unknown since only few cases of AA and early onset of telogen effluvium have been reported.[6–8]

The secretion of granzymes, perforins, granulysin and Fas ligand, which trigger programmed cell death, are common pathways involved in both viruses and alopecia areata inflammation.[5] COVID-19 could break the hair follicle immune privilege and activate $CD8^+$ cytotoxic cells and increase the secretion of IFN gamma, leading to an extensive immune response and cell disruption. An anagen effluvium during COVID-19 infection may represent a possible mechanism of the virus towards the hair follicle, as described in dengue (DENV). DENV is capable of causing a direct injury to the hair follicle leading to inflammation and cell death.[6–8]

Alopecia areata cases seem to have increased during the pandemic, but it is not clear whether this estimative is due to the psychological stress of the quarantine conditions or to subclinical infection of COVID-19.[8–10]

The continuous use of the JAK inhibitors in AA patients who develop COVID-19 infection is still controversial.

This case report demonstrates that, in patients with AA and COVID-19, the withdrawal of tofacitinib may lead to a persistent unresponsive anagen effluvium. The real effect of COVID-19 to hair follicle is still unclear, but the reactivation of the AA during COVID-19 infection may represent a possible mechanism of the virus towards the hair follicle immune privilege.

We recommend testing all patients with anagen or telogen effluvium, for COVID-19.

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## Conflict of interest

The authors have no conflict of interest to declare.

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# Updated international expert recommendations for the management of autoimmune bullous diseases during the COVID-19 pandemic

*Dear Editor,*

The SARS-CoV-2 pandemic has worsened since the publication of our initial recommendations for the management of autoimmune bullous diseases (AIBDs) during the COVID-19 outbreak in April 2020.[1] Based on the rapidly emerging increase in knowledge, this consensus of an expanded panel of international AIBD experts proposes updated recommendations to promote the optimal care of AIBD patients during the pandemic. The updated scientifically based guidance specifically pertains to the following questions:

**What do we recommend for AIBD patients considering the effects of immunomodulating therapy on SARS-CoV-2 infection?**

Patients with AIBDs treated with immunosuppressive therapies are generally prone to develop opportunistic infections,[2,3] which raised concerns that they could be more susceptible to SARS-CoV-2 infection and/or have worse COVID-19 outcomes.

Nevertheless, patients with AIBDs receiving immunomodulating therapies do not appear to have higher rates of manifest SARS-CoV-2 infection or more severe COVID-19 than the general population according to a systematic review of 732 AIBD patients on various immunomodulating treatments including rituximab.[4] While overall 9.5% of them had COVID-19 symptoms, 0.8% showed severe symptoms requiring hospitalization and 0.4% died of COVID-19, with the latter being elderly people and/or having comorbidities.[4] However, since it has been also reported that patients with AIBDs or rheumatic diseases diagnosed with and dying of COVID-19 were more likely to be receiving rituximab treatment and that each passing month from the last rituximab dose decreased the risk of getting COVID-19 and related hospitalization,[5–7] we currently do not recommend the use of rituximab as maintenance therapy to prevent relapses. This particularly applies to individuals who have not received a SARS-CoV-2 vaccine (see below). Therefore, while the decision to initiate B-cell depletion therapy remains to be made on a case-by-case basis, delays or obstructions in other important immunomodulatory treatments should be avoided during the pandemic, supporting our initial advice. Temporary changes in some immunosuppressive medications are still recommended in patients who have active COVID-19, as detailed in our previously suggested guidelines.[1]

**What should patients with AIBDs do to protect themselves from SARS-CoV-2 infection?**

Patients should continuously follow generally recommended measures to prevent SARS-CoV-2 infection (www.who.int, www.cdc.gov) and preferentially be managed with telemedicine instead of in-person visits where appropriate. The major advance in protection from COVID-19 is the recent advent of mRNA vaccines [from Pfizer-BioNTech and Moderna, authorized by the European Medicines Agency (EMA) and US Food and Drug Administration (FDA)] and adenoviral vector vaccines (from AstraZeneca and Johnson & Johnson, authorized by EMA and/or FDA), which induce an immune response to the SARS-CoV-2 spike protein, while additional vaccines are pending authorization or undergoing testing (www.who.int, www.cdc.gov). Therefore, it is recommended that every AIBD patient without contraindications to vaccination is immunized with one of the authorized vaccines to prevent COVID-19. Since the effect of AIBD treatment on the efficacy of COVID-19 vaccines is widely unknown, it is preferred to vaccinate patients while in remission or before planned immunosuppression, if feasible. In the case of rituximab, it is suggested to complete the entire vaccination series ≥4 weeks prior to the initiation of infusions or 12–20 weeks after completion of a rituximab cycle,[8] but the optimal time points are not clearly defined. Although vaccination is expected to be most effective when immunosuppression is low, we do not advise deliberately decreasing the patients' immunomodulatory medications before or during the vaccination period because of the risk of disease exacerbation.

Finally, it is worth mentioning that the European Academy of Dermatology and Venereology task force has initiated a registry for AIBD patients with confirmed COVID-19 (https://recovab.umcg.nl). This online registry, which is open to physicians worldwide, will help determine how the SARS-CoV-2 infection impacts patients with AIBDs and provide future recommendations.

## Conflicts of interest

Dr. Schmidt reports grants and personal fees from UCB, grants and personal fees from Biotest, grants from Incyte, grants from Euroimmun, personal fees from Novartis, grants and personal fees from ArgenX, personal fees from Astra Zeneca, grants and personal fees from Fresenius Medical Care, grants from Dompe, grants from Synthon, grants from Admirx and personal fees from Thermo Fisher, outside the submitted work; Dr. Amagai reports grants from Ono Pharmaceutical Company, grants from MBL and grants from RegCell, outside the submitted work; Dr. Fairley reports grants from National Institutes of Health and other from AstraZeneca, outside the submitted work; Dr. Murrell has served as a Principal Investigator and Advisor for trials with Principia Biopharma, Roche and Sanofi; Dr. Payne reports grants, personal fees and non-financial support from Cabaletta Bio and personal fees from Villaris Therapeutics, outside the submitted work; in addition, Dr. Payne has a patent Compositions and methods of chimeric autoantibody receptor T cells with royalties paid from Cabaletta Bio, a patent Compositions and methods for selective protein expression with royalties paid from Novartis, and a patent Method of redirecting T cells to treat HIV infection with royalties paid from Tmunity; Dr. Zillikens reports personal fees from UCB, Almirall, ArGEN-x, grants from Biotest, Euroimmun, Fresenius, personal fees from Biotest, Fresenius, Miltenyi, Roche, Biogen, Abbvie, UCB, Janssen, Novartis, outside the submitted work; Mr. Yale and Drs. Kasperkiewicz, Joly and Woodley have nothing to disclose.

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# Lichen planus arising after COVID-19 vaccination

Dear Editor,

A 56-year-old woman, with personal history of lichen planus 7 years prior that had been successfully treated with topical therapy, was referred to our dermatology outpatient unit from a primary care centre. She complained of pruritic lesions that had appeared 48 h after the second dose of the COVID-19 vaccine (Comirnaty, Pfizer, New York, NY, USA; BioNTech, Mainz, Germany). The lesions had developed in the ankles and subsequently extended to the flexural wrist and forearms, periumbilical area, mammary and axillary folds.

When asked about possible alternative triggers, the patient denied any signs of infection, changes in medication or stress in the weeks prior. Upon physical examination, she showed polygonal, well-delimited, erythematous papules in the ankles (Fig. 1), periumbilical area (Fig. 2), flexural wrist and forearms and mammary and axillary folds. Dermoscopy examination revealed slight desquamation and Wickham's striae. No mucosal or nail involvement was appreciated.

A punch biopsy of one of the papules on the arm was performed, showing typical findings of lichen planus with epidermal hyperplasia forming a characteristic saw-tooth appearance with wedge-shaped hypergranulosis, vacuolar degeneration of basal layer and dense lymphocytic infiltrate in the superficial dermis. The patient started treatment with high-potency topical corticosteroids.

Lichen planus is a T cell-mediated inflammatory disease of unknown origin. Its clinical presentation varies depending on the location in which it appears, affecting mostly the skin, where it shows as violaceous, shiny, polygonal, flat-topped papules that can be intensely pruritic. This condition has been related to previous exposure to certain agents such as drugs, vaccines and viruses.[1] Lately, it has also been related to the COVID-19 infection.[1] Hepatitis B virus (HBV), influenza, rabies, Diphtheria, Tetanus, Pertussis (DTaP) and measles, mumps and rubella (MMR) are some of the vaccines that have been previously associated with flares of lichen planus, with HBV being the most common agent implicated. The exact mechanism and the component responsible for this event are yet to be uncovered.[2]

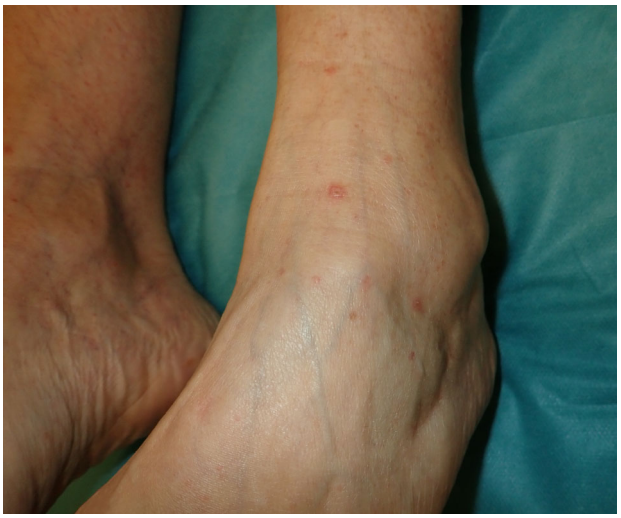

**Figure 1** Flat-topped, erythematous papules on the ankle.

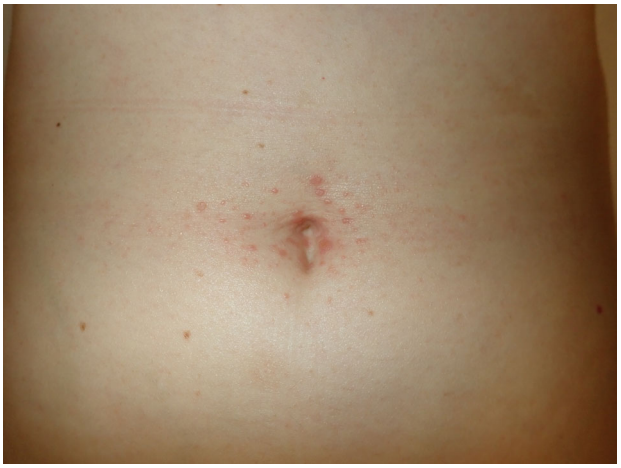

**Figure 2** Similar lesions located in the periumbilical area.