Patients with bullous pemphigoid and comorbid psoriasis present with less blisters and lower serum levels of anti-BP180 autoantibodies

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Abstract

Background Although the association of bullous pemphigoid (BP) and psoriasis is well-established, the clinical and immunological features of patients with coexisting BP and psoriasis are yet to be investigated.

Objective We aimed to estimate the prevalence of psoriasis amongst patients with BP and to elucidate the clinical and immunological characteristics of BP patients with comorbid psoriasis.

Methods A retrospective cohort study including all consecutive patients diagnosed with BP throughout the years 2009–2019 in a tertiary referral centre.

Results The study encompassed 273 patients with BP, of whom 11 (4.0%; 95% CI, 2.3–7.1%) had comorbid psoriasis. The onset of psoriasis preceded that of BP in 81.8% of patients by a median (range) latency of 26.5 (5.0–34.0) years. Compared to BP patients without psoriasis, those with BP and comorbid psoriasis were significantly younger at the onset of BP [71.8 (9.3) vs. 79.4 (9.8) years; \(P = 0.023\)], had a milder erosive phenotype [erosion/blister BPDAI mean (SD) score; 5 (4.1) vs. 22.3 (15.2); \(P = 0.025\)], lower levels of anti-BP180 NC16A serum autoantibodies [236.6 (266.3) vs. 556.2 (1323.6) U/mL; \(P = 0.008\)] and a higher prevalence of isolated linear C3 deposits (36.4% vs. 14.1%; \(P = 0.043\)) and a lower prevalence of linear immunoglobulin G deposits (36.4% vs. 68.7%; \(P = 0.025\)) along the dermal–epidermal junction by direct immunofluorescence microscopy.

Conclusions Patients with BP and comorbid psoriasis present at a younger age with milder erosive phenotype and lower levels of pathogenic autoantibodies.

Conflict of interest None.

Funding sources Clinical Research Unit Pemphigoid Diseases (KFO 303) and Cluster of Excellence Precision Medicine in Chronic Inflammation (EXC 2167), both from the Deutsche Forschungsgemeinschaft.

Introduction

The coexistence of psoriasis and autoimmune bullous diseases (AIBD) was described as early as 1929. Amongst these diseases, bullous pemphigoid (BP) and anti-p200 pemphigoid were most frequently reported to associate with psoriasis. Four controlled observational studies depicted a statistically significant association between BP and psoriasis. The findings of these studies were subsequently synthesized in a meta-analysis demonstrating that the pooled prevalence of psoriasis was 2.5-fold increased amongst patients with BP.

Despite the robust epidemiological relationship binding these conditions, it is yet to be determined whether BP patients with coexistent psoriasis are characterized by a distinct clinical and immunological profile. Moreover, the prevalence of psoriasis amongst patients with BP varied considerably between different populations and ranged between 2.1% and 11.3%, implying for the presence of ethnic predisposition to this association. Of note, this association is yet to be evaluated amongst patients in Germany.
The aim of the current study was to estimate the prevalence of psoriasis amongst a large cohort of German patients with BP, and to delineate the clinical and immunological characteristics of BP patients with comorbid psoriasis relative to other patients with BP.

**Methods**

**Study population and definition of patients**
We conducted a retrospective study including all patients diagnosed with BP between 1 January 2009 and 28 February 2020, at the Department of Dermatology, University of Lübeck, Germany. The current study was approved by the institutional review board (20-110A).

The diagnosis of BP was established based on the following criteria: (i) suggestive clinical characteristics; (ii) linear deposits of IgG and/or C3 along the dermal–epidermal junction (DEJ) by direct immunofluorescence (IF) microscopy of a perilesional skin biopsy; and (iii) detection of circulating autoantibodies binding to the epidermal side of 1 mL NaCl-split normal human skin by indirect IF microscopy and/or the presence of circulating IgG autoantibodies against BP180 and/or BP230, as identified by enzyme-linked immunosorbent assay (ELISA).8,9 The diagnosis of psoriasis was grounded on a suggestive clinical presentation, with histological analyses preserved for morphologically uncertain cases.

**Definition of covariates**

The severity of disease was assessed based on the Bullous Pemphigoid Disease Area Index (BPDAI).10 This score had been documented, including four subcomponents (cutaneous erosion/blister activity, cutaneous urticaria/erythema activity, damage and pruritus). Since this scoring system was introduced only in 2012, BPDAI scores were available for only 132 out of 273 (48.4%) patients.

The levels of circulating anti-BP180 NC16A and anti-BP230 autoantibodies were measured utilizing commercial ELISA systems (Euroimmun, Lübeck, Germany). Seropositivity was defined based on the cut-off values proposed by the manufacturer (i.e., 20 U/mL). Of note, direct IF, immunoserological essays, eosinophil counts and C-reactive protein levels were measured at the onset of the diseases prior to the administration of any new systemic therapy. The latter two biomarkers were analysed since evidence suggests that peripheral eosinophilia may predict diseases severity11 and that acute phase reactants are associated with prognostic outcomes in BP.12

**Statistical analysis**

Baseline characteristics are described by means and standard deviations (SDs) for continuous variables, whilst categorical values were signified by percentages. The comparison of clinical and immunological variables between subgroups was performed using the chi-square test and t-test for categorical and continuous variables, respectively. SPSS software, version 25 (SPSS: IBM Corp, Armonk, NY, USA), was utilized to conduct all statistical analyses.

**Results**

**Demographic characteristics of the study population**

The study population encompassed 273 patients with incident BP, of whom 119 (43.6%) were males and 154 (56.4%) females. The average age (SD) at diagnosis was 79.1 (9.9) years, and the median (range) age was 80.4 (49.6–98.2) years.

**Burden of psoriasis amongst patients with BP**

Out of the study population, 11 patients (4.0%; 95% CI, 2.3–7.1%) presented with comorbid psoriasis. The onset of psoriasis preceded that of BP in 9 (81.8%) patients with a dual diagnosis, whereas it followed BP in the remaining two (18.2%) patients. The median (range) latency separating the conditions was 26.5 (5.0–34.0) years in cases in which BP followed psoriasis and 4.5 (2.0–7.0) years when BP preceded psoriasis.

Amongst patients with comorbid BP and psoriasis, plaque type psoriasis was the most frequently encountered subtype of psoriasis (n = 8; 72.7%), followed by inverse psoriasis (n = 2; 18.2%), and palmoplantar pustular psoriasis (n = 1; 9.1%). Psoriatic arthritis was present in one (9.1%) patient and nail involvement in one (9.1%) patient. In two (18.2%) patients, the bullous lesions of BP and the plaques of psoriasis co-localized (Fig. 1).

Out of the nine patients developing BP following psoriasis, six (66.7%) had active psoriasis at the presentation with BP. With regard to anti-psoriatic therapies preceding the diagnosis of BP,
### Table 1

| Age at diagnosis; years | BP with comorbid psoriasis (n = 11) | BP without comorbid psoriasis (n = 262) | P value |
|-------------------------|-------------------------------------|----------------------------------------|---------|
| Mean (SD)               | 71.8 (9.3)                          | 79.4 (9.8)                             | 0.023   |
| Median (range)          | 75.0 (60.7-84.0)                    | 80.5 (49.6-98.2)                       |         |
| **Sex, n (%)**          |                                     |                                        |         |
| Male                    | 5 (45.5)                            | 113 (43.1)                             | 0.875   |
| Female                  | 6 (54.5)                            | 149 (56.9)                             |         |
| **Distribution of bullous lesions; n (%)** | | | |
| Head and neck           | 4 (36.4)                            | 72 (27.5)                              | 0.520   |
| Trunk                   | 11 (100)                            | 195 (74.4)                             | 0.054   |
| Limbs                   | 9 (81.8)                            | 220 (84.0)                             | 0.846   |
| Hands/feet              | 3 (27.3)                            | 106 (40.5)                             | 0.382   |
| Mucous membranes        | 1 (9.1)                             | 30 (11.5)                              |         |
| **DPP4i-associated BP, n (%)** | | | |
|                          | 0 (0.0)                             | 24 (9.2)                               | 0.293   |
| **Mean BPDAI severity score (SD)** | | | |
| Erosion/blister cutaneous activity | 5.0 (4.1)                        | 22.3 (15.2)                             | 0.025   |
| Urticaria/Erythema activity | 8.0 (4.1)                       | 12.6 (15.4)                            | 0.553   |
| Pruritus score          | 21.8 (7.1)                          | 19.0 (9.1)                             | 0.498   |
| Damage score            | 0.8 (1.3)                           | 2.2 (3.2)                              | 0.120   |
| **Main comorbidities, n (%)** | | | |
| Hypertension            | 5 (45.5)                            | 161 (61.5)                             | 0.288   |
| Diabetes mellitus       | 2 (18.2)                            | 73 (27.9)                              | 0.481   |
| Ischaemic heart disease | 3 (27.3)                            | 61 (23.3)                              | 0.760   |
| Dementia                | 1 (9.1)                             | 58 (22.1)                              | 0.305   |
| Cerebrovascular disease | 1 (9.1)                             | 29 (11.1)                              | 0.835   |
| Parkinson              | 0 (0.0)                             | 12 (4.6)                               | 0.488   |
| **Treatment modalities, n(%)** | | | |
| Topical corticosteroids monotherapy | 3 (27.3)                        | 35 (13.4)                              | 0.193   |
| Systemic corticosteroids >1 mg/kg | 0 (0.0)                       | 7 (2.7)                                | 0.582   |
| Systemic corticosteroids | 2 (18.2)                        | 83 (31.7)                              | 0.345   |
| Adjuvant immunosuppressive and immunomodulatory agents | 8 (72.7)                      | 204 (77.9)                             | 0.685   |

Bold: significant values.

BP, bullous pemphigoid; BPDAI, bullous pemphigoid disease area index; DPP4i, dipeptidyl peptidase-4 inhibitors; n, number; SD, standard deviation.

†BPDAI score was calculated in 4 BP patients with coexistent psoriasis and 128 BP patients without psoriasis.

Three (33.3%) patients had been managed by phototherapeutic modalities, but only one (11.1%) patient was exposed to it in proximity to the onset of BP [a 62-year-old man completing narrowband ultraviolet (UV) B course 3 months prior to BP]. Whilst four patients (44.4%) had no previous systemic anti-psoriatic medications, two (22.2%) patients were managed by secukinumab, one patient (11.1%) by certolizumab and one (11.1%) patient by acitretin at the onset of BP.

**Clinical and immunological profile of BP patients with comorbid psoriasis**

We aimed to assess whether patients with BP and comorbid psoriasis (n = 11) are typified by distinct clinical and immunopathological features as compared to the remaining patients with BP (n = 262). Patients with comorbid psoriasis were significantly younger at the onset of BP [71.8 (9.3) vs. 79.4 (9.8) years, respectively; P = 0.023], whilst the sex distribution was comparable between the two subgroups. No significant differences were observed in terms of the anatomical distribution of bullous lesions, the prevalence of mucosal involvement and the prevalence of dipeptidyl peptidase-4 inhibitor (DPP4i)-associated BP (Table 1).

When the disease severity was compared, patients with comorbid psoriasis were found to present with milder erosive phenotype, as estimated by lower erosion/blister BPDAI score [5.0 (4.1) vs. 22.3 (15.2), respectively; P = 0.025]. No differences were revealed between both subgroups in the severity of the erythematosus phenotype (as estimated by urticaria/erythema BPDAI) or in the pruritus and damage components of the BPDAI score (Table 1). It is noteworthy, the BPDAI score was
Table 2 Immunological characteristics and laboratory findings of BP patients with comorbid psoriasis as compared to BP patients without psoriasis

|                          | BP with comorbid psoriasis (n = 11) | BP without comorbid psoriasis (n = 262) | P value |
|--------------------------|------------------------------------|---------------------------------------|---------|
| **BP180 NC16A ELISA†**   |                                    |                                       |         |
| Seropositivity, n (%)    | 8 (72.7)                           | 212 (83.1)                            | 0.373   |
| ELISA value, mean (SD); U/mL| 236.6 (266.3)                      | 556.2 (1323.6)                       |         |
| **BP230 ELISA†**         |                                    |                                       |         |
| Seropositivity, n (%)    | 3 (60.0)                           | 38 (48.1)                             | 0.608   |
| ELISA value, mean (SD); U/mL| 44.0 (67.1)                        | 119.3 (292.9)                        |         |
| **Linear deposits of immunoreactants by direct immunofluorescence** | | | |
| IgG, n (%)               | 4 (36.4)                           | 180 (68.7)                            | 0.025   |
| IgA, n (%)               | 0 (0.0)                            | 22 (8.4)                              | 0.317   |
| IgM, n (%)               | 0 (0.0)                            | 7 (2.7)                               | 0.582   |
| Isolated C3, n (%)       | 4 (36.4)                           | 37 (14.1)                             | 0.043   |
| **Eosinophil count, mean (SD); cells/µL ‡** | 1275 (958.8) | 1234.5 (999.8) | 0.909 |
| **C-reactive protein, mean (SD); mg/L ‡** | 24.3 (19.4) | 30.1 (28.9) | 0.554 |

Anti-BP180 NC16A and anti-BP230 antibody levels were measured via enzyme-linked immunosorbent assay; cut-off: 20.0 U/mL. Bold: significant values.

BP, bullous pemphigoid; CI, confidence interval; ELISA, enzyme-linked immunosorbent assay; n, number; SD, standard deviation.

†Was performed in 11 BP patients with comorbid psoriasis and in 255 BP patients without psoriasis. ‡Was performed in 5 patients with comorbid psoriasis and in 78 patients without psoriasis. ¶Was available for 5 patients with comorbid psoriasis and in 94 patients without psoriasis.

**Figure 2** Serum levels of anti-BP180 NC16A IgG amongst bullous pemphigoid (BP) patients with and without comorbid psoriasis.

P = 0.008. Moreover, 83 (30.4%) patients were tested for circulating anti-BP230 antibodies, without disclosing statistically significant differences between the two subgroups neither in the detection rate nor in levels of circulating autoantibodies (Table 2).

We then characterized the deposition of immunoreactants along the DEJ by direct IF analysis. BP patients with comorbid psoriasis demonstrated less frequent linear deposits of immunoglobulin G (IgG; 36.4% vs. 68.7%, respectively; P = 0.025) but more frequent isolated linear deposits of C3 (36.4% vs. 14.1%, respectively; P = 0.043) along the DEJ. Deposition of the remaining immunoreactants did not vary statistically significant between the subgroups (Table 2). Circulating eosinophil counts and C-reactive protein levels were not significantly different between BP patients with and without psoriasis (Table 2).

**Discussion**

The current retrospective study demonstrated that the prevalence of psoriasis amongst this German cohort of patients with BP was estimated at 4.0%. Psoriasis preceded BP in the vast majority (81.8%) of patients with dual diagnoses, with a median latency of 26.5 years separating the onsets of both conditions. BP patients with comorbid psoriasis were significantly younger at the onset of BP and had a significantly less severe erosive phenotype, lower levels of anti-BP180 NC16A autoantibodies and more frequent isolated linear C3 deposits (without concomitant deposits of immunoglobulins) by direct IF microscopy.

systematically evaluated in 48.4% (n = 132) of the study population. The two compared subgroups were comparable with regard to the prevalence of the main metabolic, cardiovascular and neurological comorbidities as well as in the distribution of different therapeutic interventions (Table 1).

Table 2 demonstrates the immunological and laboratory features of BP patients with comorbid psoriasis as compared to BP patients without psoriasis. Overall, 267 (97.8%) BP patients were tested for the presence of circulating anti-BP180 NC16A antibodies. Whilst the seropositivity rate of this autoantibody was comparable, patients with comorbid psoriasis had lower mean (SD) levels [236.6 (266.3) vs. 556.2 (1323.6) U/mL, respectively;
The association of BP with psoriasis and the features of BP amongst patients with dual diagnosis

Whilst knowledge regarding the coexistence of BP and psoriasis was previously based mainly upon case reports and case series, four case–control and cross-sectional studies stemming from Taiwan, France, UK and Israel confirmed this association. A recent meta-analysis aimed to synthesize data across these four studies and included 4035 patients with BP and 19 215 controls. This quantitative synthesis revealed that the pooled odds ratio of psoriasis in patients with BP was 2.5 (95% CI, 1.4–4.6), signifying that the prevalence of psoriasis is 2.5-fold increased amongst patients with BP relative to their matched control counterparts. The prevalence rate of psoriasis amongst our German patients with BP (4.0%) compare with the rates reported in France (4.5%) and Israel (5.2%), outreaches the rate in Taiwan (2.1%), but is outnumbered by the rate reported in the UK (11.3%).

We have shown that psoriasis preceded BP in the great majority of patients (82.8%), aligning with the findings of Ohata et al. as well as Kridin and Bergman showing a subsequent development of BP in 97.2% (141/145) and 100% (15/15) of their patients with dual diagnoses, respectively. However, the fact that these studies had been held in tertiary referral centres specialized in AIBD renders them susceptible to overlook cases in which psoriasis emerges years after the diagnosis of BP. No evident sex predilection was observed in our patients, unlike the aforementioned meta-analysis demonstrating that the pooled prevalence of psoriasis was 1.8-fold higher amongst male than female patients with BP, and unlike the overwhelming male preponderance (82.1%) reported amongst 145 Japanese patients with coexisting AIBD and psoriasis.

The current study indicated that patients with a dual diagnosis of BP and psoriasis tended to present with a milder classical BP phenotype, i.e. less erosions/blisters, which is well reflected by lower levels of anti-BP180 NC16A autoantibodies. The latter were found to correlate with disease activity. To the best of our knowledge, the clinical and immunological features of BP have not previously been investigated amongst patients with coexistent BP and psoriasis. Additionally, the occurrence of isolated C3 deposits without concomitant deposits of immunoglobulins was more frequently observed amongst patients with comorbid psoriasis. A recent study demonstrated that the presence of C3 by direct IF microscopy was associated with increased serum levels of anti-BP180 NC16A autoantibodies and frank blisters at presentation. However, the latter study did not refer to patients with isolated C3 deposits. Further research is required to outline the mechanism underlying this observation.

Clinical features of psoriasis amongst patients with BP and comorbid psoriasis

In a case series of 145 patients with coexistent AIBD and psoriasis originating from Japan, BP was the leading associated disease (63.4%), followed by anti-p200 pemphigoid (37.2%) and their combination (6.9%). This study revealed that the prevalence of pustular psoriasis amongst patients with AIBD (9.0%) outnumbered its prevalence in the general Japanese psoriatic patients (1.3%) and that the pustular phenotype manifested in a close temporal association with the onset of BP. A systematic review, summarizing the literature between 1960 and 2006, detected 40 cases of BP with comorbid psoriasis. This review did not identify evident differences between classical psoriasis and psoriasis associated with BP. Another retrospective study describing 15 patients with coexisting BP and psoriasis revealed that 93.3% had plaque type psoriasis, whereas a single patient showed guttate psoriasis. Our study suggested that plaque type psoriasis was the leading subtype of psoriasis, with only one patient presenting with pustular psoriasis, 23 years prior to the development of BP. Genetic differences may underlie the different subtype distribution across different study populations. Larger sample sizes are highly warranted to draw firmer conclusions with respect to the features of psoriasis in patients with BP.

Putative pathomechanisms underlying the association of BP with psoriasis

Whilst UV irradiation therapy was administered to a third of our patients with BP and preceding psoriasis, only one patient developed BP in a close chronological association with phototherapy. In the aforementioned landmark Japanese study, Ohata et al. reported that in 12.1% of their patients the AIBD arose whilst receiving phototherapy. Correspondingly, Kridin and Bergman found that 40% of their 15 patients with BP and antecedent psoriasis had been previously exposed to phototherapy or climatotherapy at the Deadsea. The role of UV irradiation in precipitating BP was substantiated by the study of Muramatsu et al. reporting an evident decrease in the expression of BP180 in organ-cultured normal-appearing human skin after UVB irradiation. This observation was attributed to UV irradiation-associated configurational alteration in this autoantigen, which may eventually modify antigenicity of constituents of the DEJ and stimulate the generation of autoantibodies against hemidesmosomal autoantigens. UV irradiation, however, does not seem as the cardinal predisposing factor of BP in our patients since the majority of them presented with BP without this exposure. Of note, 33.3% of patients developing BP following psoriasis were managed by biologic agents at the onset of BP. This finding aligns with growing evidence that biologic agents may associated with the paradoxical induction of autoimmune processes. The latter include organ-specific autoimmune diseases (uveitis, multiple sclerosis, autoimmune hepatitis and peripheral neuropathy) and systemic diseases (systemic lupus erythematosus, sarcoidosis, vasculitis and antiphospholipid syndrome).

The pathomechanism underlying the association of BP with psoriasis is largely unknown. Previous studies have shown that laminin 1 and laminin α1 within the DEJ are disrupted in both
involved and uninvolved psoriatic lesions. Degradation of laminin in psoriasis is mediated by the overexpression of plasminogen activators, fibronectin and αβ1 integrin. These structural alterations may modify the antigenicity of DEJ proteins and lower the threshold for the production of autoantibodies targeting these proteins. Another hypothesis relates to the shared role of neutrophils both in BP and psoriasis, given that keratinocytes in both conditions form neutrophil chemoattractants. It was reported that neutrophils secrete different metalloproteases that may be incriminated in the degradation of matrix proteins, leading to subsequent exposure of antigenic epitopes at the DEJ. Our study denotes that patients with comitant BP and psoriasis were significantly younger at the onset of BP. We may postulate that in patients with psoriasis, the antigenicity of DEJ components may be altered at an earlier age, stimulating anti-DEJ autoantibody synthesis and development of BP earlier in life.

Strengths and limitations

The current study aimed to shed light on a previously unexplored topic utilizing data from a large cohort of patients thoroughly profiled, both clinically and immunologically. However, the retrospective data collection resulted in missing information for some variables. Selection bias cannot be refuted given that the study was held in a tertiary-care referral centre specialized in AIBD. This setting is susceptible to overlook mild cases of BP managed by outpatient dermatologists, as well as to overlook mild cases of psoriasis developing years following the initial diagnosis of BP. Data concerning serum reactivity with BP230 and BPDAI scores were missing in a considerable portion of our patients, potentially rendering the study underpowered to identify significant differences in their values between the two subgroups of interest. Moreover, the small sample size of the subgroup of patients with coexistent BP and psoriasis may have interfered with identifying further distinct features of these patients.

In conclusion, the current study shows that 4.0% of BP patients in Northern Germany have comorbid psoriasis. BP followed psoriasis in the majority of patients. Compared to BP patients without psoriasis, those with comorbid psoriasis were younger at the onset of BP, had a more severe erosive phenotype, higher levels of anti-BP180 NC16A autoantibodies, and more frequently isolated deposits of C3 and less frequently deposits of IgG by direct IF microscopy. Further research is warranted to elucidate the pathomechanisms of this association and to validate the distinct clinical and immunopathological profile of these patients in other study populations.

Acknowledgement

The patients presented in this manuscript have given written informed consent to publication of their case details. Open access funding enabled and organized by Projekt DEAL.

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