reality there is not a single case of suicide due to the "Blue Whale Challenge".[7]

Several dermatological conditions lead to depression and anxiety disorders.[8] In the same way, it is important to note the prevalence of self-induced skin cutaneous lesions among many psychiatric disorders. Detection and identification of those lesions may be the key to an early intervention of patients at risk of suicide. It would then be necessary to use a multidisciplinary approach[9] including psychiatrists, psychologists, dermatologists, and even pediatricians in child populations.

The examination of any dermatological lesion with peculiar characteristics, both by origin and by form, should alert us in order to rule out that our pediatric patient is not immersed in games of psychological manipulation. Thus, the specialist in dermatology should be aware and recognize the psychiatric aspects underlying these lesions.

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Recrudescence of livedoid vasculopathy induced by COVID-19

Dear Editor,

Livedoid vasculopathy (LV) is a rare, chronic, small-vessel occlusive disease whose pathogenesis is mediated by inflammation and coagulation disorders. It often affects young women in the lower limbs, usually manifested by *livedo racemosa*, purpuric macules, and skin necrosis. The disease progresses to superficial and painful ulcers that often evolve into characteristic atrophic scars. The management of LV is difficult, as it evolves through a chronic and recurrent course. Relapses are associated with hot summer days, prolonged orthostatic posture, and risk factors for thrombosis (e.g., weight gain, contraceptive pills, and smoking).[1]

We report on a 34-year-old female patient with histologically confirmed LV, with onset at 26 years old; no other comorbidities or coagulative disorders were identified. While the LV was refractory to anticoagulation (warfarin and rivaroxaban), the patient had been under clinical control with acetylsalicylic acid 300 mg/day, pentoxifylline 800 mg/day, cyclosporine 200 mg/day, and compression socks since 2018.

In the spring of 2020, the patient presented with an infection manifested by fever, sore throat, tinnitus, and cough, with no need for hospitalization. The nasopharyngeal swab test for SARS-CoV-2 was positive. Four days after the onset of symptoms, she experienced the recrudescence of LV (Figs. 1 and 2) with new ulcers despite maintaining the treatment. Prednisone 80 mg/day was added to the therapeutic scheme, and 21 days after the onset of symptoms, the underlying disease was subjected to clinical control, with a decrease in pain and in the diameter and healing of ulcers.

The new SARS-CoV-2 infection is a life-threatening multisystem disease, reported in China (late 2019), that evolved into a pandemic in March 2020 (COVID-19). Its cutaneous manifestations are multiple, nonspecific, and reported in up to 30% of patients.[2] In general, four patterns of cutaneous manifestation have been reported: exanthema pattern (varicella-like, maculopapular, and morbilliform), vascular pattern (chilblain-like, livedoid lesions, and purpuric or petechial lesions), acral papular eruption, and urticaria-like pattern.[3,4]

The pathogenesis of COVID-19 involves an increase in inflammatory markers, such as CRP, D-dimer, ferritin, fibrin degradation products, and prolonged prothrombin time, in addition to an increase in inflammatory cytokines (cytokine storm); this involvement indicates the activation of both Th1 and Th2 immune responses. Despite the presence of lymphopenia, there

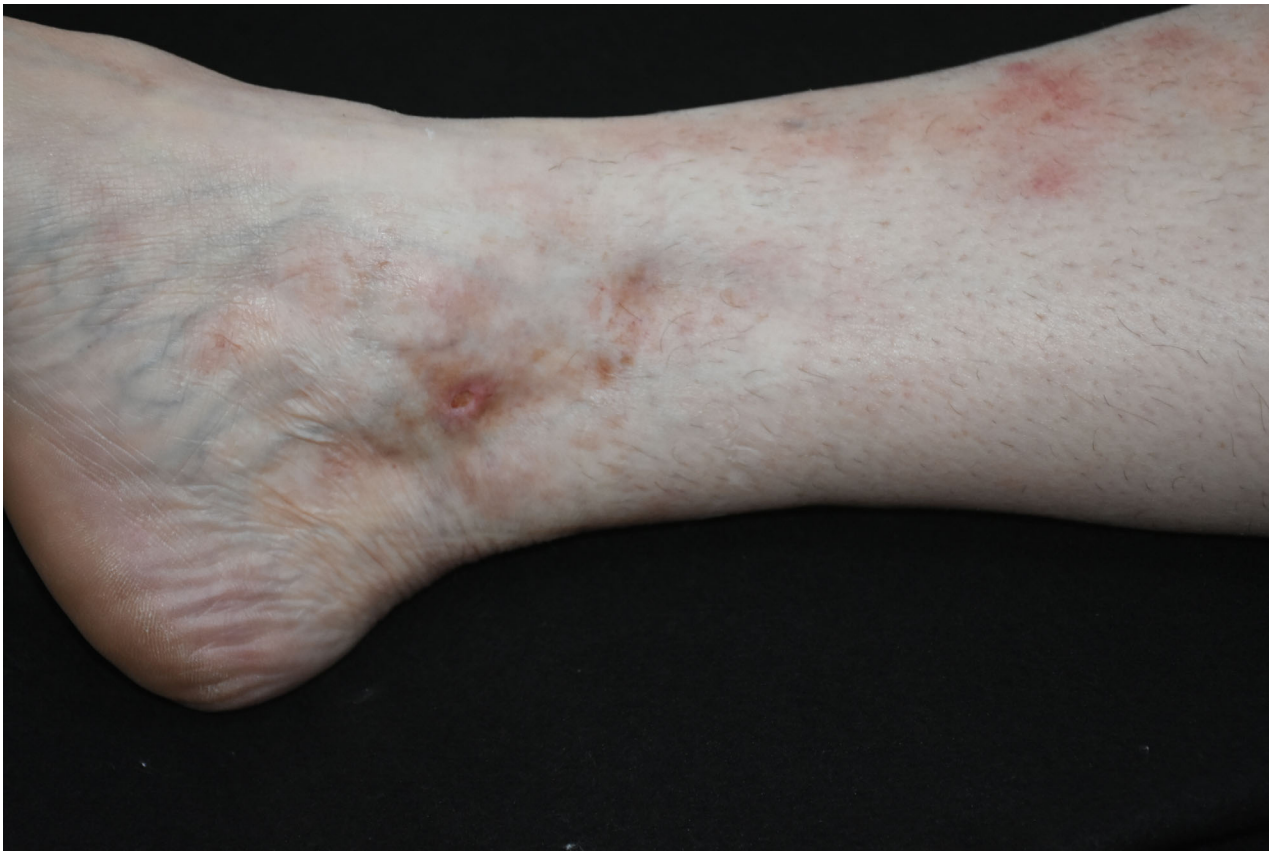

Figure 1 Livedoid vasculopathy. Ulcer with fibrinous base and well-adhered edges, purpuric lesions, and erythematous macules on the right lower limb, medial view

is evidence of CD4 and CD8 hyperactivation. As the infection progresses, immunoglobulins are produced, leading to the formation of immune complexes and increased secretion of inflammatory cytokines, mainly TNF-α, interferon γ, IL2, IL6, IL8, IL10, and IL17.[5] In addition to the inflammatory context, the virus has endothelial tropism, binding to selectins, increasing the expression of von Willebrand factor, favoring thrombotic events and, thus, aggravating vascular occlusion.[6,7]

Critically ill COVID-19 patients develop a complex state of immunologically mediated thrombosis and endothelial damage that leads to systemic compromise and a myriad of clinical manifestations, with microthrombosis in the lungs, brain, liver, kidneys, skin, and gastrointestinal system. At the inflammatory phase (cough, myalgia, and fever), there are similarities between COVID-19 and the pathological findings from thrombo-occlusive ulcers in patients with LV. Additionally, some therapeutic approaches are common to these diseases, such as anticoagulation, hyperbaric oxygen, and anti-inflammatory measures.[8] Recent studies indicate a relationship between cutaneous vascular lesions and COVID-19 severity (e.g., *livedo racemosa*, necrotic ulcers, and acro-ischemia).[4,9] The virus is believed to use the endothelial receptor of the angiotensin-converting enzyme to enter cells and, from there, initiate an inflammation cascade, inducing endothelial and myointimal proliferation.[10–12]

The recrudescence of LV in the context of SARS-CoV-2 infection as well as its remission after corticosteroid therapy and the cessation of respiratory symptoms suggest COVID-19 influences the course of inflammatory and thrombotic diseases.

This case report should be confirmed through other LV patients who have been infected with COVID. Moreover, the clinical improvement with the administration of steroids cannot be established, while we have no information from cases in which the drug was not used.

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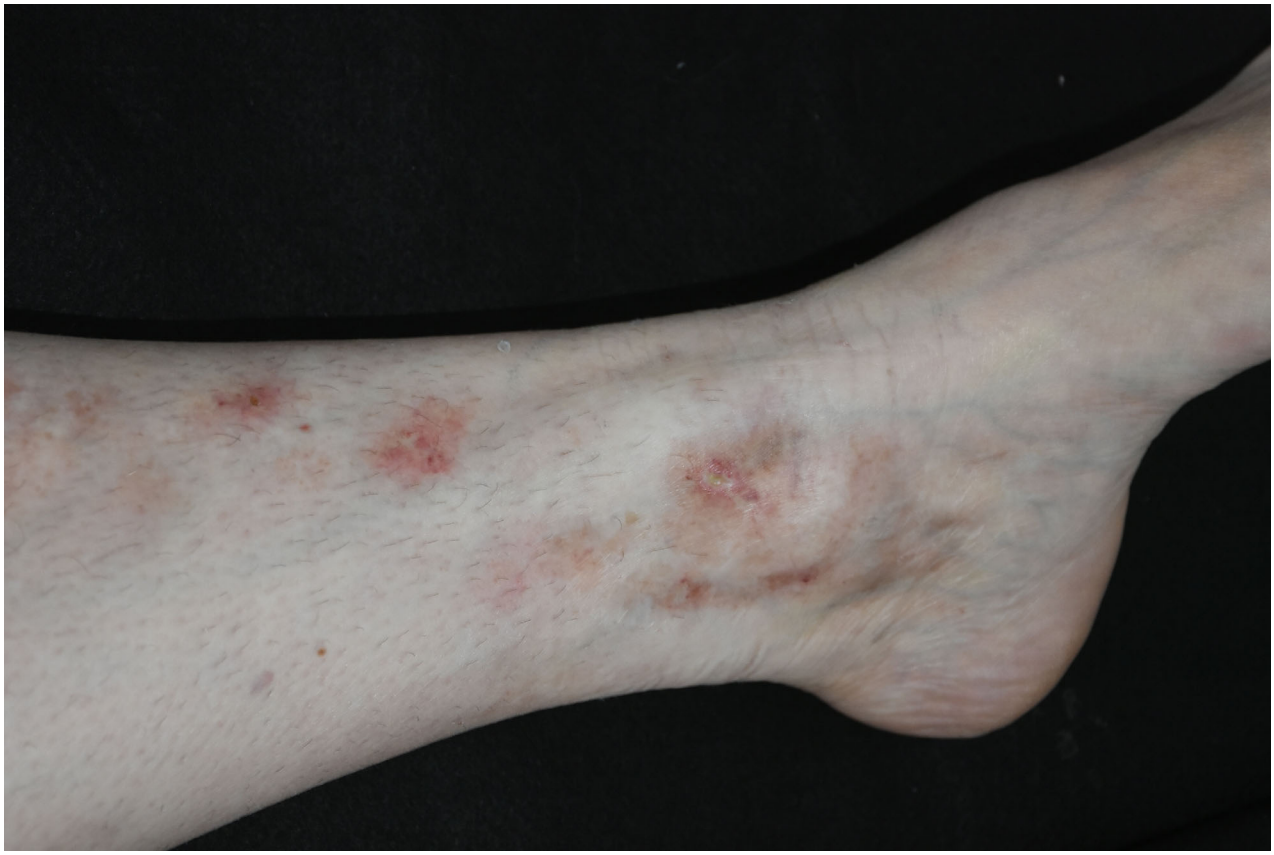

Figure 2 Livedoid vasculopathy. Purpuric lesions, two ulcers with fibrinous bases and atrophic scars on the left lower limb, medial view

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Novel keratin 16 mutation in a Chinese family with focal palmoplantar keratoderma

Dear Editor,

Palmoplantar keratodermas (PPKs) are heterogeneity disorders characterized by marked hyperkeratosis on the surface of palms and soles. According to the pattern of hyperkeratosis, PPKs are classified into diffuse, focal (including striate), and punctate forms. Among these forms, focal PPK (FPPK),