A Clinicopathologic study of Lichen Planus at Assir area, Kingdom of Saudi Arabia

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ABSTRACT

Background: Lichen planus (LP) is a subacute or chronic immunologically mediated dermatosis that involves the skin, mucous membranes, hair follicles, and nails. To date, the clinicopathologic features of these lesions in Assir region, Kingdom of Saudi Arabia are largely unknown.

Materials and Methods: To define these features, diagnostic records of dermatopathology cases received at the Pathology Department, Assir Central Hospitals (2007-2008) were reviewed. The lesions included 51 cases of lichen planus.

Results: Lichen planus was more common in males than in females (2 : 1). The average age incidence was 35.8 3.2 years, and 35.4 2.4 years for males and females respectively. The lower extremities, face and trunk were the most common sites for the lichen planus.

Conclusions: In Assir region, Kingdom of Saudi Arabia lichen planus is a common disease. It usually affects middle age populations and has a male sex predilection.

Key words: Lichen planus, Saudi Arabia

Review of literature

Normal skin

The epidermis is a stratified squamous epithelium composed predominantly of keratinocytes, and of at least three other resident cells: melanocytes, Langerhans cells and Merkel cells.

The keratinocytes differ from the dendritic cells, or clear T-cells, by having intercellular bridges and ample amount of stainable cytoplasm. The epidermis is not only the most cellular but also the most dynamic layer of the skin. As such, it continuously sheds and regenerates itself.

The keratinocytes are arranged in four layers: the basal cell layer (stratum basalis), the squamous cell layer (stratum spinosum), the granular layer (stratum granulosum) and the horny layer (stratum corneum).(18)

The epidermis forms a broadly undulating interface with the dermis. It extends into the dermis as broad folds (the rete ridges) and the dermis projects into the epidermis in finger-like projections (the dermal papillae). (18)

The basal cell layer is formed of a single layer of columnar cells. They lie with their long axes perpendicular to the dividing line between the epidermis and the dermis. They have a more basophilic cytoplasm than cells of the stratum spinosum and contain dark stained oval or elongated nuclei. They often contain melanin pigment transferred from adjacent melanocytes. The extent and distribution of this pigment correlates with skin color. The basal cells are connected with each other and with the overlying cells by intercellular bridges or desmosomes. At their base, they are attached to the subepidermal BM zone by modified desmosomes; termed hemidesmosomes. The basal cells and the overlying squamous cells contain keratin intermediate filaments termed tonofilaments, which form the developing cytoskeleton. Most of the mitotic activity in normal epidermis occurs in the basal cell layer.(1 and 18)

The second layer is the prickle cell layer. It is formed of 5 to 10 layers of polyhedral cells that become flattened toward the surface. The cells are separated by spaces that are transversed by intercellular bridges. The tonofilaments within the cytoplasm of these cells are loose bundles of electron-dense filaments. They are attached to the attachment plaque of a desmosome at one end, and the other end lies free in the cytoplasm near the nucleus. The intercellular cement substance between two adjacent keratinocytes contains glycoproteins. It has a gel-like consistency that explains why it on one hand provides cohesion between the epidermal cells and on the other hand allows the rapid passage of water-soluble substances through

the intercellular spaces. Furthermore, it allows the opening up of desmosomes and individual cell movement. (22)

Recent studies established the molecular basis for cell-to-cell adhesion within the prickle cell layer and other epidermal layers. Cadherins is a key family of adhesion molecules that are derived from multiple genes. Desmosomal cadherins are desmogleins and desmocollins that localize to desmosomes. They are linked to intracytoplasmic intermediate filaments by plakoglobin and desmoplakin. (22)

The third layer is the granular cell layer. It is composed of flattened cells and their cytoplasm is filled with keratohyaline granules that are deeply basophilic. The thickness of the granular layer in normal skin is proportional to the thickness of the horny layer. It is only 1-3 cell layers thick in areas with a thin horny layer. It reaches up to 10 layers in areas with a thick horny layer such as the palm and sole. There is often an inverse relationship between the presence and thickness of the granular cell layer and parakeratosis. For instance in psoriasis, parakeratosis is associated with markedly attenuated or absent granular cell layer. The keratohyaline granules are the precursors of the protein filaggrin that promotes aggregation of keratin filaments in the cornified layer. (15)

The fourth layer is the horny layer. It is composed of multiple layers of polyhedral cells that are arranged in a basket weave pattern. These cells lose their nucleus and cytoplasmic organelles and are composed entirely of keratin filaments. These cells are the most differentiated cells of the keratinizing system. They eventually shed from the surface of the skin. (17) The basement membrane separates the epidermal basal layer from the dermis. It is seen by light microscopy, as a continuous and thin periodic acid Schiff (PAS)-stained layer. Alternatively, by electron microscopy, the basal cells are seen attached to the basal lamina by hemidesmosomes. (15) Ultrastructurally, the basal lamina is composed of four different regions. From the epidermis to the dermis, they are respectively: i) the plasma membrane of the basal cells containing the hemidesmosomes and anchoring filaments (15), ii) the lamina Lucida which represents an electron-lucent area composed of laminin and bullous pemphigoid antigen, iii) the lamina densa, an electron-dense area composed of type IV collagen, and iv) the sublamina densa or lamina fibroreticularis containing the structures that attach the basal lamina to the connective tissue of the dermis. The latter represents extension of the lamina densa, the anchoring fibrils (type VII collagen) and the antigen to epidermolysis bullosa acquista. (22)

The supportive structure of the skin is provided by the dermis, a relatively hypocellular layer of varying thickness. It is composed of a structural collagen matrix, elastin and ground substance. Embedded within the dermis are epidermal appendages, nerve endings, resident cells and vessels. The dermis is divided into two compartments; the papillary dermis and the reticular dermis. The papillary dermis underlies the epidermis and extends around the adenexa (in which location it is also known as the adventitial dermis). It is composed of fine fibers consisting predominantly of type I and type III collagen. It moors the epidermis, interdigitates with the reticular dermis, and surrounds the epidermal appendages. It also contains a delicate branching network of fine elastic fibers, abundant ground substance, superficial capillary plexuses and fibroblasts. (21)

The reticular dermis is thicker than the papillary dermis. It is made up of densely packed coarse-fibered collagen which is predominantly type I. The collagen bundles traverse the dermis in a pattern that has not yet been defined. Associated with these two interstitial collagens are the finely filamentous collagens type V and type VI. (9) Supplementing its protective function, the skin has three specialized redundancies referred to as epidermal appendages or adnexa. These epidermis-derived structures consist of the pilosebaceous apparatus (with its hair, sebaceous and apocrine elements), the eccrine glands and the nails.

Lichen planus

Lichen planus (LP) is a subacute or chronic immunologically mediated dermatosis that involves the skin, mucous membranes, hair follicles, and nails. (6)

Pathophysiology: Although the exact cause of lichen planus is unknown, a cell-mediated immune reaction has been implicated in its pathogenesis. In support, lichen planus is associated with other diseases of altered immunity, such as ulcerative colitis, alopecia areata, vitiligo, dermatomyositis, morphea, lichen sclerosus and myasthenia gravis. In addition an association is noted among lichen planus and hepatitis C infection, chronic active hepatitis and primary biliary cirrhosis. (23,24 and 25)

Immunohistochemical studies show that the infiltrating cells in lichen planus are predominantly T lymphocytes with very few B lymphocytes. The predominant subtypes of T lymphocytes in the infiltrate are of helper-inducer or suppressor-cytotoxic T lymphocytes lineage. Both subsets participate in the immunologic reaction with the suppressor-cytotoxic T lymphocytes being predominant in the epidermotropic response, suggesting a cell-mediated cytotoxic mechanism against the epidermal cells. The basal keratinocytes adjacent to the infiltrate express intercellular adhesion molecule-1, which enhances the interaction between lymphocytes and their epidermal targets, resulting in keratinocytic destruction.(34)

This surface antigen is probably induced by cytokines released by lymphocytes from the infiltrate. In addition, a superantigen may be involved in the pathogenesis of lichen planus. (2,3,5,6,7,8,9,11 and 26)

The number of Langerhans' cells in the epidermis is increased very early in the disease. Immunoelectron studies have shown close contacts of lymphocytes with Langerhans' cells and macrophages. (14) The Langerhans' cells can process and present antigens to T lymphocytes, leading to their stimulation and thus attacking keratinocytes. These cell-to-cell interactions suggest that a cell mediated immune mechanism is operative in lichen planus. (31 and 32)

Incidence: Lichen planus has a worldwide distribution with no significant geographical variation in its incidence. It can occur at any age, with a tendency to affect middle aged and elderly individuals. No sex predilection has been noted. (6 and 7)

Clinical features: The eruption of lichen planus is characterized by small, flat-topped, shiny, violaceous papules that may coalesce into plaques. The papules are polygonal and often show a network of white lines known as Wickham's striae. They vary in size from 1 mm to greater than 1 cm. The disease has a predilection for the flexor surfaces of the forearms and legs. Pruritus is very common, but it varies in severity according to the type of lesion and extent of involvement. Hypertrophic lesions are usually extremely pruritic. The eruption of lichen planus may be localized or generalized, and Koebner's phenomenon is commonly seen. (6 and 7)

In addition to the cutaneous eruption, lichen planus may involve the mucous membranes of the buccal mucosa and tongue (oral LP), genitalia, nails, and scalp.

The oral lesions of lichen planus are common and may occur as a sole manifestation of the disease or be associated with cutaneous involvement. They usually involve the buccal and glossal mucosa in the form of a reticular network of coalescent papules. Besides this reticular type, other lesional patterns have been described in oral lichen planus, such as papular, plaquelike, atrophic, erosive, and bullous. (6 ,28and 30) Genital involvement is common in men with cutaneous lichen planus, usually in the form of an annular configuration of papules on the glans penis. Less commonly, linear white striae may be seen. The nails are involved in about 10% of cases of lichen planus, in the form of roughening, longitudinal ridging, and, rarely, thinning and destruction. (16,29 and 31)

Lichen planopilaris is a type of lichen planus that predominantly affects the scalp with follicular and perifollicular violaceous scaly pruritic papules. It may coexist with typical lichen planus lesions on the skin, mucous membranes, or nails. Progressive hair loss may occur, resulting in the development of irregularly shaped atrophic patches of scarring alopecia on the scalp (pseudopelade of Brocq). The axillae and the pubic region may also be affected. Hyperkeratotic follicular papules may also be seen on glabrous skin. The Graham Little syndrome consists of an association of scarring alopecia of hair-bearing areas and hyperkeratotic papules on glabrous skin. Linear lichen planopilaris of the face resolving with scarring has also been described. (17)

Other variants of lichen planus include hypertrophic lichen planus, atrophic lichen planus, vesicular lichen planus, lichen planus pemphigoides, ulcerative lichen planus, actinic lichen planus, annular lichen planus, linear lichen planus, and guttate lichen planus. The hypertrophic lichen planus is a common variant of lichen planus that usually affects the extensor surfaces of the lower extremities, especially around the ankles. It is a pruritic lesion that consists of thickened, often verrucous plaques that may heal with residual pigmentation and scarring. The atrophic variant of lichen planus is characterized by a few lesions that are often the resolution of annular or hypertrophic lesions. The vesicular lichen planus is a rare variant that shows vesicles situated on some of the preexisting lichen planus lesions.

The lichen planus pemphigoides differs from vesicular lichen planus by its more disseminated eruption and more extensive bullae. In addition, lichen planus pemphigoides may arise from papules of lichen planus and normal-appearing skin.

The ulcerative lichen planus is a rare variant of lichen planus, which shows bullae, erosions, and painful ulcerations on the feet and toes resulting in atrophic scarring and permanent loss of the toenails. It is usually associated with cutaneous and oral lesions of lichen planus, as well as atrophic alopecia of the scalp. The actinic lichen planus (lichen planus actinicus or pigmentosus) usually develops in spring and summer on sun-exposed areas of the skin, particularly the face. It is characterized by annular plaques with central blue to light brown pigmentation and well-defined, slightly raised, hypopigmented borders. Pruritus is minimal or absent.

Three forms of actinic lichen planus have been described: annular, pigmented, and dyschromic. (27 and 32)

The annular lichen planus is characterized by annular lesions with an atrophic center usually found on the buccal mucosa and male genitalia. Lichen planus papules that are purely annular are rare. The linear lichen planus represents a zosteriform lesion or may develop as a Koebner's effect. Finally, the guttate lichen planus develops in the form of discrete lesions which may vary in size from 1 mm to 1 cm. They almost never become chronic. Of note, the hypertrophic and actinic variants of lichen planus are commoner than the other variants. (6)

Histopathologic features: The typical papules of lichen planus show the following histologic features: 1) compact orthokeratosis, which contains very few, if any, parakeratotic cells, 2) wedge-shaped hypergranulosis with the granular cells being increased in number and size, and contain more abundant coarse keratohyaline granules, 3) irregular acanthosis, which affects the spinous layer of the rete ridges as well as the suprapapillary plates. The rete ridges show irregular lengthening, and some of them are pointed at their lower end, giving them a saw-toothed appearance, 4) destruction of the basal cell layer, which is obvious in fully developed lesions. In these lesions, the basal layer has the appearance of flattened squamous cells (squamatization of the basal layer), and 5) a band-like (lichenoid) dermal inflammatory infiltrate, which is in close approximation to the epidermis and is sharply demarcated at its lower border. It is composed mainly of lymphocytes intermingled with macrophages. Melanophages are usually seen in the papillary and upper reticular dermis as a result of destruction of the basal cells with subsequent pigment incontinence. (4)

In addition, apoptotic keratinocytes are present in the lower epidermis and papillary dermis in most cases. They appear in the form of round or oval, homogenous, eosinophilic bodies (colloid, hyaline, or Civatte bodies). Occasionally, small areas of artifactual separation between the epidermis and the dermis are present and are known as Max-Joseph spaces. In some instances, this separation occurs in vivo as a result of extensive damage to the basal cells. Wickham's striae are caused by the focal increase in the thickness of the granular layer and of the total epidermis. (20)

The typical papules of lichen planus show the following histologic features: 1) compact orthokeratosis, which contains very few, if any, parakeratotic cells, 2) wedge-shaped hypergranulosis with the granular cells being increased in number and size, and contain more abundant coarse keratohyaline granules, 3) irregular acanthosis, which affects the spinous layer of the rete ridges as well as the suprapapillary plates. In addition, variants of lichen planus have additional histological changes. In this regard, the hypertrophic lichen planus shows considerable acanthosis, papillomatosis, and hyperkeratosis. The vesicular lichen planus usually shows large Max-Joseph spaces, with subepidermal blisters. The lichen planus pemphigoides that arises from uninvolved skin shows subepidermal bullae with an inflammatory infiltrate that is not band-like and contains eosinophils. The lichen planus actinicus may show histologic features similar to those of typical lichen planus, but with a tendency toward thinning of the epidermis in the center of the lesion. In addition, more evident pigment incontinence and numerous melanophages are usually present in the papillary and upper reticular dermis. The oral lichen planus may show parakeratosis rather that orthokeratosis, with the presence of a granular layer (the buccal mucosa is normally devoid of a granular layer, except in the hard palate). The epithelium is often atrophic, and ulcerations may develop. The lichen planopilaris usually shows a focally dense, band-like perifollicular lymphocytic infiltrate. Vacuolar changes of the basal layer of the outer root sheath and necrotic keratinocytes are often seen. Advanced cases may show perifollicular fibrosis and epithelial atrophy, which may result in scarring alopecia. (10,19 and 33)

Specific Aims

In this investigation, we took an aim at studying the clinicopathologic features of lichen planus in Assir region, Kingdom of Saudi Arabia. To explore this aim and to fill this existing gap in literature, we carried out this investigation. To achieve our goals, we examined clinical and pathological characteristics of these lesions. A total of 51 lesions representing lichen planus were examined.

Materials and Methods

Tissue specimens: The formalin fixed, paraffin embedded tissues were obtained from the Department of Pathology, in Assir region, Kingdom of Saudi Arabia. The total number of specimens was 51 cases, including 51 cases of lichen planus. Clinical data were obtained from the clinical referral reports. They included: age and sex of the patient, type of lesions, and the site, and number of these lesions. All the patients were Saudi (Caucasian). No black individuals were included in this study.

Results

Clinical features of lichen planus: The study group consisted of 51 patients, including 17 females and 34 males. Evaluation of the clinical and histological profiles of the lesions in our locality (Assir region) demonstrated that they usually tend to affect the middle age groups and had male sex predilection.

Clinical features: The study group consisted of 51 patients, including 34 males (34/51, 67%) and 17 females (17/51, 33%). The clinical data were obtained from the referral clinical reports. The clinical characteristics of these lesions were summarized in Tables 1-2 and Figure 2 (next pages).

Pathological features: The typical papules of lichen planus show the following histologic features: 1) compact orthokeratosis, which contains very few, if any, parakeratotic cells, 2) wedge-shaped hypergranulosis with the granular cells being increased in number and size, and contain more abundant coarse keratohyaline granules, 3) irregular acanthosis, which affects the spinous layer of the rete ridges as well as the suprapapillary plates. The rete ridges show irregular lengthening, and some of them are pointed at their lower end, giving them a saw-toothed appearance, 4) destruction of the basal cell layer, which is obvious in fully developed lesions. In these lesions, the basal layer has the appearance of flattened squamous cells (squamatization of the basal layer), and 5) a band-like (lichenoid) dermal inflammatory infiltrate, which is in close approximation to the epidermis and is sharply demarcated at its lower border. It is composed mainly of lymphocytes intermingled with macrophages. Melanophages are usually seen in the papillary and upper reticular dermis as a result of destruction of the basal cells with subsequent pigment incontinence (Figure 3, page 17).

In addition, apoptotic keratinocytes are present in the lower epidermis and papillary dermis in most cases. They appear in the form of round or oval, homogenous, eosinophilic bodies (colloid, hyaline, or Civatte bodies). Occasionally, small areas of artifactual separation between the epidermis and the dermis are present and are known as Max-Joseph spaces. In some instances, this separation occurs in vivo as a result of extensive damage to the basal cells (Figure 3).

Discussion and Conclusions

Interface dermatitis encompasses a wide range of lesions characterized by lichenoid and vacuolar changes at the dermoepidermal junction i.e. interface zone. The former is characterized by lichenoid infiltrate and basal cell keratinocytes damage (LID). In the vacuolar subtype (VID), vacuolation of the basal cell keratinocytes is a characteristic feature. Although these lesions are thought to be autoimmune in nature, their exact pathogenetic causes are still unknown. In this vein, ID lesions are multifactorial in origin; may be

Table 1: Clinical characteristics of lichen planus in males

Case #	Age (Y)	Site	Clinical presentations
S08-0171	12	F	well-defined violaceous scaly papules
S08-2418	20	F	well-defined violaceous scaly papules
S08-0802	25	F	well-defined violaceous scaly papules and plaques
S07-2438	31	F	well-defined violaceous scaly papules and plaques
S07-0151	37	F	well-defined violaceous scaly papules and plaques
S07-3997	52	F	well-defined brownish scaly plaques
S08-0237	56	F	well-defined brownish scaly papules and plaques
S07- 0079	66	F	well-defined scaly plaques
S07-1797	17	UL	well-defined violaceous scaly plaques
S07- 0253	22	UL	well-defined violaceous scaly plaques
S08-2419	5	LL	well-defined violaceous scaly papules
S08-0005	12	LL	well-defined violaceous scaly papules
S08-0002	16	LL	well-defined violaceous scaly plaques
S07-0251	19	LL	well-defined violaceous scaly papules
S07-2147	23	LL	well-defined violaceous scaly papules and plaques
S08-0559	26	LL	well-defined brownish scaly papules and plaques
S08-1944	29	LL	well-defined brownish scaly papules and plaques
S07-2046	30	LL	well-defined purplish scaly papules and plaques
S07-0959	32	LL	well-defined violaceous scaly papules and plaques
S07-0876	33	LL	well-defined violaceous scaly papules and plaques
S07-0977	33	LL	well-defined grayish scaly plaques
S07-3183	37	LL	well-defined grayish scaly papules
S08-0877	50	LL	well-defined brownish scaly plaques
S08-0382	51	LL	well-defined grayish scaly plaques
S07-2360	55	LL	well-defined grayish scaly papules and plaques
S07-3427	57	LL	well-defined grayish scaly papules
S07-2228	60	LL	well-defined brownish scaly papules and plaques
S07-1530	71	LL	well-defined brownish scaly plaques
S08-1688	87	LL	well-defined brownish scaly papules and plaques
S08-1525	19	T	well-defined violaceous scaly papules and plaques
S08-1725	27	T	well-defined grayish scaly papules
S07-0254	28	T	well-defined violaceous scaly plaques
S08-0178	31	T	well-defined purplish scaly papules
S08-0470	51	T	well-defined brownish scaly papules and plaques

F : Face
LL : Lower Limb
UL : Upper Limb
T : Trunk

Table 2: Clinical characteristics of lichen planus in females

Case #	Age (Y)	Site	Clinical presentations
S07-0562	17	F	well-defined grayish scaly papules
S08-0294	30	F	well-defined violaceous scaly plaques
S07-1534	34	F	well-defined grayish scaly papules and plaques
S07-1800	50	F	well-defined brownish scaly plaques
S07-4347	20	UL	well-defined violaceous scaly papules
S07-0325	31	UL	well-defined violaceous scaly papules and plaques
S07-2077	50	UL	well-defined violaceous scaly plaques
S07-4451	24	LL	well-defined violaceous scaly papules
S08-0347	30	LL	well-defined brownish scaly papules and plaques
S07-0911	32	LL	well-defined violaceous scaly papules
S07-3572	33	LL	well-defined violaceous scaly plaques
S07-0147	36	LL	well-defined brownish scaly papules and plaques
S08-1853	38	LL	well-defined violaceous scaly papules and plaques
S08-0674	39	LL	well-defined brownish scaly papules
S07-0584	50	LL	well-defined violaceous scaly plaques
S07-1778	41	T	well-defined brownish scaly papules and plaques
S07-1571	47	T	well-defined brownish scaly plaques

F : Face
LL : Lower Limb
UL : Upper Limb
T : Trunk

induced by drugs; or by complex environmental, genetic and life style factors. (28)

Interestingly, a new association between these lesions and chronic liver disease has emerged. In this respect, Harman and his colleagues found a close association between LP and hepatitis C infection. (13) Also, anti hepatitis C antibodies were seen in patients with lichen planus. (13)

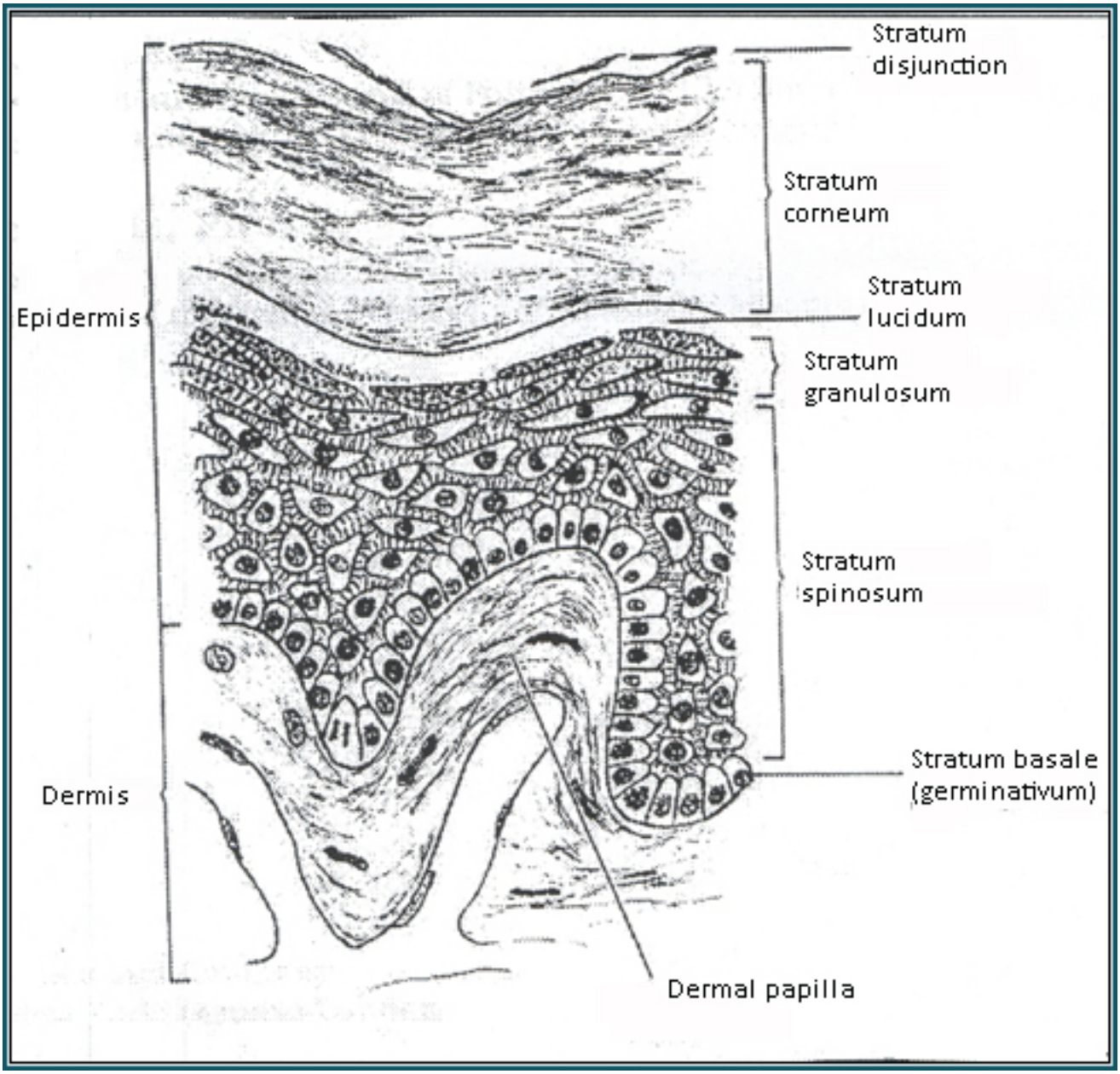
The clinical features of ID lesions in our series (age incidence, female sex predilection, and site of affection and the average duration of the diseases) are comparable to the findings in western societies. The studies performed in these societies reported an average male/female ratio of 1:1 to 1:1.3. The mean age was about 50.4 years. (6) Taken together, these findings suggest common underlying pathogenetic mechanisms in these diseases.

The female sex predilection may indicate that the susceptible genotype is probably characterized by a single inherited dominant allele on the X-chromosome. The disease chronicity, adult onset, female sex predilection and association with other autoimmune diseases suggest the autoimmune nature of ID. Familial lichen planus was reported by others in several studies and this raised the suggestion of genetic predisposition in ID. Several studies examined the role of genetic factors on the development of ID lesions like lichen planus such as HLA-associated antigens.

They showed a role of these antigens in the recruitment of lymphocytes at the site of inflammation.

The diagnosis of lichen planus can be usually made on histologic grounds in more than 90% of cases. However, a number of diseases may simulate the histologic picture of lichen planus and make some difficulties in the diagnosis. These lesions include LP-like keratosis, lichenoid drug eruption, lichenoid lupus erythematosus, chronic graft-versus-host disease, and lichen simplex chronicus. Lichen planus -like keratosis shows focal parakeratosis and adjacent solar lentigines in an otherwise typical histological picture of lichen planus. Lichenoid drug eruptions can be differentiated from lichen planus by the presence of focal parakeratosis with concomitant agranulosis, exocytosis of lymphocytes within the epidermis, and numerous eosinophils in a deeper inflammatory infiltrate. Lichenoid lupus erythematosus differs from lichen planus in the presence of epidermal atrophy in addition to acanthosis, perivascular and periadnexal infiltrate in addition to the superficial band-like infiltrate, dermal mucin deposits, and the presence of a thickened PAS-positive basement membrane. Chronic

Figure 1: The normal skin is composed of three layers: 1) epidermis, 2) dermis, 3) the subcutaneous adipose tissue. Each layer has a complex structure and function. The keratinocytes of the epidermis are arranged in four layers: 1) stratum germinatum, 2) stratum spinosum, 3) stratum granulosum, and 4) stratum corneum



graft-versus-host disease may show epidermal changes similar to lichen planus. However, the inflammatory infiltrate tends to be perivascular and the number of Langerhans' cells is decreased in chronic graft-versus-host disease. Lichen simplex chronicus can be differentiated from lichen planus by the absence of both basal cell destruction and the band-like infiltrate. (35)

The prognosis for lichen planus is good, as most cases regress within 18 months. However, some cases may recur. Hypertrophic lesions may leave residual hyperpigmentation. Alopecia is often permanent. Malignant transformation of cutaneous LP occurs in less than 1% of cases. (36) Squamous cell carcinoma may arise occasionally in long-standing lesions of lichen planus situated on mucous membranes or the vermillion border . (12) Ulcerative lesions of oral lichen planus, particularly in men, have a higher risk for malignant transformation. (37)

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Figure 2: Lichen planus, violaceous flat-topped scaly papules and plaques

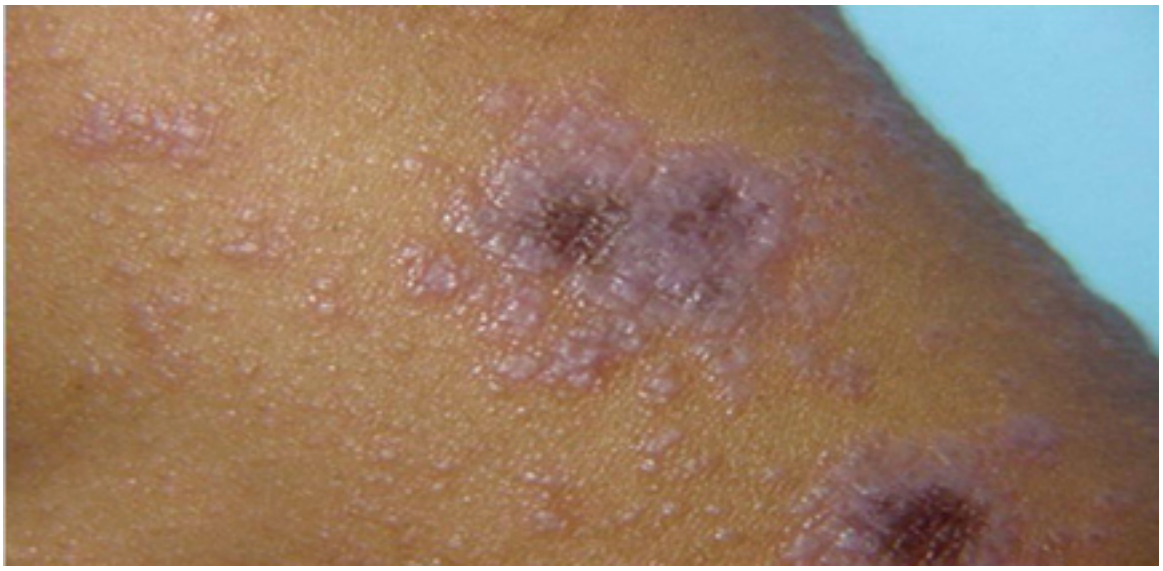
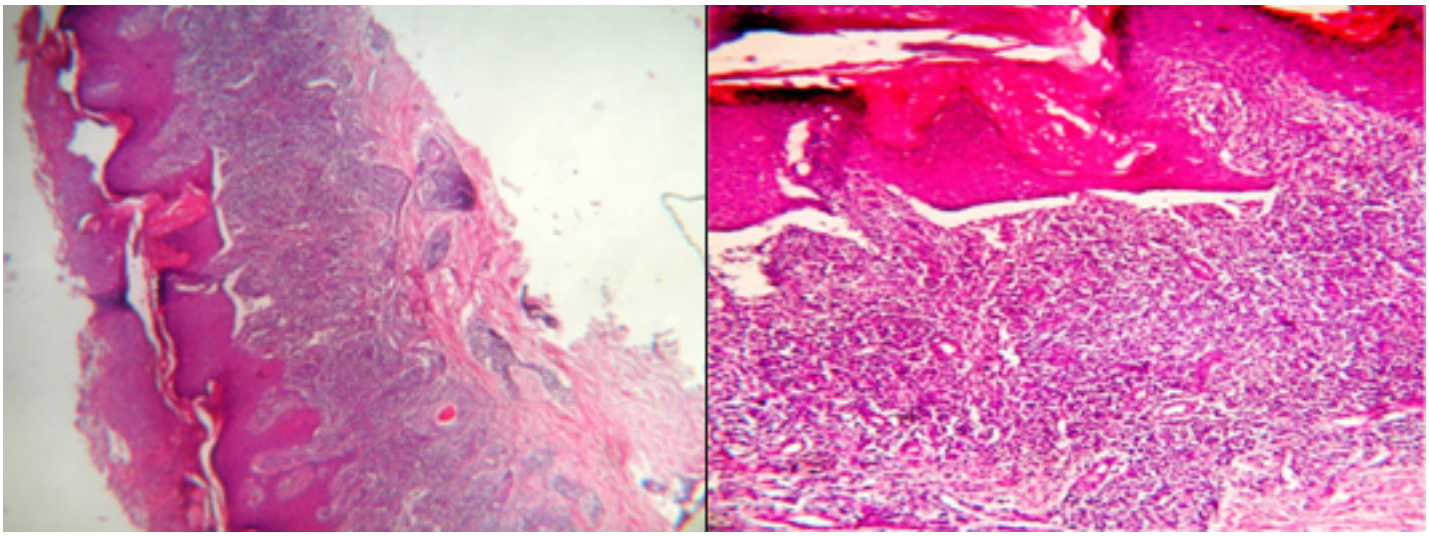


Figure 3 : Histological features of lichen planus. dense dermal inflammatory infiltrate. Occasional inflammatory cells are seen abutting on the basal cell keratinocytes together with apoptotic keratinocytes



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