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LETTER TO THE EDITOR

Necrotic lesions on the face in a patient with COVID-19

KEYWORDS
Thrombotic microangiopathy;
SARS-Cov-2;
COVID-19;
Necrotic lesions

Introduction

The main manifestations of SARS-Cov-2 are fever, asthenia and respiratory symptoms [1]. Cutaneous manifestations were unknown at the beginning of the pandemic, but are now increasingly recognized in the literature. They are reported in many sporadic cases or case series. The prevalence of cutaneous manifestations in COVID-19 patients ranges from 0.2 to 20.4% [2]. Chilblains-like lesions, maculopapular eruptions, livedo, petechiae, purpura, necrosis, wheals and vesicles are the most commonly described [3]. We are reporting the case of necrotic lesions localized exclusively on the face in a patient with COVID-19, due to thrombotic microangiopathy.

Case report

A 17-year-old patient, with no particular pathological history, was admitted in February 2021 for necrotic lesions on the face that had been evolving for 10 days. He reported a fever of 39 °C and lower back pain that appeared one week before the cutaneous symptoms. No drugs or cocaine were taken before the symptomatology. The patient didn't report any stings, bites, or applications of corrosive products. He wasn't vaccinated against the COVID-19.

On clinical examination at admission, the patient was apyretic, hemodynamically stable and eupneic. The dermatological examination revealed erythematous and purplish maculo-papular lesions with infiltration, necrotic and hemorrhagic, telangectasias and fine scales, without local inflammatory signs, on the face and the helix (Fig. 1). We also noticed a cheilitis without mucosal erosion. The abdomen was supple with no palpable hepatomegaly or splenomegaly. The rest of the clinical examination was without abnormalities. We suspected a secondary post-infectious vasculitis, a primary vasculitis (periarteritis nodosa, Churg and Strauss, cryoglobulinemia, or microscopic polyangiitis), a systemic lupus erythematosus, an antiphospholipid syndrome, or a thrombopathy associated with COVID-19.

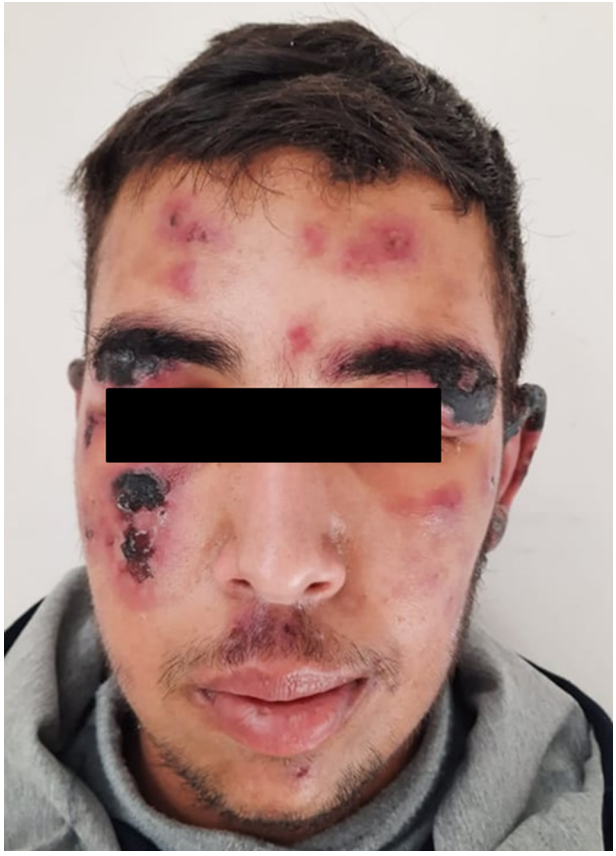

Figure 1 Necrotic erythematous and purplish maculo-papular lesions on the face and the helix.

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A cutaneous biopsy showed an acanthosic epidermis, basal vacuolation, vascular thrombosis in the dermis and hypodermis without inflammation around the vessels, and a perivascular lymphocytic infiltrate. Direct immunofluorescence was negative. Tzanck smear didn't show ballooning cells.

The biological workup showed a normal level of hemoglobin, lymphopenia at 1100 elements/μL, thrombocytopenia at 100,000 elements/μL, low prothrombin level at 55%, normal activated partial thromboplastin time, high fibrinogen level at 5 g/L, normal factor V level, and elevated D-dimer at 1113 μg/L. There was also an elevated C-reactive protein at 44 mg/L, hyperferritinemia at 373 ng/L, elevated lactate dehydrogenase at 475 U/L, cytolysis at 3 times the normal values, and total bilirubin (free and conjugated) at twice the normal values. Haptoglobin level was normal. Direct Coombs test was negative. Thus, we didn't retain enough biological signs in favour of hemolysis or disseminated intravascular coagulation. COVID-19 serology was positive. Viral hepatitis and human immunodeficiency virus serologies were negative. Tests searching for autoimmune diseases were also negative: Anti-nuclear antibodies, anti-DNA antibodies, C3, C4, CH50 complements, anti-neutrophil cytoplasm antibodies, cryofibrinogen and cold agglutinin testing, and anti-phospholipid antibodies (β2-glycoprotein, anticardiolipin antibodies, and circulating lupus coagulants). Thoracic computed tomography and computed tomography angiography were both normal. Abdominal ultrasound showed ascites of small volume. Cardiac and renal explorations were both normal. The final diagnosis was necrotic facial lesions secondary to a thrombopathy due to SARS-Cov-2. The patient was first put on corticosteroids 1 mg/kg/day (90 mg/day) since we suspected a vasculitis, acetylsalicylic acid 100 mg/day, therapeutic dose of low molecular weight heparin, vitamin C 1000 mg/day and petroleum jelly, during 2 weeks until we got the result of the cutaneous biopsy and the immunological assessment. The evolution was good with complete clinical and biological remission (Fig. 2). We kept a preventive dose of anticoagulation and vitamin C for 15 days. The corticotherapy was rapidly decreased in a period of 2 months. The follow-up is 10 months without recurrence.

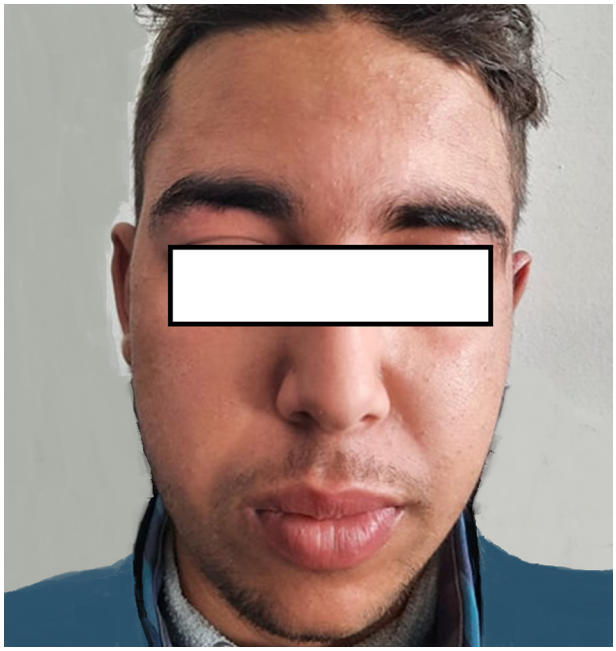

Figure 2 Complete remission after 10 days.

Discussion

The particularity of our observation is related to the rarity of cephalic necrotic lesions due to COVID-19. Only a few cases have been reported. Karagounis reported 16 cases of necrotic lesions on the face and/or ears [4], while one case of maxillary necrosis was noticed in the meta-analysis of Tan et al. [5]. The occurrence of cephalic necrotic lesions is frequent in young males, as the case of our patient. Normally, necrotic lesions in SARS-Cov-2 appear as digital necrosis or distal ischemic lesions [1,3,6,7]. However, chilblains are the most frequent acrosyndrome in the context of COVID 19 pandemic [8].

The diagnosis was retained according to a set of arguments (questioning, cutaneous biopsy and serology), and after eliminating other possible diagnosis that could explain the erythematous, infiltrated and necrotic maculopapular lesions on the face and helix: secondary or primary vasculitis, systemic lupus erythematosus, antiphospholipid syndrome, or thrombopathy associated with cocaine use. All the work-up done in this sense was negative.

The pathophysiologic mechanisms of cutaneous manifestations during COVID-19 can be explained by many theories. The virus binds to angiotensin II converting enzyme receptors leading to activation of macrophages, monocytes, and neutrophils. This leads to the secretion of cytokines: Interleukins 1, 6, 8 and tumor necrosis factor α and a prothrombotic state. Endothelial adhesion of neutrophils and Neutrophil Extracellular Traps (NET) secretion lead to vasculitis lesions [9]. The consequence is the development of tissue damage in the skin and all tissues containing angiotensin II converting enzyme receptors such as; heart, lungs, kidneys, oral cavity, pancreas, gastrointestinal tract and brain [10]. Maculopapular, urticarial and vesicular lesions are due to overproduction of cytokines, while chilblain-like, purpuric, livedoid and necrotic lesions are due either to vasculitis or to a thrombogenic vasculopathy [11].

The presence of necrotic lesions in COVID-19 is highly suggestive of vascular damage and a hypercoagulable state. This results of a cytokine storm as observed in other viral diseases. The biological assessment shows a high level of D-Dimer, prothrombin, and fibrinogen. Treatment must be rapidly started to improve the prognosis. It requires the use of a therapeutic dose of anticoagulation associated with corticosteroids. The dose and duration of oral corticotherapy is still controversial. We suggest that it depends on the clinical and biological evolution [2,4]. The prognosis of cutaneous manifestations during COVID-19 is generally good with healing times up to 10 days and without recurrence [12].

Conclusion

In the current pandemic context of COVID-19, any dermatological lesion of vascular or inflammatory origin should raise

the suspicion of SARS-Cov-2 infection, especially if there are other suggestive signs. However, this is an elimination diagnosis. This case of necrotic lesions localized exclusively on the face due to SARS-Cov-2 joins the other few cases described. It is a rare feature, hence the interest to report all observed cases.

Human and animal rights

The authors declare that the work described has not involved experimentation on humans or animals.

Informed consent and patient details

The authors declare that they obtained a written informed consent from the patients included in the article. The authors also confirm that the personal details of the patients have been removed.

Disclosure of interest

The authors declare that they have no competing interest.

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