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Sweet syndrome in post-COVID-19 infection: A case report

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Dear editor:

The novel coronavirus (SARS-CoV-2), the cause of coronavirus 2019 disease (COVID-19) pandemic, is associated with some cutaneous manifestations. The most frequently reported are: acral lesions, maculapapular rashes, urticarial rashes, vascular lesions and vesicular rashes [1]. The occurrence of sweet syndrome in the context of a covid infection of which we report a case is rare; only two cases have been reported in the literature [2,3]. We report here a case of Sweet's syndrome occurring three weeks after a simple coronavirus disease 2019 (COVID-19) infection.

A 61-year-old patient with no particular history who presented three weeks after a simple COVID-19 infection confirmed by positive RT-PCR in nasopharyngeal swabs, made up of fever, cough myalgia and without severe respiratory syndrome, painful erythematous, edematous, purplish plaques in the hands and feet (Fig. 1). The biological assessment found an inflammatory syndrome with a CRP of 105 mg, hyperleukocytosis predominantly neutrophilic. The ASLO assay was negative. A thoraco-abdomino-pelvic scanner, performed to rule out associated malignancies, showed no abnormalities. Protein electrophoresis was normal. The patient did not have any history of inflammatory bowel disease or digestive sign, and faecalcal protectin was negative. No supplementary drugs were added to what was indicated above. Pathological examination of a skin biopsy found an inflammatory dermal neutrophilic infiltrate without vasculitis. A Sweet post covid syndrome was retained in view of the clinical and anatomopathological elements. Colchicine treatment was started with marked improvement.

Sweet's syndrome also known as acute febrile neutrophilic dermatosis is a rare neutrophilic dermatosis characterized by a constellation of clinical symptoms, physical features, and pathologic findings which include fever, neutrophilia, tender erythematous skin lesions (papules, nodules, and plaques) most often on face and upper extremities. Histologically, we note a diffuse infiltrate consisting predominantly of mature neutrophils that are typically located in the upper dermis without vasculitis. The cause of this syndrome remains unknown in most cases. However, it has been associated with inflammatory diseases, haematological or visceral malignancies, pregnancy, drug exposure, upper respiratory and gastrointestinal infections [4]. Taşkın et al. [2] reported the first case of sweet's syndrome concomitant with SARS-CoV-2 infection in a 61-yearold woman. Simar Berro et al. [3] have published sweet's

syndrome three weeks after a severe COVID-19 infection. The mechanism of Sweet's syndrome remains unknown; it seems to be related to a hypersensitivity reaction triggered by infections, neoplastic processes or by drug intake leading to the secretion of cytokines and activation of PNN. Granulocyte colony stimulating factors (G-CSF) is a specific factor in the growth of neutrophils that plays a crucial role in the pathogenesis of Sweet syndrome, however several cases of Sweet syndrome have been reported following administration of GM-CSF [5] and several studies have shown an increase in GM-CSF levels in the acute phase of Sweet syndrome. GM-CSF stimulates the production, activation and chemotaxis of neutrophils that accumulate in the dermis [6,7]. The pathogenesis of sweet syndrome in Covid 19 infection is unclear but is likely the result of cytokine storm and immunologic abnormalities. The aberrant host in response in COVID-19 infection was proposed to be centred around neutrophils. In autopsy specimens, neutrophil infiltration in pulmonary capillaries, extravasation of neutrophils to alveolar space and capillaritis with fibrin deposition were shown [8,9].

We describe here a typical Sweet's syndrome occurring three weeks after COVID-19 infection and we very likely think of a causal link between the two events given that associated malignancy and inflammatory bowel disease were ruled out and the possible involvement of the administered drugs was investigated, but no strong evidence was found in terms of extrinsic and intrinsic imputability.

Statement of ethics

The patient's consent was obtained for the pictures and eventual publication.

Disclosure statement

The authors have no conflicts of interest to declare.

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Fig. 1. Edematous purplish plaques of hands.

Author contributions

All authors also state that they have read and approved the final version of the manuscript.

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