Case history

A 70 year female patient was referred by her physician to the dentist office. The patient, Mrs Bunning had complained of pain in her mouth near an 'open wound' that had not healed for several months. As Mrs Bunning's health had been her primary concern, she had not been to the dentist for over 2 years.

Medical history

The patient is currently under treatment for osteoporosis and rheumatoid arthritis which was diagnosed 3 years ago. Mrs Bunning had been prescribed oral bisphosphonates alendronate (Fosamax) to improve the bone density because of the osteoporosis and corticosteroids for the management of her rheumatoid arthritis.

Dental history

Mrs Bunning was edentulous on the maxilla because of poor oral hygiene and carious lesions, in addition to her mandibular molars. To aid with mastication the patient was fitted with a dental prosthesis on the maxillary arch 5 years ago.

Clinical situation/mouth inspection

The intra-oral examination revealed an erythematic ulceration with jagged edges approximately 8 mm in diameter surrounding exposed yellow, necrotic appearing bone on the palatal with evidence of purulent discharge. The lesion appeared to be caused by an ill fitting maxillary denture which irritated the palatal tori.

Generalized moderate accumulations of biofilm were apparent on facial and interproximal surfaces of the mandibular teeth and the upper denture. Moderate gingival inflammation was present because of the accumulation of biofilm. Periodontal depths ranged from 5–7 mm on the remaining mandibular teeth with no bleeding upon probing. Class 2 mobility was present on the mandibular right second premolar.

Radiographic image

Panoramic and periapical radiographs revealed no significant bone involvement or demineralization of the jaws. Widening of the periodontal ligament space was evident on the existing premolars. A computerized tomography scan was conducted for treatment planning. Interproximal decay was detected on the mesial surface of the left canine and distal surface of the right lateral.

Questions

1. What is the primary oral diagnosis of this patient?
2. Does the patient's prescribed medication contribute to her oral health status?
3. Discuss one possible treatment option for this patient.
4. What is the role of the dental hygienist and the dental team in the treatment of this patient?

Answers/rationale

1. The concern in dentistry is the development of osteochemonecrosis following intraoral trauma, such as surgery or simple denture irritation, in a susceptible patient (1). Patients may be considered to have bisphosphonates-related osteonecrosis of the jaw (BRONJ) if all of the following three characteristics are present (a) current or previous treatment with a bisphosphonates (b) exposed, necrotic bone in the maxillofacial region that has persisted for more than 8 weeks; (c) no history of radiation therapy to the jaws (2).
2. With post-menosausal osteoporosis as an indication for bisphosphonates use, a large percentage of the female population may be also at risk for developing bisphosphonates and jaws necrosis (2). Bisphosphonates associated osteonecrosis of the jaws, has also been referred to as bisphosphonates osteonecrosis (BON) and osteonecrosis of the jaw (3). In patients with osteoporosis, it is expected that bisphosphonates will arrest bone loss and increases bone density, decreasing the risk of pathologic fracture resulting from progressive bone loss (4). Bisphosphonates bind to bone and incorporate in the osseous matrix. During bone modeling, the drug is taken up by osteoclasts and internalized in the cell cytoplasm, where it inhibits osteoclastic function and induces apoptotic cell death (5). These properties may affect the local bone blood supply, contributing the apparent ischemic change noted in the affected patient's jawbone, or may operate in concert with the metabolic change mediated by osteoclast suppression to produce local jawbone necrosis (2). This action may well explain the great reduction on vascularity of bone as well as tumouricidal effects. The common uses of these drugs include the management of Paget's disease of bone,

hyperclcaemia of malignancy, multiple mycloma, metastatic breast, lung and prostate cancer (3).

With the physiologic function such as constant stress from mastication microfractures can occur which do not repair because of the systemic bisphosphonates which can result in oral osteonecrosis. The osteonecrosis is a complex interplay of bone metabolism, local trauma, and increased demand for bone repair, infection and hypovascularity (6).

Co-morbidities have included poorly controlled diabetes and other immuno-compromised states, concurrent use of coticosteroids, chemotherapeutic drugs, advanced age, alcohol abuse and smoking (7).

3. The objective of the treatment is to improve the quality of life of the patient by alleviating pain and treating the condition to return the patient to a state of oral health. Referral to an Oral and Maxillofacial surgeon for curettage of the necrotic bone and soft tissue closure is recommended (1) as the necrotic area acts as a portal of entry for bacteria (6). The site should be monitored every 2–3 weeks until the site is healed (1). A microbiological culture is obtained to guide the appropriate antibiotic regimen. The microbes most commonly found in BRONJ have responded to the penicillin group of antibiotic (2). Although penicillin is the first-choice antibiotic in dentistry, amoxicillin and/or clindamycin provide better bone penetration and a wider spectrum of coverage (6). Systemic antibiotic premediation should be prescribed immediately prior to debridement of the osteochemonecrosis site and followed for 10–14 days post-debridement (1). Anti-microbial mouth rinses such as 0.2% chlorhexidine guconate is also recommended for a minimum of twice daily (1) to reduce the bacterial load (6).

Discontinuing the oral administration of bisphosphonates after prolonged use is not supported by the evidence to commence dental treatment, as bisphosphonates is retained in the mineralized bone matrix within the jaw at selective levels (1, 6). Bisphosphonates nitrogen preparations are more potent and are retained longer in the bone because of the complex pathway of action which results in loss of adherence of osteoclasts to the surface of the bone (8). However, the other effect of bisphosphonates, such as the antiangiogencis activity, may be reduced when bisphosphonates are discontinued, and this may help healing the overlying mucosa after dental treatment (6).

Prosthodontic appliances should be evaluated and adjusted as needed to minimize soft-tissue trauma and pressure points and when possible a soft liner can be applied for fit and stability (1, 6).

4. The best approach to treating patients with BRONJ is prevention. Collaborative relationships are to be established between the patient, their physician and dentist prior to and during the administration of bisphosphonates. Preventive measures include a comprehensive dental examination, especially prior to the patient commencing bisphosphonates therapy. Restorative and oral surgery needs are to be addressed prior to the start of bisphosphonates therapy. A strict maintenance programme is developed with the patient for continued oral hygiene reinforcement and education on the oral risks and periodontal maintenance with atraumatic scaling, root planning and monitoring (1). Patients who have been given oral bisphosphonates within the last 3 months also should undergo a dental evaluation. Dental therapy can be provided to these patients before the risk of developing BON increases (6).

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