anti-DNA, C3, C4, IgA, and serology for hepatitis B virus and hepatitis C virus were negative or normal. Chest imaging (ie, x-ray or CT), urinalysis, and a search for stool blood were also negative. A 5 mm skin punch biopsy was performed in patient 3, which showed only a mild lymphocytic perivascular infiltrate (appendix p 3). A histological diagnosis of leukocytoclastic vasculitis could not be formally confirmed in the absence of neutrophils, yet disruption of the vessel wall, or fibrinoid necrosis, the

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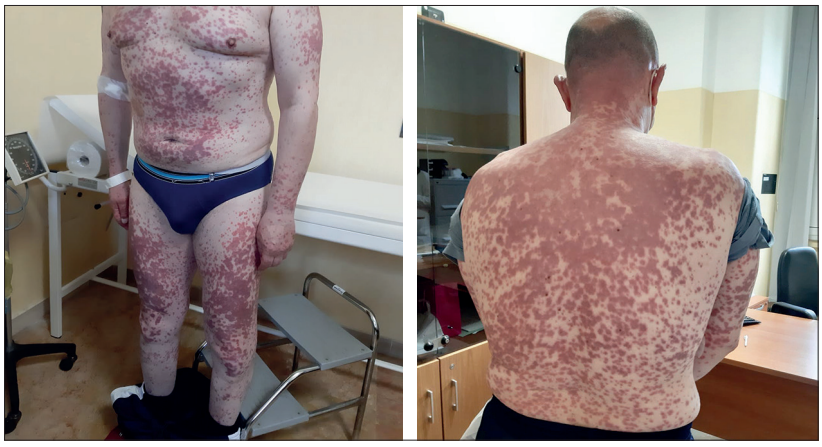

Figure: **Purpura in patient 1**