LETTERS TO THE EDITOR

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ORAL CANCER

Who best to detect oral cancer?

Sir, primary care dentistry in the UK is evolving; the role of the dental hygienists and therapists (DH-Ts) has been re-defined recently. With the introduction of direct access, DH-Ts will see patients independently and provide treatment. We noted in the paper by Brocklehurst *et al.*[1] that the performance of primary care dentists (PCDs) and DH-Ts when differentiating between mouth cancer, potentially malignant disorders and benign lesions was comparable. That said, a holistic approach is required for these diagnoses, and visual recognition of lesions is only one aspect of the diagnostic process. Two studies from Italy and Spain,[2,3] countries which have had elements of direct access for over a decade, showed deficiencies in the knowledge and training of dental hygienists (DHs) regarding risk factors for oral cancer (this study did not include hygiene therapists). In addition, 57% of DHs reported a lack of confidence in their ability to diagnose oral cancer and potentially malignant diseases.

This lack of confidence was corroborated in a UK study by Turner *et al.*[4] where the majority of participants said that they would seek a dentist's opinion of a suspicious mucosal lesion. Seeking advice is preferable to overconfidence, which could lead to mis or undiagnosed lesions but also highlights the need for further educational interventions in order to improve early detection. Brocklehurst *et al*'s study goes further and suggests that there is an equal need for improved oral cancer education and training of PCDs and DH-Ts as although both groups were comparable, there was a wide variation within each group. DH-Ts actually missed fewer mouth cancers than PCDs.

Our experience with our recently developed nurse-led review clinics has been similar. After a period of training, specialist nurses are in a position to see and examine patients that have been diagnosed with, or treated for, head and neck cancer. They have extended skills that include interventions such as nasendoscopy. When we looked at the results comparing appropriately trained specialist nurses and experienced head and neck consultants, there was no statistical difference between the two groups. This was limited to low risk clinical groups but with continuing support and training this may be applicable to all patient groups.

D. Owens, S. Wilmott and A. Kanatas, Leeds

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ORAL TUBERCULOSIS LESIONS

Sir, I read with interest the article 'Case series of extra pulmonary tuberculosis presenting as facial swelling' by E. Carter *et al.* (*Br Dent J* 2015 May 8; **218:** 519–522) about maxillofacial manifestations of tuberculosis.

Oral tuberculosis lesions, whilst uncommon, have been observed in both primary and secondary stages of the disease but have largely become a forgotten diagnosis in oral lesions. They are found in 0.05–5.00% of tuberculosis cases. Primary oral tuberculosis is more common in younger patients.[1,2]

The tissues of the oral cavity frequently reflect the condition of a person's general health and often may indicate the presence of an infectious disease, since many infection diseases occur primarily within the oral cavity.[3] Presence of atypical oral ulcerations should raise suspicion of underlying sexually transmitted infections especially in high risk group patients.[4] Dentists can play a key part in the diagnosis and management of patients and have an exceptional opportunity to become familiar with and to interpret changes in oral tissues. Health professionals must be prepared to recognise oral and maxillofacial manifestations of sexually transmitted infections and consider them in the differential diagnosis of these lesions.

A. Curto, Spain

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JAW PROBLEMS

Update on ARONJ

Sir, we wish to highlight the apparent increase in severity of osteonecrosis of the jaw (ONJ) in patients receiving the RANKL inhibitor denosumab.

Clinical evidence is mounting to support the superior efficacy of denosumab compared to bisphosphonates (BPs) for the management of metabolic and metastatic diseases of the bone. Current evidence supports the increasing use of RANKL inhibitors, as well as a low but clinically significant risk of ARONJ, particularly in the oncology setting.[1-3]

At Sheffield Teaching Hospitals' Trust we have identified several new cases of ARONJ, which have presented over a short period of time; all of these patients have received denosumab therapy. All cases appear to have a more aggressive mode of ONJ compared to that seen with IV and/or oral BPs so far. Progression of the disease occurred considerably faster with the development of widespread suppuration and teeth mobility within

weeks. One case spread rapidly across the midline of the mandible following extraction of a lower first molar, resulting in widespread bony necrosis and associated osteomyelitis. Our recent clinical experience does not support the current clinical understanding that denosumab appears to share similar oral adverse events to BPs.

By comparison the estimated risk of developing ONJ in two clinical trials between denosumab and zoledronic acid was 1.40, ie, a 40% excess risk in the denosumab group compared with IV BPs.[1] The lack of reporting of drug adverse effects and the significant lack of case report data published in oral and maxillofacial surgery literature makes it extremely difficult to ascertain the magnitude of ARONJ associated cases to date.

We want to emphasise the importance of oral health assessment and education of patients and healthcare providers in understanding the risks of ARONJ development.[4] Similarly, providers who prescribe denosumab to patients should refer them for a dental assessment to evaluate for risk of ARONJ and to initiate prophylactic dental treatment before the initiation of therapy.[2,3] Based on the mechanism of denosumab, resulting in a half-life of 6 months, it may be possible to place patients on an effective drug holiday before surgical interventions to promote bony healing, in contrast to failed BP drug holidays.[2] A diagnostic tool to assess the clinical risk may help to guide this decision as current guidelines exist specifically related to RANKL inhibitors are poor. Good communication between the dentist, general practitioner, oncologist, haematologist and metabolic bone physicians involved in the management of these patients is paramount. In the near future we predict an increased number of such cases and, therefore, optimal risk reduction strategies should be employed to prevent and best manage such cases.

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ULCERATION

More on aphthous ulceration

Sir, we were interested to read the letter on the new agent, apremilast, that has proved to be effective in treating idiopathic oral ulcers, the main manifestation of Behcet's syndrome.[1] Acting as a selective inhibitor of phosphodiesterase 4, apremilast may partially supress the production of pro-inflammatory cytokines, stimulating the production of the anti-inflammatory cytokine IL-10.[2] However, the full mechanism of action is not clear and the bioreceptor for this compound has not been identified. A preliminary, phase 2 study[3] (NCT00866359) was not designed to assess a long-term efficacy of apremilast and unfortunately, until now, this drug is not formally approved for the treatment of Behcet's disease. Currently pending, a phase-3 randomised, placebo-controlled study aims to evaluate the efficacy and safety of apremilast in about 200 patients with active Behcet's disease.[4] Apremilast is a novel analogue of thalidomide and pregnancy should be excluded before pharmacological treatment is considered to be initiated.[5]

Cimetidine, a well known drug used for the treatment of stomach ulcers as an H_2 inhibitor, can be effective as a therapeutic agent for selected forms of aphthous stomatitis.[6] When used regularly it may prevent future episodes aphthous ulcerations associated with PFAPA syndrome[7] and has immunomodulatory effects that include blocking suppressor T-cells and facilitating cell-mediated immunity.[8]

As aphthous ulcers may result from the patient's immune system reacting against the mucosal epithelium, the more common systemic use of immunomodulatory drugs would bring clinical benefits, reducing recurrence and symptoms of oral ulcers.

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SUN BED DANGERS

Ain't no sunshine

Sir, sun beds predominantly emit UVA radiation that is thought to be the least damaging of the UV radiation spectrum. However, there is growing evidence that the latest sun beds marketed produce higher levels of UVB to imitate the solar spectrum and speed the tanning process.[1]

Some 13,200 cases of malignant melanoma and 2 million cases of skin cancers occur worldwide each year and the Food and Drug Administration (United States) reclassified UV tanning devices from class I (low to moderate risk) to class II (moderate to high risk) devices in September 2014.[2] Scientific literature shows the mortality rate from skin cancer due to tanning is greater than the mortality rate from lung cancer due to smoking.[2] The individual risk of developing squamous cell carcinoma (SCC) and basal cell carcinoma (BCC) increases by 67% and 29% respectively by using one indoor tanning session. Approximately 25% of early-onset BCC could be avoided if individuals have never tanned indoors.[3] The lower lip is approximately 12 times more commonly affected as compared to the upper lip, owing to its exposure to UV radiation.

These facts suggest that the general public may be vulnerable to deadly cancers through these sun beds. Although regulations already exist regarding the use of sun beds, public awareness is lacking which could be due to a lack of pictorial and statutory warnings highlighting their harmful effects. Awareness and warnings explaining the risks involved may solve the problems associated with sun beds.

S. Arora, Malaysia

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