Oral Epithelial Dysplasia, Grading, Management and Significance

Richard C. Jordan DDS PhD FRCPath
Professor of Oral Pathology, Pathology & Radiation Oncology

What we will cover

• Leukoplakia, erythroplakia
• Causes of oral epithelial dysplasia
• Terminology & grading
• Risk of transformation to cancer
• Treatment
• Verrucous hyperplasia & carcinoma
• Proliferative verrucous leukoplakia (PVL)

Potentially malignant disorders

Leukoplakia

Erythroplakia

International Workshop on Oral Potentially Malignant Disorders
London, May 2005

Leukoplakia

‘The term leukoplakia should be used to recognise white plaques of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer’
Erythroplakia

“a red patch on the oral mucosa which cannot be characterised clinically or histologically as due to any other condition”

Tobacco smoking

- definite relationship with oral cancer
- risk is greatest in heavy users (>20/day)
- risk is greater if accompanied by alcohol use
- risk may be greater in “reverse” smoking and with pipes and cigars
Epidemiology of leukoplakia

Prevalence:
- Ranges from 0.9% to 26.9%
- Depends on site and size of study

Recent systematic review shows worldwide prevalence of:

2.6%

Petti (2003). Oral Oncology, 39, 770-780
Leukoplakia

Homogeneous
flat and plaque-like, uniformly white

Non-homogeneous
nodular, verruciform, exophytic, speckled

Leukoplakia - Histology

Up to 80% show no dysplasia

Homogeneous Leukoplakia
Only about 20% are dysplastic

Non-homogeneous Leukoplakia
About 50% are dysplastic
Dysplasia grading schemes

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Architectural (Tissue) changes:

- Loss of polarity
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- Increased cellular density
- Basal cell hyperplasia
- Dyskeratosis (premature keratinization and keratin pearls deep in epithelium)
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- Abnormal variation in nuclear size and shape (anisonucleosis and pleomorphism)
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- Increased nuclear/cytoplasmic ratio
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- Increased number and size of nucleoli

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Cellular changes

Pleomorphism of cells and nuclei

Architectural changes

- Bulbous rete pegs
- Basal cell hyperplasia
- Loss of basal polarity & cell crowding

Courtesy P. Speight U. Sheffield
Mild epithelial dysplasia

Changes are limited to the lower 1/3 of the epithelium

Moderate epithelial dysplasia

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  - Basaloid appearing
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  - No or minimal inflammation
  - Abnormal mitoses at odd levels

Is epithelial dysplasia a useful marker of potential progression of oral precursor lesions?

What becomes of dysplastic lesions?

- Malignant 20%
- Regress 20%
- No change 40%
- Increase in size 20%

What lesions progress to cancer?

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Managing dysplasia

- Remove any residual lesion
- Don’t chase microscopic margins
- Re-biopsy if lesion changes (they often recur)
- Retinoids don’t help

Verrucous carcinoma

- Rare variant of SCC 1-3/million
- Tobacco, not HPV
- Slow growing exophytic verrucous patch
- Locally destructive, rarely metastasizes
- Buccal mucosa>gingiva>tongue> palate>other
- Well differentiated carcinoma; little or no dysplasia
- Excision, prognosis excellent

Binary classification system

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Verrucous hyperplasia

Verrucous carcinoma

PVL - history

• Described by Hansen 1985 in 30 patients
• Prior to 1985 “oral florid papillomatosis”
• Slowly growing, persistent hyperkeratosis, multifocal
• Resistant to treatment
• to 2014 – 69 papers on PVL

PVL clinical

• 80 % women
• Mean age 71 years
• Gingiva > BM > palate
• Starts as a flat white lesion progressing to verruciform lesion
• Multifocal
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- Verruciform hyperkeratosis
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- Verrucous carcinoma
- Papillary SCC

### PVL & HPV

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Oral epithelial dysplasia

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Increasing severity (Hyperchromatism & crowding)

Normal keratinocytes

Atypical keratinocytes

Mild dysplasia

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Progression of dysplasia

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