Osteonecrosis Related to Intraosseous Anesthesia: Report of a Case

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Abstract

Intraosseous anesthesia is an effective and increasingly used technique with few reported complications. The technique uses a specialized drill to perforate the osseous cortex where local anesthetic can then be deposited to anesthetize teeth. It has been reported that separation of the perforation drills from their plastic bases can occur because of the friction generated during osseous perforation. Prolonged rotation of the perforator drills in the bone can also cause excessive heat, which can lead to bone necrosis. This report describes a case of focal osteonecrosis subsequent to intraosseous anesthesia and discusses possible etiologies of this sequela. *(J Endod 2009;35:288–291)*

Key Words

Anesthesia, HIV, intraosseous, osteonecrosis

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Intraosseous dental anesthesia is an effective and increasingly used technique with few reported complications. This technique is particularly useful as a supplemental technique for anesthetizing mandibular molars when traditional inferior alveolar nerve blocks are unsuccessful (1). The intraosseous anesthesia technique involves perforating the osseous cortex to permit deposition of local anesthetic within the cancellous bone where it can readily diffuse to anesthetize the sensory nerves of teeth (1).

Instruments for perforating the cortex have historically included burs, endodontic reamers, and drills (2–5). This technique has recently been made easier and more predictable with improved armamentaria. Commercial systems are now available with specialized drills to create the cortical perforations. These products include the Intra-Flow (IntraVantage Inc, Plymouth, MN), Stabident (Fairfax Dental Inc, Miami, FL), and X-Tip (Densply Tulsa Dental, Tulsa, Oklahoma).

Few complications have been reported with these commercial products (6–8). Separation of the metal perforator drills from their plastic bases has been documented (7, 8). Authors have also reported an inability to adequately perforate some mandibles with unusually thick cortices or dense cancellous bone (7–9). This report describes an osteonecrotic defect that occurred after intraosseous anesthesia in a HIV-positive patient. Possible causes of the osteonecrosis are discussed.

Case Report

A 34-year-old white male presented to the Baylor College of Dentistry Department of Graduate Endodontics for participation in a retrospective study of root canal–treated teeth. Thirteen months earlier, he had nonsurgical root canal treatment of tooth #30 performed by an undergraduate dental student. A review of his medical history revealed an HIV-positive status for the past 10 years. The patient had been on antiretroviral therapy (ART) medications, including Kaletra (Abbott Laboratories, Abbott Park, IL) and Epzicom (GlaxoSmithKline, Research Triangle Park, NC). He had an undetectable viral load and a CD4 count of 450 (normal) at the time of treatment. He reported an otherwise noncontributory medical history. No history of bisphosphonate drug use was reported. He reported no discomfort with and normal function of tooth #30. He did report difficulty keeping the #30-31 area clean and using interdental brushes daily.

A more extensive review of his dental record revealed difficulty achieving local anesthesia with a conventional inferior alveolar block during the root canal procedure for #30. A dental student without any prior experience with intraosseous anesthesia used an X-Tip device to administer intraosseous anesthesia. During cortical perforation, the X-Tip's metal perforator shaft separated from the plastic base and firmly lodged into the interdental cortex between #30 and #31. After attempts to remove the metal perforator shaft with a hemostat failed, the gingival papilla was minimally reflected without a vertical releasing incision, and a small a mount of bone was removed to facilitate the retrieval of the metal perforator. A single suture was placed to secure the papilla. The dental record indicates that multiple buccal infiltration injections were administered permitting completion of the root canal at that appointment.

Two weeks later during crown preparation procedure for tooth #30, exposed bone was noted in the area of the intraosseous injection and persisted for another 2 weeks. After consultation by a faculty member from the department of periodontics, chlorhexidine rinses and monitoring of healing were recommended. Two weeks later, during the crown cementation appointment for #30, the exposed bone was reported as appearing to be healing slowly. After this, the patient was not seen again until 13 months later when he presented for participation in the graduate endodontic research study.

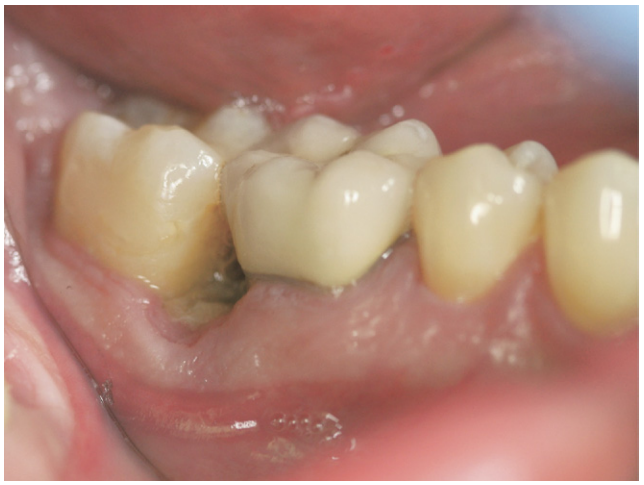

Figure 1. Initial presentation showing exposed necrotic bone.

Upon examination, a 3 × 3 mm patch of necrotic bone was noted in place of a gingival papilla in the buccal interdental space of teeth #30 and #31 (Fig. 1). Periodontally, the distobuccal of #30 probed 8 mm and the distolingual probed 6 mm. The periodontal biotype was normal. Normal physiologic mobility of both #30 and #31 was noted. Both teeth responded normally to percussion. Radiographically, the apex of the distal root of #30 showed an irregularly shaped radiolucency extending distally to the mesial of #31. A broad band of focal sclerosing osteomyelitis was evident apical to the radiolucency. The apex of the mesial root of #30 showed a widened PDL space with loss of the lamina dura (Fig. 2). Tooth #30 was diagnosed as previously endodontically treated with chronic periradicular periodontitis. Tooth #31 was diagnosed as pulpally and periradicularly normal.

The patient was referred to the department of oral and maxillofacial surgery for further evaluation and treatment. After the administration of local anesthesia, 2 mm of necrotic bone was removed with a surgical handpiece. No bleeding was encountered, and, consequently, a buccal flap was reflected, showing a section of necrotic bone extending to the depth of tooth #30's distobuccal periodontal pocket (Fig. 3). The necrotic segment was mobile and separated from the underlying bone by a layer of granulation tissue, which extended through the interradicular space to the lingual cortex. The segment of necrotic bone was removed with an elevator, and the granulation tissue was removed with curettage. A consultation from the department of periodontics suggested extraction of both #30 and #31 because of the size of the remaining osseous defect (Fig. 4). The teeth were removed with simple forcep extractions and examined for possible root fractures, with none noted. Dental implant replacements for teeth #30 and #31 were planned.

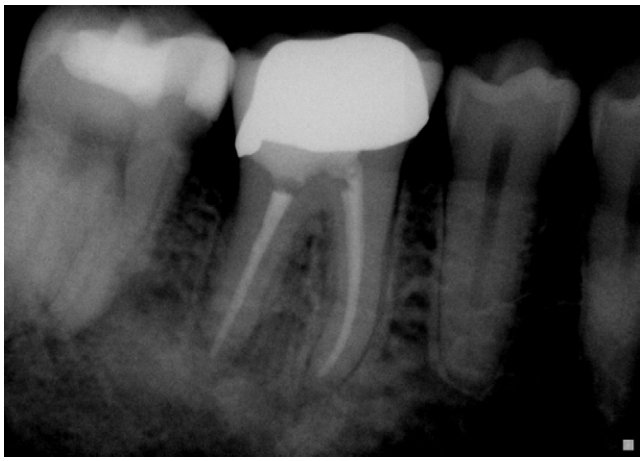

Figure 2. Periapical radiograph.

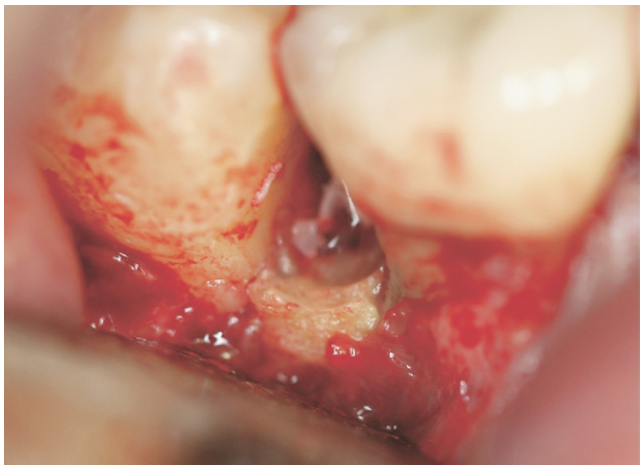

Figure 3. Reflected flap showing necrotic bone segment and granulation tissue.

Possible Effects of HIV and ART

Although tooth #30 showed periapical pathology, this lesion may not be related to the patient's HIV status. Two retrospective studies found no significant difference between the postendodontic periradicular healing of patients with HIV and noninfected controls (10, 11). Shetty et al. (12) retrospectively observed a 90% success rate at 6

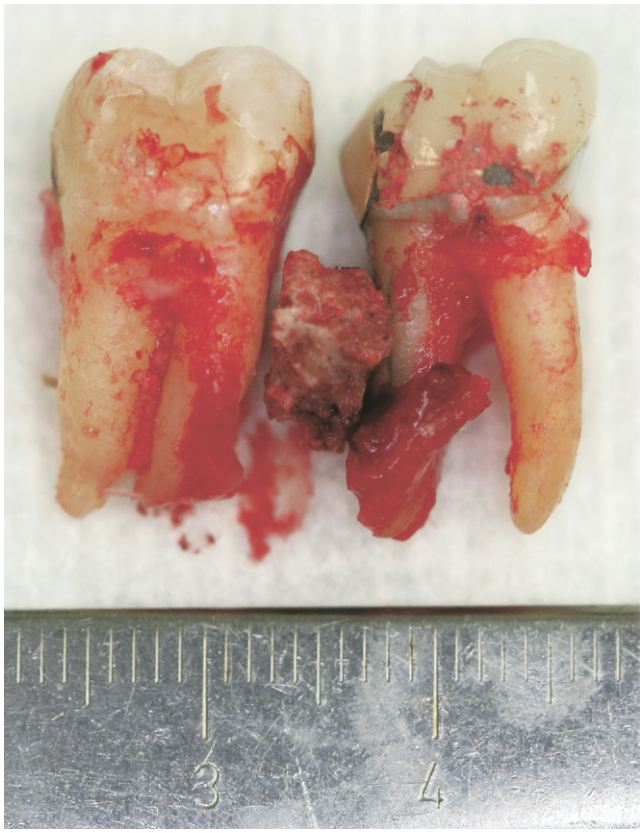

Figure 4. Extracted teeth and necrotic bone segment.

months in HIV-infected patients with no significant differences related to ART or viral load.

HIV-infected patients are known to have altered bone metabolism, but the specific etiology remains unknown (13). Osteonecrosis, also termed avascular necrosis, was first reported in the hip of a HIV-infected patient in 1990 (14). Since then, numerous case reports of osteopenia, osteoporosis, and osteonecrosis have been reported, and the prevalence of these disorders may be increasing (15–18). Estimates of the prevalence of osteonecrosis in HIV-infected patients vary and may be influenced by ART (13, 16, 19–21). Miller et al. (21) found 4.4% of HIV-infected patients had asymptomatic osteonecrosis. A similar cross-sectional study of Asian HIV-infected patients reported a 1.1% incidence of osteonecrosis of the hip, with increased incidence related to a longer history of ART therapy (18).

A recent report by Shetty (22) suggests that HIV patients may be at a higher risk for oral osteonecrosis. Other published reports document oral osteonecrosis in HIV-positive patients without ART but with oral herpes zoster infections (23–25). In these cases, herpes zoster infections are a possible comorbid factor because herpes zoster has been related to alveolar osteonecrosis in non–HIV-infected patients (26–28). Herpes zoster was not noted in the medical history of this patient.

Possible Effects of Intraosseous Anesthesia

The intraosseous anesthesia product used on this patient was the X-Tip. The X-Tip is a two-piece system with a perforating drill embedded into a plastic base that fits into a slow-speed latch-angle handpiece. A hollow metal guide sleeve overlies the metal perforator and also has a plastic base, which engages the plastic base of the perforator. This device is rotated in a slow-speed handpiece, penetrating attached gingiva and interradicular bone. The X-Tip's perforator is then withdrawn leaving the guide sleeve, through which a 27-G short needle can deposit local anesthetic.

One lawsuit alleged that an X-Tip perforator was separated, not removed and not disclosed to the patient (29). In another adverse event, an X-Tip separated during use and was reported to the Food and Drug Administration (30). Replogle et al. (8) reported that 4% of Stabident perforators separate from the plastic base. In all cases, they were easily removed with a hemostat. She specified that one separation occurred during a difficult perforation in dense cortical bone. The other two separations occurred when the handpiece was inadvertently stopped, stalling the perforator. In no case did the metal fracture. Coggins et al. (7) reported that 1 of 160 Stabident perforators separated from the plastic base and was retrieved with a hemostat. They reported that this failure occurred during a difficult perforation and suggested that the generation of heat during perforation contributed to the separation.

Undoubtedly, frictional heat develops during perforation because no water coolant is used. The bond between the plastic base and the metal perforator shaft may weaken with raised temperatures. The X-Tip is recommended to be rotated at 15,000 to 20,000 rpms and used with intermittent light pressure for 2 to 4 seconds to achieve cortical perforation (31). Difficult penetrations requiring extended perforation times may generate more heat and therefore may be at higher risk for instrument separation.

Damage to the adjacent bone is a more consequential risk than the damage to the perforator. Dentsply suggests, "To prevent 'frictional burning' of bone, always use light intermittent (forward & neutral motion) pressure, rather than continuous pressure" (31). Fister and Gross (32) showed retarded healing when cortical bone was cut without coolant. Eriksson and Albrektsson (33) showed that osseous temperature increases of as little as 10°C can cause osteonecrosis.

Dunbar et al. (6), Coggins et al. (7), and Replogle et al. (8) all reported swelling and purulence at some Stabident injection sites with an incidence of 5% or less. Reitz et al. (34) suggested that these findings may be related to gingival or bone trauma during perforation. Except for one secondhand anecdotal report of a patient who had a segment of bone removed a few weeks after a Stabident injection, a review of the published literature found no previous reports of osteonecrosis related to intraosseous anesthesia (35).

In the case report presented here, frictional heat may have contributed to the failure of the perforator. Stalling and binding of the perforator or drilling against a root may have occurred. Although intraosseous anesthesia is not an overly complex procedure, misuse or overuse of the perforator may be ascribed to iatrogenesis, with operator inexperience and error.

A gingival flap was reflected with a small amount of bone removed from this patient to retrieve the separated X-Tip perforator. Reflection of even a minimal flap may affect the periosteal vascularity and perfusion. Heat generated with the bur during the removal of bone could add thermal damage if no water coolant was used. After removal of the perforator, multiple buccal infiltration injections of anesthetic with vasoconstrictor were administered to permit completion of the root canal treatment. This may have further reduced blood flow and compromised healing potential of the traumatized bone.

Conclusions

This report presented a case of focal interdental osteonecrosis subsequent to intraosseous anesthesia with a review of possible etiologic factors. Overuse of the X-Tip perforator could have produced frictional heat leading to separation of the perforator and necrosis of the adjacent bone. Surgical retrieval of the separated instrument may have added further insult. The patient's HIV-positive status and ART medications may have limited normal bony healing. Dentists should receive proper training and understand the limitations and potential risks of intraosseous anesthesia before treating patients.

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