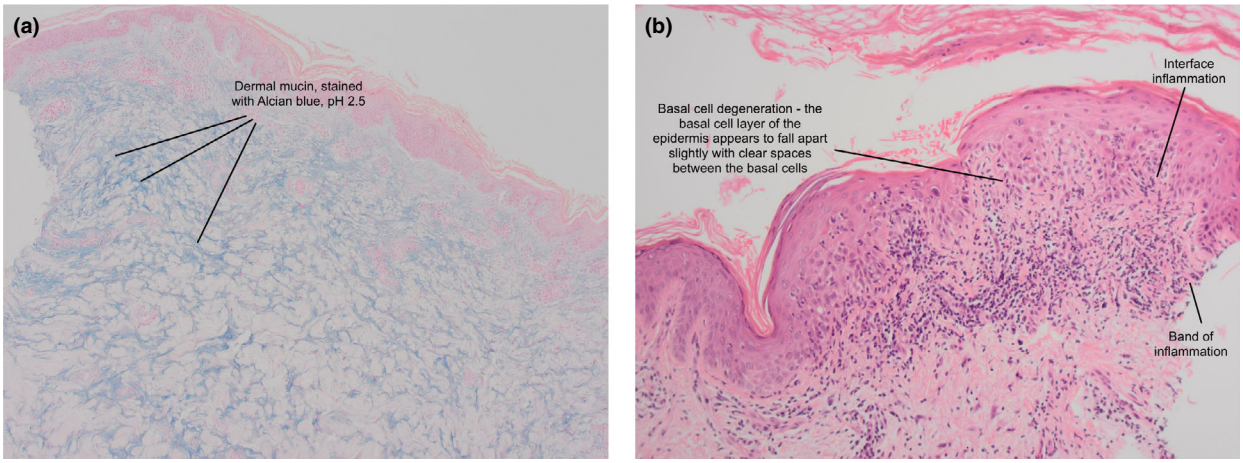

Figure 2 (a) Mucin with focal lichenoid inflammation (Alcian blue, original magnification × 40). (b) Lichenoid inflammatory infiltrate (haematoxylin and eosin, original magnification × 100).

nonresponsive to oral steroids and omission of a dose of brodalumab. Thin erythematous plaques were still present on the patient's chest. Brodalumab was stopped completely and the symptoms resolved following discontinuation.

Brodalumab, ixekizumab and secukinumab target the IL-17 pathway, with the former targeting directly at the IL-17A receptor and the latter two inhibiting IL-17A.[1] There are only three cases of secukinumab-induced LE reported in the literature.[2–4] To our knowledge, there is no report to date on SCLE associated with brodalumab.

A case report on ustekinumab-induced SCLE proposed the diversion of T-cell differentiation to T helper cell-22 production, ultimately promoting increased production of tumour necrosis factor (TNF)-α.[5] Increased production of TNF-α leading to translocation of anti-Ro/SSA and anti-La/SSB can result in deposition of immunoglobulins and complement proteins at the dermoepidermal junction. Cell-mediated keratinocyte cytotoxicity may cause SCLE.[5]

Upregulation of IL-17A may also mediate local inflammatory response in SCLE. A phase 2 clinical trial (NCT03866317) to establish its efficacy and safety of secukinumab in discoid LE is underway. The unexpected induction of SCLE with the brodalumab may be considered as a paradoxical reaction that has not been documented previously. The complexity of genetic predisposition and manipulation of immune-mediated inflammatory reactions in biological agents contributes to the advent of the specific class of adverse drug reaction.

Brodalumab can induce SCLE in a patient with psoriasis with no past history of LE. Discontinuation of the drug is advised especially in the event of symptomatic SCLE or development of systemic complication. Future research is warranted to establish a causal link between both.

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COVID-19-induced toxic epidermal necrolysis

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Cutaneous manifestations of COVID-19 are being increasingly reported in the literature, and include rashes with varying morphology including maculopapular, urticarial, chilblain-like, vesicular and livedoid/necrotic lesions.[1] To add to this compendium of associations with COVID-19,

we report a patient who developed toxic epidermal necrolysis (TEN) following a positive diagnosis of COVID-19. We believe this represents a causal association between COVID-19 and TEN.

A 53-year-old woman presented in early December 2020 with a 2-day history of a maculopapular rash. The patient had metastatic breast carcinoma and since October 2020, she had been an inpatient on the palliative care ward for management of symptoms related to her bone, brain and liver metastases. The rash was a non-blanching, mottled maculopapular rash predominantly affecting her chest, upper back and legs. The eruption had developed 5 days after she had received a positive PCR test for COVID-19 virus, following development of a new cough. There was no history of any new drugs or any other triggers. Based on this history, the initial diagnosis was that this rash was one of the cutaneous manifestations of COVID-19 virus. The patient was initiated on a potent topical corticosteroid and emollients and was also closely monitored to see if there was progression of the rash.

Ten days later, the patient developed detachment of the skin on her chest (Fig. 1a), with progression of the rash on the patient's back, arms, legs, scalp and ears (Fig. 1b). She developed erosions and haemorrhagic crusting of the mouth. The eyes and genitalia were spared. The epidermal detachment affected > 10% of the patient's surface area. The presentation was in keeping with TEN. The patient's drug history was reviewed again and there was no suggestion of any new medications. Her last chemotherapy cycle had ended 6 months earlier, she had been started on exemestane 5 months earlier, and she was due to start third-line chemotherapy, but this had been deferred due to her COVID-19 diagnosis. Her only other new medication was dexamethasone for her brain metastasis, which was continued. The SCORTEN score at the time of the diagnosis was 4 (age, malignancy, initial percentage of epidermal detachment, serum bicarbonate) indicating an expected mortality of > 50%.[2] She had punch biopsies taken from the chest for histology and immunofluorescence. Histology showed full thickness epidermal necrosis supporting the diagnosis of TEN. Immunofluorescence was negative.

Owing to her comorbidities, our patient was not transferred to the regional specialist centre, but was managed on the ward by the Dermatology and Palliative Care teams. It was challenging to strike a balance between meeting her palliative needs and the management of her TEN, and the expertise of the Palliative Care team was invaluable. The patient recovered slowly with conservative supportive management (Fig. 1c,d).

There have been many cutaneous manifestations of COVID-19 reported in the literature. A recent case report suggested possible hydroxychloroquine-induced TEN in a

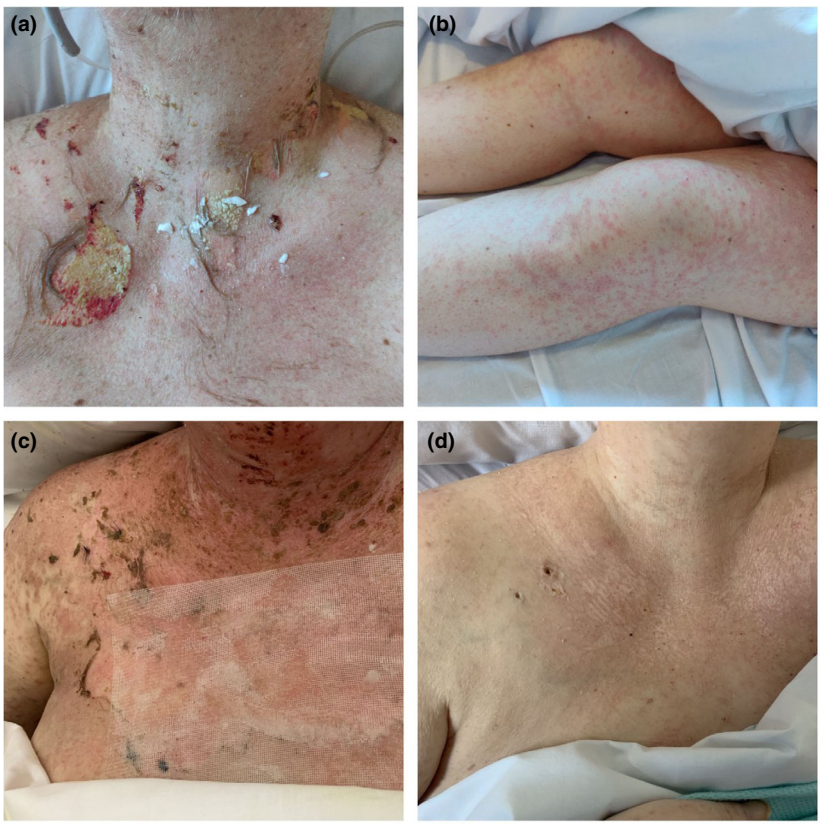

Figure 1 Progression of rash: (a) epidermal detachment of chest; (b) maculopapular rash on legs; (c) appearance of rash a week later; (d) full re-epithelialization.

patient with COVID-19, but this was not proven by histology.[3] To our knowledge, this is the first report of TEN secondary to COVID-19. Despite an initial SCORTEN score of 4, our patient improved on conservative therapy, which may indicate that TEN due to COVID-19 virus is less severe than drug-induced TEN.

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Macrodactyly in tuberous sclerosis complex

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A 19-year-old man presented with gradually progressive thickening of the fourth finger of his right hand, which was first noticed at 7 years of age. There was no pain or difficulty in moving the finger. The patient had been previously diagnosed with tuberous sclerosis complex (TSC).

On physical examination, the distal aspect of the fourth digit of the right hand appeared thicker than the other fingers. The overlying skin was loose and hyperpigmented, with a thickened velvety appearance and a doughy consistency. There was a mild flexion deformity at the distal interphalangeal joint. The fingernail showed distal horizontal splitting, while a smooth fleshy papule was noted emerging from the lateral aspect of the proximal nail fold, suggestive of a periungual fibroma (Fig. 1a, b). Radiograph of the bilateral hands showed cortical thickening and increased width of the middle phalanx of the right fourth digit, along with adjacent soft tissue thickening (Fig. 2a). Skin biopsy showed an acanthotic papillomatous epidermis, and mild thickening of the dermal collagen bundles along with ectatic vessels in the papillary dermis (Fig. 2b). In addition, the patient also had facial angiofibromas (Fig. 3), shagreen patches on his back, and multiple cortical tubers and calcified subependymal nodules detected by magnetic resonance imaging of the brain. Based on these features, a diagnosis of TSC associated with macrodactyly was made.

Macrodactyly is a rare, and perhaps under-recognized, manifestation of TSC.[1] It usually presents as a unilateral enlargement of one or few fingers, generally affecting the first, second or third digits. The overlying skin may appear unremarkable, or may be hyperpigmented and thickened as in our case. Histopathology shows thickened collagen bundles or fibrocollagenous tissue, suggestive of a collagenoma or fibrous hamartoma, respectively. Underlying bone changes are common, and include increase in bone width, periosteal reaction, irregular cortical thickening, erosive lesions and cortical cysts.[1] The mechanism for such a localized tissue hyperplasia is not completely understood, but somatic mosaicism for loss of

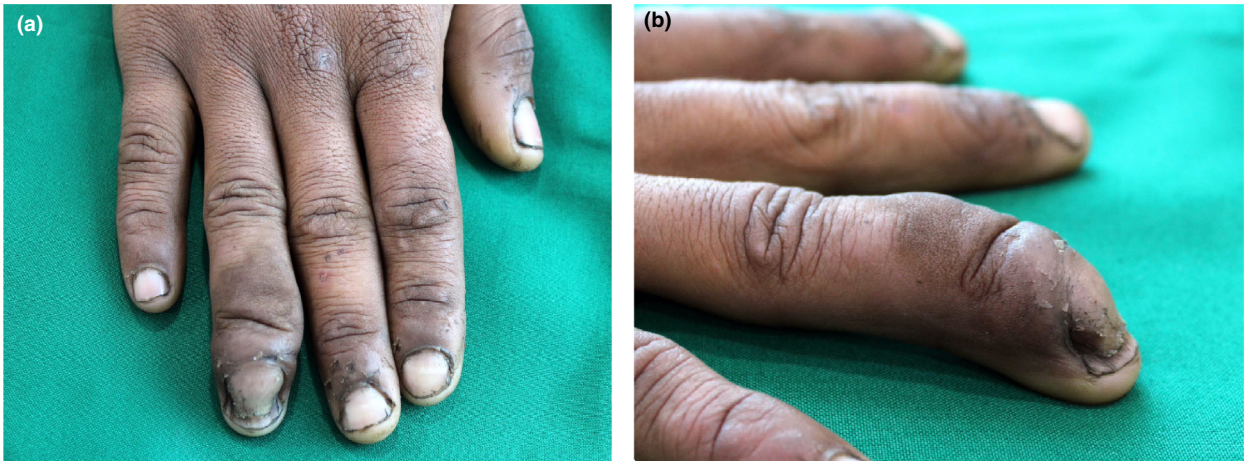

Figure 1 (a) Dorsal view showing loose hyperpigmented thickened skin of the right hand fourth digit. (b) Lateral view showing bulbous thickening of the distal finger with mild flexion deformity, and a periungual fibroma.