

moisturize in between. When prolonged glove wearing could not be avoided, we recommended to wear cotton gloves underneath their protective gloves. In case of no visible dirt, we advised to use an alcohol-based hand rub instead of soap to reduce the handwashing frequency.

After contacting various pharmaceutical companies, we were provided with multiple hand cream samples to help all hospital personnel with their basic skincare.

### Electronic consultations

When the educational information and basic skincare measures were not enough, we also provided an email account where hospital personnel could send photographs of their hands and describe their symptoms. In these cases, we either provided additional information and advice, or invited them for a consultation at our outpatient clinic.

### Outpatient clinic consultations

The next step in the treatment of hand eczema is a corticosteroid cream/ointment. In co-operation with our hospital pharmacy, we arranged a stock of corticosteroid ointments (betamethasone ointment) to be directly available at our department. This resulted in the fast treatment for our colleagues and prevented them from having to wait for GP consultation and pharmacy service.

The first consultations started on 30 March 2020. In the first two weeks, we received around 20 emails and seven co-workers visited our department. During the consultations, we performed physical examination of the hands and collected additional information. A summary of patients' characteristics can be found in Table 1. It should be noted that all of them had an atopic constitution, making them more prone to developing (hand) eczema, and a group in specific need of support. Symptoms included erythema, vesicles, itching, pain, rhagades, papules, desquamation and bleeding. During physical examination, we found severity scores of almost clear to moderate (this was after initial improvement with the above-mentioned steps). All of the seven co-workers received treatment with betamethasone ointment. Currently, we are still actively providing these consultations.

We hope that our experiences inspire more dermatologists to actively help their colleagues with hand eczema and support them in the battle against COVID-19.

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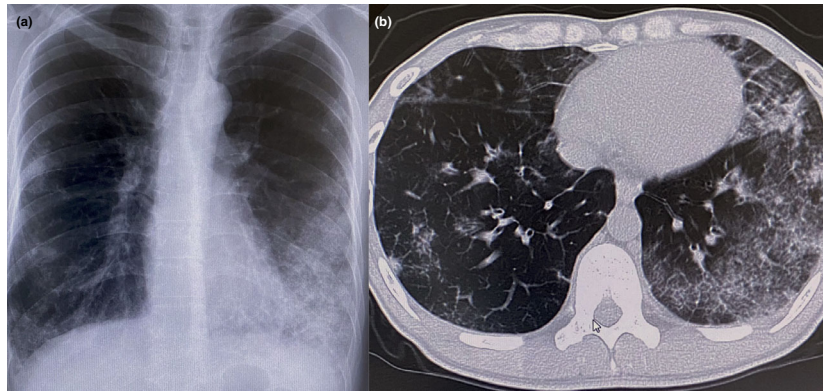
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## Eosinophilic granulomatosis with polyangiitis mimicking coronavirus disease 2019: a case report

Dear Editor,

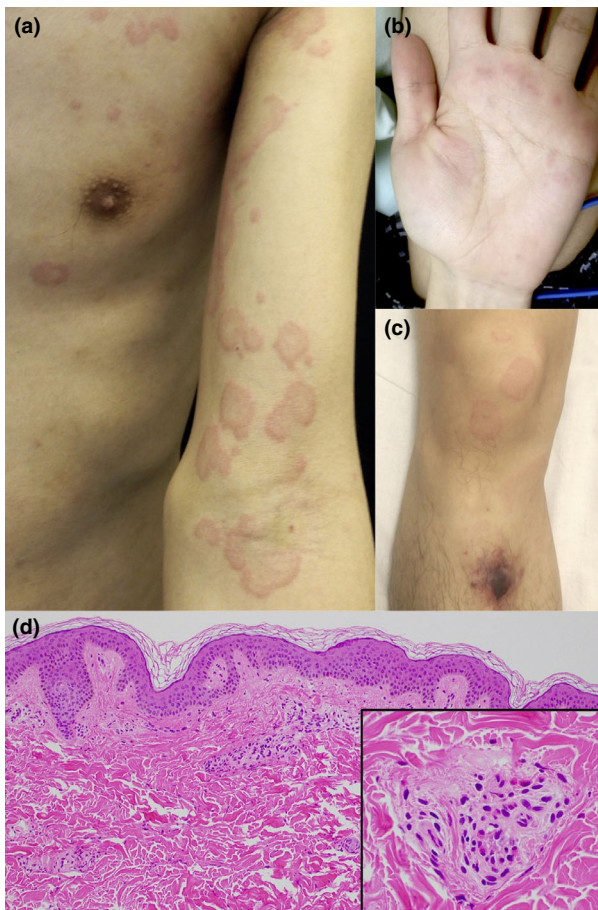
Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, is currently spreading worldwide, causing the worst pandemic experienced this century.<sup>1</sup> During the present outbreak, reports have been accumulating that various types of cutaneous manifestations were observed in COVID-19 patients.<sup>2–7</sup> We read with interest the recent article by Amatore *et al.*<sup>3</sup> describing a COVID-19 case who presented with a febrile rash consisting of annular, polycyclic and circinate erythema, presumably specific to COVID-19. Recently, we experienced a case of polycyclic erythema, which was very similar to theirs, in a patient with respiratory distress whose eosinophilic granulomatosis with polyangiitis (EAGP) was later confirmed by skin biopsy.

A 30-year-old Chinese male had a high fever (>38°C) with respiratory distress on a flight to Japan, where he worked, and was referred to us. Laboratory examination revealed elevated white ( $9.2\text{--}10.5 \times 10^4$  cells/ $\mu\text{L}$ , normal range  $3.3\text{--}8.6 \times 10^4$  cells/ $\mu\text{L}$ ) and red ( $615\text{--}692 \times 10^4$  cells/ $\mu\text{L}$ , normal range  $435\text{--}555 \times 10^4$  cells/ $\mu\text{L}$ ) blood cell counts, eosinophilia (15–28%, normal range <5%), a decreased platelet count ( $2\text{--}7 \times 10^4$  cells/ $\mu\text{L}$ , normal range  $15\text{--}42 \times 10^4$  cells/ $\mu\text{L}$ ) and high levels of D-dimers ( $10\text{--}22 \mu\text{g/mL}$ , normal range <0.1  $\mu\text{g/mL}$ ), fibrin-fibrinogen degradation products ( $6.3\text{--}35.3 \mu\text{g/mL}$ , normal range <5.0  $\mu\text{g/mL}$ ) and C-reactive protein ( $3.05\text{--}4.59 \mu\text{g/dL}$ , normal range <0.14  $\mu\text{g/dL}$ ). His high red blood cell count



**Figure 1** Chest roentgenogram (a) and computed tomogram (b) showing bilateral interstitial shadows.

had been clinically observed previously, without treatment. Neither anti-neutrophil cytoplasmic antibody nor anti-nuclear antibody was detected. The chest computed tomogram



**Figure 2** Urticarial and circinate erythema of the patient's arm (a), palm (b) and leg (c). Histologic examination showing perivascular infiltrates with lymphocytes and eosinophils (d and inset).

revealed bilateral interstitial shadows (Fig. 1), like those frequently seen in COVID-19 cases, so a pulmonologist segregated him on suspicion of COVID-19. During treatment with 6 g/day of intravenous ceftriaxone, erythema suddenly developed. Multiple circinate and annular erythema with slight pruritus were observed on his body, arms (Fig. 2a), palms (Fig. 2b) and legs (Fig. 2c). Ecchymosis after bruising was found in his right leg (Fig. 2c). A skin biopsy specimen taken from the skin lesion on his right thigh revealed mild perivascular infiltrates with lymphocytes and eosinophils. Some vessels had markedly infiltrated eosinophils with erythrocyte extravasation (Fig. 2d and inset). Afterwards, RT-PCR tests on the patient's sputum and nasopharyngeal swabs were negative for SARS-CoV-2 RNA detection. Based on these laboratory and histological findings, we diagnosed the patient with EGPA. Steroid pulse therapy for three successive days promptly relieved all his symptoms, in parallel with the normalization of his chest radiogram and laboratory data. His treatment was then changed to oral administration of 50 mg/day prednisolone for 2 weeks, after which the prednisolone dose was gradually tapered to 25 mg/day. Although the eruption reappeared transiently, it was successfully treated with olopatadine hydrochloride. No relapse occurred.

Various clinical signs, laboratory parameters and imaging modalities are suggestive of COVID-19. Additionally, recent reports have implicated distinctive skin manifestations, such as chilblain-like erythema, livedoid eruptions,<sup>2,8</sup> morbilliform rash,<sup>9</sup> urticarial rash<sup>4</sup> and varicella-like rash,<sup>6</sup> in COVID-19. Here, we found bilateral interstitial pneumonia accompanied with a hypercoagulation state, which is a typical pulmonary manifestation of COVID-19. In addition, we also observed polycyclic erythema, manifesting very similarly to the COVID-19 case described by Amatore *et al.*<sup>3</sup> These findings might have supported a diagnosis of COVID-19. However, because eosinophilia and eosinophilic infiltrates in skin lesions are an exceptional feature of COVID-19,<sup>10</sup> EGPA was the more

appropriate diagnosis for our case. The patient's complete response to steroid therapy, which usually causes deterioration in COVID-19 cases, supports our diagnosis. It is important to be aware of the similar clinical manifestations between EGPA and COVID-19.

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This patient in this manuscript has given written informed consent to the publication of his case details.

### Conflict of interest

None.

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## Histology of skin lesions establishes that the vesicular rash associated with COVID-19 is not 'varicella-like'

Editor

Several articles recently reported a 'varicella-like' rash in patients with COVID-19.<sup>1,2</sup> We observed similar cases at our institution. However, although we agree that the clinical picture is original, we reject that 'varicella-like' denomination since clinical presentation, as well as some histologic features that we wish to report here for the first time, make it clearly different from varicella.

Three patients with a vesicular rash associated with COVID-19 (RT-PCR test on a nasopharyngeal swab specimen positive for SARS-CoV-2 ARN) were seen at our institution in April, 2020. A biopsy of a vesicle was performed in each. Multiple levels with H&E stain were done; the slides were reviewed independently by two pathologists; only concordant data were validated. A test for SARS-CoV-2 was performed on a vesicle in two patients, and a direct immunofluorescence test on perilesional skin in one.

The main features of the cases are reported in the Table 1. Clinical lesions invariably consisted in small, monomorphic vesicles of 2–3 mm diameter, often excoriated at their top; trunk, especially back, was constantly involved (Fig 1a). Itching was absent or light. Evolution was sometimes irregular, until resolution that occurred without scarring after 10–22 days.

Histology showed a similar pattern in the three cases, with a prominent non-ballooning acantholysis leading to the constitution of an intraepidermal unilocular vesicle, with in two patients a clear suprabasal location (Fig 1b). Eosinophilic dyskeratosis was also constant, with on occasion a striking 'pomegranate-like' aspect (Fig 1c). Features more suggestive of a viral infection were present once (Fig 1d). No vasculitis was seen. The direct immunofluorescence performed in one patient and the two SARS-CoV-2 PCR tests performed on vesicles were negative.

The rash that we observed was similar to that reported by Marzano<sup>2</sup>, and constituted a picture that we agree to be evocative of COVID-19. But, in addition, the histologic pattern of prominent acantholysis and dyskeratosis with constitution of an unilocular intraepidermal vesicle in a suprabasal location, reported here for the first time, contributed to delineate a unique entity. Indeed, this pattern is very different from what is seen in varicella, in which major nuclear atypia, large multinucleated cells, acantholysis secondary to ballooning degeneration, involvement of the epidermis basal layer and vasculitis are regularly seen. Other acantholytic disorder (autoimmune or familial pemphigus, Grover's transient acantholytic dermatosis) do share