OSTEORADIONECROSIS AND ITS MANAGEMENT

INTRODUCTION

Definition

Osteoradionecrosis is a severe debilitating complication that results from radiotherapy to the jawbones. It is a non-healing, non-septic lesion of bone in which bone volume and density cannot be maintained by the hypovascular, hypoxic tissue, which cannot adequately meet its metabolic demands. There is a reduction in the osteocyte population and the vascularity of the bone. Such changes make the bone vulnerable to trauma and infection and impair its capacity to remodel and repair.

Radiation injury to the tissue produces a three-fold problem. First, the small blood vessels undergo endothelial death, hyalinization, and ultimately, thrombosis. Second, fibrosis occurs in the periosteum and overlying mucosa and third, radiation causes the destruction of osteocytes, osteoblasts, and fibroblasts. The combination of these factors produces what Marx calls the “three H principles of irradiated tissue” tissue that is hypovascular, hypoxic, and hypoxic.

Predisposing factors

The most common dental factors precipitating necrosis are post-radiation extractions, pre-existing periodontal disease and occasionally, pre-radiation extractions if they are poorly planned. Ill fitting dentures causing trauma to the oral mucosa and underlying bone may also contribute. Consequently, the patient’s dental state as well as the tumour site and radiation will determine susceptibility to ORN.

Although the introduction of mega-voltage radiotherapy has lead to a fail in the incidence of necrosis of the mandible (5% of long-term survivors), its consequences are extremely serious for the patient. Similarly, dental surgery can increase the risk of bone necrosis by compromising circulation to the overlying mucosa and periosteum. Tooth extractions disrupt relatively large blood vessels entering the bone form the periodontal ligament space, leaving adjacent bone relying on collateral circulation from smaller vessels that are more prone to be affected by radiation.

The effects of radiation depend on the intensity of the radiation, the age of the patient, the time intervals between treatments of the tissue being irradiated and the susceptibility of the individual patient. The mandible and the maxilla are particularly prone to these effects because of their high mineral content, with the associated increase in backscatter irradiation. The mandible is more vulnerable because of its denser structure and poorer blood supply.

incidence

A study by Morrish found that the total dosage of radiation received by the patient seemed to be a critical factor, with no reported cases using less than 6500 RADS (6000cGy) and a significant increase noted above 7500 RADS (7500cGy). They found that 85% dentulous and 50% edentulous patients developed ORN. Patients who were edentulous at the time of cancer diagnosis were at low risk for ORN, whereas dentulous patients showed an increased risk. The greater incidence of dentulous patients was associated with extractions occurring after radiotherapy. Dentulous patients with pre-radiotherapy extraction or no extraction status post XRT had a risk factor similar to that of the edentulous patients. The mandible was found to have a greater frequency of ORN cases when compared with the maxilla, presumably due to the less vascular nature of the mandible.

Articles published since 1970 suggest an incidence of ORN that ranges from 5 to 22%. The use of megavoltage radiation such as the cobalt-60 and the linear accelerators may result in a lower incidence since this type of radiation is supposed to spare the bone. The risk of developing necrosis is greatest in the first six months following treatment. However, the risk of developing ORN persists for years after radiation therapy since the reduction in healing capacity may be permanent.