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INCIDENCE OF GRANULOMA FORMATION IN DISCOID LUPUS ERYTHEMATOSUS

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Abstract:

Objective: *Lupus erythematosus is an autoimmune disease with pronounced pleotropism. If several systems are involved, the disease is called systemic lupus erythematosus (SLE) and if the skin only involved exclusively the term discoid lupus erythematosus (DLE) is used. One of several Histopathological features of DLE include periappendageal inflammation. It sometimes Eliminate the sebaceous glands that make up the granulomas.*

Aim: *To determine the frequency of sebaceous granuloma formation in discoid lupus erythematosus.*

Study Design: *An Observational prospective study.*

Place and Duration: *In the department of Medicine, Fauji Foundation Hospital Rawalpindi for one year duration from January 2019 to January 2020.*

Material and method: *This prospective observational study was conducted in pathology and Dermatology department of Services Institute of Medical Sciences, Lahore for one year duration from February 2019 to February 2020. 110 DLE cases selected for one year duration between the ages of 4 and 70 for the occurrence of sebaceous granuloma. Other DLE features, such as follicular plugging, hyperkeratosis, vacuolization of the base layer, epidermal atrophy, periappendageal inflammation, perivascular inflammation, periorbital inflammation and collagen damage was also distinguished.*

Results: *18 of these 110 cases had sebaceous granuloma. These granulomas consists of foreign body giant cells, epithelial cells containing partially digested sebaceous material and some lymphocytes.*

Conclusion: *Sebaceous granuloma formation was observed in 16.4% of DLE cases. This feature should be recognized by both pathologists and dermatologists for DLE diagnosis. Due to the presence of granulomas, an incorrect and incomplete diagnosis cannot be made.*

Keywords: *Sebaceous granulomas, hyperkeratosis, epidermal atrophy, Discoid lupus erythematosus (DLE).*

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INTRODUCTION:

Discoid lupus erythematosus (DLE) is atrophic, scar, chronic, photosensitive dermatosis. DLE can ensue in individuals with systemic lupus erythematosus (SLE), and <5% of individuals with DLE transfrom into systemic lupus erythematosus.

The primary lesion is mild to moderate scaling or erythematous papule[1, 2]. As the lesion developa, the scale may adhere and thicken, and pigmentation modifications may progress. DLE patients rarely meet the four or more criteria used to classify SLE. Serological abnormalities are rare. Sunscreen, topical corticosteroid and antimalarial drugs are often effective[3].

The study was based on sebaceous granuloma, which was not previously described as a feature of DLE in some cases observed during our weekly dermato-pathological conference at our institution[4]. Sebaceous granulomas arise as a result of the destruction of the sebaceous glands as a result of intense chronic periappendageal inflammation. The progressive fat vacuoles and lipid promote and initiate epithelioid cell differentiation, giant cell formation and phagocytic activity lastly crowning in well-differentiated granulomas[5]. This feature was obviously not visible in all cases, but in some cases was observed[6, 7]. The purpose of this study was to determine the frequency of sebaceous granulomas in discoid lupus erythematosus and to increase awareness among dermatologists and pathologists who may lead to misdiagnosis of this trait.

MATERIAL AND METHODS:

This observational study was conducted in the department of Medicine, Fauji Foundation Hospital Rawalpindi for one year duration from February 2019 to February 2020. 110 DLE patients of both sexes and ages 4 to 70 years and lasting more than one years were examined to detect the presence or absence of particularly well-formed sebaceous granulomas. All cases of clinical suspicion of skin LE were biopsied, routinely processed and stained in the pathology ward by formalin fixation and hematoxylin and eosin (H&E) staining. The slides were assessed by dermatologist and pathological consultant on multihead microscope attached with overhead projector and TV monitors. Along with other features such as hyperkeratosis, epidermal atrophy, follicular plugging, basal vacuolization, basement membrane accumulation, pigment incontinence, perivascular and peri appendageal inflammation and collagen damage, some cases had comprehensive sebaceous glands effacement with their replacement with well-formed granulomas comprising of multiple epithelioid cells, lymphocytes, giant cells along with vacuolated macrophages containing lipid droplets derivative of sebaceous glands and few neutrophils.

RESULTS:

18 of these 110 cases had sebaceous granulomas. Cases were associated with a lack of normal sebaceous glands. Residues of hair follicles and / or erector pylorum muscles muscles provided clues to the true nature of granulomas. Vacuoles with punched out cut in the giant cell cytoplasm were another useful clue (Figs. 1-4). Granulomas consist of foreign body type giant cells, some lymphocytes and epithelial cells.

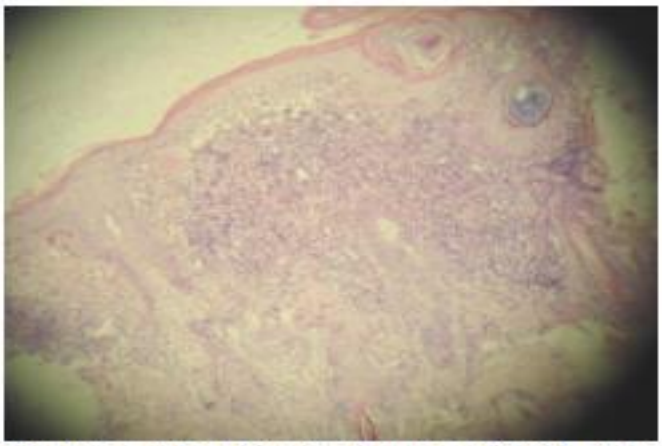

Figure 1 Skin biopsy from a discoid lesion: hyperkeratosis, keratin plugging, epidermal atrophy, basal cell vacuolization and several sebaceous granulomas (H& E X 100) .

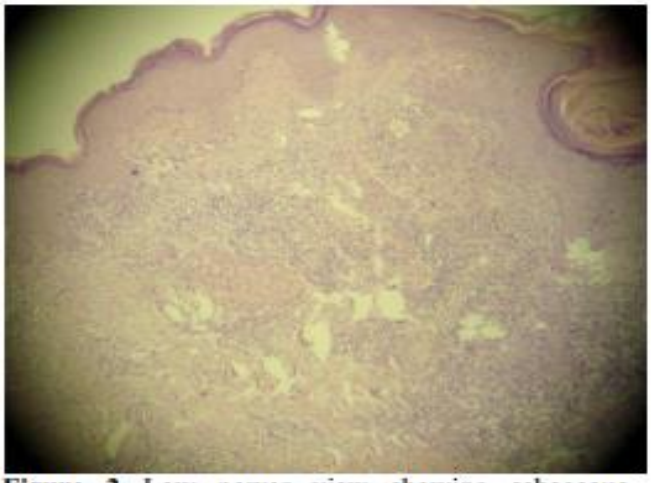

Figure 2 Low power view showing sebaceous granulomas wiith destroyed sebaceous gland visible as white tiny vacuoles.

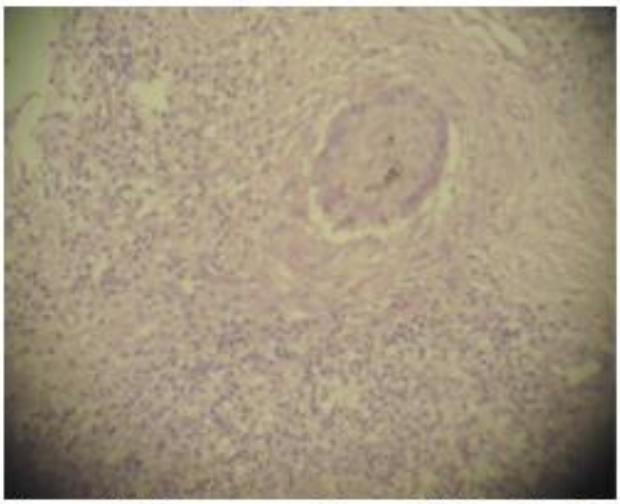

Figure 3 Sebaceous granulomas visible with giant cell. Destroyed sebaceous glands are visible as white holes.

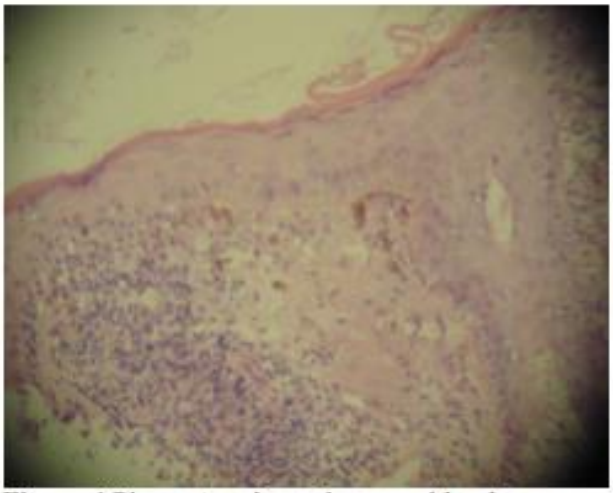

Figure 4 Pigmentary incontinence with sebaceous granulomas.

Other histopathological features include keratin plugging, hyperkeratosis, basal cell vacuolization, epidermal atrophy, and collagen damage and perivascular inflammation (Table 1).

Table 1 Frequency of histopathological features of patients (n=110).

Histopathological feature	***N & %***
Hyperkeratosis	110(100%)
Basal cell vacuolization	110(100%)
Collagen damage	110(100%)
Epidermal atrophy	107(97%)
Perivascular inflammation	106(96%)
Periappendageal inflammation	104(95%)
Follicular plugging	75(68%)
Acanthosis	50(45%)
Pigmentary incontinence	40(36%)
Pigmentary incontinence	30(27%)
Sebaceous granulomas	18(16%)

DISCUSSION:

DLE is a chronic skin condition that can cause hair loss, scarring and skin discoloration if not cured quickly and properly. This can have a significant influence on life quality. The incidence is 17.48 to 100,000 people. Females are affected three times additional than males compared to 9-10 times the frequency of SLE. 20 and 40 years of age is the age of onset observed commonly.

LE is supposed to be an autoimmune condition amongst other connective tissue disorders such as rheumatoid arthritis, scleroderma, mixed connective tissue disease and polymyositis[8]. The disease spectrum in LE is essentially a skin disease at one end and definite as DLE, and at the other end is a florid disease that is a systemic engrossment of the lungs, heart, kidneys, brain and other tissues called SLE. There are disorders such as subacute cutaneous lupus erythematosus (SCLE) between the two ends of the spectrum. SCLE usually have a sudden start with psoriasiform or annular plaques erupting on the arms, upper trunk or dorsum of hands when exposed to sunlight[9].

Early diagnosis and treatment improve prognosis. The analysis is typically based on a clinical trial. Histopathology may be needed in few cases to sanction the diagnosis[10]. Histology is the lichenoid tissue reaction with variations in the dermal and epidermal junction, including basement membrane thickening (best periodic Schiff acid staining) and vacuolar basal cells degeneration with variable periappendageal and perivascular lymphocytic infiltrate[11]. Hyperkeratosis is more pronounced in the reticular dermis, and more mature lesions have more pronounced follicular plugging. Basically, it's a lot of mucins in the dermis[12]. Histopathological features vary depending on the type and age of the lesion[13].

DLE has an enhanced prognosis and in comparison, of SLE, it has a less severe course. It is significant for GPs to distinguish DLE because it is a potentially scarring disease. Early visits by dermatologists and medical facilities increase hopes for minimizing disease progression and, consequently, for socio-economic impact on the individual.

Among the histopathological features in this study, hyperkeratosis, primary cell damage and collagen damage were the most common features occurring in 100% of cases. Epidermal atrophy was observed in 97% of cases, and periorbital and perivascular inflammation was observed in 96% of patients. Acanthosis was detected in 40% of cases. Parakeratosis was the least common feature with only 20 percent (Table 1).

The total number of cases studied over 100 years was 100. In 8 of these 100 cases, sebaceous granuloma formation was detected. This finding has not been previously discussed with the results of DLE histopathology. Another important finding in this study is the earlier destruction of the sebaceous glands from the hair follicles[14]. This was demonstrated in the form of lymphocytic infiltration of sebaceous glands, replacement of the glandular structure and sebaceous granulomas containing gigantic foreign body cells, as well as partially digested sebaceous material.

These findings increase the likelihood that an immune attack in DLE may be directed to the sebaceous glands primarily responsible for its destruction. The sebaceous glands appear to be damaged in front of the hair follicles, and therefore the permanent hair follicles and the erector pylorum muscle can be a clue to the true nature of sebaceous granulomas[15].

The skin contains many different types of granulomas. Sebaceous gland lipids such as in tuberculosis and leprosy, the lipid layer of the mycobacteria serves as a provoking factor, can lead to granulomas. In some cases, granuloma formation can be the result of simple lipids (cholesterol

granuloma). In addition to infectious agents such as Leishman-Donovan bodies and fungi, dead hair and numerous foreign bodies can cause granulomas. Endogenous substances (granulomatous mastitis) secreted from the mammary gland, semen (spermatocytic granuloma), dead or damaged collagen (cutaneous laxa, granuloma annulare) can cause granuloma. In this study, endogenous hemorrhages (giant tumor of the bone) and lipid granulomas from the sebaceous glands may occur. To more accurately determine the etiology and clinical course of the disease, each microscopic feature should be assessed.

More research should be done to assess lesions on a larger scale, as this may be related to the duration or severity of DLE clinical features and somehow play a role in the treatment and prognosis of these patients.

CONCLUSION:

Sebaceous granuloma formation is an important dermatopathological finding rare in DLE. Both dermatologists and pathologists should be aware of this rare feature (16.4 in our study) so as not to abandon the diagnosis of DLE and to make an incorrect diagnosis based on granulomas.

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