

Oral potentially malignant disorders: risk of progression to malignancy



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Oral potentially malignant disorders (OPMDs) have a statistically increased risk of progressing to cancer, but the risk varies according to a range of patient- or lesion-related factors. It is difficult to predict the risk of progression in any individual patient, and the clinician must make a judgment based on assessment of each case. The most commonly encountered OPMD is leukoplakia, but others, including lichen planus, oral submucous fibrosis, and erythroplakia, may also be seen. Factors associated with an increased risk of malignant transformation include sex; site and type of lesion; habits, such as smoking and alcohol consumption; and the presence of epithelial dysplasia on histologic examination. In this review, we attempt to identify important risk factors and present a simple algorithm that can be used as a guide for risk assessment at each stage of the clinical evaluation of a patient. (Oral Surg Oral Med Oral Pathol Oral Radiol 2018;125:612–627)

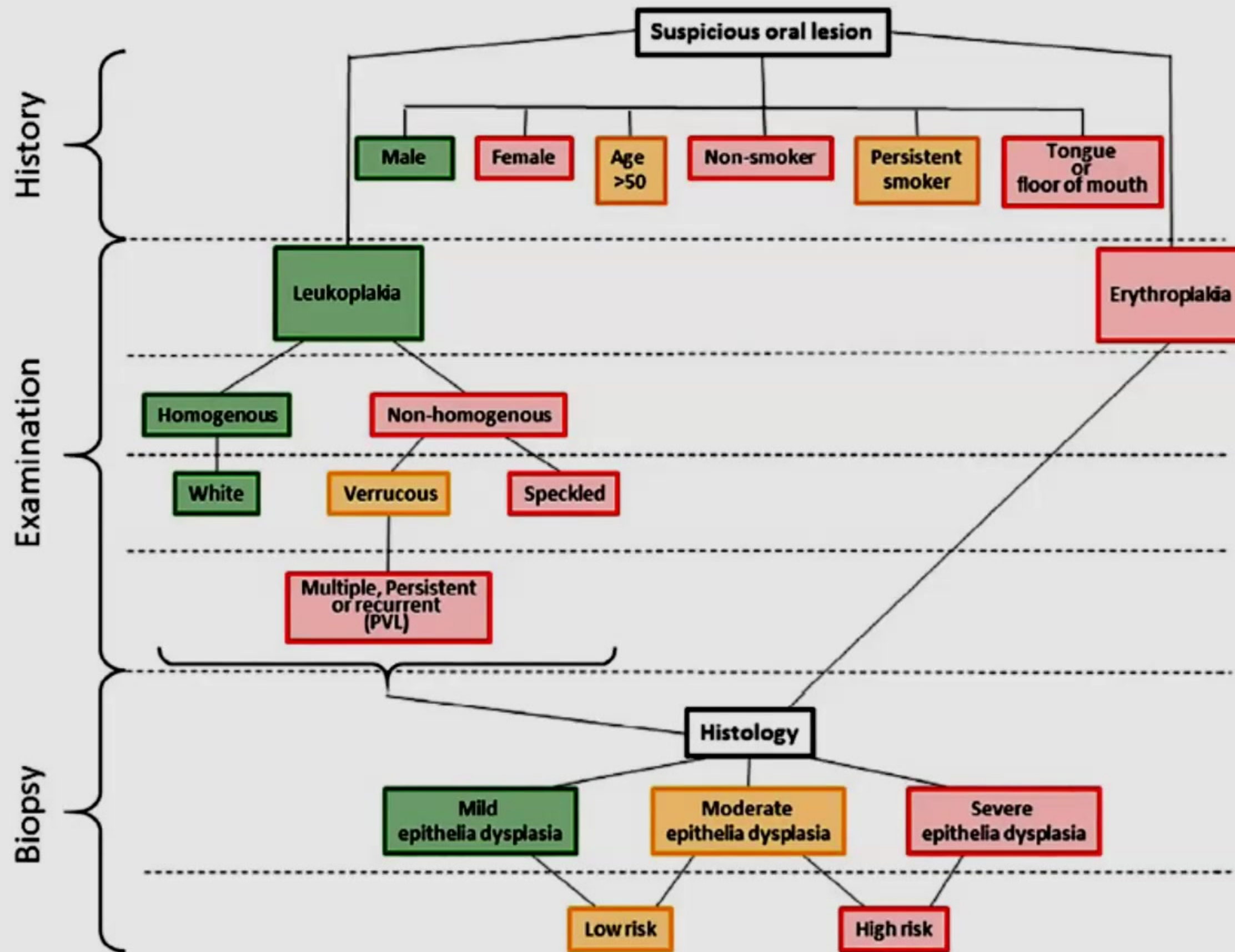


Fig. 3. A simple algorithm for clinical risk assessment of oral potentially malignant disorders (OPMDs). The clinician is faced with a suspicious oral lesion, and at each stage of the assessment process the risk of individual features are illustrated as green (low risk), amber (medium risk), or red (high risk). The levels of risk and explanations are given in the text.

Idiopathic white patches

clinically these are “leukoplakia”

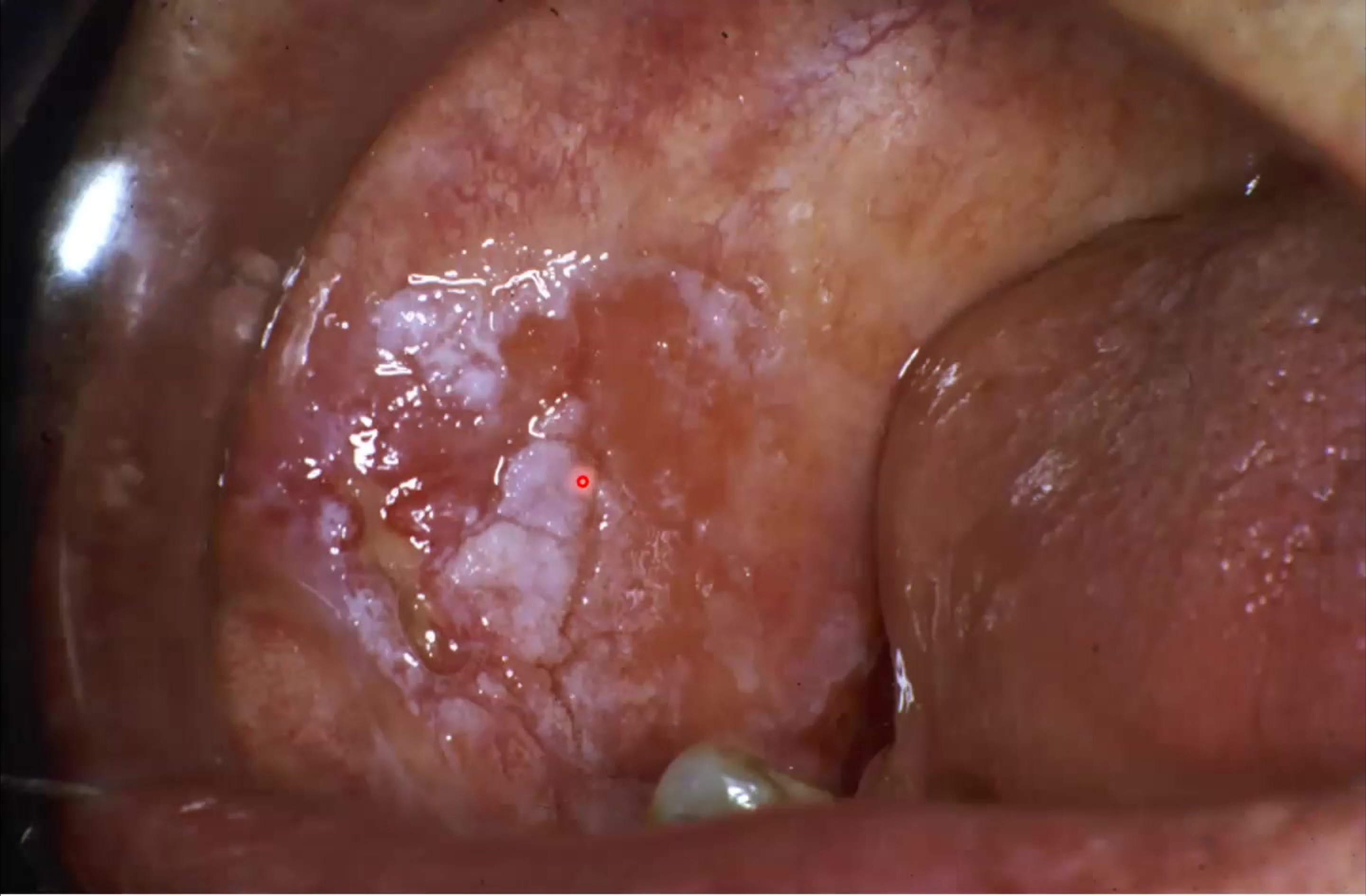


- keratosis with or without dysplasia
 - Presentation: homogeneous, nodular, verrucous, speckled
 - Site: sublingual keratosis
 - Presumed aetiology: Smokers', actinic keratoses
 - Proliferative verrucous leukoplakia

- squamous cell carcinoma



Sublingual keratosis



Speckled and nodular leukoplakia



Differential diagnosis of white lesions in the mouth



- Number of possibilities
- High frequency of insignificant lesions

Useful categories of lesion

- Not really a white lesion
- Normal
- Developmental
- Traumatic
- Infectious
- Lichen planus and similar conditions
- Idiopathic

How to differentiate lesions

- Carcinoma?
- Is it normal?
- Evidence for a developmental condition?
- Habits and causes of friction?
- Does site help - CHC, OHL, SN
- Any striae?
- Risk features for dysplasia or malignancy?
- Smear it
- Biopsy it

Clinical

PRACTICE

Evaluation of a Suspicious Oral Mucosal Lesion

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ABSTRACT

Dentists who encounter a change in the oral mucosa of a patient must decide whether the abnormality requires further investigation. In this paper, we describe a systematic approach to the assessment of oral mucosal conditions that are thought likely to be premalignant or an early cancer. These steps, which include a comprehensive history, step-by-step clinical examination (including use of adjunctive visual tools), diagnostic testing and formulation of diagnosis, are routinely used in clinics affiliated with the British Columbia Oral Cancer Prevention Program (BC OCPP) and are recommended for consideration by dentists for use in daily practice.

Introduction

- More than 350,000 patients have oral cavity and lip cancer annually in the world

Oral Malignant neoplasms

- Common: OSCC
- Less common:
 - Salivary gland tumours
 - Malignant melanoma
 - Lymphoma
 - Neoplasms of bone and connective tissue
 - Some odontogenic tumours
 - Maxillary antral carcinoma
 - Metastatic neoplasms
 - Kaposi sarcoma

Molecular alterations in OSCC

- Oral Cancer is a genetic disease.
- The genetic defects could be induced by **gene amplification** or **translocations** or point mutations or **rearrangements** and **deletions**
- Oral cancer needs~ 6-7 specific mutations

Squamous cell • carcinoma

**The most common malignant
epithelial neoplasm of the oral
region**



**Squamous cell
carcinoma:
common sites**

Features of malignant neoplasms

History: slow- or fast-growing, pain or other neurological signs +/-, impaired function, risk factors (where known)

Consistency: indurated, non-encapsulated

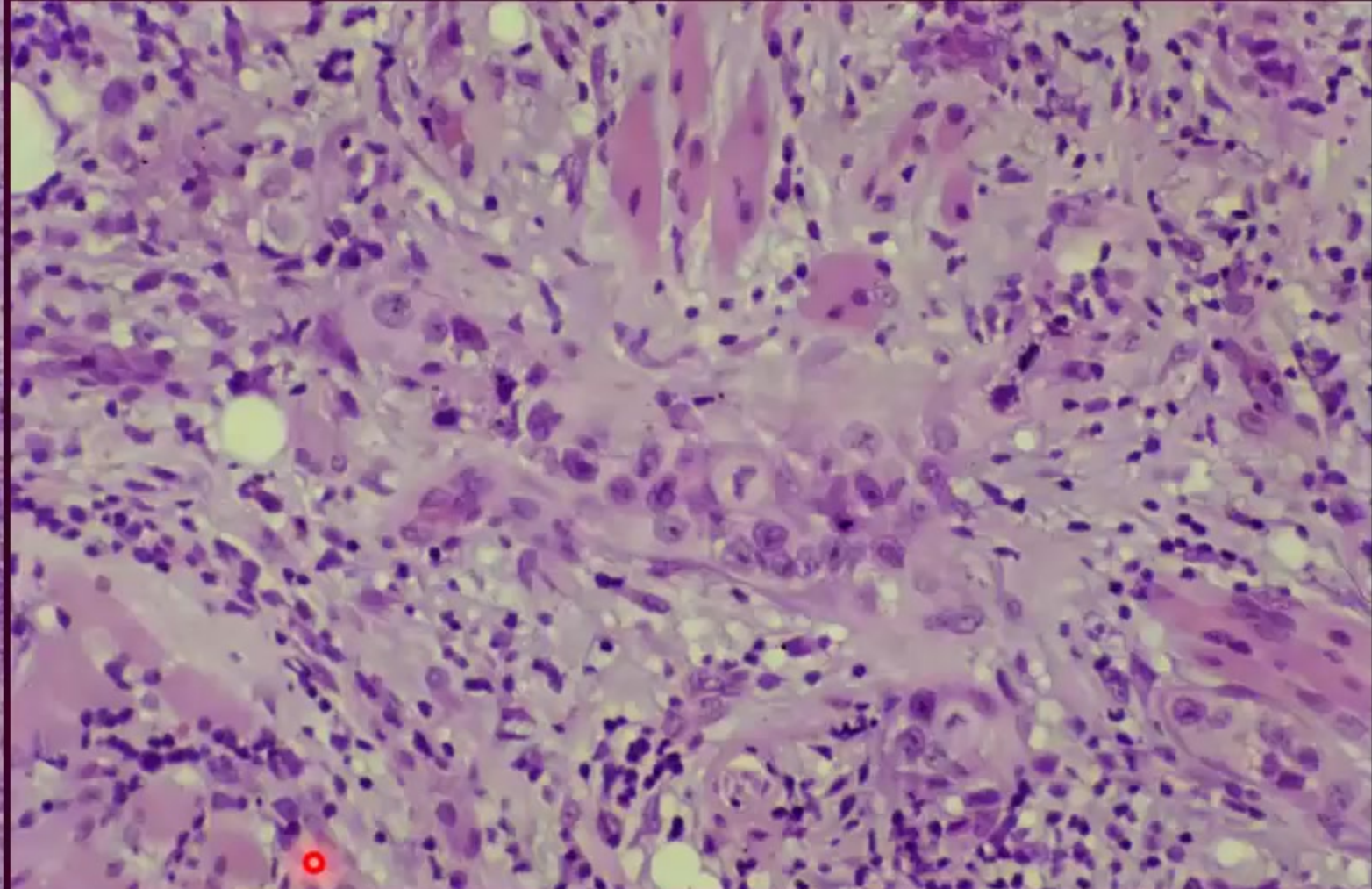
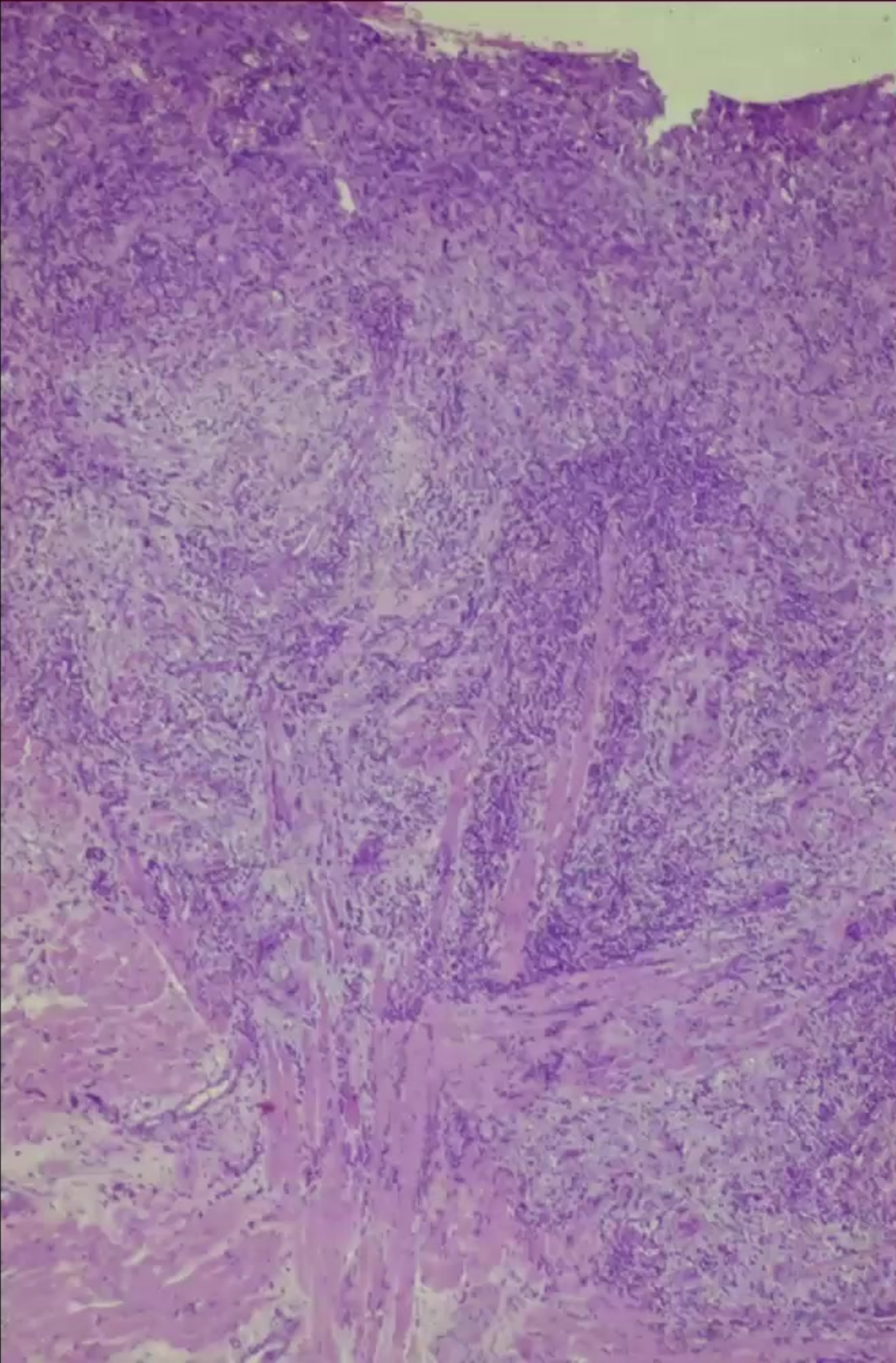
Size: progressive increase, large or small

Ulceration: +/-

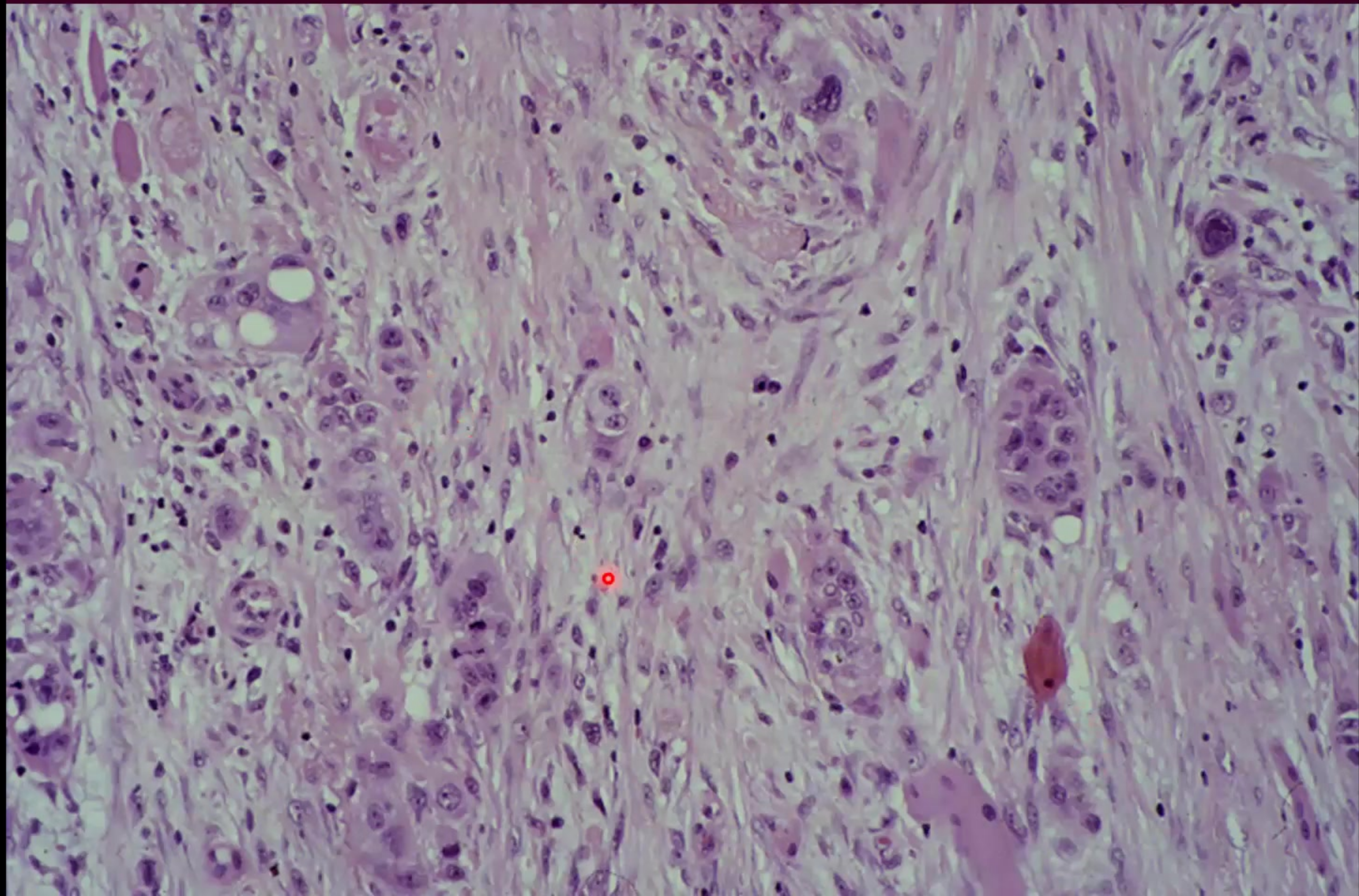
Spread: metastatic to lymph nodes

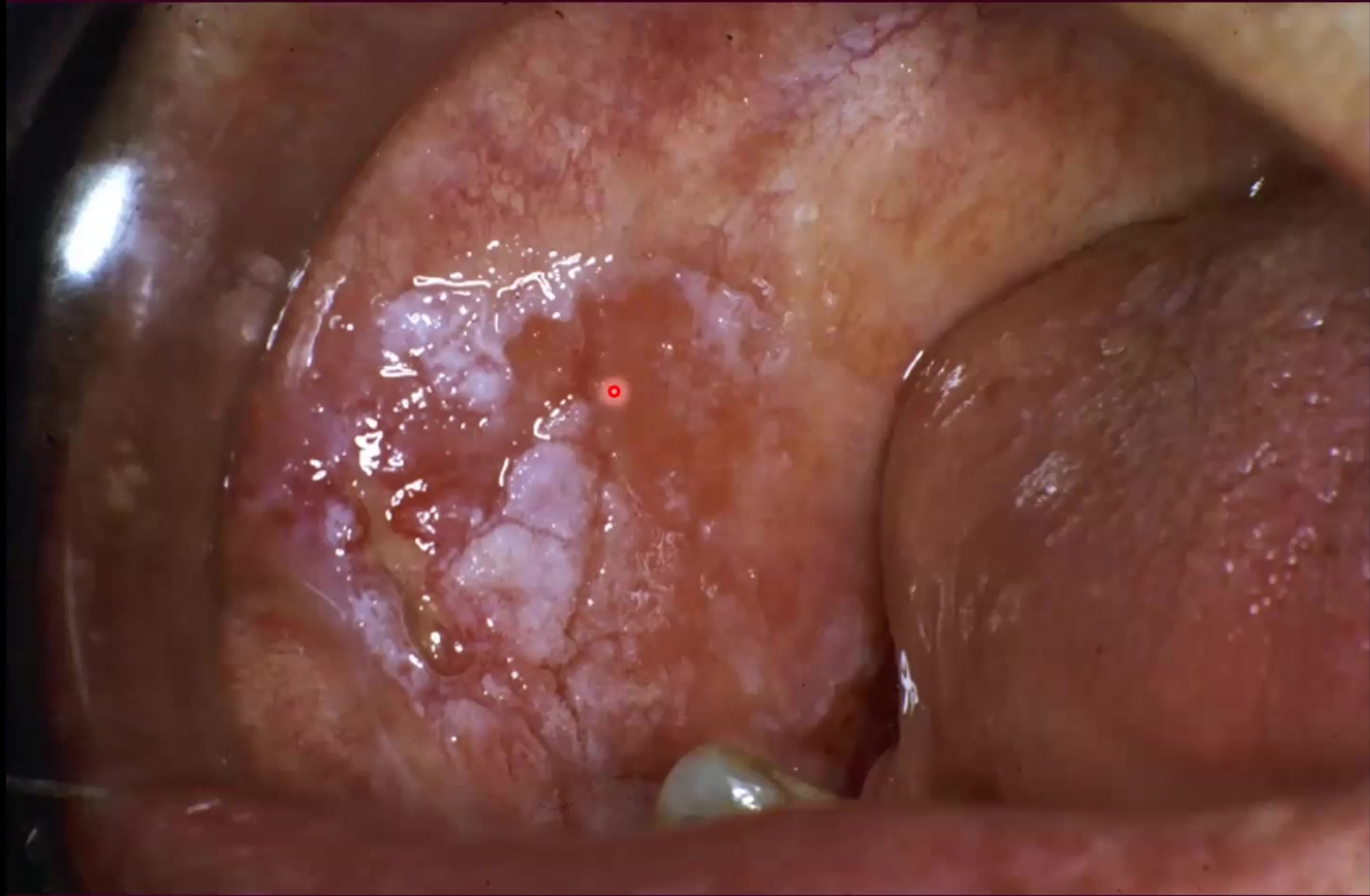
Imaging: irreg. pattern of destruction in bone or invasion in soft tissues

Poorly differentiated
squamous cell
carcinoma

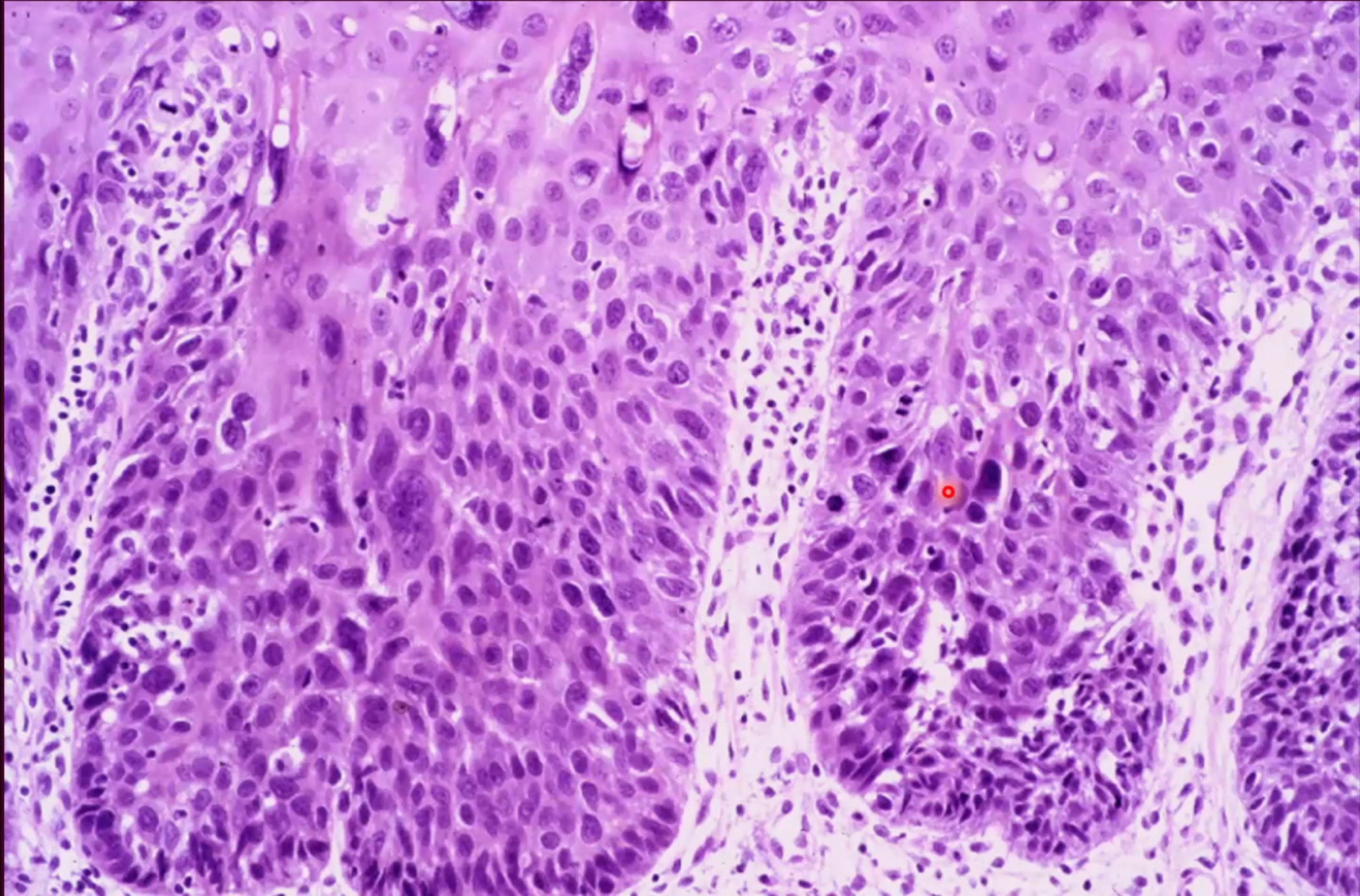


Poorly differentiated SCC





Severe epithelial dysplasia



Dysplasia

Disturbed differentiation and proliferation

Disordered architecture

Deep cell keratinisation

Cell and nuclear pleomorphism

Nuclear hyperchromatism

Increased nuclear/cytoplasmic ratio

Abnormal mitoses

ORAL CANCER - LIP

- **Most commonly affected site.**
- **Geographical variation; U-V light exposure.**
- **Susceptibility; race and immunosuppression.**

ORAL CANCER - LIP

- **Non-healing ulcer.**
- **Painless.**
- **Easily overlooked.**



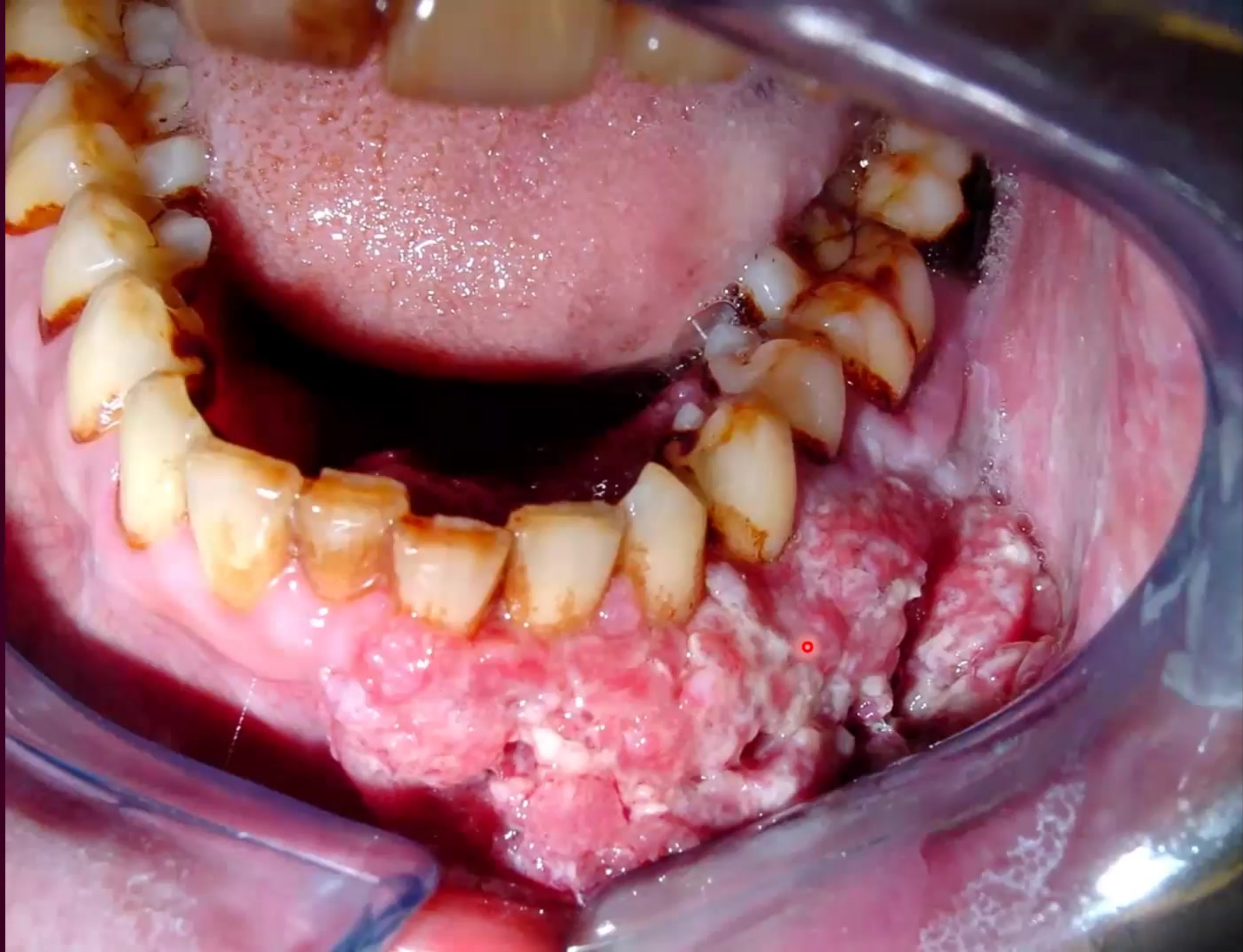




ORAL CANCER-INTRAORAL

- **Typical presentation is a painless indurated ulcer.**
- **Floor of mouth and ventral tongue are the most common intraoral sites.**









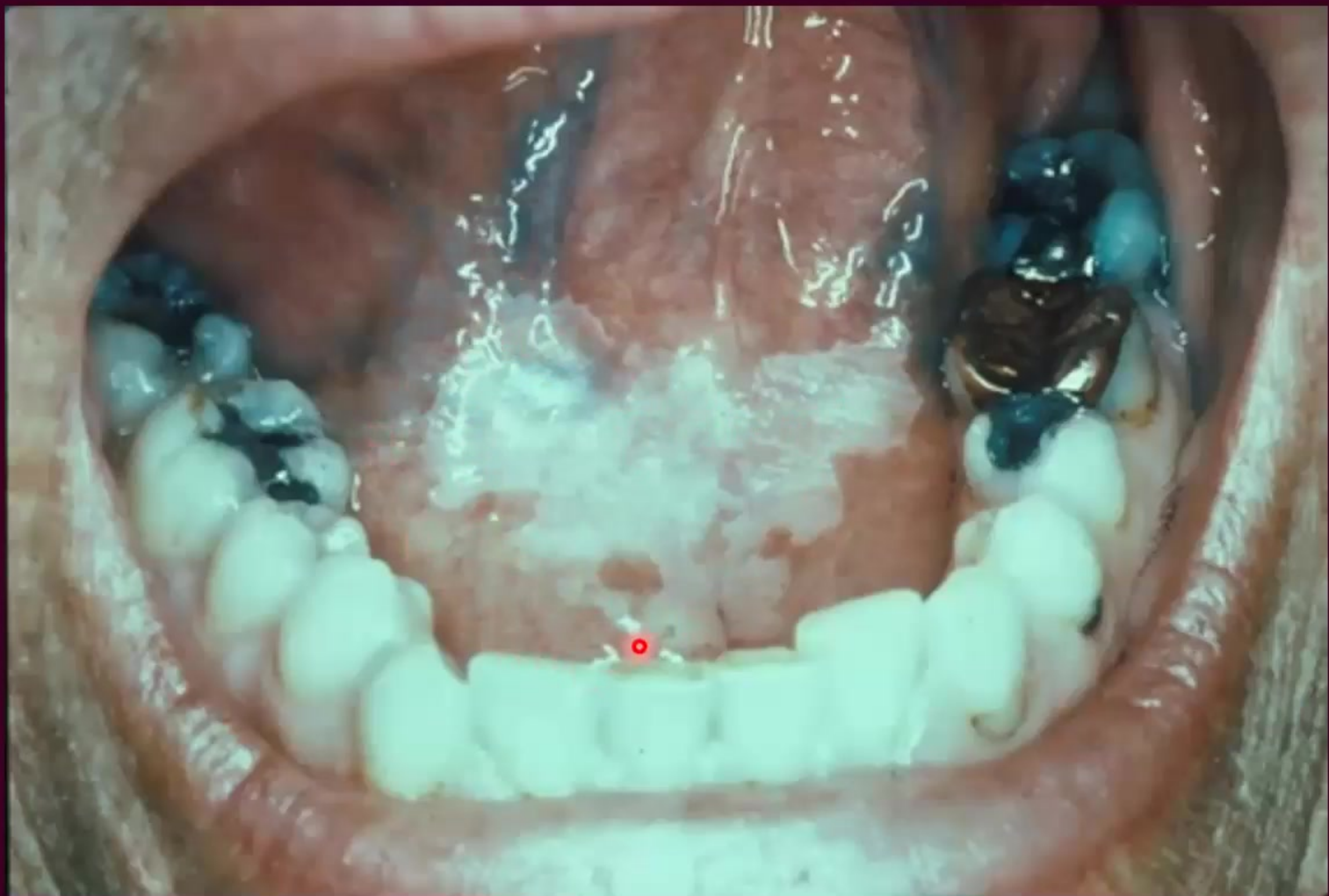
ORAL CANCER-INTRAORAL

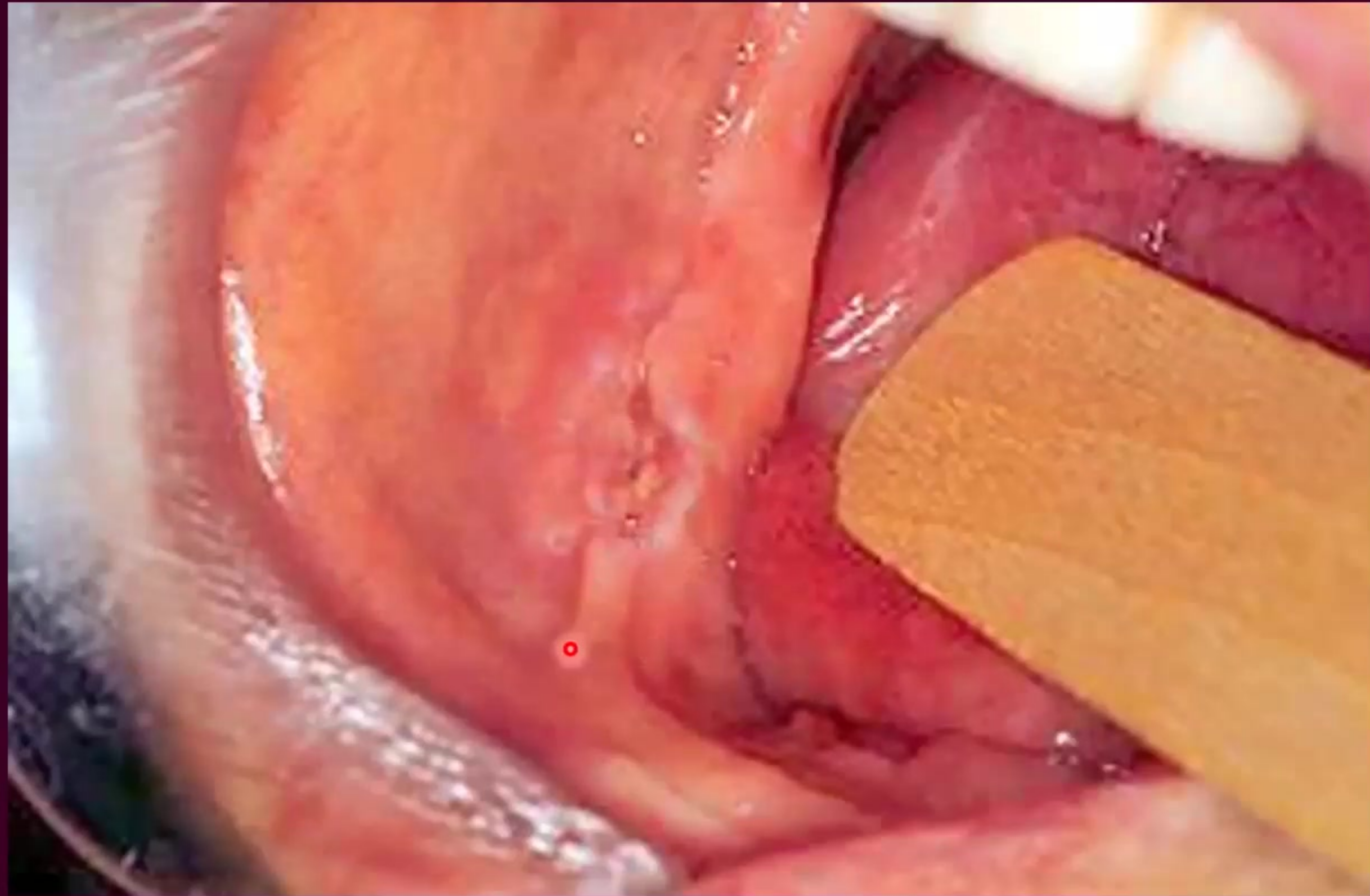
- **Less common presentation**
- **Red, soft, raised lesion**

ORAL CANCER-INTRAORAL

- **Less common presentation**
- **Indurated white patch**







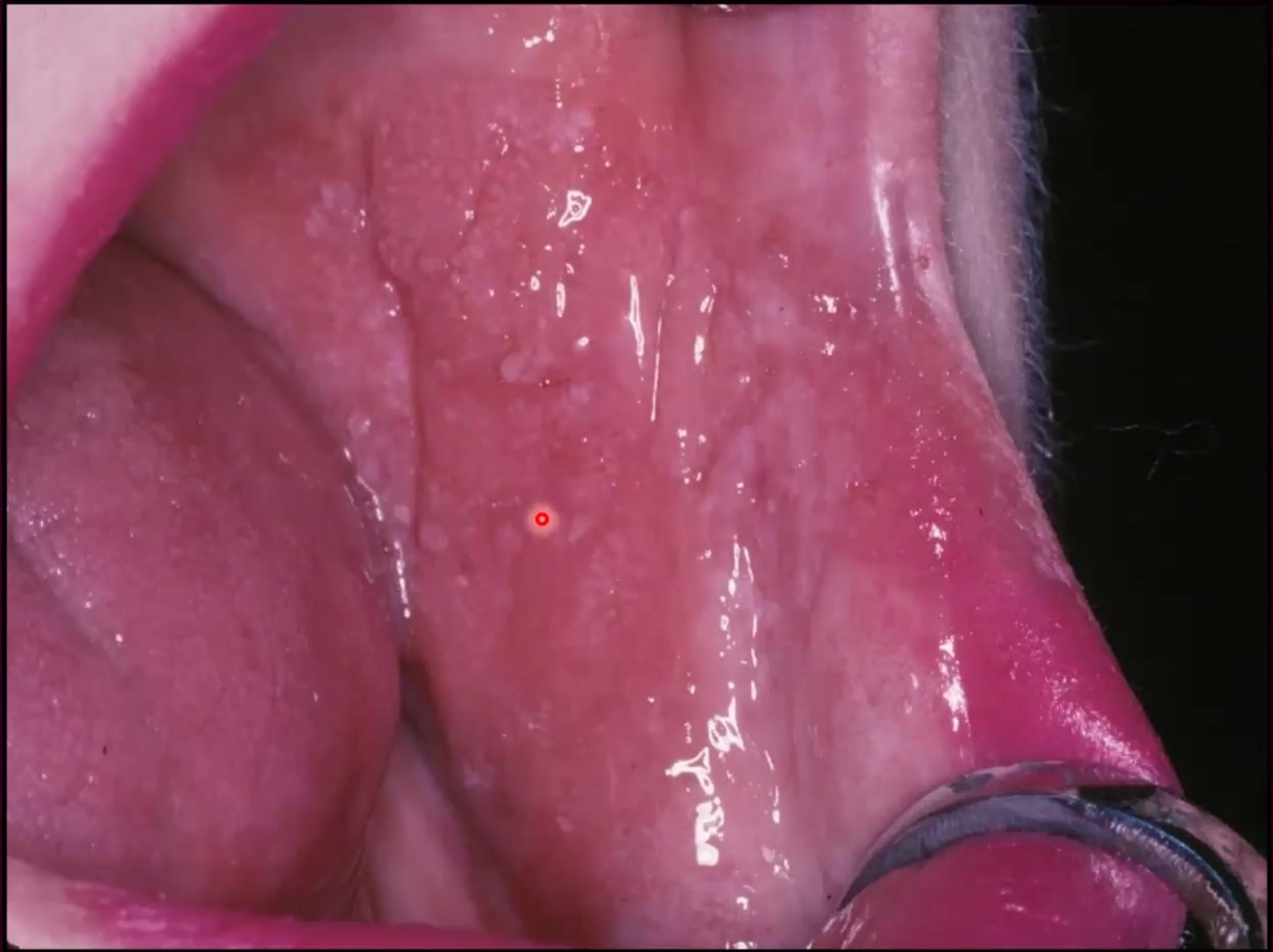


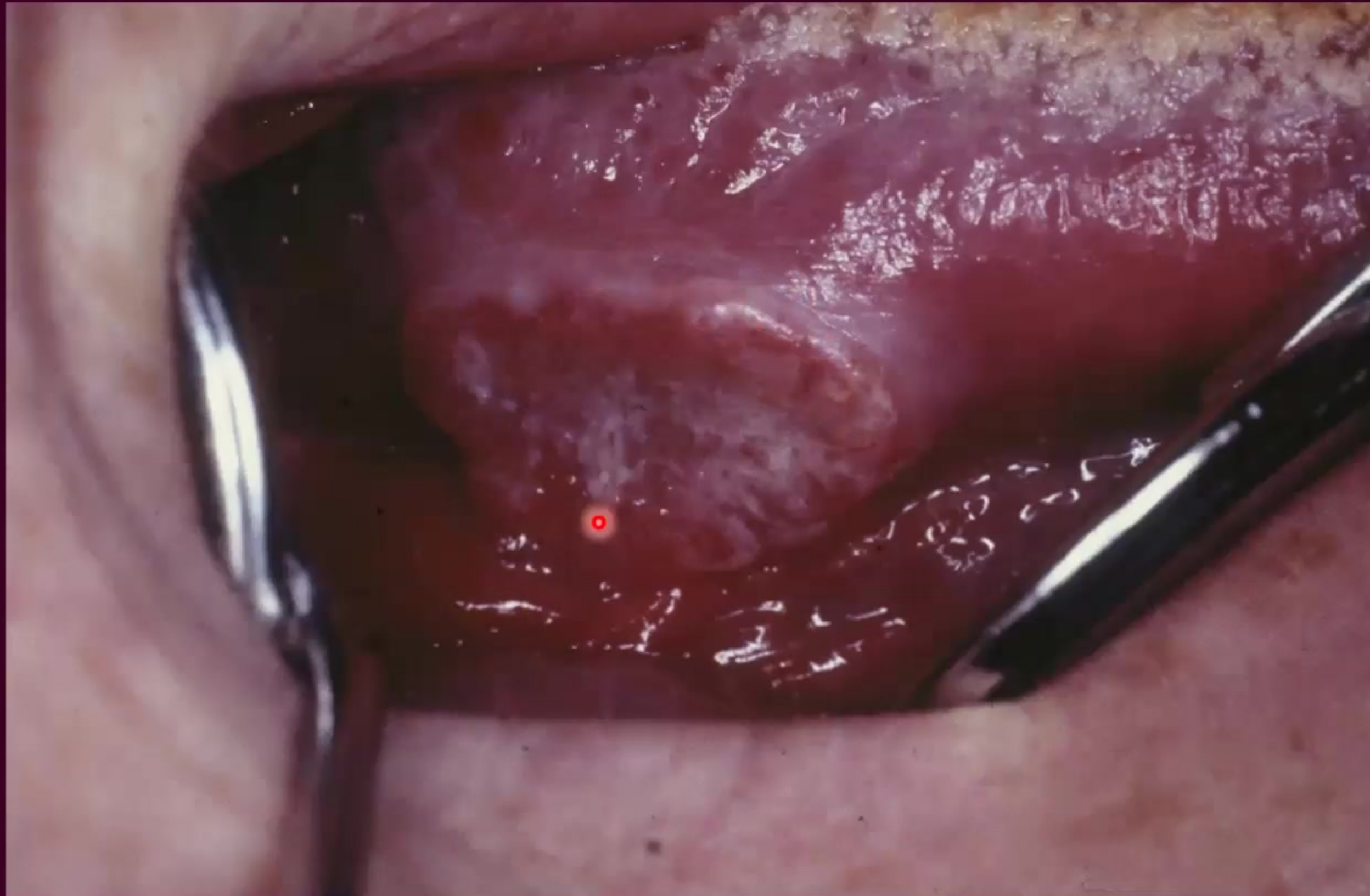
ORAL CANCER- ORO- PHARYNGEAL

- **Pain in the face and the ear.**
- **Tongue deviation.**











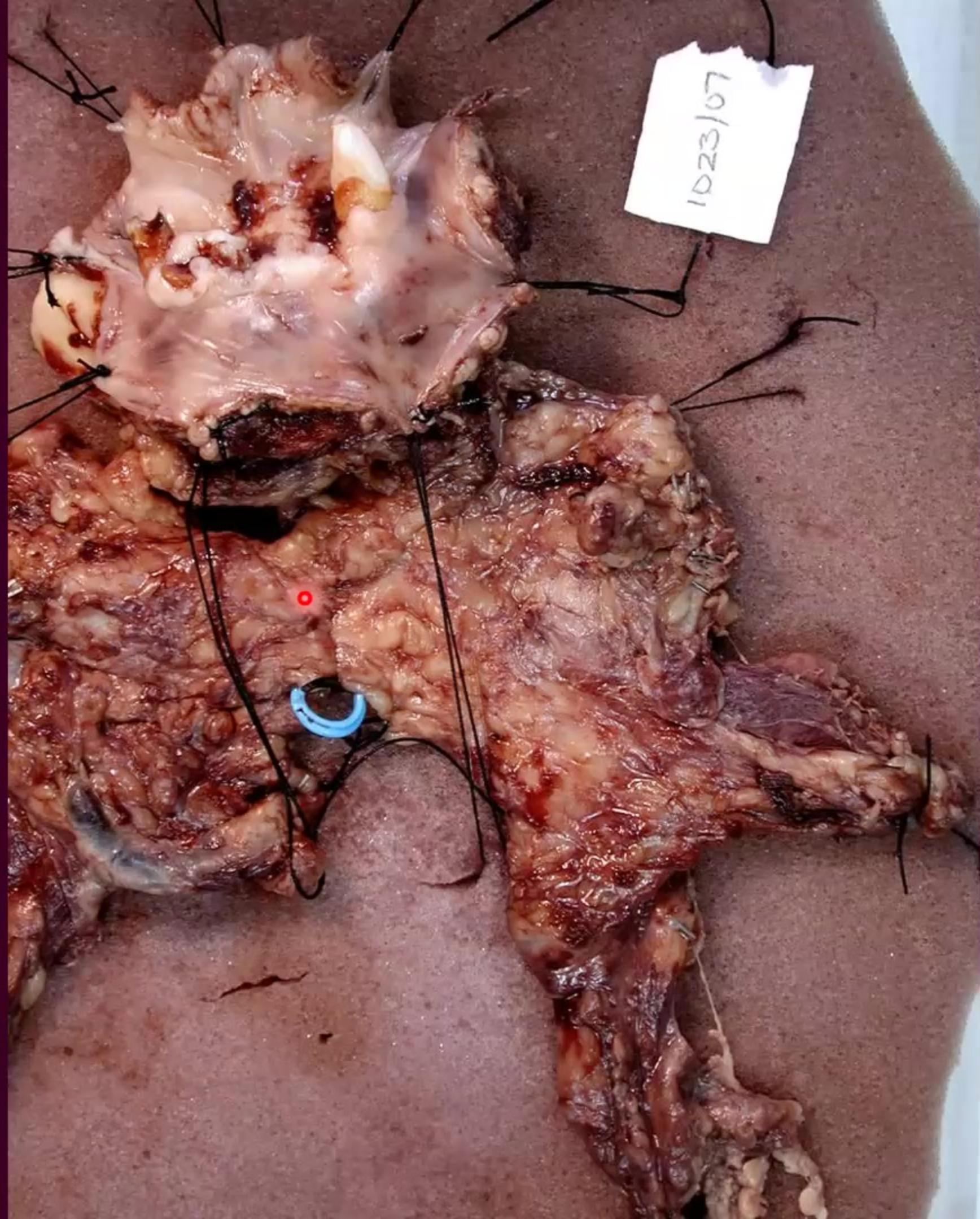


Standard clinical evaluation

- Complete history of the disease
- Alcohol and tobacco consumption
- Weight and weight loss

TNM staging

- TNM classification
- International Classification of Diseases for Oncology, 3rd Edition (ICD-O-3)



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TNM/AJCC

- Tis: Carcinoma in situ
- T1: Tumor 2 cm or less in greatest dimension
- T2: Tumor > 2 cm but ≤ 4 cm in greatest dimension
- T3: Tumor > 4 cm in greatest dimension
- T4 (lip) Tumor invades adjacent structures (through cortical bone, inferior alveolar nerve, floor of mouth, skin of face)
- T4 (oral cavity) Tumor invades adjacent structures (through cortical bone, into deep muscle of tongue, maxillary sinus, skin.)

TNM/AJCC

- N0: no regional node metastasis
- Nx: regional nodes cannot be assessed
- N1: single ipsilateral node, ≤ 3 cm
- N2a: single ipsilateral node, > 3 cm and ≤ 6 cm
- N2b: multiple ipsilateral nodes, ≤ 6 cm
- N2c: contralateral or bilateral nodes, ≤ 6 cm
- N3: node > 6 cm



TNM/AJCC

- Mx: Distant metastasis cannot be assessed
- M0: No distant metastasis
- M1: Distant metastasis

Treatment of oral carcinoma

- T1-T2 N0 :

Surgery T (only)

Brachytherapy (T<3cm, no ulceration, no infiltration, dry vermillion)

- T3-T4 N0:

Surgery T + SOH ND ± post- operative RxTh1

- T1-T4 N1:

Surgery T + SOH or radical modified ND ± post-operative RxTh1

- T1-T4 N2a-N3:

Surgery T + radical modified ND2 ± postoperative

Follow-up

- Clinical examination of head and neck mucosa (including fiberoptic) and neck palpation / performance status / nutritional assessment every 2 months (first 2 years), every 6 months (years 3-5), once a year (> 5 year)
- Dental examination and orthopantomogram every 6 months
- Chest X-ray every year
- Chest spiral CT every year
- Laboratory tests: TSH every year (if Radiotherapy delivered)

ORAL CANCER- FIVE YEAR SURVIVAL

- ❖ **Stage I > 80%**
- ❖ **Stage II ~60%**
- ❖ **Stage III ~35%**
- ❖ **Stage IV < 15%**

Second Primary Head and Neck Cancer

- Second Primary cancers of the aerodigestive pathway occur synchronously or metachronously in 20% of patients with an index of cancer of the head and neck.
- “Field cancerization”



What happens after Treatment?

- Speech and Swallowing Therapy
- Follow-up tests
- Chemoprevention
- Watch for new symptoms
- General health considerations



Prevention of Head and Neck Cancer in Primary Care Practice

1. Identify patients who use tobacco and alcohol products.
2. Counsel patients to stop using tobacco and alcohol products.
3. Maintain high index of suspicion.
4. Conduct comprehensive exams.
5. Attend to common symptoms.
6. Evaluate symptomatic patients.
7. Maintain close medical surveillance of patients in high-risk occupations.
8. Refer high-risk patients with persistent symptoms and no findings to a head and neck surgeon.

Factors Delaying the Diagnosis of Head and Neck Cancers

- Patient procrastination in seeking medical attention
- Physician delay in diagnosis
- Patient remains asymptomatic for a prolonged period



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Diagnosis and management

- Position paper from the American Academy of Oral and Maxillofacial Pathology recommends obtaining a biopsy from everyone.
- Most agree that oral lichen planus is treated
 - Symptomatically
 - With mainly topical and occasionally systemic anti-inflammatory therapy

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Lupus erythematosus

- LUPUS
 - Systemic (SLE)
 - Cutaneous
 - Acute cutaneous LE (ACLE)
 - Subacute cutaneous LE (SCLE)
 - Chronic cutaneous LE (CCLE)
 - Classic discoid LE (DLE)
- The most common form of chronic cutaneous LE
- Approximately 15-30% of SLE patients have DLE

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Lupus erythematosus

Aetiology and pathogenesis

- Autoimmune disease
 - Contribution of genetics and epigenetics
 - B and T cell signaling abnormalities
 - Dysregulated apoptosis and defective clearance of cellular debris
 - Antibody formation and perpetuation
 - Autoantibodies and organ damage

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Lupus erythematosus

Clinical features

- Begin:
 - Red purples/macules, or small plaques and rapidly develop a hyperkeratotic surface
- Evolves into:
 - Sharply demarcated discoid plaques covered by an adherent scale that extends into the orifices of dilated hair follicles




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Lupus erythematosus

Clinical features

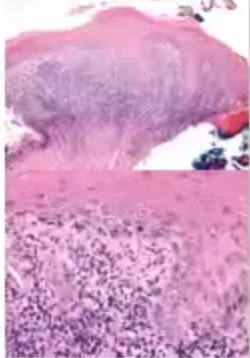
- Oral
 - Sharp margins
 - Irregular, scalloped white borders with radiating striae and telangiectasia
 - Honeycomb appearance on the palatal lesion
 - Painful ulceration is common



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Histopathologic features

- Both acanthosis and atrophy of the epithelium
- Vacuolar degeneration of basal cells
- Bandlike lymph
 - deep perivascular and perivascular lymphocytic infiltrates; eosinophilic infiltrates at the interface
- DIF
 - positive lupus band test
 - linear/juxtalinial deposition of IgG, IgM, or IgA at the basement membrane (lupus band test)



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Diagnosis and management

- Diagnosis based on clinical presentation and obtaining a biopsy
- Management
 - Unpredictable relapsing remitting course
 - Treatment with immunosuppression

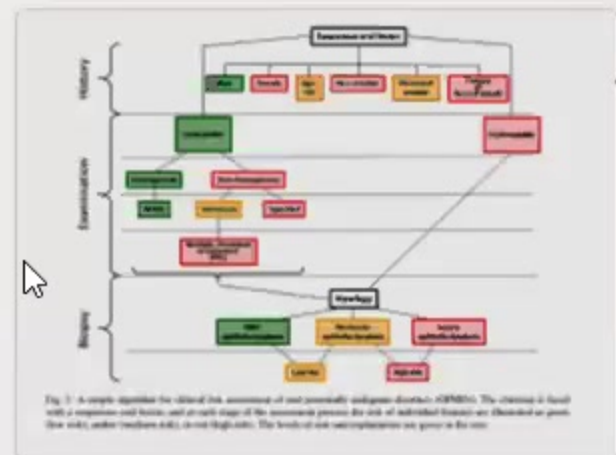
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Oral potentially malignant disorders: risk of progression to malignancy

Part 10: Speech, Swallow, Pain, Sensory, Stomatitis, Dysphagia, Oral Cancer

Oral potentially malignant disorders (OPMDs) have a variable risk of progressing to cancer. The factors that lead to a range of cancer risk are complex. It is difficult to predict the risk of progression in any individual patient with OPMD. The risk of progression to cancer is highest in those with severe dysplasia. The risk of progression to cancer is also high in those with severe dysplasia. The risk of progression to cancer is also high in those with severe dysplasia.

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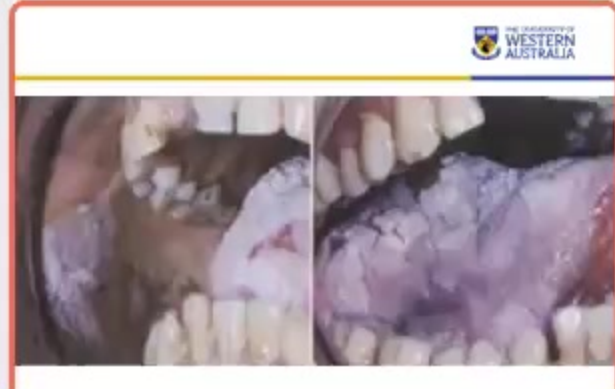


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Idiopathic white patches clinically these are "leukoplakia"

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 - Presentation: homogeneous, nodular, verrucous, speckled
 - Site: sublingual keratosis
 - Presumed aetiology: Smokers', actinic keratoses
 - Proliferative verrucous leukoplakia
- squamous cell carcinoma

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In principle every white patch in the mouth should have a biopsy sample removed

Differential diagnosis of white lesions in the mouth