Antinuclear antibodies and autoantibodies to extractable nuclear antigens (including anti-Jo-1, anti-Mi-2 and anti-TIF1-gamma) were negative. A CT scan of the chest and abdomen showed several enlarged lymph nodes in the right jugular area and a tumour in the left upper lobe. One lymph node was excised for histological examination. It revealed metastatic spread of a poorly-differentiated squamous cell carcinoma of the lung. After treatment with intravenous immunoglobulins and steroids, regression of myositis and the cutaneous lesions was achieved. The inoperable lung cancer was treated with pembrolizumab, paclitaxel, and cisplatin. The patient died two months later of pneumonia. We report a patient with squamous cell lung cancer, necrobiotic xanthogranuloma, and polymyositis. In approximately 80% of cases, necrobiotic xanthogranuloma is accompanied by monoclonal gammopathy [1]. Necrobiotic xanthogranuloma may also be associated with blood cancers or lymphoproliferative disorders [2, 3]. No cases associated with lung cancer - as in our patient - or other solid tumours have been reported so far. In our patient, monoclonal gammopathy and leukaemia were ruled out based on immuno-electrophoresis and flow cytometric analysis.

Polymyositis is one of many inflammatory myopathies and is accompanied by symmetrical proximal muscle weakness. About 30% of elderly patients with dermatomyositis/polymyositis have an underlying malignancy. Cancer is less frequent in the presence of polymyositis than dermatomyositis [4, 5]. Associated cancers include those of the ovary, lung, breast, gastrointestinal tract, pancreas, nasopharynx, testicles, and non-Hodgkin's lymphoma. An association between polymyositis and lung cancer has been rarely reported [6]. The pathophysiology of polymyositis and malignancy is not well understood. Some patients with paraneoplastic polymyositis develop autoantigens common to cancer and muscle tissue, resulting in muscle damage [7]. Paraneoplastic polymyositis is frequently resistant to treatment because of the underlying malignancy. On the other hand, the treatment of cancer may lead to the regression of myositis.

To the best of our knowledge, this is the first reported case of necrobiotic xanthogranuloma and polymyositis in a patient with lung cancer. The simultaneous occurrence of these two potentially paraneoplastic diseases in the same patient may constitute a new clinical entity.

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## Evolution of different clinical patterns of cutaneous lesions in a suspected COVID-19 patient

Cutaneous manifestations of Coronavirus disease-19 (COVID-19) were noted in >20% of hospitalized patients in a recent Italian study [1]. Heterogeneous manifestations have been reported. Suchonwanit *et al.* [2] distinguished two groups: viral exanthemas and thrombotic vasculitides. In a recent Spanish multicentre paper [3], five different patterns were identified: acral erythema (pseudochilblain), vesicular eruptions, urticarial lesions, maculopapular eruptions (the most frequent) and livedo/necrosis.

Herein, we report a patient who sequentially developed two cutaneous COVID-related manifestations; a macular exanthema followed by a livedoid vasculitic eruption, characterised by different pathogenetic and histopathological features. As far as we know, no previous cases have been reported with similar features.

A 58-year-old healthy man developed fever and chills on March 23<sup>rd</sup> and received hydroxychloroquine and minocyclin at home. On April 4<sup>th</sup>, he developed a macular rash with confluent erythema on the trunk and limbs, without itching (*figure 1A*). Because of the worsening of fever (up to 40°C), he was hospitalised on April 5<sup>th</sup>, continuing hydrox-



**Figure 1.** Clinical images of the case: **A)** confluent erythema on the trunk; **B)** livedoid lesions on the lower legs.

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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 30<sup>th</sup>.

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.

**Disclosure.** The patient presented in this manuscript has given written informed consent to publication of his case details.

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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 30<sup>th</sup> and April 30<sup>th</sup>, 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 15<sup>th</sup>. Patient 2 was a 28-year-old pregnant woman with Crohn's disease diagnosed with COVID-19 on March 10<sup>th</sup>. Her symptoms included fever, cough, coryza

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