ychloroquine with the addition of systemic steroids and meropenem. At that time, two subsequent rhino-pharyngeal swabs for SARS-CoV-2 molecular testing were performed and both resulted negative. Extensive viral and bacteriological tests were repeatedly negative; a marked increase in CRP was found. After five days, the patient was apyretic with regression of the erythema, but with onset of non-palpable purpuric and livedoid lesions on the lower limbs (*figure 1B*). Steroid treatment was continued and heparin was added, leading to an improvement in skin lesions with complete clearance on April 30th.

The interest of our case lies in the presence of the two sequential patterns of cutaneous manifestations; a confluent macular rash followed by livedoid lesions of the lower limbs, characteristic and previously reported in COVID-19 patients, but not in the same patient. Even though molecular tests were negative, the symptoms, fever, and skin lesions clearly support the diagnosis of COVID-19. Indeed, recommendations in the literature underline that a negative nasopharyngeal swab is insufficient to rule out COVID-19 [4]. Moreover, the timing of the test which was performed more than two weeks after the onset of symptoms and beginning of hydroxychloroquine treatment is consistent with the negative results.

The cutaneous lesions are strictly related to COVID infection as the macular rash represents an immune response towards viral nucleotides and the livedoid purpura is a consequence of vascular damage [2, 3, 5]; the pathogenetic mechanisms constitute the background for the different histopathological features (a perivascular lymphocytic infiltrate in the superficial dermis in the former and thrombogenic leukocytoclastic vasculitis in the deep dermis and complement deposition in the latter). The temporal evolution of the lesions, with the rash preceding the livedo, is therefore consistent with disease pathogenesis. Even though livedoid/necrotic lesions are associated with more severe disease (10% mortality) [3], this group of patients also includes mild cases such as ours, in whom heparin therapy could have played a major role in improving the disease. Awareness among dermatologists should be raised in order to correctly identify the various COVID-19 manifestations. ■

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Hidradenitis suppurativa and adalimumab in the COVID-19 era

A general concern about a potentially higher risk of COVID-19 among patients with inflammatory skin diseases, such as hidradenitis suppurativa, under treatment with biologics has promoted a number of reports in the scientific literature [1]. Recently, Blaszczak [2] found only a modestly increased risk of infections in HS patients treated with adalimumab versus those under placebo based on a review of the data published for PIONEER I and II trials [3]. Real-life data on COVID-19 risk in HS patients treated with adalimumab may be inferred only from a single-center study in which 75 HS patients under adalimumab treatment were analysed, none of whom developed COVID-19 [4].

Twenty Italian tertiary referral centers previously involved in a study on adalimumab treatment for HS [5] were asked to participate in a telephone-based survey, which was conducted between March 30th and April 30th, 2020. Patients with HS under adalimumab were asked about possible diagnosis of severe acute respiratory coronavirus disease 2 (SARSCoV-2) infection. The International HS Severity Score System (IHS4) [6] was used at the last visit and the duration of adalimumab treatment in weeks was recorded.

In total, 316 patients were included in the study, 311 of whom were under adalimumab at the time of the survey and five had temporarily discontinued adalimumab due to safety concerns related to COVID-19 on the advice of their general practitioner. There were 201 male patients (64.6%) and median age was 55.1 (range: 19-70). The median duration of adalimumab treatment was 100 (IQR: 70-132) weeks. The last median IHS4 score before the telephonic survey was 7 (IQR: 4-14). Three patients (1%) received a diagnosis of COVID-19, confirmed by nasopharyngeal swab. Using Fisher's exact test, no statistically significant differences in COVID-19 occurrence were found between patients under active treatment and patients who stopped treatment for precautionary reasons (p=1).

Patient 1 was a 65-year-old housewife without comorbidities except for moderate obesity. Her symptoms, including fever, cough, myalgia, hypogeusia and hyposmia, dated back to February 15th. Patient 2 was a 28-year-old pregnant woman with Crohn's disease diagnosed with COVID-19 on March 10th. Her symptoms included fever, cough, coryza

and pharyngodynia, hypogeusia, hyposmia and gastrointestinal symptoms. Patient 3 was a 25-year-old man who was asymptomatic and underwent a COVID-19 swab in accordance with a testing policy at work. No patients were hospitalized and all of them fully recovered; only Patient 2 temporarily discontinued adalimumab. The full protocol was approved by the Institutional Review Board of the Ethical Committee of the principal investigator's centre (Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico, Milan, Italy) with the protocol number: 464_2020. All the subjects enrolled in the study gave their informed consent.

A limitation of our study is the unavailability of data on SARS-CoV-2 infection of those in close contact with our patients.

According to the last median IHS4 score before the telephonic survey, our cohort was mainly represented by patients with moderate HS, with a median adalimumab treatment duration of a little over two years. Based on the low prevalence of COVID-19 (1%) in our cohort, as well as other relevant studies [2, 3], we hypothesize that both the disease and its therapy with adalimumab do not represent a risk factor for COVID-19. Therefore, adalimumab should not be suspended by HS patients themselves due to COVID-19-related reasons.

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