Letters to the Editor

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LETTERS

GOLDEN-CRUSTED RASH

Sir, antiresorptive agent-induced osteonecrosis of the jaws (ARONJ) has been published extensively in this journal. We treat this condition frequently. Until now, we have not seen ARONJ precipitated by herpes zoster infection.

Herpes zoster infection is caused by reactivation of varicella zoster virus following a period of dormancy in the dorsal root ganglia. It affects the trigeminal nerve in 13% of cases.¹ The most commonly involved branch is the frontal branch of the ophthalmic division.

A 55-year-old patient was referred to our department by his GP. The patient reported having a painful facial rash for two weeks. Antibiotics and aciclovir were prescribed to no effect. He had previously taken oral alendronic acid for osteoporosis prophylaxis. This ended two months previously lasting 17 months in total. The patient was in good dental health with no history of serious periodontal disease.

On examination there was a golden-crusted facial rash in the distribution of the left maxillary division of the trigeminal nerve. The buccal mucosa and maxillary gingivae were oedematous. Herpes zoster with secondary bacterial infection was diagnosed.

The patient was treated with intravenous flucloxacillin 500 mg and benzyl penicillin 1.2 g. Aciclovir therapy was stopped as the infection had been clinically apparent for over two weeks.

The patient was discharged after five days with oral antibiotics.

One month later, the patient reported he had some loose teeth and a central incisor had fallen out. On examination the attached gingiva of the left maxilla had sloughed off. This extended from the

Fig. 1 The patient's underlying bone was exposed and showed signs of osteitis. The remaining mucosa was inflamed

left central incisor to the first premolar. The underlying bone was exposed and showed signs of osteitis. The remaining mucosa was inflamed (Fig. 1). The lateral incisor, canine and first premolar were mobile. The affected area was confined to the distribution of the left maxillary division of the trigeminal nerve.

The patient was prescribed chlorhexidine mouthwash and given oral hygiene instruction.

Later, a non-eugenol periodontal dressing was applied to the exposed alveolar bone. Primarily this was aimed at reducing the discomfort associated with sharp edges of bone but it was also hoped this would limit local trauma and optimise the health of the adjacent mucosa. The patient reported this dressing did indeed reduce pain and on review the visible inflammation of surrounding tissues had reduced significantly.

Debridement of necrotic bone and extraction of affected teeth was carried out under general anaesthetic two months later. A buccal advancement flap was positioned to achieve mucosal coverage. The affected skin has developed hypertrophic scarring (Fig. 2). The patient is undergoing assessment for a temporary partial denture.

Fig. 2 Hypertrophic scarring of the affected skin

Debridement of necrotic bone was limited to what had already naturally sequestered. A mucosal flap permitted total wound coverage. This would be in keeping with the protocol presented by Markose *et al.*²

The use of a periodontal dressing was effective in reducing discomfort, prevented food packing and limited trauma to the adjacent soft tissues.

Herpes zoster infection of the maxillary division of the trigeminal nerve can result in localised osteonecrosis, tooth loss and a challenging dental restorative problem.

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2. Markose G, Mackenzie F, Currie W J R, Hislop W S. Bisphosphonate induced osteonecrosis - a protocol for management. *Br J Oral Maxillofac Surg* 2009; **47**: 294-297.

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