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Original Article

Scarring alopecia in patients with discoid lupus erythematosus versus patients with follicular lichen planus- A comparative study of clinicopathological features

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Abstract

Objective To do full clinical evaluation of follicular lichen planus (lichen planopilaris) and discoid lupus erythematosus.

Methods This cross-sectional comparative work where 100 cases of scarring alopecia of scalp were included during the period from 2014-2022. Full clinical assessment including history and examination were carried out. Confirmatory blood testing and skin biopsies were done accordingly.

Results One hundred case of scarring alopecia were included in this study and subdivided in to two groups; Discoid lupus erythematosus group: included 43 cases. The pattern of hair loss was moth eaten in 10 (23.2%) cases especially in early cases. There was no well demarcated active border as the activity of the disease was almost uniform all over the patches. Follicular lichen planus group: consisted of 57 patients. The course of the disease was chronic but ranged from 4 months to three years. The pattern of hair loss was follicular moth-eaten alopecia in all cases and the clinical picture started as small moth-eaten patches and over time these small areas of alopecia were coalesced together to form large patches simulating pseudo Palade pattern of alopecia.

Conclusion Both follicular lichen planus and discoid lupus erythematosus are common causes of scarring alopecia affecting young people more in females in DLE but more in males in follicular lichen planus. The moth eaten alopecia was in all cases in patients with LPP while was detected in 23.2% of DLE patients. The prognosis of hair growth is more favorable in DLE than FLP.

Key words
Discoid lupus erythematosus; Follicular lichen planus; Lichen planopilaris; Scarring alopecia; Moth eaten alopecia.

Introduction

Scarring hair loss denotes to a group of hair loss diseases where the follicles of hair is permanently damaged and replaced by fibrosis.[1] The term "scarring" indicates that the epithelium of hair follicles has been replaced by fibrous tissue. The broadest definition of cicatricial hair loss could encompass all types of alopecia resulting in permanent hair follicle loss.[2]

Epidemiology studies on scarring alopecia are primarily conducted in clinics involved to alopecia. The prevalence of scarring alopecia within the alopecia population is estimated to be around 7%. The relative incidence of different types of scarring hair loss of scalp differs in variable studies and is mostly depending on the clinic's population. Large race-based studies are not yet available, but certain patterns are observed, such as central centrifugal cicatricial alopecia being more common in African Americans, while lichen planopilaris is more prevalent in lighter-skinned patients. Scarring

alopecia is commonly believed to affect females more than males. Most case reports indicate that these conditions tend to occur in individuals older than 20 years.[3]

Cicatricial alopecia, also known as scarring alopecia, occurs when the stem cells in the bulge area of the follicles of hair sustain irreversible damage, usually due to inflammatory mechanisms, such as those seen in autoimmune diseases. In primary cicatricial alopecia (PCA), the immune system targets the hair follicle itself. This category of permanent loss of hair conditions can be divided into different groups based on the main type of inflammatory cells surrounding the hair follicles. The exact reasons behind the infiltration of these cells around the hair follicles in PCA remain unclear. As a result, it is challenging to halt or reverse the inflammation in PCA. Nevertheless, emerging finding indicates of stem cells of healthy hair follicles are relatively protected from attacks of inflammatory reactions by residing in an immunologically privileged environment. In PCA, this protection might lost, posing a significant obstacle in research. The primary goal in studying PCA is to find specific signaling pathways that either jeopardize or restore the relative immune protection of those stem cells.[4]

Primary scarring hair loss are characterized by inflammation that affects the upper, permanent segment of the hair follicles, known as the infundibulum, and the isthmus located below it.

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The isthmus is the region where pluripotent hair stem cells reside, and these stem cells are also located in the bulge area where the arrector pili muscle connects to the outer root sheath.[5]

Pluripotent stem cells of hair follicles play a vital role in renewing the upper segment of the hair follicles and sebaceous gland. They are responsible for restoring the lower cyclical segment of the follicles during the start of a new growth period.[5] When the bulge areas and the sebaceous glands within the isthmus are damaged, either the stem cell or sebaceous gland might be affected. As a result, an incomplete hair cycle can occur, and this disruption can lead to chronic follicular inflammation and a foreign body reaction. Ultimately, scarring alopecia becomes the consequence of this process.[5]

Scarring alopecia is a complex condition with several contributing factors, but two of the most significant ones are follicular lichen planus and discoid lupus erythematosus.

LPP also known as follicular lichen planus (FLP), is a chronic inflammatory disease that leads to the gradual scarring alopecia of the scalp. It is characterized by the loss of hair follicles due to lymphocytic inflammation. While the exact cause is not fully understood, autoimmune pathology is currently the most widely accepted theory.[6] LPP predominantly affects women and is more common in Caucasians than in individuals with darker skin tones. Only a minority of patients, fewer than 30%, develops lichen planus lesions on the glabrous skin, mucous membranes, or nail changes typically associated with lichen planus. The cause of LPP remains unknown, but it is presumed to be related to the etiology of lichen planus.[2]

The presentation of hair loss in LPP can vary significantly, with an insidious or rapid course.

Patients with mild scalp disease may not experience symptoms, while others may have pruritus (itchiness) and tenderness. Typically, there are scattered areas of partial hair loss with perifollicular erythema (redness around hair follicles), follicular spines, and scarring. The type of hair loss can resemble central centrifugal scarring alopecia or Brocq's alopecia. In early LPP lesions, a dense, band-like perifollicular lymphocytic infiltrate is observed at the level of the infundibulum and isthmus, where the hair "bulge" (containing hair follicle stem cells) is found. Inferior segment of the hair follicles is initially spared. Other common features include vacuolar changes in the basal layer of the outer root sheath and the presence of necrotic keratinocytes. Orthokeratosis (thickening of the outer layer of the skin) and follicular plugging are also often seen. In some cases, both the interfollicular epidermis and hair follicles are affected simultaneously. As the disease progresses, perifollicular fibrosis and epithelial atrophy become characteristic findings at the infundibulum and isthmus level. Damage to the hair bulge, where hair follicle stem cells reside, leads to permanent scarring alopecia. In advanced cases, fibrotic tracts containing degenerated elastic fibers replace the destroyed hair follicles, resulting in a complete lack of visible hair follicles, which has been referred to as pseudo pelade of Brocq by some researchers.[7]

Distinguishing between LPP and DLE can be challenging due to their similarities. However, several clinical and histopathological characteristics can aid dermatologists in differentiating these primary lymphocytic cicatricial conditions, as reported by Sharquie *et al*. in the Iraqi population:[8]

- Early Lesions: LPP presents with multiple pigmented follicular papules, while scalp DLE exhibits usually single but potentially multiple scaly erythematous patches, especially in advanced cases.
- Process of scarring: LPP displays early and pigmented scarring, whereas DLE scarring is typically late and erythematous. Irregular scarring areas resembling moth-eaten alopecia are distinctive features of LPP, setting it apart from DLE.
- Disease Activity: DLE lesions are mainly centralized, whereas LPP shows activity mainly at the periphery of the scarring areas in the form of hyperkeratotic follicular papules.
- The associated features: additional body sites affected by the condition can provide diagnostic clues. DLE commonly affects sun-exposed areas like nose, cheeks and ears with typical discoid rash. Conversely, LPP rash often present as asymptomatic follicular papules of trunk, which may or may not be associated with scarring.
- Histopathological differences: Both conditions show lichenoid lymphocytic infiltration of the basal epithelium and follicular structures with vacuolar interface dermatitis. However, DLE lesions display deeper follicular inflammation and perieccrine lymphocytic infiltration, while the perifollicular inflammation in LPP is more superficial and confined to the upper hair follicle area.
- Pathologic Changes: Active LPP primarily affects the hair follicles without involving the epidermis, whereas DLE exhibits changes in both the epidermal layer (vacuolar interface dermatitis) and the dermal layer with interfollicular mucin deposition.
- Bulge Region Involvement: In LPP, the process of inflammation targets the bulge areaa, causing fibrosis while sparing the lower area of the hair follicle. This finding supports the theory that the bulge site is essential for hair regeneration and may contribute to the permanent damage to the hair organ.

Basement Membrane thickening and Deposition of mucin: DLE is characterized by basement membrane thickening and deposition of mucin in the dermal layer.

Direct immunofluorescence (DIF) is typically specific for DLE, showing a positive lupus band (IgG, IgM, C3) in 63% to 100% of cases. Conversely, in LPP, the lupus band is negative. Additionally, unlike DLE, treatment with hydroxychloroquine is ineffective.[8]

DLE is a common cutaneous features of SLE. New population-based reports estimate that it can occur in around 12.4% to 15.0% of incident SLE patients and in around 16.6% to 24.3% of prevalent patients. DLE is more prevalent among black patients with SLE. The condition can lead to significant morbidity, as it often affects the scalp, face and ears, and is associated with scarring and permanent hair loss. It is worth noting that DLE might also occur independently of SLE, referred to as primary DLE.[9]

DLE lesions can either be the only manifestation of lupus or occur alongside other forms of cutaneous lupus, such as lupus panniculitis, and even in the context of systemic lupus. While DLE is commonly seen in adults, most cases with DLE do not have systemic lupus. About 50% of patients with only skin involvement will have scalp lesions, and among those with DLE confined to the scalp, only a few will develop systemic lupus.[2]

The clinical picture of lesions of scalp in DLE may simulate classic discoid rash found in other areas of the body, presenting with erythema, epidermal atrophy, and dilated, plugged hair follicles, which can contribute to hair loss. Dark-skinned individuals may show central hypopigmentation and peripheral hyperpigmentation in these lesions.

However, the distribution and degree of clinical inflammation can vary among cases, leading to a resemblance to other conditions such as alopecia areata, lichen planopilaris, linear morphea, central centrifugal cicatricial alopecia, or Brocq's alopecia. While itching or tenderness is commonly reported, some individuals with DLE may remain asymptomatic.[2]

In the early phases, typical scalp lesions appear as well-circumscribed, erythematous patches with adherent follicular hyperkeratosis, and sometimes dilated hair follicles plugged with keratinous debris. As the lesion progresses, it transforms into a sclero-atrophic, smooth, depressed, white-ivory plaque. DLE scarring alopecia (DLESA) could occur solely on the scalp or be associated with DLE lesions in other areas of the body.[10]

The epidermis in DLE shows signs of atrophy, vacuolar alteration, and an interface dermatitis. There is a superficial infiltrate consisting primarily of lymphocytes, along with prominent pigment incontinence. Hyperkeratosis and thickening of the basement membrane zone of the epidermis are also observed. Pigment-laden macrophages are present in the papillary dermis and may be seen in the subcutaneous fat at the sites of previous hair follicles. However, these findings are not specific to DLE and can be seen in other forms of alopecia. The perifollicular infiltrate is associated with eventual scarring and fibrosis at the hair follicle sites.[11]

Sebaceous glands in the affected scalp exhibit a significant reduction in size. The most prominent lymphocytic inflammation around the hair follicle occurs around the mid-follicle, specifically at the level of the sebaceous gland, which appears to be a critical functional level within the hair follicle. Notably, both normal and diseased scalp display changes in the expression of matrix molecules, particularly the

proteoglycans in the connective tissue sheath, and the keratin intermediate filaments in the outer root sheath cells at this level. The loss of a population of mid-follicular stem cells may contribute to the development of scarring alopecia in DLE.[9]

The inflammation observed in DLE primarily affects the bulge areas of the follicles of hair, indicating that damage to the stem cells in this area might contribute to the permanent loss of hair follicles. The involvement of the bulge region is part of a broader impact on hair follicles, but it is not an early event; rather, it is secondarily affected by the surrounding inflammatory cell infiltrate. Follicular stem cells damage could potentially explain the irreversible hair loss and scarring characteristic of DLE.[10]

When making a diagnosis of DLE, it is crucial to conduct screening for SLE. This screening process involves a detailed evaluation, including a medical history, physical checking, and laboratory investigations. These tests encompass blood cell count, renal function, and general urine exam. Serologic and hematological abnormalities may be evident, and in some patients, an elevated ESR may be observed. Additionally, there may be a positive result for rheumatoid factor, while complement levels could be decreased.

If abnormal results are found in kidney function tests and/ or proteinuria, it may indicate the presence of renal involvement. In approximately 20% of DLE patients, a positive antinuclear antibody (ANA) test may be detected when tested with human substrates. Furthermore, up to 20% of DLE patients may have Anti-Ro (SS-A) autoantibodies. In certain cases, anti native deoxyribonucleic acid (DNA, either double-stranded or nDNA) or anti-Sm antibodies may be present in 5-20% of DLE patients. A study has established a link between disease activity in DLE and the levels of ANA, anti-RNP, anti-dsDNA, and anti-ssDNA IgG, using the Cutaneous Lupus Disease Area and Severity Index (CLASI).

The present study aims to evaluate and compare the clinical features of scarring alopecia in patients with FLP and those with DLE in a population representing part of the Middle East countries.

Patients and Methods

This is across sectional comparative clinical and histopathological work conducted at Baghdad Medical City Complex ,the center of dermatology/ Baghdad teaching hospital during the period between 2014-2022 with a total number of 100 patients with scarring alopecia and all demographic features were recorded including personal information, Full clinical assessment including full history and examination were carried out. Confirmatory blood testing and skin biopsies were done accordingly. Formal consent was taken from patients after discussing the nature of the disease and the aim of our study.

In our study, a total of 100 cases of scarring alopecia were included and divided into two groups: Discoid lupus erythematosus and follicular lichen planus. To document the lesions, medical photographs were taken using an Iphone Xs max camera. To assess the severity of scarring, a scoring system for alopecia was employed. The scoring was based on the degree of scarring disease and adapted from estimating the percentage of scalp hair loss. The scalp was divided into four quadrants, and the percentage of the scalp surface occupied by all areas of alopecia was estimated. Based on this estimation, patients were classified into four subgroups: 1-25%, 26-50%, 51-75%, and 76-100% of scalp hair loss. For histopathological

correlation, incisional biopsy of the lesion was performed under local anesthesia. The biopsy samples were vertically sectioned and stained with Hematoxylin-Eosin. This allowed us to correlate the clinical findings with the histopathological features.

All patients with scarring alopecia having follicular lichen planus or discoid lupus erythematosus of all ages and of both sexes were included in the study. Exclusion criteria included other diseases associated with scarring alopecia like chronic folliculitis and encoupe de sabre.

Data analysis was conducted using the Statistical Package for Social Sciences (SPSS) version 26. The dataset included detailed descriptions for each variable, with participant details identified by serial numbers. Data collection occurred on a daily basis. For numerical variables, data was expressed using mean and standard deviation. Categorical variables were presented as frequency and percentage. To assess the differences between the categorical variables under study, chi-square tests or Fisher's exact tests were used alternatively. The significance level was set at a confidence level of 95%, with a P-value of equal to or less than 0.05 considered statistically significant.

Results

One 100 cases of scarring alopecia (56 males and 44 females) were included and subdivided in to two groups:

Patients with discoid lupus erythematosus: This group included 43 cases, 25 patient females (58.14 %) and 18 patient's males (41.86 %) with F:M ratio 1.38: 1 and their ages ranged from 12-60 year **(Table 1)** with a mean and SD 34.6±11.45 year. The duration of the disease ranged from 6 months to five years.

Table 1 Showing the ages distribution of patients with DLE.

	Frequency (n=43)	*Percentage*
10-19 yrs.	1	2.3
20-29 yrs.	11	25.6
30-39 yrs	10	23.3
40-49 yrs.	13	30.2
≥ 50 yrs.	8	18.6

Table 2 Showing the total clinical features of patients with DLE.

Variable	*Frequency (n=43)*	*Percentage*
Pattern of hair loss		
Single patch	17	39.53
Multiple patches	12	27.90
Moth eaten	10	23.26
Marked scarring ≥51 %	4	9.31
Extent of scalp involvement		
1-25%	27	62.8
26-50%	12	27.9
51-75%	3	7
76-100%	1	2.3
Extra cranial involvement		
None	23	53.5
Ear	6	14
Face	10	23.3
Trunk	1	2.3
Extremities	3	7
Nails	0	0
Mucous membrane	0	0
Scarring pattern		
Erythematous	29	67.4
Leukodermic	14	32.6
Pigmented	0	0
ANA		
Positive	4	67
Negative	2	33

All patients presented with scarring alopecia and they were erythematous patches in 67.4% of patients which was single or multiple, some of which is atrophic with complete or partial hair loss while 32.6 % of patients presented with leukodermic atrophic patches. Moth eaten alopecia was seen only in 10 (23.2%) cases **(Table 2)**. There was no well demarcated active border as the activity of the disease was uniform all over the patches. In 20 patients (46.5%) the scalp rash was associated with lupus rash on the face, ears and other parts of the body **(Table 2)**

in which the face was most commonly involved (23.2%) followed by the ears (13.95%). Regarding the scoring of scalp scarring, it was ranged from 1-100% **(Table 2)** in which most patients (62.8%) had scalp involvement from 1-25%. Regarding laboratory test, all patients sent for ANA test, while only 6 cases showed their results **(Table 2)**. Biopsies were taken from both areas of active lesions and late scared lesion in the same patient and showed the following histopathological changes:

The epidermis was mostly atrophic but occasionally acanthotic with compact hyperkeratosis with occasionally parakeratosis. Follicular plugging was well demonstrated in many sections. The basal layer distraction was revealed in many cases together with thickened basement membrane while melanin incontinence with melanophages were well demonstrated. Regarding the dermis, there was marked dense inflammatory infiltrate involving both superficial and deep dermis and this infiltrate were also found in perifollicular, perivascular and periadnexial areas. In addition, this infiltrate was also observed to involve the panniculus forming panniculitis. The result of late atrophic scared areas showed complete epidermal atrophy with liquefactive degeneration of basal layer while the skin appendages including hair follicles were almost completely absent leaving scarring with prominent hypertrophied arrecter pili muscles.

Patients with follicular lichen planus*:* This group consisted of 57 patients, 38 males (66.66%) and 19 females (33.34%) with M:F 2:1, their ages ranged from 10-60 year **(Table 3)** with a mean and SD 37.5±11.41 year. The course of the disease was chronic but ranged from 4 months to three years.

Table 3 Showing the ages distribution of patients with Follicular lichen planus.

Age	*Frequency (n=57)*	*Percentage*
10-19 yrs.	2	3.5
20-29 yrs.	9	15.8
30-39 yrs.	19	33.3
40-49 yrs.	15	26.3
≥ 50 yrs.	12	21.2

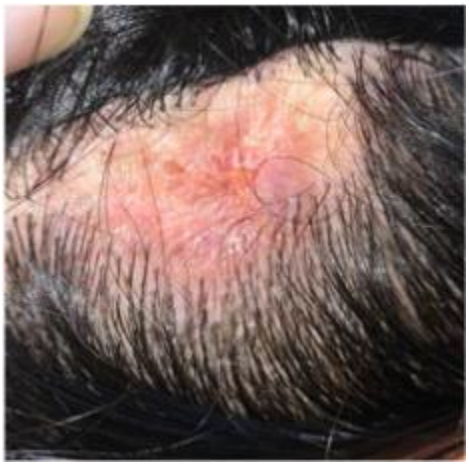

Figure 1 20 years old age male patient with discoid lupus erythematosus of scalp showing single atrophic erythematous patch of scarring alopecia about 1.5 cm diameter with central erosion & crust, & peripheral scales on left temporal area of the scalp.

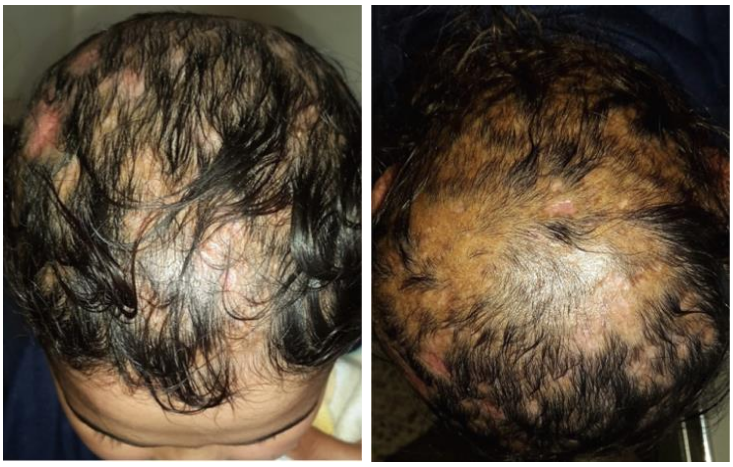

Figure 2 A. 12 years old female patient with discoid lupus erythematosus of scalp showing moth eaten alopecia, B. the same patient

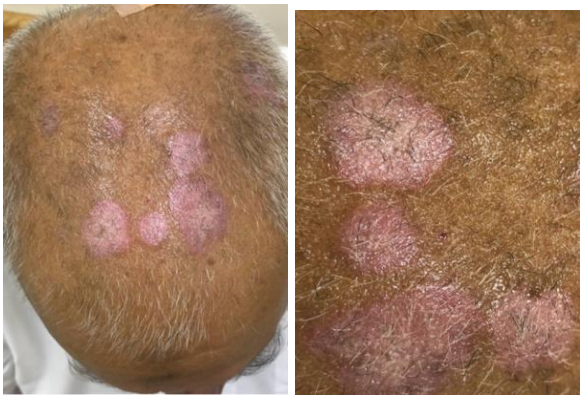

Figure 3 A. 60 years old male patient with discoid lupus erythematosus of scalp showing multiple patches of scarring hair loss with scaly erythematous atrophic center with follicular plugging, B. the same patient.

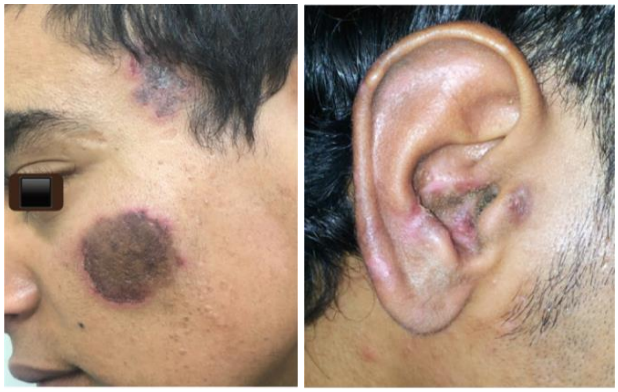

Figure 4 A. 20 years old male patient present with discoid lupus erythematosus of scalp with multiple patches of scarring alopecia associated with extra-cranial involvement including the face. The same patient in (fig5. A) showing ear involvement (B).

All patients presented with scarring alopecia and the pattern of hair loss was follicular moth-eaten alopecia in all cases and the clinical picture started as small moth-eaten patches and over time these small areas of alopecia were coalesced together to form large patches simulating pseudo Palade pattern of alopecia. involved in 2 (3.50%) cases **(Table 4)**. The scoring of scarring of the scalp ranged from 1-100% and the highest rank of severity was 1-25% which constitute 43.88% **(Table 4)**. Regarding laboratory test, all patients sent for ANA test, while only 7 cases showed their results as shown in **Table 4**.

Table 4 Showing the total clinical features of patients withfollicular lichen planus.

Variable	*Frequency N=57*	*Percent %*
Pattern of hair loss		
Single patch	8	14.03
Multiple patches	6	10.52
Moth eaten	30	52.63
Marked scarring ≥51 %	13	22.82
Extent of scalp involvement		
1-25%	25	43.9
26-50%	19	33.3
51-75%	9	15.8
76-100%	4	7
Extra cranial involvement		
None	44	77.2
Ear	0	0
Face	2	3.5
Trunk	5	8.8
Extremities	1	1.8
Nails	3	5.3
Mucous membrane	2	3.5
Scarring type		
Erythematous	0	0
Leukodermic	20	35.1
Pigmented	37	64.9
ANA		
Positive	0	0
Negative	7	100

As many of the patients were seen at the end stage of disease and the patients presented mainly with scarring with few obvious active lesions; all biopsies with their histopathological evaluation showed mainly marked fibrosis and could be summarized as the following:

The epidermis was usually normal with intact basal layer and normal dermo-epidermal junction and the inflammatory infiltrate is mainly perifollicular. While few cases showed epidermal atrophy, hyperkeratosis and follicular plugging. In addition, the very old scared lesions revealed only massive scarring and few residual melanophages. In some cases, the inflammatory reaction and fibrosis was seen to involve the panniculus.

From the current study, we found that discoid

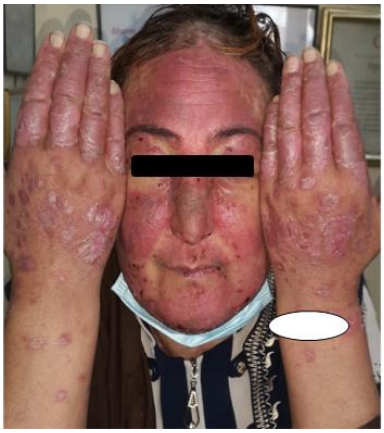

Figure 5 52 years old female patient with discoid lupus erythematosus showing facial and hands involvement in the photosensitive areas.

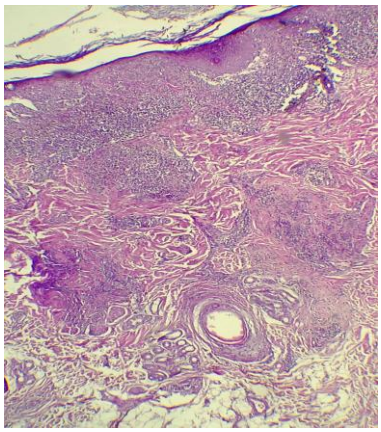

Figure 6 Patient with DLE of scalp with scarring alopecia showing epidermal atrophy, basal layer damage with melanophages and dense band-like inflammatory infiltrated dermo-epidermal junction and in the deep and superficial dermis with perifollicular infiltrate. HE stain X4

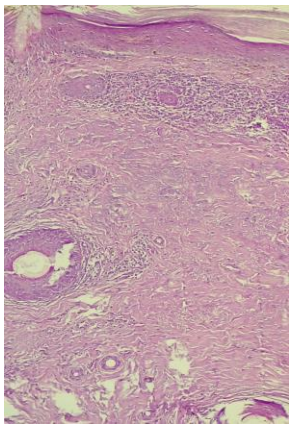

Figure 7 Patient with DLE of scalp with scarring alopecia showing epidermal atrophy, compact hyperkeratosis, band like inflammatory infiltrate at the dermo-epidermal junction and dense fibrosis with loss of skin appendages with hair follicles destruction. HE stain X10

lupus erythematosus was more common in females' patients while follicular lichen planus was more common in males' patients with significant p-value (0.013). Regarding age group, there is no statistically significant differences **(Table 5)**. Moth eaten alopecia also was significantly more in patient with follicular lichen planus than patient with discoid lupus erythematosus with significant p-value (0.000). Regarding extracranial involvement, face and ears were more commonly involved in DLE patients than patients with follicular LP. While nails and mucus membranes are more commonly involved in patients with follicular LP. Patients with DLE presented with erythematous scarring mainly (67.4%) while patients with follicular LP presented mainly with pigmented scarring (64.9%) **(Table 6)**.

Discussion

Scarring alopecia is a major health problem in both sexes but predominantly more important in females as it has a great impact in the cosmetic appearance. There are many etiological factors involved in the etiopathogenesis of the scalp

Table 5 The comparison of DLE and follicular LP regarding socio-demographic features.

Variable	*DLE*		*FLP*		*P value*
	Frequency N=43	*Percent %*	*Frequency N=57*	*Percent %*	
Age group					
10-19 y	1	2.3	2	3.5	0.671[1]
20-29 y	11	25.6	9	15.8	
30-39 y	10	23.3	19	33.3	
40-49 y	13	30.2	15	26.3	
≥50 y	8	18.6	12	21.1	
Gender					
Male	18	41.9	38	66.7	0.013*[1]
Female	25	58.1	19	33.3	

[1]Chi-square test; *Significant result.

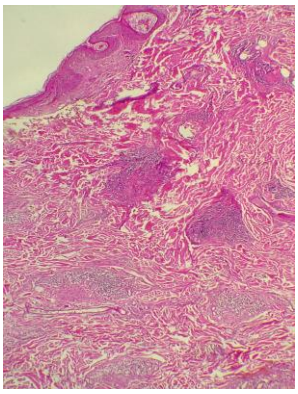

Figure 8 Patient with DLE of scalp with scarring alopecia showing multiple mass-like inflammatory infiltrates in the deep and superficial dermis with hair follicles remnants and dilated blood vessels and each area of inflammatory infiltrates possibly represent a site of a damaged hair follicle. HE stain X4.

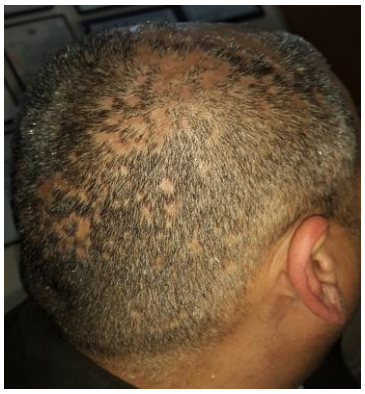

Figure 9 50 years old male patient with follicular lichen planus of scalp showing typical moth eaten alopecia.

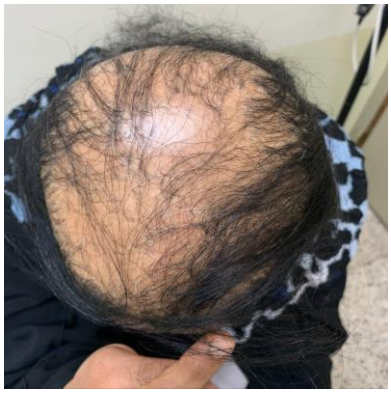

Figure 10 43 years old female patient withfollicular lichen planus of scalp showing wide patch of extensive scarring hair loss.

scaring like dermatophyte infection in children, physical problem like burn, chronic folliculitis specially folliculitis decalvans, inflammatory diseases like DLE, follicular LP, morphea in form of en coup de sabre.[8] The prevalence of scarring alopecia varies in different countries according to the frequency of different diseases like infection , inflammatory skin diseases like discoid lupus erythematosus and follicular LP.[3]

In Iraq, prevalence of hair scarring problem was not evaluated and like many other countries there are also different etiological factors involved in causation of this hair problem. While the present work focused on two important and common causes of scarring mainly DLE and follicular LP.

In this current study, we covered many aspects of DLE and follicular LP as both are a common etiological factors in scarring alopecia in Iraq. Also, we have performed epidemiological, clinical and histopathological comparison.

In the present study, we have 43 patients of DLE with scarring alopecia with a mean and SD of their ages was 34.6±11.45 year and the most common age group was (40-49 years) which constituted 30.2% followed by age group (20-29 years) which was 25.6%. The study of PFabbri *et al.* was found that age mean was 45.6 year[10] while the study of Yirong Xiang *et al.* found that the majority of patients were in their 30s.[14]

In the current work, the distribution of DLE patients according to sex, female patients was more common and constituted (58.14%) on comparison with male patients which was (41.86%) with F:M ratio 1.38:1. HyeJin Chung *et al.* also found that women predominated over men, with a ratio of 3.4:1.[15] While Josef Symon *et al.* considered that there was an equal parts of both sexes in terms of the tendency to get scarring alopecia from DLE.[16]

Regarding clinical manifestations in DLE group, in our study 39.53% of patients presented with single patch of scarring alopecia, 9.31% present with diffuse scarring alopecia, 23.2% with moth eaten alopecia and 27.9% revealed multiple patches of scarring alopecia. While The study of P Fabbri *et al.* which included 36 patients affected by DLE of scalp showing that (52.7%)

Table 6 The clinical features in patient with discoid lupus erythematosus on comparison with patients with follicular lichen planus.

Variable	*DLE*		*FLP*		*P value*
	Frequency N=43	*Percent %*	*Frequency N=57*	*Percent %*	
Pattern of hair loss					
Single patch	17	39.53	8	14.03	
Multiple patches	12	27.90	6	10.52	
Moth eaten	10	23.2	30	52.63	0.000*[1]
Marked scarring ≥51 %	4	9.31	13	22.82	
Scoring of scalp scarring					
1-25%	27	62.8	25	43.9	
26-50%	12	27.9	19	33.3	0.221[2]
51-75%	3	7	9	15.8	
76-100%	1	2.3	4	7	
Extracranial involvement					
None	23	53.5	44	77.2	
Ear	6	14	0	0	
Face	10	23.3	2	3.5	
Trunk	1	2.3	5	8.8	0.000*[2]
Extremities	3	7	1	1.8	
Nails	0	0	3	5.3	
Mucous membrane	0	0	2	3.5	
Scarring type					
Erythematous	29	67.4	0	0	
Leucodermic	14	32.6	20	35.1	0.000*[1]
Pigmented	0	0	37	64.9	
ANA					
Positive	4	67	0	0	0.029*[2]
Negative	2	33	7	100	

[1]Chi-square test; [2]Fisher's exact test; *Significant result

of patients have multiple patches of scarring alopecia, (33.3%) with a single lesion of scarring alopecia and (13.8%) with a scarring alopecia mimicking moth eaten alopecia.[9]

In the present work, there was no well demarcated active border of lesions as the activity of the disease was uniform all over the patches and these patients showed erythematous patches in (67.4 %), some of which was atrophic with complete or partial hair loss while some patients revealed leukodermic atrophic patches of scarring alopecia in (32.6 %) of cases.

A study by Filbrandt *et al.* showed that scalp lesions in patient with DLE revealed the maximum activity in center of lesions, with peri-follicular inflammation, follicular plugging, atrophy and pigmentary changes.[17] Further study by Elizabeth K *et al.* had shown that early lesion was red papule or small plaque with centrifugal spread, a coin-shaped (''discoid'') red plaque forms, with follicular plugging and adherent scale and with continued disease progression, the erythema may be diminished, while atrophy, telangiectasia, hypopigmentation or depigmentation, and loss of follicular ostia became prominent.[18]

In the present study; 46.5% of patients with DLE showed extracranial involvement, including face (23.2%), ears (13.95%), extremities (6.97%) and trunk (2.32%). A study conducted by Al-Waiz *et al.* which included 54 patients with DLE; 42.5% of patients with scarring alopecia has also found a similar result where the face and ears were more commonly involved than other body sites.[19]

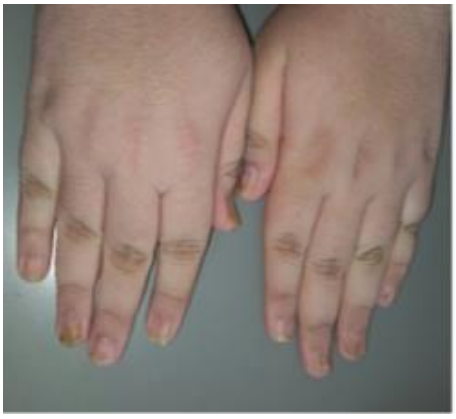

Figure 11 22 years old male patient with follicular lichen planus of scalp showing nail changes in a picture of pterygium.

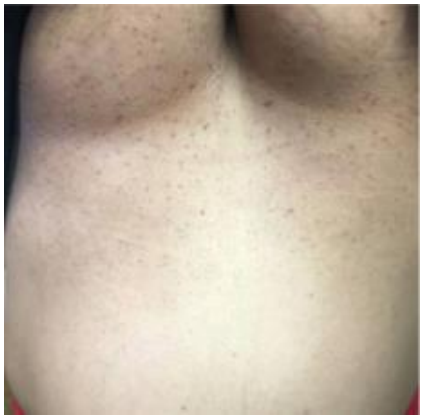

Figure 12 49 years old female patient with follicular lichen planus of scalp showing follicular lesions involving the trunk.

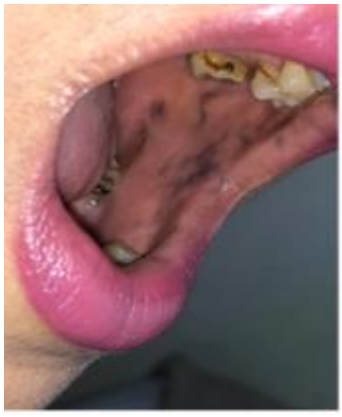

Figure 13 52 years old female patient with follicular lichen planus of scalp showing oral involvement in a form of pigmented lesions.

The histopathological findings revealed that patients with active disease, the epidermis is atrophied in most cases and showing compact hyperkeratosis, follicular plugging and parakeratosis in some cases. Basal layer destruction and melanin incontinence with melanophages were also observed, in addition there was thickening of basement membrane. The dermis showed dense cellular infiltrate in both superficial and deep dermises mainly perifollicular, perivascular and periadnexial which composed primarily of lymphocytes and this infiltrate even involved panniculus in some cases.

While in late cases in DLE, the histopathological findings were epidermal atrophy and sever dermal fibrosis with hair follicles remnants and absence of skin appendages which are replaced by a scar tissue. Wilson *et al.* found an important increase in the atrophy of the epidermis, follicular damage and scarring. Perifollicular lymphocytic inflammatory reactions were maximal around the level of the mid-follicle, while the dermal papillae were not involved by the inflammatory reactions.[20]

For patients FLP; in our study we had 57 patient with scarring alopecia, with their ages ranged from 10-60 year with a mean and SD 37.5±11.41 year which was considered younger than reported by Tan *et al.* where the mean age was 47.4 years[21] and similar to Sharquie *et al.* where the mean age±SD of onset of the disease was 36.7±5 years.[8]

In the current study, the distribution of patients with follicular lichen planus according to sex, male patients was more common and constituted (66.66%) while females patients was (33.34%) with M:F ratio 2:1.While Cevasco *et al.* found that the vast majority of patients with FLP were women (93%) in study that included 29 patients.[22] Mehregan *et al.* also found that most of patients were women which constituted (80%) of their cases.[23] Similarly Sharquie *et al.* showed that males were more affected than females with a M:F ratio of 1.4:1.[8]

This difference in sex and age distribution can be related to the difference in race as the current study was mainly among Iraqi population while the old studies were included Caucasian, American-African and other races.

In the current study, the pattern of hair loss was follicular moth-eaten alopecia in all cases and the clinical picture started as small moth eaten

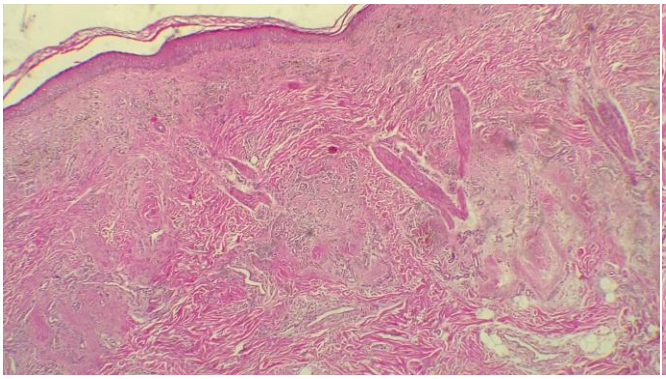

Figure 14 Patient with follicular lichen planus of scalp with scarring alopecia showing basketweave hyperkeratosis, epidermal atrophy, intact basal layer, dense scarring of deep and superficial dermis with hair follicles destruction and hypertrophied arrecter pili muscles. HE stain X4.

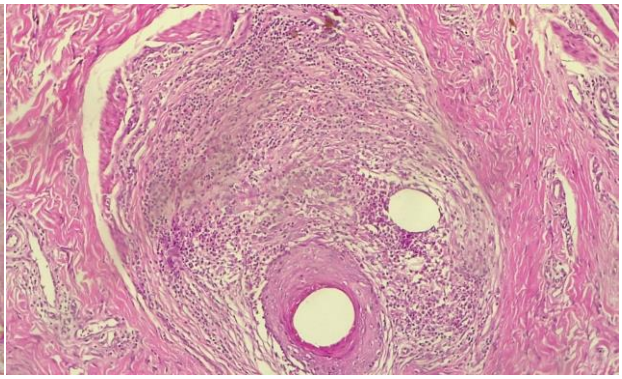

Figure 15 Patient with follicular lichen planus of scalp with scarring alopecia showing dense inflammatory infiltrate surrounding remnant of destructed hair follicle with dilated blood vessels. HE stain X40.

patches and over time these small areas of alopecia were coalesced together to form large patches simulating pseudo palade pattern of alopecia and this similar to a research conducted by Sharquie *et al.* that showing all cases had prominent and characteristic multifocal irregular areas of patchy scarring hair loss mimicking moth eaten alopecia which was similar to what has been seen in secondary syphilis and alopecia areata.[8] While HoonKank *et al.* found that small patches of alopecia of follicular lichen planus can slowly progress and become interconnected with each other, which can lead to a reticulated pattern. Furthermore, these scarring areas might unite together to produce larger scarring areas, and sometimes the whole scalp may be affected and these descriptions were similar to what had been found in the present study.[24]

Also we found that the color of the skin of alopecia in patient with follicular lichen planus was usually dark pigmented in (64.9%) of cases but more intense at the periphery of the lesion and studded with follicular lesions and over time leukodermic atrophic patches were noticed which was seen in (35.1%) of cases. Hence the progression of the disease was at the border of the patches rather than central and this similar to the study conducted by Sharquie *et al.* revealed the activity of the disease was mainly at the periphery of the scarring site in the form of hyperkeratotic follicular papules.[8]

In current study, 22.8% of patients showed extra- cranial lesions involving trunk, extremities, nail and mucus membranes in whom trunk in (8.7%) and nails in (5.26%) were most commonly involved and this is in agreement with Chieregato *et al.* which included 30 patients with FLP and revealed that 30% of patients had extracranial cutaneous involvement including skin , nails and mucus membranes.[25] Similarly Sharquie *et al.* showed that FLP of the scalp associated with other sites involvements in 26.8% of patients where trunk was most commonly involved with small asymptomatic follicular papules.[8]

In the current study, the scoring of severity of scarring of scalp in patients with follicular lichen planus ranged from 1-100% and 43.9% of patients had 1-25% scalp involvement followed by 33.3% of patients had 26-50% of scalp involved. Both Tandon *et al.*[26] and Sharquie *et al.* are revealed that the majority of patients experienced a 51-75% extent of disease in the scalp. This variation may be related to delayed diagnosis of patients with FLP where patients with early disease has less sever scoring of scalp

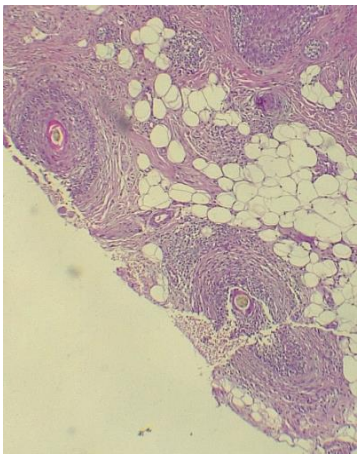

Figure 16 Patient with follicular lichen planus of scalp with scarring alopecia showing severe inflammatory infiltrate around hair follicles which extended deeply to involve the panniculus. HE stain X10.

scarring than those with delayed diagnosis.

The histopathological findings revealed that the epidermis usually normal with intact basal layer and normal dermo-epidermal junction and the inflammatory infiltrate was mainly perifollicular but only few cases showed epidermal atrophy while in others hyperkeratosis and follicular plugging were observed. In old scarred lesions, there were no obvious pathological changes apart of marked scarring and residual melanophages. Still in some cases, inflammatory reaction and fibrosis were observed reaching the panniculus.

Whiting *et al.* showed that the histopathologic changes in active follicular LP were mostly follicular without involvement of the epidermis.[3] While Sharquie *et al.* observed that the inflammatory reaction affected the site of bulge area causing scarring and leaving the lower segment of the hair follicle intact. This histopathological result explain the permanent damage to the hair organ.[8]

From the current study, we found that DLE was more common in females' patients while follicular LP was more common in males' patients with significant p-value (0.013). Regarding age group, there was no statistically significant differences. Moth eaten alopecia also was significantly more in patient with follicular lichen planus than patient with discoid lupus erythematosus with significant p-value (0.000). Regarding extracranial involvement, face and ear were more commonly involved in patients with discoid lupus erythematosus than patient with follicular lichen planus while nails and mucus membranes were more commonly involved in patients with follicular LP. Patients with DLE present with erythematous scarring mainly while patients with follicular LP showed pigmented scarring.

Conclusion

Both follicular lichen planus and discoid lupus erythematosus are common causes of scarring alopecia affecting young people more in females in DLE but more in males in follicular lichen planus. Moth eaten alopecia was the dominant picture in patients with follicular lichen planus in comparison with discoid lupus. Scarring alopecia is a progressive condition in both diseases that should be diagnosed as early as possible and treated aggressively to prevent scarring and permanent hair loss. Follicular lichen planusis a common etiology of scarring alopecia in Iraqi population. Also, it is associated with early scarring and permanent hair loss as the disease involves bulge area of hair follicles, so that by early diagnosis and management we can save hair follicles. While discoid lupus erythematosus has less tendency to cause scarring alopecia as it commonly involves lower segment of hair follicles. So the prognosis of hair growth is more favorable in discoid lupus erythematosus when compared with follicular lichen planus.

Declaration of patient consent The authors certify that they have obtained all appropriate patient consent.

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Conflict of interest Authors declared no conflict of interest.

Author's contribution

KES, MJK: Substantial contributions to study design, acquisition of data, manuscript writing, critically review, has given final approval of the version to be published.

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