

Oral Lichenoid Drug Eruptions: Their Recognition and Management

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Abstract: Lichen planus is a relatively common, often clinically distinctive, mucocutaneous condition with an uncertain aetiology. One variant of lichen planus is the so-called 'lichenoid drug eruption'. In contrast to idiopathic lichen planus, lichenoid drug eruptions, where practicable, may be managed by substitution of the offending drug. The dental clinician is in a prime position to identify these lesions and liaise with medical colleagues regarding their management. This article reviews oral lichenoid drug eruptions, emphasizing those aspects of relevance to the general dental practitioner.

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Clinical Relevance: The accurate diagnosis of drug-induced oral lichenoid eruptions, in particular differentiating them from idiopathic lichen planus, allows the dental clinician to play an important role in their successful management.

Lichenoid drug eruptions (LDEs) can be considered a variant of lichen planus (LP), and reports of such reactions date back to 1929.¹ Several cases were diagnosed during World War II, apparently related to the use of prophylactic antimalarial drugs. However, such reports were suppressed until the cessation of hostilities.^{2–5} Since then numerous drugs have been reported to be associated with LDE, although only some of these have been confirmed as causing oral involvement.^{6–8}

The diagnosis of an oral LDE (as opposed to idiopathic oral lichen planus; OLP) is not necessarily

straightforward and pathognomonic characteristics of an oral LDE are yet to be identified. In order for practitioners to develop a working knowledge of the subject and feel confident in diagnosis, oral LDEs need to be considered alongside idiopathic OLP. This article aims to review briefly the clinical, histopathological and aetiological aspects of these two conditions, thereby providing guidance to the general dental practitioner in his/her differential diagnosis and management.

DRUGS IMPLICATED IN THE DEVELOPMENT OF LICHENOID LESIONS

The clinician must be aware of the possibility of oral adverse drug events (not only as a cause of LDEs) and a detailed, accurate, drug history is mandatory in the management of all patients. It is noteworthy that, of the

patients with OLP/LDE attending the Oral Medicine Clinic at the University of Birmingham School of Dentistry, more than 50% are using some form of systemic medication. Relating the commencement of medication (or changes in dosing) to the initial onset of oral symptoms may identify aetiological clues. However, there may be a lag phase between the start of medication and onset of signs and symptoms,^{9,10} which may mislead the unwary.

Any suspicion of an adverse drug event should be reported to the Committee on Safety of Medicines using the familiar 'yellow card' system. This is particularly important with drugs not known to be associated with such events.

Although this is not the subject of this paper, it is worth mentioning that, in addition to the many drugs associated with LDE (those commonly encountered by the general dental practitioner are listed in Table 1), dental materials, including both amalgam^{11–13} and (albeit only in a few cases) composite resins,^{14,15} have also been associated with lichenoid reactions.

Confirmation of a possible LDE may be achieved by the withdrawal and subsequent rechallenge with the suspect drug, monitoring the effects on the oral lesions. However, this should be undertaken only with the agreement of the patient and his/her medical advisor. In some cases, such an approach is unrealistic and therefore the connection with certain drugs can be difficult to prove.

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| Drug group | Example |
|---------------------------------------|--|
| Antihypertensives | ACE inhibitors (e.g. captopril), beta-blockers (e.g. propranolol, atenolol), calcium-channel blockers, methyldopa , thiazide diuretics, loop diuretics (e.g. frusemide) |
| Oral hypoglycaemics (sulphonylureas) | Tolbutamide, chlorpropamide |
| Non-steroidal anti-inflammatory drugs | Ibuprofen, naproxen, phenylbutazone |
| Second line anti-arthritis | Gold, penicillamine |
| Xanthine oxidase inhibitors | Allopurinol |
| Psychoactive drugs | Lorazepam, tricyclic antidepressants, lithium, carbamazepine, phenothiazines |
| Antiparasitic agents | Antimalarials (e.g. chloroquine, pyrimethamine, levamisole) |
| Antimicrobial agents | Tetracyclines, ketoconazole, dapsone, streptomycin, mepacrine |
| Miscellaneous | Iodides, quinidine |

Table 1. Drugs associated with LDE (drugs in bold print, although not necessarily commonly prescribed, are thought to be potent oral LDE inducers).

CLINICAL FEATURES OF IDIOPATHIC OLP AND LDE

Epidemiology

A female predilection is generally seen in OLP,¹⁶⁻¹⁸ with a typical age range of 30-70 years.¹⁹ It has been reported that the prevalence of OLP is 1.9%;¹⁶ however, the figure depends on the population studied.²⁰

Site

Idiopathic OLP presents, in 90% of cases, on the posterior buccal mucosa,²⁰ often adjacent to the occlusal line²¹ (Figure 1).



Figure 1. Reticular lichen planus of buccal mucosa. Note the presence of papules, which may eventually coalesce to form striae. Typically, idiopathic OLP presents bilaterally on the posterior buccal mucosae in a reticular pattern.

The lesions are usually bilateral and symmetrical or at multiple sites in the mouth.²² Other oral sites include the tongue (30%) and/or alveolar ridge/gingiva (13%).²⁰ Idiopathic OLP rarely affects the palate²³ or vermilion border of the lip,¹⁶ although it has been reported on the lips alone.^{24,25}

Oral LDEs can appear at sites atypical for OLP, such as the palate (Figure 2) and, unlike OLP, lesions tend to be unilateral.²⁶ Some clinicians believe that labial involvement may be more frequent in LDE than in OLP. Labial involvement has also been reported as enhanced in HIV infection.²⁷

Morphology

Andreason described six clinical variants of OLP.²⁸

- The reticular form is frequently asymptomatic and characterized by a fine, lacy network of slightly elevated striae, which is sometimes accompanied by:
- The papular form.²⁸ This consists of small (0.5-1 mm) white, raised papules that can coalesce. Other patterns (e.g. annular) may coexist in the same patient.

- Erosive or ulcerative OLP presents as painful, irregular, often persistent lesions. A number of oral LDEs present as erosive lesions (Figure 3), particularly those associated with non-steroidal anti-inflammatory drugs.²⁹

The remaining variants are:

- atrophic OLP (Figure 4), which is diffuse and erythematous;
- plaque-type OLP, which mimics leukoplakia (Figure 5); and
- bullous OLP, which is rare, and (as its name suggests) presents with blisters. It has been reported that some of the lesions diagnosed as the bullous form are actually superficial mucocoeles.³⁰

Gingival involvement usually presents as a desquamative gingivitis (Figure 6). Differential diagnosis must include other mucocutaneous disorders such as pemphigoid and pemphigus.³¹

HISTOLOGY AND PATHOGENESIS

Much has been written about the

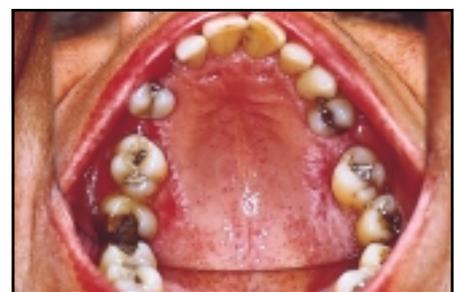


Figure 2. Palatal lichenoid lesion. LDEs may show an increased tendency to present at sites atypical for OLP, such as the palate.



Figure 3. Erosive lichenoid lesion. LDEs may be more likely to present as erosive lesions than idiopathic OLP.



Figure 4. Atrophic lichen planus of buccal mucosa. Note the presence of peripheral striae.



Figure 5. Plaque-type lichen planus of tongue dorsum.

cellular, histological and immunological aspects of oral LDE and OLP, and it is outside the scope of this article to provide a comprehensive view of this complex area.

Attempting to differentiate between the two conditions histologically may be difficult as, in common with the clinical findings, those features considered to be characteristic of an LDE can also be identified in some cases of idiopathic OLP.⁷

The histological features of OLP (Figure 7) vary according to the clinical picture and are not dissimilar from those of the cutaneous form.³² Classical



Figure 6. Gingival lichen planus presenting as a desquamative gingivitis. Not unusually, plaque-related gingivitis (superimposed because pain on tooth brushing reduces oral hygiene) makes diagnosis more difficult.

features include:

- hyperparakeratosis or hyperorthokeratosis;
- liquefaction degeneration of the basal cell layer;
- epithelial atrophy;
- a dense subepithelial band of predominantly T lymphocytes;
- elongated, widened, and flattened rete pegs;
- acanthosis;
- civatte bodies.

At a cellular level, lichen planus probably results from an immunologically induced degeneration of the basal layer.^{33,34} Whilst the basal cells are considered to be the prime target for a cell-mediated immunological reaction, the precise antigen remains unknown.³⁵⁻³⁷

In addition to a band-like infiltrate of T lymphocytes in the lamina propria (which may be particularly dense in areas of liquefaction degeneration of the basal layer), further evidence to support a cell-mediated pathogenesis is the early appearance of Langerhans' cells.³⁸ Although the numbers of these antigen-presenting cells may be normal^{39,40} or increased,⁴¹ their morphology may be more dendritic, suggesting increased surface expression of Class II major histocompatibility antigens.^{39,40} Such immunological activity may have an aetiological role to play. In LDEs, such expression of MHC Class II antigens is reduced and the expression of CD25 (marker of probable cell activation) is not seen.^{36,37}

There is little evidence to date that humoral immunity plays a significant role in the aetiopathogenesis of lichen planus. Circulating autoantibodies are not usually identifiable, and lesional plasma cells are not abundant,^{42,43} except when the lesion is ulcerated. Their presence may represent a non-specific secondary event. Aside from the colloid bodies (also known as civatte bodies, and probably derived from epithelial degeneration⁴⁴) present within the epithelial layer⁴⁵ and staining positive for IgM⁴⁶ (possibly a secondary humoral response to cell debris), direct immunofluorescence of lesional tissue is negative for the major



Figure 7. Histology of OLP. Note the dense subepithelial, lymphohistiocytic infiltrate comprising mainly T lymphocytes, reflecting the cell-mediated aetiology. (Haematoxylin and eosin stain, x250.)

immunoglobulin isotypes.⁴⁶

DIFFERENTIAL DIAGNOSIS

Certain histological features are suggestive of LDE, but whether a valid statistical correlation exists remains to be demonstrated.⁷ Each of the features listed below can also be found in OLP and, as some of these features are subjective, different pathologists may report varying observations.

The following histological features may suggest an LDE:

- Subepithelial infiltrate is more diffuse⁴⁷ and less band-like, with deeper extension into the connective tissue^{48,49}, and a more mixed cell population, including eosinophils^{5,47-51} and plasma cells.⁴⁷⁻⁴⁹
- Perivascular infiltrate.⁴⁷⁻⁵¹
- Parakeratosis.^{47-49,51,52}
- Colloid bodies in the epithelial layer.^{49,51}

Basal epithelial cell cytoplasmic autoantibodies have been detected in some patients with oral lichenoid lesions,²⁶ supporting the view that there may be a different target antigen in the pathogenesis of these two conditions. Whether a possible humoral response to such an antigen in LDE can be used as a diagnostic tool in the differentiation of these two conditions remains to be seen. It has also been demonstrated that Langerhans' cells show that the level of

| | |
|---------------------------|--|
| History | Drug history, particularly with the presence of known oral LDE inducer. |
| Examination | Unilateral lesions at atypical sites such as palate and labial mucosa. Erosive lesions, particularly if patient taking non-steroidal anti-inflammatory drugs. |
| Histopathology/immunology | More mixed, diffuse, and deeper subepithelial infiltrate; perivascular infiltrate; parakeratosis; colloid bodies in epithelial layer; presence of circulating basal cell cytoplasmic autoantibodies. |

Table 2. Factors that may suggest LDE.

HLA-DR expression in LDE is lower than in OLP, consistent with a decreased level of cell activation.^{33,53} Decreased local activation of these cells might be expected with systemic drug administration as antigen presentation may be at a remote site.

CONCLUSION

The differentiation between a drug-induced lichenoid reaction and idiopathic oral lichen planus is not always straightforward and the factors that may suggest an LDE are given in Table 2. The clinical suspicion of the presence of a lichenoid reaction is raised by an appropriate drug history, particularly if the patient is taking a 'high-risk' drug. The index of suspicion is enhanced by a clinically atypical distribution of lesions, including involvement of unusual and unilateral sites, and 'non-classical' (i.e. lichenoid) histology. The presence of basal epithelial cell cytoplasmic autoantibodies would further support diagnosis of an LDE but such a test is not routinely available.

When there is reasonable certainty of an LDE, the lesion fails to respond to topical treatment, and the patient is distressed by these oral lesions, it is appropriate to liaise with medical colleagues to consider alternative medication. The ultimate diagnostic test is improvement in symptoms on withdrawal of the suspect drug and their re-emergence on rechallenge. However, the absence of immediate resolution does not preclude an adverse drug event as, in unusual cases (notably with gold, penicillamine and methyldopa, all potent LDE-inducing agents), symptoms can persist for long periods following

drug withdrawal.^{47,54}

It is now accepted that OLP has malignant potential⁵⁵ and, in the absence of any evidence to the contrary, it may not be unreasonable to assume that LDEs may carry a similar risk. Effective management is thus important.

The general dental practitioner, armed with a little knowledge, is in a prime position to identify an oral LDE and make an early referral to a specialist in oral medicine. The opportunity may exist to effect resolution of these eruptions by appropriate manipulation of the patient's medication in conjunction with the patient's medical practitioner and specialist hospital services.

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CD-ROM REVIEW

The Last Post; Restoration of the Endodontically Treated Tooth. The Dental Channel Ltd. CPD CAL CD-ROM. Obtainable free of charge via your postgraduate deanery or order directly from the website [www.dental-channel.co.uk/PG deans.htm](http://www.dental-channel.co.uk/PG_deans.htm)

Most dentists in practice will be fitting a large number of post crowns annually, as a routine means to retain badly broken down teeth. So, is there a need for a CAL program devoted entirely to such a well-established and traditional restorative technique? The National Centre for Continuing Professional Education in Dentistry and the Department of Health obviously think so, and the generally high failure rate of post crowns in practice would suggest that there is certainly room for reviewing how and why post crowns are produced. Dr P.B. Robinson, a senior lecturer and consultant in restorative dentistry at GKT has produced this program with the Dental Channel and they are to be congratulated for producing a comprehensive educational package, which makes full use of the

opportunities afforded by a computer-based format. Video, animations, graphics and photographs complement the text and narration. There are many links between the various sections, an interactive quiz to test your knowledge, and the facility to fulfil CPD requirements online.

This program covers the importance of planning and individual assessment, and explores the relationship between the many elements that go to make up a post crown restoration, of which the post is but one part. There is a very important section dealing with the root anatomy of various teeth, and due emphasis is made on making the appropriate choice of system for each case.

Minor criticisms would include the typographic errors in some of the text, and that more comparative facts might have been included regarding core materials and cements, as these aspects are not covered in great depth and dentists need guidance in choosing from the many materials on the market. It was confusing to include composite as a suitable core material in the relevant reference section only to be told in one of the video cases that composite is precluded from use owing to its microleakage, but that it can be

used! Apart from these minor issues, this program is a very full and worthwhile resource for dentists at all levels. It is easy to follow and use, and should contribute to higher standards in this area of clinical dentistry.

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OCTOBER
 Self-Assessment
 Answers

- | | |
|------------|-------------|
| 1. A, B, C | 6. A, B, C, |
| 2. A, B, D | 7. A, B, D |
| 3. B, C | 8. A, B, D |
| 4. A, B, C | 9. A, C |
| 5. B, C, D | 10. A, D |