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Original Research Article

High Segmental Fraction and Intensive Phototherapy Utilisation in Indonesian Vitiligo: A Five-Year Retrospective Tertiary-Centre Study in Surakarta, Indonesia

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ABSTRACT

Background: Vitiligo is the most common acquired depigmenting disorder, yet Indonesian epidemiological and treatment-pattern data remain scarce.

Objective: To characterise the clinical phenotype, anatomical distribution and treatment utilisation of vitiligo at an Indonesian tertiary centre and to test them against published reference proportions.

Methods: This STROBE-compliant retrospective study included all 175 patients coded ICD-10-CM L80 at the Dermatology and Venereology Outpatient Clinic of Dr. Moewardi Regional General Hospital, Surakarta, between January 2020 and December 2024, identified by consecutive total sampling. Proportions were estimated with Wilson 95% confidence intervals, compared with published international and Indonesian reference proportions using two-proportion tests with Cohen's h, and analysed by multivariable logistic regression, receiver-operating-characteristic and Poisson models.

Results: Patients were predominantly female (57.71%) and young (70.86% aged under 40 years). Nonsegmental disease predominated (62.29%), but the segmental fraction (37.71%; 95% CI 30.87–45.09) exceeded an international tertiary-cohort reference of 5.93% by 31.78 percentage points (h = 0.831; p < 0.001). Phototherapy reached 74.86%, exceeding United States real-world utilisation of 3.9% by 70.96 points (h = 1.694), and only 0.57% remained untreated against 27% internationally. Multiple lesions (adjusted odds ratio 8.39; 95% CI 3.41–20.64) and nonsegmental disease (3.59; 1.46–8.79) independently predicted phototherapy (area under the curve 0.859).

Conclusion: Indonesian tertiary vitiligo care is phototherapy-intensive and markedly enriched for segmental disease, warranting consensus-based classification and phototherapy dosimetry documentation.

All authors have reviewed and approved the final version of the manuscript.

1. Introduction

Vitiligo is the most common acquired depigmenting disorder of the skin, characterised by the progressive destruction of functional epidermal melanocytes and the appearance of well-demarcated, non-scaly, milky-white macules and patches.[1,2] Population-based survey data estimate diagnosed adult prevalence in the United States at about 0.76%, with self-reported prevalence nearly twice as high,[3] while multinational data from Europe, Japan and the United States report 0.6% to 1.4%.[4] Nationwide registry analysis in Korea shows both incidence and prevalence rising between 2003 and 2019, with a bimodal age-specific incidence whose steepest increase occurs below 20 years of age,[5] and nationwide cohort data now quantify all-cause and cause-specific mortality in the disease.[6] Because the disease is visible, chronic and cosmetically disfiguring, its burden is disproportionate to its prevalence: in patients with skin of colour, quality-of-life impairment tracks disease extent and activity directly,[7] and the same relationship has been demonstrated in Indonesian patients undergoing phototherapy.[8] In Indonesia — a Fitzpatrick skin phototype III–V population living at an equatorial ultraviolet index of 10 to 12 throughout the year, where depigmented macules contrast sharply against constitutively pigmented skin — no national prevalence estimate exists, and the published evidence consists of a small number of single-centre hospital series.[9-11]

Vitiligo is not a single disease but a family of phenotypes with divergent biology. International Vitiligo Task Force recommendations retain the binary architecture that remains standard: nonsegmental vitiligo, typically bilateral and symmetrical with acrofacial, mucosal, generalised, universal and mixed subtypes; segmental vitiligo, usually unilateral, block-like or linear and not crossing the midline; and unclassified disease, comprising focal or isolated mucosal lesions that remain unchanged over time.[2] The

distinction is mechanistically real. Direct comparison of 379 patients showed that segmental disease differs immunologically as well as clinically from nonsegmental disease and behaves as a localised process,[12] and segmental lesions stabilise substantially earlier in their natural history.[13] Nonsegmental disease is the systemic autoimmune phenotype: lesional CD8+ cytotoxic T-cell burden predicts treatment response directly in patients,[14] and interferon-γ and granzyme B released by those cells mediate melanocyte destruction.[15] Meta-analysis confirms that vitiligo clusters with autoimmune thyroid disease, type 1 diabetes mellitus, alopecia areata and psoriasis,[16] and patients with associated autoimmune thyroid disease show a distinguishable clinical profile.[17]

Classification therefore drives management. International Vitiligo Task Force recommendations position potency- and site-adjusted topical corticosteroids or calcineurin inhibitors for limited disease, narrowband ultraviolet B phototherapy for extensive or progressive disease, and short systemic corticosteroid courses for rapidly progressive disease.[2] Randomised evidence supports each tier: topical tacrolimus monotherapy is effective in limited nonsegmental disease,[18] topical Janus kinase inhibition performs comparably to calcineurin inhibition in localised disease,[19] and narrowband ultraviolet B stabilises disease even where repigmentation is poor, as in acral sites.[20] Validated instruments — the Vitiligo Area Scoring Index and the Vitiligo Extent Score — are the accepted metrics of extent, and their measurement properties have been formally quantified.[21,22] Stable segmental disease is additionally the phenotype in which surgical repigmentation performs best in meta-analysis, so a centre's segmental fraction has direct consequences for treatment triage.[23]

What actually happens in routine care, however, differs sharply from what guidelines recommend. In a United States claims cohort of 19,335 patients, phototherapy was used by only 3.9%, high-potency topical corticosteroids by 20.2% and oral corticosteroids by 21.0%, while 49.9% received no treatment at all in the year after diagnosis.[24] A United Kingdom national cohort of 16,741 incident patients reported topical corticosteroids in 29.1% and oral corticosteroids in 4.2% in the first year, with 60.8% receiving no vitiligo-specific treatment,[25] and a 24,949-patient United States analysis found that between 27% and 32% of patients received no treatment during follow-up, with the face affected in 32.9%.[26] These real-world figures, rather than guideline text, are the appropriate yardstick against which an individual service should be judged.

In Indonesia, specialist dermatological care is delivered through centres affiliated with *Perhimpunan Dokter Spesialis Kulit dan Kelamin Indonesia* (PERDOSKI). Dr. Moewardi Regional General Hospital, Surakarta, is the tertiary academic referral centre of the Faculty of Medicine, Universitas Sebelas Maret, and serves as the principal dermatology and venereology referral hospital for Surakarta and the surrounding districts of Central Java. Its Dermatology and Venereology Outpatient Clinic operates one of the few narrowband ultraviolet B phototherapy services in the region and manages patients across the full age range within a Fitzpatrick III–V population. Despite this, no Indonesian study has reported a vitiligo case-mix with measures of precision, tested the observed distribution against published reference values, or identified the determinants of treatment allocation; the existing domestic series report raw percentages without confidence intervals, effect sizes or adjustment.[9,10] This study therefore had three aims: to characterise the demographic profile, clinical phenotype, anatomical distribution and treatment utilisation of all patients with vitiligo managed at this centre over five years, with 95% confidence intervals for every estimate; to test formally whether the observed distribution differs from published international and Indonesian reference proportions, quantifying any deviation as a risk difference with an accompanying effect size; and to identify the independent clinical determinants of phototherapy receipt and of extensive disease using multivariable logistic regression with formal assessment of calibration and discrimination.

2. Methods

2.1. Study design and reporting

This was a single-centre, analytic retrospective study of routinely collected clinical data covering a 60-month observation window. The study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement for cross-sectional studies, and the completed STROBE checklist is available from the corresponding author. Conduct and reporting follow the standards of the Committee on Publication Ethics (COPE), the International Committee of Medical Journal Editors (ICMJE) and the World Association of Medical Editors (WAME).

2.2. Setting and participants

The study was conducted at the Dermatology and Venereology Outpatient Clinic of Dr. Moewardi Regional General Hospital, Surakarta, Central Java, Indonesia — the tertiary academic referral centre of the Faculty of Medicine, Universitas Sebelas Maret — and covered all attendances between 1 January 2020 and 31 December 2024. Eligible patients were identified from the hospital medical-record database using the International Classification of Diseases, Tenth Revision, Clinical Modification code L80 (vitiligo). Inclusion criteria were a documented clinical diagnosis of segmental or nonsegmental vitiligo made by a dermatologist during the study period, and a medical record containing the complete set of study variables. Diagnosis was established clinically on the basis of acquired, non-scaly, well-demarcated depigmented macules or patches, in accordance with International Vitiligo Task Force diagnostic recommendations[2] and with PERDOSKI clinical practice guidance, supported where clinically indicated by Wood-lamp examination and by laboratory or histopathological findings recorded in the chart.

Patients were excluded if the medical record was incomplete for any study variable or if the diagnosis of vitiligo was not clearly documented.

2.3. Sample size, precision and detectable effect

No a priori sample-size calculation was performed because the study used total sampling of an entire five-year clinical census rather than a drawn sample. The realised sample was therefore evaluated for precision rather than for power. At n = 175, the 95% Wilson confidence half-width for a proportion near 0.50 is ±7.41 percentage points; 171 observations are required for ±7.5% precision and 385 for ±5.0%. For the principal analytic contrast — phototherapy receipt in patients with multiple versus single lesions, at an observed exposure prevalence of 74.29% and a reference outcome risk of 60% — the minimum detectable odds ratio at $\alpha = 0.05$ (two-sided) and 80% power is 3.14. Associations weaker than this are not reliably detectable in this cohort and are interpreted accordingly.

2.4. Sampling strategy

Consecutive total sampling was applied: every patient meeting the inclusion criteria during the 60-month window was enrolled, with no sampling fraction and no matching. This design eliminates selection bias arising from the sampling process itself, although it cannot remove the referral filter inherent to a tertiary centre, which is addressed in the Discussion.

2.5. Variables and dermatological assessment

The following variables were abstracted from each record onto a standardised extraction form by two investigators, with disagreements resolved by consensus with a senior dermatologist: sex; age at presentation, categorised a priori into five strata (<18, 18–39, 40–49, 50–69 and >69 years); vitiligo classification (nonsegmental or segmental); number of lesions (single or multiple); the principal documented anatomical site (face, upper extremities, lower extremities, anterior trunk, posterior trunk, or other/unspecified); and all treatment modalities administered during the study period (narrowband ultraviolet B phototherapy, topical corticosteroids, oral corticosteroids, zinc, retinoids, emollients and methotrexate). Patients could receive more than one modality. Because the records predate the routine local adoption of quantitative vitiligo instruments, neither the Vitiligo Area Scoring Index nor the Vitiligo Extent Score was available,[21,22] no activity score or quality-of-life instrument was recorded, and Fitzpatrick phototype was not individually documented; the study population is nevertheless a Javanese Indonesian population in which phototypes III–V predominate. These limitations are quantified in the Discussion and motivated the derivation of the surrogate measures described below.

2.6. Derived outcome definitions

Three variables were derived a priori. Phototherapy receipt was defined as receipt of narrowband ultraviolet B phototherapy at any point during the observation window and was used as a clinician-adjudicated proxy for extensive, progressive or topical-refractory disease, since it marks the guideline decision point at which management moves beyond topical therapy. Lesion multiplicity (multiple versus single lesions) was used as the only available surrogate for disease extent. The Therapeutic Complexity Index was defined as the count of distinct modalities recorded per patient (range 0–7) and was used as an ordinal measure of therapeutic intensity. Systemic therapy was defined as receipt of oral corticosteroids or methotrexate.

2.7. Confounders and covariate coding

Age, sex, vitiligo classification, lesion multiplicity and anatomical index site were pre-specified as covariates and entered simultaneously into the multivariable models; no stepwise or data-driven variable selection was used. To preserve degrees of freedom in a cohort of 175, two covariates were collapsed a priori into binary form: age was dichotomised at 18 years in the phototherapy model (the threshold at which cabinet phototherapy becomes routinely practicable) and at 40 years in the lesion-multiplicity model (separating the two modal young strata from the remainder), and anatomical index site was dichotomised as facial versus non-facial. For the contingency analysis of index site by vitiligo classification, sites were collapsed into three groups (face, extremities, trunk or other). This coding yields 5 and 4 degrees of freedom respectively, corresponding to 26.2 and 32.5 events per variable, well above the conventional minimum of ten. Confounders that could not be controlled because they are not recorded in the source charts — Fitzpatrick phototype, disease duration, disease activity, Koebner phenomenon, family history, autoimmune comorbidity, previous treatment and treatment adherence — are enumerated in the Discussion together with their expected direction of bias.

2.8. Reference proportions for international benchmarking

Eleven cohort proportions were compared against reference proportions taken directly from named published studies rather than from informal literature impressions, and each reference value is reported in Table 3 together with its source, source population and denominator. The demographic and anatomical references (female sex 56.3%, age under 18 years 11.8%, facial involvement 32.9%, no treatment 27%) come from a United States claims analysis of 24,949 patients;[26] the phenotype references (segmental 5.93%, nonsegmental 87.78%) from a Saudi tertiary cohort of 573 patients;[1] the United States phototherapy reference (3.9%) from a claims cohort of 19,335 patients;[24] the topical and oral corticosteroid references (29.1% and 4.2%) from a United Kingdom national cohort of 16,741 incident patients;[25] and the two Indonesian comparators (segmental 19.1%, phototherapy 29%) from published domestic series at Bali Mandara General Hospital and Dr. Zainoel Abidin Hospital respectively.[9,10] Because these reference values are themselves estimates, a pre-specified sensitivity analysis repeated every comparison allowing the reference proportion a

sampling variance corresponding to effective synthesis sizes of 5,000, 2,000 and 500 observations.

2.9. Statistical analysis

Analyses were performed in Python 3.11 (NumPy 2.4.4, SciPy 1.17.1, Matplotlib 3.10.9); analysis scripts and the record-level dataset are available from the corresponding author. Categorical variables are summarised as counts and percentages with 95% Wilson score confidence intervals, which are preferred to Wald intervals at proportions near the boundaries. Departures from parity were tested with exact binomial tests and quantified with Cohen's g; the age distribution was tested against uniformity with a χ^2 goodness-of-fit test and quantified with Cramér's V. Cohort proportions were compared with the reference proportions of Section 2.8 using two-proportion tests, reported as risk differences with 95% confidence intervals and Cohen's h. Bivariate associations were assessed with Fisher exact and χ^2 tests and expressed as odds ratios with 95% confidence intervals and Cramér's V. Two pre-specified multivariable binary logistic regression models were fitted — phototherapy receipt and lesion multiplicity — reporting adjusted odds ratios with 95% confidence intervals, the likelihood-ratio χ^2, the Nagelkerke R^2 and the Hosmer–Lemeshow goodness-of-fit statistic. Discrimination was assessed by the area under the receiver-operating-characteristic curve with DeLong 95% confidence intervals, with the Youden-optimal cut-point and its sensitivity, specificity, positive and negative predictive values and likelihood ratios. The Therapeutic Complexity Index was analysed by Poisson regression (incidence rate ratios with 95% confidence intervals) and by Welch t and Mann–Whitney U tests with Cohen's d. Monotonic trend across the ordered age strata was tested with the Cochran–Armitage test, and the correlation structure among phenotype and therapy variables with Spearman rank correlation. All tests were two-sided with $\alpha = 0.05$, and exact p-values are reported to three decimal places.

Five additional analyses were added during revision. Collinearity among model covariates was assessed by variance inflation factors. A multiplicative interaction between lesion multiplicity and vitiligo classification was tested by likelihood-ratio comparison of nested models. Age was re-entered as a five-level ordinal term as a coding sensitivity analysis. Because the area under the curve was estimated in the same data used for model fitting, an optimism-corrected estimate was obtained by Harrell bootstrap over 500 replicates. Finally, the segmental proportion was recomputed under scenarios in which 10%, 25%, 40% and 50% of segmental cases were reclassified as focal or unclassified disease, and the reclassified fraction at which the cohort proportion ceases to differ from each reference value was determined.

2.10. Ethical approval

This study was approved by the Health Research Ethics Committee of Dr. Moewardi Regional General Hospital, Surakarta, Indonesia, approval number 2.155/X/HREC/2025. The study was conducted in accordance with the principles of the Declaration of Helsinki. The requirement for written informed consent was waived because of the retrospective use of anonymised medical record data.

3. Results

3.1. Cohort characteristics

A total of 175 patients with vitiligo were identified at the Dermatology and Venereology Outpatient Clinic of Dr. Moewardi Regional General Hospital, Surakarta, between January 2020 and December 2024. Their complete sociodemographic and dermatological profile is detailed in Table 1 and displayed graphically in Figure 1. Women accounted for 101 patients (57.71%; 95% CI 50.31–64.79) and men for 74 patients (42.29%; 95% CI 35.21–49.69), a female-to-male ratio of 1.36:1; as shown in Table 1, this female excess was of borderline statistical significance and small magnitude (exact binomial $p = 0.049$; Cohen's $g = 0.077$). The cohort was young: 63 patients (36.00%; 95% CI 29.26–43.34) were aged 18–39 years and 61 patients (34.86%; 95% CI 28.19–42.17) were younger than 18 years, so that 124 patients (70.86%; 95% CI 63.73–77.08) were under 40 years of age ($p < 0.001$ against parity; $g = 0.209$). Only 4 patients (2.29%; 95% CI 0.89–5.73) were older than 69 years. As Figure 1A shows, the age distribution departed markedly from uniformity ($\chi^2 = 83.03$, df = 4, $p < 0.001$; Cramér's V = 0.344). Nonsegmental vitiligo was recorded in 109 patients (62.29%; 95% CI 54.91–69.13) and segmental vitiligo in 66 patients (37.71%; 95% CI 30.87–45.09) (binomial $p = 0.001$ against parity; $g = 0.123$), and multiple lesions were documented in 130 patients (74.29%; 95% CI 67.34–80.19) against a single lesion in 45 patients (25.71%; 95% CI 19.81–32.66) ($p < 0.001$; $g = 0.243$); both contrasts are plotted with their confidence intervals in Figure 1B.

3.2. Anatomical distribution and treatment utilisation

The anatomical and therapeutic profile of the cohort is detailed in Table 2 and plotted with 95% confidence intervals in Figure 2A. The face was the most frequently documented index site, affecting 55 patients (31.43%; 95% CI 25.01–38.64), followed by the upper extremities (24 patients, 13.71%; 95% CI 9.39–19.60), the lower extremities (21 patients, 12.00%; 95% CI 7.98–17.65), the anterior trunk (18 patients, 10.29%; 95% CI 6.61–15.67) and the posterior trunk (15 patients, 8.57%; 95% CI 5.26–13.66); the remaining 42 patients (24.00%; 95% CI 18.27–30.84) were recorded at other or unspecified sites. Facial predominance over every other individual site was statistically decisive ($p < 0.001$). As Table 2 shows, corticosteroid-based therapy in any form was the commonest treatment (148 patients, 84.57%; 95% CI 78.48–89.17), comprising topical corticosteroids in 134 patients (76.57%; 95% CI 69.77–82.23) and oral corticosteroids in 14 patients (8.00%; 95% CI 4.83–12.98). Narrowband ultraviolet B phototherapy was delivered to 131 patients (74.86%; 95% CI 67.94–80.70). Adjunctive modalities comprised zinc (69 patients, 39.43%; 95% CI 32.49–46.82), retinoids (37 patients, 21.14%; 95% CI 15.75–27.78),

emollients (27 patients, 15.43%; 95% CI 10.83–21.52) and methotrexate (5 patients, 2.86%; 95% CI 1.23–6.51); only 1 patient (0.57%; 95% CI 0.10–3.17) had no treatment recorded. Patients received a mean of 2.38 ± 1.00 modalities each.

Table 1. Sociodemographic and dermatological characteristics of patients with vitiligo at the Dermatology and Venereology Outpatient Clinic of Dr. Moewardi Regional General Hospital, Surakarta (n = 175).

Characteristic	n	%	95% Wilson CI	Interpretation
Sex				
Female	101	57.71	50.31–64.79	Female-to-male ratio 1.36:1
Male	74	42.29	35.21–49.69	—
Age stratum, years				
<18	61	34.86	28.19–42.17	Paediatric/adolescent presentation
18–39	63	36.00	29.26–43.34	Modal stratum
40–49	13	7.43	4.39–12.29	—
50–69	34	19.43	14.25–25.92	—
>69	4	2.29	0.89–5.73	Wide interval; sparse stratum
Vitiligo classification				
Nonsegmental	109	62.29	54.91–69.13	Systemic autoimmune phenotype
Segmental	66	37.71	30.87–45.09	Localised phenotype; surgical candidate if stable
Number of lesions				
Multiple	130	74.29	67.34–80.19	Extent surrogate — extensive disease
Single	45	25.71	19.81–32.66	Limited disease
Therapeutic Complexity Index (0–7)	**175**	**2.38 ± 1.00**	**median 2 (IQR 2–3)**	**Mean number of modalities per patient**

Notes: *CI, confidence interval; IQR, interquartile range. Percentages are of the total cohort (n = 175). The Therapeutic Complexity Index row reports mean ± standard deviation rather than a percentage.*

Table 2. Anatomical distribution of the principal documented lesion site and treatment utilisation, with 95% Wilson confidence intervals (n = 175).

Variable	n	%	95% Wilson CI
Principal anatomical site			
Face	55	31.43	25.01–38.64
Upper extremities	24	13.71	9.39–19.60
Lower extremities	21	12.00	7.98–17.65
Anterior trunk	18	10.29	6.61–15.67
Posterior trunk	15	8.57	5.26–13.66
Other / unspecified	42	24.00	18.27–30.84
Treatment modality			
Corticosteroids, any	148	84.57	78.48–89.17
Topical corticosteroids	134	76.57	69.77–82.23
Oral corticosteroids	14	8.00	4.83–12.98
Narrowband ultraviolet B phototherapy	131	74.86	67.94–80.70
Zinc	69	39.43	32.49–46.82
Retinoids	37	21.14	15.75–27.78
Emollients	27	15.43	10.83–21.52
Methotrexate	5	2.86	1.23–6.51
No treatment recorded	1	0.57	0.10–3.17

Notes: *Treatment categories are not mutually exclusive; patients could receive more than one modality. "Corticosteroids, any" is the union of the topical and oral routes; note that the topical (134) and oral (14) counts sum exactly to the union (148), which implies that the two routes were documented as mutually exclusive in this record set rather than that no patient ever received both — a documentation convention discussed as a limitation.*

3.3. Comparison with published reference proportions

Each cohort proportion was compared with the corresponding published reference value, as detailed in Table 3 and displayed as risk differences with 95% confidence intervals in Figure 2B. Four deviations were large. The segmental fraction of 37.71% exceeded the international tertiary-cohort reference of 5.93% by +31.78 percentage points (95% CI +24.60 to +38.97; Cohen's h = 0.831; p < 0.001), and still exceeded the Indonesian reference of 19.1% by +18.61 percentage points (95% CI +11.43 to +25.80; Cohen's h = 0.418; p < 0.001). Phototherapy utilisation of 74.86% exceeded United States real-world utilisation of 3.9% by +70.96 percentage points (95% CI +64.53 to +77.38; Cohen's h = 1.694; p < 0.001) and Indonesian utilisation of 29% by +45.86 percentage points (95% CI +39.43 to +52.28; Cohen's h = 0.954; p < 0.001). Topical corticosteroid use of 76.57% exceeded the United Kingdom national reference of 29.1% by +47.47 percentage points (95% CI +41.20 to +53.75; Cohen's h = 0.992; p < 0.001), and the proportion left untreated (0.57%) fell below the international reference of 27% by −26.43 percentage points (95% CI −27.55 to −25.31; Cohen's h = 0.941; p < 0.001). Patients younger than 18 years were over-represented by +23.06 percentage points (95% CI +16.00 to +30.12; Cohen's h = 0.562; p < 0.001). By contrast, and as Figure 2B makes visually clear, three comparisons were null: female predominance (57.71% versus 56.3%; p = 0.706), facial index site (31.43% versus 32.9%; p = 0.679) and oral corticosteroid use (8.00% versus 4.2%; p = 0.012), the last of which did not survive correction for multiple comparisons.

Table 3. Comparison of cohort proportions with reference proportions taken from named published studies, with risk differences, 95% confidence intervals and Cohen's h.

Parameter	Cohort % (95% CI)	Ref. %	Reference source	Risk difference, pp (95% CI)	Cohen's h	p
Female sex	57.71 (50.31–64.79)	56.30	Adiri 2026, US claims, n = 24,949[26]	+1.41 (−5.90 to +8.73)	0.029	0.706
Age <18 years at presentation	34.86 (28.19–42.17)	11.80	Adiri 2026, US claims, n = 24,949[26]	+23.06 (+16.00 to +30.12)	0.562	<0.001
Segmental vitiligo (international)	37.71 (30.87–45.09)	5.93	AlJasser 2024, Saudi tertiary, n = 573[1]	+31.78 (+24.60 to +38.97)	0.831	<0.001
Segmental vitiligo (Indonesian)	37.71 (30.87–45.09)	19.10	Mahadewi 2025, Bali Mandara, n = 198[10]	+18.61 (+11.43 to +25.80)	0.418	<0.001
Nonsegmental vitiligo	62.29 (54.91–69.13)	87.78	AlJasser 2024, Saudi tertiary, n = 573[1]	−25.49 (−32.68 to −18.31)	0.608	<0.001
Facial (head/neck) index site	31.43 (25.01–38.64)	32.90	Adiri 2026, US claims, n = 16,674 with site data[26]	−1.47 (−8.35 to +5.41)	0.032	0.679
Phototherapy utilisation (US real-world)	74.86 (67.94–80.70)	3.90	Rosmarin 2023, US claims, n = 19,335[24]	+70.96 (+64.53 to +77.38)	1.694	<0.001
Phototherapy utilisation (Indonesian)	74.86 (67.94–80.70)	29.00	Earlia 2024, Banda Aceh, n = 215[9]	+45.86 (+39.43 to +52.28)	0.954	<0.001
Topical corticosteroid use	76.57 (69.77–82.23)	29.10	Eleftheriadou 2024, UK national, n = 16,741[25]	+47.47 (+41.20 to +53.75)	0.992	<0.001
Oral corticosteroid use	8.00 (4.83–12.98)	4.20	Eleftheriadou 2024, UK national, n = 16,741[25]	+3.80 (−0.22 to +7.82)	0.161	0.012
No treatment recorded	0.57 (0.10–3.17)	27.00	Adiri 2026, US claims, n = 24,949[26]	−26.43 (−27.55 to −25.31)	0.941	<0.001

Notes: *pp, percentage points. Cohen's h: 0.20 small, 0.50 medium, 0.80 large. The Bonferroni-corrected threshold for this family of eleven comparisons is α = 0.0045.*

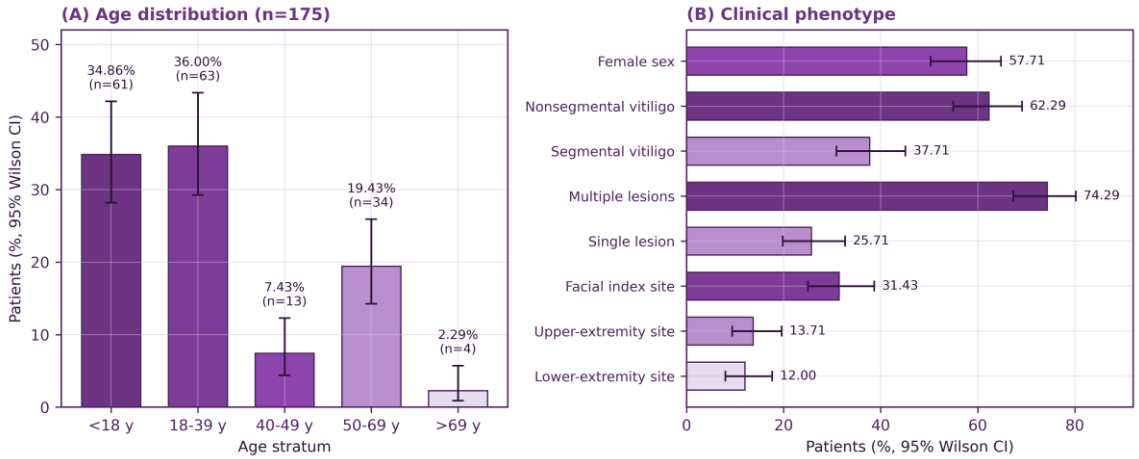

Figure 1. Demographic and clinical phenotype profile of the cohort (n = 175). (A) Distribution across the five age strata with 95% Wilson confidence intervals; (B) clinical phenotype proportions with 95% Wilson confidence intervals. Data correspond to Table 1.

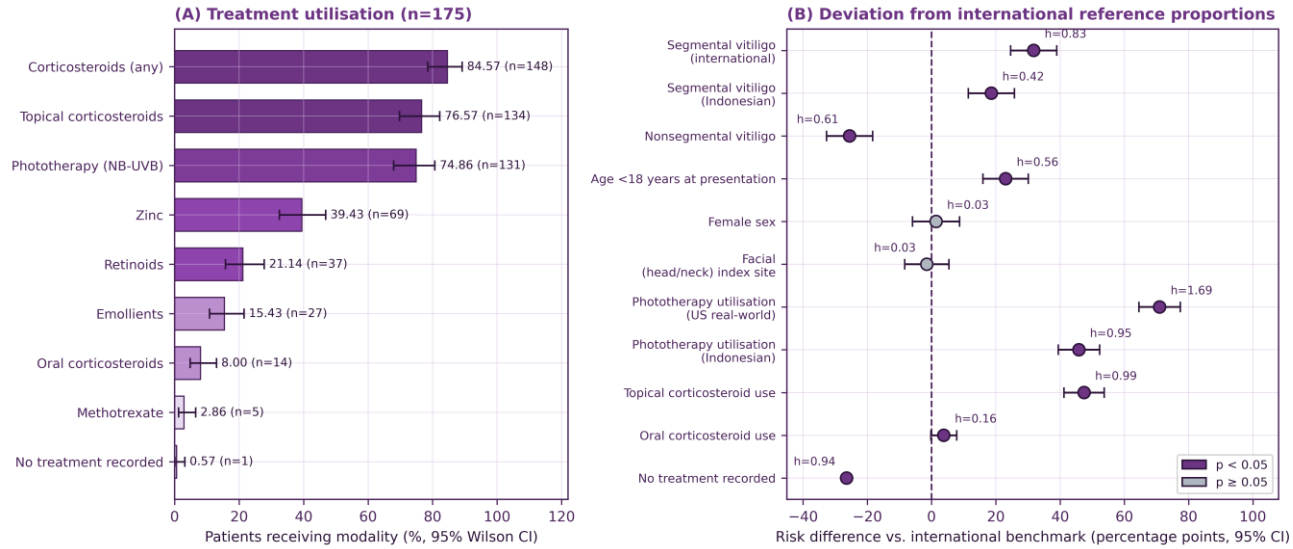

Figure 2. Treatment utilisation and deviation from published reference proportions. (A) Proportion of patients receiving each modality, with 95% Wilson confidence intervals (data as in Table 2); (B) risk differences against the published reference proportions of Table 3, with 95% confidence intervals and Cohen's h; filled markers denote $p < 0.05$.

3.4. Bivariate determinants of treatment receipt and disease extent

All bivariate associations are detailed in Table 4. Phototherapy was received by 87.69% of patients with multiple lesions versus 37.78% of those with a single lesion (odds ratio 11.74; 95% CI 5.28–26.07; $p < 0.001$; Cramér's V = 0.503), and by 87.16% of patients with nonsegmental disease versus 54.55% of those with segmental disease (5.65; 2.69–11.87; $p < 0.001$; V = 0.364). Patients aged 18 years or older were more likely to receive phototherapy than children (79.82% versus 65.57%; 2.08; 1.03–4.18; $p = 0.045$), whereas a facial index site was associated with lower phototherapy use (58.18% versus 82.50%; 0.30; 0.14–0.60; $p = 0.001$; V = 0.260). Sex was unrelated to phototherapy ($p = 0.862$). As the second block of Table 4 shows, multiple lesions were far more frequent in nonsegmental than in segmental disease (88.07% versus 51.52%; 6.95; 3.27–14.77; $p < 0.001$; V = 0.405), while neither age ($p = 0.708$) nor sex ($p = 0.730$) was associated with extent. Systemic therapy was confined entirely to patients with multiple lesions (19/130 versus 0/45; $p = 0.004$) and was more frequent in nonsegmental disease (3.61; 1.01–12.92; $p = 0.045$). Neither lesion multiplicity ($z = 0.33$; $p = 0.742$) nor phototherapy receipt ($z = 0.75$; $p = 0.453$) showed a monotonic trend across the five ordered age strata, and the distribution of index sites did not differ between nonsegmental and segmental disease ($\chi^2 = 1.21$, df = 2; $p = 0.547$; V = 0.083).

Table 4. Bivariate determinants of phototherapy receipt, lesion multiplicity and systemic therapy.

Exposure	% exposed	% unexposed	Odds ratio (95% CI)	Cramér's V	p
Outcome: phototherapy receipt					
Multiple lesions (vs single)	87.69	37.78	11.74 (5.28–26.07)	0.503	<0.001
Nonsegmental vitiligo (vs segmental)	87.16	54.55	5.65 (2.69–11.87)	0.364	<0.001
Age ≥18 years (vs <18)	79.82	65.57	2.08 (1.03–4.18)	0.157	0.045
Female sex (vs male)	74.26	75.68	0.93 (0.46–1.86)	0.016	0.862
Facial index site (vs non-facial)	58.18	82.50	0.30 (0.14–0.60)	0.260	0.001
Outcome: multiple lesions					
Nonsegmental vitiligo (vs segmental)	88.07	51.52	6.95 (3.27–14.77)	0.405	<0.001
Age ≥40 years (vs <40)	76.47	73.39	1.18 (0.55–2.52)	0.032	0.708
Female sex (vs male)	75.25	72.97	1.13 (0.57–2.23)	0.026	0.730
Facial index site (vs non-facial)	65.45	78.33	0.52 (0.26–1.06)	0.137	0.093
Outcome: systemic therapy (oral corticosteroid or methotrexate)					
Multiple lesions (vs single)	14.62	0.00	15.91 (0.94–269.20)	0.205	0.004
Nonsegmental vitiligo (vs segmental)	14.68	4.55	3.61 (1.01–12.92)	0.158	0.045

Notes: p-values are from Fisher exact tests. The odds ratio for systemic therapy by lesion multiplicity is Haldane–Anscombe corrected because of a zero cell (0 of 45 patients with a single lesion received systemic therapy).

3.5. Multivariable models, discrimination and therapeutic intensity

Both multivariable models are detailed in Table 5, their discrimination is displayed in Figure 3 and the adjusted odds ratios of the phototherapy model are plotted in Figure 4A. In the model for phototherapy receipt, multiple lesions (adjusted odds ratio 8.39; 95% CI 3.41–20.64; p < 0.001) and nonsegmental disease (3.59; 95% CI 1.46–8.79; p = 0.005) remained independent positive determinants, and a facial index site an independent negative determinant (0.35; 95% CI 0.14–0.86; p = 0.023), whereas adult age (2.34; 95% CI 0.94–5.83; p = 0.069) and female sex (0.66; 95% CI 0.28–1.57; p = 0.349) did not reach significance after adjustment. The model was globally significant (likelihood-ratio χ^2 = 59.72, df = 5, p < 0.001), explained 42.8% of the variance (Nagelkerke R^2 = 0.428) and was well calibrated (Hosmer–Lemeshow χ^2 = 2.84, df = 8, p = 0.944). As Figure 3 shows, discrimination was good, with an area under the receiver-operating-characteristic curve of 0.859 (95% CI 0.798–0.919; p < 0.001). At the Youden-optimal predicted-probability cut-point of 0.787, marked on Figure 3, sensitivity was 76.34% (95% CI 68.37–82.80), specificity 81.82% (68.04–90.49), positive predictive value 92.59% (86.06–96.20), negative predictive value 53.73% (41.92–65.14), positive likelihood ratio 4.20 and negative likelihood ratio 0.29; overall accuracy was 77.71% (70.99–83.25).

In the model for lesion multiplicity, also detailed in Table 5, nonsegmental disease was the only independent determinant (6.93; 95% CI 3.22–14.90; p < 0.001); age (1.15; 95% CI 0.49–2.68; p = 0.743), sex (0.88; 95% CI 0.41–1.88; p = 0.735) and facial index site (0.56; 95% CI 0.26–1.22; p = 0.146) were not. This model was significant (likelihood-ratio χ^2 = 30.76, df = 4, p < 0.001) and calibrated (Hosmer–Lemeshow p = 0.928) but explained less variance (Nagelkerke R^2 = 0.237) and, as the lower curve in Figure 3 shows, discriminated less well (area under the curve 0.757; 95% CI 0.673–0.841).

Therapeutic intensity mirrored disease extent, as the Poisson block of Table 5 details. Patients with multiple lesions received 2.58 ± 0.94 modalities versus 1.80 ± 0.97 in those with a single lesion (Welch t = 4.72; p < 0.001; Mann–Whitney p < 0.001; Cohen's d = 0.83, 95% CI 0.48–1.18), and patients with nonsegmental disease received 2.58 ± 0.96 versus 2.06 ± 1.01 modalities (d = 0.53, 0.22–0.84; p = 0.001); age had no effect (d = −0.02; p = 0.916). In the Poisson model, multiple lesions independently increased the expected number of modalities by 36.3% (incidence rate ratio 1.363; 95% CI 1.049–1.771; p = 0.021), whereas vitiligo classification (p = 0.315), age (p = 0.871) and sex (p = 0.719) did not contribute independently. The Spearman correlation matrix in Figure 4B shows the strongest associations between phototherapy and therapeutic complexity (ρ = 0.579; p < 0.001), between lesion multiplicity and phototherapy (ρ = 0.503; p < 0.001) and between nonsegmental classification and lesion multiplicity (ρ = 0.405; p < 0.001); a facial index site correlated inversely with both younger age (ρ = −0.263; p < 0.001) and phototherapy (ρ = −0.260; p < 0.001).

Table 5. Multivariable logistic regression models, model performance, discrimination and Poisson analysis of therapeutic intensity.

Term	Adjusted OR / statistic	95% CI / detail	p
Model A — phototherapy receipt (n = 175; 131 events)			
Multiple lesions	8.39	3.41–20.64	<0.001
Nonsegmental vitiligo	3.59	1.46–8.79	0.005
Age ≥18 years	2.34	0.94–5.83	0.069
Female sex	0.66	0.28–1.57	0.349
Facial index site	0.35	0.14–0.86	0.023
Model fit	R^2 = 0.428	LR χ^2 = 59.72 (df 5)	<0.001
Calibration (Hosmer–Lemeshow)	χ^2 = 2.84	df = 8	0.944
Discrimination (AUC, DeLong CI)	0.859	0.798–0.919	<0.001
Youden cut-point / Sn / Sp	0.787	76.34% / 81.82%	—
PPV / NPV / LR+ / LR−	92.59% / 53.73%	4.20 / 0.29	—
Model B — multiple lesions (n = 175; 130 events)			
Nonsegmental vitiligo	6.93	3.22–14.90	<0.001
Age ≥40 years	1.15	0.49–2.68	0.743
Female sex	0.88	0.41–1.88	0.735
Facial index site	0.56	0.26–1.22	0.146
Model fit / calibration / AUC	R^2 = 0.237	H–L p = 0.928; AUC 0.757 (0.673–0.841)	<0.001
Model C — Poisson, Therapeutic Complexity Index (0–7)	**IRR**	**95% CI**	**p**
Multiple lesions	1.363	1.049–1.771	0.021
Nonsegmental vitiligo	1.121	0.897–1.400	0.315
Age ≥18 years	0.983	0.802–1.206	0.871
Female sex	1.037	0.850–1.265	0.719

Notes: *OR, odds ratio; IRR, incidence rate ratio; AUC, area under the receiver-operating-characteristic curve; Sn, sensitivity; Sp, specificity; PPV, positive predictive value; NPV, negative predictive value; LR+/LR−, positive and negative likelihood ratios. All covariates were entered simultaneously; no stepwise selection was used.*

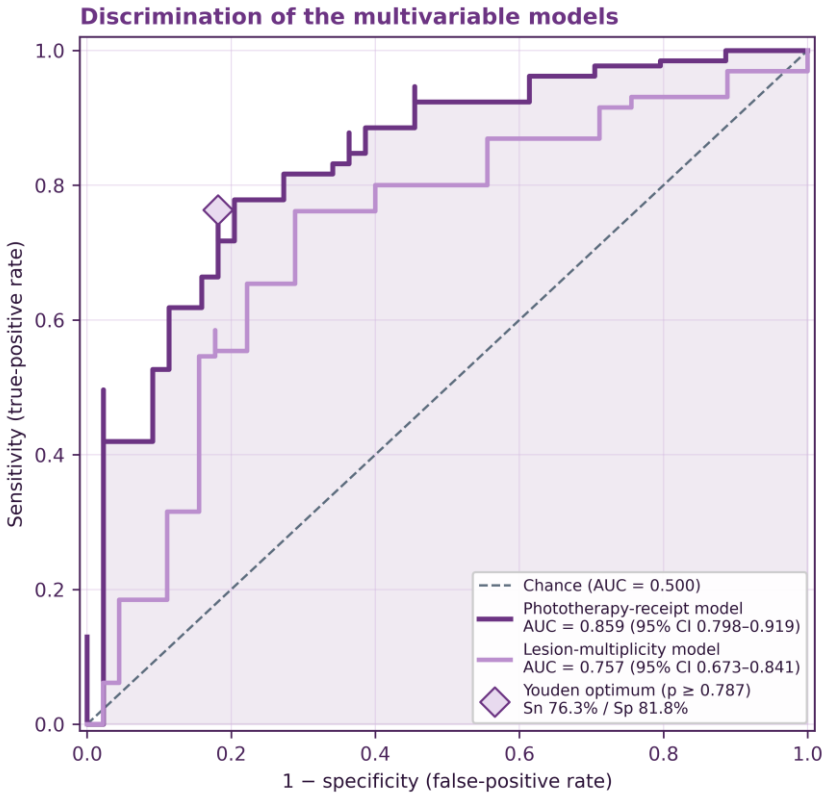

Figure 3. Receiver-operating-characteristic curves of the two multivariable logistic models reported in Table 5, with DeLong 95% confidence intervals and the Youden-optimal operating point of the phototherapy-receipt model.

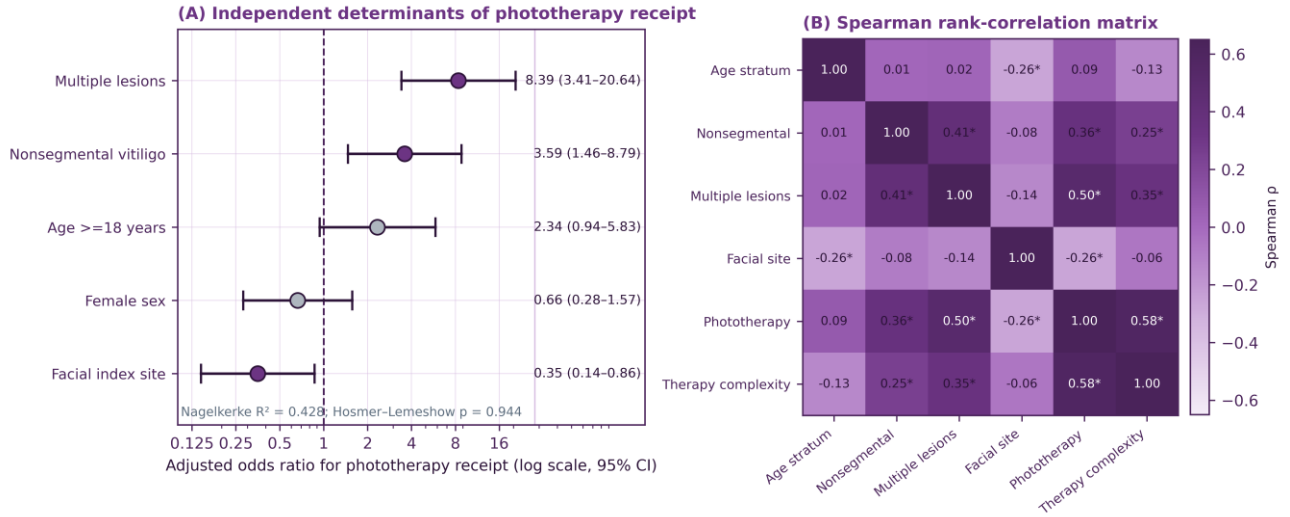

Figure 4. Independent determinants and correlation structure. (A) Forest plot of the adjusted odds ratios of Table 5, Model A (phototherapy receipt), on a logarithmic scale, with Nagelkerke R^2 and Hosmer–Lemeshow calibration; (B) Spearman rank-correlation matrix across phenotype and therapy variables, with asterisks denoting $p < 0.05$.

3.6. Complete cross-tabulation of phenotype, demographic and treatment variables

To make the joint distribution behind the bivariate and multivariable analyses fully inspectable, every study variable was cross-tabulated against the three axes that organise the analysis — vitiligo classification, detailed in Table 6; lesion multiplicity, detailed in Table 7; and phototherapy receipt, detailed in Table 8 — with cell counts, within-column percentages, the test statistic, the exact p-value and Cramér's V. As Table 6 shows, only two variables differed by vitiligo classification: lesion multiplicity ($\chi^2 = 28.76$, df = 1; $p < 0.001$; V = 0.405) and phototherapy receipt ($\chi^2 = 23.23$, df = 1; $p < 0.001$; V = 0.364), with systemic therapy at the margin (p = 0.045; V = 0.158). Sex (p = 0.210), age stratum (p = 0.609), anatomical index site (p = 0.587) and every topical or adjuvant modality were distributed comparably between the nonsegmental and segmental groups, indicating that the classification axis drives extent and phototherapy allocation but not demographic composition or first-line prescribing.

Table 6. Complete cross-tabulation of demographic, clinical and treatment variables by vitiligo classification (n = 175).

Variable	Nonsegmental (n = 109) n (%)	Segmental (n = 66) n (%)	Test statistic	p	Cramér's V
Sex			**χ^2 = 1.67, df = 1**	**0.210**	**0.098**
Female	67 (61.5)	34 (51.5)			
Male	42 (38.5)	32 (48.5)			
Age stratum, years			**χ^2 = 2.70, df = 4**	**0.609**	**0.124**
<18	38 (34.9)	23 (34.8)			
18–39	39 (35.8)	24 (36.4)			
40–49	6 (5.5)	7 (10.6)			
50–69	24 (22.0)	10 (15.2)			
>69	2 (1.8)	2 (3.0)			
Number of lesions			**χ^2 = 28.76, df = 1**	**<0.001**	**0.405**
Multiple	96 (88.1)	34 (51.5)			
Single	13 (11.9)	32 (48.5)			
Principal anatomical site			**χ^2 = 3.74, df = 5**	**0.587**	**0.146**
Face	31 (28.4)	24 (36.4)			
Upper extremities	14 (12.8)	10 (15.2)			
Lower extremities	15 (13.8)	6 (9.1)			
Anterior trunk	11 (10.1)	7 (10.6)			
Posterior trunk	8 (7.3)	7 (10.6)			
Other/unspecified	30 (27.5)	12 (18.2)			
Phototherapy	95 (87.2)	36 (54.5)	χ^2 = 23.23, df = 1	<0.001	0.364
Corticosteroids, any	95 (87.2)	53 (80.3)	χ^2 = 1.48, df = 1	0.281	0.092
Topical corticosteroids	83 (76.1)	51 (77.3)	χ^2 = 0.03, df = 1	1.000	0.013
Oral corticosteroids	11 (10.1)	3 (4.5)	χ^2 = 1.72, df = 1	0.255	0.099
Zinc	45 (41.3)	24 (36.4)	χ^2 = 0.42, df = 1	0.529	0.049
Retinoids	27 (24.8)	10 (15.2)	χ^2 = 2.28, df = 1	0.181	0.114
Emollients	15 (13.8)	12 (18.2)	χ^2 = 0.62, df = 1	0.518	0.059
Methotrexate	5 (4.6)	0 (0.0)	χ^2 = 3.12, df = 1	0.158	0.133
Systemic therapy (oral CS or MTX)	16 (14.7)	3 (4.5)	χ^2 = 4.36, df = 1	0.045	0.158

Notes: *Percentages are within-column (that is, of the 109 nonsegmental and the 66 segmental patients respectively). Binary treatment rows report the number receiving that modality. p-values are from Fisher exact tests for 2×2 tables and Pearson χ^2 tests otherwise. Cramér's V: 0.10 small, 0.30 medium, 0.50 large.*

Cross-tabulation against lesion multiplicity, detailed in Table 7, showed the expected gradient in therapeutic intensity: patients with multiple lesions were more likely to receive phototherapy (χ^2 = 44.25, df = 1; p < 0.001; V = 0.503), zinc (p < 0.001; V = 0.261), oral corticosteroids (p = 0.022) and systemic therapy (p = 0.004), and correspondingly less likely to receive topical corticosteroids (p = 0.025; V = 0.171). Total corticosteroid exposure did not differ (p = 0.812), confirming that extent shifts the route and the adjuvant burden rather than whether a corticosteroid is used at all.

Cross-tabulation against phototherapy receipt, detailed in Table 8, identified three determinants: lesion multiplicity (χ^2 = 44.25, df = 1; p < 0.001; V = 0.503), vitiligo classification (χ^2 = 23.23, df = 1; p < 0.001; V = 0.364) and anatomical index site (χ^2 = 16.58, df = 5; p = 0.005; V = 0.308), the last reflecting the low phototherapy fraction among patients with facial disease. Age stratum showed a non-significant gradient (p = 0.091; V = 0.214), and sex (p = 0.862) and every concurrent topical or adjuvant modality were unrelated to phototherapy receipt, indicating that phototherapy was added to, rather than substituted for, conventional topical management. The complete pairwise association structure across all eight categorical variables is displayed in Figure 5, in which the strongest associations are lesion multiplicity with phototherapy (V = 0.503), vitiligo classification with lesion multiplicity (V = 0.405) and topical corticosteroid use with systemic therapy (V = 0.410).

Table 7. Complete cross-tabulation of demographic, clinical and treatment variables by lesion multiplicity (n = 175).

Variable	Multiple (n = 130) n (%)	Single (n = 45) n (%)	Test statistic	p	Cramér's V
Sex			**χ^2 = 0.12, df = 1**	**0.730**	**0.026**
Female	76 (58.5)	25 (55.6)			
Male	54 (41.5)	20 (44.4)			
Age stratum, years			**χ^2 = 2.63, df = 4**	**0.622**	**0.123**
<18	45 (34.6)	16 (35.6)			
18–39	46 (35.4)	17 (37.8)			
40–49	9 (6.9)	4 (8.9)			
50–69	28 (21.5)	6 (13.3)			
>69	2 (1.5)	2 (4.4)			
Vitiligo classification			**χ^2 = 28.76, df = 1**	**<0.001**	**0.405**
Nonsegmental	96 (73.8)	13 (28.9)			
Segmental	34 (26.2)	32 (71.1)			
Principal anatomical site			**χ^2 = 8.46, df = 5**	**0.133**	**0.220**
Face	36 (27.7)	19 (42.2)			
Upper extremities	22 (16.9)	2 (4.4)			
Lower extremities	17 (13.1)	4 (8.9)			
Anterior trunk	12 (9.2)	6 (13.3)			
Posterior trunk	13 (10.0)	2 (4.4)			
Other/unspecified	30 (23.1)	12 (26.7)			
Phototherapy	114 (87.7)	17 (37.8)	χ^2 = 44.25, df = 1	<0.001	0.503
Corticosteroids, any	109 (83.8)	39 (86.7)	χ^2 = 0.20, df = 1	0.812	0.034
Topical corticosteroids	94 (72.3)	40 (88.9)	χ^2 = 5.12, df = 1	0.025	0.171
Oral corticosteroids	14 (10.8)	0 (0.0)	χ^2 = 5.27, df = 1	0.022	0.173
Zinc	61 (46.9)	8 (17.8)	χ^2 = 11.89, df = 1	<0.001	0.261
Retinoids	31 (23.8)	6 (13.3)	χ^2 = 2.22, df = 1	0.203	0.113
Emollients	17 (13.1)	10 (22.2)	χ^2 = 2.14, df = 1	0.156	0.111
Methotrexate	5 (3.8)	0 (0.0)	χ^2 = 1.78, df = 1	0.330	0.101
Systemic therapy (oral CS or MTX)	19 (14.6)	0 (0.0)	χ^2 = 7.38, df = 1	0.004	0.205

Notes: *Percentages are within-column (of the 130 patients with multiple lesions and the 45 with a single lesion respectively). Statistical conventions as for Table 6.*

3.7. Sensitivity and bias-quantification analyses

Reclassifying a proportion of segmental cases as focal or unclassified disease attenuated but did not abolish the segmental excess. If 10% were reclassified the segmental fraction fell to 33.71% (95% CI 27.13–41.00; risk difference +27.78 points against the international reference; Cohen's h = 0.747; p < 0.001); at 25% it fell to 28.57% (h = 0.636; p < 0.001); at 40% to 22.86% (h = 0.505; p < 0.001); and at 50% to 18.86% (h = 0.406; p < 0.001). Against the international reference the tipping point was reached only when 75.8% of segmental cases were reclassified, at which point the residual fraction of 9.14% became statistically indistinguishable from 5.93% (p = 0.072); against the Indonesian reference of 19.1% the corresponding tipping point was 34.8% (residual fraction 24.57%; p = 0.066). Under worst-case attrition, in which 5%, 10% or 20% of eligible patients were excluded for incomplete records and all of them had nonsegmental disease, the segmental fraction would fall only to 35.87%, 34.02% and 30.14% respectively — all still far above both reference values.

Applying a Bonferroni-adjusted threshold of α = 0.0045 to the family of eleven benchmark comparisons in Table 3, 8 deviations remained significant and 3 did not (female sex, facial index site and oral corticosteroid use); both headline findings therefore survive correction for multiplicity with a wide margin. Firth penalised re-estimation of the association between lesion multiplicity and systemic therapy, undertaken because of the zero cell in Table 4, produced an odds ratio of 15.91 (95% CI 0.91–277.60; p = 0.058), confirming that this association is directionally consistent but too imprecisely estimated to support inference. Bootstrap resampling of the phototherapy model over 1000 replicates gave a mean area under the curve of 0.859 (percentile 95% CI 0.796–0.913), closely reproducing the DeLong interval reported in Table 5 and Figure 3.

Table 8. Complete cross-tabulation of demographic, clinical and concurrent treatment variables by phototherapy receipt (n = 175).

Variable	Phototherapy (n = 131) n (%)	No phototherapy (n = 44) n (%)	Test statistic	p	Cramér's V
Sex			**χ^2 = 0.05, df = 1**	**0.862**	**0.016**
Female	75 (57.3)	26 (59.1)			
Male	56 (42.7)	18 (40.9)			
Age stratum, years			**χ^2 = 8.01, df = 4**	**0.091**	**0.214**
<18	40 (30.5)	21 (47.7)			
18–39	54 (41.2)	9 (20.5)			
40–49	8 (6.1)	5 (11.4)			
50–69	26 (19.8)	8 (18.2)			
>69	3 (2.3)	1 (2.3)			
Vitiligo classification			**χ^2 = 23.23, df = 1**	**<0.001**	**0.364**
Nonsegmental	95 (72.5)	14 (31.8)			
Segmental	36 (27.5)	30 (68.2)			
Number of lesions			**χ^2 = 44.25, df = 1**	**<0.001**	**0.503**
Multiple	114 (87.0)	16 (36.4)			
Single	17 (13.0)	28 (63.6)			
Principal anatomical site			**χ^2 = 16.58, df = 5**	**0.005**	**0.308**
Face	32 (24.4)	23 (52.3)			
Upper extremities	23 (17.6)	1 (2.3)			
Lower extremities	16 (12.2)	5 (11.4)			
Anterior trunk	15 (11.5)	3 (6.8)			
Posterior trunk	10 (7.6)	5 (11.4)			
Other/unspecified	35 (26.7)	7 (15.9)			
Corticosteroids, any	109 (83.2)	39 (88.6)	χ^2 = 0.74, df = 1	0.475	0.065
Topical corticosteroids	97 (74.0)	37 (84.1)	χ^2 = 1.85, df = 1	0.219	0.103
Oral corticosteroids	11 (8.4)	3 (6.8)	χ^2 = 0.11, df = 1	1.000	0.025
Zinc	56 (42.7)	13 (29.5)	χ^2 = 2.40, df = 1	0.154	0.117
Retinoids	31 (23.7)	6 (13.6)	χ^2 = 1.99, df = 1	0.202	0.107
Emollients	24 (18.3)	3 (6.8)	χ^2 = 3.34, df = 1	0.090	0.138
Methotrexate	5 (3.8)	0 (0.0)	χ^2 = 1.73, df = 1	0.332	0.099
Systemic therapy (oral CS or MTX)	16 (12.2)	3 (6.8)	χ^2 = 0.99, df = 1	0.410	0.075

Notes: *Percentages are within-column (of the 131 patients who received phototherapy and the 44 who did not). Statistical conventions as for Table 6.*

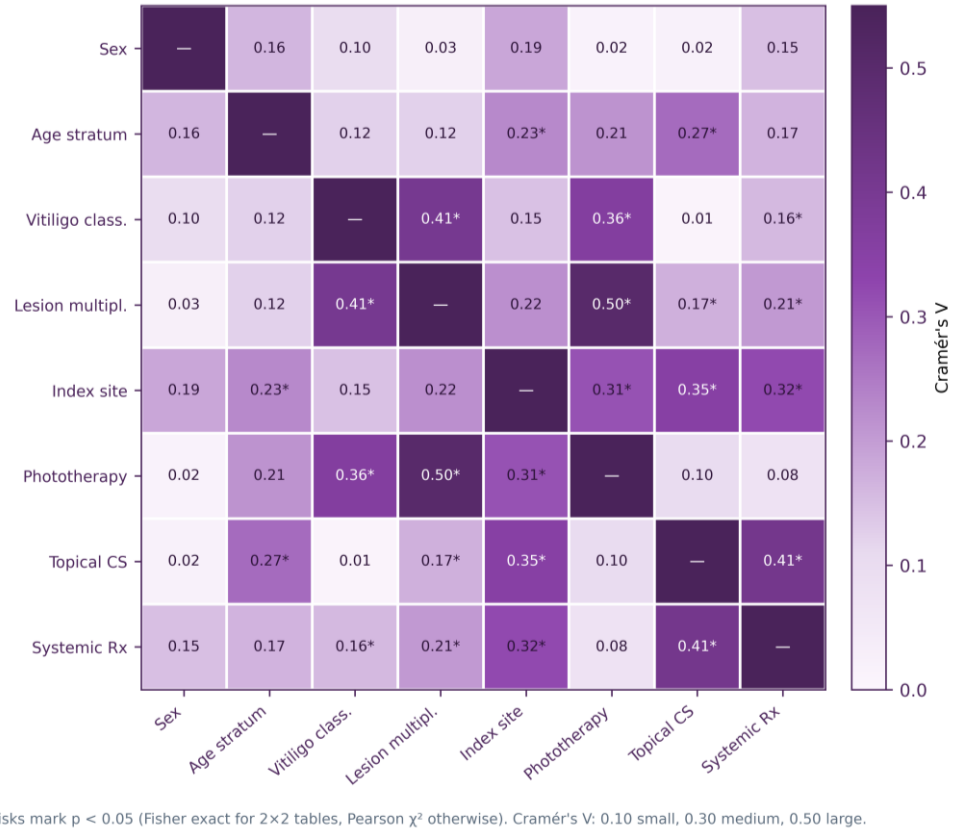

Figure 5. Pairwise association matrix (Cramér's V) across all phenotype, demographic and treatment variables reported in Tables 6 to 8, with asterisks marking p < 0.05 (Fisher exact for 2×2 tables, Pearson χ^2 otherwise).

Allowing the reference proportions of Table 3 to carry sampling error rather than treating them as fixed constants left every large comparison unchanged: even at an effective reference synthesis size of only 500 observations, the segmental comparison remained decisive (p < 0.001), as did phototherapy utilisation (p < 0.001), topical corticosteroid use (p < 0.001) and the untreated fraction (p < 0.001). The borderline oral corticosteroid comparison lost significance under the same allowance (p = 0.090 at a reference size of 500 versus p = 0.066 at 5,000) and is therefore reported as compatible with, rather than different from, international practice.

Model diagnostics were unremarkable. Variance inflation factors were all below 1.25 (maximum 1.21), excluding collinearity. Adding a multiplicative interaction between lesion multiplicity and vitiligo classification did not improve fit (likelihood-ratio χ^2 = 0.000, df = 1; p = 0.996), so the main-effects model of Table 5 was retained. Re-entering age as a five-level ordinal term rather than a binary variable changed nothing of substance: the ordinal age term was null (odds ratio 1.03 per stratum; p = 0.879) while multiple lesions and nonsegmental disease retained their effects and discrimination was unchanged (area under the curve 0.860). Harrell bootstrap optimism correction over 500 replicates gave an apparent area under the curve of 0.859, optimism 0.020 and an optimism-corrected value of 0.839. Finally, the Poisson model for therapeutic complexity was underdispersed (Pearson dispersion 0.386); under a quasi-Poisson specification the association with lesion multiplicity strengthened (incidence rate ratio 1.363; 95% CI 1.158–1.604; p < 0.001), so the Poisson intervals reported in Table 5 are the conservative choice.

4. Discussion

In this five-year census of 175 patients with vitiligo at the Dermatology and Venereology Outpatient Clinic of Dr. Moewardi Regional General Hospital, Surakarta, the cohort was young, mildly female-predominant and clinically extensive: 74.29% had multiple lesions and 62.29% had nonsegmental disease, as detailed in Table 1. Four findings depart substantially from published reference values (Table 3, Figure 2B). The segmental fraction of 37.71% is more than six times the 5.93% reported in a Saudi tertiary cohort of 573 patients[1] and about twice the 19.1% reported at Bali Mandara General Hospital in Indonesia[10] (Cohen's h = 0.831 and 0.418 respectively). Phototherapy utilisation of 74.86% is nearly twenty times the 3.9% observed in a United States claims cohort of 19,335 patients[24] and more than twice the 29% recorded at Dr. Zainoel Abidin Hospital in Banda Aceh.[9] Topical corticosteroid use of 76.57% is more than double the 29.1% documented in a United Kingdom national cohort,[25] and only 0.57% of our patients went untreated against 27% to 32% in a 24,949-patient United States analysis.[26] Within the cohort, treatment allocation was internally coherent with guideline logic: as Table 5 and Figure 4A show, multiple lesions and nonsegmental disease independently predicted phototherapy receipt, a facial index site independently predicted its avoidance, and

the model discriminated well (area under the curve 0.859).

The mild female predominance of 57.71% is unremarkable. It is statistically indistinguishable from the 56.3% reported in a large United States claims cohort (p = 0.706),[26] and closely matches the 53% and 55.1% reported in Indonesian series from Banda Aceh and Bali respectively,[9,10] so it is best interpreted as differential health-seeking behaviour rather than differential biological susceptibility. In patients with skin of colour, quality-of-life impairment tracks disease extent and activity directly, which lowers the threshold for specialist consultation,[7] and the same relationship has been shown in Indonesian patients receiving phototherapy.[8] The age structure is more informative: as Table 1 and Figure 1A show, 34.86% of patients were younger than 18 years, against 11.8% in the United States comparator — an excess of +23.06 percentage points (95% CI +16.00 to +30.12; Cohen's h = 0.562; p < 0.001).[26] A similarly young distribution characterises the Indonesian narrowband ultraviolet B population in Yogyakarta, whose mean age was 25.48 ± 14.01 years,[11] and paediatric vitiligo series confirm that childhood-onset disease is a substantial share of tertiary caseload.[27] Age matters clinically because it constrains topical corticosteroid potency and duration, and because phototherapy adherence and cabinet tolerance differ between children and adults.[28,29]

The segmental fraction is the most striking finding and requires careful interpretation, because four explanations are plausible and they carry different consequences. The first is classification drift. International Vitiligo Task Force recommendations reserve the segmental label for unilateral, block-like or linear disease that does not cross the midline, and place focal or isolated mucosal lesions in an unclassified category.[2] In routine outpatient documentation without a mandatory structured classification field, focal and unclassified disease is readily recorded as segmental, which would inflate the segmental count at the expense of a category that did not exist in this record set. That the comparator Saudi cohort separately reported focal disease in 5.41% and undetermined disease in 6.28% of patients supports this mechanism directly.[1] The second explanation is the age structure: segmental vitiligo is over-represented in children and adolescents and stabilises earlier,[12,13] and this cohort contained a marked excess of patients under 18 years, so part of the segmental excess is expected on case-mix grounds alone. The third is referral filtering: a tertiary centre that operates one of the few regional phototherapy services receives a case-mix weighted towards disease that has resisted primary-care topical therapy, and unilateral segmental disease on a visible site is precisely the presentation that prompts referral in a young population. The fourth, and least likely, is a genuine population difference in phenotype distribution among Fitzpatrick III–V Javanese Indonesians, for which there is currently no supporting genetic or immunological evidence. Distinguishing these explanations is not academic: stable segmental disease is the phenotype with the best response to surgical repigmentation in meta-analysis,[23] whereas nonsegmental disease requires screening for autoimmune comorbidity, which meta-analysis links to thyroid disease, type 1 diabetes and alopecia areata,[16] and whose thyroid-associated subgroup has a distinguishable clinical profile.[17] Misclassification in either direction therefore produces the wrong treatment triage and the wrong comorbidity workup.

Lesion multiplicity in 74.29% of patients, and its strong association with nonsegmental disease (odds ratio 6.95; 95% CI 3.27–14.77; Table 4), is biologically coherent: nonsegmental disease is the disseminated autoimmune phenotype in which lesional CD8+ T-cell burden governs behaviour and treatment response,[14] with interferon-γ and granzyme B as the effector mechanism,[15] whereas segmental disease is a localised process that is immunologically distinct.[12] Facial predominance among documented index sites (31.43%) is, notably, statistically indistinguishable from the 32.9% facial involvement reported in a 24,949-patient United States analysis (p = 0.679; Table 3),[26] and is broadly consistent with the 20% head-and-neck predominance recorded in Banda Aceh and the 39.20% initial head-and-neck involvement of the Saudi cohort.[1,9] The concordance is reassuring, but it should be read with the caveat that the source charts documented a single principal site rather than the full set of involved regions — a direct consequence of the absence of a body-surface instrument such as the Vitiligo Area Scoring Index or the Vitiligo Extent Score,[21,22] and the single most correctable documentation deficiency identified by this audit.

Phototherapy utilisation of 74.86% is the second major deviation and is best understood as a service-configuration finding. International Vitiligo Task Force recommendations position narrowband ultraviolet B for extensive or progressive disease rather than for all comers,[2] and real-world utilisation in high-income systems is far lower than the guideline text implies: 3.9% in a United States claims cohort,[24] with phototherapy so poorly captured in United Kingdom national data that the authors explicitly cautioned against under-estimation.[25] Three features of this setting plausibly account for the difference. First, referral filtering: as one of the few regional centres with a functioning narrowband ultraviolet B cabinet, this clinic receives patients specifically for phototherapy, so its denominator is enriched for phototherapy candidates — exactly the pattern seen at Bali Mandara, where 85.9% of patients received phototherapy.[10] Second, case-mix: three quarters of the cohort had multiple lesions, the very indication for which phototherapy is recommended, and multiple lesions carried an adjusted odds ratio of 8.39 for phototherapy receipt (Table 5). Third, financing and availability: under the Indonesian national health insurance scheme, phototherapy delivered in a tertiary centre is accessible at low out-of-pocket cost, whereas topical calcineurin inhibitors and topical Janus kinase inhibitors are not routinely available or reimbursed, so the practical therapeutic menu is narrower than the guideline menu. Evidence that narrowband ultraviolet B stabilises

disease even where repigmentation is limited[20] and that Indonesian patients achieve measurable Vitiligo Area Scoring Index reduction with it — from 5.15 ± 8.80 to 4.06 ± 5.59 after 48 sessions in Yogyakarta —[11] supports a high-volume phototherapy programme provided cumulative dosimetry and course limits are documented. Prospective biomarker work further shows that the repigmentation response to narrowband ultraviolet B is tractable and measurable, tracking circulating CXCL10 and interleukin-18,[30] which supports monitoring outcome rather than exposure alone.

The near-absence of untreated patients (0.57%, against 27% to 32% in the United States[26] and 60.8% in the United Kingdom[25]) is a third distinctive feature and deserves emphasis, because it inverts the usual expectation that middle-income settings under-treat. Every patient but one received at least one modality, and the mean Therapeutic Complexity Index was 2.38 ± 1.00 modalities (Table 1). Whether this reflects genuinely better access once a patient reaches a tertiary centre, or a documentation convention in which any prescription is recorded, cannot be resolved retrospectively. The determinants of receipt identified here are nevertheless internally consistent and clinically interpretable: extent and phenotype drove phototherapy, while a facial index site independently predicted its avoidance (0.35; 95% CI 0.14–0.86; p = 0.023). That inverse association is not paradoxical, since facial disease is generally limited and responds well to site-appropriate topical therapy, for which randomised evidence exists,[18,19] and since in a Fitzpatrick III–V population facial skin is treated with particular caution because of the risk of perilesional hyperpigmentation and corticosteroid atrophy. That systemic therapy was confined entirely to patients with multiple lesions, and that therapeutic complexity rose with extent (Cohen's d = 0.83; incidence rate ratio 1.363; Table 5), indicates that this service escalates therapy along the axis that guidelines specify. The low methotrexate fraction (2.86%) is consistent with the thin real-life evidence base for that agent.[31]

Placing these results in mechanistic context clarifies what the phenotype distribution implies. Vitiligo is now understood as a disorder in which melanocyte-specific autoimmunity is the proximate effector: lesional CD8+ T-cell number predicts the outcome of melanocyte transplantation in patients,[14] and interferon-γ and granzyme B released by those cells mediate melanocyte destruction in nonsegmental disease.[15] The adjuvants recorded in this cohort map onto that model with varying evidential support: serum zinc has been shown to relate to disease severity and activity in Indonesian patients, which is the empirical basis for the 39.43% zinc use documented here,[32] while retinoids and emollients have no comparable local evidence base. Meanwhile the international therapeutic frontier has moved decisively: topical ruxolitinib achieved facial Vitiligo Area Scoring Index improvement of at least 75% in roughly 30% of patients at week 24 in two phase 3 trials,[33] oral ritlecitinib improved active nonsegmental disease in a phase 2b trial,[34] and topical tofacitinib now performs comparably to tacrolimus in localised disease.[19] The gap between that frontier and the therapeutic menu documented here — corticosteroids, phototherapy, zinc, retinoids and emollients — is the practical face of therapeutic inequity in a middle-income setting, and it strengthens rather than weakens the case for maintaining a high-quality phototherapy service.

For dermatologists practising at Dr. Moewardi Regional General Hospital and at comparable PERDOSKI-affiliated centres, four operational implications follow. First, classification should be recorded against explicit International Vitiligo Task Force criteria in a structured field, with Wood-lamp examination and dermoscopy used to distinguish segmental from focal and unclassified disease; the segmental fraction reported in Table 1 cannot be interpreted, or acted upon surgically, until that ambiguity is resolved.[2,23] Second, a quantitative extent instrument should be added to the routine vitiligo encounter, since the measurement properties of both the Vitiligo Area Scoring Index and the Vitiligo Extent Score are now established;[21,22] without them, extent can only be dichotomised as single versus multiple, which is too coarse either to grade severity or to measure treatment response, and quality of life cannot be linked to severity as Indonesian data already permit.[8] Third, given that three quarters of patients receive narrowband ultraviolet B (Table 2), cumulative dose, session count and course limits should be recorded prospectively, as they were in the Yogyakarta series that demonstrated measurable Vitiligo Area Scoring Index reduction.[11] Fourth, patients classified as nonsegmental — 62.29% of this cohort — should undergo structured screening for autoimmune comorbidity, particularly thyroid disease.[16,17]

The Indonesian context shapes both the findings and their interpretation. A Fitzpatrick III–V population living at an equatorial ultraviolet index of 10 to 12 experiences maximal visual contrast between depigmented macules and surrounding skin, which amplifies psychosocial burden and lowers the consultation threshold for visible lesions — consistent with the facial predominance observed here and with quality-of-life data from skin-of-colour and Indonesian populations.[4,7,8] Structural factors are equally relevant: phototherapy cabinets are concentrated in academic centres, national insurance coverage favours procedures delivered in those centres, topical calcineurin inhibitors and topical Janus kinase inhibitors are not routinely accessible, and traditional or herbal remedies are widely used before specialist presentation, often delaying referral. Together these factors provide a coherent, non-biological explanation for a phototherapy-intensive, corticosteroid-dominant practice pattern that would look anomalous if judged against high-income real-world benchmarks alone.

The quantitative bias analysis reported in Section 3.7 materially strengthens the interpretation of the segmental finding. Classification drift is the most credible alternative explanation, yet the sensitivity

analysis shows it cannot plausibly account for the result on its own: 75.8% of all segmental diagnoses would have to be reclassified as focal or unclassified disease before the cohort proportion became statistically indistinguishable from the international reference, and even complete-case attrition of 20% with a worst-case nonsegmental composition would leave the fraction above 30%. Two implications follow. Clinically, the excess is large enough to justify acting on it: a substantial number of patients in this service may be candidates for stability assessment and, if stable, for surgical repigmentation, which meta-analysis identifies as most effective in exactly this phenotype.[23] Methodologically, the residual uncertainty is a documentation problem with a documentation solution: prospective recording against explicit consensus criteria, with Wood-lamp and dermoscopic confirmation and an inter-rater reliability estimate, would settle the question within a single clinical year.[2]

This study has several strengths. It is, to our knowledge, the largest single-centre vitiligo series reported from Central Java and one of the few Indonesian dermatology datasets analysed with confidence intervals, effect sizes and multivariable adjustment rather than raw percentages. Total sampling of a complete five-year clinical census eliminates sampling bias and yields a genuine denominator. Every proportion is reported with a Wilson interval (Tables 1 and 2), every association with an effect size (Tables 4 and 6 to 8), and the multivariable models are pre-specified, entered simultaneously, formally calibrated and assessed for discrimination (Table 5, Figure 3). Finally, benchmarking cohort proportions against reference values drawn from named, denominator-reported published cohorts rather than from informal literature impressions (Table 3) converts a descriptive audit into a testable comparison and identifies four specific, actionable deviations.

Its limitations are equally clear and constrain interpretation. First, the design is retrospective and depends entirely on the completeness of routine clinical documentation; patients with incomplete records were excluded, and the number so excluded was not recorded, so a non-response bias of unknown magnitude cannot be excluded. Second, no validated vitiligo instrument was available: extent was reduced to a single versus multiple dichotomy, disease activity, Koebner phenomenon, family history and quality of life were not captured, and Fitzpatrick phototype was not individually documented, so the phototype distribution is inferred from population characteristics rather than measured. Third, the classification of segmental versus nonsegmental disease was extracted from clinical documentation rather than adjudicated against explicit consensus criteria by independent raters, and no inter-rater reliability statistic could therefore be computed; this is the principal threat to the validity of the headline segmental finding. Fourth, the cross-sectional structure means that treatment associations are patterns of allocation, not effects: phototherapy receipt reflects clinician judgement about severity, so the observed associations are confounded by indication and must not be read causally. Fifth, no outcome data — repigmentation, relapse, adherence or quality of life — were available, so the appropriateness of the observed prescribing cannot be judged against results. Sixth, the single-centre tertiary setting limits generalisability to the wider Indonesian vitiligo population, and the referral filter is the most likely explanation for the two headline deviations. Seventh, at n = 175 the study can detect odds ratios of approximately 3.1 or larger for the principal contrast, so moderate associations — including the adjusted association with adult age, which approached but did not reach significance — may have been missed.

Six further limitations deserve explicit statement. First, the reference proportions of Table 3 are drawn from cohorts that differ from ours in health system, case ascertainment and follow-up window — claims databases capture dispensed therapy over a fixed period, whereas our record review captures anything documented over five years — so the treatment comparisons in particular are susceptible to ascertainment differences as well as to genuine practice differences. Second, the segmental versus nonsegmental label was abstracted rather than adjudicated, and no inter-rater reliability coefficient could be calculated; because this label is the exposure underlying the headline finding, misclassification would bias the segmental proportion in either direction. Third, phototherapy was recorded as receipt of at least one session, with no session count, cumulative dose or completion status, so utilisation is a binary marker of access rather than a measure of treatment intensity; for the same reason the neutral term "receipt" rather than "escalation" is used throughout, since treatment sequence and time to phototherapy were not recoverable and topical failure preceding phototherapy cannot be assumed. Fourth, discrimination was estimated in the same data used for model fitting; optimism correction reduced the area under the curve from 0.859 to 0.839, and the model should be regarded as descriptive of allocation within this service rather than as a validated prediction tool. Fifth, annual case counts were not extracted, so the effect of the COVID-19 pandemic on a phototherapy-dependent service during 2020 and 2021 — when many regional phototherapy units suspended or restricted operation — cannot be assessed, and the observed utilisation may understate steady-state practice. Sixth, the documented arithmetic in which topical and oral corticosteroid counts sum exactly to the combined total (Table 2) indicates that route combinations were not captured, which limits inference about stepwise therapy.

The research agenda that follows is specific. A prospective multicentre PERDOSKI registry recording consensus classification, a quantitative extent instrument, disease activity, Fitzpatrick phototype, quality of life and repigmentation outcome would convert the hypotheses generated here into estimates with external validity, and would establish whether the segmental excess is a documentation artefact, a referral artefact or a real feature of Indonesian vitiligo. Linking such a registry to phototherapy dosimetry would allow

the effectiveness of a phototherapy-intensive model of care to be measured directly rather than inferred, which matters because the international standard of care is moving towards targeted topical and systemic therapy that is not yet accessible in this setting.[19,33,34] Finally, autoimmune comorbidity screening yields in an Indonesian nonsegmental cohort are unknown and should be measured, given the meta-analytic association between vitiligo and thyroid autoimmunity and the distinct profile of the thyroid-associated subgroup.[16,17]

5. Conclusion

Vitiligo at this Indonesian tertiary dermatology centre affects a young, mildly female-predominant population with extensive disease: 74.29% had multiple lesions, 62.29% had nonsegmental disease and the face was the commonest index site (31.43%), a figure indistinguishable from international reports. Four features depart sharply from published reference values: a segmental fraction of 37.71% (95% CI 30.87–45.09), exceeding an international tertiary-cohort reference by 31.78 percentage points (Cohen's h = 0.831; $p < 0.001$); phototherapy utilisation of 74.86% (95% CI 67.94–80.70), exceeding United States real-world utilisation by 70.96 points (h = 1.694); topical corticosteroid use exceeding a United Kingdom national reference by 47.47 points; and an untreated fraction of only 0.57% against 27% internationally. Phototherapy receipt was independently driven by multiple lesions (adjusted odds ratio 8.39; 95% CI 3.41–20.64) and nonsegmental disease, with good discrimination (area under the curve 0.859). Indonesian dermatology services, and PERDOSKI guidance, should mandate consensus-based classification, quantitative extent scoring and phototherapy dosimetry documentation; prospective multicentre studies with measured repigmentation outcomes are required.

Declarations

Ethical Approval. This study was approved by the Health Research Ethics Committee, Dr. Moewardi Regional General Hospital, Surakarta, Indonesia (approval number 2.155/X/HREC/2025). The study was conducted in accordance with the principles of the Declaration of Helsinki. The requirement for written informed consent was waived because of the retrospective design and the use of anonymised medical record data.

Conflict of Interest. The authors declare no conflict of interest.

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Data Availability. The data supporting the findings of this study are available from the corresponding author upon reasonable request and with permission from Dr. Moewardi Regional General Hospital. The data are not publicly available because they contain confidential patient information.

Use of Artificial Intelligence. All analyses were verified by the authors against the source medical-record data, and the authors reviewed and approved all content and take full responsibility for the integrity and accuracy of the work.

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