Original Article

The Correlation Between Anti-desmoglein Autoantibody Titers, IgG Antibody Levels, and the Pemphigus Disease Area Index by Location in Patients with Pemphigus Vulgaris

Quach Thi Ha Giang[1], Tran Thi Huyen[2], Pham Quynh Hoa[3], Nguyen Thi Phuong Hoa[4], Pham Thi Lan[5]

Abstract

Background: Quantifying anti-desmoglein (anti-Dsg) antibodies and indirect immunofluorescence (IIF) are valuable methods for diagnosing and assessing the severity of pemphigus vulgaris (PV). Location-specific Pemphigus Disease Area Index (PDAI) scoring may better reflect disease activity than total PDAI scores.

Objectives: To explore correlations between anti-Dsg1, anti-Dsg3 antibody levels, IIF titers, and location-specific PDAI scores in Vietnamese patients with PV.

Methods: A cross-sectional study was conducted on 48 PV patients, including newly diagnosed or those off immunosuppressive therapy for at least one month. Anti-Dsg1 and anti-Dsg3 levels were measured by ELISA, and IgG antibodies were assessed by IIF.

Results: Among 48 patients (mean age 56.15 years; 64.58% female), anti-Dsg1 positivity was 91.67%, and anti-Dsg3 positivity was 66.67%. Anti-Dsg1 positivity was significantly higher in patients with cutaneous lesions (97.73% vs. 25.00%; P=0.0009), while anti-Dsg3 positivity was higher in those with mucosal lesions (92.86% vs. 30.00%; P<0.0001). Anti-Dsg1 levels correlated with cutaneous PDAI (r=0.378; P=0.0081), and anti-Dsg3 levels strongly correlated with mucosal PDAI (r=0.795; P<0.0001). IIF titers correlated with total PDAI (r=0.377; P=0.0082) and mucosal PDAI (r=0.389; P=0.0062), and were associated with anti-Dsg3 levels (r=0.444; P=0.0015). Higher anti-Dsg1 levels were observed in IIF-negative patients compared to IIF-positive ones. Serum levels of anti-Dsg3 were higher in the IIF-positive group compared to the IIF-negative group (median 114.63 RU/ml vs. 13.9 RU/ml, P=0.0369).

Conclusion: Severity of PV, assessed by location-specific PDAI scores, correlates significantly with anti-Dsg ELISA levels and IIF titers. Integrating clinical scoring with serological and immunofluorescence assays enhances disease monitoring in PV.

Keywords: Desmoglein 1, desmoglein 3, indirect immunofluorescence, Pemphigus Disease Area Index, pemphigus vulgaris.

Received: 15-06-2024 **Revision:** 25-03-2025 **Accepted:** 17-06-2025

Authors Affiliation:
[1,2,5]National Hospital of Dermatology and Venereology, Hanoi, Vietnam/Department of Dermatology and Venereology, Hanoi Medical University, Hanoi, Vietnam; [3,4]National Hospital of Dermatology and Venereology, Hanoi, Vietnam
Corresponding Author: Tran Thi Huyen, National Hospital of Dermatology and Venereology, Hanoi, Vietnam/ Department of Dermatology and Venereology, Hanoi Medical University, Hanoi, Vietnam. **Email:** drhuyentran@gmail.com

Introduction

Pemphigus vulgaris (PV) is the most prevalent form of pemphigus diseases, which are autoimmune conditions marked by blistering on the skin and mucous membranes.[1,2] In PV, autoantibodies (Ab) play a critical role by targeting desmoglein (Dsg) molecules 1 and 3 (anti-Dsg1 and anti-Dsg3), which are essential for maintaining intercellular adhesion in the epidermis.[2,3] Both indirect immunofluorescence (IIF) and enzyme-linked immunosorbent assay (ELISA) are important diagnostic methods for PV, with ELISA being more sensitive and specific than IIF. ELISA is also beneficial for classifying the disease, evaluating its severity, and monitoring disease activity. The severity of skin lesions is associated

with anti-Dsg1 antibody levels, while that of mucosal lesions is linked to anti-Dsg3 antibody levels. Predicting disease progression solely through clinical evaluation is challenging.[4,5] Therefore, quantifying the concentrations of anti-Dsg1 and anti-Dsg3 antibodies using ELISA offers an objective approach to disease monitoring. However, some studies have shown no correlation in certain patients. Some patients remain anti-Dsg positive during stable disease stages, at lower levels compared to active disease stages.[6] Harman's study demonstrated high ELISA values in patients during the quiescent disease period.[7]

Antibody titers measured by IIF are also related to disease severity but are not sufficient for selecting treatment methods, prognosis, or monitoring disease activity. Meanwhile, anti-Dsg1 and anti-Dsg3 are related to IIF results, disease phenotype, and treatment status.[5] The Pemphigus Disease Area Index (PDAI) is one of the most reliable scoring systems for assessing disease severity. Scoring PDAI according to cutaneous and mucosal lesions separately provides better representativeness than the total PDAI score.[8,9] Previous studies have mainly assessed disease severity based on the total PDAI, and the severity of the disease by location has not been thoroughly demonstrated.

In Vietnam, the IIF technique has been a standard procedure for some time, whereas the use of anti-Dsg ELISA is a more recent development. Consequently, this research seeks to assess the relationship between anti-Dsg1 and anti-Dsg3 ELISA results with IIF and region-specific PDAI scores.

Methods

This is a cross-sectional descriptive study. A total of 48 patients with PV were selected for the study. Of these, 23 had previously been treated with one or more immunosuppressive medications, which had been stopped taking at least a month before the study began. The remaining 25 patients were newly diagnosed and had not yet received any treatment. Diagnosis was confirmed through typical clinical signs, histopathological analysis, and immunofluorescence (indirect and direct).[10] Patients with malignancies, infections, autoimmune diseases, or immune deficiencies were excluded from the study. All participants were provided informed consent. Recruitment took place between April 2023 and December 2023.

Patients with pemphigus were categorized according to the location of their lesions-whether they had cutaneous, mucosal, or both cutaneous and mucosal lesions-as well as based on the presence of either cutaneous or mucosal lesions. Disease severity was evaluated using the PDAI score, categorized as mild (0 to 8 points), moderate (9 to 24 points), and severe (25 points or higher).[11] The scoring included both cutaneous PDAI and mucosal PDAI, where total PDAI = cutaneous PDAI + mucosal PDAI.[9]

ELISA kits (IgG) EUROIMMUN anti-Dsg1 and anti-Dsg3 (EUROIMMUN Medizinische Labordiagnostika, Lübeck, Germany) were used. Serum from PV patients was collected and stored at -80°C until analysis, diluted at a ratio of 1:100 in sample buffer. Results were measured in relative units (RU): < 20 RU/ml-negative; > 20 RU/ml-positive. If the serum sample measurement surpassed the standard substance 1 value (200 RU/ml), samples were retested at dilutions of 1:400, 1:800, and 1:1600, with the resulting RU/ml values multiplied by factors of 4, 8, and 16, respectively.

The serum was sequentially diluted with PBS (phosphate-buffered saline) buffer to quantify IgG antibodies against keratinocyte cells by IIF technique. The human dermal substrate was incubated with serum plus a secondary antibody, Polyclonal Rabbit Anti-Human IgG/FITC (Nr. F 100202 Edition/Ausgabe 01.01.032.3, Denmark). Each serum sample was examined under fluorescence microscopy. Positive anti-intercellular antibodies were indicated by fluorescent staining patterns on desmosomes of the epidermal intercellular substance. Each serum sample was diluted starting from 1/20, and if fluorescence was observed, dilution levels were increased gradually to 1/40, 1/80, 1/160, 1/320, etc. Serum samples were considered positive if immunofluores-

cence was detected at a dilution of 1/20 or higher.

Descriptive statistics were reported as mean (standard deviation, SD) for continuous variables with normal distribution, or median (interquartile ranges, IQR) for non-normal continuous variables, and frequency (percentages) for categorical variables. Normality of continuous variables was checked using graphical methods and the Shapiro-Wilk test. Absolute and relative frequencies (%) of IIF, anti-Dsg1 Ab, and anti-Dsg3 Ab were presented. Differences in positive rates between subgroups based on lesion location were analyzed using the Chi-square test or Fisher's exact test if over 20% of the contingency table cells had expected frequencies less than 5. The Mann-Whitney U test compared absolute values of anti-Dsg1 Ab and anti-Dsg3 Ab between subgroups by relative IIF, while the Kruskal-Wallis test compared lesion locations. Spearman's correlation assessed relationships between IIF, anti-Dsg1 Ab, anti-Dsg3 Ab, and PDAI (total, cutaneous, and mucous) in absolute values.

The study received approval from the Research Ethics Committee of Hanoi Medical University under decision No. 838/GCN-HDDDNCYSH-DHYHN, dated March 27, 2023.

Results

The study involved 48 individuals diagnosed with PV, their characteristics were shown in Table 1. The median total PDAI score was 22.50 (IQR: 21.50), with cutaneous PDAI at 18.00 (IQR: 21.50) and mucosal PDAI at 2.00 (IQR: 7.50). The median anti-Dsg1 Ab level was 232.00 RU/ml (IQR: 410.14) with 91.67% positivity, and the median anti-Dsg3 Ab level was 65.75 RU/ml (IQR: 218.22) with 66.67% positivity. The IIF positivity rate was 85.42%, with a median antibody titer of 1/160 (IQR: 1/600).

Table 1: *General characteristics of pemphigus vulgaris patients (n=48).*

Characteristics		
Age	**mean (SD)**	**56.15 (15.15)**
Age groups, n (%)	**<30 years**	**1 (2.08)**
	30-39 years	6 (12.5)
	40-49 years	14 (29.17)
	50-59 years	9 (18.75)
	60-69 years	7 (14.58)
	≥70 years	11 (22.92)
Gender, n (%)	Female	31 (64.58)
	Male	17 (35.42)
Diseases duration (months)	median (IQR)	5.5 (34)
Age at onset (years)	mean (SD)	53.90 (15.24)
Location, n (%)	Cutaneous	20 (41.67)
	Mucosal	4 (8.33)
	Both cutaneous and mucosal	24 (50)
Disease severity, n (%)	Mild	14 (29.17)
	Moderate	18 (37.5)
	Severe	16 (33.33)
Treatment, n (%)	No treatment	25 (52.08)
	Corticosteroids	23 (47.92)
	Azathioprine	4 (8.33)
	Methotrexate	5 (10.42)
	Mycophenolat mofetil	0 (0)
	Rituximab	1 (2.08)
	Traditional medicines	14 (29.17)
Comorbidities, n (%)	Hypertension	7 (14.58)
	Diabiates	3 (6.25)
PDAI (pemphigus disease area index)		
Total PDAI	median (IQR)	22.50 (21.50)
Cutaneous PDAI	median (IQR)	18.00 (21.50)

Mucosal PDAI	median (IQR)	2.00 (7.50)
Serum levels of anti-Dsg1 Ab		
Quantification (RU/ml)	median (IQR)	232.00 (410.14)
Qualitative (positive)	n (%)	44 (91.67)
Serum levels of anti-Dsg3 Ab		
Quantification (RU/ml)	median (IQR)	65.75 (218.22)
Qualitative (positive)	n (%)	32 (66.67)
IIF (Indirect immunofluorescence)		
Antibody titer	median (IQR)	1/160 (1/600)
Qualitative (positive)	n (%)	41(85.42)

IQR: interquartile range; SD: standard deviation

Table 2: *Positive ELISA rates with anti-Dsg Ab according to location with or without lesions.*

Locations	Anti-Dsg1 Ab positive n (%)	Anti-Dsg3 Ab positive n (%)
Cutaneous lesion		
Yes (n=44)	43 (97.73)	28 (63.64)
No (n=4)	1 (25.00)	4 (100.00)
P-value	0.0009*	0.2863*
Mucous lesion		
Yes (n=28)	25 (89.29)	26 (92.86)
No (n=20)	19 (95)	6 (30)
P-value	0.6309*	<.0001†
Cutaneous lesion (n=20)	19 (95)	6 (30)
Both cutaneous and mucous lesion (n=24)	24 (100)	22 (91.67)
P-value	0.0004*	<.0001*

*Fisher's exact test; †Chi-square test; Dsg: Desmoglein

Among PV patients, 91.67% tested positive for anti-Dsg1 Ab, while 66.7% tested positive for anti-Dsg3 Ab. The overall positive rate for indirect immunofluorescence (IIF) was 85.42%. Analysis by lesion location showed a higher anti-Dsg1 Ab positivity in patients with cutaneous lesions compared to those without (97.73% vs. 25.0%; P=0.009). Similarly, patients with mucosal lesions had a higher rate of anti-Dsg3 Ab positivity compared to those without (92.86% vs. 30%; P<0.0001). Significant differences in anti-Dsg1 Ab and anti-Dsg3 Ab positivity were observed across cutaneous, mucosal, and combined lesion groups (P=0.0004 and P=0.0001, respectively) (Table 2).

Serum levels of anti-Dsg3 Ab were higher in the IIF-positive group compared to the IIF-negative group (median 114.63 RU/ml vs. 13.9 RU/ml, P=0.0369). Conversely, anti-Dsg1 Ab levels were higher in the IIF-negative group compared to the IIF-positive group (median 726.08 RU/ml vs. 194.84 RU/ml, P=0.0029). Anti-Dsg1 and anti-Dsg3 antibody levels differed between at least two of the three subgroups according to lesion location (P < 0.05). Anti-Dsg1 Ab level was highest in patients with cutaneous lesions, anti-Dsg3 Ab level was highest in patients with only mucosal lesions (as shown in Figure 1).

There were correlations between anti-Dsg1 Ab and cutaneous PDAI (r=0.378, P=0.0081); anti-Dsg3 Ab and mucosal PDAI (r=0.7948, P<0.001); anti-Dsg3 Ab and IIF (r=0.4447, P=0.0015). Anti-Dsg3 Ab ELISA correlated with IIF (r=0.444, P=0.0015). IIF showed correlation with total PDAI (r=0.377, P=0.0082) and mucosal PDAI (r=0.389, P=0.002), illustrated in Figure 2.

Discussion

In our study, all 48 patients diagnosed with PV displayed either skin or mucosal lesions. The notably high prevalence of positive anti-Dsg1 (91.67%) likely stems from the majority of patients presenting both types of lesions. The correlation between autoantibody findings and clinical manifestations can be elucidated by the desmo-

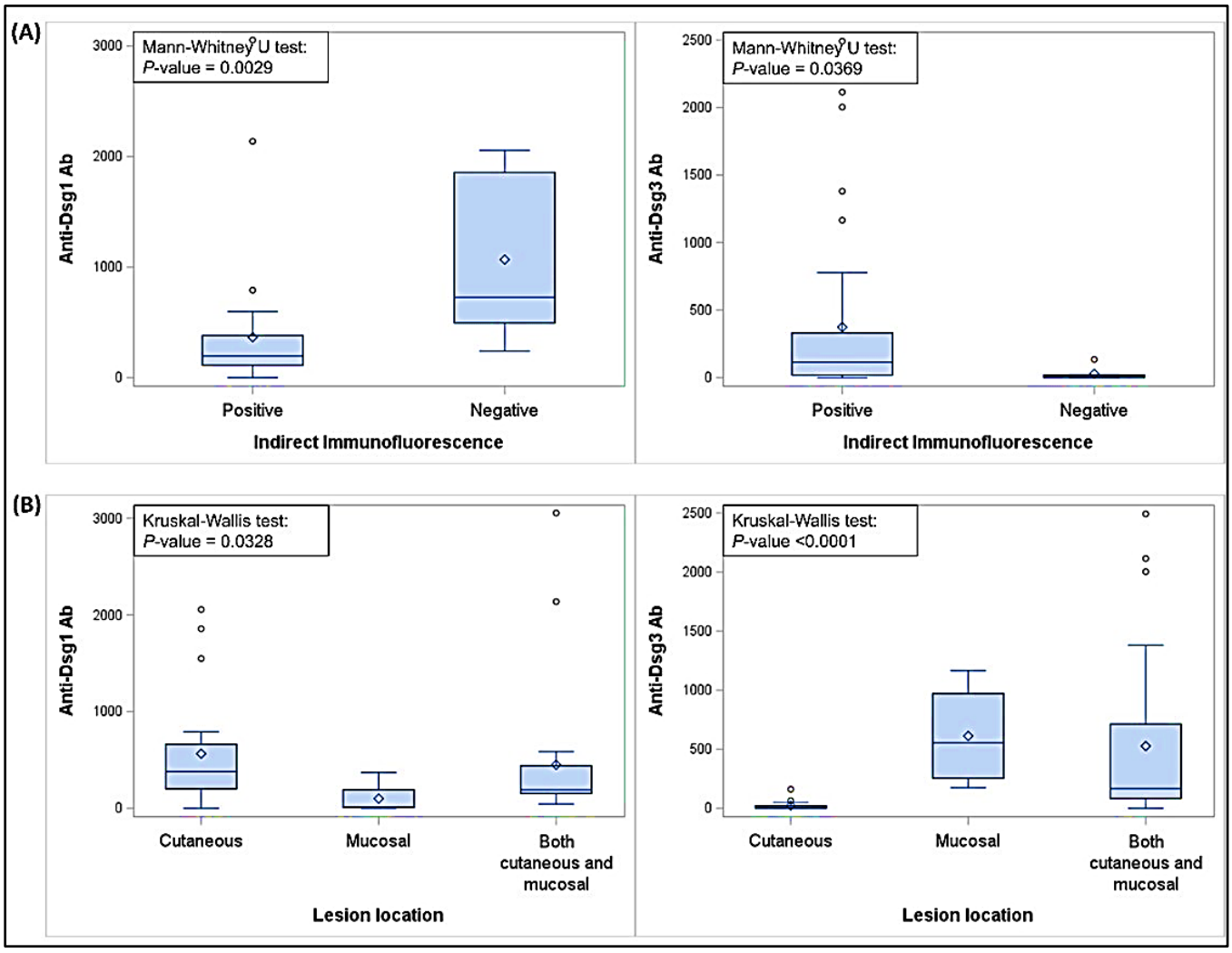

Figure 1: Desmoglein 1 and 3 liters according to (A) Indirect Immunofluorescence and (B) Lesion location.

glein compensation theory, which suggests that desmoglein 1 and desmoglein 3 can compensate for each other's function in cell adhesion when co-expressed, thereby impacting adhesive capabilities when one is compromised.[3,12,13]

There exists a direct relationship between the positivity rates of IIF and the presence of anti-Dsg3 Ab, but not with anti-Dsg1 Ab. This observation aligns with Avgerinos's findings.[14] However, some participants exhibited divergent outcomes in this investigation. For instance, one individual presented with skin lesions despite testing negative for both anti-Dsg1 and anti-Dsg3 Ab. Conversely, certain patients with skin lesions lacked anti-Dsg1 Ab, and others with mucosal lesions were devoid of anti-Dsg3 Ab. Notably, a patient with skin lesions (cutaneous PDAI=5) tested negative for both anti-Dsg1 and anti-Dsg3 Ab, despite showing a positive IIF result. Hence, a comprehensive approach integrating clinical criteria, IIF, and ELISA outcomes proves essential for precise diagnosis in these instances.[10]

In PV cases, IIF sensitivity ranges between 70% and 90%, contingent upon substrate type.[15] Research underscores ELISA's utility as a diagnostic adjunct in PV cases where IIF yields negative results.[16] ELISA demonstrates heightened sensitivity and specificity for detecting serum IgG against anti-Dsg1 and anti-Dsg3 compared to IIF.[15,17] It furnishes an objective means to quantify

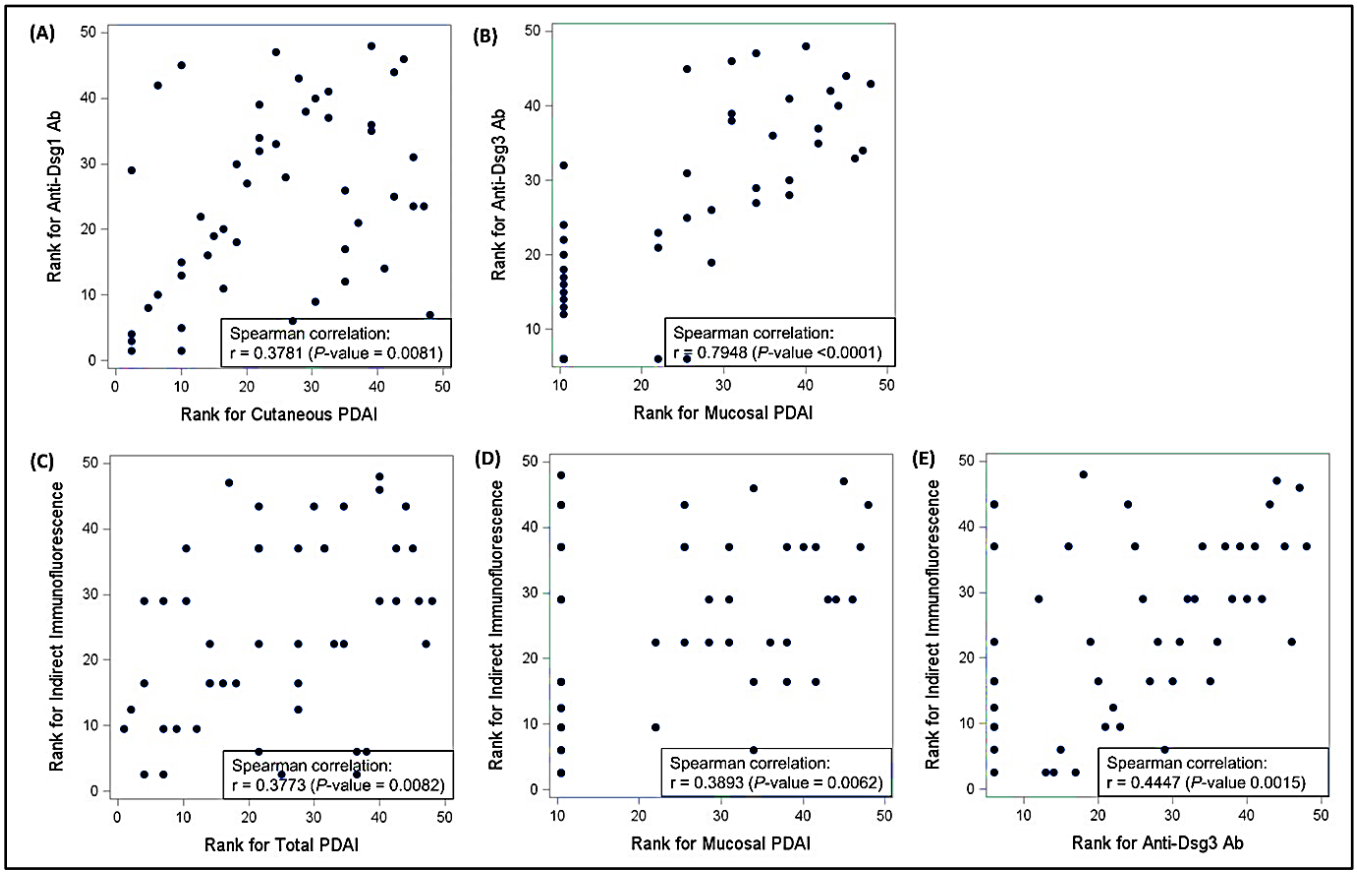

Figure 2: Correlations between (A) Anti-Dsg1 Ab and cutaneous PDAI, (B) Anti-Dsg3 Ab and mucosal PDAI, (C) Indirect immunofluorescence and total PDAI, (D) Indirect immunofluorescence and mucosal PDAI, and € Indirect immunofluorescence and Anti-Dsg3 Ab.

autoantibodies, discerning between pemphigus phenotypes. Conversely, IIF, a semi-quantitative technique for serum IgG autoantibody assessment, involves subjective interpretation based on fluorescence intensity. Studies on the correlation between IIF results and PV severity have yielded mixed outcomes. Some indicated that PV antibody titers measured via IIF effectively gauge disease activity, while others argue IIF lacks consistent correlation with disease severity, thus proving inadequate for guiding treatment selection or disease progression monitoring.[15]

In this study, we identified a correlation between anti-Dsg3 levels and IIF, whereas no such correlation was observed between anti-Dsg1 levels and IIF. Živanović's research demonstrated strong correlations between IIF values and anti-Dsg3 Ab (r=0.742, P<0,01); and anti-Dsg1 Ab (r=0.372, P=0.02).[16] However, we noted higher levels of anti-Dsg1 in the IIF-negative group compared to the IIF-positive group. This discrepancy may be attributed to the small sample size of the IIF-negative group (only seven patients), which limited our ability to draw definitive conclusions. Notably, approximately half of our study participants had received prior immunosuppressive therapy, predominantly corticosteroids, with a third using traditional remedies, potentially influencing our findings.

To comprehensively evaluate disease activity, it is crucial to consider both anti-Dsg Ab and IIF. Our findings on the relationship between Dsg ELISA outcomes and clinical phenotypes have had a similar patterns which were observed in Avgerinou's research, highlighting marked differences in serum anti-Dsg3 levels between groups exhibiting only mucosal lesions and those with both cutaneous and mucosal lesions (P=0.004).[14]

The highest anti-Dsg1 levels were observed in the cutaneous phenotype and the highest anti-Dsg3 levels were observed in the mucosal phenotype. These observations align with global studies that link serum anti-Dsg1 levels to the severity of cutaneous lesions and anti-Dsg3 levels to the severity of mucosal lesions.[5,18] Patsatsi A's study in Greece emphasized a strong correlation between total PDAI scores and anti-Dsg1 levels in patients with cutaneous-mucosal lesions, albeit with a weaker correlation noted with anti-Dsg.[3,19]

Nevertheless, the correlation between anti-Dsg Ab and disease severity remains contentious.[5,17,18] Li reported no significant correlation between conventional anti-Dsg3 Ab and PDAI scores.[20] Although the overall study cohort showed a significant association between anti-Dsg and disease severity, specific cases exhibited discrepancies. For instance, Zhelyazkova documented a case of newly diagnosed mucosal PV with initially negative levels of anti-Dsg3 (10.911 RU/ml) and anti-Dsg1 (16.776 RU/ml).[21] Similarly, Balighi identified eight female pemphigus patients with initially negative anti-Dsg1/anti-Dsg3 Ab levels; one patient with both cutaneous and mucosal lesions initially tested negative for both types of anti-Dsg Ab ELISA.[22] In this study, one patient presented with either cutaneous or mucosal lesions; however, ELISA results were negative for both types of anti-Dsg. This phenomenon may be elucidated by the presence of non-Dsg IgG autoantibodies implicated in the disease pathogenesis.[2,3,23] Furthermore, the disease pathogenesis involves interactions beyond antibodies, including T cells, myeloid cells, and various cytokines.[3,24]

The present study reiterated earlier discoveries on the link between anti-Dsg1 Ab and the extent of skin lesions, between anti-Dsg3 Ab and the severity of mucosal lesions. Nevertheless, akin to prior research, the correlation between clinical presentations and antibody levels was not uniform. What sets this study apart from its predecessors is its distinct examination of cutaneous and mucosal PDAI in relation to both IIF and anti-Dsg Ab.

Nevertheless, this study faces several limitations. It includes data from both newly diagnosed PV patients and those previously treated with immunosuppressive medications, which may influence antibody levels. Additionally, our cross-sectional study design was constrained by a relatively small sample size. Consequently, correlations with $P<0.05$ should be interpreted cautiously and require validation in larger, prospective studies.

Conclusion

There was a correlation between disease severity as assessed by PDAI location scores, IIF, and anti-Dsg Ab. The combination of these two tests provided more accurate information for diagnosing the disease and assessing overall severity across affected areas (skin and mucosa).

Ethical Approval: The Institutional Ethical Review Board of Hanoi Medical University under decision No. 838/GCN-HDDDNCYSH-DHYHN.

Conflict of Interest: There was no conflict of interest to be declared by any author.

Funding Source: None.

Author's Contribution

All authors (QTHG, TTH, PQH, NTPH, PTL) contributed equally as follow:

1. Substantial contributions to study design, data collection, or data analysis and interpretation.
2. Draft or critically revise the manuscript for important intellectual content.
3. Approve the final version to be published.
4. Accept responsibility for the work's accuracy, integrity, and resolution of any related issues.

References

1. Ellebrecht CT, Maseda D, Payne AS. Pemphigus and Pemphigoid: From Disease Mechanisms to Druggable Pathways. Journal of Investigative Dermatology. 2022;142(3):907–14.
2. Strandmoe AL, Bremer J, Diercks GFH, Gostyński A, Ammatuna E, Pas HH, et al. Beyond the skin: B cells in pemphigus vulgaris, tolerance and treat-

ment. British Journal of Dermatology. 2024 ;191(2):164–76.
3. Lim YL, Bohelay G, Hanakawa S, Musette P, Janela B, Yen Loo Lim, et al. Autoimmune Pemphigus: Latest Advances and Emerging Therapies, Front. Mol. Biosci. 8:808536. Frontiers in Molecular Biosciences. 2022;8:26.
4. Pradeep A, Eapen M, Jagadeeshan S, Kani K. Correlation of desmoglein 1 and 3 immunohistochemistry with autoantibody levels and clinical severity in pemphigus. J Cutan Pathol. 2023; 50(12):1104–9.
5. Delavarian Z, Layegh P, Pakfetrat A, Zarghi N, Khorashadizadeh M, Ghazi A. Evaluation of desmoglein 1 and 3 autoantibodies in pemphigus vulgaris: correlation with disease severity. J Clin Exp Dent. 2020;e440–5.
6. Kwon EJ, Yamagami J, Nishikawa T, Amagai M. Anti-desmoglein IgG autoantibodies in patients with pemphigus in remission. J Eur Acad Dermatol Venereol. 2008;22(9):1070–5.
7. Harman KE, Seed PT, Gratian MJ, Bhogal BS, Challacombe SJ, Black MM. The severity of cutaneous and oral pemphigus is related to desmoglein 1 and 3 antibody levels. Br J Dermatol. 2001;144(4): 775–80.
8. Boucher* D, Wilson A, Murrell* DF. Pemphigus scoring systems and their validation studies – A review of the literature. Dermatologica Sinica. 2023;41(2):67–77.
9. Mohebi F, Tavakolpour S, Teimourpour A, Toosi R, Mahmoudi H, Balighi K, et al. Estimated cut-off values for pemphigus severity classification according to pemphigus disease area index (PDAI), autoimmune bullous skin disorder intensity score (ABSIS), and anti-desmoglein 1 autoantibodies. BMC Dermatol. 2020;20(1):13.
10. Joly P, Horvath B, Patsatsi A, Uzun S, Bech R, Beissert S, et al. Updated S2K guidelines on the management of pemphigus vulgaris and foliaceus initiated by the European academy of dermatology and venereology (EADV). Acad Dermatol Venereol. 2020;34(9):1900–13.
11. Shimizu T, Takebayashi T, Sato Y, Niizeki H, Aoyama Y, Kitajima Y, et al. Grading criteria for disease severity by pemphigus disease area index. The Journal of Dermatology. 2014;41(11):969–73.
12. Yamagami J. B-cell targeted therapy of pemphigus. The Journal of Dermatology. 2023;50(2): 124–31.
13. Lin X, Li X. Assessment of anti-desmoglein antibodies levels and other laboratory indexes as objective comprehensive indicators of patients with pemphigus vulgaris of different severity: a single-center retrospective study. Clin Exp Med. 2023;23(2):511–8.
14. Avgerinou, G. Correlation of antibodies against desmogleins 1 and 3 with indirect immunofluorescence and disease status in a Greek population with pemphigus vulgaris. Journal of the European Academy of Dermatology and Venereology. 2013; 27(4): p. 430-435.
15. Ng PP, Thng ST, Mohamed K, Tan SH. Comparison of desmoglein ELISA and indirect immunofluorescence using two substrates (monkey oesophagus and normal human skin) in the diagnosis of pemphigus. Aust J Dermatology. 2005; 46(4):239–41.
16. Zivanovic D, Medenica L, Soldatovic I. Correlation of antibodies against desmogleins 1 and 3 with indirect immunofluorescence and disease activity in 72 patients with pemphigus vulgaris. Acta Dermatovenerol Croat. 2017; 25(1):8–14.
17. Marinovic, B. Comparison of diagnostic value of indirect immunofluorescence assay and desmoglein ELISA in the diagnosis of pemphigus. Acta Dermatovenerol Croat. 2010; 18(2), 79-83.
18. Ravi D, Prabhu SS, Rao R, Balachandran C. Comparison of Immunofluorescence and Desmoglein Enzyme-linked Immunosorbent Assay in the Diagnosis of Pemphigus: A Prospective, Cross-sectional Study in a Tertiary Care Hospital. Indian J Dermatol. 2017;62(2):171–7.
19. Patsatsi A, Kyriakou A, Giannakou A, Pavlitou-Tsiontsi A, Lambropoulos A, Sotiriadis D. Clinical Significance of Anti-desmoglein-1 and -3 Circulating Autoantibodies in Pemphigus Patients Measured by Area Index and Intensity Score. Acta Derm Venerol. 2014;94(2):203–6.
20. Li Z, Zhang J, Xu H. Correlation of conventional and conformational anti-desmoglein antibodies with phenotypes and disease activities in patients with pemphigus vulgaris. Acta Derm Venereal. 2015; 95(4):462–5.
21. Zhelyazkova ZH, Abadjieva TI, Gardjeva PA, Murdjeva MA, Miteva-Katrandzhieva TM. Desmoglein autoantibodies and disease severity in pemphigus patients – correlations and discrepancies. FM. 2023;65(6):969–74.
22. Balighi K, Ashtar Nakhaei N, Daneshpazhooh M. Pemphigus patients with initial negative levels of anti-desmoglein: A subtype with different profile? Dermatol Ther. 2022; 35(4):e15299. Available from: https://doi.org/10.1111/dth.15299.

23. Maho-Vaillant M, Lemieux A, Arnoult C, Lebourgeois L, Hébert V, Jaworski T, et al. Prevalence and pathogenic activity of anti-desmocollin-3 antibodies in patients with pemphigus vulgaris and pemphigus foliaceus. Br J Dermatol. 2025 ;ljaf021.

24. Takahashi H, Iriki H, Asahina Y. T cell autoimmunity and immune regulation to desmoglein 3, a pemphigus autoantigen. J Dermatol. 2023 Feb; 50(2):112–23.