characterization of COVID-19 skin lesions is important to prompt suspicion of SARS-CoV-2 infection [9]. Thus, dermatologists at Harvard Medical School and Massachusetts General Hospital created the COVID-19 Dermatology Registry and, in collaboration with the American Academy of Dermatology (AAD) and International League of Dermatologic Societies (ILDS), invited health care workers globally to submit data for possible cutaneous manifestations of confirmed or suspected COVID-19 [10]. Freeman et al. [11] analysed this data aiming to characterize internationally representative cutaneous manifestations of confirmed/suspected SARS-CoV-2 infection and to gain insight into the pathophysiological course of COVID-19. The registry collected 716 cases of new-onset dermatological symptoms in patients with confirmed/suspected COVID-19. Of the 171 patients in the registry with laboratory-confirmed COVID-19, the most common morphologies were morbilliform (22%), pernio-like (18%), urticarial (16%), macular erythema (13%), vesicular (11%), papulosquamous (9.9%), and retiform purpura (6.4%). Pernio-like lesions were common in patients with mild disease, whereas fixed livedo racemosa, retiform purpura, and true acral ischaemia appeared in critically ill patients, corroborating others’ findings [12]. This suggests that thrombotic disease in critically ill patients with COVID-19 may extend to the skin in patients with livedo racemosa/retiform purpura/acro-ischaemia, with a morphology distinct from pernio [12]. Previously, Suchonwanit et al. [13] proposed that cutaneous manifestations of COVID-19 might present as two mechanistic patterns: clinical features similar to viral exanthemas and cutaneous eruptions secondary to COVID-19 systemic symptoms, especially vasculitis and thrombotic vasculopathy. Although this proposed dichotomy is a loose framework, it may provide a good starting point to consider further findings. Although the study by Freeman and colleagues [11] has obvious limitations (a case series, reliance on the providers - only half of whom were dermatologists - judgment in entering data, only 25% of patients with laboratory-confirmed disease), it highlights the wide variety of cutaneous manifestations of COVID-19 reported and should prompt further investigations.

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A recent nodule on the forehead

Cécile LESORT1, Kinda FATTOUH1, Denis JULLIEN1, Jean KANITAKIS1,2

1 Department of Dermatology, Edouard Herriot Hospital, Hospices Civils de Lyon, Lyon, France
2 Department of Pathology, Lyon Sud Hospital Centre, Pierre Bénite, France
jean.kanitakis@univ-lyon1.fr

A 69-year-old Caucasian man with a past history of systemic vasculitis treated with rituximab and cyclophosphamide, as well as several cutaneous keratinocytic carcinomas treated by surgical excision, presented with a cutaneous lesion on the forehead that had appeared one month prior to consultation and was increasing in size. Clinical examination revealed a 0.8-cm, well-defined, redd-brown keratotic nodule (figure 1A). Dermoscopy revealed

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Figure 1. A) An erythematous, pigmented and keratotic nodule on the forehead. B) Histological aspect showing a dermal-based tumour connected to the epidermis (haematoxylin-eosin stain; original magnification: ×40). C) Tumour cells are large and polygonal, with an eosinophilic or clear cytoplasm, several mitotic figures and significant nuclear atypia (haematoxylin-eosin stain; magnification: ×250). D) Diffuse expression of p53 by tumour cells (original magnification: x100).

...some atypical vessels. The lesion was surgically excised under local anaesthesia and submitted for microscopic examination with the clinical diagnosis of squamous cell carcinoma.

Histological examination showed an endophytic tumour with an ulcerated surface. This consisted of more or less atypical keratinocytes with hyperchromatic, often mitotic, nuclei and abundant eosinophilic cytoplasm, occasionally containing melanin deposits. The cells formed large tumour lobules which abutted the basal layer of the epidermis and invaded the upper and mid dermis (figure 1B-C). Within the tumour lobules, the keratinocytes often formed rounded nests showing features of pilar-keratinization. PAS staining was positive in the most differentiated proliferative cells with clear cytoplasm. On immunohistochemistry, the tumour cells demonstrated diffuse expression of the oncoprotein p53 (figure 1D) and focal, heterogeneous expression of epithelial membrane antigen; they did not express keratin 15 or CD34 antigen. These features were suggestive of trichilemmal carcinoma.

Trichilemmal carcinoma (TC), also known as tricholemmal carcinoma and trichilemmocarcinoma, is a rare cutaneous adnexal tumour first described by Headington in 1976 [1], and represents the malignant counterpart of trichilemmoma. Hamman et al. [2] reviewed, in 2014, all the 103 reported TC cases. TC develops mainly on sun-exposed skin in elderly patients, most often on the scalp, forehead and neck. It usually manifests as a single lesion and may be ulcerated, mimicking basal cell carcinoma, squamous cell carcinoma, keratoacanthoma or amelanotic melanoma. It can also present as an indurated erythematous plaque. Histological examination [3] shows intradermal cell proliferation, composed of multiple lobules connected to the epidermis with a pushing lower border. Tumour cells are polygonal, with abundant, eosinophilic, often clear, glycogen-rich cytoplasm. They form nests showing trichilemmal keratinization and a peripheral palisading pattern, indicating the follicular outer root sheath origin of the tumour (absence of granular layer). Cytologic atypia and high mitotic activity are frequent. Hyperkeratosis, parakeratosis, dyskeratotic cells and pagetoid intraepidermal spread are variably present. Actinic damage is generally associated.

Despite a malignant cytological appearance, the clinical course of TC is usually indolent. The recommended treatment is surgical excision with clear margins [4]. Recurrence has been reported, and was also treated with surgical excision. Rare cases of local lymph node or distant metastases have been reported [5]. Various chemotherapy agents have been used, such as cisplatin, 5-fluorouracil, vindesine, and cyclophosphamide, but the prognosis of metastatic TC is poor.

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Acute generalised exanthematous pustulosis secondary to prednisolone: an unlikely suspect

Sanju ARIANAYAGAM1, Eleni IEREMIA2, Stephanie ARNOLD1

1 Department of Dermatology, Churchill Hospital, Oxford, UK
2 Department of Cellular Pathology, John Radcliffe Hospital, Oxford, UK
sanju.ari@outlook.com

A 75-year-old female patient was referred to the urgent dermatology service by her general practitioner (GP). She described an erythematous rash which had first appeared five days prior. The rash initially developed on the neck and then spread to include the arms, chest, back and groin. She described some pruritus, although not as a prominent feature, and some coryzal symptoms, but no fever. Just over two weeks prior to the rash appearing, she had been prescribed prednisolone by her GP at a dose of 60 mg once daily for presumed giant cell arteritis. She had been referred to the rheumatology team for assessment, but had not yet been reviewed. Her past medical history included fibromyalgia, asthma and hay fever. Her regular medication included cetrizine, dosulepin, fluoxetine, gabapentin, omeprazole and a combined beclometasone/formoterol inhaler. She had no known drug allergies.

On examination, there was a widespread papular erythematous rash with superimposed superficial, fragile pustules, affecting the trunk, upper limbs and groin. The rash showed confluence over the chest and back (figures 1A-C). The legs and face were relatively spared on initial presentation. There was no mucosal involvement. She was aperyrexial and there was no lymphadenopathy.

The clinical differential included acute generalised exanthematous pustulosis (AGEP) and pustular psoriasis. Initial blood tests were unremarkable except for a neutrophilia of 20.74 × 10^9/L (normal: 4-11). The high dose of prednisolone likely contributed to this degree of neutrophilia, but a neutrophilia can also be seen in neutrophilic dermatoses such as AGEP. Although C-reactive protein was only 20 mg/L (normal <5) at initial presentation, this did peak at 200 mg/L on blood tests taken around two weeks after initial presentation. All regular medication was discontinued and the dose of prednisolone was reduced to 40 mg once daily, after discussion with the rheumatology team.

Skin biopsies from the back and thigh were taken at initial presentation and one week later, respectively. Figure 2A shows the biopsy taken from the thigh which revealed extensive subcorneal neutrophilic pustules with intraepidermal extension. There was subepidermal vesiculation due to papillary dermal oedema in association with extravasated red blood cells. A mild superficial perivascular inflammatory cell infiltrate, composed of lymphocytes and neutrophils, was present in the underlying dermis. Eosinophils were

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Figure 1. A-C) Clinical photographs taken at initial presentation showing a rash composed of pustules on an erythematous background.