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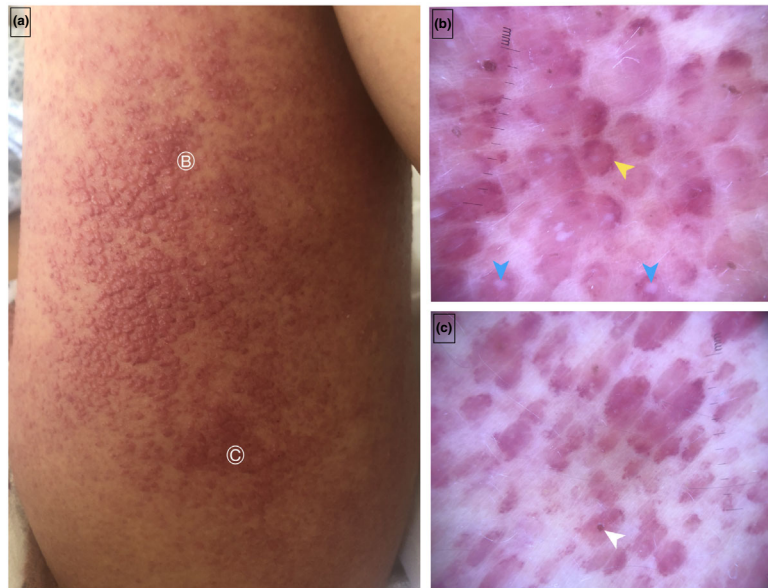
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## Papular-purpuric exanthem in a COVID-19 patient: clinical and dermoscopic description

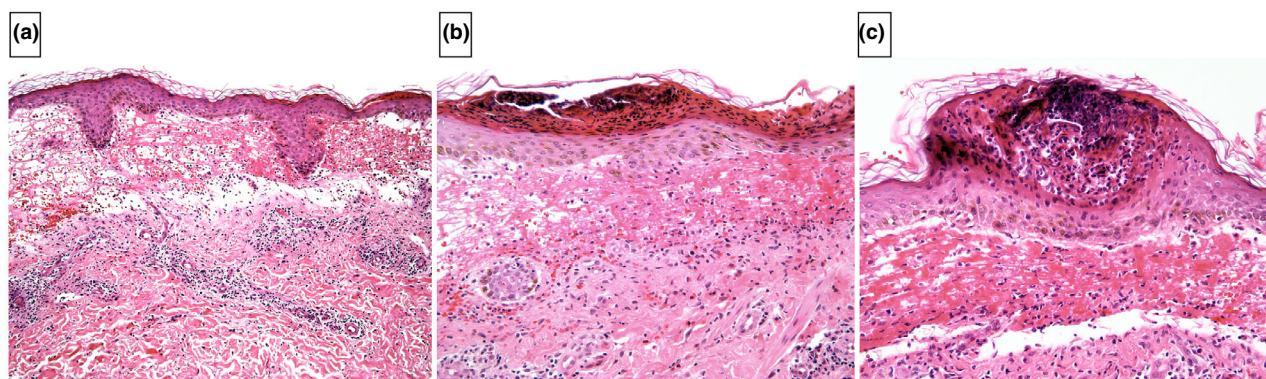
### Editor

Acute coronavirus disease 2019 (COVID-19) has become a global health concern associated with a broad spectrum of clinical presentations. Cutaneous lesions in COVID-19 are still under continuous study. We report a case of COVID-19 (+) patient with late-onset cutaneous rash associated with a systemic inflammatory response during the second hospitalization.

A 39-year-old woman with known COVID-19 exposure presented to the emergency department with a seven-day history of fatigue, fever, dry cough and shortness of breath. Physical examination revealed pulmonary bilateral basal crepitations. Laboratory tests showed high levels of C-reactive protein 6.4 mg/dL (reference range, 0.1–0.5 mg/dL) and D-dimer 604 ng/mL (reference range, 0–500 ng/mL). Nasopharyngeal swab for COVID-19 was positive (genesig<sup>®</sup> Real-Time PCR assay, Primerdesign, UK). Chest computed tomography (CT) showed peripheral ground-glass opacities in the lower zones of the lungs and mild opacities in the right upper lobe. The patient was hospitalized with the diagnosis of pneumonia due to COVID-19. She had a previous history – since adolescence – of autonomic dysfunction with recurrent episodes of hypotension. She was treated with



**Figure 1** Papular-purpuric exanthem on COVID-19(+) patient (second day of readmission). Erythematous-purpuric papular rash on patient's left thigh. Biopsies were taken on zones B and C (a). Dermoscopy shows multiple monomorphic papules with an incomplete violaceous rim at the periphery (yellow arrowhead), and a central yellow globule in some papules (blue arrowheads) (b). Other papules had a central purpuric globule with an erythematous background (c).



**Figure 2** Histopathologic findings. Histopathology shows acute oedema, extravasation of red blood cells and neutrophils in the papillary dermis, a perivascular mononuclear infiltrate around superficial plexus, endothelial swelling (a,b, H&E 100 $\times$ ) and a subcorneal pustule (c, H&E 20 $\times$ ).

rivaroxaban, acetaminophen, ipratropium bromide and fenoterol hydrobromide. On day 9 of the first hospitalization, the C-reactive protein level diminished to 2 mg/dL, dysautonomia was controlled, and later she was discharged.

After 10 days, the patient was re-hospitalized due to hypotension episodes with intense malaise and myalgia. During the second hospitalization, both C-reactive protein (6.3 mg/dL) and D-dimer (3602 ng/mL) showed very high levels. A computed tomography pulmonary angiography (CTPA) ruled out a pulmonary embolism. On the second day of rehospitalization, the patient developed an acute symmetric purpuric rash on the buttocks, thighs and axillae characterized by petechiae and multiple erythematous-purpuric papules, forming well-defined plaques (Fig. 1a). Following strict sanitary measures, we obtained dermoscopic images (DermLite<sup>®</sup> DL4, 3gen, USA). Under dermoscopy (10 $\times$  magnification), multiple monomorphic papules with an incomplete violaceous rim at the periphery and a central yellow globule were found (Fig. 1b). Other papules had a central purpuric globule with an erythematous background (Fig. 1c). The skin biopsy of the papules in zone 'b' showed mild spongiosis, subcorneal micropustules, focal exocytosis of neutrophils, marked oedema of the papillary dermis, red cells extravasation, perivascular and interstitial lymphocytic inflammatory infiltrate and endothelial swelling (Fig. 2). No signs of vasculitis were observed, and direct immunofluorescence was negative. The patient received supportive therapy and high-potency topical corticosteroids for the rash. After six days of treatment, D-dimer (581 ng/mL) and C-reactive protein level (0.5 mg/dL) diminished to almost normal levels, and skin lesions disappeared.

Recent studies<sup>1,2,3</sup> have described various cutaneous patterns in COVID-19 patients; pseudo-chilblain, vesicular, urticarial, maculopapular and livedo/necrotic. Petechial rash has also been associated with COVID-19 infection<sup>4</sup>. In our

patient, we have described a papular-purpuric pattern. High levels of D-dimer and C-reactive protein were associated with this papular-purpuric exanthem. Clinically, we found papules with a central micropustule that under dermoscopy was seen as a yellow globule. Other papules had a homogenous red-violaceous colour that under dermoscopy was characterized by an incomplete dark red-violaceous rim at the edge of the papule. D-dimer and C-reactive protein levels had a direct correlation with skin lesions. During the acute phase of the exanthem, characterized by red papules with dermoscopic signs of epidermal and dermal damage, both levels were high. During the fading period, these markers diminished along with the rash. To our knowledge, this is the first published observation of a papular-purpuric rash using dermoscopy in a COVID-19 patient.

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### Conflict of interest

The authors state no conflict of interest for this work.

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J. Larrondo,<sup>1,2,\*</sup>  R. Cabrera,<sup>1</sup> M. Gosch,<sup>2</sup> F. Larrondo,<sup>3</sup> M. Aylwin,<sup>4</sup> A. Castro<sup>5</sup>

<sup>1</sup>Department of Dermatology, Clínica Alemana-Universidad del Desarrollo, Santiago, Chile, <sup>2</sup>Department of Dermatology, Hospital Del Salvador, Santiago, Chile, <sup>3</sup>Department of Cardiology, Clínica Alemana-Universidad del Desarrollo, Santiago, Chile, <sup>4</sup>Department of Infectology, Clínica Alemana-Universidad del Desarrollo, Santiago, Chile, <sup>5</sup>Department of Pathology, Clínica Alemana Universidad del Desarrollo, Santiago, Chile

\*Correspondence: J. Larrondo. E-mail: jlarrondo@alemana.cl

This work was conducted at the Clínica Alemana, Santiago, Chile.

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## Histological findings in chilblain lupus-like COVID lesions: in search of an answer to understand their aetiology

To the Editor,

We read with great interest the article by Piccolo *et al.*<sup>1</sup> describing chilblain-like lesions (CLL) on feet and hands during the COVID-19 pandemic. They mention the rate of association to autoimmune conditions was very low, which led to exclude a note autoimmune disorder as main cause of CLL. Here, we hypothesize the possible relationship between the development of these lesions and immune-chained phenomena following viral infection in a certain group of patients.

Although perniosis is a frequent phenomenon, it seems reasonable to establish a causal relationship between these lesions and the coronavirus given the significant increase in these lesions in the epidemiological context that we are living in.<sup>1,2</sup>

Kolivras *et al.*<sup>3</sup> have recently described the histological manifestations in a 23-year-old caucasian male with pernicious lesions and a confirmed rt-PCR positive for COVID-19 without other analytical alterations.

We reaffirm the similarity between these lesions and those found in lupus chilblain. We present another case of an 17-year-old male, caregiver of a patient convalescing from COVID pneumonia, who presented acral lesions of 2 days' evolution compatible with the acromanifestations described (Fig. 1). Blood analysis revealed an elevation of IgA. Antinuclear antibodies were negative, and cryoglobulins were not detected. Rt-PCR for COVID resulted negative and serologies showed positive IgG with negative IgM. CBC showed no cytopenias and haemostasis, including D-dimer, was normal. A 4 mm punch biopsy was performed on one of the lesions (Fig. 2a–d), where marked

hydropic degeneration of the basal layer was observed with isolates necrotic keratinocyte. In the papillary and reticular dermis, a moderate lymphocyte infiltration was observed around the vessels as sleeves. The endothelium was conspicuously prominent, without visualizing fibrinoid necrosis. A dense perieccrine infiltration was also evident. Immunohistochemistry with CD 123 resulted positive, notably around vessels and sweat glands. No direct immunofluorescence was performed.

The similarity of these acral lesions with chilblain lupus lesions in this population group, mostly young patients, may be due to the type of immune response triggered by the interaction of COVID-19 with the immune system of these individuals. IFN type I levels are known to correlate with age.<sup>4</sup> Infection of COVID-19 (as well as other viruses such as respiratory syncytial virus) would produce, in paediatric patients, an IFN-mediated response that, when induced prematurely, would control the viral infection. However, in adults, SARS-Cov-2 infection would produce a mute of interferon pathway regulatory genes (ISG) which prevents successful inhibition of viral spread.<sup>4</sup>

The understanding of the molecular basis of innate immunity has led to identification of IFNs as a central mediator in the pathogenesis of systemic lupus erythematosus (SLE).<sup>5</sup> Type I



**Figure 1** Periungueal erythema in second and third finger toe.