Review Article

Updates in causes, risk factors, diagnosis and management of lichen planus

Abstract:

A pigmented abnormal of lichen planus (LP) was first reported from India in 1974 by Bhutani *et al*. who coined the term LP pigmentosus (LPP) to rigidity a descriptive nomenclature to it. LP has a number of variants, one of which is LPP. This sickness has also later been described from the Middle East, Latin America, Korea, and Japan, especially in people with dimmer skin. Erythema dyschromicum perstans (EDP) is considered as the principal differential diagnosis of LPP. Other differences are fixed drug eruption, macular amyloidosis, urticaria-pigmentosa, tar melanosis, frictional melanosis, berloque dermatitis, pigmented beautifying dermatitis (Reihl's melanosis). Cutaneous LP typically clears spontaneously within 1 to 2 years, so management is aimed at reducing pruritus and time to resolution. For limited LP, first-line management is superpotent interesting steroids (clobetasol 0.05%) twice daily for 2 to 4 weeks.

Keywords: *Erythema dyschromicum perstans*, *lichen planus*, *lichen planus pigmentosus* lichenoid, LP, planopilaris, variants

Introduction:

Lichen planus (LP) is a mutual mucocutaneous disease affecting stratified squamous epithelia. The aetiology of the condition is intricate and multifactorial, with histopathological features more archetypal in cutaneous than mucosal lesions, where ulceration is more apparent. LP most normally affects middle-aged adults, in particular perimenopausal women and is rare in children. The scratches usually include the skin (cutaneous lichen planus), the oral cavity (oral lichen planus), the

genitalia (penile or vulvar lichen planus), the scalp (lichen planopilaris), nails (lichen unguis), or extracutaneously (e.g. the oesophagus). The diagnosis and controlling of lichen planus will be studied here.

Despite the high prevalence of the disease and the variability of therapeutic options available, no national or international evidence-based guidelines for management exist. That is why the European Dermatology Forum (EDF) introduced a project to develop guidelines for the treatment of lichen planus. Based on expert estimation and literature search, therapeutic recommendations were advanced through round mailing (Delphi method). This process was subject to an approval of the plans by all the memberships of the subcommittee.

The purpose of the guideline is to deliver all healthcare professionals with a tool for choosing an efficacious and safe therapy for various subcategories of patients, presenting with unlike subtypes of lichen planus. Healthcare professionals include dermatologists, dentists, gynaecologists, urologists, general physician in clinics, as well as in private practice and other specialists who are involved in the management of patients with lichen planus [1].

A pigmented variant of lichen planus (LP) was first described from India in 1974 by Bhutani *et al*. who coined the term LP pigmentosus (LPP) to bounce a descriptive nomenclature to it. LP has a number of variants, one of which is LPP. This sickness has also later been described from the Middle East, Latin America, Korea, and Japan, especially in people with darker skin. It has an insidious onset. Originally, small, black or brown macules appear on sun-exposed areas. They later combine to form large hyperpigmented patches. The disease principally touches the sun-exposed areas of the body such as the face, trunk, and upper edges. The oral mucosa may rarely be involved. However, the palms, soles, and nails are not pretentious. Histologically, the epidermis is atrophic along with vacuolar disintegration of basal cell layer. The dermis exhibits incontinence of pigment with disseminated melanophages and a sparse follicular or perivascular infiltrate. There is a considerable correspondence in histopathological findings among LPP and erythema dyschromicum perstans. However, there are immunologic and clinical alterations between the two. These

observations have led to a controversy regarding the uniqueness of the two entities. While some dermatologists consider them to be the similar, others have opined that the two should be considered as distinctly different diseases. A amount of relations such as hepatitis C virus infection, forward fibrosing alopecia, acrokeratosis of Bazex and nephrotic syndrome have been reported with LPP. A rare irregular, LPP inversus, with similar clinical and histopathological findings was described in 2001. As opposed to LPP, this variant occurs in covered intertriginous positions such as groins and axillae and mostly affects white-skinned individuals [2].

Definition:

Lichen planus I ,s an inflammatory skin illness affecting the skin, mucous membranes, hair and nails. The term lichen planus is derived from the Greek word 'leichen', which incomes *to lick or what eats around itself*, describing the characteristic way this skin illness appears and evolves and the Latin word 'planus', which means 'flat', depicting the exact appearance of this disorder. The dermatosis was first labelled by Erasmus Wilson in 1869 [3].

Lichen planus is a distinctive entity that affects numerous areas of the body, either concomitantly or sequentially. It is a chronic inflammatory sickness, with the exception of most cutaneous forms that often resolves spontaneously indoors one to two years. Skin hypertrophic and mucosal lichen planus is considered a probable premalignant condition, as the incidence of squamous cell carcinoma in these LP variants is around 1% [4-5].

Three systematic reviews were prepared on the behavior of oral and erosive mucosal LP, respectively [6-7], four on the treatment of cutaneous lichen planus [8-9] and unique on the therapeutic management of lichen planopilaris [10]. Data from randomized, measured trials are limited, and management choices are based mainly on clinical knowledge [11-12].

Etiology:

Lichen planus is an idiopathic disease. Its pathogenesis is not completely understood, but it appears to represent a T-cell-mediated autoimmune sickness. The prevailing theory is that exposure to an exogenous agent such as a virus, drug, or communication allergen causes alteration of epidermal self-antigens and activation

of cytotoxic CD8+ T cells. The altered self-antigens cross-react with ordinary self-antigens found on basal keratinocytes resulting in T-cell directing and apoptosis.[12]

A variety of agents have been associated with the expansion of LP, but a particular note has been made of the link with viruses, expressly the hepatitis C virus (HCV). Patients with LP are 5 times as likely to test progressive for HCV as the general population, and those with HCV-seropositivity are 2.5 to 4.5 whiles as likely to develop LP.[13-14]

Epidemiology:

Estimations on the incidence of lichen planus are among 0.14 and 1.27% of the general population [15]. At least two-thirds of the cases occur among the ages of 30 and 60 years. The disease is uncommon in children; however, it container occur at any age. No sexual or racial prevalence is obvious in the cutaneous form, whereas 60 to 75% of patients with oral lichen planus are females [16]. The commonness of oral lichen planus is approximately 1.5% [17]. Familial cases are infrequent, but have been labelled [18].

Risk Factors:

1. Immunogenetic Factors:

LP is a complex disease and thus can be instigated or triggered by genetic malfunction and/or environmental factors. The existence of ancestral cases of LP may suggest a possible genetic predilection. Gene polymorphisms of different HLA markers as well as the inflammatory cytokines and chemokines must been associated with the presence of LP. The interconnection of these polymorphisms, although unclear, ropes the autoantigen hypothesis.

2. Clinical Factors:

Associated factors and disease conditions seen in LP contain but are not limited to stress/anxiety, hepatitis C virus (HCV), autoimmune diseases, interior malignancies, dyslipidemia, and viral infections. Nervousness is a well-established risk factor or accompanying factor in LP patients. Some studies have specified that stressful events can induce LP lesions in otherwise healthy folks. In a case-control study, more than 67% of LP patients experienced a stressful event while nearby 21% of matched healthy controls experienced such events. Other studies more or less designate a similar trend for stress, anxiety, and depression. précises the coexistence of some clinical circumstances and LP.[46]

Histopathology:

Skin biopsy and microscopic analysis are valued in confirming the diagnosis in atypical and severe cases as the histopathologic features are principally the same regardless of the distribution or subtype. Principal findings entail of thickening of the stratum corneum without nuclei present (hyperkeratosis deprived of parakeratosis); irregular thickening of the stratum granulosum; liquefactive deterioration of the stratum basale; alteration or loss of rete ridges subsequent in a sawtooth appearance; and a dense band of lymphocytes infiltrating the dermis lengthways the dermo-epidermal intersection (interface dermatitis).[19-20]

Prognosis

The long-term outlook (prognosis) for people with lichen planus pigmentosus (LPP) is generally good. Aside from the characteristic macules and patches on the skin, many people do not have any other symptoms. In those who do experience itching and/or burning of the skin or other features of lichen planus, symptoms usually improve with treatment. Unfortunately, LPP is generally a chronic, relapsing condition with periods of exacerbations (worsening symptoms) separated by periods of remission (a decrease in or disappearance of symptoms).[48-49]

The various cutaneous and mucosal variants of lichen planus:

Oral lichen planus

Oral lichen planus (OLP) is a common display of lichen planus that can occur alone, but often occurs concurrently with skin lesions. The spoken form affects females more than males and normally affects patients of internal age. Exact prevalence is unknown, but has been estimated to be amid 0.5% and 2.6% of various populaces.

First labeled in 1869 by British physician Wilson Erasmus, lichen planus (LP) is an autoimmune condition current on the skin, hair, eyes, mucous membranes, and nails. LP lesions on the skin have a purplish elevated flat appearance with no particular pattern. When the lesions present in the oral cavity, it is mentioned to as oral lichen planus (OLP), with OLP being originate in 53.6% of cutaneous LP patients. OLP lesions appear as inflamed ulcerations that might have a white linear or lacy pattern. Usually, there is a constant attendance of the lesions; however, the lesions will not remain in one area of the mouth or skin and incline to migrate over time. The lesions characterize by remissions and flares, "flare," connotation that the lesions will become much more proliferative and sore. When OLP is present, it is challenging for the patient to eat, drink, and function because of continuous pain. OLP is more common in females ended the age of 40 and in non-Asian countries. It is a chronic T-cell mediated disease of the oral mucosa. Augmented numbers of mast cells with significant degranulation are a reliable finding in OLP. The mechanism of action appears to be mediated by an antigen-specific device that activates T-cells. There is also a non-specific device of mast cell degranulation. In a systematic review and meta-analysis, OLP had a worldwide occurrence of 1.01%. In another systematic review and meta-analysis of 46 studies, the general global prevalence of OLP was 0.89%.

There are numerous possible causative agents for LP/OLP, the most mutual being pharmaceuticals and dental materials that produce a lichenoid answer. [26]

Nail lichen planus

Nail involvement is a common appearance of disseminated LP, affecting up to 10% of patients with LP lesions involving other sites. Nail LP can also be the only manifestation of the condition. Nail LP typically looks during the fifth or sixth decades of life and affects masculinities equally.

Linear lichen planus

Linear LP is a rare variant that moves fewer than 1% of all patients with LP. However, up to 10% of Japanese patients with LP may have the line form. Gender predominance has not been described**.**

Annular lichen planus

Annular LP is a morphological variant of typical LP. Approximately 3 to 7% of patients with LP have the annular variant, although the true commonness is likely underrated.

Atrophic lichen planus

Atrophic LP is a rare irregular that may occur in areas previously affected by other LP variants. There are very few reports of atrophic LP in recent prose, making prevalence largely mysterious. Etiology of the atrophic subtype is not yet elucidated.

Hypertrophic lichen planus

The hypertrophic variant of LP, also baptized lichen planus verrucous or lichen planus hypertrophicus, is branded by thickened papules and plaques and is of unknown prevalence among adults. While meticulous pathophysiology has not been clarified, eosinophils may drama a larger role in hypertrophic LP than in other variants.

Inverse lichen planus

Inverse LP is a variant moving the intertriginous zones. Prevalence is unknown, as is the exact instrument behind these site-specific eruptions. Inverse LP lesions are confined to the intertiginous zones, counting the axillae, inguinal creases, gluteal cleft, limb flexures, and submammary area

Eruptive lichen planus

The eruptive variant of LP, also called exanthematous or generalized LP, has seldom been reported in English-language literature in adults. Etiology late the generalized nature of this form of LP is not well tacit.

Bullous lichen planus

The bullous form of LP is a rare variant considered by development of vesico-bullous lesions. Only a few cases have been reported in the prose, making prevalence difficult to estimate. However, the etiology of bullae development in bullous LP (BLP) is consistent with widespread vacuolar change of the basal cell layer.

Ulcerative lichen planus

The ulcerative or erosive variant of LP may be initiate on mucosal surfaces, but also occurs on the plantar surface of the feet. Frequency is unfamiliar. Etiology behind the ulcerative nature of this variant is poorly unspoken, but a potential inductive relationship amongst metoprolol and erosive LP may exist.

Lichen planus pigmentosus

Lichen planus pigmentosus (LPPi) is an unusual variant that affects all races but seems to favor darker-skinned individuals. It may be more mutual in Indian and Middle Eastern populations and may occur more regularly in females during the third and fourth periods of life .

Lichen planopilaris

Lichen planopilaris (LPP) is a morphological irregular of LP involving the hair follicles that has been classified as a chief lymphocytic cicatricial alopecia. It occurs more frequently in Caucasian and Indian populations, with lower frequency in Asian inhabitants.

Vulvovaginal lichen planus

Vulvovaginal LP is an uncommon variant that includes the vulva and vagina. This disease seems to largely affect Caucasian womenfolk of perimenopausal age. Most patients present in the sixth period of life.

Actinic lichen planus

Actinic LP, also called lichen planus subtropicus or lichen planus actinicus, is a erratic variant that affects sun-exposed areas of the skin. Gossips of actinic LP are more common in darker-skinned African, Middle Eastern, and Indian populaces, while very few cases have been described in Caucasians .

Lichen planus-lupus erythematosus overlay syndrome. Lichen planus-lupus erythematosus overlap syndrome is a rare variant that exhibitions features of both LP and lupus erythematosus in the same patient or in the identical lesion in a single enduring.

Lichen planus pemphigoides

Lichen planus pemphigoides (LPPe) is a infrequent autoimmune subepidermal blistering dermatosis that may be a single variant of LP or a heterogeneous blistering response to various antigens exposed by multiple injuries to the basal crust sector.

Pathophysiology:

The pathogenesis of LP remains unclear, but it is possible to be of a multifactorial nature. It is generally considered an immunologically mediated complaint. It affects surfaces roofed by stratified squamous epithelium.

There is evidence that cell-mediated immune reply plays a major role in the development of the disease. T cells, both CD4+ and CD8+, accrue in the dermis, while CD8+ T cells infiltrate the epidermis in LP lesions. The mainstream of lymphocytes in the LP infiltrate consists of CD8+ and CD45RO+ cells and couriers the α-β T-cell receptor (TCR), and to a lesser extent the γ-δ receptor.[21] These cells are responsible for the most characteristic change experimental in the lichenoid reaction, apoptosis.[22]

History and Physical:

Lichen planus can display a variety of lesion types, but the most mutual presentation is an zone of polygon-shaped, itchy, violaceous, flat-topped papules a few millimeters wide. This classic presentation is recognized as The Six Ps of LP: purple, polygonal, planar, pruritic papules, and plaques. The cuts have a shiny surface covered in fine white lines known as Wickham striae and are secure on palpation. They may be seen as a few individual lesions, found scattered broadly, grouped in plaques, or arranged in annular, linear, or actinic (sun-exposed) designs. The isomorphic response (i.e., Koebner phenomenon) can be seen in LP where new lesions arise in lines where scratching occurs, just as is understood in psoriasis. The most common areas of involvement include the flexor wrists, dorsal hands, inferior back, ankles, and shins. Regularly a grayish-brown hyperpigmentation can be found after lesions resolve due to deposition of melanin in the shallow dermis.[23-24]

Differential Diagnosis:

Erythema dyschromicum perstans (EDP) is measured as the principal differential diagnosis of LPP. Other differentials are fixed drug eruption, macular amyloidosis, urticaria-pigmentosa, tar melanosis, frictional melanosis, berloque dermatitis, pigmented cosmetic dermatitis (Reihl's melanosis), postinflammatory hyperpigmentation, and idiopathic eruptive macular pigmentation and hyperpigmentation due to drugs and weighty metals.[25]

Management:

Lichen planus is a chronic disease, and the main focus of treatment is to control symptoms and minimize damage. The management should be associated with the severity of the disease and the less conceivable side-effects and should improve the patients' superiority of life. In these guidelines, we give recommendations about management modalities of the various forms of LP trying to achieve the highest author's accord level in the order of penchant.

All the drugs, except topical steroid preparations, found off-label treatment modalities. Cutaneous LP typically clears spontaneously within 1 to 2 years, so

management is aimed at reducing pruritus and time to resolution. For limited LP, first-line usage is superpotent topical steroids (clobetasol 0.05%) twice daily for 2 to 4 weeks. Inadequate response to topical steroids may be increased with intralesional steroid injections (triamcinolone 5 to 10 mg/mL). For diffuse LP, first-line management is daily oral corticosteroids (prednisone 30 to 60 mg) elongated over 2 to 6 weeks. If no change is seen, second-line therapy should be considered. Second-line remedy may include metronidazole (500 mg twice daily for 3 to 8 weeks), sulfasalazine (500 mg twice daily increased in 500 mg increments every 3 days pending 2.5 grams daily is reached, for 3 to 6 weeks), isotretinoin (10 mg twice everyday for 2 months), acitretin (30 mg daily for 8 weeks), PUVA, UVB, up-to-date calcineurin inhibitors, or methotrexate (15 mg per week for adults, 0.25 mg/kg per week for children). Third line treatment may comprise trimethoprim-sulfamethoxazole, griseofulvin, terbinafine, antimalarials, tetracyclines, ciclosporin, mycophenolate mofetil, azathioprine, etanercept, adalimumab, or low-molecular-weight heparin.[27-28]

Oral LP may spontaneously resolution within 5 years, but many cases are chronic and never resolve. Treatment-induced remission is characteristically followed by relapse. Thus, asymptomatic oral LP should not be treated as the side-effect weight of treatment is high. The goal for treatment of indicative oral LP is to heal erosive lesions to reduce pain and allow normal food intake. Patients must be instructed to avoid spicy or acidic foods as fine as alcohol and tobacco as these exacerbate symptoms. First-line treatment is very high potency up-to-date steroids three times daily until remission. No improvement after 6 weeks should quick escalation of therapy. Second-line treatment is oral corticosteroids or claim of topical calcineurin inhibitors. Third-line treatment may comprise cyclosporine, azathioprine, mycophenolate mofetil, or methotrexate[29-30]

Consideration of drug-induced LP must always be travelled prior to starting therapy. Withdrawal of the suspected drug leading to the regular disappearance of lesions confirms the diagnosis, although it may take some time for grazes to fully resolve. [31]

LPP is a disease which is basically recalcitrant to management and therapies attempted in this disorder are quite ineffective. Vitamin A was optional by Bhutani *et al*. for the treatment of LPP. Other workers have claimed that up-to-date and systemic corticosteroids clear the lesions quickly. Al-Mutairi and El-Khalawany *et al*. found tacrolimus ointment (0.1%) to be effective in 53.8% of patients. A few suitcases have responded well to pigment laser. Sehgal *et al*. suggested a combination of oral diamino-diphenyl-sulfone (dapsone) along through oral immunomodulator, topical tacrolimus along with photoprotection for the management of LPP.[2]

A complete history and physical scrutiny are required, as well as identification of activating factors and comorbidities, such as variants of LP, hepatitis C virus-induced liver disease, endocrine disorders, and autoimmune diseases. This will permit prompt and appropriate treatment to prevent serious sequelae such as marking alopecia in frontal fibrosing alopecia. It is important to avoid identified triggers such as topical or systemic contactants, as well as sun experience.

Topical treatment includes medium to high effectiveness corticosteroids, tacrolimus, and skin lightening creams containing hydroquinone and retinoids. Solitary of the most commonly used topical treatments is tacrolimus. Universal treatment includes corticosteroids in pulse doses or continuous direction with gradual tapering, as well as dapsone. Isotretinoin has been newly reported as a promising therapy with a relatively better side effect shape.

Currently, it is unknown if it is appropriate to maintain anti-inflammatory conduct for a prolonged period of time to prevent relapses since this could be damaging by altering the migration and phagocytic function of macrophages, lengthening pigmentary incontinence, and consequently perpetuating pigmentation. Though slow to respond, a combination of topical and systemic conducts, as well as the avoidance of recognized triggers, will recover pigmentation and the aesthetic appearance in the majority of patients with LPP, thereby educating their quality of life. [48].

First-line treatments

Symptomatic treatment

Oral Antihistamines

Sedating antihistamines can be more effective in Spartan pruritus, but the reported adverse reports (safety problems/sleep disturbance/accidents) minimalize their use.

Topical antipruritic agents

Menthol, camphor, doxepin, polidocanol, etc., can be prearranged as an adjuvant to the main conduct.

Second-line treatments

Although numerous treatment modalities exist, the physician should reflect the benefits of the prescribed treatment against the possible side-effects, because cutaneous LP is a self-limited sickness with very few complications.[1]

CUTANEOUS LICHEN PLANUS TREATMENT:

Cutaneous lichen planus may determination spontaneously in one to two years, although lichen planus moving mucous membranes may be extra persistent and resistant to treatment. Recurrences are mutual, even with management. summarizes the management of nongenital cutaneous lichen planus scratches. High-potency topical corticosteroids are first-line remedy for cutaneous lichen planus[32-33]. Oral antihistamines (e.g., hydroxyzine [Vistaril]) may be used to switch pruritus. Hypertrophic lesions are preserved with intralesional triamcinolone acetonide (Kenalog), 5 to 10 mg per mL injection (0.5 to 1 mL per 2-cm lesion)[32]

Acitretin (Soriatane) is an expensive and toxic verbal retinoid that is secondhand in more severe cases of cutaneous lichen planus which do not respond to topical treatment.[32] Acitretin is a strong teratogen that remnants in the body for at least three months after the last dose; consequently, women who may developed pregnant are not candidates for the therapy. Acitretin is not permitted by the U.S. Food and Drug Administration (FDA) for the management of lichen planus, and the label includes an FDA boxed warning recommending that it be rummage-sale only by

physicians with experience treating severe psoriasis, recommending oral retinoids, and handling teratogenic capsules. Referral to a dermatologist is warranted for patients with severe lichen planus requiring systemic treatment with acitretin or an oral immunosuppressant.

For genital lichen planus lesions, triamcinolone ointment (Triderm) is a decent firstline agent. Topical tacrolimus (Protopic) and clobetasol (Temovate) seem to be effective actions for vulvovaginal erosive lichen planus.[34] Aloe vera gel has been deemed a safe and effective management for patients with vulvar lichen planus [35].Topical lidocaine (Xylocaine) may be rummage-sale as desirable for pain relief, and a water-based lubricant may be used to prevent pain throughout communication.

The scarring alopecia of lichen planopilaris is hard to converse. A case series showed that interesting high-potency corticosteroids and intralesional corticosteroids are commonly secondhand [36].

The aim of the administration of cutaneous lichen planus is to reduce itching and shorten the duration between onset of the disease and determination of the grazes.

Topical glucocorticoids are the treatment of choice, though their efficacy has not been proven in well designed, randomized, controlled trials. When interesting glucocorticoids are ineffective, oral corticosteroids are administered. Oral corticosteroids are also favorite from the beginning of treatment, when atrophic lesions appear early in the growth of the disease.

ORAL LICHEN PLANUS TREATMENT:

Mucosal LP is often difficult to treat, predominantly when ulcerations and erosions are present. For many years, treatment modalities for mucosal LP had been intended at palliation rather than cure of oral symptoms. However, existing treatments should intend to the elimination of symptoms and potentially reduce the jeopardy of malignant alteration.[1]

Various treatments have been employed to delicacy suggestive oral lichen planus, but complete resolution is problematic to achieve.[37] summarizes action options for oral lichen planus. Topical corticosteroids are first-line treatment.[32-38] High-potency topical steroids are the most effective, with response charges up to 75 percent likened with placebo.[39] Topical corticosteroids are too first-line therapy for mucosal erosive lichen planus. [32] High-potency corticosteroids practical to the oral mucosa do not appear to cause significant adrenal conquest, straight with comparatively long-term use. Systemic corticosteroids, such as oral prednisone, would be considered only for severe, prevalent oral lichen planus and for lichen planus involving other mucocutaneous positions.[40-32]

Topical calcineurin inhibitors, such as tacrolimus and pimecrolimus (Elidel), remain second-line treatments for oral lichen planus. [41-42] A comparative study presented that topical tacrolimus is as actual as the high-potency corticosteroid clobetasol in the treatment of oral lichen planus.[41-42] A randomized measured trial revealed that pimecrolimus 1% cream effectively treats erosive oral lichen planus with continuing therapeutic effects. [43][

In a randomized controlled trial, aloe vera gel was meaningfully more effective than placebo in the scientific and symptomatologic development of oral lichen planus.[44] If topical corticosteroids are ineffective, carbon-dioxide laser disappearance can lead to long-term remission of symptoms, and may be appropriate as first-line therapy in patients with tender oral lichen planus.[45]

General measures

Based on studies and expert opinions, dealings of general care can be discussed before the onset and during the management. Patients should be advised of the need to maintain good oral hygiene and to circumvent mucosal trauma. Depending on the severity of the disease, steady personal and professional dental care, replacement of amalgam or gilt dental restorations,[87] avoidance of smoking, spicy food and alcohol may be specified for some patients with oral lichen planus.

There is some evidence to advise that stress and anxiety are possible risk factors for the development of oral lichen planus (OLP). However, this connotation remains controversial.[88] It is assumed that psychological support may be useful to some patients with recurrent oral lichen planus. In case mucosal lesions continue despite treatment, frequent biopsies are necessary to discount malignant transformation.

Management of oral LP

First-line treatments

Topical application of potent or ultrapotent steroids is the backbone of treatment in the case of contained OLP. Clobetasol propionate 0.05%, triamcinolone, betamethasone, fluocinonide, fluticasone, dexamethasone and prednisolone in different forms have been proved to be effective and safe. They can be practical topically either in as lozenges. They have been also secondhand as an ointment, as an oral suspension or aqueous solution, pellets, aerosol or sprig, mouthwashes and usually in an adhesive paste. The frequency of claim and the duration of maintenance treatment is a topic of discussion. Usually, twice-daily request of topical steroids for 1-2 months, and then administered as needed, is a mutual practice.

Intralesional shot of corticosteroids (triamcinolone acetonide hydrocortisone, dexamethasone and methylprednisolone) in ulcerative OLP is also an active treatment approach. Injections can be painful; to avoid mucosal atrophy, we habitually administer a corticosteroid attenuation of 10 mg/mL.

Systemic corticosteroids, methylprednisolone or prednisone (30–80 mg/day) are the most effective treatment modality for patients with verbose recalcitrant erosive OLP or multisite lesions of severe erosive OLP. This must be used in short burst to induce remission rather than as a long-term maintenance treatment.

Systemic retinoids, such as acitretin (25–50 mg/day) initially, trailed by isotretinoin (0.5–1 mg/kg/day), have been used in the management of OLP. Topical retinoids (isotretinoin 0.05–0.1%) or other forms of vitamin A derivatives can eradicate white

lesions, but in all cases reported the lesions relapsed 2–5 weeks after termination of treatment.

Systematic use of cyclosporine (3–10 mg/kg/day) has been originate to be effective in different studies and for some authors is measured to be the drug of choice. Topical cyclosporine was used in the form of gargles or adhesive base, 2–3 times daily for 1 month. However, the application of cyclosporine answer proved to be less effective than the application of clobetasol or triamcinolone acetonide, with no important differences between the two managements. Furthermore, a large patient-to-patient variability regarding the efficacy of topical cyclosporine was experimental in both studies[1].

Second-line treatments

In OLP recalcitrant to topical corticosteroids, the use of interesting calcineurin inhibitors, tacrolimus and pimecrolimus, is recommended. Twice-daily application for 4–6 weeks has been proven innocuous and efficacious.

In few patients treated with topical calcineurin inhibitors, alteration in squamous cell carcinoma has been labelled, but it is not clear whether it can be attributed to the medications applied or to the sickness or to any other motive.[1]

Role of modern imaging techniques for the in vivo diagnosis of lichen planus: Lichen planus (LP) is a chronic inflammatory skin disease that can occasionally affect mucosal surfaces, with unknown pathogenesis, even though it looks to be an autoimmune disease. The diagnosis of lichen planus is usually founded on histopathological examination of the lesions. Nowadays, the traditional invasive diagnostic methods are replaced by modern non-invasive techniques. In this appraisal, we present the main non-invasive imaging methods (dermoscopy, reflectance confocal microscopy, optical consistency tomography, ultrasound and diffuse reflection spectrophotometry) used in the diagnosis and beneficial monitoring of lichen planus. Dermoscopy is a non-invasive process initially used for diagnosis of pigmented tumors but now is used also for inflammatory and transferable skin diseases. In lichen planus, the dermoscopy increases the accuracy

of diagnosis, avoids skin biopsies commonly rummage-sale and can be useful in the therapeutic monitoring by repeated investigation at different phases of treatment. Reflectance confocal microscopy (RCM) is a novel non-invasive imaging method that is prevalently used for the diagnosis of skin tumors and provocative skin diseases. This technology has been mostly employed for bedside, real-time minute evaluation of psoriasis, lichen planus, contact dermatitis, revealing detailed confocal features to support clinical diagnosis and assist with patient administration. Optical coherence tomography (OCT) is an emergent imaging technique, advanced over the last decade, based on the communication of the infrared radiation (900-1,500 nm) and the living tissues. A limited information exists on the assistances of OCT technology for the in vivo diagnosis of LP but could be a beneficial auxiliary tool in the in vivo differential diagnosis, especially in clinical equivocal sceneries like mucosal lesions, and in monitoring the response to treatment. Our review shows the opportunity of using modern imaging techniques for the in vivo identification and also for evaluation of the management response.[47]

Conclusion:

LPP a hyperpigmentary disorder related with lesions of LP was first reported from India in 1974 by Bhutani *et al*. Whether this was the same disorder of learned hyperpigmentation which had been reported periodically during the preceding four decades remains a matter of conjecture. After 1974, a controversy has exploded as to whether LPP and EDP are the same disorder. Lichen planus is an seditious skin disease with characteristic clinical and histopathological conclusions. In addition to classic LP, a myriad of LP variants exist, counting oral, nail, linear, annular, atrophic, hypertrophic, inverse, eruptive, bullous, ulcerative, LP pigmentosus, lichen planopilaris, vulvovaginal, actinic, LP-lupus erythematosus overlay syndrome, and LP pemphigoides. The pruritic, many-sided, violaceous, flat-topped papules and plaques of classic LP are the most common performance of the disease, but morphology and location vary importantly among the alternatives.

COMPETING INTERESTS DISCLAIMER:

Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

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