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Original Article

Dermatology Section

Clinical, Dermoscopic and Histopathologic Features of Cicatricial Alopecia- A Prospective Cohort Study

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ABSTRACT

Introduction: Cicatricial Alopecia (CA) is a scarring form that arises due to permanent hair follicle destruction. It can either primarily affect the follicles or by an external process leads to secondary alopecia causing scarring and considerable disfigurement of the scalp. Hence initial diagnosis, aetiology of the contributing factors and earlier intervention are vital.

Aim: To identify the diagnostic features of CA based on clinical, dermoscopic and histopathological (HP) findings and the underlying aetiological causes.

Materials and Methods: This prospective cohort study was conducted in a tertiary care centre of Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, Tamil Nadu, India from November 2016 to September 2017. Total 50 clinically diagnosed CA cases were randomly selected and case history, related skin findings, predisposing factors and other findings associated with scarring were documented. Dermatological examination and Hair pull test was done. Trichoscopic examination was done using a non polarised dermoscopy with specific emphasis on the follicular ostia and morphological patterns. Scalp biopsy was done. The collected data were analysed in terms of frequency and percentage and Chi-square test was also performed with p-value <0.05 as statistically significant.

Results: Out of total 50 patients, 22 (44%) were males, and 28 (56%) females with the mean age of 35.46±15.03 years. Total 29 (58%) patients had multiple lesions and 21 (42%) had localised involvement of alopecia in the scalp. Focal type was noted in 86%, and diffuse type in 14%. In this study, 80% of CA were of primary causes with only 20% secondary causes. Lichen Plano Pilaris (LPP) and trauma were the most common primary and secondary causes. The dermoscopic features of absent follicular orifice, arborising thicker blood vessel and pustule formation were statistically significant (p-value <0.001) for the diagnosis of CA. The most common histopathological feature was basal cell vacuolization (54%), followed by hyperkeratosis (52%), and follicular plugging (46%).

Conclusion: The clinical, dermoscopic and histopathological findings of this study were consistent for diagnosis and differentiating primary CA. Histopathology is the final confirmatory diagnostic tool when characteristic clinical and dermoscopic features are absent.

Keywords: Permanent hair follicle destruction, Primary scarring alopecia, Skin of colour, Trichoscope

INTRODUCTION

Cicatricial Alopecia (CA) is a scarring form that arises due to permanent hair follicle destruction leading to localised loss of hair [1]. Due to the destruction of stem cell niche of the bulge region of follicle, hair regrowth is impaired. It can either primarily affect the follicles or by an external process leads to secondary CA [2]. The common etiology of primary CA includes Lichen plano pilaris [LPP], Discoid lupus erthematosus [DLE], Pseudopelade of Brocq [POB] in the descending frequency order. Infrequent causes are central centrifugal cicatricial alopecia, Keratosis Spinulosa Declavans [KSD], alopecia mucinosa, Acne Keloidalis Nuchae (AKN), folliculitis declavans, frontal fibrosing alopecia, dissecting cellulitis, erosive pustular dermatosis and non specific end stage CA [3]. Causes for secondary CA include infections, trauma, granulomatous condition, sclerosing disorders, neoplasm, hereditary disorders and developmental defects. Patients with CA generally have symptoms like burning, itching, discharge and scaling leading to significant scarring of their scalp which can be a major psychosocial concern for them [4]. Hence, early diagnosis, identification of the causative factors and prompt treatment is crucial.

A non invasive modality like Dermoscopy is very useful to identify and diagnose scalp conditions leading to alopecia [5]. Dermoscopic examination shows features like loss of follicular opening, follicular pustules and scattered single hair follicle [6]. Histopathological correlation aids as a confirmatory tool for diagnosis [7]. In addition to distinguish cicatricial alopecia from non cicatricial alopecia, it is classified as lymphocytic and neutrophilic based on the predominant infiltrate [8]. Even though the aetiolopathogenesis of CA have been noted, there is a paucity of published literature in it. Dermoscopy may aid in early diagnosis, monitor the disease progression, response to therapy and to identify the reactivation of the disorder. This study was done to identify the characteristic features for the diagnosis and differentiation of primary CA and to find the concordance between the dermoscopic and histopathological findings. Though similar studies are found in literature, they are done mostly in caucasian skin type. The novelty of this study is that it is conducted in skin of colour that has significantly distinct features in reaction patterns and also in clinical predominance of certain dermatoses.

MATERIALS AND METHODS

This prospective cohort study was conducted in Dermatology Outpatient Department of a tertiary care centre of Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, Tamil Nadu, India from November 2016 and September 2017. Prior to the commencement of the study, Institutional Ethical Committee approval (No.08122016) and informed consent was obtained from patients. Total 50 new clinically diagnosed CA cases were randomly selected for the study.

Inclusion criteria: Patients having following findings for diagnosis of CA -

a) On clinical examination, absence of follicular ostia

b) Histologically, the presence of follicles replaced by fibrous stellae and

c) Dermoscopic features of interrupted honeycomb pattern of pigmentation, absence of follicular openings, presence of fibrotic bright white dots and irregularly distributed/absent pin point white dots were included in the study.

Exclusion criteria: Patients who have been treated earlier with either topical or systemic therapy were excluded.

Study Procedure

Detailed case history with reference to the duration, its onset and development, other associated dermatological findings and symptoms like itching, scarring and pain were documented. Presence of predisposing features like infection and trauma along with relevant family and personal history were noted. Dermatological examination including the lesion morphology, location, dimensions and number were documented along with Hair pull test. Presence of hair loss in other sites were noted along with a complete dermatological examination. Routine blood investigations were done. In pertinent cases Culture and Sensitivity Pus (C&S), Woods lamp examination, scraping to rule out fungal infections, Mantoux, HIV, VDRL and antinuclear antibodies were done. Using a non polarised dermoscope trichoscopic examination was performed in the scalp. The alopecia lesions were clinically examined and both the central margin and the peripheral margin were noted and photo images were documented. Scalp biopsy was taken from active border with both horizontal and vertical sections stained with Haematoxylin and Eosin (H&E).

STATISTICAL ANALYSIS

Using IBM Statistical Package for Social Sciences software (SPSS) version 23.0, the data collected was analysed. For categorical variables, frequency and percentage analysis were done. In categorical data to note the significance, Chi-square test was done. A p-value <0.05 was taken as statistically significant.

RESULTS

Fifty cases diagnosed clinically as CA which had not undergone any prior treatment, were enlisted for this study. Among 50 patients, 22 (44%) were males, and 28 (56%) females with the mean age of 35.46±15.03 years. The number of cases with CA were maximum in the age group of 31-40 years 15 (30%) followed by 21-30 years 11 (22%), 41-50 years 10 (20%), 4-20 years 7 (14%), 51-60 years 5 (10%) and above 60 years 2 (4%). Total 48 patients (96%) had a gradual onset. The range of disease duration was from 8 months to 20 years, with a mean duration of 3.96±4.5 years. Total 29 (58%) patients had multiple lesions and 21 (42%) had localised involvement of alopecia in the scalp. The focal type (patchy involvement of alopecia) was noted in 86%, and diffuse type (diffuse involvement of the scalp) in14%. The vertex of the scalp was the most common site involved (60%). Concomitant skin lesions were noted in 20% of CA cases and the most common being DLE [Table/Fig-1].

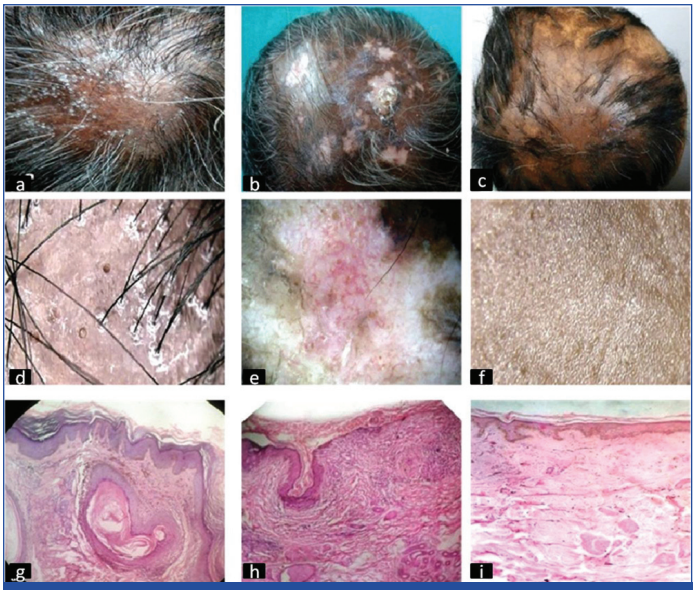
The aetiology of various type of CA are summarised in [Table/Fig-1]. The most common cause of primary CA was LPP 15 (30%). The other less frequent causes noted were AKN 2 (4%) and 1(2%) each of KSD, Graham Little Picardi syndrome (GLP) and Systemic Lupus Erythematosus (SLE). Amongst the secondary causes of CA, naevus sebaceous 2 (4%) and trauma 2 (4%) were the most common.

The clinical features of various type of CA are summarised in [Table/Fig-2,3]. In patients with LPP, 11 were males and 4 females. The involvement was most common in the vertex followed by parietal area [Table/Fig-4]. Irregular areas of scarring alopecia associated with perifollicular erythema and papules around the emergence of hair shaft were noted [Table/Fig-2a]. Itching was a predominant symptom in 7 (46.6%) patients [Table/Fig-4]. Oral Lichen planus was noted in 1 (6.6%) and cutaneous lesions of Lichen planus in 2 (13.3%) patients. DLE was noted in 4 male and 9 female cases. The vertex was the commonest site of involvement in both sexes. There were many erythematous, scaly, depigmented and atrophic plaques with marginal hyperpigmentation. In 3 (23%) cases other body site lesions were noted. One patient has scalp DLE with associated SLE [Table/Fig-2b].

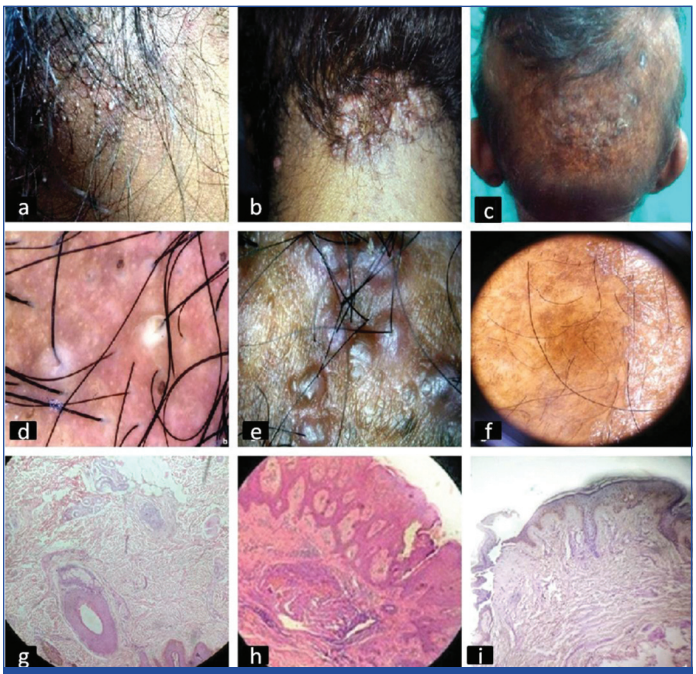
One patient presented with features suggestive of GLP like violaceous lesions of scarring alopecia of the scalp along with axillary non scarring alopecia and follicular type of lichen planus in the back. Features suggestive of POB were seen in one male and two females, presenting as multiple reticular irregular lesions of varying sizes that were white and atrophic. Classical foot print in snow appearance was noted in two cases. The lesions were asymptomatic with no signs of inflammation [Table/Fig-2c]. A single case of KSD was presented with cicatricial plaques with few pustules [Table/Fig-3a]. Both patients of AKN were males and presented with cicatricial plaque with papulonodules and pustules in the occipital scalp and posterior neck [Table/Fig-3b]. Lipoid proteinosis were clinically presented with atrophied waxy papules seen over the occiput [Table/Fig-3c].

Cicatricial alopecia aetiology	N=50	Mean age (years)	Gender		Duration (years)	Skin lesions
			Male n=22 (%)	Female n=28 (%)		
Primary Cicatricial Alopecia (N=40) 80%						
Lichen plano pilaris	15 (30%)	39.50	11 (50%)	4 (14.2%)	1.04	2 (4%)
Discoid lupus erythematosus	13 (26%)	39.38	4 (18%)	9 (32.2%)	4.5	3 (6%)
Pseudopelade of brocq	3 (6%)	48.6	1 (4.5%)	2 (7%)	5.5	0
Acne keloidalis nuchae	2 (4%)	35.5	2 (9%)	-	2.25	0
Keratosis spinulosa declavans	1 (2%)	17	-	1 (3.5%)	7.5	0
Graham little syndrome	1 (2%)	68	-	1 (3.5%)	0.5	1 (2%)
SLE with DLE	1 (2%)	24	-	1 (3.5%)	0.16	1 (2%)
Non specific CA	4 (8%)	30.75	2 (9%)	2 (7%)	3.25	0
Secondary Cicatricial Alopecia (N=10) 20%						
Lipoid proteinosis	1 (2%)	15	1 (4.5%)	-	14	1 (2%)
Naevus sebaceous	2 (4%)	12.5	1 (4.5%)	1 (3.5%)	12.5	0
Trichilemmal cyst	1 (2%)	4	-	1 (3.5%)	4	0
En coup de sabre	1 (2%)	18	1 (4.5%)	-	4	0
Trauma/Radiotherapy	3 (6%)	32.3	2 (9%)	1 (3.5%)	11.6	1 (2%)
Gunther's disease	1 (2%)	21	1 (4.5%)	-	0.08	0
Seborrheic keratosis	1 (2%)	67	1 (4.5%)	-	10	1 (2%)

[Table/Fig-1]: Aetiology of various types of cicatricial alopecia.



[Table/Fig-2]: Global view (a-c), Dermoscopy (d-f) and Pathological features (g-i) of LPP, DLE and POB. Histopathology H&E (X100). (g) LPP showing hypergranulosis, basal cell degeneration with pigmentary incontinence and interface dermatitis (h) DLE showing Keratotic plugging, epidermal atrophy, focal basal cell degeneration and plenty of lymphocytic infiltrate (i) POB showing epidermal atrophy, atrophied hair follicles and absence of inflammatory infiltrate.
LPP: Lichen plano pilaris; DLE: Discoid lupus erythematosus; POB: Pseudopelade of brocq



[Table/Fig-3]: Global view (a-c), Dermoscopy (d-f) and Pathological features (g-i) of KSD, AKN and LP. Histopathology H&E (X100). (g) KSD showing empty hair follicles with sparse peri follicular lymphocytic infiltrate and dermal edema (h) AKN showing flaky hyperkeratosis, plenty of lymphocytic infiltrate around epidermal appendages (i) LP showing by atrophy of epidermis, pink hyaline material and atrophied hair follicle.
KSD: Keratosis spinulosa declavans; AKN: Acne keloidalis nuchae; LP: Lipoid proteinosis

On dermoscopy, follicular opening was absent in all 50 (100%) cases and epidermal atrophic changes in 41 (82%). Thick peripilar cast surrounding most of the hairshafts, erythema and blue grey dots were typically noted in cases with LPP [Table/Fig-2d]. Linear and thick arborising blood vessels were mostly seen in cases of DLE along with loss of pigment and brownish discolouration in the interfollicular area [Table/Fig-2e]. POB was characterised by absent follicular opening and background shades of white and beige [Table/Fig-2f]. Classic white dots, pustules and atrophy were seen in KSD [Table/Fig-3d]. AKN was characterised by erythemaous papules, peripilar cast and whitish atrophic areas [Table/Fig-3e]. Lipoid proteinosis presented with features like brownish discolouration, atrophy and whitish areas [Table/Fig-3f]. Statistically significant differences in frequency of dermoscopic signs were noted among

Cicatricial alopecia aetiology N=50	Predominant symptoms									Region				
	Thinning/Shedding	Recession of hair line	Pain/Burning	Itching	Photosensitivity	Scaling	Discharge	Scaring	Regrowth of hair	Vertex	Frontal	Parietal	Occipital	Temporal
Lichen plano pilaris (15)	2	0	0	7	0	1	0	15	0	10	7	8	4	2
Discoid lupus erythematosus (13)	0	0	0	4	7	2	0	13	0	10	6	7	4	1
Pseudopelade of brocq (3)	3	0	0	0	0	0	0	3	0	3	2	3	2	1
Acne keloidalis nuchae (2)	2	0	2	0	0	0	2	2	0	0	0	0	2	0
Keratosis spinulosa declavans (1)	1	0	1	0	0	0	0	1	0	0	0	0	1	0
Graham little syndrome (1)	1	0	0	1	0	0	0	1	0	1	0	1	0	0
SLE with DLE (1)	1	0	0	1	1	1	0	1	0	0	1	1	1	0
Non specific CA (4)	2	0	0	1	0	0	0	4	0	3	1	2	1	1
Lipoid proteinosis (1)	1	0	0	0	0	0	0	1	0	0	0	1	1	1
Naevus sebaceous (2)	0	0	0	0	0	0	0	1	0	1	0	0	1	0
Trichilemmal cyst (1)	1	0	0	0	0	0	0	1	0	1	0	0	0	0
En coup de sabre (1)	0	0	0	0	0	0	0	1	0	0	1	0	0	0
Trauma/ Radiotherapy (3)	0	0	0	0	0	0	0	3	0	1	1	2	0	1
Gunther's Disease (1)	1	0	1	1	1	1	1	1	0	0	1	1	0	0
Seborrheic keratosis (1)	1	0	0	0	0	0	0	1	0	0	0	1	0	0

[Table/Fig-4]: Predominant symptoms and regional involvement of cicatricial alopecias.

the various causes of primary CA [Table/Fig-5]. Significant follicular features similar to all subtypes on trichoscopic examination was absent. Significant variation across various subtypes under follicular features noted were follicular pustules in KSD, follicular plugging in LPP, AKN and peripilar cast in LPP. In perifollicular and interfollicular features, significant variation across various subtypes documented were thick arborising blood vessels in DLE and perifollicular erythema in both DLE and AKN. Fibrous connective tissue in place of hair follicles was documented in all 50 (100%) cases.

The most common histopathological feature was basal cell vacuolization (54%), followed by hyperkeratosis (52%), and follicular plugging (46%). Others like pigmentary incontinence (36%), perifollicular lymphocytic infiltrate and hypergranulosis each (30%), epidermal atrophy (28%) were also noted. LPP was characterised by hypergranulosis, basal cell degeneration with pigmentary incontinence and interface dermatitis [Table/Fig-2g]. Epidermal atrophy, keratotic plugging, focal basal cell degeneration with infiltrate consisting of plenty of lymphocytes were noted in DLE [Table/Fig-2h]. In POB epidermal atrophy, hair follicle atrophy with absent inflammatory infiltrate were noted [Table/Fig-2i]. In KSD, there was empty follicles with scant perifollicular lymphocytic infiltrate in a oedematous dermis [Table/Fig-3g]. Flaky hyperkeratosis, plenty of lymphocytic infiltrate around epidermal appendages were noted in AKN [Table/Fig-3h]. Lipoid proteinosis was characterised by epidermal atrophy, pinkish hyaline deposits with atrophic hair follicle [Table/Fig-3i]. Follicular atrophy was the most common HP

Trichoscopic features	DLE (n=13) (%)	LPP (n=15) (%)	POB (n=3) (%)	KSD (n=1) (%)	AKN (n=2) (%)	Non specific CA (n=4) (%)	p-value (Chi-square test)
Follicular features							
Yellow dots	-	-	-	-	-	1 (25)	0.7
Black dots	-	-	-	-	-	-	-
Classic white dots	-	3 (20)	1 (33)	1 (100)	-	2 (50)	0.8
Absent follicular openings	13 (100)	15 (100)	3 (100)	1 (100)	2 (100)	4 (100)	0.002†
Follicular pustules	-	-	-	1 (100)	-	-	<0.001†
Follicular hyperkeratosis	5 (38)	6 (40)	-	-	-	1 (25)	0.2
Follicular plugging	-	1 (7)	-	-	2 (100)	-	0.002†
Peripilar cast	-	14 (93)	-	-	-	-	<0.001†
Perifollicular and interfollicular features							
Thick arborising blood vessels	10 (77)	-	-	-	-	-	0.003†
Elongated linear blood vessels	4 (31)	-	-	-	-	-	0.7
Scattered brown discoloration	3 (23)	6 (40)	-	-	-	2 (50)	0.446
Perifollicular erythema	13 (100)	-	-	-	2 (100)	-	<0.001†
Perifollicular scaling	10 (77)	8 (53)	-	-	-	-	0.092
Epidermal atrophy	13 (100)	11 (73)	2 (67)	1 (100)	2 (100)	3 (75)	0.025*
Cicatricial white patches	12 (92)	8 (53)	2 (67)	-	-	1 (25)	0.045*
Blue grey dots	-	8 (53)	-	-	-	-	0.153

[Table/Fig-5]: Trichoscopic features of various types of primary cicatricial alopecias.
p-value <0.05, †p-value <0.01. DLE: Discoid lupus erythematoses; LPP: Lichen plano pilaris; POB: Pseudo pelade of brocq; KSD: Keratosis spinulosa declavans; AKN: Acne keloidalis nuchae

feature noted in all the subtypes. Keratotic follicular plugging varied significantly across the various lesions (DLE, LPP, AKN) and most common lesion associated with the feature was DLE. Pigmentary incontinence varied significantly across DLE, LPP and most common lesion associated with the feature was LPP. Hypergranulosis was significantly noted only in LPP [Table/Fig-6].

Histopathological features	DLE (n=13) (%)	LPP (n=15) (%)	POB (n=3) (%)	KSD (n=1) (%)	AKN (n=2) (%)	p-value (Chi-square test)
Epidermal atrophy	6 (46)	5 (33)	3 (100)	-	-	0.158
Hyperkeratosis	12 (92)	14 (93)	-	-	-	0.607
Keratotic follicular plugging	12 (92)	10 (67)	-	-	1 (50)	0.041*
Hypergranulosis	-	15 (100)	-	-	-	0.005*
Perifollicular lymphocytic infiltrate	7 (54)	5 (33)	-	1 (100)	2 (100)	0.765
Basal cell vacuolization	12 (92)	15 (100)	-	-	-	0.532
Necrotic keratinocytes	8 (62)	5 (33)	-	-	-	0.231
Pigmentary incontinence	4 (31)	14 (93)	-	-	-	0.035*
Follicular atrophy	4 (31)	5 (33)	3 (100)	1 (100)	2 (100)	0.03*
Dermal sclerosis	-	-	-	-	-	-
Absent sebaceous gland and hair	4 (31)	-	1 (33)	-	2 (100)	0.134

[Table/Fig-6]: Histopathological features of various types of primary cicatricial alopecias.

DISCUSSION

Primary CA is a group of disorders where the follicles are replaced by fibrosis resulting in permanent hair loss [3]. The follicle is the primary focus of the inflammatory reaction [9]. Whiting DA [2] has described that primary CA comprises 7.3% of all patients of hair loss. United Kingdom (UK) survey conducted by Griffin LL et al., [10] reported about 9.6% new patients of primary CA detected every year. There are very few documented studies from India on CA [11]. This study was undertaken to identify the features diagnostic of CA primarily based on clinical findings, dermoscopic patterns and histopathological examination and to identify the basic aetiological causes. In this study, 80% of CA were of primary causes with only 20% secondary. LPP and trauma were the most common primary and secondary causes, respectively. The dermoscopic features of absence of follicular opening, thick arborising blood vessel and pustules were significant along with histopathological features of follicular plugging, basal cell vacuolization and hyperkeratosis in this study.

A retrospective study done by Whiting DA [2] reported CA in 7.3% of all cases of hair loss with a primary to secondary CA ratio of 4:1 which was in concordance with present study. Minor variations were noted when comparing similar research conducted in China by Qi S et al., [12]. The constitution of age group ranged from 5-74 years in this study, with 31.7 years as the mean age, and extent of disease ranging from 1 month to 10 years with an average duration of 1.8 years. In the index study, the age range was 4-68 years, with 35.46 years as the mean age, and period of disease ranging from 8 months to 20 years, with an average duration of 3.96 years. A majority of CA patients (30%) were in the age group of 31-40 years. This clinical difference may be due to the varied ethnic and demographic features reported in Caucasian skin type when compared to skin of colour in the index study. The longer disease duration indicates the slower and progressive development of CA. Hence an earlier clinical diagnosis of CA can halt the disease development. The female (56%) to male (44%) ratio in the present study was 1.3:1 which was similar to studies by Olsen EA et al., [13] (1.6:1), and Thakur BK et al., [14] (2:1). This shows a female predominance when compared to the males affected. The slight variation in the ratio can be due to the geographical differences.

The most common primary CA subtype noted in this study was LPP 15 (30%) followed by DLE 13 (26%), POB 3 (6%), AKN 2 (4%) and GLP1 (2%). Also, the most common subtypes were DLE 13 (22%), LPP 2 (3.4%), POB 9 (15.3%) as reported by Qi S et al., [12]; DLE 10 (41.7%), LPP 5 (20.8%), POB 2 (8.3%) as reported by Thakur BK et al., [14]; and DLE 38 (33.9%), POB 27 (24.1%), LPP 25 (22.3%) as reported by Tan E et al., [9]. This finding can be ascribed to the increased occurrence of Lupus erythematoses in skin of colour when compared to Caucasian skin [15]. Further, due to the limited time frame of this study and lesser number of primary CA cases noted there was paucity of subtypes like alopecia mucinosa, folliculitis declavans and dissecting cellulitis of scalp.

Very limited research on secondary CA have been documented in India. In a study conducted by Kumar UM and Yelikar BR [16] in

secondary CA, there were 4 cases of morphea and one each case of lupus vulgaris, congenital absence of skin, burn and sarcoidosis. On comparison with the present study, there were 3 cases of trauma/radiation, 2 cases of naevus sebaceous and one each case of trichilemmal cyst, lipoid proteinosis, seborrheic keratosis, En coup de sabre and Gunther's disease. This variation may be because the present study was based on clinical, dermoscopic and histopathological findings, whereas the other study was based on histopathological features only. Further due to the shorter duration of the present study it was not possible to find a substantial number of cases with secondary CA presentation.

Dermoscopy is a non invasive instrument which provide hints for clinical diagnosis and biopsy site localisation [6]. The characteristic feature of CA in dermoscopy is lesser density of hair and loss of follicle ostia in almost all patients [4]. LPP is characterised by peripilar casts and blue grey dots and patches. GLP is a variant of LPP with CA of the scalp and non CA type in associated other sites with follicular type of lichen planus. There is similarity with the dermoscopic and histopathological findings of LPP. These features of dermoscopy had a positive correlation with LPP (p-value <0.01). Unique dermoscopic feature of thick arborising and linear blood vessels was noted only in DLE (77%), and this finding was statistically significant (p-value <0.001) when compared to other subtypes of CA. This was comparable to research done by Thakur BK et al., [14] (80%). Also red dots, corresponding to follicular openings surrounded by dilated vessels, considered a good prognostic factor was not seen in the index patients [17]. This is similar to the findings by Thakur BK et al., [14] but in contrast to that by Qi S et al., [12]. The latter study reported the feature in 25% of DLE cases. This may be due to the factor that red dots may not be a prominent dermoscopic feature of DLE in skin of colour [14].

Pseudopelade of Brocq (POB) is a slowly progressive scarring alopecia characterised by asymptomatic, non inflammed reticulate lesions localised to the central scalp. The dermoscopic finding of POB, noted in the present study, was white atrophic areas with loss of follicular opening. This dermoscopic feature was similar to the study conducted by Thakur BK et al., [14]. A study by Qi S et al., [12] reported the dermoscopic features of POB like arborising vessels and pinkish white appearance. This finding was not documented in the present study, because of the darker skin type of the patients. The classical dermoscopic features of AKN (hair follicle tufting, ingrown follicles) were not documented in the current study, due to the lack of cases. Absence of follicular opening was the common dermoscopic feature shared along with secondary CA. Seborrheic keratosis and naevus sebaceous were presented with yellowish globules, crypts, cerebriform surface changes and fat finger structures. Thus, dermoscopy may aid as a screening tool for a presumptive diagnosis that further can be established with biopsy.

Biopsy of the scalp is vital for the precise clinical diagnosis and in differentiating the types of CA [18]. They are classified primarily into lymphocytic, neutrophilic and mixed type based on the histopathology [8]. In the present study, lymphocytic infiltration was seen in 38 (95%) cases of primary CA. A mixed inflammatory infiltrate was seen in only 2 (5%) cases of AKN. This is similar to a study conducted by Kumar UM and Yelikar BR [16] who reported 23 (96%) cases of lymphocytic infiltration and one (4%) case of mixed infiltrate (folliculitis decalvans). Qi S et al., [5] reported a predominant neutrophilic infiltration in 33 (56%) cases which may be due to the higher proportion of folliculitis decalvans and dissecting cellulitis was documented. The reason for these differences could be related to the shorter time limit of this study and other attributable causes like geographical variations and ethnic factors.

Hypergranulosis, basal cell vacuolization, pigmentary incontinence and lichenoid interface dermatitis in upper perifollicular region were seen in most of the cases of LPP [19]. In a study conducted by Thakur BK et al., [14], similar features were documented but in addition perifollicular fibrosis was also noted. This variation may be due to the scalp biopsy done at an earlier stage of LPP in this study as concentric perifollicular fibrosis develops at a later stage as the disease progress. Patches of DLE showed epidermal atrophy, keratotic follicular plugging, focal basal cell vacuolization and both dermal and periadnexal lymphocytic infiltrate. Apart from similar findings thickened basement membrane was also noted in a study conducted by Kumar UM and Yelikar BR [16], which may signify a late stage of DLE. Atrophy of epidermis and absent follicles and sebaceous glands were noted in POB cases. Fibrous tract replacing follicle was also seen in addition to the above findings in a study by Qi S et al., [12]. These variations may be due to the scalp biopsy done in different stages of CA. Flaky hyperkeratosis with predominant periappendageal lymphocytic infiltrate was seen in AKN. Characteristic perifollicular lymphocytic infiltrate with limited atrophic follicles was noticed in KSD. No other dermatological features or loss of eyebrows were documented in the patient that are predominantly seen in males. KSD is a X-linked genodermatoses and females are carriers or have milder symptoms. In secondary CA, features like scantiness of hair follicles with atrophic epidermis were common. Lipoid proteinosis cases had hyaline material deposition amid rete ridges which stained with Periodic Acid-Schiff (PAS). Both the naevus sebaceous cases had epidermal hyperplasia with papillomatosis and multiple sebaceous glands that were mature. Obvious acanthosis and papillomatosis were noted in seborrheic keratosis. Trichilemmal cyst has abrupt keratinisation with foci of calcification. Among the above features atrophic follicles, hypergranulosis, follicular keratotic plugs and pigment incontinence were statistically significant (p-value <0.05). Similar statistical significance in keratotic follicular plugging and follicular atrophy was noted by Qi S et al., [12] and Thakur BK et al., [14]. In their study vacuolization of basal cells, neutrophilic folliculitis, perifollicular lymphocytic infiltration, parakeratosis was also found to be significant (p-value <0.05). These differences can be ascribed to the number variations and morphological staging of specific CA among these studies. Four patients in the present study had clinically cicatrised lesion with absence of follicular ostia in dermoscopy. Histopathologically, apart from epidermal thinning and atrophic follicles, other findings were indecisive. They were documented as non specific primary CA. Moure ERD et al., [20] reported 13 cases of non specific CA amongst 38 patients.

Limitation(s)

A limitation of this study is lack of documenting clinical types of primary CA like folliculitis decalvans, dissecting cellulitis and alopecia mucinosa. Also, the small number of cases were noted in secondary CA. This can be attributed to the inadequate time duration of the present study.

CONCLUSION(S)

Primary CA are rare conditions and hence an increased awareness is essential to improve its outcome. Dermoscopy provides diagnostic clues in differentiating DLE and LPP from other primary CA and biopsy site localisation. Histopathology is the final confirmatory diagnostic means to identify the etiology of primary CA when characteristic clinical and dermoscopic features are absent. Some patients of primary CA may be indecisive in histopathology also and are categorised under non specific CA. These cases must to be periodically followed-up as they may evolve into a specific type at a latter point.

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