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DOI: 10.1111/jdv.15212

Psoriasis dermatitis: an overlap condition of psoriasis and atopic dermatitis in children

To the Editor,

Some children present with a so-called overlap condition with clinical features of both psoriasis and atopic dermatitis (AD), previously described as 'psoriasis dermatitis' (PD).^{1–3} Though its existence is not questioned in literature, only one study demonstrated clinical characteristics of children with coexisting psoriasis and AD,² and its course over time has not been described so far.

We conducted a single-centre cohort study to assess demographical information, clinical characteristics and follow-up of children with PD (defined as children presenting with clinical features of psoriasis and AD at two or more consecutive consultations) seen at our paediatric psoriasis outpatient clinic between November 2010 and September 2017. The PD population was compared to children included in the Child-CAPTURE registry, a long-term prospective daily practice cohort of children with a confirmed diagnosis

of psoriasis.⁴ This study has been approved by the local ethics committee. A two-tailed $P < 0.05$ was considered statistically significant.

Fifteen patients {86.7% female, median age 7.4 years [interquartile range (IQR) 7.4]} presented with PD (3.7% of the patients referred to our paediatric psoriasis outpatient clinic; Table 1). Comparison of children with PD vs. children with psoriasis (Child-CAPTURE cohort; $n = 395$) showed a significantly greater proportion of females in the PD cohort (86.7% vs. 57.2%; $P = 0.030$) and a younger age at first consultation [median age 7.4 (7.4) vs. 11.1 (6.9); $P = 0.028$]. A positive family history of atopic disease was more frequently reported by children with PD vs. psoriasis (53.3% vs. 20.2%; $P = 0.007$), whereas the family history for psoriasis did not differ between groups. None of the children with PD reported a positive Koebner phenomenon (0.0% vs. 29.1%; $P = 0.023$). There was no substantial difference in the presence or intensity of itch between groups (80.0% vs. 87.5%; $P = 0.421$; VAS itch 59.5 vs. 47.0; $P = 0.392$). With respect to the location of skin lesions, the PD cohort presented more often with facial lesions than the psoriasis cohort (33.3% vs. 11.1%; $P = 0.024$). The scalp, however, was less frequently involved in PD vs. psoriasis patients (46.7% vs. 76.2%; $P = 0.015$). Nail changes did not differ between groups (26.7% vs. 18.2%; $P = 0.494$). When the presence of AD characteristics was retrospectively scored with the diagnostic criteria of Hanifin and Rajka (HR),⁵ the PD population met remarkably more AD criteria compared to the paediatric psoriasis population [PD cohort 4.0 (3.0); psoriasis cohort 3.0 (1.0), $P = 0.008$; Fig. 1a]. In contrast, psoriasis criteria (17 criteria based on characteristics of paediatric psoriasis described in literature, data available upon request) were less frequently reported by children with PD compared to psoriasis [PD cohort 4.0 (2.0); psoriasis cohort 5.0 (1.0), $P = 0.001$; Fig. 1b].

Median follow-up time of children with PD was 1.2 years (2.55), in which patients had between two and 11 consultations [median visit number 5.0 (4.0)]. In this period, characteristics of both AD and psoriasis persisted and the treating physicians could not assign the diagnosis of either AD or psoriasis. Only one patient was diagnosed with AD after two follow-up visits.

Recent publications on underlying pathogenic disease processes of AD reported similarities in immune activation in psoriasis and AD.^{6–8} Based on these publications, it was hypothesized that AD, psoriasis and associated subtypes might exist across a disease spectrum, resulting in overlapping disease characteristics.⁹ This concept, primarily based on pathogenic disease mechanisms, is supported by our clinical work. Future research to further identify underlying immune activation in children with PD could strengthen the hypothesis of a disease spectrum.

In conclusion, 4% of children referred to our paediatric psoriasis outpatient clinic showed clinical features of both

Table 1 Characteristics of children with psoriasis dermatitis (PD) vs. psoriasis at baseline (*N* = 410)

Characteristics	No. (%) of patients with PD (<i>N</i> = 15; 3.7%)	No. (%) of patients with psoriasis (<i>N</i> = 395; 96.3%)	<i>P</i> -value
Sex			
Male	2 (13.3)	169 (42.8)	0.030
Female	13 (86.7)	226 (57.2)	
BMI (<i>N</i> = 306)[†]			
Normal	11 (64.7)	229 (78.2)	0.430
Overweight	4 (23.5)	41 (14.0)	
Obesity	2 (11.8)	23 (7.8)	
Family history[‡]			
Psoriasis	4 (26.7)	138 (34.9)	0.592
Atopic disease [§]	8 (53.3)	83 (20.2)	0.007
Trigger factors (<i>N</i> = 411)			
No trigger	14 (93.3)	263 (66.6)	0.282
Infection	0 (0.0)	60 (15.2)	
Stress	1 (6.7)	51 (12.9)	
Koebner phenomenon (<i>N</i> = 369)[¶]	0 (0.0)	102 (29.1)	0.023
Itch	12 (80.0)	344 (87.5)	0.421
Allergies	3 (20.0)	78 (19.7)	1.000
Atopy (<i>N</i> = 401)[§]	2 (13.3)	83 (21.5)	0.747
Localization of skin lesions^{††}			
Facial	5 (33.3)	44 (11.1)	0.024
Scalp	7 (46.7)	301 (76.2)	0.015
Trunk	5 (33.3)	225 (57.0)	0.109
Upper extremities	9 (60.0)	277 (57.5)	1.000
Lower extremities	11 (73.3)	231 (58.5)	0.296
Body folds	5 (33.3)	150 (38.0)	0.793
Palmoplantar	1 (6.7)	2 (0.5)	0.106
Nails	4 (26.7)	72 (18.2)	0.494
	Median (IQR) in patients with PD (<i>N</i> = 15)	Median (IQR) in patients with psoriasis (<i>N</i> = 395)	<i>P</i>-value
Age, years	7.4 (7.4)	11.1 (6.9)	0.028
Age at onset, years	4.9 (10.0)	7.0 (6.0)	0.194
BMI (<i>N</i> = 306)	16.6 (4.6)	17.8 (5.9)	0.142
Severity at baseline			
BSA (<i>N</i> = 403)	3.5 (9.5)	5.4 (8.5)	0.157
CDLQI (0–30; <i>N</i> = 392)	5.5 (7.0)	8.0 (7.0)	0.095
VAS			
Disease severity (<i>N</i> = 243)	51.0 (36.0)	58.0 (39.0)	0.373
Itch (<i>N</i> = 309)	59.5 (35.0)	47.0 (50.0)	0.392
Pain (<i>N</i> = 309)	2.5 (4.0)	4.0 (25.0)	0.486
Fatigue (<i>N</i> = 307)	25.0 (37.0)	11.0 (44.0)	0.243

P-values printed in bold were regarded statistical significant ($P < 0.05$)

[†]Body mass index cut-off points for normal weight, overweight and obesity are based on the extended International Obesity Task Force (IOTF).¹⁰

[‡]Family history of psoriasis or atopy in parents or siblings.

[§]Atopy was defined as the presence of atopic dermatitis, and/or allergic asthma and/or allergic rhinitis.

[¶]Positive Koebner phenomenon was defined as having an isomorphic response regularly or often.

^{††}Total number of patients does not equal sum of patients reporting different localizations because more than one localization can be reported in the same patient.

BMI, body mass index; BSA, body surface area; CDLQI, Children's Quality of Life Index; VAS, visual analogue scale.

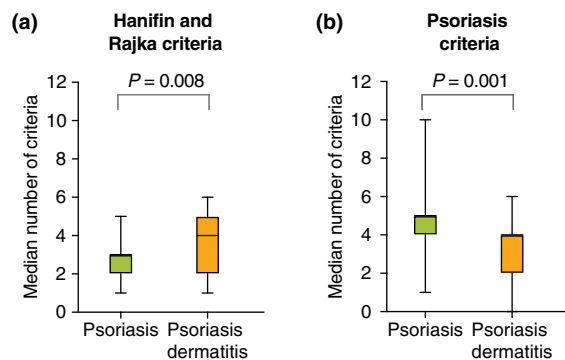


Figure 1 Median number of diagnostic AD and psoriasis criteria met in children with psoriasis dermatitis ($N = 15$) vs. psoriasis ($N = 395$) at baseline. (a) Median number of diagnostic AD criteria (Hanifin and Rajka criteria⁵) met in children with psoriasis dermatitis ($N = 15$) vs. psoriasis ($N = 395$) at baseline. (b) Median number of diagnostic psoriasis criteria met in children with psoriasis dermatitis ($N = 15$) vs. psoriasis ($N = 395$) at baseline (17 criteria based on characteristics of paediatric psoriasis; based on Kapila *et al.*²; Lysell *et al.*¹¹; Bronckers *et al.*¹²; Burden-Teh *et al.*¹³; Cohen *et al.*¹⁴; Fortina *et al.*¹⁵).

psoriasis and AD, even after a longer follow-up. Recognition of PD as a distinct subtype not only adds to the scientific insight in both AD and psoriasis, but is also important for affected patients and their parents, as PD patients might need different therapeutic interventions from paediatric psoriasis patients.

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DOI: 10.1111/jdv.15213

Chromoblastomycosis: an autochthonous case of a tropical disease

Editor

Chromoblastomycosis (CBM) is a chronic granulomatous fungal infection caused by melanized or brown-pigmented fungi. It is mainly a tropical or subtropical disease. CBM is considered an implantation mycosis because its mode of infection comprises transcutaneous inoculation from an environmental source. The group of implantation mycosis includes other more aggressive fungal infections such as phaeohyphomycosis (PHM). Several species of the genera *Exophiala*, *Cladophialophora*, *Fonsecaea*, *Phialophora* and *Rhinocladiella* are possible aetiological agents of CBM and PHM.¹ We report a case of a patient who acquired CBM in a non-tropical area after bipulmonary transplant.

The patient was a 52-year-old man with a history of diffuse interstitial lung disease who had received his second bipulmonary transplant 30 days ago after the first one complicated with a bronchopulmonary aspergillosis. He presented with an asymptomatic cutaneous lesion with progressive ulceration in his right forearm where an intravenous line had been inserted. Physical examination disclosed a 4 × 2.5-cm-diameter violaceous plaque, with ulceronecrotic tissue and exudative small abscesses (Fig. 1a).