CASE REPORT

Osteoradionecrosis of the Anterior Cranium

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ABSTRACT—Osteoradionecrosis occurs in approximately 10% to 15% of patients following radiation therapy for head and neck cancer.[1] In these patients, it is most commonly reported in sites involving the mandible, but it has also been reported in the maxilla, sphenoid, and temporal bones. The majority of these cases are related to some type of trauma such as dental extraction or intraoral biopsies.[2] However, approximately 40% of these entities occur spontaneously and are felt to be secondary to cell kill in intermediate tissues such as bone and periosteum.[1] Our literature review yielded no previously reported cases of osteoradionecrosis involving the anterior cranium. The following two cases present patients who experienced osteoradionecrosis of their frontal bone flaps following subcranial approaches for tumor resection. Both patients suffered from carcinomas involving the ethmoid sinuses; one tumor was a moderately well-differentiated squamous cell carcinoma, the other a mucinous adenocarcinoma. One patient's radiation therapy consisted of external beam photons; the other patient received external beam neutrons. Treatment for these patients, as well as possible causative factors regarding their osteoradionecrosis, are discussed. (*Skull Base Surgery, 6(4):259–266, 1996*)

Osteoradionecrosis occurs in approximately 10% to 15% of patients following radiation therapy for head and neck cancer.[1] It is most commonly reported in sites involving the mandible, but it has also been reported in the maxilla, sphenoid, and temporal bones. The majority of these cases are related to some type of trauma such as dental extraction or intraoral biopsies.[2] However, approximately 40% of these entities occur spontaneously and are felt to be secondary to cell kill in intermediate tissues such as bone and periosteum.[1] Our literature review yielded no previously reported cases of osteoradionecrosis involving the anterior cranium. The following two cases present patients who experienced osteoradionecrosis of their fronto-naso-orbital bone flaps following subcranial approaches for tumor resection and postoperative radiation therapy. The subcranial approach, as popularized by Raveh et al,[3] has been utilized for traumatic reconstruction, as well as for resection of malignant and benign tumors of the anterior skull base. Both patients suffered from carcinomas involving the ethmoid sinuses; one being a moderately well-differentiated squamous cell carcinoma, the other a mucinous adenocarcinoma. Radiation therapy for one patient consisted of external beam photons; the other patient received external beam neutrons. Treatment for these patients, as well as possible causative factors regarding their osteoradionecrosis, are discussed.

CASE REPORTS

Case 1

A 66-year-old African-American female presented with a 3-week history of frontal headaches accompanied by pain and increased swelling. She complained of tenderness over the frontal bone and nasal ridge with episodes of purulent nasal discharge and congestion approximately three times per year. She had a prior history of allergic rhinitis and occasional periorbital swelling. Her social history was significant: She smoked two packs of cigarettes per week. She was on acetominophen, calcium replacement, and conjugated estrogens (Premarin). She

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had no known drug allergies. Her past medical history was significant for angina, hypertension, bundle branch block, chronic obstructive pulmonary disease, and alcohol abuse.

Her physical examination at the time of admission revealed tenderness over the frontal and maxillary sinuses, without evidence of visual changes or periorbital cellulitis. A screening x-ray in the clinic revealed an air-fluid level bilaterally in the frontal sinuses. She was therefore admitted for intravenous antibiotics and further work-up. A computerized axial tomography scan and a magnetic resonance imaging study of the sinuses was obtained which revealed acute frontal sinusitis and a calcified tumor in the frontal and ethmoid sinuses with evidence of erosion and possible involvement of the anterior cranial fossa (Fig. 1).

In the operating room, a biopsy of the sinus mass was performed. The tissue was felt to be most consistent with a neuroendocrine tumor. Shortly thereafter, a definitive resection was performed. The subcranial approach was used for resection of the entire tumor, including the superior nasal septum. This included both a bifrontal craniotomy and fronto-naso-orbital segment removal prior to the tumor resection. Reconstruction was accomplished with split calvarial bone graft. The postoperative course was complicated by a left frontal lobe infarct and a significant amount of pneumocephaly (Fig. 2). She had no neurologic changes, and was discharged approximately 11 days following resection of the tumor. The final pathology report was consistent with a moderately well-differentiated, squamous cell carcinoma of the septum, ethmoids, and frontal sinus.

The patient subsequently underwent postoperative radiation therapy to maximize local control. A dose of 4600 centiGray was delivered utilizing a single fraction of 6 MV photons prescribed to a depth of 1.5 cm, with the remainder of the prescribed dose delivered with 16 MeV electrons prescribed to the 80% isodose line (5.5 cm). She was treated from 14 May 1993 to 16 June 1993. Following completion of radiation therapy, the patient was noted to

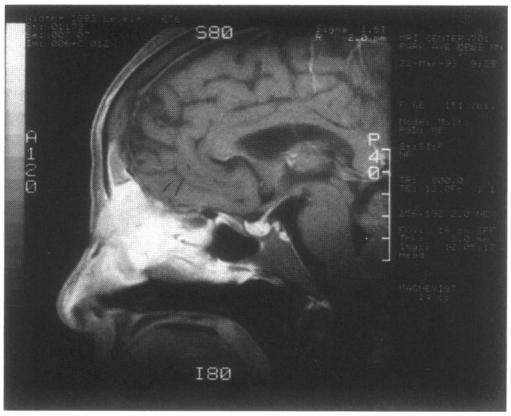


Figure 1. Saggital MRI scan of case 1 frontoethmoidal tumor showing erosion into anterior cranial fossa.

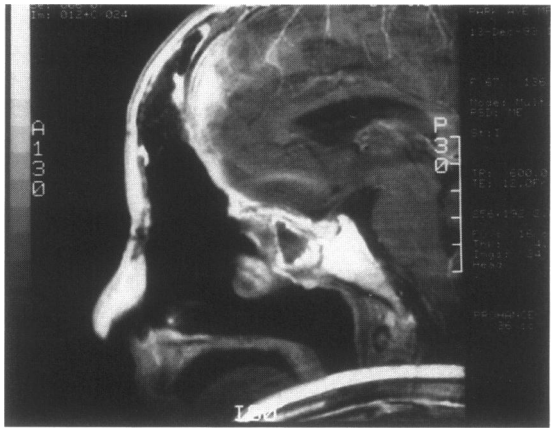


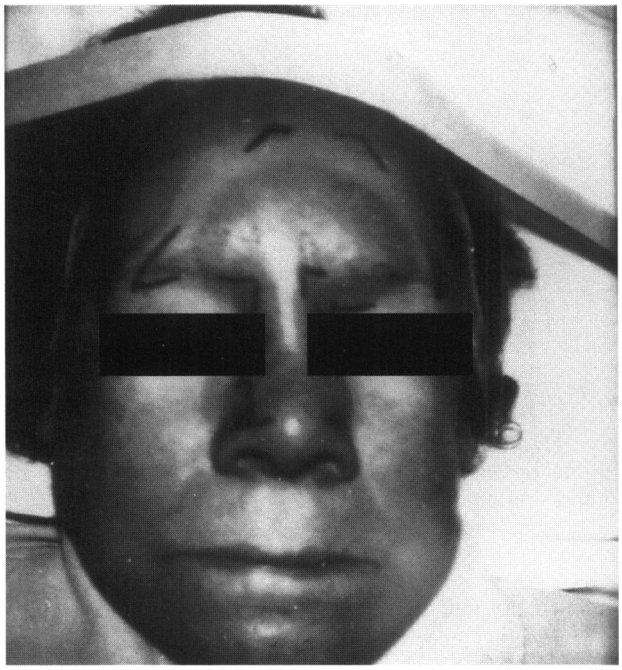
Figure 2. Saggital MRI scan of case 1 demonstrating persistent pneumocephaly behind fronto-naso-orbital bone flap.

have severe induration, ecchymosis, and supraorbital pain. Two months postradiation therapy, she complained of persistent pain, nasal discharge, and development of fistulae in the canthal region bilaterally (Fig. 3). Retrospective analysis of the treatment plan indicated that for each electron treatment prescribed to the 80% isodose line, the skin received approximately 232 centiGray, and the underlying bone received approximately 248 centiGray. Additional review of postoperative computerized axial tomography scans confirmed the presence of an air gap below the bone, allowing the possibility of a 20% to 30% "hot spot" to exist, delivering daily doses of 298 to 325 centiGray and potential total electron doses of 6556 to 7150 centiGray to the anterior cranium and fronto-naso-orbital segment.

Two months following radiation therapy, she was taken to the operating room for examination under anesthesia to clean her nasal-sinus cavity. At this time, it was noted that there was no mucosa lining the inner surface of the bone in the area of the air cavity space. She was started on hyperbaric oxygen therapy, as well as on ciprofloxacin 500 mg BID to treat the possible occurrence of both osteitis or osteoradionecrosis. She received a total of 33 treatments of hyperbaric oxygen, one treatment per day, five times per week. Each dive lasted 90 minutes at 2.4 atmospheres.

Follow-up radiographic evaluation with a nuclear medicine scan (technetium 99m) revealed a very marked decrease uptake in the area of the previous bone flaps. Computerized axial tomography scan and magnetic resonance imaging studies were also obtained, and showed a marked amount of encephalomalacia with a continued pneumocephaly and/or air pocket behind the frontal bone flap (Fig. 4). The patient still had some scar tissue present in the right frontal lobe, which was felt to be most likely secondary to the previous postoperative infarct.

The patient returned to the operating room in the following weeks for removal of the bone flap and titanium hardware. The bone was found to be completely avascular



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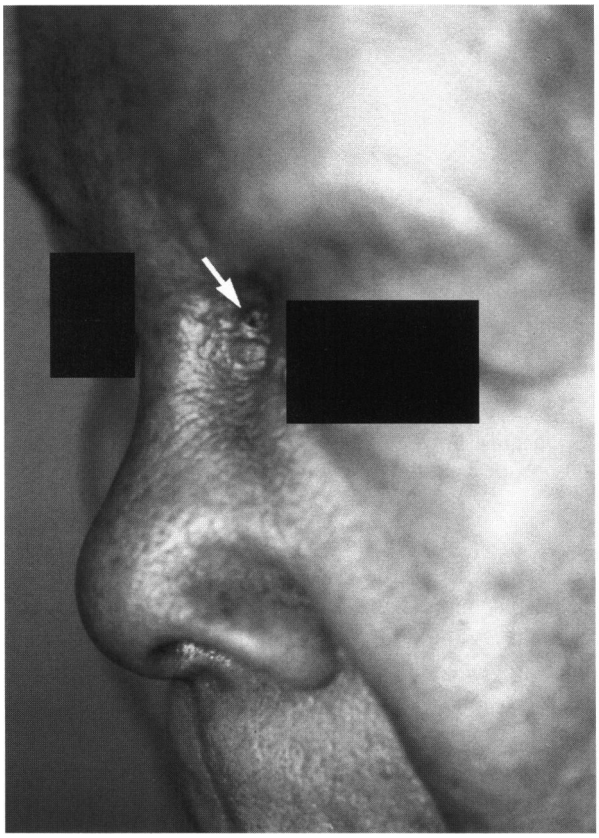

B

Figure 3. A: Patient in case 1: postradiation therapy showing induration and ecchymosis over naso-frontal region. B: Close-up of left medial canthal fistula.

and had a green appearance with purulent material in the diploic layer. This dead bone was removed until bleeding bone was encountered at the margins of the previous frontal craniotomy sites. Cultures at the time of surgery revealed *Enterobacter cloacae*, coagulase positive, and negative staphylococcus and diphtheroids. All were sensitive to trimethoprim-sulfamethoxazole, and enteral therapy was started. The surgical defect was reconstructed with a rectus abdominis free flap with anastamosis to the superficial temporal artery and vein. Her postoperative course was otherwise unremarkable. She did experience significant atrophy of the rectus abdominis flap, resulting in a loss of her frontal contour (Figs. 5, 6).

Eleven months later, methyl methacrylate cranioplasty was performed to establish a proper frontal contour. Approximately 2 months postimplantation, this patient developed skin breakdown and infection over the methyl methacrylate. This necessitated full removal of the implant. Presently, she remains free of disease.

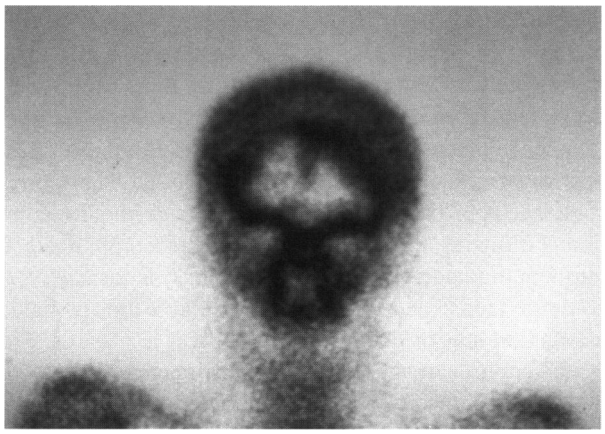

Figure 4. Nuclear medicine scan (technetium 99m) of patient in case 1 showing very decreased uptake in area of fronto-naso-orbital segment consistent with osteoradionecrosis.

Case 2

A 61-year-old male presented to a community otolaryngologist with a 2½-month history of sinus complaints, chronic nasal discharge, occasional epistaxis, and worsening hyposmia over the previous year. The patient's only medication consisted of propranolol for migraine headaches. The patient had stopped smoking in 1964. The patient's past medical history was significant only for a depressed right maxilla since birth, followed by osteomyelitis and sequestration of the right malar and zygomatic processes at 1 year of age. Examination at that time revealed a deviated septum to the left with a large polyp in the right nasal cavity. The patient was treated medically for presumed nasal polyposis and chronic sinusitis. There was some improvement in his symptoms, though the

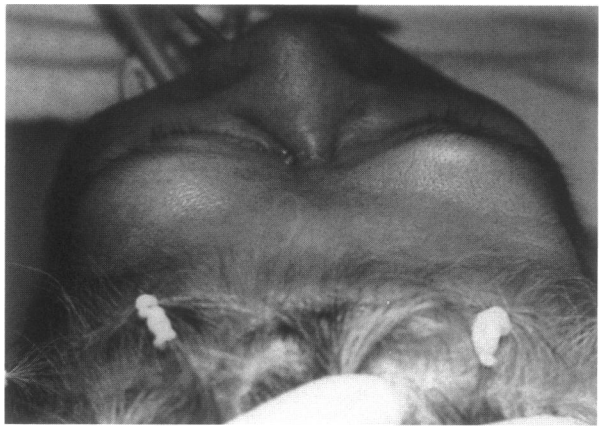

Figure 5. Patient in case 1 showing atrophy of free flap, prior to methyl methacrylate cranioplasty.

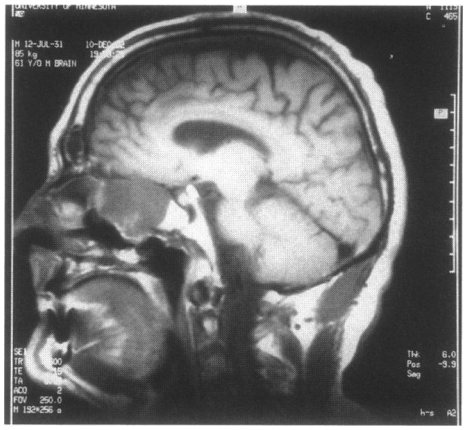

Figure 7. Sagittal MRI scan of case 2 frontoethmoidal tumor demonstrating location in sphenoid sinus and ethmoid sinuses with appearance of dural involvement.

nasal discharge with occasional epistaxis continued. A coronal computerized axial tomography scan of the sinuses revealed disease in the area of the ethmoid sinuses with erosion of the medial wall of the right orbit. Three months after starting medical treatment, the patient was taken to the operating room for an endoscopic biopsy, which was consistent with mucinous adenocarcinoma. The patient was then referred to the Department of Otolaryngology at the University of Minnesota Hospital and Clinics for further work-up and treatment.

A magnetic resonance imaging study of the head, with and without gadolinium, was obtained. This revealed a large soft tissue mass centered in the sphenoid sinus extending into the ethmoid sinuses bilaterally, abutting the anterior aspects of both carotid arteries, suggesting right orbital involvement through the lamina papyracea. The mass also extended along the cribriform plate with dural enhancement of the inferior surface of the frontal lobe in this region. However, there was no direct evidence of intracranial extension of the tumor (Fig. 7).

The patient was scheduled for radical surgical excision of the mucinous adenocarcinoma in January 1993. The patient underwent preoperative carotid balloon occlusion. He tolerated this well, without any clinical sequelae or evidence in the Xenon computerized axial tomography scan suggesting ischemia. Thus, the patient underwent a combined neurosurgical and otolaryngologic procedure with a subcranial approach utilizing a bifrontal craniotomy and removal of a fronto-naso-orbital segment for resection of the entire tumor from the ethmoid and sphenoid sinuses, right medial orbital wall, nasal septum, superior nasal bones, and superior nasopharynx. The tumor did not involve either carotid artery, and though the lamina papyracea was dehiscent on the right, there was no actual orbital involvement. Split calvarial bone grafts were used to reconstruct the anterior cranial fossa floor, and the anterior craniotomy flap and nasal bone flaps were replaced and secured using a standard miniplating system. The patient's postoperative course was unremarkable. The final pathology revealed mucinous adenocarcinoma involving the sphenoid sinus, both ethmoid sinuses, septum, and the superior aspect of the nasopharynx.

A review of the pathology indicated close margins at the posterior aspect, measuring 1.0 mm or less. As the patient was believed to be at excessive risk for local failure, he was referred for external beam neutron radiation therapy 1 month postoperatively. He received a fractionated course of 1800 neutron centiGray to the surgical bed and right orbital area over 2 months. This corresponds to approximately 7000 centiGray of photon radiation. Following this therapy, the patient complained of purulent drainage from his right maxillary sinus cavity. A computerized axial tomography scan at this time revealed a large amount of debris in this cavity.

A routine magnetic resonance imaging study obtained 6 months following his surgery showed a 1 cm tissue mass in the left frontal sinus region. The patient was taken to the operating room to rule out recurrence. The frontoethmoid cavity was inspected through an external trephination, and no gross lesion or bone erosion was

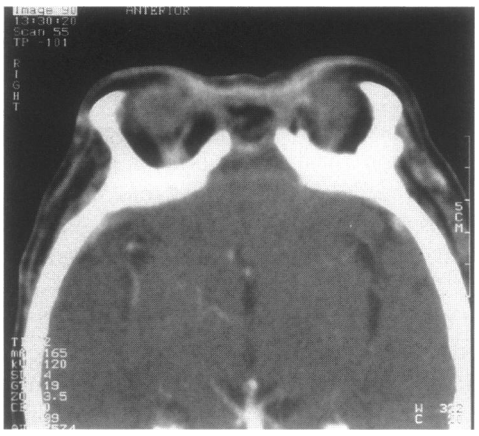

Figure 6. CT scan demonstrating identical area.

noted. The final pathology of the tissue specimens revealed chronic inflammation only.

Six months later, this patient continued to have persistent pain and drainage in the nasal area. The surgical wound from the frontoethmoid biopsy was open, with a green discharge. Examination revealed a large amount of nasal crusting within the cavity, left canthal wound breakdown with expressible pus, and facial erythema and induration on the left nasal maxillary process. A magnetic resonance imaging study at this time showed no evidence of tumor recurrence. A triple phase bone scan utilizing technetium 99m revealed decreased uptake in the nasofrontal bone area consistent with osteoradionecrosis (Fig. 8). Hyperbaric oxygen therapy was initiated at this point along with ciprofloxacin at a dose of 500 mg BID.

The patient underwent a total of 30 treatments with hyperbaric oxygen, one treatment per day, 5 days per week. Each treatment lasted 90 minutes at 2.4 atmospheres. While there was some clinical improvement, exposed bone remained in the left canthal area with continued nasal drainage. Follow-up computerized axial tomography scan showed only air-fluid levels in the sinuses bilaterally. This was managed medically. A follow-up bone scan utilizing technetium 99m showed no change in the area of uptake in the nasofrontal region. On exam, there was persistent exposed nonviable bone in the left canthal area. There was decreased erythema and induration over the face when compared to the time prior to hyperbaric oxygen therapy.

One month later, the patient underwent debridement of the anterior cranial fossa, including the fronto-naso-orbital segment, and the split calvarial graft was removed, showing gross evidence of osteoradionecrosis. The entire graft was removed, and reconstruction was accomplished via a rectus abdominis free flap (Fig. 9). Intraoperative cultures revealed *Xanthomonas maltophilia*, *Klebsiella oxytoca*, and coagulase-negative staphylococcal species. The patient was placed on enteral trimethoprim-sulfamethoxazole postoperatively and had an uneventful course.

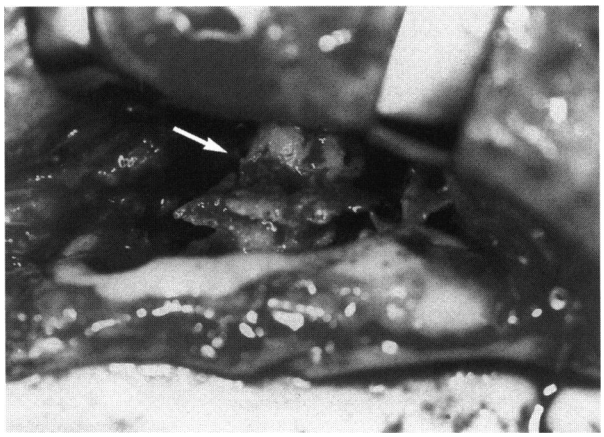

Figure 9. Surgical view of patient in case 2 showing necrotic fronto-nasal bone segment prior to removal.

The patient did experience atrophy of the rectus abdominis free flap (Fig. 10). Dermal fat grafts from the patient's abdomen were used to recontour the forehead. There is no evidence of disease recurrence or remaining osteoradionecrosis, and the fistula is now closed.

DISCUSSION

Osteoradionecrosis of the anterior cranium has not previously been reported. There are two different types of osteoradionecrosis, depending on etiology: posttraumatic osteoradionecrosis and spontaneous osteoradionecrosis. One of the notable differences between these types of osteoradionecrosis is the time of onset. The vast majority of spontaneous osteoradionecrosis cases occurs between 6 and 24 months after radiation treatment. It is felt that spontaneous osteoradionecrosis is the result of outright cellular kill by radiation. Trauma-related osteoradionecrosis, on the other hand, is more likely a mixture of cell death and injury (from surgical insult or other trauma)

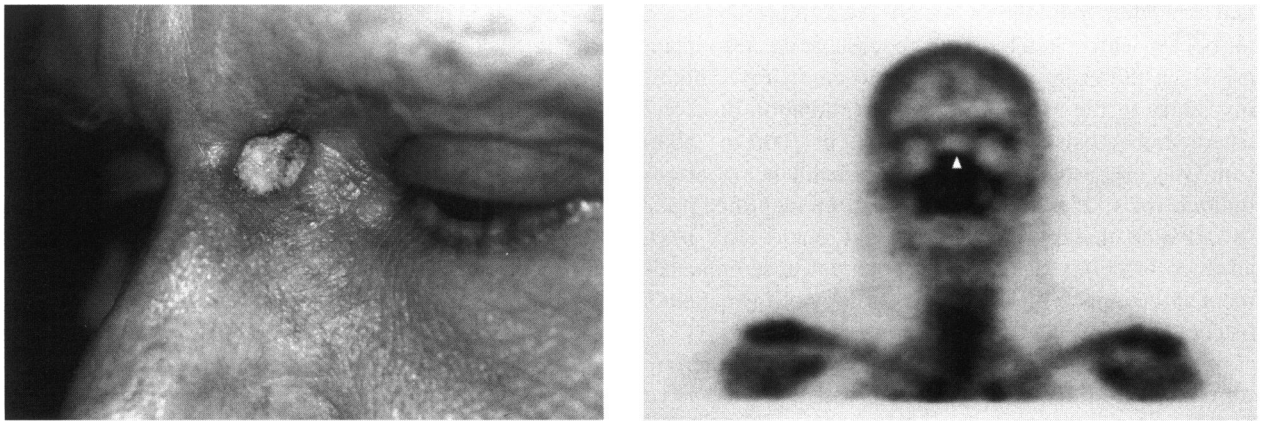

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Figure 8. A: Patient in case 2 showing nonhealing trephination site in area of left medial canthus. B: Nuclear scan (technetium 99m) of patient in case 2 showing decreased uptake in naso-frontal region consistent with osteoradionecrosis.

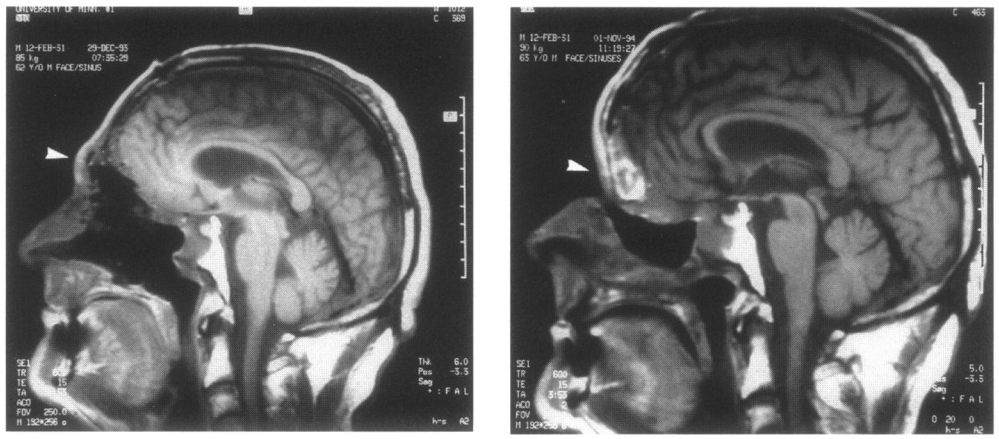

Figure 10. A: Sagittal MRI scan of patient in case 2 showing preosteoradionecrosis debridement contour. B: Profile after free flap placement and subsequent atrophy.

before or during radiation therapy.[4] This type of osteoradionecrosis has a bimodal peak of incidence. The first peak occurs at 3 months, and the second rise of trauma-related osteoradionecrosis starts around 2 years later and peaks at 5 years later.[1,4]

The two cases presented represent diagnostic and treatment dilemmas. The first dilemma involves the diagnosis of osteitis versus osteoradionecrosis. The bone scan utilizing technetium 99m seemed to give evidence in support of osteoradionecrosis, but there was a margin of strong enhancement that could also have been consistent with an osteitis. The ability to diagnosis osteoradionecrosis with bone scans is not reliable, especially in the early stages. As osteoradionecrosis is closely related to decreased vascularity of the involved tissue, evaluation of blood supply is difficult to assess with bone scans.

In the first case, osteoradionecrosis developed between 3 to 6 months postradiation treatment. The etiology was likely secondary to excessive electron beam dose to the frontal bone flap. The air pocket behind the bone was an unusual postoperative sequelae and was secondary to the cerebral infarct along with the subsequent encephalomalacia. This prevented the brain tissue from enlarging and expanding to fill the air pocket. It was this unexpected air pocket which resulted in the excessive electron beam dose—in the order of 6556 to 7150 centiGray. Studies investigating the association of total radiation dose and osteoradionecrosis show that dosages of 7000 to 75000 centiGray are associated with a higher incidence of osteoradionecrosis.[1] If this air pocket had been identified prior to radiation therapy, then the dosage could have been adjusted to compensate for the potential dose enhancement at the inner table. Thus, the etiology of the patient's osteoradionecrosis was spontaneous.

In the second case, neutron beam radiation therapy for treatment of the mucinous adenocarcinoma may have had an impact on the development of osteoradionecrosis. The patient experienced osteoradionecrosis at approximately 10 months postradiation therapy. His case most likely also represents solely spontaneous osteoradionecrosis. It should be noted that the dosage the patient received, 1800 neutron centiGray, was well within an established tolerance dose for neutron beam therapy in the head and neck region.[5] Calculation of equivalent dosage of photon radiation therapy from external beam neutron therapy can be performed using relative biologic effectiveness data. Responses of pulmonary metastases to treatment with neutrons versus treatment with Cobalt 60 photon therapy has allowed some of these relative biologic effectiveness values to be found. Fractionated courses of neutrons for salivary gland cancer (adenoid cystic type) resulted in a relative biologic effectiveness of 8.0, with a relative biologic effectiveness of approximately 3.0 to 3.5 for normal tissues. In other words, a fractionated course of 1800 neutron centiGray to a salivary gland malignancy would result in a biologically equivalent effect of approximately 5400 to 6300 photon centiGray to normal tissues, and approximately 14 400 photon centiGray to the salivary tumor.[6]

Assuming a relative biologic effectiveness of approximately 4.0, the patient received an equivalent of approximately 7000 centiGray of photon radiation. While this may be an acceptable level of radiation dosage, there are published reports of significant complications following neutron beam therapy. In one report of 38 complications resulting from neutron beam therapy for head and neck cancers, there were 8 cases of osteoradionecrosis. Seven of these involved the mandible, and one involved the maxilla.[7] In another study, six patients with recurrent pleomorphic adenomas of the parotid gland were treated with neutron beam radiotherapy with a median dose of 1800 neutron centiGray. One of these six patients developed osteoradionecrosis of the mandible.[8]

Radiation therapy has both acute and chronic effects on soft tissues and bone. Decreased vascularity of radiated tissues is secondary to decreased number of blood vessels and obliterative endarteritis. Eventually, fibrosis within marrow vessels and nutrient vessels occurs. It

should be noted that progressive loss of vascularity proceeds in an almost linear fashion for the rest of the patient's life.[9] Additionally, there is a lack of a steep oxygen gradient in radiated tissues, a necessary factor for macrophage chemotaxis. In turn, the macrophages secrete important factors for angiogenesis. This loss of macrophage chemotaxis leads to poor healing in response to injury. It is well established that radiation therapy has a detrimental effect on periosteum and marrow of bone. Some authors feel that direct radiation effects on osteocytes have a major impact on the development of osteoradionecrosis. This is due to an imbalance favoring osteoclasts leading to bone resorption. Thus vascular changes, as well as a possible osteocyte imbalance, are factors which play major roles in osteoradionecrosis development.[1] The classic sequence is described as radiation, hypoxic-hypocellular-hypovascular tissue formation, and tissue breakdown.[10] Overall, the tissue is less resistant to infection and traumatic insult. Basically, necrosis occurs when the rate of healing is exceeded by the rate of breakdown. Thus, multiple factors are involved in the pathogenesis of osteoradionecrosis.[10]

Traditionally, hyperbaric oxygen therapy has been helpful in the initial treatment and stabilization of osteoradionecrosis because it increases fibroblast proliferation as well as vascular neogenesis. The mechanism by which this is accomplished is thought to be as follows. In radiated tissues, there is only a gradual change in oxygen tensions between normal and radiation-injured tissues. Hyperbaric oxygen magnifies the shallow oxygen gradients found in radiated tissues, producing steep ones capable of allowing the macrophages to recognize the irradiated tissue as a true wound leading to angiogenesis.[9] Studies have shown that this steep oxygen gradient is produced after approximately 10 hyperbaric oxygen treatments and plateaus after 20 to 24 treatments. Long-term studies are needed to assess the permanence of the new vascularity after hyperbaric oxygen treatment. Currently, one study has shown that the effect of hyperbaric oxygen is still present 4 years after completion of treatment.[9] Thus, some authors feel that pretreatment of irradiated tissues with hyperbaric oxygen prior to surgical intervention should be prescribed.[4] There are small risks of hyperbaric oxygen therapy. Approximately 2% to 5% of patients will have middle ear difficulties requiring myringotomy.[1] Hyperbaric oxygen-induced toxicity seizures occur in less than 0.5%. of patients.[1]

The subcranial approach used in both of our cases may have also contributed to the development of osteoradionecrosis. Perhaps this might be described more accurately as osteoradiosurgical necrosis. The surgical technique used in both these cases involved removal of a bifrontal craniotomy bone flap and a fronto-naso-orbital segment. These bone flaps, once removed, actually become free bone grafts when replaced at the conclusion of the case. The grafts rely on nutrient periosteal and dural blood supply. In the first case, the air pocket between the bone flap and the dura resulted in a loss of this important blood supply to the inner table, making the bone more susceptible to radiation-related injury. In this case the entire bone flap, with the exception of a 1-cm margin at the osteotomy site, was necrotic. In the second case, the main bone loss was the fronto-naso-orbital segment, which lacked a sufficient nutrient inner mucosal lining. This, in conjunction with the radiation injury, may have contributed to the necrosis and eventual bone loss.

In both cases, the bony changes and destruction were not significantly altered by the hyperbaric oxygen treatments. These treatments probably did little to decrease the amount of bone that was eventually removed. The inability of hyperbaric oxygen therapy to reverse the process of osteoradionecrosis in the head and neck has been previously reported.[7] While soft-tissue radionecrosis has been shown to respond extremely well to hyperbaric oxygen therapy, the amount of osseous healing varies.[9] Farmer et al[11] found resolution in 54% and improvement in 23% of the patients treated with hyperbaric oxygen. Mansfield et al[12] reported favorable responses in 92% of cases. Surgical debridement along with hyperbaric oxygen therapy is particularly useful in large areas of osteoradionecrosis.[10] Marx and Johnson[4] reported successful resolution in all 58 patients treated with hyperbaric oxygen in combination with surgery. In fact, in major cases of osteoradionecrosis, removal of the bone is still the primary treatment.[1] Thus, lesions that do not fully respond to hyperbaric oxygen should be subjected to surgical debridement. After a full course of hyperbaric oxygen therapy, the first patient we discussed still had decreased uptake on her nuclear medicine scan and therefore was a candidate for surgical debridement and free flap reconstruction. In the second case, the patient had a persistent nonhealing area despite 30 treatments of hyperbaric oxygen. He too was a candidate for surgical debridement and free flap reconstruction. Thus, conservative therapy and hyperbaric oxygen alone did not appear to salvage either case.

Prevention of osteoradionecrosis in these cases may have been enhanced by using the pericranial flap to resurface the inner bone layer of the fronto-naso-orbital segment. A soft-tissue free flap could also have been used to augment and fill in the air space between the dura and frontal bone prior to initiation of radiation therapy, although a radiation modality other than electrons could also have been used to decrease the potentiation due to the air pocket. Unfortunately, acuity is always better in hindsight.

Patients with extensive osteoradionecrosis necessitating surgical debridement will usually need reconstruction. This can best be accomplished by vascularized free flaps. Nonvascularized bone grafts placed in irradiated beds have been shown to undergo a high rate of resorption and/or infection. Ademo and Szal had only a 50% success rate with these bone grafts. Hyperbaric oxygen therapy did increase these success rates significantly. Growth factors such as beta fibroblast growth factor (bFGF) and

bone morphogenetic protein-2 (MBP-2) may also enhance the healing of bone grafts in irradiated tissues. The combination of hyperbaric oxygen therapy and growth factors to enhance healing would seem additive, but no studies have yet investigated this. Vascularized flaps are a more popular method for reconstruction, which can be accomplished by pedicle flaps or free flaps. By bringing their own blood supply to the wound area, these flaps are not dependent on the local vascularity. Major arteries are still adequate for anastamosis even after significant doses of radiation.[1] This has inherent advantages, as the ability of hyperbaric oxygen to achieve local angiogenesis in an irradiated tissue bed is variable. Placing a vascularized free flap eliminates the potential lack of adequate local vascularity in supporting a nonvascularized graft. However, free flaps do carry their own risk. Thrombosis of the artery or vein may cause loss of flap. The large defects and unusual location of the osteoradionecrosis defects in both of our cases prevented pedicle flap reconstruction. Thus, rectus abdominis free flaps were used in each case. In both cases, the rectus flap underwent significant atrophy, necessitating second-stage recontouring with methyl methacrylate or dermal fat grafts. Unfortunately, the extrusion of the prosthesis in the first case creates the dilemma of a third surgical reconstruction in an irradiated bed.

Osteoradionecrosis of the anterior cranium has not been previously reported, and this would allow some presumption that this area, although often included in radiation ports, is relatively resistant to osteoradionecrosis. The blood supply via the diploic layer, as well as the periosteal blood supply, may be protective. The loss of the inner table blood supply and creation of free bone grafts in conjunction with large radiation doses may have contributed to the development of necrosis in both of these cases. Thus, osteoradiosurgical necrosis is a combined outcome of both treatment modalities.

CONCLUSION

Osteoradionecrosis of the anterior skull is an unfortunate and previously unreported complication of surgery in this area. The grim prognosis for cancers of the cribriform plate often necessitates multimodality treatment, including craniofacial resection and postoperative radiation therapy. Prevention of osteoradionecrosis is probably the most important issue. Pretreatment of the irradiated tissue with hyperbaric oxygen prior to any further surgical intervention may play a role. However, it is possible that the effects of hyperbaric oxygen will not be seen until significant vascular damage has occurred. Further studies to define at what point hyperbaric oxygen will be of benefit to irradiated damaged tissue will clarify this issue. The proper calculation of radiation dosage also needs to be adjusted when an air pocket exists in the treatment field. Osteoradionecrosis following subcranial tumor resection is an unexpected sequelae of this type of surgery. While hyperbaric oxygen treatment may stabilize this process, surgical debridement and reconstruction with vascularized tissue transfers will continue to be the best treatment. Finally, the subcranial approach, which necessitates devascularized bone flaps, may not be the ideal approach for patients requiring radiation therapy.

REFERENCES

1. Sanger JR, Matloub HS, Yousif NJ, Larson DL: Management of osteoradionecrosis of the mandible. Clin Plast Surg 20(3):517–530, 1993
2. Larson DL, et al: Major complications of radiotherapy in cancer of the oral cavity and oropharynx. Am J Surg 146:531, 1983
3. Raveh J, Laedrach K, Speiser M, et al: The subcranial approach for fronto-orbital and anteroposterior skull-base tumors. Arch Otolaryngol Head Neck Surg 119:385–393, 1993
4. Marx RE, Johnson RP: Studies in the radiobiology of osteoradionecrosis and their clinical significance. Oral Surg 64(4):379–390, 1987
5. Cohen L, et al: Normal tissue reactions and complications following high-energy neutron beam therapy: I. Crude response rates. Int J Radiation Oncol Biol Phys 16:73–78, 1989
6. Batterman J, Breur K, Hart G, Van Peperzeel H: Observations on pulmonary metastases in patients after single doses and multiple doses of fast neutrons and cobalt-60 gamma rays. Eur J Cancer 17:539–548, 1981
7. Stafford N, et al: Complications following fast neutron therapy for head and neck cancer. J Laryngol Otol 106:144–146, 1992
8. Buchholz TA, Laramore GE, Griffin TW: Fast neutron radiation for recurrent pleomorphic adenomas of the parotid gland. Am J Clin Oncol 15(5):441–445, 1992
9. Kindwall EP: Hyperbaric oxygen's effect on radiation necrosis. Clin Plastic Surg 20(3):473–483, 1993
10. Epstein JB, Wong FL, Stevenson-Moore P: Osteoradionecrosis: Clinical experience and a proposal for classification. J Oral Maxillofac Surg 45:104–110, 1987
11. Farmer JC, Shelton DL, Bennett PD, et al: Treatment of radiation-induced tissue injury by hyperbaric oxygen. Ann Otolaryngol 87:707, 1978
12. Mansfield MJ, Sanders DW, Hemback RD, et al: Hyperbaric oxygen as an adjunct in the treatment of osteoradionecrosis of the mandible. J Oral Surg 39:585, 1981