characterized by an exaggerated inflammatory response.[9] Another experiment in mouse has documented an inhibitory effect of apremilast on the release of profibrotic cytokine from macrophages, including interleukin-6.[10] During COVID19, pneumonia has been documented a 'cytokines storm', with markedly higher levels of IL-6, and TNF-α, suggesting the use of interleukin-6 receptor blocker tocilizumab in severe cases.[11] Recently, another Italian psoriasis patient contracting COVID-19 under IL-23 inhibitor treatment (guselkumab) has been reported, and completely recovered from the infection.[12]

From our experience, apremilast confirms its safety in very critical patients with severe infections, including COVID-19. Its efficacy in our sub-erythrodermic psoriasis was not completely satisfactory, but other treatments were contraindicated for the recurrent brain oligodendroglioma. Further studies are warrant to explore the intriguing immune modulating activities of this very manageable drug.

Acknowledgement

The patient in this manuscript has given written informed consent to the publication of his case details.

Funding source

none.

C. Mugheddu, L. Pizzatti, S. Sanna, L. Atzori,* F. Rongioletti

Dermatology Clinic, Department of Medical Sciences and Public Health, University of Cagliari, Cagliari, Italy

*Correspondence: L. Atzori. E-mail: atzoril@unica.it

References

1 Gisondi P, Piaserico S, Conti A, Naldi L. Dermatologists and SARS-CoV-2: The impact of the pandemic on daily practice. *J Eur Acad Dermatol Venereol* 2020. https://doi.org/10.1111/jdv.16515. [Epub ahead of print].
2 Tartari F, Guglielmo A, Fuligni F, Pileri A. Changes in emergency service access after spread of COVID19 across Italy. *J Eur Acad Dermatol Venereol* 2020. https://doi.org/10.1111/jdv.16553. [Epub ahead of print].
3 Radi G, Diotallevi F, Campanati A, Offidani A. Global coronavirus pandemic (2019-nCOV): implication for an Italian medium size dermatological clinic of a II level hospital. *J Eur Acad Dermatol Venereol* 2020. https://doi.org/10.1111/jdv.16386. [Epub ahead of print].
4 Atzori L, Mugheddu C, Addis G *et al*. Psoriasis health care in the time of the coronavirus pandemic: insights from dedicated centers in sardinia (Italy). *J Eur Acad Dermatol Venereol* 2020. https://doi.org/10.1111/jdv.16473. [Epub ahead of print].
5 Di Altobrando A, Patrizi A, Bardazzi F. Should SARS-CoV-2 influence immunosuppressive therapy for autoimmune blistering diseases? *J Eur Acad Dermatol Venereol* 2020. https://doi.org/10.1111/jdv.16491. [Epub ahead of print].
6 Price KN, Frew JW, Hsiao JL, Shi VY. COVID-19 and Immunomodulator/ Immunosuppressant use in dermatology. *J Am Acad Dermatol* **82**: e173–e175.
7 Zerilli T, Ocheretyaner E. Apremilast (Otezla): a new oral treatment for adults with psoriasis and psoriatic arthritis. *Pharm Ther* 2015; **40**: 459–500.
8 Sp R Shah VV, Wu Jj. Apremilast for a psoriasis patient with HIV and hepatitis C. *J Eur Acad Dermatol Venereol* 2017; **31**: e481–e482.
9 Imam F, Al-Harbi NO, Al-Harbi MM *et al*. Apremilast ameliorates carfilzomib-induced pulmonary inflammation and vascular injuries. *Int Immunopharmacol* 2019; **66**: 260–266.
10 Maier C, Ramming A, Bergmann C *et al*. Inhibition of phosphodiesterase 4 (PDE4) reduces dermal fibrosis by interfering with the release of interleukin-6 from M2 macrophages. *Ann Rheum Dis* 2017; **76**: 1133–1141.
11 Zhang C, Wu Z, Li JW, Zhao H, Wang GQ. The cytokine release syndrome (CRS) of severe COVID-19 and Interleukin-6 receptor (IL-6R) antagonist Tocilizumab may be the key to reduce the mortality. *Int J Antimicrob Agents* 2020: 105954. https://doi.org/10.1016/j.ijantimicag.2020.105954
12 Messina F, Piaserico S. SARS-CoV-2 infection in a psoriatic patient treated with IL-23 inhibitor. *J Eur Acad Dermatol Venereol* 2020. https://doi.org/10.1111/jdv.16468. [Epub ahead of print].

DOI: 10.1111/jdv.16625

Personal protective equipment induced facial dermatoses in healthcare workers managing Coronavirus disease 2019

Editor,

During the coronavirus disease 2019 (COVID-19) pandemic, frontline healthcare workers (HCW) are working tirelessly for long hours to provide patient care. Although COVID is not dermatotropic, prolonged contact with personal protective equipment (PPE, i.e. goggles, face-shield/visor, N 95 respirator, double-layered gloves, coverall/gowns, head cover and shoe cover) may cause various dermatoses. Several dermatoses have been reported due to PPE, such as pressure injury, contact dermatitis, pressure urticaria and exacerbation of pre-existing skin diseases, including seborrheic dermatitis and acne.[1,2] We report a preliminary data of HCW who experienced facial dermatoses due to the use of PPE.

From 24 March 2020 to 16 April 2020, we came across with 43 patients comprising physicians, nurses and paramedical staff who involved (directly/indirectly) in managing patients of COVID-19. We used telemedicine to consult these patients. Their history, clinical findings including onset, duration, location, clinical features and other associated symptoms of dermatoses and type of PPE used were recorded. However, patch could not be performed. Final diagnosis was based on history, clinical findings and pattern of dermatoses and symptoms.

The most commonly noted dermatoses were irritant contact dermatitis (ICD; 39.5%) followed by friction dermatitis (25.5%). Goggles were the most common culprit agent among all PPE causing any one of the dermatoses (51.92%), followed by N95

Table 1 Clinical features and other data of health care workers suffered from dermatoses

Variables	Number (*n*)	Percentage (%)
Number of patients	43	–
Mean age	32.78 ± 14.51	–
Sex (Male: Female)	1.39	–
Average duration PPE worn per day (hours):	8.76 ± 2.31	–
Average time after which rotation/doffing done (hours):	4.08 ± 1.01	–
Healthcare role:		
i. Doctors	31	72.09
ii. Nurses	9	20.93
iii. Miscellaneous like ward assistants/cleaners/ transport teams	3	6.98
Type of dermatoses:		
i. Irritant contact dermatitis	17	39.53
ii. Allergic dermatitis	03	6.98
iii. Pressure/friction marks/rhagades	11	25.58
iv. Sweat dermatitis	07	16.28
v. Facial acne	05	11.63
vi. Lip lick dermatitis	04	09.30
Site:		
i. Forehead	09	20.93
ii. Eyelids/canthus (goggle dermatitis)	12	27.91
iii. Nasal bridge (goggle and mask dermatitis/pressure dermatosis)	27	62.79
iv. Temple (Visor/mask straps)	09	20.93
v. Medial concha of ear (N95 respirator straps)	07	16.28
vi. Cheeks and chin	11	25.58
vii. Lips/angle of mouth	04	09.30
Symptoms:		
i. Itching	29	67.44
ii. Rash/redness	21	48.84
iii. Burning	11	25.58
iv. Smarting	08	18.60
v. Skin tightness/dryness	16	37.21
Signs:		
i. Erythema	23	53.49
ii. Vesicles	09	20.93
iii. Scaling	19	44.19
iv. Urticaria	02	04.65
v. Papules	07	16.28
vi. Pustules	04	09.30
vii. Pressure indentations and bruising	20	46.51
viii. Excessive lacrimation	08	18.60
ix. Rhinorrhoea	06	13.95
x. Skin discontinuity (Erosions/fissuring/excoriation)	31	72.09
Systemic features:		
i. Nausea	05	11.63
ii. Headaches	27	62.79
iii. Sneezing	04	09.30
iv. Feeling of intense heat	37	86.05
v. Claustrophobia/agitation with PPE suit	31	72.09
vi. Facial rubeosis/suffusion	09	20.93
Allergic predisposition	07	16.28
Treatment given:		
i. Spontaneous resolution with frequent breaks alone	08	18.60
ii. Topical calamine with aloe vera extracts	10	23.26
iii. Topical tacrolimus	09	20.93
iv. Topical low to mid potency steroids	04	09.30
v. Oat colloidal meal based moisturizer	24	55.81
vi. Petroleum jelly	04	09.30
vii. Systemic antihistamines	15	34.88
Patients requiring rescue steroids	04	09.30
Work absenteeism	09	20.93

masks (30.77%) and face shields (17.31%). Nasal bridge (63%) was the commonest anatomical site affected due to dermatoses followed by cheeks and chin (26%). However, there was a considerable overlap of different dermatoses with affliction of multiple sites. The most common symptom experienced by patients was pruritus (67.44%), while erythema (53.49%) was the most common sign observed. Interestingly, we observed two distinct dermatoses, i.e. whole face erythema (suffusion; 21%) attributed to doffing after a long shift and lip lick dermatitis due to constant licking of lips, because of feeling of intense thirst due to restricted fluid intake after donning PPE. The duration of wearing the goggles and mask, excessive sweating and ill-fitting masks, all were associated with increased sensation of irritation. Most of these dermatoses responded well to topical moisturizer, calamine lotion and oral antihistamines. Overall, 21% patients suffered from work absenteeism due to one of the dermatoses (Table 1).

Personal protective equipment-induced dermatoses occur mainly due to the occlusion and hyper-hydration effect of PPE and friction leading breach in the epidermal integrity.[1] Recently, in China, authors noted a very high prevalence, i.e. 97% of skin damages in first-line HCW fighting COVID-19.[3] Yin Z in his study found N-95 mask-induced pressure sore on the nasal bridge in HCW managing COVID-19 patients.[4] Skin barrier may create a portal of entry for COVID-19, as angiotensin-converting enzyme 2, the cell receptor for Severe acute respiratory distress-related Coronavirus-2 (SARS-CoV-2), is abundantly present in blood vessels of the skin and the basal layer of the epidermis.[5] As cases of COVID-19 are on rise with exponential

phase, we fear that the dermatoses-induced skin breach and irritation, and frequent touching of face due to latter, may increase the exposure and entry of SARS-CoV-2 infection in the healthcare workers.

Air-conditioning, proper fitting masks, use of better material (latex straps to be avoided) in the goggles and frequent rotation and regular breaks with removal of the mask and wiping of skin to remove sweat, may help in alleviation of dermatoses in HCW. Adequate hydration is useful to avoid dehydration-induced dermatoses and dry skin.[6] Moisturizers or emollients are needed to restore the integrity of skin barrier and should be applied at least 30 min before wearing mask, to prevent the damage to mask. Staff is advised not to smoke if they have applied emollient containing white soft paraffin, as it is flammable.[6] Low potency topical steroid or tacrolimus is required in some cases if above measures fail.[7]

M. Singh,[1] M. Pawar,[2,*] A. Bothra,[3] A. Maheshwari,[4] V. Dubey,[5] A. Tiwari,[6] A. Kelati[7]

[1]Department of Dermatology, JK Medical College & LN Hospital, Bhopal, India, [2]Department of Dermatology, MVP's Dr. V.P Medical College& Hospital & Research Center, Nashik, India, [3]Department of Dermatology, Gauhati Medical College & Hospital, Guwahati, India, [4]Consultant Dermatologist, Private Practice, New Delhi, India, [5]Department of Orthopaedic surgery, JK Medical College & LN Hospital, Bhopal, India, [6]Department of Medicine, All India Institute of Medical Sciences (AIIMS), Bhopal, India, [7]Cheikh Khalifa International University Hospital, Mohammed VI University of Health Sciences (UM6SS), Casablanca, Morocco

*Correspondence: M. Pawar. E-mail: manojpawar624@yahoo.com

References

1 Darlenski R, Tsankov N. Covid-19 pandemic and the skin - What should dermatologists know? *Clin Dermatol* 2020. https://doi.org/10.1016/j.clindermatol.2020.03.012. [published online ahead of print, 2020 Apr 18].

2 Gheisari M, Araghi F, Moravvej H, Tabary M, Dadkhahfar S. Skin reactions to non-glove personal protective equipment: an emerging issue in the COVID-19 Pandemic [published online ahead of print, 2020 Apr 18]. *J Eur Acad Dermatol Venereol* 2020. https://doi.org/10.1111/jdv.16492

3 Lan J, Song Z, Miao X *et al*. Skin damage among healthcare workers managing coronavirus disease-2019. *J Am Acad Dermatol* 2020; **82**: 1215–1216.

4 Yin Z. Covid-19: Countermeasure for N95 mask-induced pressure sore [published online ahead of print, 2020 Apr 18]. *J Eur Acad Dermatol Venereol* 2020. https://doi.org/10.1111/jdv.16490

5 Hamming I, Timens W, Bulthuis ML, Lely AT, Navis G, van Goor H. Tissue distribution of ACE2 protein, the functional receptor for SARS coronavirus. A first step in understanding SARS pathogenesis. *J Pathol* 2004; **203**: 631–637.

6 https://www.england.nhs.uk/coronavirus/wp-content/uploads/sites/52/2020/04/C0129_Preventing-skin-damage-under-PPE_9-April.pdf (last accessed: 17 April 2020).

7 https://www.bad.org.uk/shared/get-file.ashx?itemtype=document&id=6675 (last accessed: 17 April 2020).

DOI: 10.1111/jdv.16628

LETTERS TO THE EDITOR

Effectiveness and safety of dupilumab for the treatment of atopic dermatitis in adult cohort: a real-life Italian tertiary centre experience

Dear Editor,

Atopic dermatitis (AD) is a chronic inflammatory skin disease affecting 7–10% of adults.[1] The systemic treatment for moderate-to-severe AD was limited, but a new biologic drug (dupilumab) was recently approved. It is a fully human monoclonal antibody directed against the alpha-subunit of the interleukin-4 receptor, blocking signalling of both Th2 cytokines IL-4 and IL-13. The effectiveness and safety were demonstrated in clinical trials,[2–4] but these studies do not reflect conditions in daily practice.

The aim of this study was to analyse the activity of dupilumab in a real-world setting. A total of 128 adult patients were enrolled prospectively between January and September 2019, with moderate-to-severe AD [Eczema Area and Severity Index (EASI) ≥24, or less, but with involvement of sensitive areas], collected from the Dermatology Clinic of the University of Turin. All patients received dupilumab at standard dose, due to inefficiency, side-effects or contraindication of cyclosporine.

This study was approved by the local ethical committee of the Turin University Hospital (No.CS2/1359). Clinical documentation was collected at baseline and every 16 weeks by validated scores[5]: EASI, Scoring Atopic Dermatitis (SCORAD), Investigator Global Assessment (IGA), Patient Oriented Eczema Measure (POEM) and Dermatology Life Quality Index (DLQI). Itch and sleep disorders were evaluated on a numerical rating scale (itch-NRS and sleep-NRS), as peak value during the previous 24 h. Furthermore, for the first month, the peak of itch-NRS was completed daily by the patients[2] to evaluate the weekly median reduction of the values. In addition, during follow-up, blood chemical tests and adverse events (AEs) were collected.

The baseline characteristics are reported in Table 1. Males, at baseline, reported a significantly higher mean EASI (M : F = 29.2 : 24.5-P = 0.01), mean SCORAD (M : F = 65.8 : 60.4-P = 0.01), total IgE mean levels (M : F = 3958 : 1977-P = 0.007) and LDH mean value (M : F = 372 : 271-P = 0.02), while no difference in itch-NRS, sleep-NRS or eosinophil count was detected. However, females reported higher mean POEM (M : F = 19.4 : 21.7-P = 0.03). No patients were previously included in clinical trials and all were treated with conventional treatment (95% cyclosporine).