noticed at the biopsy site, corresponding to the area covered by the dressing plaster (Fig. 1c). Treatment with topical steroids improved the skin lesions, and after the Choreito was discontinued. the symptoms did not recur.

Although the pathomechanisms underlying GCD are unknown, certain cytokines are likely to be involved.[1,2] Histopathological examination of this case showed neutrophilic and eosinophilic infiltration. Neutrophils release inflammatory cytokines, including tumour necrosis factor-α, and interleukin (IL)-1, IL-6 and IL-7.[5] Furthermore, epidermal keratinocytes, stimulated by inflammatory cytokines, are reported to produce C3.[1] Therefore, a possible pathogenesis of GCD is the cascade of keratinocyte stimulation by neutrophil-derived cytokines and overproduction of C3. In our case, a systemic drug allergy or mechanical stress from skin biopsy might have increased inflammatory cytokines, followed by triggering granular C3 deposition at the BMZ. Further studies are required to unravel the pathogenesis of GCD.

This case highlights Choreito as a causative agent for GCD, supported by a positive DLST. Cessation of the drug resulted in remission. GCD may be associated with KP as demonstrated in our patient, which is observed as the emergence of new skin lesions at the site of pre-existing skin diseases, as seen in psoriasis, vitiligo, lichen planus and pemphigus.[3] We are not aware of other reports of KP in GCD.

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Linear IgA bullous dermatosis following Oxford AstraZeneca COVID-19 vaccine

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Dear Editor,

Linear IgA bullous dermatosis (LABD) is a subepithelial vesiculobullous disease, characterized by linear deposition of IgA along the basement membrane zone of the skin and/or mucosae.[1] Most cases of LABD are idiopathic,[1] but drugs, infections and malignancies have all been implicated as possible inductors.[2] To date, only two cases of LABD following vaccination have been reported, one after influenza[3] and one after human papillomavirus[3] vaccine administration. We describe the first case of LABD developing 3 days after the second dose of Oxford AstraZeneca COVID-19 vaccine in an adult patient, suggesting a possible causal association.

A 61-year-old man presented with a 22-day history of a bullous eruption, which had first appeared 3 days after his second dose of Oxford AstraZeneca COVID-19 vaccine. He reported no symptoms of infection, medication intake or sources of stress in the previous weeks.

Physical examination revealed tense blisters with serous content on a background of erythematous, urticarial and purpuric skin on his legs, and target-like lesions on his abdomen, trunk and thighs (Fig. 1). The oral and genital mucosae were also involved.

Histopathological examination revealed a subepidermal split with an inflammatory infiltrate composed of lymphocytes, histiocytes and some eosinophilic polynuclear lymphocytes. Direct immunofluorescence demonstrated predominant linear IgA deposition at the dermoepidermal junction (Fig. 2), while indirect immunofluorescence revealed antibasement membrane zone IgA antibodies binding to the roof of salt-split skin.

Laboratory examination revealed eosinophilia (1450 cells/μL; normal < 500/μL). ELISA results for anti-desmoglein (Dsg)1 and Dsg3, and for bullous pemphigoid (BP)180 antibodies were negative.

A diagnosis of LABD was made. The patient was treated with oral prednisolone, with marked clinical improvement and normalization of polynuclear eosinophil count.

There are multiple reports in the literature on drugs that cause LABD, particularly vancomycin.[2] Drug-

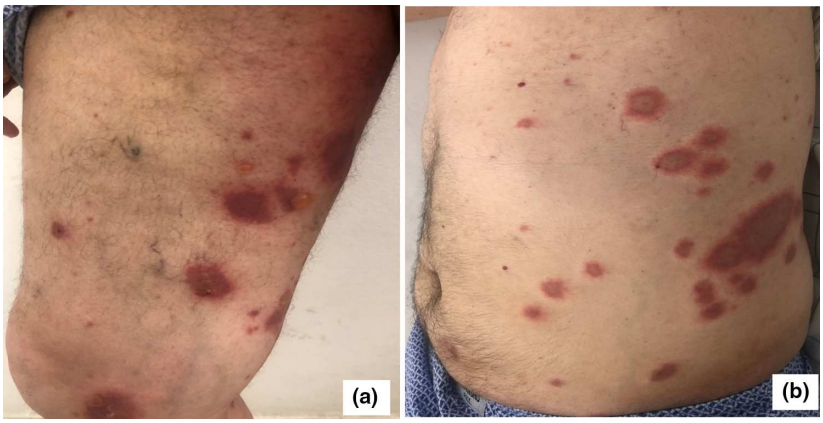

Figure 1 (a) Tense blisters on a background of urticarial skin; (b) multiple target-like lesions on the abdomen and trunk.

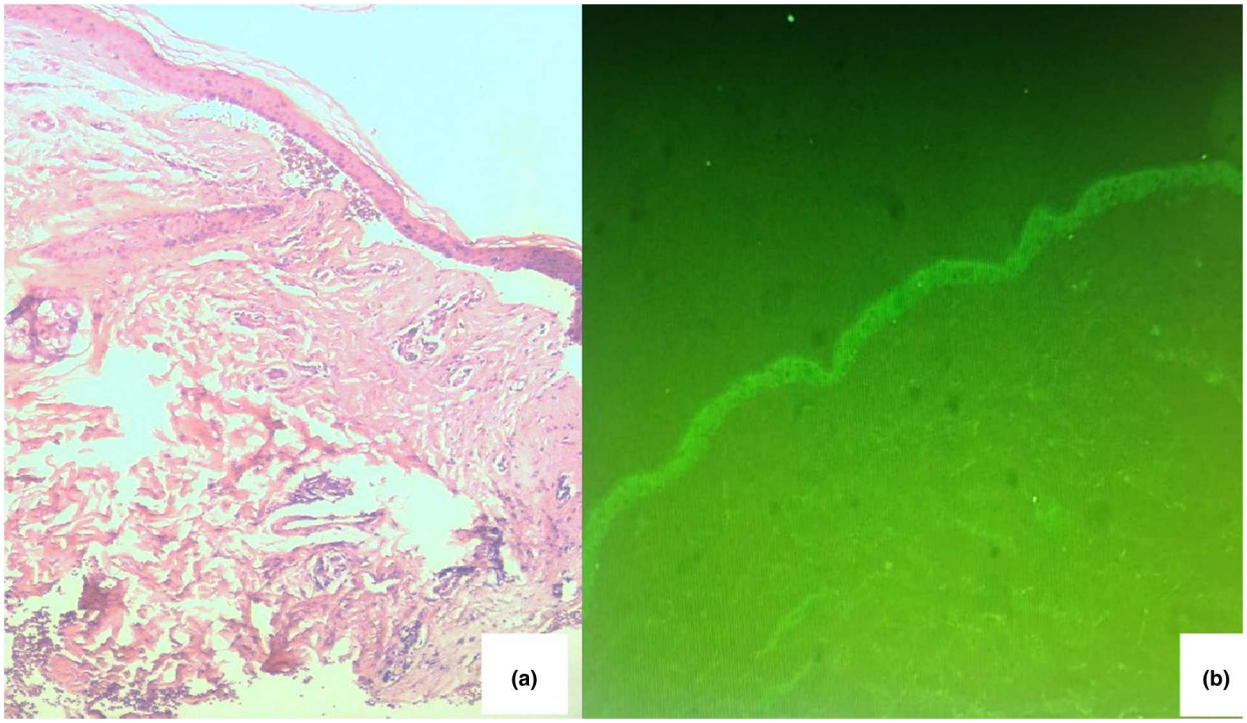

Figure 2 (a) Deep junctional detachment with bulla (haematoxylin and eosin, original magnification × 40); (b) direct immunofluorescence showed linear IgA deposits along the basement membrane zone (original magnification × 20).

induced LABD is defined by its polymorphic clinical features, which are more severe than in spontaneous forms.

A large range of cutaneous adverse events after COVID-19 vaccine is being reported as the vaccination programmes widen. Regarding bullous dermatoses, Coto-Segura *et al.* reported three cases of BP and one case of LABD induced by the BioNTech/Pfizer mRNA COVID-19 vaccine.[4] Tomayko *et al.* reported 12 cases of subepidermal blistering eruptions, including BP, following COVID-19 vaccination.[5]

Among the possible explanations for LABD developing after vaccination is molecular mimicry, where a viral antigen shares sequence or structural similarity with a host antigen. Another is direct or indirect activation of the host's immune system by viral antigens or cytokines, such as interleukins and transforming growth factor-β, which increase IgA synthesis.[3]

To our knowledge, this is the first case of LABD after Oxford AstraZeneca COVID-19 vaccine (adenoviral vector vaccine). It is possible, given the absence of triggers, that

the condition was spontaneous but considering the temporal association between COVID-19 vaccination and the development of the eruption, we suggest that immunization was the most probable trigger. Vaccination is a rare trigger for this condition. While this case may be a simple coincidence, it is worth keeping in mind that COVID-19 vaccination could induce immune-mediated bullous disease in susceptible people.

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Scurvy in a patient with thalassaemia

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Dear Editor,

A 23-year-old man presented with a 2-month history of intermittent gum bleeds and purpuric lesions over his arms and legs. He had pain in the knee and ankle joints of both legs, causing difficulty in walking. He had been diagnosed with beta-thalassaemia major at the age of 5 months, which was treated with monthly packed red cell transfusions. He was also on irregular treatment with iron chelation therapy. He had no concomitant psychiatric illness or history of drug abuse, smoking or alcohol intake. His dietary history revealed no intake of fruit and vegetables.

Physical examination revealed scattered perifollicular haemorrhagic and hyperkeratotic papules over the arms, back, buttocks and legs, and large confluent ecchymoses on both lower legs (Fig. 1a,b) and posterior thigh of both legs. Oral examination revealed multiple discrete areas of punctate haemorrhages over the oral mucosa with gingivitis (Fig. 1c), which bled on touch.

Laboratory investigations for erythrocyte sedimentation rate, levels of C-reactive protein, cryoglobulins, complement rheumatoid factor, antiphospholipid antibodies, anticitrullinated peptide antibodies, antinuclear antibodies and antineutrophil cytoplasmic antibodies, and hepatitis B and C and HIV infections, were within normal limits. The patient had anaemia (haemoglobin 9.2 g/dL; reference range 13.5–17.5 g/dL) and high ferritin (7885 μg/mL; 15–300 μg/mL). The clinical findings of gum bleeding and purpura on background of thalassaemia major raised the suspicion of scurvy. Serum vitamin C level was found to be very low (< 0.1 mg/dL; 0.2–2.0 mg/dL), confirming scurvy.

Scurvy is a manifestation of vitamin C deficiency, which leads to impaired collagen synthesis and capillary fragility, resulting in perivascular oedema and red cell extravasations. Clinical features include generalized weakness and fatigue, hyperkeratosis of hair follicles, perifollicular haemorrhages, petechiae and ecchymoses, gingivitis with gum bleeding, delayed wound healing and easy fractures in bones.[1] Musculoskeletal manifestations including arthralgias, particularly in the knees, ankles and wrists, as well as myalgias, are seen in up to 80% of patients. Haemarthroses are also commonly seen in hips, knees and ankles and may be the first reason for seeking medical attenton.[2]

Vitamin C deficiency can be easily diagnosed with proper dietary history, history of pre-existing illness, with typical examination findings. In this case, the patient had iron overload, which led to non-transferrin-bound iron, inducing lipid peroxidation with subsequent depletion of vitamin C, which resulted in scurvy.[3] The prognosis of scurvy is excellent with a dramatic response to vitamin C therapy; usually, patients become asymptomatic by 3–5 days with resolution of physical findings by 1–2 weeks.[4] In patients with thalassaemia, a well-balanced diet that includes citrus fruits eliminates the need for routine supplementation of vitamin C. We started our patient on supplementation with oral vitamin C 500 mg twice daily, resulting in dramatic improvement in gingivitis and purpura, with resolution of joint symptoms within 1 week. Vitamin C supplementation was continued for a month, along with a vitamin C-rich diet and regular iron chelation therapy. There was no recurrence of symptoms at follow-up after 6 months.