Conflict of interest
None.

Funding sources
None.

N. Martínez Campayo,1,* J.I. Bugallo Sanz,2 I. Mosquera Fajardo3
1Department of Dermatology, University Hospital of A Coruña, A Coruña, Spain, 2Department of Plastic and Reconstructive Surgery, University Hospital of A Coruña, A Coruña, Spain, 3Department of Intensive Care Unit, University Hospital of A Coruña, A Coruña, Spain
*Correspondence: N. Martínez Campayo. E-mail: nieves.mtnez.campayo@gmail.com

References
1 Phelan AL, Katz R, Gostin LO. The novel coronavirus originating in Wuhan, China: challenges for global health governance. JAMA 2020; 323: 709.
2 Munshi L, Del Sorbo L, Adhikari NKJ et al. Prone position for acute respiratory distress syndrome. a systematic review and meta-analysis. Ann Am Thorac Soc 2017; 14(Supplement_4): S280–S288.
3 Ghelichkhani P, Esmaeili M. Prone position in management of COVID-19 patients: a commentary. Arch Acad Emerg Med 2020; 8: e48.
4 Zingarelli EM, Ghiglione M, Pesce M, Orejuela I, Scarrone S, Panizza R. Facial pressure ulcers in a COVID-19 50-year-old female intubated patient. Indian J Plast Surg 2020; 53: 144–146.
5 McCormick J, Blackwood B. Nursing the ARDS patient in the prone position: the experience of qualified ICU nurses. Intensive Crit Care Nurs 2001; 17: 331–340.
6 Mervis JS, Phillips TJ. Pressure ulcers: Prevention and management. J Am Acad Dermatol 2019; 81: 893–902.
7 Galván Casas C, Catalá A, Carretero Hernández G et al. Classification of the cutaneous manifestations of COVID-19: a rapid prospective nationwide consensus study in Spain with 375 cases. Br J Dermatol 2020. [Epub ahead of print]. https://doi.org/10.1111/bjd.19163

DOI: 10.1111/jdv.16755

| Table 1 Adherence rates, clinical and demographic data |
|-----------------------------------------------|
| Adherence | Number of cases (n) | Percentage (%) |
| Yes | 181 | 76.4 |
| No | 56 | 23.6 |
| Age group | |
| 15–30 | 12 | 5.1 |
| 31–45 | 50 | 21.1 |
| 46–60 | 65 | 27.4 |
| 61–75 | 96 | 40.5 |
| 76–90 | 14 | 5.9 |
| Type of treatment | |
| MTX | 16 | 6.8 |
| CyS | 20 | 8.4 |
| APREM | 54 | 22.8 |
| ADA | 44 | 18.6 |
| SECUK | 38 | 16 |
| UST | 24 | 10.1 |
| BROD | 28 | 11.8 |
| ETA | 13 | 5.5 |
| Type of comorbidities | |
| None | 102 | 43 |
| Psoriatic arthritis | 7 | 2.9 |
| Arterial hypertension | 34 | 14.3 |
| Diabetes mellitus | 22 | 9.3 |
| Cardiovascular disease | 10 | 4.2 |
| Depression | 6 | 2.5 |
| Dyslipidemia | 18 | 7.6 |
| Obesity | 14 | 5.9 |
| Other | 24 | 10.1 |
| Number of comorbidities | |
| None | 102 | 43 |
| 1 | 50 | 21.1 |
| 2–3 | 41 | 17.3 |
| >3 | 44 | 18.6 |
| Total | 237 | 100 |

Dear Editor
COVID-19 pandemic raised questions both in dermatologists and in patients about the use of immunosuppressive medications. Although dermatologic societies recommend the continuing of psoriatic systemic therapies and biologics, little is known about treatment adherence in psoriatic patients during COVID-19 outbreak.1 Medication self-management may feel burdensome to patients with psoriasis due to the nature of treatments and many of them face additional challenges as they may suffer from comorbidities. Under these already difficult conditions, COVID-19 disease puts extra pressure on individuals and may undermine adherence. Acknowledging treatment non-adherence as a consequence of conflicting goals may help to find the reasons for but, most important, solutions to non-adherence especially during public health crises. The objective of our study was to evaluate the adherence of psoriatic patients in traditional systemic treatment as well as biologics and identify possible influencing factors of drug interruption during COVID-19 pandemic.

This observational, single-institution study was conducted between 15 March 2020 and 30 April 2020 at the 1st Dermatology Department (Aristotle University of Thessaloniki, Greece). A total of 237 psoriatic patients were interviewed through phone calls about their adherence to medication (methotrexate, cyclosporine, apremilast, adalimumab, etanercept, brodalumab, ...

© 2020 European Academy of Dermatology and Venerology
essential, and the approach to psoriatic patients had to be reduced resistance to infection. This concern is inevitably heightened during COVID-19 outbreak (1). Of comorbidity did not appear to influence the therapeutic routine (2). However, drug discontinuation in our patients seemed to be driven exclusively by concerns about the potential for coronavirus infection. The most important risk factors that worsen the prognosis of COVID-19 disease are comorbidities that are also present in psoriatic patients. Our results are agreed with relevant reports which suggest that the presence of comorbidities are linked to sustained drug survival. Several authors support the rationale that the more patients are accustomed to medication use for coexisting health issues, the higher and more long-lasting their adherence to additional treatments. Our study suggests that necessity beliefs about therapy are a prerequisite for taking medicines and that the need for treatment appears to outweigh the fears about the medication.

Conclusively, we recommend embracing a non-judgmental approach that acknowledges difficulties in adherence, especially in situations of increased public health risks, and encouraging patients to discuss factors contributing to non-adherence. This approach may assist patients to determine conflicting goals and find possible solutions, support psychological welfare and improve adherence.

Conflicts of interest
Dr. Vakirlis, Dr. Bakirtzi, Dr. Papadimitriou, Dr. Vrani, Dr. Sideris, Dr. Lallas Dr. Ioannides and Dr. Sotiriou have nothing to disclose.

Funding sources
None.

References
1 Torres T, Puig L. Managing cutaneous immune-mediated diseases during the COVID-19 pandemic. Am J Clin Dermatol 2020; 21: 307–311
2 Belinchón I, Rivera R, Blanch C, Comellas M, Lizán L. Adherence, satisfaction and preferences for treatment in patients with psoriasis in the...
An unusual case of bullous haemorrhagic vasculitis in a COVID-19 patient

Dear Editor

A novel Coronavirus strain, named ‘Severe Acute Respiratory Syndrome Coronavirus 2’ (SARS-CoV-2) was recently identified as the etiologic agent of the COronaVIrus Disease 2019 (COVID-19). Interestingly, a consistent number of COVID-19-associated skin manifestations seem to share a certain degree of vascular damage as common pathogenetic mechanism. Vascular injury may be due to the direct damage of endothelial cells by the virus or may represents an epiphenomenon of a dysregulated host inflammatory responses triggered by the infection. Here, we describe an unprecedented case of leukocytoclastic vasculitis presenting with a haemorrhagic bullous eruption in a patient affected by COVID-19.

A 79-year-old man with a history of hypertension, myocardial infarction and chronic obstructive pulmonary disease has been hospitalized for acute heart failure. The patient was tested for COVID-19 (RT-PCR on nasopharyngeal swab sample) and resulted negative. Medical treatment for heart failure was started and patient’s conditions progressively improved. On day 15 of the hospitalization, he rapidly developed fever and dyspnea. Chest radiograph and CT scan revealed a radiologic pattern suggestive for COVID-19 pneumonia and nasopharyngeal swab RT-PCR confirmed SARS-CoV-2 infection. Treatment with hydroxychloroquine (400 mg bid), prophylactic anticoagulation (enoxaparin 4000 IU qd), empiric antibiotics (ceftraxone 600 mg bid) and intravenous corticosteroids (methylprednisolone 80 mg qd) was started. Concomitantly, oxygen therapy was initiated at 8 liters/minute (approximately 40% FiO2) via a non-rebreathe mask. After ten days, the patient developed multiple non-itching vesiculobullous lesions on neck and dorsal areas of hands (Fig. 1a,b). Laboratory tests including whole blood count, biochemical and coagulation parameters were within normal limits. Antinuclear antibody, antineutrophil cytoplasmic antibody and cryoglobulins resulted negative and serum protein electrophoresis as well as complement levels were normal. Moreover, the patient tested negative for enzyme-linked immunosorbent assay (ELISA) for detecting BP180 and BP230 antibodies. A punch skin biopsy was performed. Histopathologic examination demonstrated irregular hyperplasia of the epidermis and abundant erythrocytes extravasation with formation of intraepithelial haemorrhagic bullae. The epidermis was partly necrotic with keratinocytes focally showing nuclear hyperchromasia and cytoplasmic eosinophilia (Fig. 1c). Within the superficial dermis, there were marked erythrocytes extravasation and severe neutrophilic infiltrate within the wall of small vessels and in their proximity with scant leukocytoclasia (acute vasculitis). Endothelial cells were activated showing nuclear enlargement and hyperchromasia (Fig. 1d). Eosinophils and lymphocytic infiltration were not observed. Fibrinoid vascular changes and thrombi were absent as well as no viral cytopathic changes were observed. The histopathologic findings demonstrated a typical picture of leukocytoclastic vasculitis. Unfortunately, in the following days patient’s respiratory conditions deteriorated and, despite intensive care support, he died of respiratory insufficiency.

The case described is an unusual case of bullous haemorrhagic vasculitis in a COVID-19 patient. The macroscopic characteristics of the lesions were compatible with a localized bullous pemphigoid (BP) or a heparin-induced bullous haemorrhagic dermatosis (BHD). In our patient, absence of eosinophilic infiltrates as well as negativity of ELISA for BP180/BP230 autoantibodies reasonably rule out the hypothesis of localized BP. The second diagnostic diagnosis was BHD. Nevertheless, focally necrotic epidermis and vasculitis observed in our case have never been reported in BHD and thus we excluded this diagnosis. Histopathologic features observed in our patient are characteristic of an evolving leukocytoclastic vasculitis (LCV). Interestingly, capillary injury and/or neutrophilic infiltrates have been described in lung tissues from COVID-19 and, in one recent report, also in the skin. Nonetheless, we can expect that the number of reports concerning COVID-19-related vasculitis is likely to increase since inflammatory vascular damage is emerging as one of the main pathogenic mechanisms of SARS-CoV-2 infection, including its cutaneous manifestations. However, only further studies, novel reports including clinical images and detailed histology as well as data from international dermatology registries will be able to confirm this hypothesis.