An 83-year-old woman presented with a painless, discharging, swollen nodule on the right side of the jaw of 6 months’ duration. She had a history of osteoporosis diagnosed 3 years prior for which she was taking alendronate and cholecalciferol. Bone mineral density test scores were -3.93 (spine) and -2.81 (hip) (reference range, -2 and above). She also had hypertension that was treated with amlodipine. On examination there was fetor oris and a discharging sinus with purulent discharge at the jaw. The lower jaw was edentulous. A 5-mm area of red beefy granulation tissue was attached to underlying bone.

An exposed sequestrum was seen intraorally with a 3-cm opening at the mandible. There also was submandibular lymphadenopathy.

What’s the diagnosis?

a. basal cell carcinoma
b. dental sinus secondary to osteonecrosis of the jaw
c. furuncle
d. pyogenic granuloma
e. squamous cell carcinoma

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The Diagnosis: Dental Sinus Secondary to Osteonecrosis of the Jaw

Cone beam computed tomography revealed an area of lucency measuring 40×20 mm in the body of the right mandible (Figure). The patient subsequently underwent curettage of the wound with sequestrectomy of the involved area.

Osteonecrosis of the jaw is a form of avascular necrosis. It is an uncommon but potentially serious side effect of bisphosphonate use.1 Bisphosphonates commonly are used as first-line therapy for osteoporosis, with proven efficacy to reduce fracture risk by exerting an antiresorptive effect on bones.2 Bisphosphonate-related osteonecrosis of the jaw (BRONJ) is defined by the American Association of Oral and Maxillofacial Surgeons as the presence of exposed necrotic bone in the maxillofacial region that has persisted for more than 8 weeks, with current or prior treatment with a bisphosphonate and absence of prior radiation therapy to the jaw.1 Bisphosphonate-related osteonecrosis of the jaw can be associated with infections, pathologic fractures, extraoral fistulae, or osteolysis extending to the inferior border.

Our patient had a dental sinus that resulted from the underlying BRONJ. The jawbones, unlike the long bones, are in a special environment in that both acute and chronic infections occur often within the bone, and surgical procedures as well as masticatory trauma expose the bone to a bacteria-laden environment.4 Infection around the root apex of a tooth results in a dental abscess and a sinus tract can develop from the abscess, draining either intraorally or extraorally.5 Facial sinus tracts can be either odontogenic or nonodontogenic, and sometimes the lesions of dental origin may be confused with dermatological lesions.

Bisphosphonates inhibit osteoclasts, which are responsible for bone resorption. Antiangiogenetic effects also have been reported in bisphosphonates, resulting in devitalized bone.6 The potent and prolonged inhibition of bone remodeling likely plays an important role in BRONJ. The more frequently occurring microdamage inflicted on the lower jawbone with mastication also may represent a contributory factor.7

Bisphosphonate-related osteonecrosis of the jaw more often is associated with the use of high-dose intravenous (IV) bisphosphonate in cancer-related hypercalcemia and less so with oral bisphosphonates, which are generally used to treat osteoporosis.3 In a Swedish study conducted from 2003 to 2010, 55 cases of BRONJ were documented in a population of 1.2 million individuals. The prevalence of BRONJ in patients on oral bisphosphonates and IV bisphosphonates was estimated to be 0.024% and 2.8%, respectively.8

Bisphosphonates are widely used worldwide as the main treatment of osteoporosis. The association between osteonecrosis of the jaw and oral bisphosphonates is contentious among the osteoporosis population, as most studies focus on IV bisphosphonate use in cancer patients.9 Bisphosphonate-related osteonecrosis of the jaw adversely affects the patient’s quality of life, producing notable morbidity in afflicted patients. Thus, a complete dental assessment and treatment is recommended before the initiation of bisphosphonate treatment. The risk for developing BRONJ associated with oral bisphosphonates increases when the duration of therapy exceeds 3 years.3 It has been reported that antifracture efficacy would persist for 1 to 2 years following discontinuation of alendronate or risedronate that had
been taken for 3 to 5 years, but patients with low bone mineral density at the femoral neck (T-score below –2.5) after 3 to 5 years of treatment of bisphosphonates are at the highest risk for vertebral fractures and therefore appear to benefit most from continuation of therapy.10 For dental procedures, the American Association of Oral and Maxillofacial Surgeons suggests that if systemic conditions persist, the clinician might consider discontinuation of oral bisphosphonates for a 3-month period before and after elective invasive dental surgery to lower the risk for BRONJ.3 When possible, invasive dentoalveolar procedures such as extractions should be avoided; conservative endodontic treatment is preferable.

Bisphosphonate-related osteonecrosis of the jaw is a devastating condition that is difficult to treat and manage, thus the focus should be on prevention through dental clearance prior to starting bisphosphonates. It also is crucial to have a high index of suspicion for BRONJ in patients presenting with orofacial lesions so that they can be treated expediently.

REFERENCES