

Microbial Profiles, Antibiotic Resistance and Mortality in Autoimmune Blistering Disorders: A Cross-sectional Observational Study

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ABSTRACT

Introduction: Autoimmune Blistering Disorder (AIBD) is characterised by autoantibodies targeted against structural proteins of the skin and mucosa. The prognosis of this disorder has significantly improved with the introduction of steroids, immunosuppressives, biologics and antibiotics. Despite these advances, septicaemia remains a leading cause of mortality in these patients.

Aim: To evaluate microbial profiles, antibiotic resistance patterns, and mortality in hospitalised patients with AIBDs.

Materials and Methods: The present cross-sectional observational study was conducted at the Department of Dermatology in Saveetha Medical College and Hospital in Chennai, Tamil Nadu, India, from March 2021 to May 2025. The study included 43 inpatients above 18 years with an established AIBD diagnosis based on clinical, histopathological and Direct Immunofluorescence (DIF) findings. All patients were subjected to cutaneous and blood cultures at admission prior to initiating antibiotics, and antibiotic susceptibility testing was performed when bacterial growth was detected. Disease severity was assessed using the Autoimmune Bullous Skin Disorder Intensity score (ABSIS) and demographic parameters, including age,

sex, AIBD subtype, Body Surface Area (BSA) involvement and co-morbidities were recorded. Data analysis utilised Chi-square test and Analysis of Variance (ANOVA), with $p < 0.05$ considered statistically significant.

Results: Among 43 patients, 31 (72.1%) had growth in cutaneous culture, with *Staphylococcus aureus* (11/31, 35.5%) most common; blood culture growth occurred in three patients (7.0%), yielding four organisms. *Staphylococcus aureus* demonstrated high sensitivity to cefazolin, cefuroxime, and cefotaxime in 11 isolates (100%), followed by cotrimoxazole and gentamicin in 10 isolates (90.9%). The overall mortality rate observed in this study was 1 (2.33%). The mortality rate among patients with positive blood cultures was 1 (33.33%).

Conclusion: All patients with growth in blood culture had growth in cutaneous culture, but the vice-versa was not true. Three patients had growth in both cultures, of which one patient died. Secondary bacterial infection/bacteraemia in AIBD remains the leading cause of mortality, especially in patients with co-morbidities and greater BSA involvement. This necessitates the judicious and prompt use of antibiotics and periodic updates on changing bacteriological profiles in AIBD to reduce mortality.

Keywords: Autoimmune bullous disorders, Blood culture, Pemphigoid, Pemphigus, Pus culture, Septicaemia

INTRODUCTION

The AIBD encompass diverse group of potentially life-threatening mucocutaneous diseases characterised by autoantibodies directed against the skin's structural proteins [1]. Among these, the most common are the pemphigus and pemphigoid groups of disorders [2]. The pemphigus group of disorders shows intraepidermal acantholysis, whereas the pemphigoid group is characterised by subepidermal blister formation.

Over the years, there has been a significant improvement in the prognosis of these diseases with the introduction of steroids, immunosuppressants, and biologics [3]. Also, the recent understanding of the alteration in skin microbiome caused by blistering disease could help us choose appropriate antibiotics to manage secondary bacterial infection [4]. Despite these advances, the mortality rate persists at 5-15% [5]. Currently, the average static mortality rate is 6.2% [6], with septicaemia stemming from cutaneous *Staphylococcus aureus* infection being the primary contributory factor.

Several studies have identified *Staphylococcus aureus* as the predominant pathogen in cutaneous bacteriological profiles [3,7]. However, the majority of the research that is currently accessible concentrates on wound infection or surface colonisation, with little information assessing concurrent bacteraemia verified by blood culture. Although septicaemia is frequently reported as a leading cause of mortality in AIBDs, there is limited literature evaluating

culture-proven bloodstream infections and their correlation with clinical outcomes and antibiotic resistance patterns [8-10].

Moreover, existing studies have evaluated antimicrobial resistance patterns in either cutaneous or bloodstream isolates separately, with a paucity of studies integrating both within a single framework. Available literature primarily consists of cutaneous microbiological studies or infection-related outcome analyses, highlighting the lack of comprehensive studies assessing both skin and bloodstream isolates together [3,10,11]. This gap is particularly relevant in the Indian context, where regional variations in antimicrobial resistance patterns may significantly influence empirical antibiotic selection [12,13]. The association between the risk of bacteraemia and disease severity indices, such as BSA involvement and ABSIS, remains inadequately explored in the existing literature [10]. The extent to which patients are at risk for systemic infection due to severe skin involvement has not been determined.

Given these gaps, it is crucial to assess microbial profiles, trends in antimicrobial resistance, and their association with mortality in AIBD. It may be possible to optimise antibiotic stewardship and enhance patient outcomes by comprehending the relationship between cutaneous colonisation, bloodstream infection and clinical severity.

The study aimed to evaluate the microbial profile and antibiotic resistance patterns of cutaneous and blood culture isolates in patients with AIBDs. The primary objective of the study was to determine the spectrum of bacterial isolates obtained from

cutaneous and blood cultures in patients with AIBDs and the secondary objectives were to analyse the antibiotic susceptibility patterns of the isolated microorganisms and to describe mortality among patients with AIBDs.

MATERIALS AND METHODS

The present cross-sectional observational study was conducted at the Department of Dermatology, Saveetha Medical College and Hospital in Chennai, Tamil Nadu, India, from March 2021 to May 2025. Institutional Ethical Committee clearance (protocol no. 473/21) was obtained, and written informed consent was obtained from all patients.

Sample size calculation: Sample size was calculated using the single proportion formula:

$$n = Z^2 \times p \times (1-p) / d^2$$

where, n =sample size, Z =standard normal deviate corresponding to 95% confidence interval (1.96), p =anticipated proportion and d =absolute precision.

The sample size was calculated using an anticipated proportion of 10%, based on previously published literature reporting bloodstream infection rates and mortality associated with autoimmune bullous disorders [10]. An absolute precision of 10% and a 95% confidence level were considered. Given the rarity of the disease, limited inpatient recruitment pool, and feasibility constraints during the study period, a minimum sample size of 44 patients was calculated.

Inclusion criteria: Inpatients aged ≥ 18 years with confirmed AIBD based on clinical, histopathological, and DIF findings.

Exclusion criteria: Patients who had received immunosuppressants or systemic/topical antibiotics within two weeks prior to admission were excluded to avoid false-negative culture results and altered antimicrobial susceptibility patterns. Pregnant or lactating women and patients seropositive for Human Immunodeficiency Virus (HIV), Hepatitis B Virus (HBV) or Hepatitis C Virus (HCV) were also excluded from the study.

Study Procedure

Demographic data and clinical characteristics, including age, sex, AIBD subtype, co-morbidities, disease duration, and disease severity, were assessed using percentage BSA involvement estimated clinically using the rule of nine [14] and ABSIS (total score ranges from 0-206: skin 0-150, oral extent 0-11, oral discomfort 0-45 and interpreted as: mild <17 , moderate 17-53, severe >53) [15]. ABSIS scoring was performed by a single trained dermatologist in all cases, and hence inter-rater reliability analysis was not applicable.

On admission, prior to starting antibiotics, cutaneous and blood cultures were collected. Empirical antibiotic therapy was initiated at admission based on institutional protocol and clinical suspicion of infection. The commonly used empirical antibiotics include intravenous cefotaxime (1 g twice daily) or ceftriaxone (1 g twice daily) depending on disease severity and suspected source of infection. Antibiotic therapy was subsequently modified according to culture and sensitivity results, typically within 48-72 hours. The average duration of antibiotic treatment ranged from 7 to 14 days, tailored to clinical response and microbiological clearance. Precise time intervals between culture collection and initiation of antibiotics were not consistently documented and hence could not be reliably analysed. Pus was collected from clinically infected lesions, which were defined as skin lesions demonstrating one or more of the following features: purulent discharge with surrounding erythema, crusting, foul odour or delayed healing suggestive of secondary bacterial infection [16]. Under strict aseptic precautions, blood cultures were collected by obtaining 10-15 mL of venous blood from adults and inoculated into sterile nutrient broth culture bottles.

Samples were processed in the institutional microbiology laboratory using standard aerobic culture systems. Anaerobic cultures were not routinely performed as part of institutional protocol for suspected bloodstream infection in dermatology inpatients.

Pus samples were inoculated onto routine bacteriological media, including blood agar and MacConkey agar, and incubated aerobically at 35-37°C for 24-48 hours. Blood culture bottles were incubated at 35-37°C and monitored for microbial growth for up to 5-7 days. Positive samples were sub-cultured onto appropriate media for organism isolation. Antibiotic susceptibility testing was determined using the Kirby Bauer disk diffusion method, and results were interpreted according to Clinical and Laboratory Standards Institute (CLSI) guidelines, 2022 [17] followed by the laboratory during the study period.

STATISTICAL ANALYSIS

Collected data was entered in a Microsoft Excel spreadsheet and analysed using Statistical Package for Social Sciences (SPSS) Version 20.0 for Windows. Data were analysed for statistical significance by the Chi-square test and ANOVA test. A p -value of <0.05 was considered statistically significant.

RESULTS

A total of 43 patients with Autoimmune Blistering Disorders (AIBD) were included in the study. The mean BSA involvement was $61.88\% \pm 11.14\%$ (range: 43-85%), indicating extensive disease in most patients. Co-morbidities were present in 16 patients (37.2%), while 27 patients (62.8%) had no associated co-morbid conditions. Diabetes mellitus was the most frequently observed co-morbidity, affecting seven patients (16.3%), followed by the coexistence of diabetes mellitus and hypertension in five patients (11.6%). None of the participants reported a positive family history of AIBD.

Most patients belonged to the 41-60 years age group (19/43, 44.2%), with a female predominance observed in 26 patients (60.5%). Pemphigus vulgaris was the most common subtype, seen in 17 patients (39.5%), followed by bullous pemphigoid in 13 patients (30.2%) and pemphigus foliaceus in nine patients (20.9%) [Table/Fig-1].

Characteristics	Range	Value (n)
1. Age (years)	≤ 40	12 (27.9%)
	41-60	19 (44.2%)
	>60	12 (27.9%)
2. Sex	Male	17 (39.5%)
	Female	26 (60.5%)
3. Type of bullous disorder	Pemphigus vulgaris	17 (39.5%)
	Bullous pemphigoid	13 (30.2%)
	Pemphigus foliaceus	9 (20.9%)
	Linear IgA bullous dermatosis	2 (4.7%)
	Dermatitis herpetiformis	1 (2.3%)
	IgA pemphigus	1 (2.3%)
4. BSA involvement (Mean $\pm$ SD)		61.88 $\pm$ 11.14 (43-85)
5. ABSIS score (Mean $\pm$ SD)	Mean cutaneous score	85.87 $\pm$ 21.39 (46-127.5)
	Mean mucosal severity score	31.23 $\pm$ 6.58 (20.5-41.5)
	Average mucosal extent score	3.26 $\pm$ 1.66 (1-7)
6. Co-morbidity profile	Diabetes mellitus	7 (16.3%)
	Diabetes mellitus+hypertension	5 (11.6%)
	Others*	4 (9.3%)
	No co-morbidities	27 (62.8%)

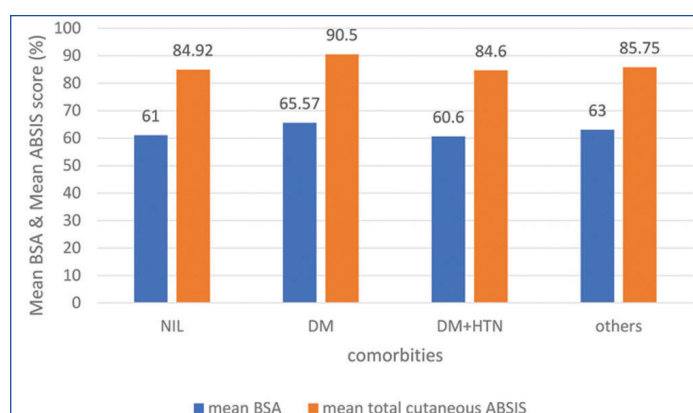
[Table/Fig-1]: Demographic details of the patients.

BSA: Body surface area; ABSIS: Autoimmune bullous skin disorder intensity score; SD: Standard deviation; *Other co-morbidities include hypothyroidism (n=2), coronary artery disease (n=1), chronic kidney disease (n=1)

Clinical images of a patient with pemphigus vulgaris and a patient with bullous pemphigoid are presented in [Table/Fig-2a,b], respectively. Patients with type 2 DM had showed highest disease severity by mean total ABSIS score of 90.50% and mean cutaneous BSA involvement of 65.57% [Table/Fig-3]. The mean duration of hospital stay was 13±4 days. ICU care was required in 5 (11.63%) of patients, primarily due to extensive body surface involvement.

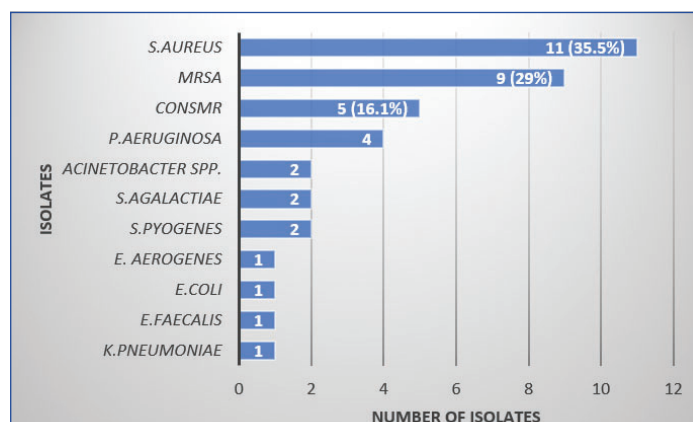


[Table/Fig-2]: Clinical images depicting: a) Pemphigus vulgaris. b) Bullous pemphigoid.



[Table/Fig-3]: Comparison of mean BSA (%) and mean total ABSIS Score (%) across co-morbidity groups.

Among the 43 patients, 12 (27.9%) had no growth in cutaneous culture, 23 (53.5%) showed growth of a single organism, and 8 (18.6%) exhibited growth of two organisms each. A total of 39 bacterial isolates were identified from 31 (72.1%) patients. The most common organism isolated from cutaneous culture was *Staphylococcus aureus*, identified in 11/31 (35.5%) patients with positive cultures. In blood culture, 3 (7%) patients exhibited growth, of which one patient had growth of two organisms [Table/Fig-4].



[Table/Fig-4]: Cutaneous culture analysis.

Concordance analysis between empirical antibiotic therapy and culture sensitivity results showed that empirical antibiotics were appropriate in 18 of 31 culture-positive cases, yielding a concordance rate of 58.1%. In polymicrobial infections, concordance was taken as the sensitivity of the empirical antibiotic to at least one of the isolated organisms.

Among the 11 *Staphylococcus aureus* isolates recovered from cutaneous cultures, all isolates were sensitive to cefazolin, cefuroxime, and cefotaxime (100% each). High susceptibility was also observed to cotrimoxazole and gentamicin (90.9% each) [Table/Fig-5].

Organism	Sensitive
<i>Staphylococcus aureus</i> (n=11)*	Cefazoline 11 (100%), cefuroxime 11 (100%), cefotaxime 11 (100%), cotrimoxazole 10 (90.9%), gentamicin 10 (90.9%), clindamycin 9 (81.81%), teicoplanin 9 (81.81%), linezolid 9 (81.81%)

[Table/Fig-5]: Antibiotic sensitivity profile of *Staphylococcus aureus* isolated from cutaneous cultures.

*As per CLSI, the number of isolates less than 30 was not significant. However, this data has been presented here for epidemiological reasons, so that it can be later on pooled in a different meta-analysis

Antibiotic Sensitivity Profile of Blood Culture

Blood culture positivity was observed in 3 (7%) patients, yielding four isolates: *Burkholderia pseudomallei*, *Klebsiella pneumoniae*, *Proteus mirabilis* and Methicillin Resistant *Staphylococcus aureus* (MRSA).

The gram-negative isolates demonstrated variable sensitivity. *Burkholderia pseudomallei* showed broad susceptibility to multiple antibiotics including ceftazidime, carbapenems, and beta-lactamase inhibitor combinations, while *Klebsiella pneumoniae* exhibited extensive resistance with only intermediate sensitivity to tigecycline. *Proteus mirabilis* retained sensitivity to several beta-lactams and carbapenems but demonstrated resistance to cotrimoxazole and polymyxin B. MRSA isolated from blood culture showed sensitivity to vancomycin, linezolid, clindamycin, and cotrimoxazole, but resistance to cephalosporins and fluoroquinolones.

Comparison of Growth in Blood and Cutaneous Culture

Only 3 (7%) of the 31 (72.1%) patients with positive cutaneous cultures had growth in blood cultures, but no significant association was found [Table/Fig-6].

Cutaneous culture	Blood culture no growth	Blood culture growth	Total
No growth	12 (27.9%)	0	12 (27.9%)
Growth	28 (65.1%)	3 (7%)	31 (72.1%)
Total	40 (93%)	3 (7%)	43

[Table/Fig-6]: Cross-tabulation between cutaneous and blood culture number of isolates.

Fisher's-exact test was applied, p-value=0.548

Among the three patients with concurrent growth in cutaneous and blood cultures, two patients demonstrated polymicrobial growth. One patient had *Streptococcus pyogenes* and MRSA in cutaneous culture with *Proteus mirabilis* and *Klebsiella pneumoniae* in blood culture. Another patient showed MRSA in both cutaneous and blood cultures. The third patient had *Pseudomonas aeruginosa* and MRSA in cutaneous culture, with *Burkholderia pseudomallei* isolated from blood culture. The patient with *Burkholderia pseudomallei* isolated from blood culture succumbed to septicemia within two weeks of admission.

Analysis of the overall antibiotic susceptibility pattern, based on the total number of isolates obtained from both cutaneous and blood cultures (n=43), showed that linezolid exhibited the highest sensitivity 27 (62.8%), followed by vancomycin 24 (55.8%) and teicoplanin 17 (39.5%). These findings indicate that glycopeptides and oxazolidinones demonstrated relatively higher activity against the isolated organisms.

Notably, all patients with positive blood cultures had a BSA involvement greater than 75% and an ABSIS mucosal severity score greater than 35. One of the patients 1/43 (2.33%) succumbed during the study period due to septicemia. The patient demonstrated a BSA involvement (>70%), high ABSIS mucosal severity score (>28), positive blood culture for *Burkholderia pseudomallei*, and clinical evidence of systemic sepsis. Despite initiation of appropriate antimicrobial therapy and supportive management, the patient

developed progressive septicaemia resulting in mortality. The mortality rate among patients with positive blood cultures was 1/3 (33.3%).

DISCUSSION

The destruction of skin and mucosal barrier by AIBD plays a pivotal role in contributing to infectious complications [5]. The alteration in the homeostasis leads to colonisation and biofilm formation, with microorganisms embedded in a self-secreted extracellular polysaccharide matrix that facilitates immune evasion [7]. Systemic spread can occur passively through the dispersal of cell-free microbes in lymph or plasma. It can also occur actively through the infection of mucosal, submucosal, dermal, or subcutaneous macrophages, lymphocytes, and dendritic cells, leading to the dispersal of cell-associated microbes in the lymphatic or circulatory systems [18]. Understanding these changes at the disease site becomes essential, in guiding the selection of appropriate antibiotics to reduce mortality in AIBD [4].

Staphylococcus aureus was consistently the most common cutaneous isolate across different reports from various regions of India (Ahmedabad, Assam, Karnataka, Gujarat, Odisha) and other countries such as Egypt and Iran, indicating that the trend of isolation of microorganism has not changed [3,11-13,19-23].

In the current study, three of the four isolates from blood culture were gram-negative, similar to the findings of Lehman JS et al., [10]. In the present study, all patients with bloodstream infection had extensive disease with significant mucosal involvement, which may increase susceptibility to systemic infection. The patterns of antimicrobial susceptibility found in this study emphasise the importance of regional resistance trends when selecting empirical treatment. Glycopeptides and linezolid retained high activity against MRSA and other Gram-positive pathogens, which is consistent with the finding of Abdallah M et al., [21]. The sensitivity profile of *Staphylococcus aureus* contrasts with Solanki RB et al., Kiran KC et al., and Esmaili N et al., study [3,11,22]. Maximum resistance was observed with fluoroquinolones and certain third generation cephalosporins like ceftriaxone, reflecting evolving trends caused by overuse of these agents in hospital and community settings. *Staphylococcus aureus* resistance pattern aligns with Abdallah M et al., and Kiran KC et al., study [11,21]. This emphasises the need for culture-guided therapy whenever possible.

Gram-negative isolates including *Pseudomonas aeruginosa*, *Acinetobacter spp.*, *Enterobacter aerogenes*, *Escherichia coli* and *Klebsiella pneumoniae*, showed better sensitivity to carbapenems like imipenem, meropenem and beta-lactamase inhibitor combinations like piperacillin-tazobactam, cefoperazone-sulbactam, aligning with the findings of Kiran KC et al., study [11]. However, ampicillin showed high resistance rates which aligns with Abdallah M et al., study [21].

Klebsiella pneumoniae and MRSA were cultured from blood and cutaneous samples in current study. *Klebsiella pneumoniae* isolates from blood culture were resistant to all tested antibiotics except for intermediate sensitivity to tigecycline contrary to the findings of cutaneous culture in this study, while the resistance and sensitivity patterns of MRSA isolated from blood and cutaneous culture were similar. The sensitivity pattern of *Proteus mirabilis* aligned with the cutaneous culture findings in the Kiran KC et al., study [11]. *Burkholderia pseudomallei* showed sensitivity to most tested antibiotics as detailed in the results.

Patients with diabetes mellitus had a longer duration of illness and greater BSA involvement compared to non diabetics, which was comparable to the findings of Esmaili N et al., and Belgnaoui FZ et al., [22,24]. The present study's mortality stands at 2.33%, with a mean BSA involvement of 61.88%. These rates are notably lower than those reported by Solanki RB et al., Nair SP, Kanwar AJ et al., [3,25,26] Interestingly, contrary to Kridin K et al., who found that older age at diagnosis correlated with shorter survival, the present

study featured a middle-aged patient [27]. Septicaemia remained the leading cause of death similar to findings of Kanwar AJ et al., Chowdhury S et al., Kridin K et al., and Nair SP [9,25-27] The low mortality observed may be attributed to several factors: the relatively low BSA involved at diagnosis, early and appropriate use of antibiotics based on culture results, availability of biologics, and favourable patient compliance.

The source of bacteraemia in blood cultures may be due to active mucosal involvement, substantiated by the lack of corresponding isolates in cutaneous cultures, negative urine cultures and the absence of central lines in patients at the time of sample collection. The identification of *Burkholderia pseudomallei* also reflects regional epidemiological patterns, as sporadic cases of melioidosis have been reported in southern India. These findings highlight the importance of routine blood culture in patients with extensive disease, systemic symptoms, or high severity indices. This practice can prevent delays in treatment, reduce morbidity and mortality associated with AIBD, and prevent antibiotic misuse.

Limitation(s)

The study was limited by the small sample size, especially for bloodstream infections, restricting detailed statistical analysis and subgroup comparisons. Its single-centre design may limit generalisability. Prior antibiotic exposure could not be fully verified and may have affected culture yield. Anaerobic cultures and molecular identification methods were not routinely performed, possibly leading to under-detection of certain pathogens. Despite these limitations, the study provides useful insight into the microbial spectrum and antimicrobial susceptibility patterns in AIBD patients from a South Indian tertiary-care setting.

CONCLUSION(S)

The persistent challenge of secondary bacterial infection leading to septicaemia remains a major cause of morbidity and mortality despite the advanced therapeutic options available for AIBD, especially in patients with extensive BSA, mucosal involvement and co-morbidities. In the present study, cutaneous bacterial colonisation was common, whereas bacteraemia occurred infrequently. In skin cultures, Gram-positive organisms like *Staphylococcus aureus* and MRSA predominated, whereas bloodstream isolates showed variable microbial patterns. Glycopeptides and linezolid retained high activity against Gram-positive organisms, while carbapenems and beta-lactamase inhibitor combinations showed comparatively better efficacy against Gram-negative isolates. Resistance to fluoroquinolones and selected cephalosporins was observed across several organisms. Although higher disease severity appeared to be associated with bacteraemia, the limited number of bloodstream infections restricts definitive inference. These findings highlight the importance of culture-guided antibiotic therapy and periodic local antimicrobial surveillance in optimising management of AIBDs.

REFERENCES

- [1] Zhao CY, Murrell DF. Autoimmune blistering diseases in females: A review. Int J Womens Dermatol [Internet]. 2015;1(1):04-12. Available from: <http://dx.doi.org/10.1016/j.ijwd.2015.01.002>
- [2] Griffiths C, Barker J, Bleiker T, Chalmers R, Creamer D, editors. Rook's Textbook of Dermatology. 9th ed. Hoboken, NJ: Wiley-Blackwell; 2016. p.50.1-41.
- [3] Solanki RB, Shah YB, Shah AN, Jain V. Bacterial culture and sensitivity in pemphigus. Indian J Dermatol Venereol Leprol. 1997;63(2):89-90.
- [4] Belheouane M, Hermes BM, Van Beek N, Benoit S, Bernard P, Drenovska K, et al. Characterization of the skin microbiota in bullous pemphigoid patients and controls reveals novel microbial indicators of disease. J Adv Res [Internet]. 2023;44:71-79. Available from: <http://dx.doi.org/10.1016/j.jare.2022.03.019>.
- [5] Herbst A, Bystryn JC. Patterns of remission in pemphigus vulgaris. J Am Acad Dermatol [Internet]. 2000;42(3):422-27. Available from: [http://dx.doi.org/10.1016/s0190-9622\(00\)90213-5](http://dx.doi.org/10.1016/s0190-9622(00)90213-5).
- [6] Sacchidanand S, Oberai C, Inamadar AC, editors. IADVL Textbook of Dermatology. 4th ed. Mumbai: Bhalani Publishing House; 2018. p. 933-91.
- [7] Wilson JW, Schurr MJ, LeBlanc CL, Ramamurthy R, Buchanan KL, Nickerson CA. Mechanisms of bacterial pathogenicity. Postgrad Med J [Internet]. 2002;78(918):216-24. Available from: <http://dx.doi.org/10.1136/pm.j.78.918.216>.

- [8] Kanwar AJ, Dhar S. Factors responsible for death in patients with pemphigus. *J Dermatol* [Internet]. 1994;21(9):655-59. Available from: <http://dx.doi.org/10.1111/j.1346-8138.1994.tb01811.x>.
- [9] Chowdhury S, Podder I, Saha A, Bandyopadhyay D. Inpatient mortality resulting from dermatological disorders at a tertiary care center in eastern India: A record-based observational study. *Indian J Dermatol* [Internet]. 2017;62(6):626-29. Available from: http://dx.doi.org/10.4103/ijid.IJD_284_17.
- [10] Lehman JS, Khunger M, Lohse CM. Infection in autoimmune bullous diseases: A retrospective comparative study. *J Dermatol* [Internet]. 2013;40(8):613-19. Available from: <http://dx.doi.org/10.1111/1346-8138.12175>.
- [11] Kiran KC, Madhukara J, Abraham A, Muralidharan S. Cutaneous bacteriological profile in patients with Pemphigus. *Indian J Dermatol* [Internet]. 2018;63(4):301-04. Available from: http://dx.doi.org/10.4103/ijid.IJD_152_17.
- [12] Motgi MB, Janagond AB, Inamadar AC, Adya KA. A prospective cross-sectional study to determine the bacteriological profile and antibiotic sensitivity patterns of cutaneous lesions in immunobullous disorders. *Clin Derm Rev* [Internet]. 2025;9(4):332-35. Available from: http://dx.doi.org/10.4103/cdr.cdr_29_25.
- [13] Mohanty S, Saha S, Firdaus S, Sirka CS. Profile of cutaneous bacterial flora in Pemphigus patients. *J Lab Physicians* [Internet]. 2023;15(4):616-20. Available from: <http://dx.doi.org/10.1055/s-0043-1768635>.
- [14] Tiling-Grosse S, Rees J. Assessment of area of involvement in skin disease: A study using schematic figure outlines. *Br J Dermatol*. 1993;128(1):69-74.
- [15] Boulard C, Duvert Lehembre S, Picard-Dahan C, Kern JS, Zambruno G, Feliciani C, et al. Calculation of cut-off values based on the Autoimmune Bullous Skin Disorder Intensity Score (ABSIS) and Pemphigus Disease Area Index (PDAI) pemphigus scoring systems for defining moderate, significant and extensive types of pemphigus. *Br J Dermatol* [Internet]. 2016;175(1):142-49. Available from: <http://dx.doi.org/10.1111/bjd.14405>.
- [16] Healy B, Freedman A. Infections. *BMJ* [Internet]. 2006;332(7545):838-41. Available from: <http://dx.doi.org/10.1136/bmj.332.7545.838>.
- [17] Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing. 32nd ed. Wayne (PA): CLSI; 2022. CLSI supplement M100.
- [18] Church D, Elsayed S, Reid O, Winston B. Burn wound infections. *Clin Microbiol Rev*. 2006;19(2):403-34.
- [19] Huda MM, Afsar MI. A clinicopathological study of pemphigus. *Indian J Dermatol*. 2001;46(2):75-79.
- [20] Chauhan K, Mehta K, Shingala H. Cutaneous bacteriological profile and its antibiotics sensitivity pattern in patients of Pemphigus vulgaris attending tertiary care hospital, Jamnagar, India. *Saudi J Pathol Microbiol* [Internet]. 2019;04(10):738-40. Available from: <http://dx.doi.org/10.36348/sjpm.2019.v04i10.007>.
- [21] Abdallah M, Zaki SM, El-Sayed A, Erfan A. Evaluation of secondary bacterial infection of skin diseases in- & outpatient & their sensitivity to antimicrobials. *Egypt Dermatol Online J*. 2007;3(2):01-15.
- [22] Esmaili N, Mortazavi H, Noormohammadpour P, Boreiri M, Soori T, Vasheghani Farahani I, et al. Pemphigus vulgaris and infections: A retrospective study on 155 patients. *Autoimmune Dis* [Internet]. 2013;2013:834295. Available from: <http://dx.doi.org/10.1155/2013/834295>.
- [23] Qadim HH, Hasani A, Zinus BM, Orang NJ, Hasani A. Etiology of pyrexia in pemphigus patients: A dermatologist's enigma. *Indian J Dermatol Venereol Leprol* [Internet]. 2012;78(6):774. Available from: <http://dx.doi.org/10.4103/0378-6323.102392>.
- [24] Belgnaoui FZ, Senouci K, Chraïbi H, Aoussar A. Predisposition to infection in patients with pemphigus. Retrospective study of 141 cases. *Presse Med*. 2007;36:1563-69.
- [25] Nair SP. A retrospective study of mortality of pemphigus patients in a tertiary care hospital. *Indian J Dermatol Venereol Leprol*. 2015;79(5):706-09.
- [26] Kanwar AJ, Ajith AC, Narang T. Pemphigus in North India. *J Cutan Med Surg* [Internet]. 2006;10(1):21-25. Available from: <http://dx.doi.org/10.1007/7140.2006.00010>.
- [27] Kridlin K, Sagi SZ, Bergman R. Mortality and cause of death in patients with Pemphigus. *Acta Derm Venereol* [Internet]. 2017;97(5):607-11. Available from: <http://dx.doi.org/10.2340/00015555-2611>.

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- iThenticate Software: Jun 03, 2026 (4%)

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