原文題目(出處)：	Oral mucosal disease: Lichen planus Br J Oral & Maxillofac Surg 2008;46:15–21
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報告日期：	97/12/22

內文：

Introduction

What is Lichen Planus?

- Affects **stratified squamous epithelium** virtually exclusively
- **An auto-cytotoxic T lymphocytes** trigger apoptosis of epithelial cells leading to chronic inflammation.
- Diagnosis of OLP can be **made from the clinical features**, particularly if typical skin or other lesions are present
- Mostly in the **fifth to sixth decades** of life
- **twice as common in women** than in men
- Treated with **anti-inflammatory agents**, mainly the **topical corticosteroids.**

Aetiology and pathogenesis

- **T cell-mediated autoimmune disease** but its cause is unknown in most cases.
- The **increased production of TH1 cytokines** is a key and early event in LP, it is genetically induced, and genetic polymorphism of cytokines:

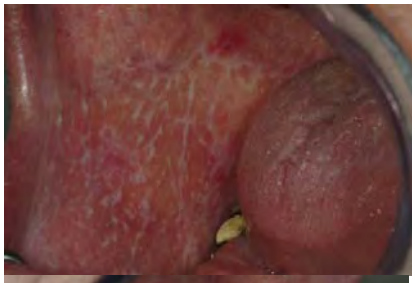
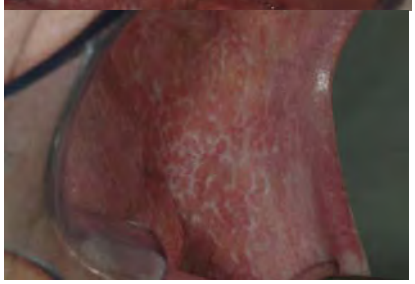
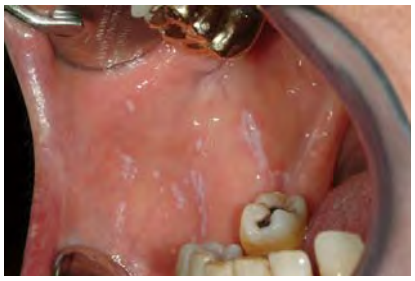
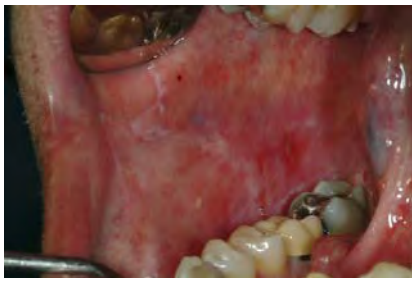
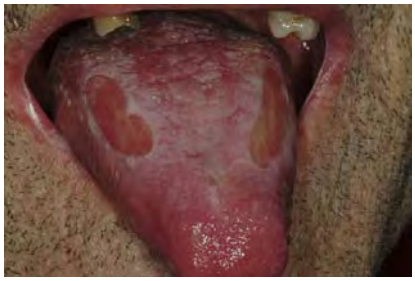
Interferon-gamma (IFN-) associated	**lesions develop in the mouth alone**
Tumor necrosis factor-alpha (TNF-)	in the **mouth and skin**

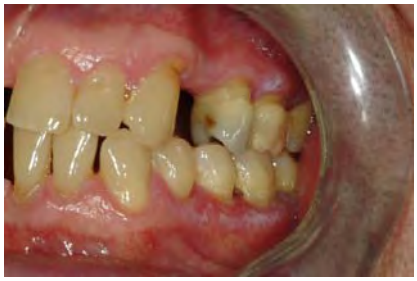
- Activated T cells are then attracted and migrate towards the oral epithelium, further **attracted by intercellular adhesion molecules (ICAM-1 and VCAM)**
- **Upregulation** of **epithelial basement membrane extracellular matrix proteins**(collagen types IV and VII, laminin and integrins, and possibly by CXCR3 and CCR5 signalling pathways.)
- **Cytokines** secreted by keratinocytes **such as TNF- and interleukins (IL)-1,IL-8, IL-10, and IL-12 are also chemotactic for lymphocytes.**
- The **T cells then bind to keratinocytes and IFN-** , and subsequent **upregulation of p53**, matrix metalloproteinase 1 **(MMP1) andMMP3** leads to **programmed death of cells (apoptosis)**, which destroys the epithelial basal cells.
- The inhibition of the transforming growth factor control pathway **(TGF-beta/smad)** may **cause keratinocyte hyperproliferation** that l**eads to the white lesions.**

Association with systemic disease

- **HCV-specific T cells** may have a role in the pathogenesis of some cases of OLP
- Patients with LP had about a **five-fold greater risk** of being infected with HCV

Oral lesion

<table>
<tr><td rowspan="4">Morphology</td><td colspan="2">small, raised, white, lacy lesions</td><td> 
  </td></tr>
<tr><td colspan="2">Papules, plaques, and can resemble keratotic diseases such as leukoplakia.</td><td>  </td></tr>
<tr><td>Atrophic lesions</td><td rowspan="2">Cause pain</td><td>  </td></tr>
<tr><td>Erosions</td><td>  </td></tr>
<tr><td>Site</td><td colspan="2">buccal mucosae,
tongue (mainly the dorsum)
gingival
labial mucosa
vermilion of the lower lip.</td><td></td></tr>
</table>

	About **10% of patients** with OLP have the disease **confined to the gingiva**	

- **Erythematous lesions** that affect the gingiva cause **desquamative gingivitis**, the most common type of gingival LP
- **Uncommon：**
  ---Lesions on the **palate, floor of the mouth, and upper lip**
  ---isolated to a **single oral site** other than the gingival
- **Striated white lesions**, with or without erosions can mimic **lupus erythematosus.**

Malignant potential of OLP

- At least three studies using strict diagnostic criteria have shown a significant risk of malignant transformation of OLP to **squamous cell carcinoma (SCC).**
- The risk of malignant transformation varies between **0.4 and 5%** over periods of observation from **0.5 to 20 years,** and seems to be **independent of the clinical type of OLP** or **the treatment used.**

Extraoral lesion

	Skin	**Skin appendages**	**Extraoral mucosa**	
Rate	15%		20% of women with OLP	male equivalent
Site	flexor surfaces of the forearms	Scalp and nails	vulvovaginal-gingival syndrome	penogingival syndrome
morphology	Erythematous to violaceous, flat-topped, pruritic, polygonal papules that have a network of fine lines (Wickham's striae)	➢ Scarring alopecia, lichen planopilaris ➢ thinning and ridging of the nail plate, and splitting of the distal free edge of the nail.	Burning, pain, discharge, dyspareunia. **(May become malignant)**	may also become malignant.
Duration	several months			

- Oesophageal LP has been well-documented and is **relatively common** in patients with oral LP.

Oral lichenoid reaction

- A term used for lesions that resemble OLP clinically and histologically, but **have an identifiable aetiology.**
- Precipitants include **chronic graft-versus-host disease (cGVHD)**, some **dental materials**, and a range of **drugs**.
- To be **unilateral** and **erosive**, and histological examination may show a **more diffuse lymphocytic infiltrate** with **eosinophils and plasma cells**, and with

more colloid bodies than in classic LP

	Chronic graft-versus-host disease (cGVHD)	**Dental restorative materials**	**Drugs**
Origin	Haematopoietic stem cell transplantation	Amalgams composite resins cobalt, and gold	--non-steroidal anti-inflammatory agents --angiotensinconverting enzyme inhibitors
Side effect	--high risk of developing **secondary neoplasms** **--leukaemias,lymphomas** --risk of **squamous cell carcinomas**	There have also been reports of **malignant transformation** of restoration-related lichenoid lesions	

Diagnosis of OLP

- **Oral biopsy** with histopathological examination is recommended both to **confirm the clinical diagnosis** and particularly **to exclude dysplasia and malignancy**

Management of OLP

- Depends on **symptoms**, the **extent of oral and extra-oral clinical involvement**, **medical history**, and other factors.
- Patients with **reticular and other asymptomatic** OLP lesions
  --- no active treatment
- Patients **with symptomatic** lesions
  --- need treatment, usually with **drugs**, but **occasionally surgery has a role**.
- **Mechanical injury** or irritants such as **rough restoration margins or badly fitting dentures**
  ---given attention, and an optimal programme of oral hygiene instituted, particularly in patients with gingival LP.

Drug treatment	➢ Topical agents ➢ Fewer adverse effects. ➢ Systemic agents may be required if lesions are widespread, or there is recalcitrant disease
Topical corticosteroids	➢ Midpotency topical corticosteroids ➢ superpotent halogenated steroids ➢ Elixirs ➢ apply the steroid several times daily, to maintain the drug in contact with the mucosa for a few minutes, and they should refrain from eating and drinking for 1 hour afterwards.
Other topical agents	➢ calcineurin inhibitors ➢ retinoids: particularly **atrophic-erosive forms**, with considerable improvement ➢ Cyclosporine ➢ Tacrolimus: accelerates skin carcinogenesis in mice
Systemic drug treatment	usually reserved for cases where **topical approaches have failed**, where there is r**ecalcitrant, erosive, or erythematous OLP**, or for widespread OLP when skin, genitals, oesophagus, or scalp are also involved.

Surgery	➢ **Resection** has been recommended for isolated plaques or non-healing erosions ➢ **Free soft-tissue grafts** have also been used for localised areas of erosive OLP. ➢ **Cryosurgery** has been used particularly in erosive drugresistant OLP ➢ **Lasers** have also been used to treat OLP
Cancer surveillance	It seems prudent to monitor patients with OLP in the long term.

題號	題目
1	下列關於lichen planus的敘述何者正確? (A) 臨床上最常出現在單側的頰黏膜 (B) 組織病理學上，在表皮及黏膜下層的交接處有衣帶狀的發炎細胞浸潤，大部分為plasma cell (C) 組織病理學上，帶狀的發炎細胞浸潤侵蝕表皮的基底細胞，形成coagulation necrosis (D) 臨床上，可以出現紅斑周圍有網狀的白色細線稱為Wickham's striae
答案(D)	出處：94年第二次檢覆筆試試題
題號	題目
2	Lichen Planus有可能與下列何種系統性疾病相關? (A) HAV (B) HBV (C) HCV (D) HEV
答案(C)	出處：