When Is Lichen Planus Lichen Planus*

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Dermatoses of diverse etiology have been reported to give rise to skin and mucous membrane lesions whose morphology, symptomatology, course, and microscopic picture cannot be distinguished from lichen planus. Drugs,\textsuperscript{1-8} vaccines,\textsuperscript{7} contact with chemicals,\textsuperscript{8-13} and exposure to ultraviolet light and Grenz rays\textsuperscript{14} are prominent among the causative agents which have been observed to produce lichenoid or lichen planus-like eruptions. The cause of lichen planus is not known, but the muco-cutaneous lesions produced by these agents can simulate it in minute detail. Could lichen planus then represent a symptom complex or disease which can result from diverse causes in susceptible individuals? It is the purpose of this paper to discuss this possibility.

DISCUSSION

Lichen Planus: The flat angular papule bearing an adherent scale with Wickham's striae is the classical lesion of lichen planus. Less commonly follicular accumulate spinous papules, bullous lesions, atrophic or hypertrophic lesions, and erosive lesions are found. Rings, plaques, and other morphological types commonly seen result from enlargement of coalescence of existing lesions. Itching is common and the Koebner phenomenon somewhat less so. Solitary lesions, lesions localized to a small area, or mililiary lesions involving almost all of the cutaneous surface may be found.\textsuperscript{15} Lichen planus has been reported in association with lichen nitidus, the malignant reticuloses, and skin cancer.\textsuperscript{16} Lesions of the shins and erosive mucosal lesions tend to persist, but spontaneous remission may be expected in two-thirds of the cases.\textsuperscript{17}

Mucous membrane lesions, which occur in about 30 per cent of the cases, may precede, accompany, or follow the appearance of the skin lesions and, in some instances, occur without them.\textsuperscript{17} The buccal mucosa is the most frequent site of oral lesions (85 per cent), but the tongue, floor of the mouth, gingiva, hard palate, and lower lip may also be involved. Symptoms with oral lesions are usually absent, but the sensation of burning or irritation may be present. Bluish-white reticulated striate, annular, or plaque-like lesions are the ones most frequently seen. Other mucosal sites in which lesions of lichen planus have been observed include the pharynx, larynx, stomach, rectum, bladder, vagina, vulva, and glans penis.\textsuperscript{8}

The nail changes most frequently observed are onychorrhexis, longitudinal splitting, central grooving, and thinning of the nail plate to the point of atrophy.\textsuperscript{18-20} The nail changes occur in approximately 10 per cent of the patients while the disease process is active. These and other nail changes, while not diagnostic, are highly characteristic of the disease. Lichen planus limited to the nails and confirmed by biopsy has been reported.\textsuperscript{21} All of the nails may be involved, but not at the same time or to the same degree. Discoloration, pitting, onycholysis, and shedding of the nail may also take place. Kilgman has expressed his opinion that onychial lichen planus is primarily a disease of the nail plate in contrast to fungus infections in which the nail bed is primarily involved.\textsuperscript{22}

Scalp lesions occur in somewhat less than 5 per cent of the patients.\textsuperscript{17} Known variably as follicular lichen planus, lichen plano-pilaris, or the Graham-Little syndrome, it occurs as follicular spinous papules predominantly in females (2:1).\textsuperscript{23} These lesions may be manifested by the triad of follicular accumulate papules, atrophy, and finally alopecia (4 per cent). Whereas cutaneous lichen planus may clear spontaneously, the scalp lesions seem to remain stationary or progress relentlessly.\textsuperscript{24} Typical cutaneous lesions may accompany those of the scalp, but more often they too are of the follicular accumulate or lichen spinulosus variety. Lichen plano-pilaris, among other disease processes, may eventuate into the pseudo pelade of Brocq.

The etiology of lichen planus is not known. Race, sex, and climate do not appear to bear any
relationship to the onset or course of the disease.\textsuperscript{17,20} Age may be a factor in that lichen planus is found most frequently in the young to middle-aged adult (ages 30-70). On occasion it has been reported in young children.\textsuperscript{7} Precipitating stressful situations have been observed by many investigators to precede or coincide with the onset or exacerbation of the disease. The death of a dear friend or relative, severe pressure on the job, and intolerable economic, social, or political pressures represent a few sample situations. Chronic nervous tension has similarly been incriminated as causal.

Lichenoid or Lichen Planus-like Eruptions: Numerous drugs,\textsuperscript{1-6,26} some chemical contactants,\textsuperscript{8,9,12} a vaccine,\textsuperscript{7} ultraviolet light and Grenz rays\textsuperscript{2,14} have been observed and reported to produce eruptions quite similar to, if not identical, with lichen planus. The clinical lesions produced by them have mimicked the classical form of the disease with respect to morphology, distribution, and symptoms. Pruritus and discomfort, frequent concomitants of lichen planus, have or have not accompanied the drug eruption. Gradual clearing following cessation of the drug has been the rule, but occasionally persistent hyperpigmentation or atrophy have resulted.\textsuperscript{27} A partial list of drugs so incriminated includes the following:

1. Penicillin
2. Bismuth
3. The antimalarial—Quinacrine and chloroquine, and quinine
4. Quinidine
5. Para-aminosalicylic acid and Amiphenazole
6. Barbiturates
7. Arsenic
8. Gold
9. Phenothiazines
10. Tetracycline

In the past 12-15 years many American, European, and Asian observers have reported cases of lichen planus-like eruptions among workers in the color photo industry. Evidently, prolonged contact over several months or years is necessary to produce the dermatoses with the chemicals CD-2, a Kodak product (3-methyl-4 amino-N-diethyl-aniline monohydrochloride), and TSS, an AGFA produce (4-amino-N-diethyl-aniline sulfate). The eruptions were not limited to areas of contact but eventually involved the backs of the hands, forearms, arms, abdomen, trunk, neck, face, legs, genitalia, and buccal mucosa. The lesions persisted for long intervals after these individuals withdrew from their sources of contact.\textsuperscript{8,9,12}

Ultraviolet light and Grenz rays have also been reported to give rise to dermatoses resembling lichen planus or else to produce isomorphic responses in already established cases.\textsuperscript{14} Lichen planus has developed in sites of herpes zoster treated extensively with x-rays or ultra-violet lights.\textsuperscript{8}

Gougerot and Civatte in 1953 cited their uncertainty whether lichen planus was a distinct skin disease or a dermatologic syndrome on special terrain secondary to diverse etiological agents.\textsuperscript{1} The uncertainty still exists. Most clinicians will nod in agreement that stressful situations have precipitated or possibly triggered attacks in susceptible patients. Yet, drugs (administered by various routes) and other agents have demonstrated their ability to trigger similar attacks in susceptible individuals.

Lichenoid drug eruptions, contact dermatoses, and photodermatoses must be at least in some measure, allergic dermatoses for the following reasons:

1) Long intervals exist between exposure and disease (incubation period?) 2) Shorter intervals are required for subsequent attacks. 3) The dosage needed to precipitate an initial attack varies, but subsequent attacks require small amounts. 4) Some drugs (e.g., the phenothiazaines) are known photosensitizers. 5) Lichenoid lesions produced by drugs are often allergic and are not expected pharmacological side effects. 6) The lichenoid contact dermatoses and photodermatoses lesions develop at mucocutaneous sites at a distance from contact. 7) Cross sensitivity with CD-2 to para phenylenediamine has been demonstrated.

None of the foregoing reasons offer direct proof of etiology. The editor of Yearbook of Dermatology, 1965, suggested that an allergic reaction probably precedes the appearance of lichen planus in at least some patients, and is probably caused by small molecular compounds in susceptible individuals.\textsuperscript{26}

The criteria for histological diagnosis of lichen planus have long been established and universally accepted.\textsuperscript{28} They consist of hyperkeratosis, keratotic plugging, hypergranulosis, acanthosis or atrophy of the epidermis, edema of the epidermis and upper dermis, liquefaction degeneration of the basal cell layer, band-like inflammatory infiltrate
of the upper corium of lymphocytes and histiocytes, and incontinence of pigment. Eosinophiles and plasma cells are found frequently in classical lichen planus but probably more so in the drug eruptions. The density of the dermal infiltrate often obscures all other structures. The microarchitecture of Wickam's striae possesses all of the foregoing features plus dimpling of the horny layer. The ungual pathology resembles that of the skin, whereas the scalp lesions tend to be perifollicular.

TREATMENT

Therapy in lichen planus and lichen planus-like eruptions is not altogether satisfactory, yet symptomatic relief or even cure may result from appropriate management. Caution must be used in prognosticating because of the great variability of the course of the disease.

Topical:
1. Shake lotions with phenol, menthol, or camphor may relieve itching considerably.
2. Menthol 1/4 per cent in triamcinolone .01 per cent may also be useful to allay pruritus.
3. Synalar .01 per cent, Cordran 0.5 per cent, or Aristocort 0.1 per cent creams under occlusive dressings are especially valuable for hypertrophic lesions or those localized to a small area.

Systemic:
1. ACTH, 80 units IM weekly for 4-6 weeks, IM Aristocort forte (triamcinolone diacetate), 80 mg q. 14 days, are useful for widespread or acute efflorescences.
2. Tranquilizers such as Atarax and/or the antihistamines may be useful as adjunctive therapy.
3. Intralesional injections for localized eruptions with Aristocort or Kenalog may bring prompt relief.

Oral lesions: Erosive lesions of the oral mucosa have been treated with considerable success with triamcinolone acetonide 0.1 per cent in orabase. To prevent recurrences, maintenance therapy b.i.d. may be necessary for weeks or months.

Radiation:
1. Localized areas may benefit from superficial x-ray or Grenz ray therapy, but hyperpigmentation may result.

Older modalities such as bistrimate, thorium-X, radicular x-ray therapy, or mercury salts are generally less effective than the newer ones. When drugs, contactants, or other agents are incriminated, their removal or discontinuance is imperative.

CONCLUSION

Drugs, chemicals, vaccines, and radiation have been observed and reported to give rise to skin and mucous membrane lesions clinically and histologically similar to and indistinct from lichen planus. When in those instances a cause can be established, they should be referred to appropriately as drug eruptions, contact dermatitis, or photodermatitis of a lichenoid type. Many of these are probably allergic dermatoses. Thus, lichen planus in the future may become a diagnosis of exclusion as more causes of lichenoid eruptions become known.

LITERATURE CITED
of the therapeutic task at hand. Reality is not ignored but the strengthening effect of separating intra-psychic conflict from external social pressure aids the patient in dealing with the often harsh reality.

Of all the strange and horrible outgrowths of slavery in America, this surely is the most bizarre—that fantasies of forbidden instinctual gratification should have become so intimately linked with the experience of "being Negro"—"Negroeness"—that 100 years later such fantasies offer dark children an additional barrier to psychological growth.

(Grier, from page 418)


(Srong et al., from page 451)

Metabolism and Vaginal Cytology in Humans. Metabolism, 8:709, 1959.


(Keene, from page 456)


