Osteonecrosis of the jaw (ONJ) was first associated with antiresorptive therapy (preventing or slowing the destruction of bone using bisphosphonate drugs, such as Fosamax, Actonel, Boniva and zoledronic acid) in 2003. At that time, dentists and oral surgeons at a dental clinic specializing in the treatment of cancer patients noted an increased incidence of ONJ in their patients who were receiving high doses of bisphosphonates and subsequently had dental surgery such as tooth extraction or implantation. High doses of antiresorptive drugs (such as bisphosphonates and denosumab/Prolia) are also used to treat certain cancers.

It is worth noting that some patients who did not have recent dental surgery also developed ONJ. ONJ is defined as exposed necrotic jaw bone that has still not healed eight weeks after dental surgery in patients who have received potent antiresorptive drugs and have not also undergone radiation therapy of the jaw. Cancer patients may receive doses of antiresorptive therapy that are 10-12 times higher than the doses used to treat osteoporosis. Still, some patients receiving traditional osteoporosis treatment doses of these medications have experienced ONJ.

The risk of ONJ in the general population has been reported at less than .001% and taking osteoporosis medications only marginally raises the risk to between .001 and .01%. Evidence does suggest some association between the risk of ONJ in patients on long-term bisphosphonate and denosumab (Prolia) therapy. The highest risk group for ONJ is cancer patients on bisphosphonates with rises to 1 to 15%.

ONJ often is without symptoms initially but symptoms may appear weeks to months later due to local inflammation. Symptoms may include jaw pain, loose teeth, jaw bone enlargement, red gums and gum ulcers. ONJ may occur at the site of a recent dental surgery and occurs more frequently in the lower versus the upper jaw.

In addition to the association of ONJ with chronic bisphosphonate and denosumab use in the treatment of osteoporosis, other risk factors include smoking, poor oral hygiene, diabetes, steroids, chemotherapy and dental surgery. Fortunately, the course of ONJ in most patients is limited, with more than 90% of patients responding to conservative management using antibiotics and oral rinses. In rare cases, limited surgical debridement of oral tissue is necessary.
Prevention of ONJ in osteoporosis patients on antiresorptive treatments involves regular prophylactic dental care and avoidance of invasive dental procedures, if possible. Even proper fitting of dentures is important. Routine dental care, such as cleaning, cavity remediation, crowns, whitening and even root canal surgery, do not appear to increase the risk.

The cause of ONJ is not understood. Some theories include over-suppression of bone production, dental infection, inhibition of new blood vessel growth, soft tissue injury and compromised immunity. ONJ has been seen in a variety of cancers as well as following head and neck radiation therapy. While there appears to be an association between antiresorptive drugs and ONJ, the evidence is unclear.

If you develop ONJ while on antiresorptive therapy, a number of clinical decisions need to be addressed. Patients with metastatic cancer to their bones may not be able to stop their antiresorptive therapy safely. Osteoporosis patients may be able to stop their antiresorptive treatment and/or substitute bone active therapy with Forteo (teriparatide), which has not been associated with ONJ. These clinical decisions need to be made with your physician.

Osteoporosis patients on medication who are facing elective dental procedures (extraction and implantation) should discuss options with their physician and their dentist. Antiresorptive therapy may be stopped two to three months prior to dental surgery and restarted two to three months following the dental procedures, provided mouth tissues have healed completely. However, this will depend on the medication the patient is taking, the severity of their osteoporosis, the risk of fracture and the urgency of the dental problems. There is no evidence that stopping osteoporosis medication prior to dental surgery reduces the risk of ONJ even though this is a common practice. Some dentists recommend bone resorption marker measurement to help determine the risk of ONJ in advance of dental surgery. Although theoretically an attractive concept, the effectiveness of this strategy is unknown.

How long should patients on bone-active osteoporosis medications continue their medications? Most of the benefit from long-term bisphosphonate use is realized in the first five years of oral treatment and the first three years of IV therapy (with zoledronic acid). Patients at lower risk of osteoporotic fracture (those with no previous fractures) might be able to take a drug holiday of two years with reassessment of fracture risk and bone density. Patients at high fracture risk (those who’ve had previous and/or multiple fractures) probably should continue with medication and be reassessed every one to two years. It is very important that each patient is assessed independently, with treatment and follow-up tailored to that unique patient.