

Clinical Showcase

This month's Clinical Showcase highlights common oral conditions that should be included in the differential diagnosis for squamous cell carcinoma or salivary gland tumours. The author sounds a cautionary note about the dangers of misdiagnosing oral lesions and reminds oral health professionals that they can detect oral cancer in its early stage, when treatment may be most effective. Clinical Showcase is a new section that features case demonstrations of clinical problems encountered in dental practice. If you would like to propose a case or recommend a clinician who could contribute to Clinical Showcase, contact editor-in-chief Dr. John O'Keefe at jokeefe@cda-adc.ca.

Oral Cancer

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Practising dentists should concentrate on competently diagnosing and treating routine conditions like caries, gingivitis, periodontitis, malocclusion and tooth loss. However, dentists and dental hygienists are also in the best position to detect and diagnose relatively rare and life-threatening oral lesions such as carcinoma. The dental team should therefore always maintain a high index of suspicion. The cases presented here highlight some of the key factors essential for the early detection and most effective treatment of oral cancer.

Squamous cell carcinoma (SCC) of the lower lip vermilion is most prevalent among those who spend a lot of time outdoors: farmers, fishermen, construction workers, sailors and golfers, among others. Poor tanning ability, which is often but not always associated with red or blond hair, markedly increases the risk for SCC. Lesions occur at a younger age in this higher-risk population. Smoking is also a risk factor. The patient in **Fig. 1** had been aware of “chapping and occasional bleeding” for 2 years. He was only 43, had red hair and could not tan at all, yet spent a lot of time outdoors without taking any measures to protect his lips (or skin) from ultraviolet (UV) exposure. Despite the innocent appearance of the lesion, biopsy revealed carcinoma in situ — the most advanced stage of precancer, immediately preceding invasive SCC. When a worrisome lesion becomes nodular (**Fig. 2**), the probability of cancer increases markedly. Biopsy of this lesion revealed early invasive SCC. Depending on the stage of the SCC and the condition of the lip adjacent to the lesion, treatment involves wedge excision, with or without vermilionectomy (“lip shave”). Earlier detection of lesions and appropriate referral leads to proper UV protection and prevention of cancer.

Only by routinely and systematically examining the entire oral cavity can asymptomatic lesions be detected. The patient in **Fig. 3** was referred specifically for diagnosis and treatment of a clinically important tongue lesion. A complete, systematic oral examination was performed, without which this asymptomatic SCC would have remained undetected, causing death or at least increased

morbidity. Instead, radiation therapy was curative, leaving minimal local scarring. Patients can remain completely unaware of surprisingly large intraoral lesions if the lesions are slow-growing and asymptomatic. Unfortunately, even when patients become aware of an intraoral lesion, they often erroneously assume that if it's painless, it's not dangerous. The patient with the mucoepidermoid carcinoma in **Fig. 4** had been aware of the asymptomatic swelling for 18 months. As was the case here, palatal salivary gland neoplasms can be misdiagnosed as a subperiosteal abscess. Salivary gland neoplasms (half of which are malignant), antral carcinoma and lymphoma must be included in the differential diagnosis of enlarging nodules at the hard and soft palate junction.

Epulis fissuratum is a common reaction to chronic injury from unstable dentures. The linear folds of mucosa feel rubbery because of the underlying fibrosis. Epulides tend to be asymptomatic, but can become very tender if acutely injured. Epulis fissuratum and SCC are not causally related, but a patient can have more than one unrelated oral lesion at the same time. Induration and persistent pain despite avoidance of trauma are danger signs that SCC may have developed, as was the case for the patient in **Fig. 5**. This elderly woman, with a 20 pack-year smoking history, had ignored the constant pain arising from the lesion for more than a year.

Pyogenic granuloma (PG) tends to arise when a blood clot forms after accidental injury or surgery and persists as a nodule of granulation tissue. PGs therefore tend to present suddenly as surprisingly large ulcerated nodules. The lesion in **Fig. 6** arose shortly after a large leukoplakia was excised. PG was considered to be much more likely, but the biopsy revealed that it was SCC. Cancer must be thought of in terms of probabilities and risk factors, hence the necessity of a high index of suspicion, differential diagnosis, appropriate referral to specialists and judicious use of biopsies.

Mucoceles are common submucosal nodules of the lower lip that are caused by the accidental traumatic

rupture of a minor salivary gland duct, resulting in pooling of mucus. Typically, these nodules are superficial, fluctuant and bothersome; they can reach up to 3 cm in diameter. Not every submucosal nodule of the lip is a mucocele, hence the necessity for generating a differential diagnosis. The mucoepidermoid carcinoma in Fig. 7 shared some of the clinical features of a mucocele. Though mucoceles contain mucus, so do most benign and malignant salivary gland neoplasms. Therefore, aspiration of mucus is neither diagnostic nor curative. Mucoceles are definitively diagnosed and treated by complete excisional biopsy.

Gingival SCC tends to be diagnosed very late, partly because the early clinical features are obscured by coexisting gingivitis or periodontitis. The subtle nodularity of the complex gingival lesion in Fig. 8 was the key clinical feature that ultimately led to the biopsy diagnosis of SCC. This area had been bleeding and tender for a year. The patient also had developed oral lichen planus (OLP), which classically exhibits thin white striae with or without painful erosion or ulceration; it can be seen in 1% to 2% of the general population (mostly adults). The precise relationship between OLP and SCC remains controversial. Remember that histologically typical multifocal OLP can, as in this case, be present at the same time as oral SCC. One does not exclude the other, any more than periodontitis (or epulis fissuratum) precludes oral cancer.

The vast majority of sore, highly mobile teeth represent end-stage periodontitis or periodontal abscess. It is all too easy to skip from these symptoms immediately to treatment — simple extraction. At the very least, a pre-extraction radiograph should be taken, and other possibilities considered in the differential diagnosis: primary intraosseous carcinoma, osteomyelitis, lymphoma, central hemangioma and metastatic disease. Failure to diagnose these diseases increases patient morbidity and mortality. The primary intra-alveolar carcinoma (primary intraosseous carcinoma) in Fig. 9 clinically mimicked periodontitis — pain, loose tooth and no evidence

of any surface lesions, aside from minimal swelling of the surrounding ridge. The periapical radiograph, however, showed diffuse ill-defined alveolar bone destruction typical of an infiltrative process quite distinct from periodontitis. The primary intra-alveolar carcinoma in Fig. 10 exhibited similar clinical features. Again, the ill-defined radiolucency extended too far beyond the roots to be consistent with apical abscess or periodontitis. In both cases, the treating dentists astutely recognized the radiographic warning signs and promptly referred the patients for further work-up.

In summary, for oral cancer to be treated effectively, it must be detected early. Among health care professionals, dentists and dental hygienists have the best opportunity to detect oral cancer at its earliest. It therefore behooves the dental team to maintain a high index of suspicion, routinely perform complete oral examinations, generate differential diagnoses, take and interpret necessary radiographs, make appropriate referrals and regularly upgrade their knowledge in oral medicine, oral pathology and oral radiology. ♦

Further Reading

- Neville BW, Damm DD, Allen CM, Bouquot JE. Oral & maxillofacial pathology. 2nd ed. Philadelphia: WB Saunders Co.; 2002.
- Regezi JA, Sciubba JJ, Jordan RC. Oral pathology. Clinical pathologic correlations. 4th ed. St. Louis: Saunders/Elsevier Science; 2003.
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- White SC, Pharoah MJ. Oral radiology. Principles and interpretation. 4th ed. St. Louis: Mosby - Year Book Inc.; 2000.

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The views expressed are those of the author and do not necessarily reflect the opinion or official policies of the Canadian Dental Association.



Figure 1: Persistent scaling and crusted plaques of the lower lip vermilion — actinic cheilitis.



Figure 2: Persistent fissuring with subtle nodules of the lower lip vermilion — carcinoma developing in actinic cheilitis.



Figure 3: Persistent white plaque with subtle nodules of the soft palate and uvula — carcinoma developing in leukoplakia.



Figure 4: Rubberly submucosal nodule at the junction of the hard and soft palate — mucoepidermoid carcinoma.



Figure 5: Persistent painful hard nodule within folds of rubbery tissue — carcinoma (slightly darker) and epulis fissuratum.



Figure 6: Ulcerated nodule that arose rapidly on the tip of tongue — squamous cell carcinoma.



Figure 7: Painful, firm, fixed, bluish submucosal nodule of the right lower lip — mucoepidermoid carcinoma.



Figure 8: Subtle nodules, red erosions, fine white plaques and striae of the attached gingivae — squamous cell carcinoma and oral lichen planus.



Figure 9: Ill-defined (“moth-eaten”) alveolar bone loss, not closely conforming to the root surface — primary intra-alveolar carcinoma. Radiograph courtesy of Dr. H. Carr.

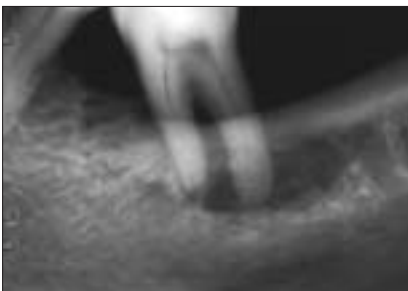


Figure 10: Ill-defined periapical radiolucency extending posteriorly, well beyond the roots — primary intra-alveolar carcinoma. Radiograph courtesy of Dr. B. Hart.