

Clinical features of 109 patients with primary cicatricial alopecia: a 15-year retrospective study

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Abstract

Introduction: Given the limited data on primary cicatricial alopecias (PCAs), which cause permanent hair loss, this study investigates the demographic and clinical characteristics, applied treatments, and disease course of PCA patients.

Methods: Medical records of 109 PCA patients that attended an outpatient clinic between 2010 and 2025 were retrospectively reviewed. Demographic and clinical characteristics, and treatment approaches were retrieved from patient files.

Results: Lymphocytic PCA (83.5%) was the most frequent type, followed by neutrophilic PCA (15.6%). Classic lichen planopilaris (LPP-C) was the leading lymphocytic subtype (39.4%), and folliculitis decalvans (12%) was the most common neutrophilic PCA. Lymphocytic PCAs showed female predominance, whereas male predominance was present in neutrophilic PCAs ($p < 0.001$). Disease onset occurred at an older age in lymphocytic PCAs compared to the neutrophilic group (42.0 ± 11.8 and 30.2 ± 11.4 years, $p < 0.001$). Pain and nodule/pustule formation were more frequent in neutrophilic PCAs ($p < 0.001$). Topical/intralesional corticosteroids and hydroxychloroquine were mainly used in lymphocytic PCAs, whereas oral antibiotics and isotretinoin were preferred in neutrophilic PCAs.

Conclusions: PCA frequency may vary geographically. LPP-C was the most common subtype. Early diagnosis and timely treatment are essential to prevent irreversible hair loss and optimize cosmetic outcomes.

Keywords: alopecia, cicatricial alopecia, hair loss, lichen planopilaris, scarring alopecia

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Introduction

Cicatricial alopecias (CAs) are relatively rare disorders characterized by irreversible destruction of hair follicles (HFs) and permanent hair loss (1). Clinically, loss of visible follicular ostia within alopecic patches is observed, and epidermal atrophy or other textural skin changes may be present. Histologically, damaged HFs replaced with fibrous tissues are present (1).

Cicatricial alopecias may be classified as primary, in which the HF itself is the direct target of inflammation, or secondary, occurring due to incidental follicular damage from unrelated inflammatory processes such as infections, sarcoidosis, burns, radiation, or malignancies (1, 2). Although irreversible destruction of the stem cells of epithelial HFs is thought to play a role in primary CA (PCA), its exact etiopathogenesis remains unknown (1). PCA

is further histologically classified according to the predominant inflammatory cell type into lymphocytic, neutrophilic, mixed, or nonspecific forms. Lymphocytic types include chronic cutaneous lupus erythematosus (CCLE), lichen planopilaris (LPP-C: classic type; FFA: frontal fibrosing alopecia; Graham-Little syndrome), pseudopelade of Brocq (PPB), central centrifugal cicatricial alopecia (CCCA), keratosis follicularis spinulosa decalvans (KFSD), and alopecia mucinosa. Neutrophilic types include folliculitis decalvans (FD) and dissecting cellulitis (DC), while mixed forms include acne keloidalis, acne necrotica, and erosive pustular dermatosis (Fig. 1) (3).

Although the exact prevalence is unknown, the frequency of PCA among patients presenting with hair loss has been reported to range between 2.1% and 7.3% (4–6). Özkesici Kurt et al. reported that PCA accounted for 6% of all hair diseases (7). Given the

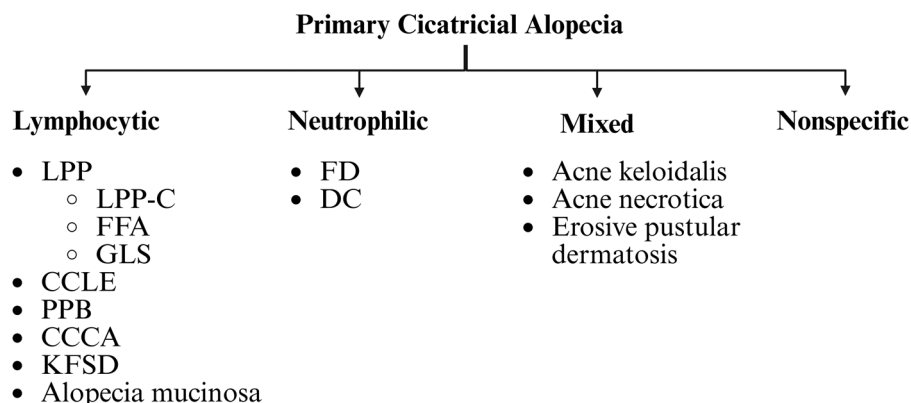


Figure 1 | Histopathologic classification of primary cicatricial alopecia by predominant inflammatory infiltrate.

CCCA = central centrifugal cicatricial alopecia, CCLE = chronic cutaneous lupus erythematosus, DC = dissecting cellulitis, FD = folliculitis decalvans, FFA = frontal fibrosing alopecia, GLS = Graham-Little syndrome, KFSD = keratosis follicularis spinulosa decalvans, LPP = lichen planopilaris, LPP-C = classic lichen planopilaris, PPB = pseudopelade of Brocq.

large sample sizes, and the challenges in both diagnosis and management, identifying the demographic and clinical characteristics of patients is essential to better understand the disease. Therefore, we aimed to investigate the demographic and clinical features, applied treatment modalities, and disease course of patients with PCA.

Methods

Patients

Medical records of patients with histopathologically confirmed PCA that were admitted to our outpatient clinic between 2010 and 2025 were retrospectively evaluated. Patients with missing clinical data, uncertain diagnosis, or lacking histopathological confirmation were excluded.

Histopathological diagnosis and subtype classification were retrieved from scalp biopsy reports and categorized according to the North American Hair Research Society (NAHRS) working classification (3). Subtypes were primarily distinguished by the predominant inflammatory infiltrate (lymphocytic, neutrophilic, mixed, or nonspecific) together with supportive histopathologic features, including but not limited to the pattern and level of perifollicular inflammation, perifollicular fibrosis, evidence of follicular destruction, dermal changes (e.g., fibrosis and hyalinization), and changes in adnexal structures / sebaceous glands. Nonspecific PCA was defined as advanced idiopathic scarring alopecia with minimal residual inflammation and inconclusive clinical and histopathologic findings, which may represent the end stage of various inflammatory CAs (1–3).

Demographic variables (age at disease onset and sex) and clinical data (time until diagnosis; diagnostic subtype; lesion location; symptoms such as pruritus, burning sensation, or pain; trichoscopic findings such as perifollicular or interfollicular erythema, and perifollicular scaling; clinical signs such as atrophy, or nodule or pustule formation; non-scalp skin findings related to the diagnosis; accompanying comorbidities; treatment modalities, follow-up duration; and disease course) were retrieved from medical records.

Ethical approval

We obtained ethical approval from the local ethics committee (decision no. 2025.34.02.12, date: February 25th, 2025).

Statistical analysis

Categorical variables were presented as n (%). The Shapiro–Wilk test was used to assess the normality of continuous variables. Continuous variables were reported as the mean \pm standard deviation or median (interquartile range, IQR) depending on the normality. An independent samples t -test or the Mann–Whitney U test was used to compare normally distributed variables (age at disease onset) or non-normally distributed variables (time until diagnosis and follow-up duration) between lymphocytic and neutrophilic groups of PCA, respectively. Categorical variables were compared with the Pearson chi-square or Fisher–Freeman–Halton exact test, as appropriate. Inferential analyses were restricted to the lymphocytic versus neutrophilic PCA groups; subtype-level results were reported descriptively due to small numbers.

SPSS v.25 software (IBM Corp. Armonk, NY, USA) was used for

statistical analysis. A p -value < 0.05 was considered statistically significant.

Results

A total of 109 patients with clinically and histopathologically confirmed PCA were included in the study. The female-to-male ratio was 2.4:1, and the mean age at diagnosis was 40.3 ± 12.5 years. The most frequent PCA type was lymphocytic, observed in 91 (83.5%) patients, followed by neutrophilic PCA in 17 (15.6%) patients.

Among lymphocytic PCAs, LPP-C (39.4%), discoid lupus erythematosus (DLE) (24%), and FFA (13%) were the leading subtypes. On the other hand, FD was the most frequent neutrophilic PCA, observed in 13 (12%) patients. The mixed PCA category was represented only by acne keloidalis ($n = 1$), and no cases of acne necrotica or erosive pustular dermatosis were observed. The demographic and clinical features of the patients are summarized in Table 1.

Demographic and clinical features of PCA subtypes

The demographic and clinical characteristics of lymphocytic and neutrophilic PCAs, as well as PCA subtypes, are presented in Tables 2 and 3. A female predominance was observed in lymphocytic PCA, including FFA, DLE, and LPP-C, whereas a significant male predominance was present in neutrophilic PCA, especially in FD ($p < 0.001$). All patients with FFA were female. The age of disease onset was significantly lower in neutrophilic PCA than in lymphocytic PCA ($p < 0.001$).

Most patients had multifocal alopecia lesions across all subtypes. The vertex was the most commonly involved region in LPP-C (72.1%) and DLE (73.1%). All patients with FFA had frontal region involvement, and eyebrow involvement was observed in six of 14 patients with FFA (42.9%). In neutrophilic PCAs, the vertex and occipital regions were the most commonly affected sites in FD (84.6% and 61.5%, respectively) and in DC (50% and 75%, respectively).

Symptomatic presentation was significantly more common in the neutrophilic group than in the lymphocytic group (16/17 [94.1%] vs. 29/91 [31.9%], $p < 0.001$). Among lymphocytic subtypes, pruritus was the most common symptom, especially in LPP-C (44.2%) and DLE (26.9%). On the other hand, pain in the lesional areas was the leading symptom in neutrophilic PCAs (84.6% in FD and 75% in DC; Table 3). Although the frequencies of perifollicular or interfollicular erythema were similar between lymphocytic and neutrophilic groups, these findings were less observed in the patients with FFA (35.7%) and PPB (33.3%) than in other subtypes. Conversely, atrophy was more commonly seen in DLE (61.5%). Nodule or pustule formation was the predominant clinical sign in neutrophilic PCAs ($p < 0.001$).

A total of 24 (26.4%) patients in the lymphocytic group and six (35.3%) patients in the neutrophilic group had accompanying comorbidities ($p = 0.556$). The details of the comorbidities are summarized in Table 4.

Treatment methods applied in PCA subtypes

The previous treatments, treatment methods applied, and disease courses are presented in Table 5. A total of 39 (35.8%) patients had received at least one treatment before being admitted to our clinic. Topical corticosteroids (TCSs) were the most commonly

used treatment in LPP-C, PPB, and DC. In addition, intralesional corticosteroids (ILCS) or hydroxychloroquine (HCQ) had previ-

Table 1 | Patient demographics and clinical characteristics.

Variables	Values
Sex, <i>n</i> (%)	
Female	77 (70.6)
Male	32 (29.4)
Age at disease onset, years, mean \pm <i>SD</i>	40.3 \pm 12.5
Time until diagnosis, years, median (IQR)	1 (0.3–2.0)
Type of primary cicatricial alopecia, <i>n</i> (%)	
Lymphocytic	91 (83.5)
Neutrophilic	17 (15.6)
Mixed	1 (0.9)
Subtypes, <i>n</i> (%)	
Lichen planopilaris	
Classic type	43 (39.4)
Frontal fibrosing alopecia	14 (12.8)
Discoid lupus erythematosus	26 (23.9)
Pseudopelade of Brocq	6 (5.5)
Central centrifugal cicatricial alopecia	1 (0.9)
Keratitis follicularis spinulosa decalvans	1 (0.9)
Folliculitis decalvans	13 (11.9)
Dissecting cellulitis	4 (3.7)
Acne keloidalis	1 (0.9)
Involvement, <i>n</i> (%)	
Unifocal	35 (32.1)
Multifocal	74 (67.9)
Alopecia locations, <i>n</i> (%)	
Vertex	62 (56.9)
Parietal	30 (27.5)
Occipital	25 (22.9)
Frontal	23 (21.1)
Temporal	23 (21.1)
Eyebrow	9 (8.3)
Symptoms, <i>n</i> (%)	
Asymptomatic	63 (57.8)
Pruritus	29 (26.6)
Burning sensation	1 (0.9)
Pain	16 (14.7)
Signs, <i>n</i> (%)	
Peri/interfollicular erythema	83 (76.1)
Perifollicular scaling	73 (67.0)
Atrophy	28 (25.7)
Nodule/pustule	22 (20.2)
Previously treated patients,* <i>n</i> (%)	39 (35.8)
Topical corticosteroid	18/39
Intralesional corticosteroid	4/39
Minoxidil	4/39
Oral corticosteroid	3/39
Hydroxychloroquine	8/39
Systemic isotretinoin	5/39
Acitretin	2/39
Systemic antibiotics	4/39
Methotrexate	1/39
Treatments, <i>n</i> (%)	
Topical corticosteroid	83 (76.1)
Topical calcineurin inhibitor	14 (12.8)
Intralesional corticosteroid	46 (42.2)
Minoxidil	7 (6.4)
Finasteride	2 (1.8)
Oral corticosteroid	17 (15.6)
Hydroxychloroquine	68 (62.4)
Systemic isotretinoin	13 (11.9)
Acitretin	12 (11.0)
Systemic antibiotics	13 (11.9)
Methotrexate	13 (11.9)
Dapsone	1 (0.9)
Follow-up, months, median (IQR)	13 (6–29)
Relapse, <i>n</i> (%)	23 (21.1)

*Patients that received ≥ 1 treatment prior to presentation at our clinic. IQR = interquartile range, *SD* = standard deviation.

ously been prescribed for DLE patients. On the other hand, ILCS, oral antibiotics, or oral isotretinoin were the most commonly prescribed treatments for FD patients.

At our clinic, combination therapy was used in most patients with LPP-C (72.1%), FFA (78.6%), DLE (88.5%), and FD (69.2%). TCS, ILCS, and HCQ were the most frequently used treatment methods in LPP-C, FFA, DLE, and PPB. Minoxidil and oral finasteride were prescribed to 28.6% and 14.3% of patients with FFA, respectively. On the other hand, oral antibiotics and oral isotretinoin were the most commonly used systemic treatments in neutrophilic PCA (Table 5).

Remission was achieved in six (23.1%) patients with DLE, four (9.3%) with LPP-C, one (7.1%) with FFA, and one (16.7%) with PPB. In DLE, remission was observed after treatment with HCQ ($n = 2$), combination of HCQ and TCS ($n = 1$), combination of HCQ, TCS, and topical calcineurin inhibitor (TCI) ($n = 1$), combination of HCQ, TCS, ILCS, and oral corticosteroid ($n = 1$), and methotrexate ($n = 1$). In LPP-C, remission occurred after combined HCQ, TCS, and ILCS ($n = 2$), combined HCQ and TCS ($n = 1$), and combined methotrexate and TCI ($n = 1$). One patient with FFA achieved remission following combined HCQ, TCS, and ILCS. Moreover, one patient with PPB experienced remission with ILCS monotherapy. However, recurrences occurred, especially in neutrophilic PCA (Table 5).

Discussion

Primary CA is an important group of scarring hair disorders that leads to permanent hair loss. Therefore, accurate diagnosis and rapid management are crucial (1). In our study, lymphocytic PCA was more common than the other types. The leading subtypes were LPP-C, DLE, FFA, and FD. The overall female-to-male ratio was 2.4:1. A female predominance was present in lymphocytic subtypes, whereas males were significantly more frequent in FD.

Demographics and clinical features of the PCA

The frequency of PCA subtypes varies across studies from different geographical regions (Table 6). Lymphocytic subtypes are more common in most studies (5–12). In a study from the United States including both primary and secondary CA, PCA constituted the majority of cases, and the three most common diagnoses were PPB, LPP-C, and FD. However, the authors classified all nonspecific CAs as pseudopelade (5). In studies from Canada, Brazil, and Iran, DLE was reported as the most frequent PCA subtype (6, 9, 11). However, in another study from Iran, Nejad et al. reported that FD was the most common subtype (10). Similarly, Qi et al. stated that the most frequent PCA was FD, and neutrophilic PCAs were more common than lymphocytic PCAs among 59 Chinese patients (4). Moreover, in a study from Taiwan, Su et al. found that the total ratio of neutrophilic and mixed types was higher than that of lymphocytic subtypes, with DC being the most common subtype (12). On the other hand, Özkesici Kurt et al. found that the most frequent subtypes were LPP-C, FFA, and DLE in their study conducted at a tertiary hair disease clinic in Turkey (7). Similarly, in our study, the leading three diagnoses were LPP-C, DLE, and FFA. These differences may reflect a combination of population- and study-related factors (7, 13). Population-specific genetic predisposition and sociodemographic/ethnic differences, including variations in hair texture, hair-care practices, and environmental exposures, may influence susceptibility to certain subtypes.

Morerelative rarity of PCA, the limited number of controlled trials with large sample sizes, and the challenges in both diagnosis and management, identifying the demographic and clinical characteristics of patients is essential to better understand the disease. Therefore, we aimed to investigate the demographic and clinical features, applied treatment modalities, and disease course of patients with PCA.

The age of disease onset varies among previous studies. Usually, patients with neutrophilic PCA are the youngest, whereas the oldest group differs across studies (4, 5, 7, 9, 12). Whiting et al. stated that patients with acne necrotica were the oldest (5). However, patients with FFA, PPB, or LPP were reported as the oldest groups in the other studies (4, 7, 9, 12). In our study, the age at onset was lowest in neutrophilic types, except KFSD, and highest in acne keloidalis, followed by FFA. Among women with FFA, six were postmenopausal, two were perimenopausal, and six were of reproductive age. Although FFA is usually reported in postmenopausal women, it may also occur in younger patients (14, 15).

Prompt diagnosis and management of PCA are important to prevent further scarring and irreversible HF damage. Nevertheless, diagnostic delays may occur due to difficulties in establishing patients' diagnosis, or due to misdiagnosis or late presentation by patients. Özkesici Kurt et al. reported that 1 to 5 years elapsed between the onset of symptoms and admission to a hair disease clinic (7). In our patients, the median time between the onset of alopecia and the confirmed diagnosis was 1 year. Although this median delay may appear modest, it may still be clinically meaningful because ongoing follicular inflammation can lead to permanent scarring and hair loss. Misdiagnosis, presentation to clinics other than dermatology or specialized hair disease units, or sociocultural factors may contribute to diagnostic delays. The exact reasons should be investigated in further studies. Increasing awareness of early clinical and trichoscopic clues, encour-

aging timely referral to dermatology / hair-disorder clinics, and earlier consideration of scalp biopsy in suspected cases may help shorten the diagnostic pathway and facilitate prompt initiation of appropriate treatment to minimize further follicular damage.

In previous studies, as in ours, women were generally affected by PCA more frequently than men. However, this distribution is likely related to the ratio of lymphocytic to neutrophilic types because lymphocytic PCAs are reported to be more common in women, whereas neutrophilic or mixed CAs are more frequent in men (4-7, 9-12).

In addition to alopecic patches, patients with PCA may present with pruritus, tenderness, burning sensation, pain, or pustular/nodular lesions (1, 16, 17). Although most of our patients were asymptomatic, itching was the most frequent symptom in those with LPP-C and DLE. On the other hand, pain in the lesional area was the predominant symptom in FD, DC, and acne keloidalis. In addition, nodule or pustule formation was significantly more common in neutrophilic PCA, consistent with the literature (1, 6, 7, 12).

Non-scalp skin lesions may assist in the diagnosis of LPP or CCLE (1). Lichen planopilaris may occur alone or in association with other forms of lichen planus (1, 18). It was reported that 17% to 28% of LPP patients may have other forms of lichen planus, although the incidence of co-occurrence varies among studies (6, 18). Similarly, patients with CCLE may have disease-associated lesions on the other body sites. Tan et al. reported concomitant involvement of additional body areas in 29% of patients with CCLE (6). We observed non-scalp mucocutaneous lichen planus lesions in 9.3% of the patients with LPP-C. In addition, 30.8% of DLE patients had non-scalp disease-associated skin lesions. None of the patients with DLE developed systemic lupus during the follow-up period.

An association between LPP and thyroid disease, particularly hypothyroidism, was previously reported (19-21). In our study,

Table 2 | Demographics and clinical characteristics of lymphocytic and neutrophilic primary cicatricial alopecia.

Variables	Lymphocytic (n = 91)	Neutrophilic (n = 17)	p-value
Sex, F/M	73/18	4/13	< 0.001
Age at disease onset, years, mean ± SD	42 ± 11.8	30.2 ± 11.4	< 0.001
Time until diagnosis, years, median (IQR)	1 (0.3-2)	1 (0.4-1.8)	0.757
Involvement, n (%)			
Unifocal	32 (35.2)	4 (23.5)	0.422
Multifocal	59 (64.8)	13 (76.5)	
Alopecia locations, n (%)			0.402
Vertex	51 (56)	11 (64.7)	0.543
Parietal	27 (29.7)	3 (17.7)	< 0.001
Occipital	14 (15.4)	10 (58.8)	0.019
Frontal	23 (25.3)	0 (0)	0.509
Temporal	21 (23.1)	2 (11.8)	0.347
Eyebrow	9 (9.9)	0 (0)	
Symptoms, n (%)			< 0.001
Asymptomatic	62 (68.1)	1 (5.9)	
Symptomatic	29 (31.9)	16 (94.1)	0.744
Signs, n (%)			0.084
Perifollicular/interfollicular erythema	70 (76.9)	13 (76.5)	0.005
Perifollicular desquamation	65 (71.4)	9 (53.0)	< 0.001
Atrophy	27 (29.7)	0 (0)	0.556
Nodule/pustule	4 (4.4)	17 (100.0)	
Comorbidities, n (%)	24 (26.4)	6 (35.3)	0.385
Treatment, n (%)			0.552
Monotherapy	25 (27.5)	7 (41.2)	0.008
Combined therapy	66 (72.5)	10 (58.8)	
Follow-up, months, median (IQR)	13 (6-31)	15 (4-26)	
Recurrence, n (%)	16/55 (29.1)	7/9 (77.8)	

F = female, M = male, SD = standard deviation, IQR = interquartile range. Recurrence data are presented as n/N (%), with N indicating the number of patients with available recurrence data.

Table 3 | Demographics and clinical characteristics of the primary cicatricial alopecia subtypes.

Variables	Lymphocytic (n = 91)						Neutrophilic (n = 17)			Mixed (n = 1)
	LPP-C (n = 43)	LPP-FFA (n = 14)	DLE (n = 26)	PPB (n = 6)	CCCA (n = 1)	KFSD (n = 1)	FD (n = 13)	DC (n = 4)	AK (n = 1)	
Sex (F/M)	32/11	14/0	24/2	2/4	1/0	0/1	2/11	2/2	0/1	
Age at onset, years, mean ± SD	41.6 ± 12.6	44.8 ± 11.6	39.3 ± 9.3	40.2 ± 10.5	56	15	30.5 ± 13.5	30.5 ± 12.3	57	
Years to diagnosis, median (IQR)	1 (0.2–2)	1 (0.2–2)	0.8 (0.2–2.8)	2 (0.5–3)	0	5	1 (0.3–1)	1.8 (0.5–6.5)	2	
Involvement, n (%)										
Unifocal	15 (34.9)	6 (42.9)	10 (38.5)	0 (0)	1 (100.0)	0 (0)	3 (23.1)	1 (25.0)	0 (0)	
Multifocal	28 (65.1)	8 (57.1)	16 (61.5)	6 (100.0)	0 (0)	1 (100.0)	10 (76.9)	3 (75.0)	1 (100.0)	
Alopecia locations, n (%)										
Vertex	31 (72.1)	1 (7.1)	19 (73.1)	5 (83.3)	0 (0)	1 (100.0)	11 (84.6)	2 (50.0)	0 (0)	
Parietal	11 (25.6)	0 (0)	10 (38.5)	6 (100.0)	1 (100.0)	1 (100.0)	4 (30.8)	0 (0)	0 (0)	
Occipital	10 (23.3)	0 (0)	4 (15.4)	0 (0)	0 (0)	1 (100.0)	8 (61.5)	3 (75.0)	1 (100.0)	
Frontal	7 (16.3)	14 (100.0)	2 (7.7)	0 (0)	0 (0)	1 (100.0)	0 (0)	0 (0)	0 (0)	
Temporal	8 (18.6)	6 (42.9)	7 (26.9)	1 (16.7)	0 (0)	0 (0)	2 (15.4)	0 (0)	0 (0)	
Eyebrow	1 (2.3)	6 (42.9)	1 (3.9)	0 (0)	0 (0)	1 (100.0)	0 (0)	0 (0)	0 (0)	
Symptoms, n (%)										
Asymptomatic	24 (55.8)	14 (100.0)	17 (65.4)	5 (83.3)	1 (100.0)	1 (100.0)	1 (7.7)	0 (0)	0 (0)	
Pruritus	19 (44.2)	0 (0)	7 (26.9)	1 (16.7)	0 (0)	0 (0)	1 (7.7)	1 (25.0)	0 (0)	
Burning	0 (0)	0 (0)	1 (3.9)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	
Pain	0 (0)	0 (0)	1 (3.9)	0 (0)	0 (0)	0 (0)	11 (84.6)	3 (75.0)	1 (100.0)	
Signs, n (%)										
PFE/IFE	40 (93.0)	5 (35.7)	25 (96.2)	2 (33.3)	0 (0)	0 (0)	10 (76.9)	3 (75.0)	1 (100.0)	
PFS	37 (86.1)	4 (28.6)	24 (92.3)	1 (16.7)	0 (0)	1 (100.0)	8 (61.5)	1 (25.0)	0 (0)	
Atrophy	7 (16.3)	3 (21.4)	16 (61.5)	2 (33.3)	0 (0)	1 (100.0)	0 (0)	0 (0)	0 (0)	
Nodule/pustule	4 (9.3)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	13 (100.0)	4 (100.0)	1 (100.0)	
Non-scalp skin findings, n (%)	4 (9.3)	0 (0)	8 (30.8)	0 (0)	0 (0)	1 (100.0)	0 (0)	0 (0)	0 (0)	
Comorbidities, n (%)	13 (30.2)	3 (21.4)	6 (23.1)	1 (16.7)	0 (0)	0 (0)	6 (46.2)	0 (0)	0 (0)	

AK = acne keloidalis, CCCA = central centrifugal cicatricial alopecia, DC = dissecting cellulitis, DLE = discoid lupus erythematosus, F = female, FD = folliculitis decalvans, IQR = interquartile range, KFSD = keratosis follicularis spinulosa decalvans, LPP-C = classic lichen planopilaris, LPP-FFA = lichen planopilaris–frontal fibrosing alopecia, M = male, PFE = perifollicular erythema, PFS = perifollicular desquamation, PPB = pseudopelade of Brocq, SD = standard deviation.

Table 4 | Accompanying comorbidities.

PCA subtypes	Comorbidities (n)
LPP-C (n = 13)	Hypothyroidism (n = 4), HBV-inactive carrier (n = 2), RAS (n = 2), Hashimoto thyroiditis (n = 1), hypertension (n = 1), hyperlipidemia (n = 1), DM (n = 1), CAD (n = 1), psoriasis (n = 1)
FFA (n = 4)	Hashimoto thyroiditis (n = 2), hyperlipidemia (n = 1), breast cancer (n = 1)
DLE (n = 6)	Epilepsy (n = 2), IDA (n = 2), breast cancer (n = 2), hypertension (n = 2), hypothyroidism (n = 1), DM (n = 1), CVD (n = 1), gout (n = 1)
PPB (n = 1)	Hypertension (n = 1), DM (n = 1), asthma (n = 1)
FD (n = 6)	Epilepsy (n = 1), HBV-inactive carrier (n = 1), type 1 DM (n = 1), vitiligo (n = 1), HS (n = 1), Sjögren's disease (n = 1)

DLE = discoid lupus erythematosus, FD = folliculitis decalvans, FFA = frontal fibrosing alopecia, HS = hidradenitis suppurativa, LPP-C = classic lichen planopilaris, PCA = primary cicatricial alopecia, PPB = pseudopelade of Brocq, HBV = hepatitis B virus, DM = diabetes mellitus.

Table 5 | Previous and current treatments, follow-up, and disease courses of primary cicatricial alopecia subtypes.

Variables	Lymphocytic (n = 91)						Neutrophilic (n = 17)				Mixed (n = 1)
	LPP-C (n = 43)	LPP-FFA (n = 14)	DLE (n = 26)	PPB (n = 6)	CCCA (n = 1)	KFSD (n = 1)	FD (n = 13)	DC (n = 4)	AK (n = 1)		
Previous treatments,* n (%)	9 (20.9)	4 (28.6)	11 (42.3)	3 (50.0)	0 (0)	1 (100.0)	8 (61.5)	2 (50.0)	1 (100)		
Topical corticosteroid	7/9	2/4	0	2/3	0	1	2/8	2/2	0		
Topical calcineurin inhibitor	1/9	1/4	0	0	0	0	0	0	0		
Minoxidil	1/9	3/4	0	0	0	0	0	0	0		
Intralesional corticosteroid	1/9	1/4	5/11	1/3	0	0	4/8	0	0		
Hydroxychloroquine	1/9	0	7/11	0	0	0	0	0	0		
Oral corticosteroid	1/9	1/4	0	0	0	0	1/8	0	0		
Methotrexate	1/9	0	0	0	0	0	0	0	0		
Oral antibiotics	1/9	0	0	0	0	0	3/8	1/2	1		
Oral isotretinoin	1/9	0	0	0	0	0	4/8	0	0		
Acitretin	1/9	0	0	0	0	0	1/8	0	0		
Treatment, n (%)	40 (93.0)	10 (71.4)	23 (88.5)	4 (66.7)	1 (100.0)	1 (100.0)	7 (53.9)	1 (25.0)	0 (0)		
Topical corticosteroid	3 (7.0)	3 (21.4)	7 (26.9)	0 (0)	0 (0)	0 (0)	2 (15.4)	0 (0)	0 (0)		
Topical calcineurin inhibitor	2 (4.7)	4 (28.6)	1 (3.9)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		
Minoxidil	24 (55.8)	10 (71.4)	9 (34.6)	1 (16.7)	1 (100.0)	0 (0)	3 (23.1)	1 (25.0)	0 (0)		
Intralesional corticosteroid	31 (72.1)	12 (85.7)	24 (92.3)	2 (33.3)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		
Hydroxychloroquine	9 (20.9)	2 (14.3)	5 (19.2)	0 (0)	0 (0)	0 (0)	1 (7.7)	0 (0)	0 (0)		
Oral corticosteroid	7 (16.3)	1 (7.1)	5 (19.2)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		
Methotrexate	3 (7.0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	7 (53.9)	4 (100.0)	0 (0)		
Oral antibiotics	2 (4.7)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	7 (53.9)	3 (75.0)	0 (0)		
Oral isotretinoin	7 (16.3)	0 (0)	1 (3.9)	0 (0)	0 (0)	0 (0)	2 (15.4)	1 (25.0)	1 (100.0)		
Acitretin	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (7.7)	0 (0)	0 (0)		
Dapsone	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		
Oral finasteride	0 (0)	2 (14.3)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		
Monotherapy, n (%)	12 (27.9)	3 (21.4)	3 (11.5)	5 (83.3)	1 (100.0)	1 (100.0)	4 (30.8)	3 (75.0)	1 (100.0)		
Combined therapy, n (%)	31 (72.1)	11 (78.6)	23 (88.5)	1 (16.7)	0 (0)	0 (0)	9 (69.2)	1 (25.0)	0 (0)		
Follow-up, months, median (IQR)	13.5 (7.5-31)	10 (6-16)	23.5 (8-72)	6 (4.5-6.5)	6	3	13 (4-25)	22 (13-24)	5		
Remission, n (%)	4 (9.3)	1 (7.1)	6 (23.1)	1 (16.7)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		
Without treatment	2/4	1	3/6	1	0	0	0	0	0		
Under treatment	2/4	0	3/6	0	0	0	5/7 (71.4)	2/2 (100)	1 (100.0)		
Recurrence, n (%)	7/26 (26.9)	2/8 (25.0)	7/18 (38.9)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		

*Treatments received prior to presentation to our clinic. Recurrence data are presented as n/N (%), with N indicating the number of patients with available recurrence data.
 AK = acne keloidalis, CCCA = central centrifugal cicatricial alopecia, DC = dissecting cellulitis, DLE = discoid lupus erythematosus, FD = folliculitis decalvans, IQR = interquartile range, KFSD = keratosis follicularis spinulosa decalvans, LPP-C = lichen planopilaris classic type, LPP-FFA = lichen planopilaris-frontal fibrosing alopecia, PPB = pseudoplaque of Brocq.

Table 6 | Previous studies on PCA from different countries compared with the current study.

Study	Year	Country	Sample size	L/N primary CA ratio	F/M ratio	Mean age of onset or age range	PCA subtypes (%)							Histopathologic confirmation, n/N (%)		
							LPP-C	FFA	DLE	PPB	CCCA	KFSD	FD		DC	AK
Whiting (5)	2001	United States	358	5.7/1	2.6/1	41	10.0	—	8.7	32.4	7.8	—	8.9	1.1	8.4	358/358 (100.0)
Tan (6)	2004	Canada	112	4.7/1	1.6/1	5-70	19.6	—	33.9	14.3	9.8	—	11.6	5.4	2.7	109/112 (97.3)
Trachsler (8)	2005	Switzerland	136	3.1/1	—	—	26.0	5.0	21.0	10.0	—	—	20.0	—	1	136/136 (100.0)
Moure (9)	2008	Brazil	38	8.3/1	3.1/1	17-75	10.5	—	44.7	31.6	—	—	8.0	2.6	—	38/38 (100.0)
Nejad (10)	2013	Iran	100	1.9/1	0.9/1	20-70	14.0	—	25.0	18.0	—	—	30.0	—	5	100/100 (100.0)
Qi (4)	2014	China	59	0.8/1	0.6/1	31.7 (5-74)	3.4	—	22.0	15.3	—	—	40.7	15.3	—	38/59 (64.4)
Beheshtiroy (11)	2015	Iran	97	8.2/1	1.8/1	37 (10-70)	14.4	—	63.9	6.2	—	—	9.3	10.0	1.0	97/97 (100.0)
Su (12)	2018	Taiwan	89	1.2/1	1.1/1	35.7	14.6	7.9	7.8	3.3	12.4	—	10.1	30.3	12.4	89/89 (100.0)
Özkesici Kurt (7)	2018	Turkey	97	6.3/1	1.8/1	44.2 (14-82)	41.2	15.4	7.2	5.2	2.1	—	2.1	9.3	—	50/97 (51.5)
Current study	2025	Turkey	109	5.4/1	2.4/1	40.3 (14-69)	39.4	12.8	23.9	5.5	0.9	0.9	11.9	3.7	0.9	109/109 (100.0)

AK = acne keloidalis, CCCA = central centrifugal cicatricial alopecia, DC = dissecting cellulitis, DLE = discoid lupus erythematosus, F = female, FD = frontal fibrosing alopecia, KFSD = keratosis follicularis spinulosa decalvans, L = lymphocytic, LPP-C = classic lichen planopilaris, M = male, N = neutrophilic, PCA = primary cicatricial alopecia, PPB = pseudopelade of Brocq. Histopathologic confirmation rates are reported as stated in the original articles.

thyroid disorders were more frequent in patients with LPP. We found that 9.3% and 2.3% of patients with LPP-C had hypothyroidism and Hashimoto thyroiditis, respectively. In addition, Hashimoto thyroiditis was present in 14.3% of patients with FFA. On the other hand, although an association between lichen planus and hepatitis B or C virus is well known, this relationship was not demonstrated for LPP (7, 22). We observed that 4.7% of LPP-C and 7.7% of FD patients were inactive HBV carriers. However, given the small sample size, these associations should be investigated in further studies with larger sample sizes.

Brown-Korsah et al. reported that women with CCCA were almost three times more likely to have a history of breast cancer compared to controls. However, cancer treatments and other risk factors for breast cancer, such as obesity in postmenopausal women, were not considered in that study (23). In our study, one patient (7.1%) with FFA and two patients (7.7%) with DLE had a history of breast cancer.

In addition, hidradenitis suppurativa may accompany FD and DC in some cases (24, 25). In our study, among 13 patients with FD, one patient (7.7%) had concomitant hidradenitis suppurativa.

Treatment methods for PCA

The main goals of treatment are to alleviate symptoms, halt progression of the scarring process, and prevent permanent alopecia, thereby achieving a satisfactory cosmetic outcome. However, there are no definitive stepwise treatment guidelines, and the level of evidence supporting one treatment over another is low for most PCA entities (26). Therefore, the treatment and management of PCA often depend on the preference and experience of the individual clinicians and patient-related factors, such as the extent of disease, rate of progression, treatment adherence, and comorbidities.

Topical corticosteroids or ILCS are usually the first-line treatments for localized LPP and DLE (26-28). Systemic corticosteroids are used in cases of rapid progression or severe symptoms in LPP (28). Oral HCQ, cyclosporine, methotrexate, and tetracycline are among the other treatment options for LPP (26-29). In FFA, topical, intralesional, or systemic corticosteroids, 5 α -reductase inhibitors such as finasteride, and oral HCQ may be used (26, 28). For DLE, oral HCQ is often chosen as the first-line systemic treatment, and mycophenolate, methotrexate, and thalidomide are additional therapeutic options (26-28). Although there is no specific treatment algorithm for PPB, TCS, ILCS, HCQ, or thalidomide may be considered (26, 27). For FD, topical or oral antibiotics are proposed as first-line treatment, and oral isotretinoin may be used in resistant cases. In DC, oral isotretinoin is recommended (26-28).

In our study, combination therapies were used in most patients. In lymphocytic PCA, TCS, ILCS, and HCQ were the most frequently used treatments. In contrast, oral antibiotics and oral isotretinoin were the most commonly prescribed systemic treatments in neutrophilic PCA. Primary CA often follows a chronic and recurrent course. Recurrences were more frequent in neutrophilic PCA in our cohort ($p = 0.008$), which may reflect a more aggressive and fluctuant inflammatory course, potentially driven by persistent follicular infection/dysbiosis and ongoing neutrophil-mediated inflammation. In addition, therapeutic responses can be variable, and relapses may occur when anti-inflammatory and/or antimicrobial therapy is insufficient, discontinued, or not tolerated. These factors may collectively contribute to the higher recurrence rates seen in neutrophilic PCA.

Study limitations

The retrospective design and the small sample size in some subtypes of PCA were the main limitations of this study. Given the very small numbers in some individual subtypes, we did not perform inferential subtype-level comparisons; therefore, subtype-specific findings are descriptive and should be interpreted cautiously. In addition, patients without histopathological confirmation of PCA were excluded. Therefore, the prevalence of certain PCA subtypes, such as FD, DC, or acne keloidalis, may have been underestimated because the diagnosis and management of some subtypes can be based solely on clinical findings. Another limitation was the lack of a standard treatment protocol; treatments were applied according to the individual clinicians' preferences at our center.

Conclusions

Primary CAs are important hair disorders that result in permanent alopecia. Therefore, accurate diagnosis and early treatment are essential to achieve better cosmetic outcomes and improve patients' quality of life. In our study, LPP-C was the most frequent subtype of PCA, followed by DLE. In addition to alopecia, pruritus, especially in lymphocytic types, and pain, especially in neutrophilic types, were the most common symptoms. Recurrence rates were higher in neutrophilic subtypes.

Future studies with larger sample sizes and from various geographic regions are needed to better clarify the prevalence, clinical course, and optimal treatment approaches for PCA.

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