Hence, corticosteroids could have prevented the initial development of AEGP.

Lastly, to date SCARs have been reported with the use of ceftriaxone, but there is only one reported case of DRESS related to cefditoren, among other causes.\(^4,5\)

Surrounding the COVID-19 pandemic, many dermatoses are being reported as possibly SARS-CoV-2 induced. Here, we highlight the importance of considering other etiologies as causes of skin lesions arising on the background of SARS-CoV-2 infection. Special effort has to be made to identify drugs as the source of these events, as they may lead to SCARs.

Finally, further studies should investigate cross-reactions between cephalosporins, and the role of cefditoren as causative agent of SCARs.

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Nothing to disclose.

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**Erythema multiforme and Kawasaki disease associated with COVID-19 infection in children**

Dear Editor

We read with interest the publications in the JEADV which reported dermatological manifestations associated with COVID-19, such as pityriasis rosea, urticaria, rash, vascular signs or chillblain-like lesions.\(^1,6\) Herein, we report two life-threatening cases of children presenting with fever and eruptions with mucous membrane involvement – erythema multiforme and Kawasaki disease – associated with COVID-19.

Case 1: A 6-year-old male was hospitalized for painful cheilitis that develops during the week before admission and rapidly became associated with a rash of the extremities, and conjunctivitis. The patient was reported to have had a loss of appetite, without any other symptoms. The father reported having transient anosmia 2 weeks before. There was no history of recent medication. At admission, clinical examination revealed severe erosive cheilitis (Fig. 1a) with diffuse gingival erosions and thick haemorrhagic crusts, bilateral conjunctivitis, associated with multiple target lesions (Fig. 1b,c). Respiratory function was normal. The clinical picture led to a diagnosis of erythema multiforme. *Mycoplasma pneumoniae* serology was negative. The herpes simplex virus (HSV) polymerase chain reaction (PCR) test, on buccal erosions, was also negative. A first COVID-19 test, carried out by PCR, was negative; however, a second test was positive. The child’s condition improved, and he was discharged 2 weeks after.

Case 2: A 3-year-old male was hospitalized for fever >39.0°C for 8 days. The fever was associated with asthenia, generalized exanthema, cheilitis, stomatitis and bilateral conjunctivitis. His mother had been diagnosed with COVID-19, 3 weeks earlier. Clinical examination revealed generalized exanthema (Fig. 2a), bilateral palmar oedema, glossitis and cervical lymphadenopathy. Desquamation of the extremities was noted during a subsequent examination (Fig. 2b). Laboratory tests showed an increase in inflammatory biomarkers: CRP = 195 mg/L and hyperleukocytosis (leucocytes = 17 400/mm\(^3\)). A COVID-19 PCR test performed at admission was negative. The CT scan revealed ground-glass opacities and consolidation in the right posterobasal area (<10% of the lung parenchyma), suggestive of COVID-19 pneumonia (Fig. 2c). We concluded a final diagnosis of COVID-19-associated Kawasaki disease. The child was treated with an initial dose of intravenous gamma globulin (2 g/kg).

This case report provides a detailed description of severe cutaneous manifestations occurring in two children with COVID-19. The manifestations reported in our first case are typical of erythema multiforme, with particularly severe mucosal lesions being noted in this child. The main causes of erythema...
multiforme in children are *Mycoplasma pneumoniae* and HSV. Other viruses and bacteria, together with some vaccines, have also been reported to trigger erythema multiforme. In our case, the absence of any serologic and biologic evidence of HSV or of mycoplasma infection, together with the positive result for the second COVID-19 test, led us to a final diagnosis of COVID-19-associated erythema multiforme.

The skin condition in our second case was diagnosed as Kawasaki disease. Kawasaki disease is a systemic vasculitis for which the aetiology remains unknown, although it seems likely that immunologic and infectious mechanisms, associated with either viruses or bacteria, are involved. Several studies have investigated whether there is a link between the common human coronavirus (HCoV) NL-63 and Kawasaki disease, but no associations have been identified so far. A 2014 study indicated that HCoV-229E could be a possible causative agent for Kawasaki disease. Although the COVID-19 PCR test performed on the child described in our case report was negative, the results of the CT scan were very suggestive of COVID-19 pneumonia. This case strongly suggests that SARS-CoV-2 is a trigger for Kawasaki disease.

Finally, it is interesting to note that neither of the two children with COVID-19 described in this report had respiratory symptoms and that cutaneous manifestations were at the forefront of the clinical picture.

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Cutaneous small vessel vasculitis secondary to COVID-19 infection: a case report

Dear editor

We present a case of an 83-year-old woman with a history of hypertension, transient ischaemic attack (TIA), atrial fibrillation and chronic renal impairment presented to our dermatology emergency room on 9 April 2020, for evaluation of purple palpable papules and serohaematous blisters on both her lower legs, feet and toes that had appeared 5 days earlier (Fig. 1). She denied having any new medications, and she had been undergoing long-term treatment with acenocoumarol and furosemide for her prior conditions. A month before the cutaneous lesions developed, she had a history of one week of sore throat, followed by malaise and nausea. She did not present dyspnoea, chest pain, diarrhoea, smell or taste alterations, nor fever. A clinical diagnosis of cutaneous vasculitis was made. We performed a 4-mm punch biopsy of one palpable purple papule. A complete blood test and urinalysis were performed. The patient was started on 30 mg/day prednisone therapy with clinical improvement achieved after 10 days, after which the medication was progressively tapered. Laboratory testing showed a normal white blood cell count. Thrombocytopenia was not observed. C-reactive protein was 8.0 mg/L (normal value 0.0–5.9), and serum lactate dehydrogenase was 279 U/L (normal value 100–190). Hepatic function was not impaired. Creatinine was 1.44 mg/dL (normal value 0.50–1.10), and this finding was related to prior chronic renal disease. Urinalysis showed no signs of proteinuria nor haematuria. Serum IgG, IgM and IgA levels were within normal ranges. Immunological laboratory results showed negative values for anti-nuclear antibodies and anti-neutrophil cytoplasmic antibodies (ANCAs). IgM cardiolipin antibodies were slightly increased: 21.29 (MPL; normal value < 20.00). Complement C3 and C4 levels were not decreased. Serum cryoglobulins were increased: 21.29 (MPL; normal value < 20.00). Complement C3 and C4 levels were not decreased. Serum cryoglobulins were

Figure 1 Cutaneous finding of our patient. (a) Purpuric papules on both distal legs and feet with some haematoic blisters. (b) Closer look at toes showing petechiae and purpuric papules.