European Journal of Clinical Pharmacy
Print ISSN: 2385-409X **Online ISSN**: 3105-0409
Website: https://farmclin.com/

Original Research Article

A prospective study to find out epidemiological spectrum, Etio-clinico and histopathological correlation of cicatricial alopecia encountered in tertiary care centre of Western Rajasthan

Article History:

Name of Author:
Manish Sethiya[1], Chandraprakash Chouhan[2], Pankaj Rao[3], Manjulata Verma[3] and Meenakshi Rao[4]

Affiliation: *[1]MD Skin, VD & Leprosy, Balotra*
[2]Associate Professor, Department Of Dermatology, Venereology And Leprosy, Dr S.N.Medical College, Jodhpur
[3]Professor And Head, Department Of Dermatology, Venereology And Leprosy, Dr S.N.Medical College, Jodhpur
[4]Assistant Professor, Department Of Dermatology, Venereology And Leprosy, Dr S.N.Medical College, Jodhpur
[5]Professor, Department Of Pathology, Aiims, Jodhpur.

Corresponding Author:
Chandraprakash Chouhan

Received: 12-12-2025
Revised: *30*-12-2025
Accepted: *18*-01-2026
Published: *30*-01-2026

Abstract: Introduction: Cicatricial alopecia is an enigmatic group of hair disorders in which follicular units are replaced by fibrous tissue, resulting in progressive and permanent scarring hair loss. It has always been a diagnostic and therapeutic challenge to the dermatologists. Aim: to find out Etio-clinico and histopathological correlation of cicatricial alopecia encountered in tertiary care centre. Methods: A prospective and clino-histopathologic evaluation of 84 consecutive patients of cicatricial alopecia attending the dermatology outdoor was performed who meet inclusion criteria. Results: disease varies from 7 years to 59 years with a mean age of 27.57 years. 85.7% cases of lymphocyte mediated group and the ratio of lymphocytic mediated versus all other: 6:1. In lymphocytic group Lichen planopilaris (35.7%), DLE (31%), Pseudopelade (16.7%) are mainly present. Conclusion: Cicatricial alopecia is a trichological emergency, hence required meticulous management to prevent further complications and follicular destruction and for a prompt and accurate diagnosis, combined clinico-pathological and immunologic approaches are needed.

Keywords: Cicatricial alopecia, clinico-pathological, immunologic.

INTRODUCTION

Cicatricial alopecia is an enigmatic group of hair disorders in which follicular units are replaced by fibrous tissue, resulting in progressive and permanent scarring hair loss[1-4]. It has always been a diagnostic and therapeutic challenge to the dermatologists[2,5]. Historically, one of the first descriptions of type of cicatricial alopecia was given by Brocq in 1885 which was later known as Pseudopelade[6]. In 1888, Quinquaud described pustular folliculitis with scarring which is now known as Folliculitis decalvans[7]. Cicatricial alopecia is also classified according to a consensus-issued classification scheme based on the predominant cell type present: lymphocytic, neutrophilic, or mixed and hence histopathology has important role in the diagnostic evaluation[8]. It may result from a disease process affecting the hair follicles primarily or secondary to external disease process including trauma, burns,

chemicals, chronic infections, systemic disease or malignancy[7, 9].

Various classification schemes exist in literature with distinction based on clinical features, age of onset and pathology using different terminologies[5, 7-9]. In 2001, NAHRS issued a working classification based on predominant type of inflammatory infiltrate so that uniformity can be achieved in terminologies thus allowing better correlation[11]. The disorders were categorized in lymphocytic, neutrophilic, mixed and non-specific groups. Epidemiology of cicatricial alopecia is widely unknown with prevalence ranging from 3.2-7.3%[1, 12-13]. Majority of the patients belong to primary cicatricial alopecia[1-2, 12]. Females are affected 3-4 times more commonly than males[11]. Most common causes of primary cicatricial alopecia include Discoid lupus erythematosus (30-35%), Pseudopelade (25-30%), lichen-planopilaris (20-25%), and folliculitis decalvans (10-15%) [1, 12-13]. Majority of cicatricial alopecias, differing clinical presentations which change over time and often overlap thus making accurate diagnosis challenging for both the clinician. Cicatricial alopecia is trichologic emergency as the resultant alopecia is often irreversible[1]. A perfect diagnosis is usually reached with the help of clinico-pathological features, stigmata of other cutaneous disorder along with special investigations[1-3]. Thus a scalp biopsy is warranted in all cases[1, 7-8]. For histopathological evaluation, minimum 4 mm biopsy sample from active site are required while, another sample is needed for direct immunofluorescence (DIF)[8]. The aim of this study was to find out epidemiological spectrum and to describe prospectively the histopathological outcomes of cicatricial alopecias & correlate with clinical diagnosis. Aditionally, on the basis of etiology it can be classify as primary and secondary cicatricial alopecia.

MATERIAL & METHODS: -

A prospective and clino-histopathologic evaluation of 84 consecutive patients of cicatricial alopecia attending the dermatology outdoor was performed who meet inclusion criteria. All patients with clinical evidence of cicatricial alopecia irrespective of age, sex and duration of the disease were enrolled in the study. The patients with prescence of thrombocytopenia (<50,000/mm3) or disorders of coagulation, HIV or any immunodeficiency, Pregnancy and nursing women, predisposition to keloid formation and psychological problems were exclude from the study. After written consent, detailed history, clinical examination and the baseline investigations like complete blood count, ESR, KOH for fungus were done in each case and relevant investigations like ANA, culture for fungus and bacteria were performed when required. Based on history and clinical and laboratory finding, diagnosed as cicatricial alopecia. Then a punch biopsy 4-5 mm in size was done from the active advancing margin of latest alopecic patch to include both the scarred area and nearby clinically normal skin. The biopsy was sent to pathological department for histopathological evaluation and compare to clinical diagnosis. In present study, the informed consent document and any subsequent modifications were reviewed and approved by the Institutional ethics committee (IEC).

RESULTS: -

In present study, table -01 communicated that total 84 patients comprised of females 69.1% and males 30.9%. The age at the onset of disease varies from 7 years to 59 years with a mean age of 27.57 years. Maximum numbers of cases were seen between the age group of 10-30 years**.** The cases were categorized into various groups i.e. Lymphocytic [mediated (DLE, LPP, PPB), Neutrophilic mediated (FD) and miscellaneous groups in accordance with NAHRS working classification for primary cicatricial alopecia. After final diagnosis there were 85.7% cases of lymphocyte mediated group and all the other groups showed equal frequency of 4.76%. According to table-02, the ratio of lymphocytic mediated versus all other: 6:1**.** In the lymphocyte mediated group maximum numbers of cases were found out to be of Lichen planopilaris (35.7%), DLE (31%), Pseudopelade (16.7%)**.** The duration of the disease varied markedly ranging from as short as 15 days to 16 years, with a mean duration of 2.42 years.

Table No. 1: Age and Sex distribution of patients with cicatricial alopecia. N=84

Variables		No. of patients
Age group (years)	0-10	10 (11.9%)
	11-20	20(23.81%)
	21-30	24(28.57%)
	31-40	12(14.29%)
	41-50	10 (11.20%)
	>50	8 (9.52%)
Gender	Male	26 (30.95%)
	Female	58(69.05%)

Clinical profile: - The present study highlighted that the most common characteristic was asymptomatic to erythematous plaques and hair loss patches in 88.1% cases. The most common presenting features of DLE (figure-1) present in >50% cases were itching, scaling, follicular plugging, hyperpigmentation, telangiectasia and atrophy & LPP presented with itching, tenderness, follicular erythema, hyperpigmentation and activity at margin (figure-2). All Pseudopelade cases and end stage disease presented with asymptomatic well defined atrophic patches. Patients of folliculitis decalvans (figure-3) presented with papules, pustules with crusting and few atrophic patche. Pseudopelade (16.7%) presented with irregular, shiny and atrophic patches of alopecia with islands of normal hair (figure-4).

Table No. 2: Distribution of cases according to NAHRS working classification

Category	No. of cases (n=84)
Lymphocyte-associated	72(85.71%)
LPP	30(35.7%)
DLE	26(31%)
PB	14(16.7%)
AM	2(2.4%)
Neutorphil-associated	
FD	4(4.76%)
Undiagnosed	4(4.76%)
Fungal folliculitis	4(4.76%)

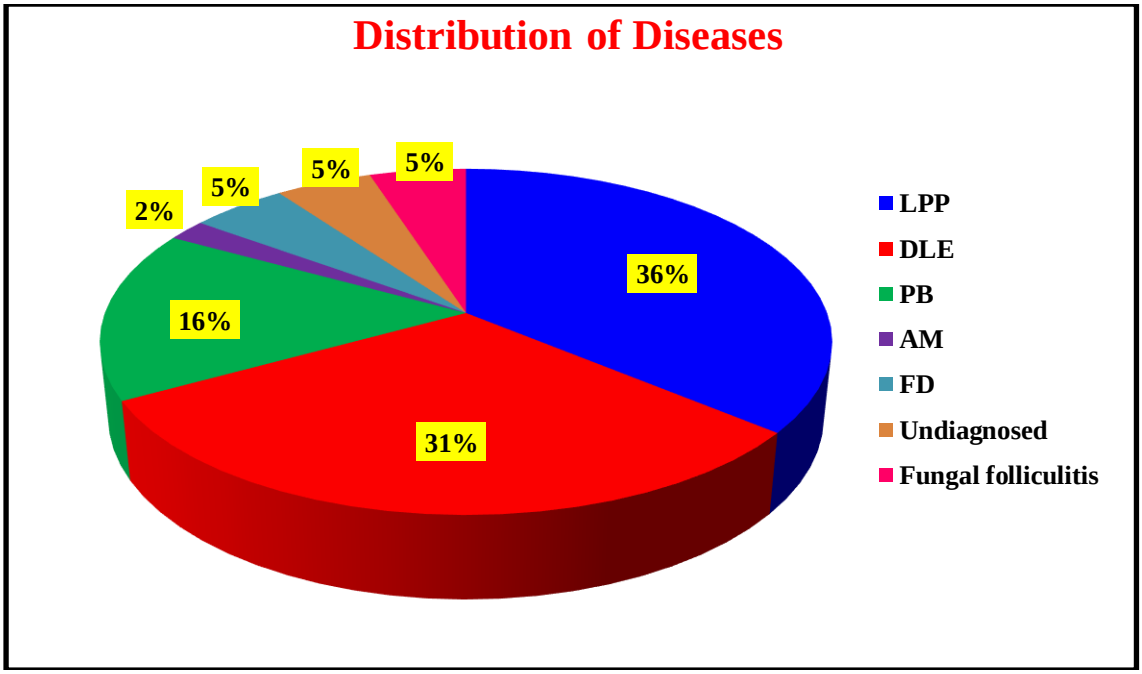

Table No. 3: CLINICAL FINDINGS

Clinical findings	LPP (N=30)	DLE (N=26)	PB (N=14)	AM (N=2)	FD (N=4)	UNDIAG (N=4)	FUNGAL(N=4)
Type Patches and plaques Papules Pustules	 28 (93.4%) 2 (6.6%) 0	 22 (84.6%) 4 (15.4%) 0	 14 (100%) 0 0	 1(50%) 1(50%) 0	 2 (50%) 2 (50%) 0	 4 (100%) 0 0	 2 (50%) 0 2 (50%)
Itching	16 (53.4%)	9 (69.2%)	0	1(50%)	2 (50%)	0	2 (50%)
Tenderness	16 (53.4%)	3 (23.1%)	0	0	2 (50%)	0	2 (50%)
Scaling	20 (66.7%)	20 (76.9%)	0	1(50%)	2 (50%)	0	2 (50%)
Follicular erythema	18 (60.0%)	6 (23.1%)	0	2(100%)	2 (50%)	0	0
Follicular plugging	16 (53.4%)	16 (61.5%)	0	1(50%)	0	0	0

Hyperpigmentation	20 (66.7%)	22 (84.6%)	0	0	0	0	2 (50%)
Atrophy	10 (33.3%)	10 (38.5%)	12 (85.7%)	0	0	4 (100%)	0
Telangiectasia	0	10 (38.5%)	0	0	0	0	0
Crusting	0	0	0	2(100%)	4 (100%)	0	2 (50%)

Table No. 4: HISTOPATHOLOGICAL FIDINGS

Histopathological Findings	**LPP (N=30)**	**DLE (N=26)**	**PB (N=14)**	**AM (N=2)**	**FD (N=4)**	**UNDIAG (N=4)**	**FUNGAL (N=4)**
Epidemal Involvement Hyperkeratosis Acanthosis Epidermal Atrophy	20 (66.7%) 8(26.7%) 8(26.7%)	22(84.6%) 18(69.2%) 6(23.1%)	10(71.4%) 0(0%) 4(28.6%)	0(0%) 0(0%) 0(0%)	4(100%) 2(50%) 0(0%)	4(100%) 4(100%) 0(0%)	4(100%) 2(50%) 0(0%)
Pigment incontinence	26 (86.7%)	8 (30.8%)	0	0	0	2 (50%)	2 (50%)
Follicular plugging	6(20%)	16(61.5%)	0	0	0	0	2(50%)
Vacuolar degeneration	14(46.7%)	22(84.6%)	0	0	0	0	2(50%)
Composition Lymphocytic Mixed No inflame	30(100%) 0 0	26(100%) 0 0	10(71.4%) 0 4(28.6%)	2(100%) 0 0	0 4(100%) 0	4(100%) 0 0	1(25%) 3(75%) 0
Perifollicular location Infundibulum and isthmus Deep follicle	28(93.4%) 0	22(84.6%) 0	10(71.4%) 0	2(100%) 0	2(50%) 2(50%)	4(100%) 0	3(75%) 1(25%)

Table No. 5: Correlation between clinical histopathological and final diagnosis. N=84

FINAL DIAGNOSIS (combined clinical & histological findings)	HISTOPATHOLOGICAL DIAGNOSIS	CLINICAL DIAGNOSIS
LPP (30)	26/30 (86.6%)	18/30 DEFINITE 6 LPP/DLE 6 LPP/PB
DLE (26)	22/26 (84.6%)	12/26 DEFINITE 10 LPP/DLE 4 LPP/PB
PB (14)	14/14 (100%)	8/14 DEFINITE 4 LPP/PB 4 LPP/DLE
AM (2)	2/2 (100%)	1/2 DEFINITE 1 AM/FD
FD (4)	4/4 (100%)	2/4 DEFINITE 2 FD/Fungal folliculitis
UNDIAG (4)	2/4 (50%)	2 PB

		2 LPP
Fungal Folliculitis (4)	4/4 (100%)	2/4 DEFINITE 2 FD/Fungal folliculitis

FIGURES: CLINICAL AND HISTO-PATHOLOGICAL

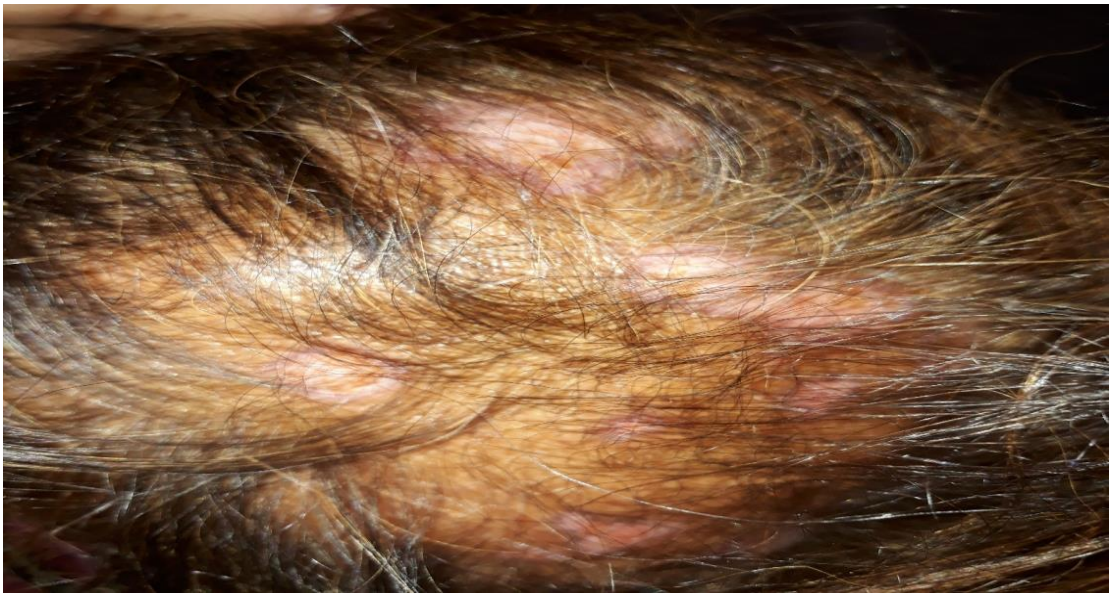

Fig 1- Scalp DLE, showing thin atrophic skin with white atrophic plaque with loss of follicular ostia.

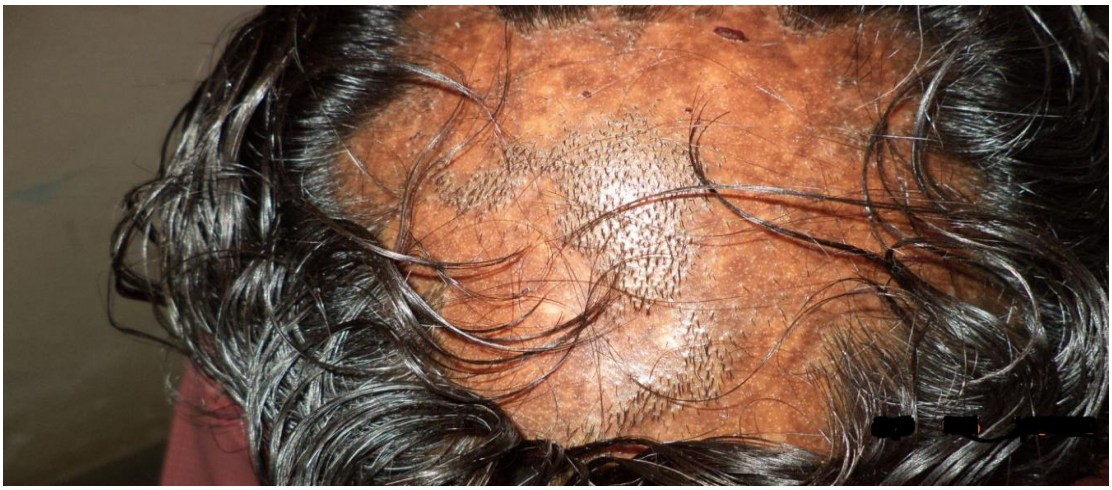

Fig 2-Patch of LPP, scarred dyspigmentaion violaceous skin with few areas of loss of follicular ostia.

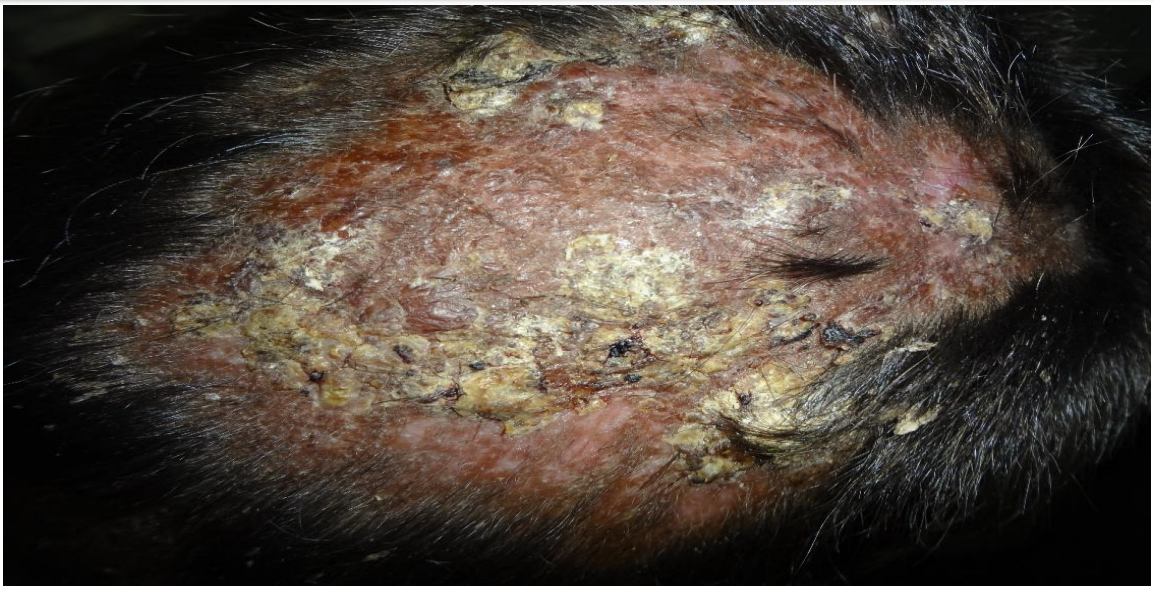

Fig 3-Folliculitis Decalvans showing scarred scalp with yellowish, crusted pustules at the periphery with matted and tufted hairs in the centre of plaque.

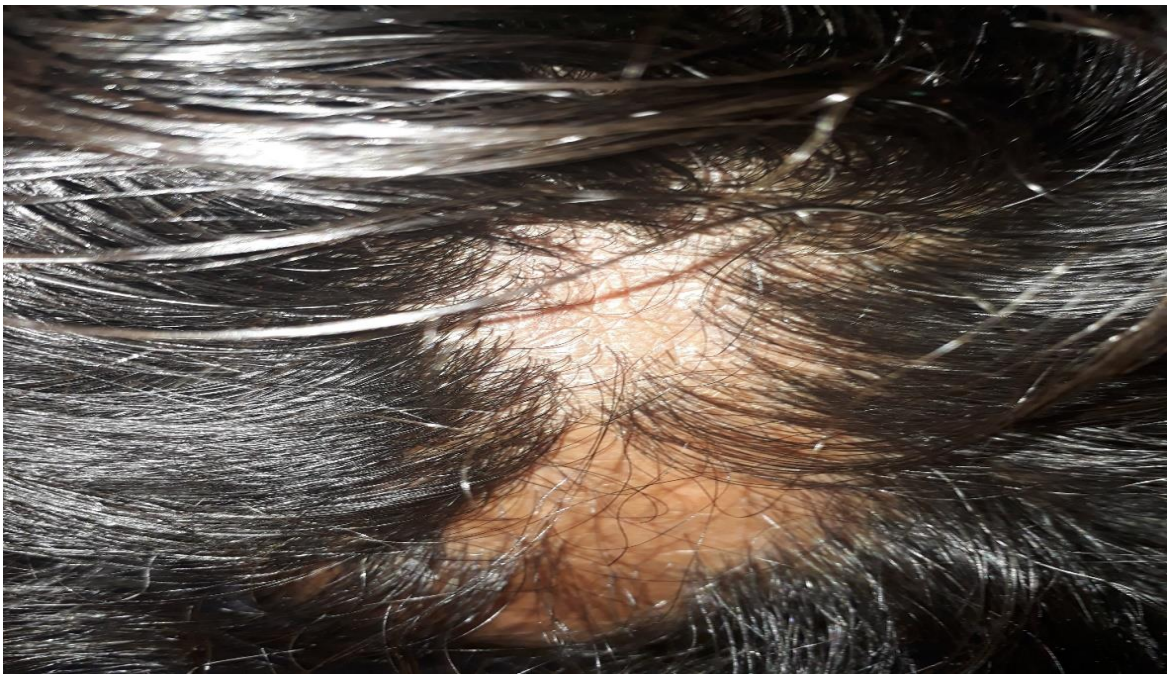

Fig 4 -Multifocal, skin-colored plaques of pseudopelade of Brocq with complete loss of follicular ostia and no signs of inflammation

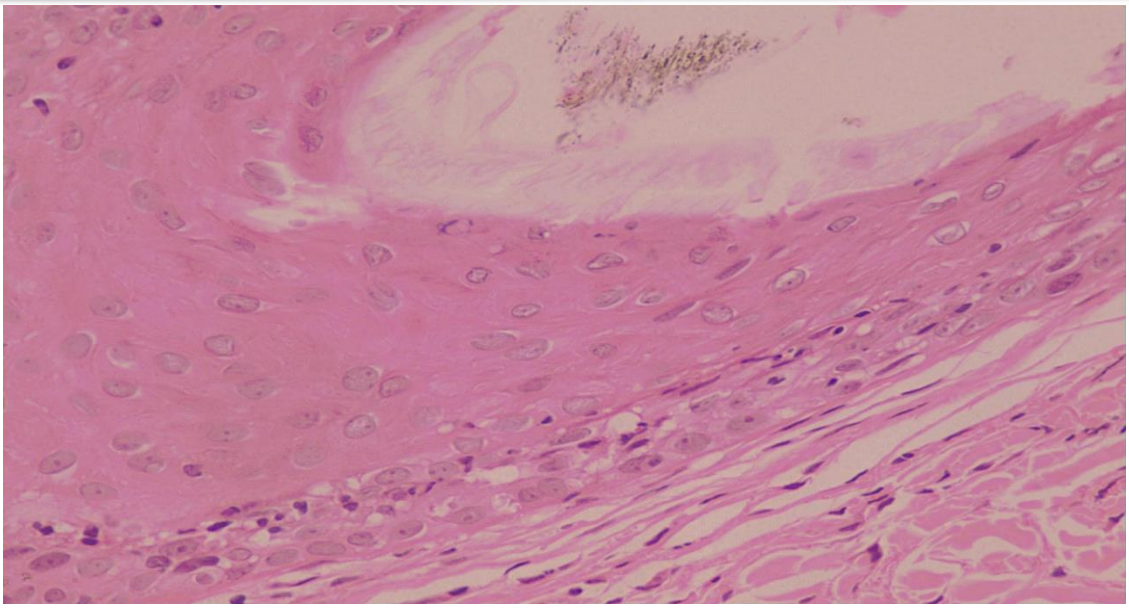

Fig 5 -A case of lichen planopilaris. Hair follicle infiltrated by lymphocytes (seen in lower half of the image). H&E 40X

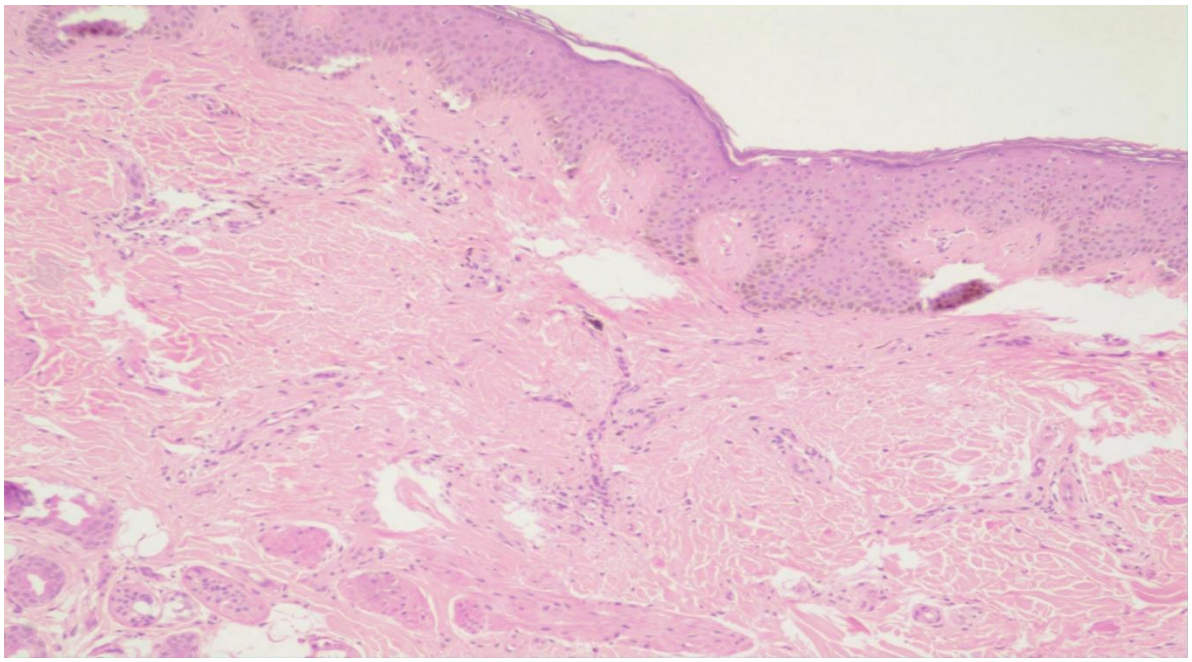

Fig 6 -Cicatricial alopecia. The image shows a vertical fibrotic band in the centre, with isolated Arrector pili muscle, without accompanying pilosebaceous units. H&E 40X

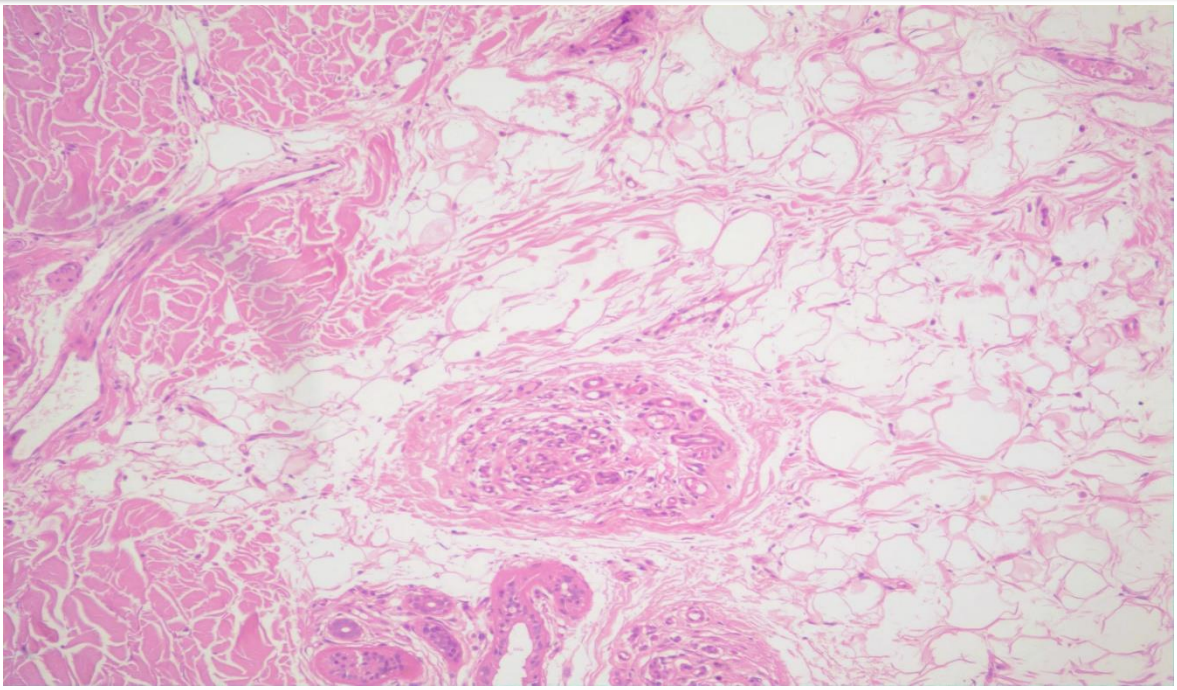

Fig 7 -scalp biopsy of cicatricial alopecia shows hair follicles in deep dermal-subcutis junction replaced by fibrovascular areas. H&E 40X

Histopathological findings:

Inflammatory infiltrate was composed of mainly lymphocytes in 90.5% (76) of total cases which included all the cases of DLE and LPP and 71.4% cases of Pseudopelade. Both the cases of folliculitis decalvans showed mixed inflammation composed mainly of neutrophils, lymphocytes and plasma cells. Mostly, 84.6% cases of DLE and 93.4% of cases of LPP lymphocytic infiltrate present at the dermo-epidermal junction surrounding the follicular infundibulum and isthmus. It was also seen in 71.4% cases of Pseudopelade and 2 cases each of folliculitis decalvans and fungal folliculitis. Deep follicular involvement was seen in 1 case each of FD but it was not found to be statistically significant (Table 4). By clinical examination the cases showed marked overlapping features. 18 cases of LPP could be correctly diagnosed on clinical examination where as 6 cases of LPP showed overlapping features with DLE and 6 presented with end stage features and resembled Pseudopelade clinically. DLE was diagnosed definitely by clinical examination in 12 cases whereas in rest all cases definite diagnosis was not possible as there were overlapping features with LPP. On clinical examination, Pseudopelade could be definitely diagnosed in majority of the cases. Only, 4 cases presenting within one year of onset showed some features of activity with mild erythema and clinically LPP was also kept as differential diagnosis. Folliculitis decalvans was diagnosed correctly in 2 cases when presented early with features of papules and pustules While, other 2 cases showed overlapping features with fungal folliculitis (Table 5). By histopathological examination, majority of cases (90.47%) were correctly diagnosed. Histopathology alone correctly diagnosed 86.6% cases of LPP and 84.6% cases of DLE. All the cases of Pseudopelade, folliculitis decalvans and fungal folliculitis were correctly diagnosed. The histopathology shows all features of end stage scarring alopecia, with concentric perifollicullar fibrosis, no sebaceous glands, loss of follicular units and minimal residual inflammatory cell infiltrate (figure-5, 6, and 7).

DISCUSSION:

There is paucity of data on cicatricial alopecia worldwide. Limited studies are available regarding comparative clinical, histopathological and immunological findings of cicatricial alopecia in past. Epidemiology of cicatricial alopecia is largely unknown with only two retrospective studies available worldwide with no study done so far in the Indian context. The aim of conducting this study was to evaluate the clinical characteristics, histopathological findings in patients with cicatricial alopecia in Western Rajasthan. In our study, there were 69% females and 31% males and this was in accordance with most of the studies in western literature [12, 13, 21, 23]. Whereas, in some studies, the male female ratio was comparatively high[12-13]. In terms of age group, cicatricial alopecia was reported in 10-30 years as compared to 31-40 years in most western studies [12-15, 19]. Only 5 children (11.9%) had cicatricial alopecia with female to male ratio of 1.5:1 similar to adults. In this context, few studies reported only 2-4% pediatrics cases [12-13, 15]. There is no study available from Indian subcontinent illustrating the epidemiology except a study on lichen planopilaris demonstrating that prevalence in children is more than west. We found that LPP contributed to 40% cases in children

followed by 20% each of Pseudopelade, folliculitis decalvans and fungal folliculitis. We found that lymphocyte mediated group constituted 85.7% of all the cases after categorizing the cases into various groups according to NAHRS classification[11]. This was slightly higher as compared to study by Whiting[13] and Tan et al[14] where this group comprised 77.1% and 80.3% cases respectively. In lymphocyte mediated group, maximum number of patients (35.7%) belonged to LPP. This was in accordance to study by Amato et al which concluded it as 33.3%[15]. This was unlike the observations in other studies by Tan et al[13]. This may be due to the fact that end stage LPP is categorized as secondary pseudopelade in these studies. In the present study DLE comprised 31% of total cases, which was in accordance with studies by Tan et al[13] and Moure et al[16] where they reported it as 33.9% and 37.2% respectively.

In present study, Pseudopelade comprised 16.7% of total cases while, various studies communicated that number of cases of Pseudopelade varies from 10% to 33%. [11-14, 22, 25].

Folliculitis decalvans was diagnosed in 4.76% cases. This was in accordance with study by Moure et al where they reported Folliculitis decalvans in 6.7% cases[16]. In present study, all the case of Pseudopelade, majority of patients of LPP and 84.6% cases of DLE presented with patches and plaques. The present finding was supported by various studies. [13,14,22,25].

Histopathological findings

Hyperkeratosis was present in 84.6% cases of DLE which is in accordance with studies by Moure et al[16], Fabbri et al[26]. Two-third of cases with LPP showed hyperkeratosis which is consistent with previous studies.[20, 23-26]. It was also seen in 71.4% cases of Pseudopelade. There is no statistical significance between various groups and HK.

In our study epidermal atrophy was noticed in 69.2% cases of DLE which was consistent with observations of Fabbri et al[26] and Annessi et al[27]. Only, 26.7% cases of LPP reported epidermal atrophy while, in the biopsy in Pseudopelade group 28.2% cases had epidermal atrophy which is in accordance with previous studies[13, 14].

Additionally, all the cases of DLE, LPP and majority of cases of Pseudopelade highlighted lymphocytic infiltrate. Whereas, Neutrophilic and mixed infiltrate was seen in folliculitis decalvans.

Among the lymphocyte mediated group, 93.4% cases of LPP showed lichenoid infiltrate involving the isthmus and infundibulum only. One-fifth of cases showed band like infiltrate at dermo-epidermal junction. This is consistent with findings of Moure et al[16], Annessi et al[27] and Tandon et al[22]. In cases of DLE, variably dense lymphocytic infiltrate mixed with plasma cells in 15.4% was seen around superficial part of follicle in 84.6% which is in context with previous studies[13, 14, 20-22]. Only 15.4% cases of DLE showed complete loss of hair follicle with diffuse dense fibrosis. Furthermore, in 71.4% cases of Pseudopelade there was mild lymphocytic infiltrate around isthmus and infundibulum. While, some studies reported that it varies from 0-64%[21-27]. This could be contributed to varying duration of presentation of patients with minimal inflammation in end stages. Moreover, all the cases of folliculitis decalvans showed neutrophilic infiltrate with mixed inflammation in one case. This has been reported in previous study by Whiting[13], Tan et al[14] and Otberg et al[28].

Correlation of clinical, histological and final diagnosis

By clinical examination the cases showed marked overlapping features. Only 18 cases of LPP were correctly diagnosed on clinical examination whereas, 6 cases of LPP showed overlapping features with DLE and 6 presented with end stage features and resembled Psedopelade clinically. DLE was diagnosed definitely by clinical examination in 12 cases while, in rest all cases definite diagnosis was not possible as there was overlapping features with LPP. Additionally, Pseudopelade could be definitely diagnosed in majority of the cases on the basis of clinical examination. Four cases presenting within one year of onset showed some features of activity with mild erythema and clinically LPP was also kept as differential diagnosis.

Moreover, Folliculitis decalvans was diagnosed correctly in two cases when presented early with features of papules and pustules. Other two cases showed overlapping features with LPP as the patient presented with atrophic patches with papules. This is consistent with findings of Otberg et al[28]and Powell et al[29]. Furthermore, in mostly cases (90.5%) accurate diagnosis was done by histopathological examination. On histopathology basis, correctly diagnosed cases of LPP and DLE were 86.6% and 84.6% respectively. This is consistent with findings of Trachsler and Trueb[30] and Moure et al[16]. All the cases of Pseudopelade, folliculitis decalvans and fungal folliculitis were correctly diagnosed, in this context, Trachsler and Trueb[30] also reported that the accuracy to diagnosis was 97% on histopathology ground.

Limitation: The present study was conducted on small same size with lack of dermatoscopy and trichoscopic findings, lack of immunoflourence study and other special stains.

CONCLUSION: -

The primary scarring alopecia is diagnostic challenge,

which is faced by the clinician and pathologist. Overlapping clinical features are there because of heterogeneous group. Cicatricial alopecia is a trichological emergency, hence required meticulous management to prevent further complications and follicular destruction. The study, recommended that scalp biopsy should be done in each case from an active lesion. Furthermore, multiple sections should be analyzed in each case. The histological features vary drastically with stage of the disease hence, should be supplemented with special stains PAS, Methyl Trichrome and VVG. DIF has both supplemental and diagnostic role in Lymphocytic group especially in histologically suspected cases. Moreover, for a prompt and accurate diagnosis, combined clinico-pathological and immunologic approaches are needed.

REFERENCES: -

1. Sellheyer K, Bergfeld WF. Histopathological evaluation of alopecias. Am J Dermatopatohology. 2006; 28:236-59.
2. Shapiro J. Cicatricial (scarring) alopecias. In: Shapiro J, editor. Hair loss: principles of diagnosis and management of alopecia. London: Martin Dunitz; 2002. pp. 155-74.
3. Bergfeld WF, Elston DM. Cicatricial alopecia. In: Olsen E, editor. Disorders of hair growth: diagnosis and treatment. 2nd Ed. New York: McGraw-Hill; 2003. pp. 363-98.
4. Fatemi-Naeini F, Mokhtari Fatemeh, Saber Mina, Basiri Akram, Matin Marzieh. An Epidemiological Study of 198 Cases of Primary Cicatricial Alopecia in Iran British Journal of Medicine & Medical Research 2017;20(4): 1-6.
5. Templeton SF, Solomon AR. Scarring alopecia: a classification based on microscopic criteria. J Cutan Pathol 1994; 21:97-109.
6. Braun-Falco O, Imai S, Schmoeckel C. Pseudopelade of Broq. Dermatologica 1986; 172:18-23.
7. Scribner MD. Folliculitis devalvans. Arch Dermatol 1971; 104:451-452.
8. SornaKumar L, Shanmuga Sekar C, Vignesh S. Correlation between clinical, histopathological and direct immunoflourescence findings in cases of cicatricial alopecias. Int J Res Dermatol 2016;2:99-102.
9. Sperling LC, Solomon AR, Whiting DA. A new look at scarring alopecia. Arch Dermatol 2000; 1336:235-42.
10. Newton RC, Hebert AA, Freese TW, Solomon AR. Scarring alopecia. Dermatol Clin 1987; 5:603-18.
11. Headington JT. Cicatricial alopecia. Dermatol Clin 1996; 14:773-82.
12. Olsen E, Bergfeld W, Cotsarlis G, Price V, Shapiro J, Sinclair R, et al. Summary of North American Hair Research Society (NAHRS)-sponsored workshop in cicatricial alopecia, Duke University Medical Center. J Am Acad Dermatol 2003; 48:103-10.
13. Whiting DA. Cicatricial alopecia: clinico-pathological findings and treatment. Clin Dermatol 2001; 19:211-25.
14. Tan E, Martinka M, Ball N, Shapiro J. Primary cicatricial alopecias: clinocopathology of 112 cases. J Am Acad Dermatol 2004; 50:25-32.
15. Amato L, Mei S, Massi D, Gallerani I, Fabbri P. Cicatricial alopecia; a dermatopathologic and immunopathologic study of 33 patients (pseudopelade of Brocq is not a specific clinico-pathologic entity). Int J Dermatol. 2002;41(1):8-15. doi: 10.1046/j.1365-4362.2002.01331.x. PMID: 11895507.
16. Moure ER, Romiti R, Machado MC, Valente NY. Primary cicatricial alopecias: a review of histopathologic findings in 38 patients from a clinical university hospital in Sao Paulo, Brazil. Clinics (Sao Paulo). 2008;63(6):747-752. doi:10.1590/s1807-59322008000600007
17. Yell JA, Mbuagbaw J, Burge SM. Cutaneous manifestations of systemic lupus erythematosus. Br J Dermatol 1996; 135:355-62.
18. Sperling LC. Chronic cutaneous lupus erythematosus (discoid lupus erythematosus). In: Sperling LC, editor. An atlas of hair pathology with clinical correlations. New York: Parthenon Publishing Group; 2003. pp. 109-14.
19. Mehregan AH. Histopathology of alopecias. Cutis 1978; 21:249-53.
20. Sharma R, Maheshwari V. Childhood lichen planus: a report of fifty cases. Pediatr Dermatol 1999; 16:345-8.
21. Nayar M, Schomberg K, Dawber RP, Millard PR. A clinicopathological study of scarring alopecia. Br J Dermatol 1993; 128:533-6.
22. Tandon YK, Somani N, Cevasco NC, Bergfeld WF. A histologic review of 27 patients with lichen planopilaris. J Am Acad Dermatol 2008; 59:91-8.
23. J. Postmenopausal frontal fibrosing alopecia. Br J Dematol 1999; 140:1181-2.
24. Crickx B, Blanchet P, Grossin M, Belaich S. Lassueur-Graham-little syndrome. 2 cases. Ann Dematol Venereol 1990; 117:907-9.
25. Sperling LC. Brocq's alopecia (pseudopelade of Brocq) and 'burnt out' scarring alopecia. In: Sperling LC, editor. An atlas of hair pathology with clinical correlations. New

York: Parthenon Publishing Group; 2003. pp. 115-8.
26. Fabbri P, Cardinali C, Giomi B, Caproni M. Cutaneous lupus erythematosus: Diagnosis and management. Am J Clin Dermatol 2003; 4(7): 449-65.
27. Annessi G, Lombardo G, Gobello T, Puddu P. A clinicopathologic study of scarring alopecia due to lichen planus: comparison with scarring alopecia in discoid lupus erythematosus and pseudopelade. Am J Dermatopathol. 1999:21:324–31.
28. Ortega del Olmo R, Aneiros-Cachaza J, Fernández-Pugnaire MA, Buendía Eximan A, Serrano Ortega S. Alopecia frontal postmenopáusica. Piel 2002;17:160-4.
29. Powell JJ, Dawber RPR, Gatter K. Folliculitis Decalvans including tufted folliculitis: Clinical, histological and therapeutic findings. Br J Dermatol 1999; 140:328-33.
30. Trachsler S, Trueb RM. Value of direct immunofluorescence for differential diagnosis of cicatricial alopecia. Dermatology. 2005;211:98–102.

