Review Article

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

Abstract:

Bhutani et al. originally described a pigmented variety of lichen planus (LP) from India in 1974, and created the label LP pigmentosus (LPP) to better describe it. LPP is one of several variations of the LP gene. This skin lesion has also been reported in the Middle East, Latin America, Korea, and Japan, and is more common in those with darker skin. The most common differential diagnosis for LPP is erythema dyschromicum perstans (EDP). Fixed drug eruption, macular amyloidosis, urticaria pigmentosa, tar melanosis, frictional melanosis, berloque dermatitis, pigmented beautifying dermatitis (Reihl's melanosis), and pigmented beautifying dermatitis (Reihl's melanosis) are some of the other differences. Cutaneous LP usually clears up on its own after 1 to 2 years, thus care focuses on lowering pruritus and speeding up the healing process. First-line management for limited LP is superpotent interesting steroids (clobetasol 0.05%) twice daily for 2 to 4 weeks.

This study aims to:

1- Recognize the causes and risk factors for lichen planus

2- Correct diagnosis of lichen planus

3- Provide effective treatment for lichen planus

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

Introduction:

Lichen planus (LP) affects stratified squamous epithelia and is a mutual mucocutaneous disease. Lichen planus (LP) is an autoimmune disease that affects the skin, hair, eyes, mucous membranes, and nails. It was first identified in 1869 by British physician Wilson Erasmus. On the skin, LP lesions have a purplish raised flat appearance with no discernible pattern. The aetiology of the illness is complex and multifaceted, with cutaneous lesions having more typical histological traits than mucosal lesions, which have more obvious ulceration. LP is most common in middle-aged adults, especially perimenopausal women, and is uncommon in children. The skin (cutaneous lichen planus), the oral cavity (oral lichen planus), the genitalia (penile or vulvar lichen planus), the scalp (lichen planopilaris), the nails (lichen unguis), or extracutaneously (e.g. the scalp) are the most common sites for scratching.

Despite the disease's significant prevalence and the wide range of therapy choices available, no national or international evidence-based management guidelines exist. As a result, the European Dermatology Forum (EDF) launched a study to produce treatment guidelines for lichen planus. Therapeutic recommendations were progressed by round mailing based on expert estimation and literature search (Delphi method). The plans had to be approved by all of the subcommittee's members before the process could begin.

The goal of the guideline is to provide all healthcare providers with a tool for selecting an effective and safe treatment for various subcategories of people who have lichen planus in different subtypes. Dermatologists, dentists, gynaecologists, urologists, and general practitioners work in clinics and in private practise. [1]

Bhutani et al. described a pigmented variation of lichen planus (LP) from India in 1974, coining the term LP pigmentosus (LPP) as a descriptive descriptor. LPP is one of several variations of the LP gene. This disease has also been reported in the Middle East, Latin America, Korea, and Japan, with darker skin being the most common. It creeps up on you slowly at first. Small black or brown macules form on sun-exposed areas at first. They eventually meld into enormous hyperpigmented regions. The condition primarily affects regions of the body that are exposed to the sun, such as

the face, trunk, and upper margins. Rarely, the oral mucosa is implicated. The palms, soles, and nails, on the other hand, are not ostentatious. In terms of histology, the epidermis is the outermost layer of the skin. The epidermis is atrophic histologically, with vacuolar breakdown of the basal cell layer. With dispersed melanophages and a scant follicular or perivascular infiltration, the dermis reveals pigment incontinence. LPP and erythema dyschromicum perstans have a lot in common when it comes to histological findings. There are, however, differences in immunology and clinical manifestations between the two. The uniqueness of the two entities has sparked debate as a result of these observations. While some dermatologists believe they are the same thing, others believe they are two distinct disorders. LPP has been linked to a number of conditions, including hepatitis C virus infection, forward fibrosing alopecia, Bazex acrokeratosis, and nephrotic syndrome. In 2001, the LPP inversus, a rare abnormality with identical clinical and histological features, was discovered. Unlike LPP, this form is seen in covered intertriginous areas such as the groynes and axillae, and it predominantly affects white people [2].

Lichen planus (LP) is a squamous epithelial inflammatory illness with an unknown cause. The oral mucosa is most commonly affected, however other mucosa and the skin may also be affected. The term lichen planus is derived from the Greek word 'leichen,' which means 'to lick or to eat around itself,' indicating the distinctive manner this skin disease appears and evolves, and the Latin word 'planus,' which means 'flat,' defining the disorder's specific look. Erasmus Wilson was the first to name the dermatosis [3].

Lichen planus is a unique condition that affects a variety of bodily locations simultaneously or sequentially. With the exception of most cutaneous types, it is a chronic inflammatory illness that usually heals spontaneously inside within one to two years. Because the prevalence of squamous cell carcinoma in these LP variations is roughly 1%, hypertrophic and mucosal lichen planus is thought to be a possible premalignant disease [4-5].

There have been three systematic reviews on the treatment of oral and erosive mucosal lichen planus [6-7], four reviews on the treatment of cutaneous lichen planus [8-9], and one review on the therapeutic management of lichen planopilaris [10]. Because the number of randomised, controlled studies is limited, management decisions are based mostly on clinical data [11-12].

Etiology:

The exact etiology of lichen planus is unknown at this time. Its cause is unknown; however it appears to be a T-cell-mediated autoimmune disease. Exposure to an exogenous substance, such as a virus, medication, or allergy, alters epidermal self-antigens and activates cytotoxic CD8+ T cells, according to popular belief. T-cell directing and apoptosis are caused by altered self-antigens reacting with regular self-antigens located on basal keratinocytes. [12]

A variety of substances have been linked to the spread of LP, but the association with viruses, namely the hepatitis C virus, has received special attention (HCV). Patients with LP are 5 times more likely than the general population to test positive for HCV, and those with HCV seropositivity are 2.5 to 4.5 times more likely to acquire LP [13-14].

Epidemiology and risk factors of PL:

Lichen planus is estimated to affect between 0.14 and 1.27 percent of the general population [15]. At least two-thirds of occurrences occur in people between the ages of 30 and 60. The condition is uncommon in youngsters, but it can strike anyone at any age. In the cutaneous form, there is no evident sexual or racial preponderance, whereas 60 to 75 percent of individuals with oral lichen planus are females [16]. Oral lichen planus affects about 1.5 percent of the population [17]. Although familial instances are uncommon, they have been documented [18].

Because LP is a complicated illness, it can be caused or provoked by hereditary or environmental factors. The presence of LP cases in the family tree might indicate a hereditary predisposition. The occurrence of LP must be linked to gene variations in distinct HLA indicators, as well as inflammatory cytokines and chemokines. The autoantigen theory is tied together by the connectivity of various polymorphisms, which is unknown.

Stress/anxiety, hepatitis C virus (HCV), autoimmune disorders, internal tumours, dyslipidemia, and viral infections are among the associated causes and disease conditions identified in LP. In LP patients, nervousness is a well-known risk factor or concomitant condition. According to certain research, stressful situations might cause LP lesions in otherwise healthy people. A case-control research found that more than 67 percent of LP patients encountered a stressful incident, whereas only 21% of matched healthy controls did [46].

Histopathology:

Because the histopathologic findings are mostly the same independent of distribution or subtype, skin biopsy and microscopic examination are valuable in confirming the diagnosis in unusual and severe cases. The stratum corneum thickening without nuclei (hyperkeratosis without parakeratosis); irregular thickening of the stratum granulosum; liquefactive deterioration of the stratum basale; alteration or loss of rete ridges resulting in a sawtooth appearance; and a dense band of lymphocytes infiltrating the dermis lengthways the dermo-epidermal intersection are the main findings (interface dermatitis) [19-20].

Fortunately, people with lichen planus pigmentosus (LPP) have an excellent long-term outlook (prognosis). Many people have no more symptoms beyond the telltale macules and patches on their skin. Symptoms of itching and/or burning of the skin, as well as other lichen planus symptoms, normally improve with therapy. Unfortunately, LPP is a relapsing, chronic disorder marked by times of aggravation (worsening symptoms) followed by periods of remission (a decrease in or disappearance of symptoms) [48-49].

Forms of LP:

Oral lichen planus (OLP) is a frequent kind of lichen planus that can appear on its own or in combination with skin lesions. Females are affected more than males, and patients of all ages are affected. The exact prevalence is unclear, however it has been estimated that between 0.5 and 2.6 percent of diverse populations are affected. OLP lesions appear as inflammatory ulcers that might be infectious or not. Lichen planus (LP) is an inflammatory disease that affects the skin, hair, eyes,

mucous membranes, and nails. It was first identified in 1869 by British physician Wilson Erasmus. On the skin, LP lesions have a purple raised flat appearance with no discernible pattern. Mouth lichen planus (OLP) occurs when the lesions appear in the oral cavity, and it affects 53.6 percent of cutaneous lichen planus patients. Inflamed ulcerations with a white linear or lacy pattern are common OLP lesions. The pleasant remark is usually there at all times; however, the lesions do not stay in one place in the mouth or on the skin and tend to move over time. Remissions and flares describe the lesions, with "flare" implying that the lesions will become considerably more proliferative and painful. Because of the constant discomfort, it is difficult for the patient to eat, drink, and function while OLP is present. OLP is more frequent in non-Asian nations and in women over the age of 40. It's an oral mucosa illness caused by persistent T-cell activation. In OLP, increased numbers of mast cells with considerable degranulation are a consistent observation. An antigen-specific mechanism that activates T-cells appears to be the mechanism of action. Mast cell degranulation also has a non-specific mechanism. OLP was found to have a global prevalence of 1.01 percent in a comprehensive review and meta-analysis. A comprehensive review and meta-analysis of 46 papers was conducted in another study, 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 lesion [26].

Nail involvement is a frequent symptom of disseminated LP, affecting up to 10% of individuals with LP lesions that have spread to other locations. Nail LP might occasionally be the only symptom of the disease. Nail LP is most common in people in their fifth or sixth decades of life, and it affects both men and women equally.

Linear LP is an uncommon form that affects less than 1% of all people with LP. However, the line form may be present in up to 10% of Japanese individuals with LP. There is no mention of gender preponderance.

The morphological variation of normal LP is an annular LP. The annular form affects between 3 to 7% of people with LP, while the exact frequency is likely underestimated.

Atrophic LP is an uncommon variety that can appear in places where other LP variants have previously been present. The incidence of atrophic LP is mostly unknown because to the scarcity of data in contemporary literature. The atrophic subtype's aetiology has yet to be determined.

The hypertrophic variety of LP is characterised by thicker papules and plaques and is also known as lichen planus verrucous or lichen planus hypertrophicus. It's not known how common it is. While the pathogenesis of hypertrophic LP is unknown, eosinophils may play a bigger role in this variation than in others.

The intertriginous zones are involved in Inverse LP. The frequency of these site-specific eruptions is unclear, as is the exact process driving them. The axillae, inguinal creases, gluteal cleft, limb flexures, and submammary region are all intertiginous zones where inverse LP lesions occur.

In adults, the eruptive type of LP, also known as exanthematous or generalised LP, has been recorded seldom in English-language literature. The specific cause of the disease is uncertain.

The bullous type of LP is an uncommon variety that manifests as vesico-bullous lesions. Because just a few cases have been recorded in the literature, it's impossible to assess prevalence. Bullae formation in bullous LP (BLP) is, nevertheless, linked to broad vacuolar changes in the basal cell layer.

The ulcerative or erosive variety of LP affects both the mucosal and plantar surfaces, with no recognised cause or frequency.

Lichen planus pigmentosus (LPPi) is a rare lichen planus pigmentosus form that affects people of all ethnicities but tends to favour those with darker skin. It may be more common in Indian and Middle Eastern communities, and it may strike women more frequently in their third and fourth decades.

Lichen planopilaris (LPP) is a morphological form of Lichen planopilaris (LP) that affects the hair follicles and is characterised as a primary lymphocytic cicatricial

alopecia. It is more common in Caucasian and Indian populations, with Asian residents having a lesser prevalence.

Vulvovaginal LP is a rare form that affects both the vulva and the vagina. This condition appears to mostly affect Caucasian perimenopausal women. The majority of patients are in their sixties.

Actinic LP, also known as lichen planus subtropicus or lichen planus actinicus, is an uncommon lichen planus variation that affects sun-exposed skin. The majority of instances of actinic LP have been reported in darker-skinned African, Middle Eastern, and Indian populations, with only a few occurrences reported in Caucasians.

Overlay syndrome of lichen planus with lupus erythematosus. Lichen planus-lupus erythematosus overlap syndrome is a rare form in which a patient or a single lesion has symptoms of both LP and lupus erythematosus.

Lichen planus pemphigoides (LPPe) is a rare autoimmune subepidermal blistering dermatosis that can be a single variety of lichen planus (LP) or a heterogeneous blistering reaction to numerous antigens exposed by several lesions to the basal crust sector.

Pathophysiology of LP:

The cause of LP is unknown, however it is most likely complex. It's usually thought to be an immune-mediated ailment. It affects stratified squamous epithelium-lined surfaces.

There is evidence that the disease's progression is aided by the cell-mediated immune system. In LP lesions, CD4+ and CD8+ T cells accumulate in the dermis, whereas CD8+ T cells penetrate the epidermis. The majority of lymphocytes in the LP infiltration are CD8+ and CD45RO+ cells that express the - T-cell receptor (TCR), as well as the - receptor to a lesser proportion. [21] Apoptosis, the most prominent experimental alteration in the lichenoid response, is caused by these cells [22].

Manifestations of LP:

Lichen planus can manifest itself in a variety of ways, but the most common is a zone of polygon-shaped, itchy, violaceous, flat-topped papules that are a few millimetres broad. The Six Ps of LP refers to the purple, polygonal, planar, pruritic papules, and plaques that characterise this typical appearance. On palpation, the wounds have a gleaming surface covered in thin white lines known as Wickham striae. They might appear as a few isolated lesions, clustered in plaques, or organised in circular, linear, or actinic (sun-exposed) patterns. In LP, when new lesions appear in lines where scratching occurs, the isomorphic response (also known as the Koebner phenomenon) can be visible, exactly as it is in psoriasis. The flexor wrists are one of the most typical regions of involvement. 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].

The most common differential diagnosis for LPP is erythema dyschromicum perstans (EDP). 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 heavy metals are some of the other differentials [25].

Management of different types of LP:

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' quality of life. In these guidelines, we give recommendations about management modalities of the various forms of LP.

All the drugs, except topical steroid preparations, are 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) prolonged 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 resolve 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 well as alcohol and tobacco as these exacerbate symptoms. First-line treatment is very high potency topical steroids three times daily until remission. No improvement after 6 weeks should lead to quick increased of therapy. Second-line treatment is oral corticosteroids or topical calcineurin inhibitors. Third-line treatment may comprise cyclosporine, azathioprine, mycophenolate mofetil, or methotrexate[29-30]

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

LPP is a disease which is basically recalcitrant to management and therapies tried in this disorder are quite ineffective. Vitamin A was optional by Bhutani et al. for the treatment of LPP. Other authors have claimed that topical 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 cases 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 provoking 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.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 retinoids. Solitary of the most commonly used topical treatments is tacrolimus. Universal treatment includes corticosteroids in pulse doses or continuous use with gradual tapering, as well as dapsone. Isotretinoin has been newly reported as a promising therapy.

Currently, it is unknown if it is appropriate to maintain anti-inflammatory use 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 drugs, as well as the avoidance of recognized triggers, will restore pigmentation and the aesthetic appearance in the majority of patients with LPP, thereby improving their quality of life. [48].

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.

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]

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, 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.

In oral type, 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].

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]

Conclusion:

Lichen planus is a type of inflammatory skin disease with distinct clinical and histological characteristics. Oral, nail, linear, annular, atrophic, hypertrophic, inverse, eruptive, bullous, ulcerative, LP pigmentosus, lichen planopilaris, vulvovaginal, actinic, LP-lupus erythematosus overlap syndrome, and LP pemphigoides are among the many LP types. Classic LP is characterised by pruritic, polygonal, violaceous, flat-topped papules and plaques, however the form and location of the variants differ significantly. The histological findings of the variations, on the other hand, are essentially consistent. While clinical examination may be sufficient in some situations, histological investigation is frequently useful in verifying the diagnosis of LP variations. The treatment of classic LP and its variants is largely constant, with topical TCS as the first-line therapy among a wide range of therapeutic modalities documented in the literature with varied degrees of efficacy. Finally, understanding

the characteristics of LP and its variants is critical for timely detection and appropriate management.

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.

References:

1-D. Ioannides,E. Vakirlis,L. Kemeny,B. Marinovic,C. Massone,R. Murphy,A. Nast,J. Ronnevig,T. Ruzicka,S.M. Cooper,R.M. Trüeb,R.M. Pujol Vallverdú,R. Wolf,M. Neumann(2020): European S1 guidelines on the management of lichen planus: a cooperation of the European Dermatology Forum with the European Academy of Dermatology and Venereology. First published: 17 July 2020 **https://doi.org/10.1111/jdv.16464** Citations: 30

2- **Aparajita Ghosh and Arijit Coondoo** (2016): Lichen Planus Pigmentosus: The Controversial Consensus

Indian J Dermatol. 2016 Sep-Oct; 61(5): 482–486. doi: 10.4103/0019-5154.190108

3-**Kanwar AJ, De D. Lichen planus in children.** ***Indian J Dermatol Venereol Leprol*** **2010;** 76**: 366– 372.**

4-Giuliani M, Troiano G, Cordaro M *et al*. Rate of malignant transformation of oral lichen planus: A systematic review. ***Oral Dis*** **2019;** 25**: 693– 709.**

5-Friedl TK, Flaig MJ, Ruzicka T, Rupec RA. Verrucous squamous cell carcinoma complicating hypertrophic lichen planus. Three case reports and review of the literature. *Hautarzt* 2011; **62**: 40– 45.

6- Thongprasom K, Carrozzo M, Furness S, Lodi G. Interventions for treating oral lichen planus. *Cochrane Database Syst Rev* 2011; (7); CD001168.

7-García-Pola MJ, González-Álvarez L, Garcia-Martin JM. Treatment of oral lichen planus. Systematic review and therapeutic guide. *Med Clin (Barc)* 2017; **149**: 351– 362.

8-Antiga E, Caproni M, Parodi A, Cianchini G, Fabbri P. Treatment of cutaneous lichen planus: an evidence based analysis of efficacy by the Italian Group for Cutaneous Immunopathology. *G Ital Dermatol Venereol* 2014; **149**: 719– 726.

9-Atzmony L, Reiter O, Hodak E, Gdalevich M, Mimouni D. Treatments for cutaneous lichen planus: a systematic review and meta-analysis. *Am J Clin Dermatol* 2016; **17**: 11– 22.

10-Errichetti E, Figini M, Croatto M, Stinco G. Therapeutic management of classic lichen planopilaris: a systematic review. *Clin Cosmet Investig Dermatol* 2018; **27**: 91– 102.

11-Le Cleach L, Chosidow O. Clinical practice. Lichen planus. *N Engl J Med* 2012; **366**: 723– 732.

12-10Fazel N. Cutaneous lichen planus: A systematic review of treatments. *J Dermatol Treat* 2015; **26**: 280– 283.

12-Shiohara T, Moriya N, Mochizuki T, Nagashima M. Lichenoid tissue reaction (LTR) induced by local transfer of Ia-reactive T-cell clones. II. LTR by epidermal invasion of cytotoxic lymphokine-producing autoreactive T cells. J Invest Dermatol. 1987 Jul;89(1):8-14. [PubMed]

13-Le Cleach L, Chosidow O. Clinical practice. Lichen planus. N Engl J Med. 2012 Feb 23;366(8):723-32. [PubMed]

14-Alaizari NA, Al-Maweri SA, Al-Shamiri HM, Tarakji B, Shugaa-Addin B. Hepatitis C virus infections in oral lichen planus: a systematic review and meta-analysis. Aust Dent J. 2016 Sep;61(3):282-7. [PubMed]

15-Giuliani M, Troiano G, Cordaro M *et al*. Rate of malignant transformation of oral lichen planus: A systematic review. *Oral Dis* 2019; **25**: 693– 709.

16-Le Cleach L, Chosidow O. Clinical practice. Lichen planus. *N Engl J Med* 2012; **366**: 723– 732.

17-McCartan BE, Healy CM. The reported prevalence of oral lichen planus: a review and critique. *J Oral Pathol Med* 2008; **37**: 447– 453.

18-Pittelkow MR, Daoud MS. Lichen planus. In GK Wolff, L Goldsmith, S Katz, B Gilchrest, A Paller, eds. *Fitzpatrick's Dermatology in General Medicine*, 7th edn, Mc-Graw-Hill, New York, 2008: 244– 255.

19-Scully C, el-Kom M. Lichen planus: review and update on pathogenesis. J Oral Pathol. 1985 Jul;14(6):431-58. [PubMed]

20-Van den Haute V, Antoine JL, Lachapelle JM. Histopathological discriminant criteria between lichenoid drug eruption and idiopathic lichen planus: retrospective study on selected samples. Dermatologica. 1989;179(1):10-3. [PubMed]

21-Wenzel J, Tuting T. An IFN-associated cytotoxic cellular immune response against viral, self-, or tumor antigens is a common pathogenetic feature in "interface dermatitis". *J Invest Dermatol* 2008; **128**: 2392– 2402.

22-Giuliani M, Troiano G, Cordaro M *et al*. Rate of malignant transformation of oral lichen planus: A systematic review. *Oral Dis* 2019; **25**: 693– 709.

23-Le Cleach L, Chosidow O. Clinical practice. Lichen planus. N Engl J Med. 2012 Feb 23;366(8):723-32. [PubMed]

24-Boyd AS, Neldner KH. Lichen planus. J Am Acad Dermatol. 1991 Oct;25(4):593-619. [PubMed]

25- Sehgal VN, Verma P, Bhattacharya SN, Sharma S, Rasool F. Lichen planus pigmentosus. *Skinmed.* 2013;11:96–103. [PubMed] [Google Scholar]

26-

27- Atzmony L, Reiter O, Hodak E, Gdalevich M, Mimouni D. Treatments for Cutaneous Lichen Planus: A Systematic Review and Meta-Analysis. Am J Clin Dermatol. 2016 Feb;17(1):11-22. [PubMed]

28- Le Cleach L, Chosidow O. Clinical practice. Lichen planus. N Engl J Med. 2012 Feb 23;366(8):723-32. [PubMed]

29- Thongprasom K, Carrozzo M, Furness S, Lodi G. Interventions for treating oral lichen planus. Cochrane Database Syst Rev. 2011 Jul 06;(7):CD001168. [PubMed]

30- Giannetti L, Dello Diago AM, Spinas E. Oral Lichen planus. J Biol Regul Homeost Agents. 2018 Mar-Apr;32(2):391-395. [PubMed]

31- Seehafer JR, Rogers RS, Fleming CR, Dickson ER. Lichen planus-like lesions caused by penicillamine in primary biliary cirrhosis. Arch Dermatol. 1981 Mar;117(3):140-2. [PubMed]

32- Cribier B, Frances C, Chosidow O. Treatment of lichen planus. An evidence-based medicine analysis of efficacy. *Arch Dermatol*. 1998;134(12):1521–1530.

33-Carbone M, Arduino PG, Carrozzo M, et al. Topical clobetasol in the treatment of atrophic-erosive oral lichen planus: a randomized controlled trial to compare two preparations with different concentrations. *J Oral Pathol Med*. 2009;38(2):227–233.

34- Jensen JT, Bird M, Leclair CM. Patient satisfaction after the treatment of vulvovaginal erosive lichen planus with topical clobetasol and tacrolimus: a survey study. *Am J Obstet Gynecol*. 2004;190(6):1759–1763.

35-Rajar UD, Majeed R, Parveen N, Sheikh I, Sushel C. Efficacy of aloe vera gel in the treatment of vulval lichen planus. *J Coll Physicians Surg Pak*. 2008;18(10):612–614.

36- Cevasco NC, Bergfeld WF, Remzi BK, de Knott HR. A case-series of 29 patients with lichen planopilaris: the Cleveland Clinic Foundation experience on evaluation, diagnosis, and treatment. *J Am Acad Dermatol*. 2007;57(1):47–53.

37-Thongprasom K, Chaimusig M, Korkij W, Sererat T, Luangjarmekorn L, Rojwattanasirivej S. A randomized controlled trial to compare topical cyclosporin with triamcinolone acetonide for the treatment of oral lichen planus. *J Oral Pathol Med*. 2007;36(3):142–146.

38-Carbone M, Arduino PG, Carrozzo M, et al. Topical clobetasol in the treatment of atrophic-erosive oral lichen planus: a randomized controlled trial to compare two preparations with different concentrations. *J Oral Pathol Med*. 2009;38(2):227–233.

39- Voûte AB, Schulten EA, Langendijk PN, Kostense PJ, van der Waal I. Fluocinonide in an adhesive base for treatment of oral lichen planus. A double-blind, placebo-controlled clinical study. *Oral Surg Oral Med Oral Pathol*. 1993;75(2):181–185.

40- **10.** Lodi G, Scully C, Carrozzo M, Griffiths M, Sugerman PB, Thongprasom K. Current controversies in oral lichen planus: report of an international consensus meeting. Part 2. Clinical management and malignant transformation. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2005;100(2):164–178.

41-Corrocher G, Di Lorenzo G, Martinelli N, et al. Comparative effect of tacrolimus 0.1% ointment and clobetasol 0.05% ointment in patients with oral lichen planus. *J Clin Periodontol*. 2008;35(3):244–249.

42- Radfar L, Wild RC, Suresh L. A comparative treatment study of topical tacrolimus and clobetasol in oral lichen planus. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2008;105(2):187–193.

43-Volz T, Caroli U, Lüdtke H, et al. Pimecrolimus cream 1% in erosive oral lichen planus—a prospective randomized double-blind vehicle-controlled study [published correction appears in *Br J Dermatol*. 2008;159(4):994]. *Br J Dermatol*. 2008;159(4):936–941.

44- Choonhakarn C, Busaracome P, Sripanidkulchai B, Sarakarn P. The efficacy of aloe vera gel in the treatment of oral lichen planus: a randomized controlled trial. *Br J Dermatol*. 2008;158(3):573–577.

45-van der Hem PS, Egges M, van der Wal JE, Roodenburg JL. CO2 laser evaporation of oral lichen planus. *Int J Oral Maxillofac Surg*. 2008;37(7):630–633.

46-Gorouhi,F., Davari, P., Fazel, N., (2014): Cutaneous and Mucosal Lichen Planus: A Comprehensive Review of Clinical Subtypes, Risk Factors, Diagnosis, and PrognosisVolume 2014 |Article ID 742826 | https://doi.org/10.1155/2014/742826