(Fig. 1e). There was no posterior enhancement or increased vascularity. The thickness of the hypoechoic area decreased under compression by the US transducer. The post-treatment US scan of the same area showed that the thickened area had reduced to the same thickness (0.8 mm) as the nonlesional area, and there was no detectable hypoechoic material in the dermis (Fig. 1f).

US has become a helpful diagnostic technique to assess the activity of dermatological diseases.[1] In addition to increased dermal thickness, the US scan revealed a partially compressible hypoechoic area located in the upper to mid-dermis, which we speculate is associated with the mucin deposition seen by histopathology. The post-treatment US changes reflected the clinical improvement and resolution of mucin deposition.

The clinical manifestation of LLM should be differentiated from that of other mucin deposition or sclerotic disorders, and US may assist in the differential diagnosis. For example, the US findings of scleroedema include thickening dermis, dermal echogenic spots and a hypoechoic area in lower dermis,[2] whereas pretibial myxoedema shows increased skin thickness, hypoechogenic deposition in the mid to lower dermis, and a blurred boundary between the dermis and subcutis.[3] The hypoechoic areas seen in US scans of dermal mucinoses correlate with the location of mucin deposition, helping to differentiate LLM from other dermal mucinoses. US of acute-phase morphoea reveals increased dermal thickness and decreased whole-layer dermal echogenicity, but increased subcutaneous echogenicity, increased vascularity, and a blurred border between the dermis and hypodermis.[4] The hypoechoic areas are associated with mobile mucin and compact fibrosis in LLM and morphoea, respectively. Therefore, the hypoechoic area is compressible in LLM but not compressible in morphoea. US can provide another option to assist diagnosis in cases where skin biopsy is not possible, or to assess the effects of therapy for LLM.[1]

In conclusion, we present a case of LLM with its US findings. US can be a useful noninvasive tool for assisting in the differential diagnosis and monitoring of the therapeutic response of LLM.

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COVID-19 vaccine-induced Stevens–Johnson syndrome

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Dear Editor,

Stevens–Johnson syndrome (SJS) is a severe cutaneous adverse drug reaction. There is scant information about its occurrence following vaccine.[1] We report a case of SJS induced by a COVID-19 vaccine in an adult.

A 60-year-old man presented with complaints of fever, oral ulceration and skin rash, which had started 3 days after he had received his first dose of the recombinant ChAdOx1 nCoV-19 (Covishield, which is a patent product of AstraZeneca, manufactured by Serum Institute of India) COVID-19 vaccine. He had consulted a local physician who had prescribed paracetamol and levocetrizine; however, the symptoms were not controlled and gradually the rashes became generalized in distribution. The patient presented to the emergency department 3 days following the first onset of the lesions (10 days after the vaccine administration) throughout which period the fever had persisted.

Physical examination revealed multiple purpuric macules present all over the body with perilesional erythema. The lesions had coalesced to form large sheets of necrosed skin over the front and back of the patient's trunk, with a few areas showing bullae. Mucosal involvement was present in the form of oral erosions, haemorrhagic crusting over the lips, redness of and slight discharge from the eyes, and erosions on the glans (Fig. 1).

Based on the disease course and morphology, SJS was suspected and a detailed drug history was elicited, which revealed that for the past 6 months the patient had been taking teneligliptin and metformin for diabetes and amlodipine for hypertension. His other medications had been

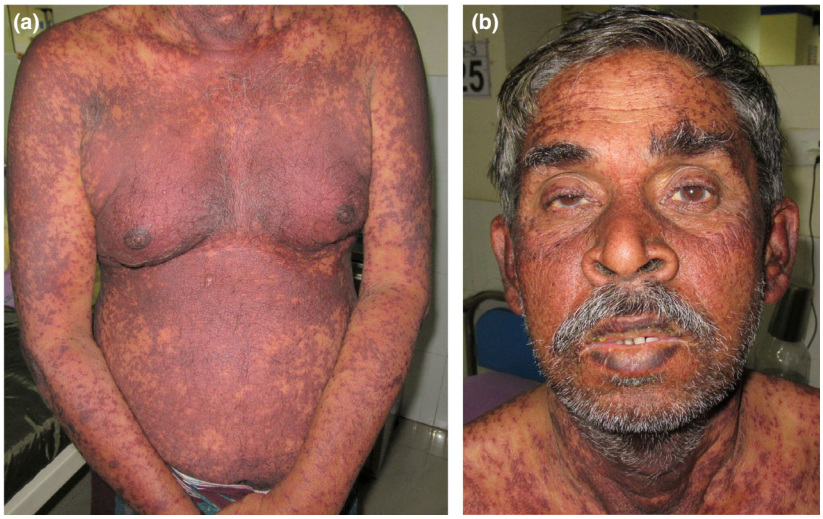

Figure 1 (a) Large sheets of necrosed skin in front of trunk, with a few areas showing bullae; (b) involvement of the face with erosions in palpebral conjunctiva and necrotic crusting over lips.

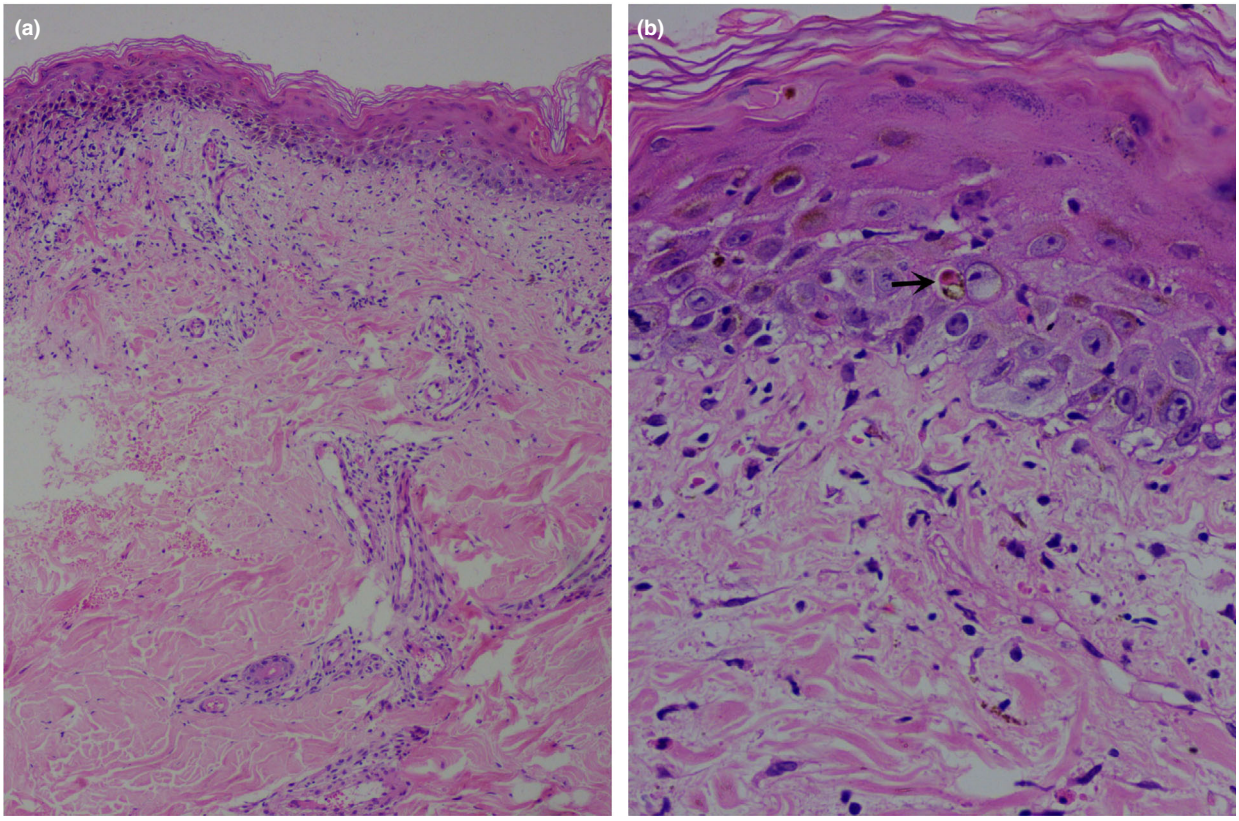

Figure 2 (a) Orthokeratosis with epidermal atrophy, scattered degenerated apoptotic keratinocytes, patchy areas of basal cell degeneration and interface dermatitis, perivascular and periadnexal inflammatory cell infiltrate, and extravasation of erythrocytes in the dermis; (b) apoptotic keratinocytes (arrow), upper dermal oedema and extravasation of erythrocytes. Haematoxylin and eosin, original magnification (a) × 50; (b) × 400.

prescribed after he had developed fever and skin rash, and he denied any other drug intake before the development of his symptoms. The SCORE of Toxic Epidermal Necrosis was 1 on the day of admission, and the Naranjo algorithm revealed a causal association of 2 (possible association) between the vaccine and the adverse drug reaction.

Histopathological examination from the erythematous lesion revealed orthokeratosis with epidermal atrophy, moderate intraepidermal infiltration of lymphocytes and neutrophils with moderate spongiosis, scattered degenerated apoptotic keratinocytes, patchy areas of basal cell degeneration and interface dermatitis, perivascular and periadnexal inflammatory cell infiltrate, and extravasation of erythrocytes in the dermis (Fig. 2).

The diagnosis of SJS was thus confirmed and the patient was started on oral ciclosporin 300 mg, which led to complete resolution after 7 days (Fig. S1). The patient was issued a drug card and advised to defer the second dose of vaccine.

Diagnosis of SJS is made on the basis of clinical suspicion and histological findings. In this case, suspicion of SJS was based on the sudden appearance of erythematous, reticulate patches on the skin, the mucosal ulceration and the constitutional symptoms. The diagnosis was confirmed by the presence of epidermal keratinocyte necrosis. Chahal *et al.* adopted a similar diagnostic approach to SJS, which included clinical findings, corroborative history and histopathological findings.[1] The Naranjo algorithm score is widely used for assessing causal association in drug reaction.[2] Our patient was known to have diabetes and hypertension, and was on teneligliptin, metformin and amlodipine. He had taken the antihypertensive drug and received the vaccine prior to development of SJS but the continued intake of the antihypertensive drug did not aggravate the condition. The Naranjo scale score of 2 suggested possible association of vaccine in the development of SJS.

All COVID-19 vaccines have two components (virotopes and excipients) and both can cause severe drug reaction.[3] We believe that in our patient, it was the virotopes that caused the SJS, as we did not find severe delayed-type hypersensitivity to other vaccine ingredients. In a previous study,[1] Chahal *et al.* hypothesized that expression of the virotopes on the surface of keratinocytes leads to a CD8+ T-lymphocyte response against epidermal cells and causes apoptosis of keratinocytes and detachment at the dermoepidermal junction, leading to SJS in genetically susceptible individuals. This is further supported by the ability of the ChAdOx1 nCoV-19 vaccine to induce a T-cell-specific response, which is predominantly T helper 1-based, which may have induced an immune response with consequent keratinocyte cell damage.[4] Further, in an extensive review we did not find any evidence of any of the excipients (L-histidine, L-histidine hydrochloride, sucrose, sodium chloride, magnesium chloride, polysorbate 80, edetate disodium, ethanol and water) causing severe delayed type hypersensitivity reactions such as SJS.[3]

In conclusion, we report the first case, to our knowledge, of COVID-19 vaccine-induced SJS. This case illustrates an exceedingly rare complication of the vaccine. As the benefits far outweigh the risk of the vaccine in the current pandemic, such rare reactions should not deter people from receiving the vaccine.

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The patient provided written informed consent to publication of the case details and photographs.

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Supporting Information

Additional Supporting Information may be found in the online version of this article:
Figure S1 (a,b) Healed lesions after treatment.

Personal Health Records as a tool to support patient-initiated follow-up: a dermatology perspective

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Dear Editor,
One of the few positive outcomes of the COVID-19 pandemic has been an unprecedented flexibility and willingness to embrace innovation in the delivery of healthcare services in the UK National Health Service (NHS). This change in mindset has presented a unique opportunity to rethink and transform outdated models of delivering