## LICHEN PLANUS: HISTOPATHOLOGICAL STUDY OF 57 CASES

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A voluminous literature has accumulated since the first description of lichen ruber planus by Erasmus Wilson, 1868<sup>1</sup> when he separated this entity from a choatic group as approved by Graham Little 1919<sup>2</sup>, <sup>3</sup> at the American dermatologic association meeting. The term lichen has been abused by different investigators, who applied to quite a large group of dermatologic entities that are not at all or partially related to the classical lichen planus of Wilson. The term lichen ruber accuminatus coined by Besiner 1889<sup>‡</sup> was universally recognised as synonymous with pityiasis rubra pilaris particularly as such term is now being known to have no relation to the lichen planus of Wilson.

The relation of lichen sclerosus et atrophicus to localized scleroderma or lichen planus has been the matter of controversy among different authorities. Hallopeau 1887<sup>5</sup> and Darier 1892<sup>6</sup> considered lichen sclerosus et atrophicus as being a modification of lichen planus due to the close similarity between clinical and morphological findings in both conditions. An almost similar view was expressed by other workers e.g. Gahan 1954<sup>7</sup> and Miller et al 1957<sup>8</sup> who claimed a transitional inter relationship between lichen sclerosus et atrophicus, localized scleroderma and lichen planus. The modern trend, however, is to place lichen sclerosus et atrophicus with the group of sclerotic atrophies denying any relation to lichen planus.

The use of several terms in referring to one and the ame clinical entity leads to severe confusion and misunderstanding between different investigators. Lieberthal 1916<sup>5</sup> was the first to describe the condition known as lichen planus ocreaformis which was discussed by the French authors for example Darier 1945<sup>10</sup> under the name of lichen obtusus while Pautrier 1936<sup>11</sup> called it lichen corneus hypertrophicus. Unna 1929<sup>12</sup> on the other hand considered it as a variant of eczema and called it eczema verruca collosum, and limited the term lichen obtusus to the variety of the disease in which the papule of lichen ruber planus has an obtuse form.

In an attempt to limit the use of the term lichen to the large family of lichen planus, Fred Wise 1919<sup>13</sup> in an interesting discussion of neurodermatitis and the pseudolichens intelligently expressed that neurodermatoses are not lichens despite the remarkable resemblence that might, occasionally be present between both entities.

A line has to be drawn between the true lichen lesions and lichenification. While the formar denotes persistence of papular character of the eruption, Winer and Leeb 1954<sup>14</sup>, the latter term indicates a mosaic thickening of the skin in

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patches, deepening of the skin furrows and loss of elasticity, features that are most frequently recognized as atypical lichen planus.

Several types of lichen planus have been included in this big family but there is considerable doubt as to whether they are variant of this disease e.g. lichen planus erythematosus, lichen aureus or lichen purpuracious and lichen ruber moniliformis. Wise and Rein 1936<sup>16</sup> reviewed 17 cases of lichen ruber moniliformis which was first described by Kaposi 1886<sup>17</sup> and expressed their or inion that it should be considered a disease sui genereis. They suggested the name morbus moniliformis lichenoides.

The subject of structural alterations in lichen planus has been the field of extensive studies during a period of over one hundred years and voluminous data have been collected on that particular matter. The vast majority of such studies were directed more toward the clinical aspects rather than histologic changes. Our principal aim is to evaluate lichen planus and its different types from the histopathologic point of view.

Method and Material. 57 cases of lichen planus have been studied. 43 of the cases were females and 14 were males. The youngest patient was 7 years and the oldest was 72 years. The 57 cases were distributed among the following types of lichen planus as follows: 19 cases of classical 1. p. or 1. ruber planus; 7 cases of 1. p. verruccsus; 12 cases of 1. p. actinicus, 2 cases of annular 1. p.; 2 cases of lichen planopilaris, one case of atrophic lichen planus; 2 cases of lichen linearis and 5 cases of post lichen pigmentation.

82 percent of the patients had subjective sensation of different degrees of mild to severe itching. 18 percent had no subjective sensation. 74 percent of the patients attributed their condition to a psychic trauma and the rest failed to mention any reason. 18 percent of patients were presented with mucous membrane affection. Scalp and hair affection were found in 9 percent of the cases. Nail affection was noted in 7 percent of cases.

In our selection of cases it was decided that thorough clinical examination should be our criteria in denoting the disease as lichen planus. The clinical features of some of these cases had been published before <sup>18,19</sup> and the detailed histopathological of features clinical forms of lichen planus of mucous membrane will be published in a separate article.

In our cases, the shortest duration for which the lesion persisted prior to taking biopsies was one week, while the longest was about 10 years. It was taken in consideration that the lesion would not be presenting noticeable secondary changes.

Biopsy tissue was obtained under local anesthesia, paraffin tissue section was done using standard technical procedures and stained with hematoxylin and eosin. We will now describe the detailed reports of 7 cases as examples of different types of lichen planus.

Case reports. Case 1. male, 26 years, a clinical case of hichen ruber planus. Histopathological study of biopsy from typical papule of lichen planus from right forearm (Fig. 1) and from extensor surface of left leg (Fig. 2) showed: hyperkeratosis, increase of thickness of granular layer, acanthosis, liquifaction degeneration of basal cell layer and band like inflammatory infiltrate of round cells which is sharply demarcated at its lower border and hugs the epidermis.

Case 2. Male, 30 years, a case of lichen planus actinicus. Histopathological study from an annular lesien from the forehead showed (Fig. 3) hyperkeratosis, increase of thickening of stratum granulosum. The infiltrate was band like sharply demarcated at its lower border composed mainly of lymphocytes and plasma cells.

Case 3. Female, 38 years, a case of lichen plano-pilaris.

Histopathological study of biopsy taken from patches of spiny papules on left leg showed (Fig. 4,5) cellular infiltrate predominantly lymphocytic in nature surrounding the pilosebaceous structure at the level of the sebaceous glands. Associated with this infiltrate there was disappearance of sebaceous glands. The follicle was dilated and filled with a keratotic plug. There was also a band like infiltrate beneath the epidermis.

Case 4: Female aged 34 years. The clinical diagnosis was bullous lichen planus. Histopathological study of a recent vesicle and a bullae showed (Fig. 6) that in both subepidermal bullae were present and the dermal papillae were fairly preserved. Band like inflammatory infiltrate of round cells mainly lymphocytes was present in dermis of both lesions. The epidermis was thinned in the center but little in the periphery (Fig. 7).

Case 5: Female, 50 years. The clinical diagnosis was atrophic l. p.

Histopathological study of the atrophic area showed (Fig. 8) hyperkeratosis, atrophic epidermis, inflammatory infiltrate band-like, mainly lymphocytes, in addition fibroblasts were present in the dermis. The adnexial structure were destroyed.

 $Case\ 6$ : Female, 65 years. The provisional diagnosis was lichen corneus hypertrophicus.

Histopathological study of papule from left leg showed (Fig. 9) marked hyperkeratosis, acanthosis, and much thickening of granular cell layer. Intactness of the basal cell layer, sclerosing changes in the connective tissue and confinement of the inflammatory infiltrate to the perivascular zones. Beside the previously described picture, another biopsy of the same case from recent papule from the right thigh showed (Fig. 10) the presence of hyperkeratosis, thickness of granular layer, acanthosis and focal inflammatory patch in the dermis not exhibiting the band like pattern but causing destruction of few layers of epidermis including the basal layer. The inflammatory patch consists chiefly of lymphocytes and few histiocytes.

Case 7: Female, 18 years. A case of post lichen pigmentation.

Histopathological study of post lichen pigmentation lesion showed (Fig. 11) hyperkeratosis, diminution of thickness of stratum granulosum, acanthosis, intactness of basal cell layer. The inflammatory infiltrate seems to subside before epidermal involuting changes. Regenerative changes of collagen fibers presumably were indicated by the presence of fine newly formed fibers, lying side by side with remnants of destroyed ones. Melanophores were present in large numbers, having picked up pigment which has been dropped from the basal cell layer.

Comment. Lichen planus has a fairly specific picture which enables one to make a diagnosis from histologic examination alone.

(1) Classical lichen planus: lichen ruber planus-epidermis: There is a general agreement between investigators that hyperkeratosis forms a constant finding in lichen planus and "unless the lesion has been subjected to vigorous treatment, parakeratosis is never seen even in the bullous form." of the disease. Ormsby and Montgomery, 1955<sup>20</sup>.

Remarkable epidermal feature in all cases of lichen planus is increased thickening of stratum granulosum in which the unequal distribution of elidin granules is clinically reflected in Wickham striae, Darier  $1909^{21}$ . Seif-El-Nasr  $\epsilon$ t al  $1962^{22}$  attributed those striae to abnormal thickening of the granular layer in some areas and its absence in other areas with the appearance of alternating white and violet areas respectively.

Recently Summerly 1964<sup>2,3</sup>, in an interesting article "the microarchitecture of Wickham's striae" ended with the conclusion that Wickham striae are related to a focal hyperactivity in lesions rather than to thickening of the stratum granulosum. Glickman 1964<sup>2,4</sup> attributed the pallor which produces the appearance of Wickham striae to the absence of papillary capillaries in the center of the lesion. Ryan 1966<sup>2,5</sup> on the other hand attributed the violaceous hue which surrounds the area of pallor to radially arranged horizontal oriented capillaries of proliferative type.

Besides irregular acanthosis, the basal cell layer, characteristically shows varying degrees of liquifactive degeneration (Fig. 12). The degenerated epithelial cells according to Gougerot and Civatte 1953<sup>23</sup> play a role in the mechanism of formation of colloid bodies.

The demonstration of colloid bodies at the epidermodermal junction by Thyresson and Moberger 1957<sup>9,7</sup> was considered by Samman<sup>4,6</sup> as the most important addition to histopathological description of lichen planus since Dariers description in 1890<sup>2,1</sup>. Such bodies however were con idered by Thyresson and Moberger 1957<sup>2,7</sup> to be due to epidermal intracytoplasmic viral inclusion bodies as they are more present in early lesions. This explanation, however, contradicts Lever's 1961<sup>2,8</sup> view that colloid bodies are Fuelgen negative.

Dermis. It is commonly accepted that the lesional process in lichen planus primarily starts in the corium by perivascular round cell infiltration and vascular

dilatation resulting in papillary body enlargement. The infiltrate is quite sharply demarcated at its inferior border while having a hazy dermal epidermal border.

Regarding the nature of cellular infiltrate much controversy exists among various investigators. Pinkus 1903<sup>29</sup> described the infiltrate as comprising lymphocytes, plasma and endothelial cells with preponderance of leucocytes. While according to Fordyce 1910<sup>30</sup> the cells also form principal part of the infiltrate together with connective tissue cells. Torok<sup>31</sup> and Sabroud 1910<sup>32</sup> emphasized the mononuclear feature of the infiltrate. Occasional giant cells have been demonstrated by Sabroud 1910<sup>32</sup> and Seif-El-Nasr et al 1962<sup>22</sup>. The author, however, failed to demonstrate such cells except in one case (Fig. 13). Polymorphonuclear leucocytes were stated by Winer and Levitt 1947<sup>33</sup> to be usually present in early lesions. Lumpkin et al 1965<sup>34</sup> considered that one of the criteria for diagnosing lichen planus histopathologically was the absence of eosinophil leukocytes or plasma cells. The presence of plasma cells and absence of eosinophils was mentioned by Oberste & Lehn 1954 35. The author on the otherhand noted a few eosinophils in several tissue sections (Fig. 14). Diversity of opinion concerning the nature of cellular infiltrate has been attributed to examination of different lesions in different developmental stages2.

With the exception of lichen planopilaris, no hair follicle affection was in the lesional tissue sections examined, this is in disagreement with Crocker<sup>36</sup> and Little<sup>2</sup>, <sup>3</sup> who related the umblication of lichen planus papule present in some cases, to the presence in the center of the papule a hair follicle. Our finding of absence of hair follicles also disproves the belief of Bisiadecki and Crocker<sup>37</sup> that umblication of the papule is due to tetanic contraction of arrector pilorum muscle. Such umblication was attributed by Joseph<sup>38</sup> to absorption of pseudovesicles while Torok<sup>31</sup> described holding down of the center of the papule by the coil gland duct. (Fig. 15).

(2) Lichen planus pemphigoides. Several histopathological explanations for the bullous formation in lichen planus had been proposed. Allen 1901<sup>39</sup>, attributed the mechanism of vesicle formation to vitiated coil gland conforming with Crocker's 1900<sup>36</sup>. This was in agreement with some observations<sup>38</sup> of the centering of umblicated papule around sweat duct.

The presence of lacunae between the corium and the epidermis as a common microscopical finding were believed to lead to vesication from the exudate separating the basal cell layer from the round cell inflammatory zone. The occasional presence of intraepidermal clefts as described by Fordyce 1910<sup>30</sup>, Whitfield 1902<sup>40</sup> Reute (1929)<sup>41</sup>, and sometimes at the level of granular layer leading to vesicle formation, was against the lacunar explanation.

Lever 1961<sup>28</sup> believed that the destruction of basal cells results in hole formation which is the starting point in production of bullae; a view against which is the vesicle formation on the skin not involved with lichen planus papule. Blair 1948<sup>42</sup> however, demonstrated in the latter cases that the microscopic findings

were identical with those manifested when vesicles were superimposed on papules. This varifies the hypothesis that papules are not necessary prerequesites for vesicle formation.

(3) Lichen planopilaris. The atrophy in lichen planoplaris has been the matter of debate between various investigators. Silver and associates 1953<sup>43</sup> attributed such atrophy to the loss of pilosebaceous apparatus with which the scalp is very rich. They also considered loss of such pilosebaceous apparatus to be secondary incident resulting from the pressure of perifollicular infiltrate

Inflammatory reaction combined with counter pressure produced by hyper-keratosis, plugging the dilated follicle caused interference with vascular supply. Feldman 1936<sup>44</sup>, in this respect referred clinical atrophy to fragmentation and diminution of elastic fibres. He<sup>44</sup> added that these changes were present not only in the affected area but in the uninvolved areas as well. On the other hand Silver and co-workers 1953<sup>43</sup> stated that there was absence of elastic fibres in areas of cellular infiltrate attributing such phenomena to displacement. Waldorfe 1966<sup>45</sup> found in area of clinical absence of hair no evidence of scar formation; only complete absence of any remnant of pilosebaceous structure and presence of arrector pili muscles but no abnormalities detected in sweat glands and blood vessels.

In this regard the author attributed the atrophy in cases of lichen planoplaris to combination of previously mentioned factors, namely destruction of elastic fibers in the affected area and loss of pilosebaceous apparatus. The author thinks the destruction of elastic fibres plays a significant role in the production of atrophy due to the derangement of the high degree of elasticity of elastic fibres as a result of disease.

(4) Lichen planus actinicus Histopathologically lichen planus actinicus was almost compatible with the classical lichen planus (Fig. 3). A point of difference was first cleared by Seif-Ei-Nasr et al 1962<sup>22</sup> who noted a sparse infiltrate below the epidermis, a condition they called a space. They <sup>22</sup> also noted that the capillaries in the papillary, subpapillary layers were dilated at the sides of the lesion while at the center the endothelium was usually proliferated. In our series only 5 cases showed (Fig. 16) the presence of an area of normal connective tissue free from infiltrate below the epidermis while the rest of 7 cases showed a picture similar to that of lichen planus (Fig. 3). Also in the annular lesions, in addition, the dermal infiltrate was present in relation to the clinically elevated whitish blue edge while the darkly pigmented center showed no similar dermal infiltration, but only increase in pigment in basal cell layer and in dermal melanophores.

Sometimes, transtitional alteration in the picture of lichen planus actinicus occurs and ultimately leading to establishment of lesions histologically identical with chronic discoid lupus erythematosus and solar dermatitis<sup>47</sup>. Such observation is in agreement with the view of some investigators<sup>22</sup>, <sup>48</sup> that lichen planus

actinious should be grouped with light sensitive dermatores or considered to be a peculiar form of chronic polymorphic light eruption (Katzenellenbogen<sup>47</sup>). The author opines that this type has to be put as light sensitive dermatosis and its separation into an entity of lichen planus was due to its occurrence more in children and young adults<sup>18</sup>, <sup>47</sup>, <sup>48</sup>, characteristic geographical, racial and seasonal incidence<sup>22</sup>, <sup>47</sup>, <sup>48</sup> and the lack of chief symptom of lichen planus, the itching. <sup>18</sup>, <sup>22</sup>, <sup>47</sup>.

(5) Post lichen pigmentation. It has been noted in involutional changes that inflammatory reaction in the connective tissue subsides before epidermal involuting changes. This observation could be explained by the occurence initially of the inflammatory process in connective tissue, cometimes before the epidermis exhibits any clinically visible alteration. Such explanation is compatible with what has been frequently stated by different investigators that lesion in lichen planus is essentially lesion of the corium rather than epidermal pathology

The authors, observation of the presence of fine newly formed connective tissue fibres lying side by side with remnants of destroyed elements, disproves what has been stated by Winer 1955<sup>49</sup> that both collagen and elastic fibres are seen to disappear when there is plasma cell or round cell infiltrate. If actual disappearance of fibre elements has occured how could the presence of remnants of destroyed collagen and elastic fibres in addition to the new fibre elements be explained?

Summary. 57 cases of different types of lichen planus had been histopathologically studied. Trial of limitation of the abused term lichen was done. Review of the literature concerning the histopatholoic studies and their evaluation for different types of lichen planus had been done.

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