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Data available on request from the authors.

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Primary cutaneous lymphoma and risk for severe COVID-19: a prospective study of 48 cases in Morocco

Editor,

Primary cutaneous lymphomas (PCLs) are rare non-Hodgkin's lymphomas that are present in the skin without any extracutaneous involvement at the time of initial diagnosis.[1] The group of PCLs shows distinct clinical, histological, immunophenotypic and genetic characteristics.[2]

Coronavirus Disease 2019 (COVID-19) is the disease caused by SARS-CoV-2 infection. It has been accelerating since the beginning of 2020 and is still challenging the healthcare systems worldwide.

Studies suggest that patients with older age and malignancy have a higher risk of severe events including death due to COVID-19.[3,4] Patients with primary cutaneous lymphoma receive immunosuppressive therapy long term for disease control, have potential underlying predisposing conditions (e.g. hypertension and diabetes) and tend to be older.

There are no enough data in the literature about COVID-19 infection and cutaneous lymphomas.

The aims of our study were to evaluate the incidence of COVID-19 and severe outcomes of patients with PCL, and describe changes in lymphoma staging after COVID-19.

We performed a prospective study of patients with PCL at the Dermatology venerology Department, Military Hospital Instruction Mohammed V between June 2020 and June 2021.

We collected all patients with COVID-19 and described their clinical data and evolution. All statistical calculations were performed using Jamovi ver. 2.2.2.

Table 1 Baseline clinical characteristics and evolution of the PCL patients COVID positive (n = 36)

Variables	Characteristics
Comorbidity	
Smoking	4 (11%)
Diabetes mellitus	10 (27%)
Hypertension	8 (22%)
Bronchial asthma	2 (5%)
Ischaemic heart disease	3 (8%)
Clinical manifestations	
Diarrhoea	11 (30%)
Abdominal pain	8 (22%)
Anosmia	14 (38%)
Dysgeusia	10 (27%)
Fever	27 (75%)
Dyspnoea	7 (19%)
Headache	18 (50%)
Fatigue	19 (52%)
Rhinorrhoea	6 (16%)
Skin rash	1 (2%)
COVID formes	
Mild	9 (25%)
Moderate	12 (33%)
Severe	15 (42%)
Outcome at end of lock down	
Improvement	2 (6%)
Stationary	30 (85%)
Progression	4 (9%)

COVID-19 outcomes included mild, moderate and severe. This classification was established based on clinical, biological and radiographic evidence:

- Mild Illness: Individuals who have any of the various signs and symptoms of COVID-19 but who do not have shortness of breath, dyspnoea or abnormal chest imaging.
- Moderate Illness: Individuals who show evidence of lower respiratory disease during clinical assessment or imaging and who have an oxygen saturation (SpO2) ≥ 94%.
- Severe Illness: Individuals who have SpO2 < 94% on room air at sea level.

Our study included 48 patients (96% Mycosis fungoides/Sézary (MF/SS), 4% non-MF/SS primary cutaneous lymphomas); 40% were IA stage, and 21 patients (60%) received systemic treatment.

Nine Patients (18%) did not receive COVID-19 vaccines, 17 patients (35%) received partial immunization and 22 patients (45%) received complete immunization.

Thirty-six patients (75%) suffered from COVID-19, median age of 55.2 years (SD = 15.5); The sex ratio of males to females was 2.9.

The most common clinical manifestations of COVID-19 were fever (75%), fatigue (52%), headache (50%), anosmia (38%), diarrhoea (30%), dysgeusia (27%) and dyspnoea (19%).

Comorbidities including smoking, diabetes, hypertension, bronchial asthma and ischaemic heart disease were common in the PCL patients COVID-19 positives.

All baseline clinical characteristics and evolution are listed in Table 1.

The haematological and coagulation parameters are presented in Table 2. In all, 25% patients experienced mild disease, 33% moderate disease and 42% severe disease: 46% required hospitalization, 26% needed ICU and 20% died.

The proportion of mortality in our patients is 8% vs. 1.7% in our Hospital.

Totally 85% of patients reported stability of their PCLs, 6% improvement and 9% worsening.

There were limited data regarding PCL and COVID-19.

Table 2 Laboratory data of PCL patients COVID-19 (*n* = 48)

Laboratory data	Normal range	COVID-19 Negative (*n* = 12)	COVID-19 Positive (*n* = 36)	*P* value
Creatinine (mg/L)	6–13	8.83 ± 1.64	11.1 ± 2.57	0.008*
CRP (mg/L)	<5	5.67 ± 2.10	234 ± 62.4	<0.001*
ALT (Units/L)	<40	36.92 ± 14.24	34.1 ± 8.71	0.409
AST (Units/L)	<35	31 ± 9.18	29.9 ± 15.3	0.858
LDH (Units/L)	125–243	174.58 ± 29.17	343 ± 78.8	<0.001*
hs Troponin-I (ng/L)	2–34	12.33 ± 8.8	29.7 ± 15.9	<0.001*
Serum Ferritin (ng/mL)	23.9–336.2	133 (53.3–263)	554 (467–847)	<0.001*
Fibrinogen (g/day)	2–4	4.53 ± 2.68	5.02 ± 1.57	0.561
WBC (× 10^3/μL)	4–10	9.24 ± 6.71	9.48 ± 5.53	0.918
Hb (g/dL)	13–17	11.13 ± 2.47	12.27 ± 5.65	0.341
Platelet (× 10^3/μL)	150–450	258.90 ± 136.12	219.37 ± 111.14	0.407
Lymphocytes (× 10^3/μL)	1.5–4	1.78 ± 1.19	4.56 ± 17.3	0.319

*$P < 0.05$.

Our study suggests that patients who suffer from PCLs may represent a risk group for potential life-threatening complications in case of infection with SARS-CoV-2.

Risk factors for infections in PCL patients include lymphopenia, chronic organ failure (renal, cardiac or respiratory), Sezary syndrome, other comorbidities (e.g. diabetes and hypertension), aggressive immunosuppressive treatment, advanced/aggressive disease and older age leading to severe COVID-19 symptoms.[5]

Several guidelines for the management and the treatment of cutaneous lymphoma during the COVID-19 pandemic have been recently established by the United States CL Consortium and the EORTC CLTF.[6,7]

In conclusion, physicians should consider that PCL patients are at risk for severe COVID-19; therefore, reinforced preventive measures and prioritization in vaccination strategies are required.

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Data availability statement

Data openly available in a public repository that issues datasets with DOIs.

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Case of lichen planus pigmentosus–inversus after Oxford–AstraZeneca COVID-19 vaccine: cause or coincidence?

Dear Editor,

While the COVID-19-vaccinated population is increasing globally, more vaccine-associated cutaneous adverse events are reported. Immunogenic effects of vaccines lead to altered levels of chemokines and cytokines, which activate different key players of the immune system. The skin and mucosa as boundary surfaces to the environment are largely affected by the general activation of the immune system sparked by vaccines.[1] Lichen planus (LP) has been documented after COVID-19 mRNA vaccines.[2,3] Recently, a lichenoid eruption after Oxford–AstraZeneca COVID-19 vaccine was also described.[4]

We report a peculiar variant of lichen planus after Oxford–AstraZeneca vaccination.

A 64-years-old phototype III women, with no relevant past medical history, presented to our department with a three-month history of persistent hyperpigmented lesions in the intertriginous areas. The lesions were slightly pruritic and developed 2 weeks after the inoculation of the first dose of Oxford–AstraZeneca COVID-19 vaccine. She did the second dose of the same vaccine and reported clinical worsening. She denied prior history of medication use, trauma or sun exposure, and her family history was unremarkable. Physical examination revealed symmetrically distributed, dark-brown macules, papules and plaques on the folds (Fig. 1). Mucous membranes and nails were not involved. Routine laboratory tests and hepatitis serology showed no anomalies. A skin biopsy of the lumbar area was consistent with lichen planus (Fig. 2). Based on the clinical picture and the histopathology findings, the diagnosis of lichen planus pigmentosus–inversus (LPPI) was made. Topical betamethasone 0.05% ointment was prescribed, and a minor clinical improvement was observed after two months of follow-up, such as a decline in pigmentation.

LPPI, first described in 2001 by Pock et al.,[5] is a rare subvariant of lichen planus pigmentosus (LPP), with only a few cases reported in medical literature so far. It has been most frequently reported in light-skinned patients with the development of asymptomatic to slightly pruritic, hyperpigmented patches in an intertriginous distribution.[6] The histopathology is generally characterized by a hyperorthokeratotic epidermis with variably band-like inflammatory infiltrate on the superficial dermis, containing lymphocytes and histiocytes with a prominent pigmentary incontinence.[6,7] The pathogenesis of LPPI was suggested to be related to a T-lymphocyte-mediated, cytotoxic activity against basal keratinocytes, similar to classic LP.[8] Although the exact aetiology of LPP remains