CLINICOPATHOLOGICAL CORRELATION OF ORAL LICHEN PLANUS

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CERTIFICATE

This is to certify that **Dr. G.SUCHITHRA** PG Student (2005 - 2008) in the department of Dermatology, Stanley Medical College, Chennai has done this dissertation titled "**CLINICO PATHOLOGICAL CORRELATION OF ORAL LICHEN PLANUS"** under direct guidance and supervision in partial fulfilment of the regulations laid down by the TamilNadu Dr.M.G.R.Medical University, Chennai for M.D. Br.XII-A, Dermatology, Venereology & Leprology degree examination.

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I, **Dr. G.SUCHITHRA** hereby declare that the Dissertation titled "**CLINICO PATHOLOGICAL CORRELATION OF ORAL LICHEN PLANUS"** was entirely done by me, under the guidance of **PROF. KIRUBA MOHAN,M.D.,D.D.** at Government Stanley Medical College and Hospital, in partial fulfillment of regulation for MD Dermatology, Venereology & Leprosy Degree Examination of the Tamil Nadu Dr.M.G.R. Medical University to be held in March 2008.

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CONTENTS

INTRODUCTION

Oral lichen planus is a chronic inflammatory disease of oral mucosa with associated skin involvement. Oral lichen planus is difficult to treat and typically lasts longer than cutaneous lichen planus. It is a disease of remissions and exacerbations and is commonly a disease of the elderly.

The oral mucosa forms an interface between the milieu interior and the external environment. The clinical and morphological changes seen in the oral mucosa are due to the interaction of both extrinsic and intrinsic forces. Careful examination of the oral cavity should be an integral part of a detailed physical examination.

Lichen planus has a significant association of the skin lesions with those of mucous membrane. The diagnosis of oral lichen planus may pose a challenge because of the difficult visualization of the oral lesions. Other factors that interfere with diagnosis are alteration in the original

appearance due to maceration from moisture, abrasion due to sharp tooth, food and infections.

Oral lichen planus is often diagnosed in dental clinics during routine check up, because asymptomatic patients may be unaware of the lesions in the mouth. Patients with severe types like erosive lichen planus present earlier to a dermatologist.

The pathological significance of the disorders affecting the oral mucosa may be a reflection of internal disease. Oral lichen planus has a premalignant potential. Hence, knowledge about the various precipitating factors and modes of presentation of oral lichen planus may help in predicting the course of the disease. Further, it helps in early treatment and prevention of further complications.

Bearing this in mind, an analytical study of oral lichen planus has been done and presented here…….

REVIEW OF LITERATURE

DEFINITION

Lichen planus is a chronic, autoimmune, mucocutaneous disease which can affect the oral mucosa, skin, genital mucosa, scalp and nails. Oral lichen planus can occur without skin lesions or skin lesions may precede, follow or appear at the same time as those in the oral cavity. Oral lichen planus has most often been reported in middle aged patients, more commonly in females than males.

HISTORICAL ASPECTS

Lichen Planus was first described in 1869 by Erasmus Wilson[1]. Wickham described the characteristic striae in 1895 and Graham little described scalp involvement in lichen planus in 1919. The histological findings in lichen planus were defined in 1909 by Darier[1].

The word "Lichen" is derived from a Greek verb 'leikhein' which means "to lick". In latin it denotes a kind of plant. This word is used as a noun in Greek and Latin to

denote those symbiotic forms of life (combined growth of algae and fungus) that are now called lichens.

Trautmann described the vesiculo bullous form of lichen planus in the oral mucosa in 1911[2]. lichen planus has also been reported involving other mucous membranes. The occurrence of oral lesions in the absence of dermal lesions in lichen planus was first pointed out by Audry (1894)[2]

EPIDEMIOLOGY

Erasmus Wilson, in his original description of lichen planus reported two cases with tongue lesions among fifty patients with lichen planus. Oral involvement in lichen planus is reported in 10% to 50% of patients. The prevalence of oral lichen planus is 2.6% in the Indian population[3] and 1.9% in Swedish population[4].

Oral lichen planus occurs as a disease of adults, aged twenty and above with most between the age of forty and sixty. An overall female preponderance has been noted in oral lichen planus.

ETIOLOGY

The precise etiology of oral lichen planus is unknown. However, several predisposing factors have been implicated in the pathogenesis of oral lichen planus.

They are as follows :

- Autoimmunity
- Drugs
- Dental Materials
- Stress
- Hepatitis C Virus
- Genetics
- Miscellaneous
  - Tobacco Chewing
  - Human Papilloma Virus
  - Helicobacter pylori
  - Human Herpes Virus
  - Graft Versus Host Disease (GVHD)

AUTOIMMUNITY

The oral lichen planus antigen may be a self peptide or an altered self peptide. The role of autoimmunity in the pathogenesis of oral lichen planus is supported by many of its autoimmune features such as chronicity, onset in adults, prediliction for females, association with other autoimmune disorders, occasional tissue type associations, depressed immune suppressor activity in the affected patients and the presence of autocytotoxic T-cell clones in the Lichen Planus lesions [1].

It has been suggested that keratinocytes express a lichen planus antigen but only at the lesional site i.e., the clinical distribution of lichen planus lesions is determined by the distribution of lichen planus antigen. Hence, an early event in lichen planus lesion formation may be keratinocyte antigen expression or unmasking, at the future lesion site which may be induced by systemic drugs, allergens in dental restorative materials or tooth pastes, mechanical trauma, bacterial or viral infection or an unidentified agent.

Various autoimmune disorders associated with lichen planus are alopecia areata, dermatomyositis, hashimoto's thyroiditis, lupus erythematosus, myasthenia gravis, pemphigus vulgaris, pernicious anaemia, rheumatoid arthritis, scleroderma and vitiligo.

DRUGS

The drugs implicated in oral lichen planus are anti hypertensives and anti arthritic agents and a number of miscellaneous groups such as antimalarials and phenothiazines. These include beta blockers, captopril, methyl dopa, spirinolactone, arsenic, gold, naproxen, NSAIDs, penicillamine, allopurinol, quinidine, tetracycline, sulphonyl ureas like chlorpropamide and tolbutamide[6, 9].

Grinspan's syndrome, which is a triad of oral lichen planus, diabetes mellitus and hypertension, could be an iatrogenically induced syndrome since drug therapy for both the conditions can result in oral lichen planus[7, 8].

Lichenoid drug eruptions of the mouth and the skin can look like classical lichen planus and are not necessarily atypical in appearance. Clinical identification of lichenoid drug eruption has been largely on

subjective criteria. Oral lichenoid lesions may be unilateral. Oral lichenoid eruptions may have more diffuse lymphocyte infiltrate and contain eosinophils and plasma cells and there may be more colloid bodies than in classical lichen planus.

DENTAL MATERIALS

Dental materials such as amalgam, gold, silver and composite restorations have been associated with oral lichen planus. Amalgam restorations induce oral lichen planus. Thornhill et al found that 3.9% of patients with oral lichen planus were patch test positive for amalgam or mercury. Dental materials like mercury and silver are implicated in the etiopathogenesis of oral lichen planus due to their electrogalvanic effect[4,10,11]. Metals like nickel, gold, palladium, cobalt or copper released from certain dental cast alloys and orthodontic appliances have been reported to play a role in pathogenesis of oral lichen planus and lichenoid reations [12].

Oral lichen planus occurs due to cell mediated contact hypersensitivity to dental materials in susceptible individuals who have been sensitized through long exposure. Dental materials in direct contact with oral mucosa may directly alter the antigenicity of basal keratinocytes by the release of mercury[4].

STRESS

Stress has been widely held to be an important etiological factor in oral lichen planus. Exacerbations of oral lichen planus have been linked to periods of psychological stress and anxiety. A study by Ivanovski et al in 2005 shows that prolonged emotional stress in many oral lichen planus patients leads to psychosomatization contributing to initiation and clinical expression of oral lichen planus[13]. On the contrary, Humphris and Macleod reported no statistically significant association between oral lichen planus and anxiety[4,14,15,16].

HEPATITIS C VIRUS

The association between oral lichen planus and hepatitis C virus was first reported in France by Mokni et al in 1991. Carorozzo et al have demonstrated a strong association between hepatitis C virus infection and oral lichen planus and also an association with HLA-DR6 allele[17]. The association of oral lichen planus with hepatitis C virus infection seems to be dependent on geographical heterogenecity and is more common in the Mediteranean and Japan.

The proposed mechanism for the causation of lichen planus by hepatitis C virus are as follows : (a) HCV is capable of cytopathic replication in cell types outside the liver (b) It may trigger an autoimmune response that is directed against antigens expressed on extra hepatic cells (c) Persistent infection can lead to immune complex formation with antibodies, followed by deposition on small blood vessels (d) The trigger of immunological processes leading to dermatological manifestations are the activated $CD8^+$ T cells, cytokines and expansion of certain B cell clones[18,20].

Among the clinical types of oral lichen planus, reticular type is common in HCV positive patients while plaque type is common in HCV negative patients. No significant difference is seen with erosive or atrophic types of oral lichen planus[19].

GENETICS

Genetic background seems to play a role in oral lichen planus pathogenesis as several familial cases have been reported. An increased frequency of HLA-DR3 was found in erosive oral lichen planus. The familial cases of lichen planus differ from the classic non familial form of the disease by affecting younger persons and usually presenting with a great number of lesions[21,22].

Familial cases of oral lichen planus have a frequent expression of HLA-B7 antigens. In non familial cases, an association between the disease and HLA-DRw9/DR9 in Asian patients has been described. HLA –B8 is more common in patients with oral lichen planus as a sole manifestation[1].

MISCELLANEOUS

Tobacco chewing

Oral lichen planus has been reported among Indian betel-tobacco chewers during an epidemiological study in Kerala. The prevalence was 1.5% among the villagers of Kerala. Patients presented with white, linear, parallel streaks in the buccal mucosa[23,24].

Human papilloma virus

In a study published in 1990, the presence of human papilloma virus types 6, 11 and 16 were demonstrated in 20 patients with oral lichen planus[25].

Helicobacter pylori

Helicobacter pylori is one of the most important bacterial etiologies that has been suggested in the pathogenesis of oral lichen planus. A study in Iranian population demonstrated active Helicobacter pylori infection in 82.5% of patients with lichen planus[26,27].

Human herpes virus

Human herpes virus 7 may play a role in pathogenesis of oral lichen planus. HHV 7 DNA has been detected in lichen planus lesions[28].

Graft versus host disease (GVHD)

GVHD is a common but serious complication following allogenic bone marrow transplantation. GVHD is initiated by donor T-cells that recognize a subset of host peptide called minor histocompatibility antigens (miHAs). Oral involvement occurs in 33% to 75% of patients with acute GVHD , while it is up to 80% in chronic GVHD.

Oral mucosal GVHD resembles oral lichen planus both clinically and histologically. Although the antigen specificity of lichen planus and mucocutaneous GVHD are probably distinct, it is likely that they share similar immunological effector mechanisms, resulting in T-cell infiltration, basal keratinocyte apoptosis, epithelial basement membrane disruption and clinical disease[29].

PATHOGENESIS

Antigen Specificity

The following factors are responsible for antigen specificity in oral lichen planus:

1. CD 8+ T-cells.
2. Initial approximation between CD 8+ T-cells and keratinocytes.
3. Heat shock proteins.
4. Mechanisms of keratinocyte killing.
5. CD 4+ T-cells.
6. Types of antigen presentation.

CD 8^+ T-Cells

The lymphocytic infiltrate in oral lichen planus is composed almost exclusively of T-cells, and the majority of T-cells within the epithelium and adjacent to damaged basal keratinocytes are activated $CD8^+$ lymphocytes. The activated $CD8^+$ T-cells trigger keratinocyte apoptosis in oral lichen planus. The majority of cytotoxic clones from lichen planus lesions were $CD8^+$, and the majority of non-cytotoxic

clones were $CD4^+$. The cytotoxic activity of $CD8^+$ lesional T-cell clones was partially blocked with anti-MHC class 1 monoclonal antibody.

Hence, early in oral lichen planus lesion formation, $CD8^+$ lesional T-cells may recognize an antigen associated with MHC class 1 on lesional keratinocyte. Following antigen recognition and activation, $CD8^+$ cytotoxic T-cells may trigger keratinocyte apoptosis. Activated $CD8^+$ T-cells may release chemokines that attract additional lymphocytes and other immune cells into the developing oral lichen planus[30].

Initial Approximation between $CD8^+$ T-Cells and Keratinocytes

Following altered keratinocyte antigen expression, an antigen-specific $CD8^+$ T-cell may be either (1) on routine surveillance in the epithelium and encounter the keratinocyte antigen by chance ("CHANCE ENCOUNTER" hypothesis) or (2) attracted to the epithelium, along with T-cells of irrelavent specificity, by keratinocyte derived chemokines ("DIRECTED MIGRATION" hypothesis). The "chance encounter" hypothesis is supported by finding of $CD8^+$ T-cells in normal human epidermis and basal cell degeneration in the absence of a dense inflammatory infiltrate in lichen planus lesions. Conversely, the "directed

migration" hypothesis is supported by finding of constitutive chemokine receptor expression on naïve T-cells and a dermal T-cell infiltrate prior to the appearance of intra-epithelial lymphocytes and epithelial damage in lichen planus lesions.

The "directed migration" hypothesis predicts that keratinocyte-derived chemokines attract T-cells of irrelavent specificity along with antigen-specific T-cells into the developing oral lichen planus lesion. In summary, the initial event in oral lichen planus lesion formation may be keratinocyte antigen expression in association with MHC class 1 at the future lesion site, with or without up-regulated keratinocyte chemokine production.This is followed by keratinocyte apoptosis triggered by antigen-specific $CD8^+$ cytotoxic T-cells[30].

Heat Shock Proteins(HSP)

Oral lichen planus lesional keratinocytes express up-regulated Heat Shock Proteins (HSP). Keratinocyte HSP expression in oral lichen planus may be an epiphenomenon associated with pre-existing inflammation. Alternatively, up-regulated HSP expression by oral mucosal keratinocytes may be a common final pathway linking a variety of exogenous agents (systemic drugs, contact allergens, mechanical trauma etc.,) in the

pathogenesis of oral lichen planus. The HSP expressed by oral keratinocytes may be auto-antigenic[30].

Mechanism of Keratinocyte Killing

The exact mechanism used by $CD8^+$ cytotoxic T-cells to trigger keratinocyte apoptosis in oral lichen planus are unknown. Possible mechanisms include **(1)** T-cell secreted TNF-α binding TNF-α receptor 1 (TNF R1) on the keratinocyte surface **(2)** T-cell surface CD95L (Fas ligand) binding CD95(Fas) on the keratinocyte surface, or **(3)** T-cell secreted granzyme B entering the keratinocyte via perforin-induced membrane pores. All these mechanisms may activate the keratinocyte caspase cascade, resulting in keratinocyte apoptosis[30,32].

$CD4^+$ T-Cells

Though the majority of intra-epithelial lymphocytes in oral lichen planus are $CD8^+$ cytotoxic T-cells, most lymphocytes in the lamina propria are $CD4^+$ helper T-cells. Hence, an early event in oral lichen planus lesion formation may be MHC class 2 antigen presentation to $CD4^+$ helper T-cells, followed by keratinocyte apoptosis triggered by $CD8^+$ cytotoxic T-cells. MHC class 2 antigen presentation in oral lichen planus may be mediated by langerhans cells or keratinocytes.

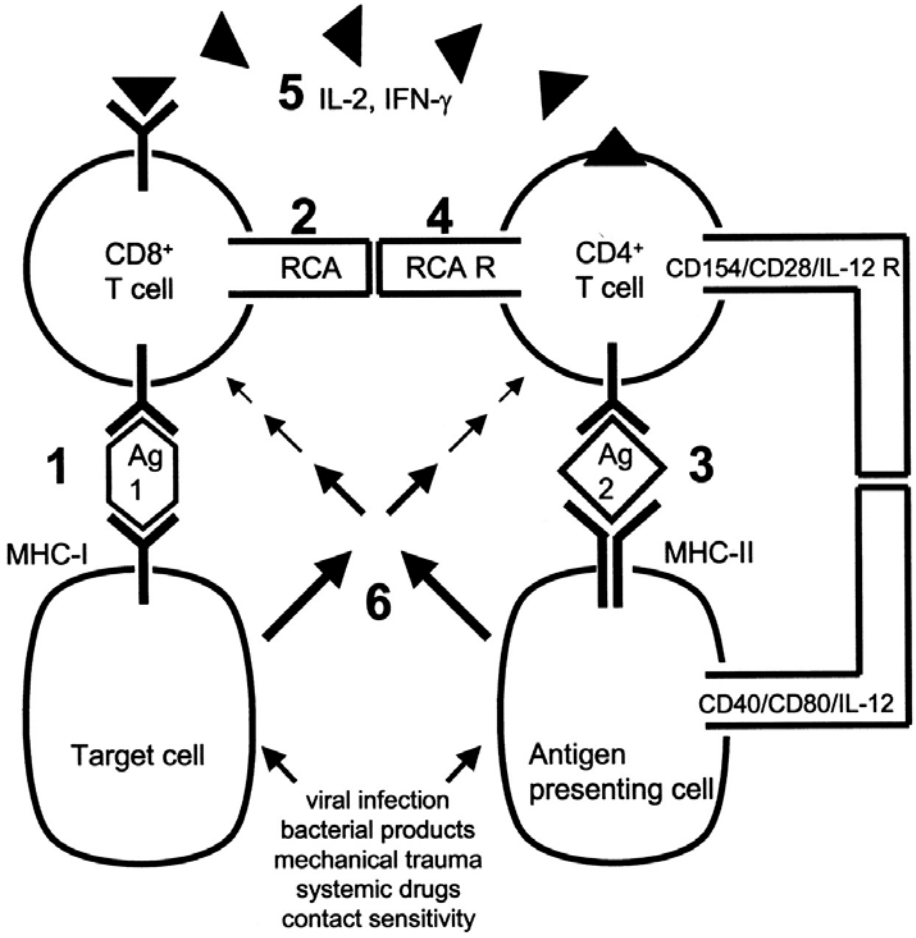
There are increased number of langerhans cells in oral lichen planus lesions. High levels of antigen expression, CD40 and CD80 expression and IL-12 secretion by MHC class 2 antigen-presenting cells(APC) in oral lichen planus may promote a T-helper-1(Th1) $CD4^+$ T-cell response with IL-2 and IFN-γ secretion[30,33].

Types of Antigen Presentation

Antigens that are presented by MHC class II are processed through an endosomal cellular pathway. In contrast, antigens that are presented by MHC class I are processed through a cytosolic cellular pathway. Hence, the putative antigen presented by MHC class II to $CD4^+$ helper T-cells in oral lichen planus may differ from that presented by MHC class 1 to $CD8^+$ cytotoxic T-cells. MHC class I antigen presentation alone may result in transient cytotoxic T-cell response.

MHC class II antigen presentation alone may generate Th1 $CD4^+$ T-cells. However, in the absence of MHC class I antigen presentation to $CD8^+$ cells, the IL-2 and IFN-γ secreted by the activated Th1 $CD4^+$ T-cells would be cytotoxically inert. Therefore, simultaneous antigen presentation to $CD8^+$ and $CD4^+$ T-cells is required to develop persistent T-cell infiltration and $CD8^+$ cytotoxic T-cell activity in oral lichen planus[30].

HYPOTHESIS FOR ANTIGEN PRESENTATION AND T-CELL ACTIVATION IN OLP



Initially, the CD8+ T-cell antigen receptor engages a specific foreign antigen (Ag 1) in the context of MHC class I on the basal keratinocyte target cell in OLP [1]. The CD8+ T-cell may then seek CD4+ T-cell confirmation by expressing the hypothetical "request cytotoxic activity" (RCA) cell surface molecule [2]. The CD4+ T-cell expresses the hypothetical "RCA receptor" (RCA R) [4], but only following CD4+ T-cell antigen receptor engagement of a related foreign antigen (Ag 2) in the context of MHC class II on the antigen-presenting cell (basal keratinocyte or Langerhans cell in OLP) [3].

Ligation between RCA and RCA R in combination with co-stimulatory signals from the MHC class II^+ antigen-presenting cell (*e.g.*, CD40, CD80, and IL-12 binding CD154, CD28, and IL-12 R, respectively, on the $CD4^+$ T-cell) initiates Th1 differentiation of the $CD4^+$ T-cell that then secretes IL-2 and IFN-γ [5]. Receptors for IL-2 and IFN-γ are expressed by the $CD8^+$ T-cell, but only following (i) specific engagement of the $CD8^+$ T-cell antigen receptor in the context of MHC class I

and/or (ii) ligation between RCA and RCA R. The $CD4^+$ Th1 cytokines (IL-2 and IFN-γ) are detected by the $CD8^+$ T-cell and interpreted as confirmation to proceed with target cell (basal keratinocyte) lysis. Keratinocyte activation by (i) the $CD4^+$ or $CD8^+$ T-cell following receptorantigen- MHC trimerization or (ii) exogenous agents such as viral infection, bacterial products, mechanical trauma, systemic drugs, or contact sensitivity up-regulates keratinocyte cytokine and chemokine secretion [6] that promotes lymphocyte extravasation and directs lymphocyte migration into the site of the developing OLP lesion.

Non Specific Mechanisms in Oral Lichen Planus

The non specific factors involved in the pathogenesis of oral lichen planus are as follows :

1. The Epithelial basement membrane.
2. Matrix Metalloproteinases.
3. Mast cells.
4. Chemokines.

The Epithelial Basement Membrane

Keratinocytes contribute to the structure of the epithelial basement membrane by secreting collagen IV and laminin V into the basement membrane zone. Keratinocyte apoptosis triggered by intra-epithelial $CD8^+$ cytotoxic T-cells may result in basement membrane disruption in oral lichen planus. Conversely epithelial basement disruption may trigger keratinocyte apoptosis in oral lichen planus. Such a cyclical mechanism may underlie the disease chronicity[34].

Matrix Metalloproteinases (MMPs)

Matrix metalloproteinases are a family of Zinc-containing endoproteinases. The principal function of MMPs is the proteolytic degradation of connective tissue matrix proteins. MMP-2 and MMP-3 were expressed mainly in the oral lichen planus epithelium. MMP-9 was identified within the inflammatory infiltrate in the lamina propria. The T-cells in oral lichen planus may be stimulated by TNF-α to secrete MMP-9.[30,35]

T-cell secreted MMP-9 may disrupt the epithelial basement membrane in oral lichen planus lesions. MMP-9 induced basement membrane disruption may facilitate the passage of antigen-specific $CD8^+$ cytotoxic T-cells into the oral lichen planus epithelium, where they trigger further keratinocyte apoptosis[30,35].

Mast Cells

Approximately 60% of mast cells were degranulated in oral lichen planus compared with 20% in normal buccal mucosa. Mast cell degranulation releases a range of pro-inflammatory mediators such as TNF-α, chymase and tryptase. TNF-α may upregulate endothelial cell

adhesion molecule [CD62E, CD54 and CD106] expression in oral lichen planus that is required for lymphocyte adhesion to the luminal surfaces of blood vessels and subsequent extravasation[36].

Mast cells and intra- epithelial CD8+ T-cells have been identified at the sites of basement membrane disruption in oral lichen planus. This suggests that mast cells may play a role in epithelial basement membrane disruption and CD8+ T-cells may migrate through basement membrane breaks to enter the oral lichen planus epithelium. MMPs are secreted as inactive proenzymes and are rapidly degraded after activation. Chymase, a mast cell protease, is a known activator of MMP-9. hence basement membrane disruption in oral lichen planus may be mediated by mast cell proteases directly or indirectly via activation of T-cell secreted MMP-9.

Chemokines

Chemokines are a superfamily of pro-inflammatory cytokines that are produced by all somatic cells. RANTES [regulated on activation, normal T-cell expressed and secreted], which is a member of the CC

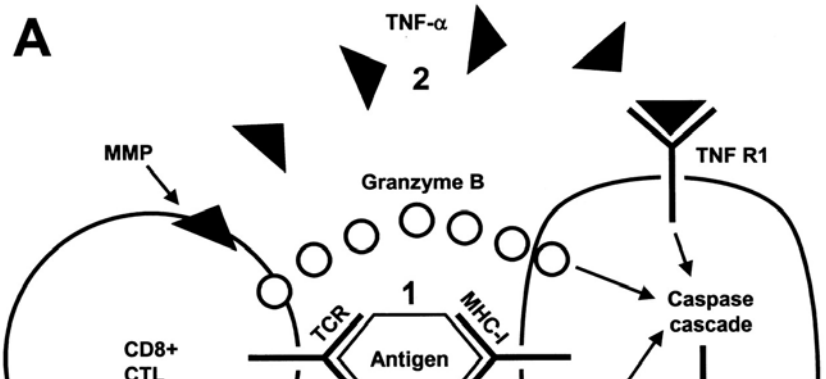
chemokine family, is produced by activated T-lymphocytes, bronchial epithelial cells, oral keratinocytes and mast cells.

RANTES plays a critical role in the recruitment of lymphocytes, monocytes, natural killer cells, eosinophils, basophils and mast cells. Chemokines mediate their biological effects by binding to cell-surface receptors. Several RANTES receptors including CCR1, CCR3, CCR4, CCR5, CCR9 and CCR10 have been identified. The CC chemokines, including RANTES activate mast cell migration and degranulation via these G protein-coupled receptors[37].

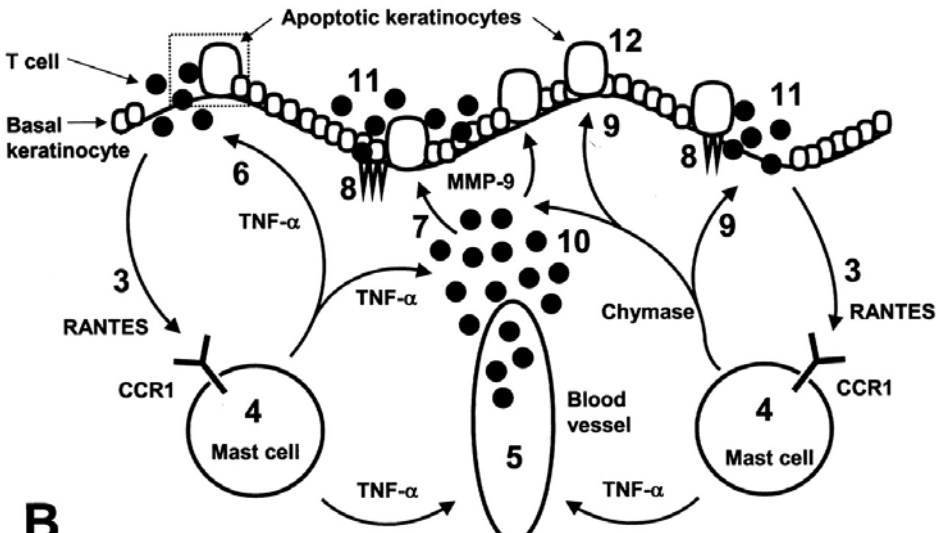
RANTES secreted by oral lichen planus lesional T-cells may attract mast cells into the developing oral lichen planus lesion and subsequently stimulate mast cell degranulation. Degranulation of mast cells would release TNF-α, which upregulates oral lichen planus T-cell RANTES secretion. This cyclical mechanism is the cause for chronicity of the disease. RANTES also prolongs the survival of inflammatory cells in oral lichen planus[30].

A Unifying Hypothesis for the Pathogenesis of Oral lichen planus

A lichen planus antigen is expressed in association with MHC class I molecules on basal keratinocytes at the oral lichen planus lesion site [1]. Antigen-specific CD8+ cytotoxic T-lymphocytes (CTLs) are activated in the oral lichen planus epithelium (possibly with help from Th1 CD4+ T-cells) and trigger keratinocyte apoptosis *via* secreted TNF-α binding the TNF-α receptor (TNF-R1) [2], although roles for granzyme B and Fas cannot be excluded at this stage. TNF-α may be activated and released from the CTL surface by lesional MMPs.



Activated T-cells undergo intra-lesional clonal expansion and release RANTES and other cytokines [3] that up-regulate mast cell CCR1 expression and stimulate intra-lesional mast cell migration and degranulation [4]. Degranulating mast cells release TNF-α, which up-regulates endothelial cell adhesion molecule expression for lymphocyte adhesion and extravasation [5]. Mast cell TNF-α also upregulates RANTES [6] and MMP-9 [7] secretion by oral lichen planus lesional T-cells. Activated lesional T-cells (and possibly keratinocytes) secrete chemokines that attract extravasated lymphocytes toward the OLP epithelium [8].



Degranulating mast cells release chymase that damages the epithelial basement membrane directly [9] or indirectly *via* activation of MMP-9 secreted by oral lichen planus lesional T-cells [10]. Epithelial basement membrane disruption facilitates the passage of lymphocytes into the oral lichen planus epithelium [11] and denies keratinocytes a cell survival signal, resulting in further keratinocyte apoptosis [12].

CLINICAL FEATURES

Oral lichen planus affects primarily middle aged adults and the prevalence is greater among women[38]. Children are rarely affected. 30% to 50% of patients with oral lichen planus also have cutaneous involvement. More than 50% of patients have bilateral involvement.

Any location in the oral cavity may be involved in lichen planus, with the most common site being the posterior buccal mucosa[39]. Other common sites are tongue, gingival, retromolar area, vestibule, palate, floor of mouth and lip[5, 39].

The onset of oral lichen planus is insidious. Symptoms like roughness of the lining of the mouth, sensitivity to hot or spicy foods, painful oral mucosa, sore gums, red or white patches on the oral mucosa or oral ulcers may be present. At times, patient can be completely asymptomatic.

The World Health Organization recognizes seven different clinical forms of oral lichen planus. They are : reticular, papular, plaque, atrophic, erosive, ulcerated and bullous. Andreasen's classification[5] of oral lichen planus is as follows :

(1) Reticular type

(2) Erosive type

(3) Plaque type

(4) Atrophic type

(5) Papular type

(6) Bullous type

RETICULAR

This is the most common type of oral lichen planus. It presents as interlacing white keratotic lines, also known as Honiton lace or Wickham's striae. The interlacing lines have an erythematous border. The striae are typically located bilaterally on the buccal mucosa, mucobuccal fold, gingival and less commonly the tongue, palate and lips. This type is usually asymptomatic[40,41].

EROSIVE

It is the second most common type of oral lichen planus. It presents as a mix of erythematous and ulcerated areas surrounded by finely radiating keratotic striae. The erosion may be covered by a pseudomembrane. When erosive oral lichen planus involves the attached gingival tissue, it is called desquamative gingivitis[42,43]. The lesions of erosive oral lichen planus migrate over time and tend to be multifocal.

Patients present with symptoms ranging from episodic pain to severe discomfort that can interfere with normal masticatory function. The triad of erosive or desquamative lichen planus involving vulva, vagina and gingiva is called vulvo-vaginal-gingival syndrome[44,45]. Malignant transformation is common with this type of oral lichen planus.

PLAQUE TYPE

The clinical presentation of plaque type of oral lichen planus can range from smooth, flat areas to irregular, elevated areas. It is commonly found on the dorsum of the tongue and on the buccal mucosa. This type of oral lichen planus is usually asymptomatic. This form of oral lichen planus is frequently found in smokers[40].

ATROPHIC TYPE

This type of oral lichen planus presents as diffuse, erythematous patches surrounded by fine white striae. This can cause significant discomfort and burning sensation[40].

PAPULAR TYPE

The papular form of oral lichen planus is rare and it consists of small whitish papules, about 0.5 mm in diameter. They may also precede Whickham's striae and they are usually asymptomatic[40,46].

BULLOUS TYPE

This is the least frequent form of oral lichen planus. The lesions appear as vesicles or bullae of variable size, that easily burst leaving behind painful erosions. It occurs mainly in the buccal mucosa, in

particular in the area next to the second and third lower molar teeth. The lateral portion of the tongue may also be involved[40,42].

Oral lichen planus may present as hyperpigmented macular lesions in the midst of white striae. Though there is no separate entity called hyperpigmented oral lichen planus, this could be an expression of a post inflammatory hyperpigmentation[47]. Multiple clinical subtypes can be present in the same patient.

Oropharyngeal involvement in lichen planus can involve the esophagus also. Clinically it manifests as dysphagia and formation of benign strictures. Endoscopic findings show papular or erosive lesions. Lichen planus of esophagus may have a risk of malignant transformation similar to oral lichen planus.

Lichen planus can involve eye lids and conjunctiva also. It causes a foreign body sensation. They can be seen as fine, white lacy pattern in the tarsal conjunctiva. Rarely, Kerato conjunctivitis sicca may be seen. Inflammatory meatal fibrosing otitis can be seen in the years.

70% of patients with cutaneous lichen planus develop oral lichen planus. The characteristic cutaneous lesion is a flat topped, erythematous or violaceous pruritic papule typically involving flexor surfaces of arms

and legs. Oral lichen planus may be the only manifestation in 20 to 30 % of patients.

25% of female patients with oral lichen planus also have genital involvement, while only 2 to 4% of men with oral lichen planus have genital lesions[3]. Male patients present with lesions over the penile shaft, glans penis, prepuce or scrotum. Female patients present with lesions over the vulva either as fine reticulate papules or severe erosions leading to dyspareunia and scarring[47].

Nail involvement in patients having oral lichen planus alone is uncommon. It may present as ridging, thinning, subungual hyperkeratosis, pitting, onycholysis, shedding of nails and discolouration. But the characteristic nail change is pterygium formation.[47].

COURSE

Oral lichen planus usually has a long evolution, persisting for 25 years or more. The atrophic, bullous and erosive types are usually painful and may present with a burning sensation when in contact with spicy food or smoke[4]. Oral lichen planus presents with periods of exacerbation and quiescence. During periods of exacerbation, the area of erythema and erosion increases. Both erosive and atrophic types present with shorter

period of evolution along with larger areas and multiple sites of involvement[4,5].

COMPLICATIONS

Oral lichen planus patients are predisposed to oral candida albicans super infection. Candida species are commensal organisms in 40% of healthy individuals. Various factors involved in colonization by candida include immunosuppression, medications, nutritional deficiencies, malignancies, age, poor oral hygiene, dental prostheses, salivary changes and epithelial alterations. Normally, intact oral mucosa provides an effective barrier against fungal or bacterial invasion[50.]

In oral lichen planus, the oral mucosal pathology alters the integrity of the oral epithelium and predisposes the patients to fungal infections. Also, the alteration in the cell mediated immunologic response to antigenic changes in the basal layer alters the immunologic response against candida albicans as well. Superinfection with candida albicans usually exacerbates the signs and symptoms of oral lichen planus. There is an increased chance of malignant transformation in oral lichen planus patients who have candidial superinfection. Hence, such patients should be initiated on antifungal treatment in addition to treatment of oral lichen planus[50].

Oral squamous cell carcinoma is a dreaded complication of oral lichen planus. The most important risk factor for development of malignancy is concomitant use of alcohol and tobacco products. Atrophic, erosive and plaque types of oral lichen planus are at greater risk of malignant change. The rate of malignant transformation in oral lichen planus is between 0.5 to 2%[3,4,40].

The World Health Organization has categorized oral lichen planus as a precancerous condition. Mignogna et al. have suggested that regular follow up of patients with oral lichen planus be performed up to 3 times a year[38,39,40]. The signs that may be indicative of transformation, such as the extent of symptoms and the loss of homogeneity should be assessed thoroughly. When there is evidence of changes in clinical appearance, the follow-up period should be shortened and additional biopsy should be performed[50,51].

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of oral lichen planus includes pemphigus vulgaris, benign mucosal bullous pemphigoid, cicatricial pemphigoid, secondary syphilis, chronic candidiasis, discoid lupus erythematosus, erythema multiforme, oral leukoplakia, smoker’s patches, squamous cell

carcinoma, hypersensitivity mucositis, leukokeratosis and white sponge nevus[47].

Unlike oral lichen planus, leukoplakia is more common in males and it affects persons above forty years of age. Local irritative factors are essential for the development of leukoplakia but there is no pattern of location. Associated skin lesions are absent in leukoplakia, while cutaneous involvement is common with oral lichen planus.

Candida albicans is a commensal in the mouth. Factors like extremes of age, prolonged use of steroids, prolonged use of antibiotics and immunosuppresants and endocrine disorders cause a host parasite imbalance and result in infection. It can be confirmed by scraping.

Oral white sponge naevus may manifest at birth or chidhood with white plaques in oral mucosa and usually it is asymptomatic. Biopsy helps in differentiating between the two conditions.

Oral lesions of pemphigus vulgaris resemble ulcerative or bullous type of oral lichen planus. But oral lichen planus lesions may present with white striations around the ulcers. Biopsy is useful for diagnosis. Bullous pemphigoid produces oral lesions similar to bullous type of oral lichen planus, but associated skin lesions help in differentiating between them.

Oral lesions in secondary syphilis may manifest as pseudomembranous lesions or erosions of tongue, hard and soft palate and tonsils. It may be associated with generalized lymphadenopathy. The histopathological features of secondary syphilis are variable. Often the changes are non specific, although they may include perivascular infiltrates with a preponderance of plasma cells and epidermal psoriasiform hyperplasia. Specific tests can be done to confirm syphilis.[53]

Discoid lupus erythematosus may present with oral lesions resembling lichen planus, but coexisting skin lesions in the face and sun exposed areas help in diagnosis.

Erosive type of oral lichen planus involving the gingival as erosive gingivitis is not pathognomonic of lichen planus. Erosive gingivitis could be an oral manifestation of other diseases like cicatricial pemphigoid, bullous pemphigoid, pemphigus vulgaris and lupus erythematosus.

HISTOPATHOLOGY

The histological features of oral lichen planus are similar to those of cutaneous lichen planus. It was first described by Dubrenill in 1906 and later by shklar[48]. Shklar described three classic histological features

which are overlying keratinization, a dense band-like layer of lymphocytic infiltrate within the underlying connective tissue and liquefactive degeneration of basal cell layer.

Pindborg et al have further described the histological features of idiopathic oral lichen planus. It consists of hyperkeratosis, parakeratosis, focal hypergranulosis, acanthosis, liquefaction degeneration of basal keratinocytes and a band like inflammatory lymphohistiocytic infiltrate of the upper lamina propria. Within the basal cell layer, degenerating basal keratinocytes form colloid (civatte, hyaline or cytoid) bodies that appear as homogenous eosinophilic globules. The ultra structure of colloid bodies suggest that they are apoptotic keratinocytes. B cells and plasma cells are rare features of idiopathic lichen planus[30,52].

Eisenberg[49] suggested the essential histologic features for definitive diagnosis of oral lichen planus. They are : band-like inflammatory infiltrate consisting of lymphocytes, normal maturation epithelium, saw-tooth appearance of rete ridges, civatte bodies and hyperkeratosis. The exclusion criteria for oral lichen planus are: absence of basal cell liquefaction degeneration, heterogeneous population of infiltrate, atypical cytomorphology, blunted rete ridges, absence of civatte bodies and abnormal keratinization.

Epithelial basement membrane changes are common in oral lichen planus and include breaks, branches and duplications. The basal keratinocyte anchoring elements (hemidesmosomes, filaments and fibrils) are disrupted in oral lichen planus. Degeneration of basal keratinocytes and disruption of the epithelial basement membrane and basal keratinocyte anchoring elements in oral lichen planus produces weakness at the epithelial – connective tissues interface which may result in histological cleft formation (Max-Joseph space).

Ulcerative or erosive type of oral lichen planus shows typical inflammatory degeneration and necrosis of epithelium adjacent to the ulcer, while bullous type shows blister formation at the level of the basement membrane. Atypical cytomorphology, nucleus enlargement, increased mitotic figures, blunted rete ridges and abnormal keratinization are features suggestive of dysplasia in oral lichen planus.

The histological features of oral lichen planus and lichenoid drug reaction are similar. Distinguishing features seen in lichenoid drug reaction are an inflammatory infiltrate located deep in the dermis, a focal perivascular infiltrate and abundance of plasma cells in the connective tissue.

DIRECT IMMUNOFLUORESCENCE

Direct immunofluorescence is used to detect autoantibodies that are bound to the patient's tissue. Direct immunofluorescence studies in oral lichen planus have shown a linear pattern along the basement membrane zone. Under direct immunofluorescence, oral lichen planus lesions show presence of IgM and eventually IgG, IgA, C3 and fibrin in the colloid corpuscles. But this pattern is not specific [4,30].

INDIRECT IMMUNOFLUORESCENCE

Indirect immunofluorescence is used to detect the presence of antibodies that are circulating in blood. Though this technique may not help in diagnosing oral lichen planus, it can be used for diagnosing lichenoid drug reactions. In lichenoid drug reactions, the circulating antibodies reactive with basal cells give rise to an annular fluorescence pattern[30,39].

IMMUNOHISTOCHEMISTRY

The infiltrating cells in oral lichen planus are predominantly T-lymphocytes with very few B-lymphocytes. More than 90% are activated T-lymphocytes expressing HLA - DR antigen and some interleukin-2 receptor. In older lesions of oral lichen planus, the

suppressor T-lymphocytes predominate. In the epidermis adjacent to the infiltrate, basal keratinocytes express HLA - DR surface antigen and intercellular adhesion molecule - 1 (ICAM-1), both of which are implicated in augmentation of interaction between lymphocytes and their epidermal targets resulting in keratinocyte destruction.[54]

These surface antigens are induced by cytokines IFN-γ and TNF-α that are released by lymphocytes from the infiltrate. Immunophenotyping studies on T-lymphocyles shows that the majority of clones were CD8+ T-lymphocytes. The number of Langerhans cells in the epidermis is increased very early in the disease, especially near keratinocytes expressing HLA-DR + antigen. Specific conjugations between CD4+ T-lymphocytes and dendritic cells and between CD8+ T-lymphocytes and degenerated basal keratinocytes have been observed in the lesional epithelium of oral mucosa. These cell-to-cell interactions suggest that a cell - mediated immune mechanism is responsible for the evolution of oral lichen planus.[54]

HISTOPATHOLOGICAL DIFFERENTIAL DIAGNOSIS

Lichenoid drug eruptions in the oral mucosa can be differentiated from classical oral lichen planus by the

presence of focal parakeratosis with the absence of granular layer. Necrotic keratinocytes are seen in the basal and spinous layer with exocytosis of lymphocytes to the upper layers of epidermis. The inflammatory infiltrate is deep with predominance of eosinophils.

The epidermal changes in chronic graft versus host disease (GVHD) may be similar to oral lichen planus. But the inflammatory infiltrate tends to be more perivascular while it is band-like in lichen planus. The number of langerhans cells in chronic GVHD is decreased and all the intra epidermal lymphocytes are cytoloxic - suppressor T-cells.

In early Squamous cell carcinoma *in situ*, the histological findings are similar to oral lichen planus. Both diseases show hyperkeratosis and an inflammatory infiltrate close to the epidermis. But a thorough microscopic examination reveals more atypical keratinocytes in squamous cell carcinoma *in situ*. Moreover, it shows irregular downward proliferation of the rete ridges and numerous plasma cells.[54]

AIMS OF STUDY

A prospective, analytical study of oral lichen planus was planned.

Aims of this study

1. To study the age and sex incidence of oral lichen planus.
2. To look for any known provocative factors in the onset of oral lichen planus.
3. To study the various types of clinical presentation of oral lichen planus.
4. To study the various sites of distribution of oral lichen planus.
5. To study the associated disorders in oral lichen planus
6. To study the histopathological features of various clinical types of oral lichen planus

MATERIALS AND METHODS

The patients for this study were selected from the Outpatient Department of Dematology, Government Stanley Hospital, Chennai. All cases of Lichen Planus were screened for oral mucosal lesions and other patients who presented with oral lesions were examined specially to rule out Lichen Planus.

Patients were enrolled and consent obtained for study, biopsy and clinical photo. The cases were given a thorough clinical examination. Detailed enquiries were made with regards to the symptoms and their duration, nature of occupation and history of taking any drugs prior to the development of lesion.

Detailed personal history regarding other skin diseases, personal habits, exposure to sexually transmitted diseases and the possibility of emotional or physical stress prior to the onset of the lesions were recorded. Family H/o similar skin lesions, Diabetes Mellitus and Hypertension were asked for. History of remissions and exacerbation with or without treatment was noted.

All the cases were given a routine general check up with special reference to skin and nail changes.

Hypertension was ruled out by appropriate measures. Patients were examined under good light, magnifying lens and retractor. The sites affected, the types of changes in the mucous membrane, skin and nail changes along with associated skin disorders if any were made note of. Systemic examination was done in all cases to rule out systemic disorder. All patients were referred to ENT and Dental department to rule out focal sepsis.

LABORATORY INVESTIGATIONS

Routine Haematological, urine and stool examinations were done in all cases. Serology for Sexually Transmitted Diseases was done. Fasting and post prandial blood sugar were done in all the cases. Scraping for candidiasis was done from the mucosal lesions to rule out oral candidiasis.

Biopsy of the oral mucosal lesions were done and the specimen preserved in 10% formalin and sent for histopathological examination. Biopsy specimens were studied with Haematoxylin and Eosin staining. Skin Biopsy was done when skin lesions were present with oral lichen

planus. Clinical photographs were taken prior to the commencement of investigations and treatment.

OBSERVATION

A prospective analytical study of oral lichen planus was carried out at the Department of Dermatology, Govt. Stanley Medical College Hospital during the period of June 2006 to May 2007. Among the patients who attended the skin OPD during the study period, thirty patients who had oral mucosal lichen planus were taken up for study. Among the cases of oral lichen planus, 50% of patients presented with cutaneous manifestations of lichen planus.

Among the thirty patients with oral lichen planus, twenty were males and ten were females. The youngest patient was a sixteen-year-old male, while the oldest was a seventy-year-old male. The youngest female patient was twenty-one years old while oldest was forty-six years old. The majority of patients belonged to the age group of twenty and forty. A total of twenty patients belonged to that age group.

In nine out of thirty patients, history suggestive of emotional stress was found to be associated with the onset of the oral lesions. Six out of twenty male patients were chronic smokers. Two patients had been on treatment for hypertension with atenolol and captopril for six months prior to the onset of oral lesions. Three patients were known diabetics and were on treatment with tolbutamide.

On dental examination, nine patients had oral sepsis. Four patients had chronic maxillary sinusitis. None of the patients had a positive history of exposure to sexually transmitted diseases. In two patients vitiligo was found in association with oral lichen planus. Two patients had positive family history for lichen planus. Two patients had dental fillings. Six patients had oral candidiasis.

Among thirty patients, only fourteen were symptomatic of which nine were males and five were females. Routine general and systemic examination of the cases in this study did not reveal any other abnormality. Fifteen out of thirty cases, presented only with oral mucosal lesions. In eleven cases skin and oral mucosa were affected. In four cases in addition to skin and oral mucosa, genital mucous membrane was also affected(Figure-11).

Among the oral lichen planus lesions, fourteen patients had reticular type (Figure-3,5,9), eight patients had erosive type (Figure-1,4), two patients had papular lesions (Figure-9), one atrophic type, two plaque type of lesions (Figure-2,6,7). Three patients had both reticular and erosive types of oral lichen planus.

Buccal mucosa was found to be commonly affected in this study, as seventeen patients had buccal mucosa lesions. Along with that, lower

lips, tongue, gingival and hard palate were found to be affected. Six patients had palatal involvement, three had gingival lesions (Figure-10), two had tongue lesions and two patients had lower lip lesions.

In the skin involvement, three patients had hypertrophic type of lichen planus while rest had classical type of lichen planus. Linear type of lichen planus was found in two patients. One patient had palmoplantar keratoderma. (Figure-13 & 14)

Characteristic nail change – pterygium was present in one patient. Twenty nail dystrophy was present in one patient (Figure-12). Seven patients showed longitudinal ridging and three had thinning of nail plate.

Routine haematological examination did not show any abnormality. Urine analysis were found to be normal in all the cases. On routine examination of the stools, two cases had cyst of entamoeba histolytica. Serological test for syphilis was found to be non-reactive for all the cases. ELISA for HIV was negative for all the patients.

Clinical photographs were taken for all the cases. After eliminating the predisposing factors and treating the focus of infection such as oral sepsis and ear, nose, throat infection, the patients showed improvement of the lesions. The patients were followed up with

symptomatic and topical treatment. Patients with erosive and atrophic type of oral lichen planus are still being followed up for malignant transformation.

Oral mucosal biopsy was done to demonstrate the histopathological features which were compatible with features of oral lichen planus. It presented with parakeratosis with atrophic epithelium, irregular elongation of rete ridges with a band like inflammatory infiltrate containing lymphocytes (Figure-15-20).

Reticular type – it showed features of classical lichen planus with a band like inflammatory infiltrate whose lower margin was not well defined. (Figure -17 & 18).

Erosive type – it showed marked basal cell degeneration and there was evidence of spongiosis. There was no acanthosis. The band like inflammatory infiltrate was not well defined in the lower border. There was no necrosis. (Figure -16)

Plaque type – It presented with marked acanthosis along with a band like inflammatory infiltrate with a well defined lower margin as against the erosive type. (Figure - 15 & 20)

Atrophic type – Significant epidermal thinning along with classical features of lichen planus.

Papular type – Significant pigmentary incontinence was seen. (Figure-19)

Skin biopsy was done wherever skin lesions were present and the classical histopathological features of cutaneous lichen planus were made note of. Patients with hypertrophic type of lichen planus presented with acanthosis, papillomatosis, hypergranulosis and hyperkeratosis.

DISCUSSION

Thirty cases of oral lichen planus were taken up for this study which included lichen planus lesions occurring either in oral mucosa alone or in combination with skin and genital mucous membrane. The percentage of oral lichen planus patients presenting with cutaneous lichen planus in this study was found to be 50%.

As per literature, the age group commonly affected in oral lichen planus is said to be twenty and above, with a peak incidence between twenty to sixty years of age[29,42]. In the current study, the maximum number of cases were in the age group of twenty one and thirty. In this study the youngest age was sixteen in males and twenty one in females. The oldest male patient was seventy years old, while it is forty six in females. The peak age incidence in males is thirty one to forty years, while in females it is twenty one to thirty years(Table-1, Graph-1).

Table-1

Age Group	***Male***	***Female***	***Total***	***Percentage***
10 – 20	2	0	2	7%
21 – 30	5	7	12	40%
31 – 40	6	2	8	27%
41 – 50	5	1	6	20%
51 – 60	1	0	1	3%
61 – 70	1	0	1	3%
Total	**20**	**10**	**30**	**100%**

It has been reported in literature that oral lichen planus has a female preponderance. Some studies have reported an equal sex incidence of oral lichen planus[29,31]. But in this study there is a male predominance. Out of the thirty patients taken up for this study, only ten were females. The reason for this male predominance could be due to the health seeking behaviour of males when compared to females in our country (Graph-1 & 2).

Among the thirty patients studied, fourteen had symptoms such as pain and burning sensation while taking spicy food. Five out of ten female patients were symptomatic. The longest duration of symptoms was eighteen months, the shortest being four months. Out of twenty male patients, nine were symptomatic. In most of the asymptomatic patients, oral lichen planus was diagnosed when they presented with cutaneous lesions (Graph-3 & 4).

Twelve out of fifteen patients who presented with only oral lichen planus were symptomatic. Various studies have reported that in many patients the onset of oral lichen planus is insidious and the patients are unaware of their oral condition. Approximately two thirds of patients

with oral lichen planus report oral discomfort, especially in association with erosive and atrophic lesions[4,38,40].

Oral lichen planus is said to be a multi factorial disease. In our study, two patients had positive family history of lichen planus. Emotional stress is an important predisposing factor in oral lichen planus. Even in patients with oral lichen planus, periods of exacerbations are associated with anxiety[13]. Nine out of thirty patients gave history suggestive of emotional stress prior to the onset of the disease. So, almost 30% of patients had history of emotional stress (Graph-5).

Drugs like antimalarials, anti arthritic drugs, anti hypertensives, antidiabetic drugs, cardiovascular drugs and certain dental amalgams and dyes used in colour developers are said to be predisposing factors in oral lichen planus[6,8]. The period between the commencement of of the drug therapy and the clinical appearance of oral lichen planus varies. In the current study, two patients were on beta blockers for hypertension and three were on oral hypoglycaemic agents. Two patients had dental fillings. Nine patients had dental sepsis and four had chronic sinusitis.

Smoking and tobacco chewing have been reported as predisposing factors in the development of oral lichen planus(Graph-6). Various Indian studies have reported this association. Smoking also increase the chances

of developing oral malignancy later[3,4,40]. In this study, six out of twenty male patients were chronic smokers. Candidial superinfection has been reported in oral lichen planus and in this study six patients had oral candidiasis.

Oral lichen planus can occur alone or can be associated with skin lesions. Up to 70% of patients with cutaneous lichen planus can present with oral mucosal involvement[40]. In the current study, among thirty patients with oral lichen planus, fifteen patients presented with cutaneous lesions. So, the percentage of patients presenting with cutaneous lichen planus lesions in patients with oral lichen planus is 50 in this study. Fifteen cases presented with oral lesions only. Eleven patients had skin and oral mucosal lesions. Four patients had genital lesions in addition to cutaneous and oral lichen planus(Graph-7).

Literature reports that 25% of female patients with oral lichen planus present with genital lesions. Among males, 2% to 4% of patients with oral lichen planus present with genital lesions[38,40]. In the present study, three out of ten female patients presented with genital lesions, while one out of twenty male patients had genital lesions. So, the percentage of genital involvement was thirty in females and five in males in the present study.

Various studies report that reticular type is the most common type of oral lichen planus. The second most common is the erosive type[40,41]. Our study also has a similar presentation with 47% of patients presenting with reticular type lesions while 27% of patients had erosive lesions.

10% of patients had both reticular and erosive lesions. 7% of patients presented with popular lesions, while another 7% had plaque type lesions. Atrophic type was present in 2% of patients. Plaque type lesions were present among smokers(Graph-8).

The histopathological features in all the cases was similar to classical oral lichen planus . Various clinical subtypes showed certain differences in their histopathology.

Reticular type – it showed features of classical lichen planus with a band like inflammatory infiltrate whose lower margin was not well defined.

Erosive type – it showed marked basal cell degeneration and there was evidence of spongiosis. There was no acanthosis. The band like inflammatory infiltrate was not well defined in the lower border. There was no necrosis. The absence of necrosis could you be due to relative shorter duration of the disease.

Plaque type – It presented with marked acanthosis along with a band like inflammatory infiltrate with a well defined lower margin as against the erosive type.

Atrophic type – Significant epidermal thinning along with classical features of lichen planus.

Papular type – Significant pigmentary incontinence was seen and this could be attributed to the site of the lesion in the lip.

Various conditions have been associated with oral lichen planus. They are pernicious anaemia, alopecia areata, vitiligo, rheumatoid arthritis, lupus erythematosus and scleroderma[30,40]. In this study two patients had vitiligo associated with oral lichen planus. Three patients had diabetes mellitus.

SUMMARY

1. The number of male patients affected is more than female patients.

2. The peak age incidence in males is thirty one to forty years.

3. The peak age incidence in females is twenty one to thirty years.

4. The youngest age affected is sixteen years and oldest age is seventy years.

5. Emotional stress is the predisposing factor in 30% of patients.

6. The most common morphological type is reticular pattern.

7. Male patients, who were smokers had plaque type of oral lichen planus.

8. Fifteen patients had cutaneous lichen planus and four patients had genital lesions.

9. Two patients had hypertension, three had diabetes mellitus and two patients had vitiligo.

10. Classical histopathological changes are seen in oral as well as cutaneous lesions.

CONCLUSION

In this study of oral lichen planus conducted with a sample of thirty patients, a male predominance has been observed. But previous studies report a female predominance or equal sex incidence in oral lichen planus. Male preponderance observed in our study could be attributed to the smaller sample size or could be due to health seeking behaviour of males in our country. More over, the proximity of our institution to the industrial belt, facilitates easy access for males to the skin OPD.

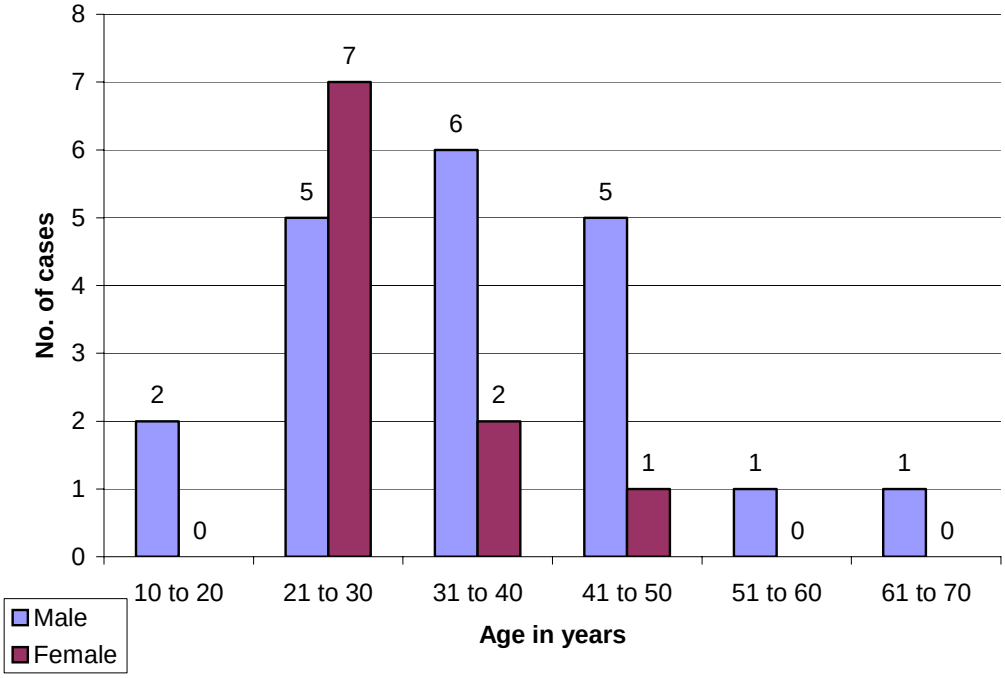
Reticular type of oral lichen planus was the commonest type as per literature. Erosive type was the second most common type showing marked basal cell degeneration. Plaque type showed features of classical lichen planus along with marked acanthosis. Papular type showed significant pigmentary incontinence and atrophic type showed significant epidermal thinning along with features of lichen planus. None of the patients showed features of dysplasia.

The patients with ulcerative and erosive type of oral lichen planus have to be followed up at 6 monthly intervals and preferably with histo pathological correlation, because it has been reported that malignant transformation is common in these types of oral lichen planus.

In our study risk factors like stress and smoking played a significant role in the pathogenesis of oral lichen planus. In future, studies with larger sample size and longer duration have to be undertaken for better analysis of the risk factors involved and for better clinico pathological correlation which will help in early detection of malignancy and management.

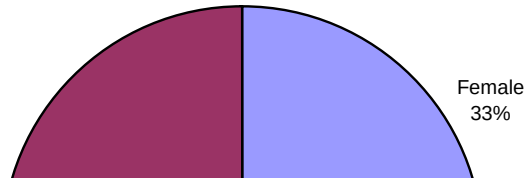
GRAPH-1

SEX AND AGE INCIDENCE OF ORAL LICHEN PLANUS



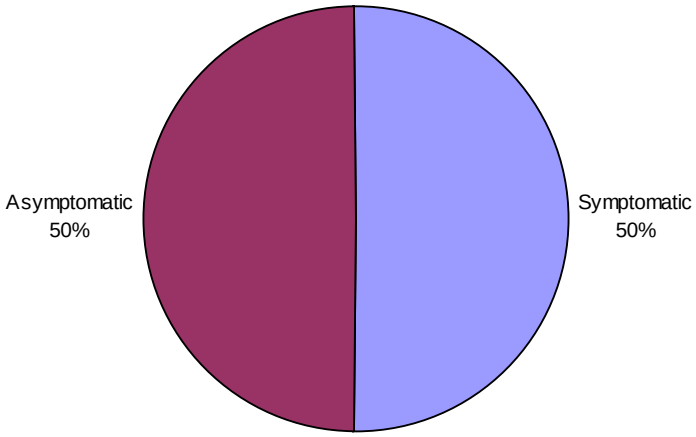
GRAPH-2

SEX INCIDENCE OF ORAL LICHEN PLANUS



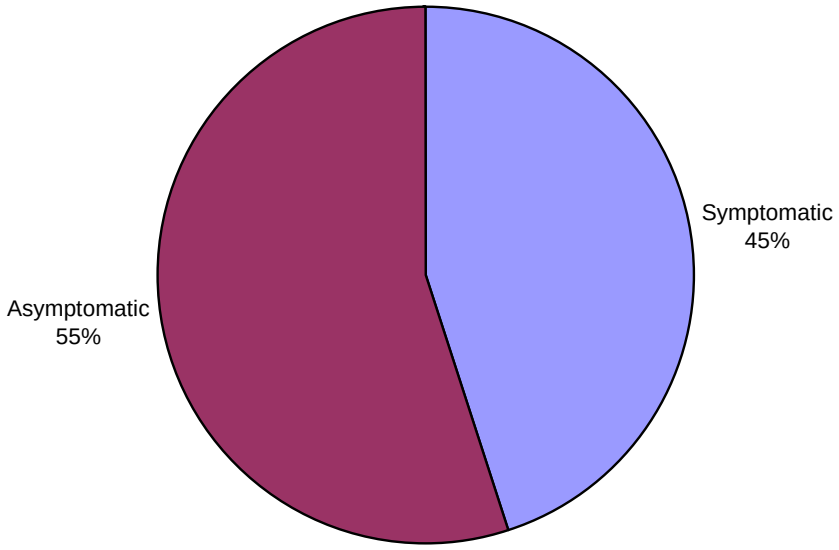
GRAPH-3

PERCENTAGE OF SYMPTOMATIC FEMALE PATIENTS



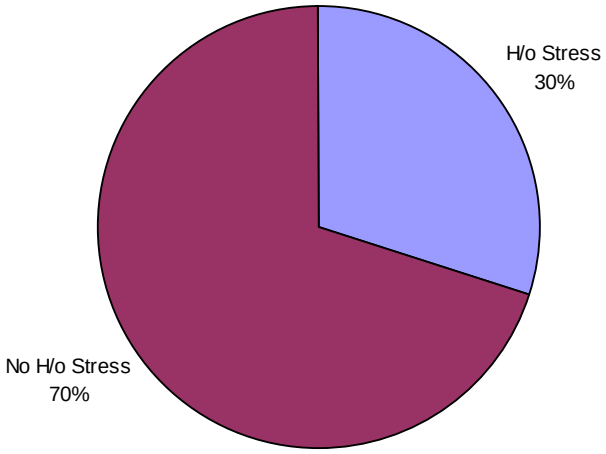
GRAPH-4

PERCENTAGE OF SYMPTOMATIC MALE PATIENTS



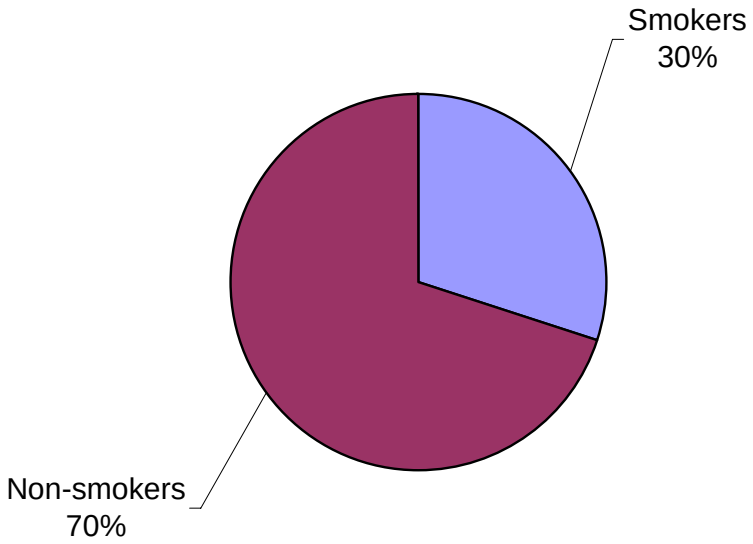
GRAPH-5

ROLE OF STRESS



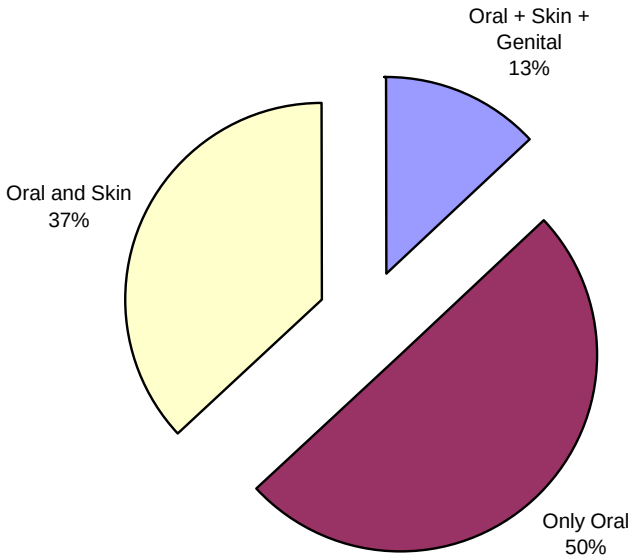
GRAPH-6

IMPACT OF SMOKING



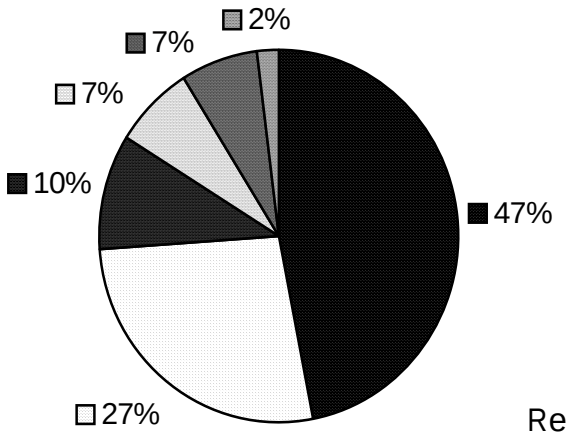
GRAPH-7

VARIOUS PRESENTATION OF ORAL LICHEN PLANUS



GRAPH-8

Clinical types of Oral Lichen planus



Reticular	47%
Erosive	27%
Reticular + Erosive	10%
Papular	7%

MASTER CHART

S. No.		1	2	3	4	5	6	7	
Age		41	29	46	39	19	48	70	2
Sex		M	M	F	M	M	M	M	F
Symptoms & Duration		Burning sensation 6 months	asymptomatic	asymptomatic	Pain 4 months	Pain & discomfort 1 year	asymptomatic	Burning sensation 6 months	a
History of Drugs		+	_	_	_	_	+	_	_
ENT opinion		_	_	Chr.sinusitis	_	_	_	_	_
Dental opinion		gingivitis	_	_	_	_	gingivitis	_	_
Emotional stress		+	__	_	+	_	_	_	_
H/o Diabetes mellitus		+	_	_	__	_	_	_	_
H/o Hypertension		_	_	_	_	_	+	_	_
Family H/o Diabetes		_	_	_	+	_	_	_	+
Oral lesions	Type	Plaque	Papular	Reticular, erosive	Erosive	Reticular, erosive	Plaque	Atrophic	R
	Site	tongue	Lower lip	buccal	gingival	buccal	tongue	buccal	H
Skin lesions		Nil	Nil	Classical lichen planus	Nil	Nil	Nil	Nil	C li p
Genital lesions		_	_	+	_	_	_	_	_
Nail changes		Long. ridging	_	_	_	Thinning	Long. ridging	20 nail dystrophy	_
Associated diseases		_	_	_	_	_	_	_	_
Scraping for candida		_	_	_	+	_	+	_	_
Blood sugar		Normal	Normal	168 mg%	Normal	Normal	225 mg%	Normal	N
Urine examination		Normal	Normal	Sugar- 1+	Normal	Normal	Sugar- 3+	Normal	N
Biopsy – Skin		Nil	Nil	Classical LP	Nil	Nil	Nil	Nil	C
Biopsy – Oral		Compatible with OLP	Compatible with OLP	Compatible with OLP	Compatible with OLP	Compatible with OLP	Compatible with OLP	Compatible with OLP	C w

*

S. No.	11	12	13	14	15	16	17	
Age	21	48	23	26	30	29	25	1

Sex		F	M	M	M	F	F	M	M
Symptoms & Duration		asymptomatic	Pain 1 year	asymptomatic	asymptomatic	White patch 18 months	asymptomatic	asymptomatic	W p 1
History of Drugs		_	+	_	_	_	_	_	_
ENT opinion		_	__	_	_	_	_	_	_
Dental opinion		_	gingivitis	__	_	_	_	_	_
Emotional stress		_	_	_	_	+	_	_	_
H/o Diabetes mellitus		_	+	_	_	_	_	_	_
H/o Hypertension		_	_	_	_	_	_	_	_
Family H/o Diabetes		_	_	_	_	_	_	_	_
Oral lesions	*Type*	Papular	Erosive	Reticular	Reticular	Reticular	Reticular	Reticular	R
	Site	Lower lip	buccal	buccal	buccal	Hard palate	buccal	buccal	b
Skin lesions		Classical lichen planus	Nil	Classical lichen planus, linear lesions	Classical lichen planus	Nil	Classical lichen planus	hypertrophic	N
Genital lesions		__	_	_	_	_	_	_	_
Nail changes		_	Long. ridging	_	_	_	_	_	_
Associated diseases		_	_	_	_	_	_	_	_
Scraping for candida		_	+	_	_	_	_	_	_
Blood sugar		Normal	Normal	Normal	Normal	Normal	Normal	Normal	N
Urine examination		Normal	Normal	Normal	Normal	Normal	Normal	Normal	N
Biopsy – Skin		Classical LP	Nil	Classical LP	Classical LP	Nil	Classical LP	Classical LP	N
Biopsy – Oral		Compatible with OLP	Compatible with OLP	Compatible with OLP	Compatible with OLP	Compatible with OLP	Compatible with OLP	Compatible with OLP	C w

S. No.	***21***	***22***	***23***	***24***	***25***	***26***	***27***	
Age	50	24	55	40	26	28	37	2
Sex	M	F	M	M	F	F	M	M

Symptoms & Duration		Pain 8 months	asymptomatic	Burning sensation 6 months	Pain 4 months	asymptomatic	asymptomatic	Pain 4 months	a
History of Drugs		_	_	_	_	_	_	+	_
ENT opinion		_	_	_	_	_	_	Chr.sinusitis	_
Dental opinion		gingivitis	_	_	_	_	gingivitis	_	_
Emotional stress		+	_	+	+	_	_	_	_
H/o Diabetes mellitus		_	_	_	_	_	_	+	_
H/o Hypertension		_	_	_	_	_	_	_	_
Family H/o Diabetes		_	_	_	_	_	_	_	_
Oral lesions	***Type***	Erosive	Reticular	Erosive	Reticular, erosive	Reticular	Reticular	Erosive	R
	Site	buccal	Hard palate	gingival	buccal	Hard palate	buccal	buccal	H
Skin lesions		Nil	Classical lichen planus	Nil	hypertrophic	Classical lichen planus	Classical lichen planus	Nil	C li p li le
Genital lesions		_	_	_	+	_	_	_	_
Nail changes		Long. ridging	_	Long. ridging	_	thinning	_	Pterygium	_
Associated diseases		_	_	_	_	_	vitiligo	_	_
Scraping for candida		+	_	_	_	_	_	_	_
Blood sugar		Normal	Normal	Normal	196 mg%	Normal	Normal	Normal	N
Urine examination		Normal	Normal	Normal	Sugar- 2+	Normal	Normal	Normal	N
Biopsy – Skin		Nil	Classical LP	Nil	Classical LP	Classical LP	Classical LP	Nil	C
Biopsy – Oral		Compatible with OLP	Compatible with OLP	Compatible with OLP	Compatible with OLP	Compatible with OLP	Compatible with OLP	Compatible with OLP	C w

PROFORMA

1. Serial No. Date:

2. (a) Name

(b) Age

(c) Sex: Male / Female

(d) O.P. No.

(e) Address

(f) Occupation

(g) Percapita Income : Higher
Middle
Lower

3. Complaints

Skin:

Itching Duration

	Mucous Membrane	1.Burning sensation
		2.Pain
		3.Onset – Acute / Slow

4. H/o Presenting Complaints

(a)	H/o Trauma	Sharp Tooth
		Sepsis
(b)	H/o Drugs	
(c)	H/o Emotional Stress	
(d)	H/o Use of Dental Amalgams	
(e)	Remission and Exacerbation	With treatment / Without treatment
(f)	H/o Exposure to the risk of STD	
(g)	H/o Diabetes	

5. History of Past illness

6. Family History H/o diabetes mellitus

	Other members affected
	other skin diseases
7. Personal History	Married / Unmarried
	No. of siblings
	Smoker / Non-smoker
	Alcoholic / Non-alcoholic
	Tobacco / Pan chewing
	Menstrual Histroy
	Diet
8. General Examination	Anaemic / Not anaemic
	Jaundiced / Not Jaundiced
	Cyanosis Present or Absent
	Clubbing Present or Absent

Pedal Oedema present or Absent

Lymphadenopathy

Pulse

BP

9. Systemic Examination

CVS

RS

Abdomen

CNS

10. Dermatological Examination

Primary — Macules / Papules / Patches / Plaques / Vesicle / Bullae

Secondary — Erosion / Ulcer

Sites involved

Face	Right	Left	
Neck (Front)	Right	Left	
Neck (Back)	Right	Left	
Upper Extremity	Right	Flexor	Extensor
	Left	Flexor	Extensor

Lower Extremity		Right	Flexor	Extensor
		Left	Flexor	Extensor
Trunk	Anterior	Right	Left	
	Posterior	Right	Left	

Gluteal Region

Mucous Membrane

Oral

- Buccal
- Gingival
- Hard Palate
- Tongue
- Lip

Size	Surface
Shape	Borders
Distribution	Colour
Bleeding	

Scarring

Genital

Tympanic membrane

Others

Palms

Soles

Hair

Nails

Any associated skin lesions

11. Investigation

(a) Blood : TC/ DC/ ESR/ Hb/ Platelet Count

(b) Urine : Albumin

Sugar

Deposits

(c) Blood Sugar : Fasting and Postprandial

(d) Motion : Ova, Cyst

(e) Scraping of Oral Mucosa for Candida

(f) Blood VDRL :

(g) ELISA for HIV :

(h)	Skin Biopsy	:	
(i)	Oral Mucosal Biopsy	:	
(j)	Clinical Photo	:	
(k)	Treatment and Prognosis	:	
(l)	Consent	:	Clinical photo
			Biopsy

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