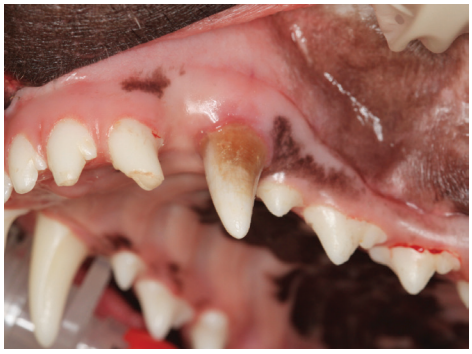

Figure 1—Photograph of the left maxillary canine tooth in a 1-year-old Bull Terrier that was evaluated because of tooth discoloration. Gray-brown intrinsic discoloration is present, and the gingiva along the mesial aspect of the tooth is inflamed and edematous. Notice the enamel fractures at the cusps of the left maxillary second and third incisor teeth.

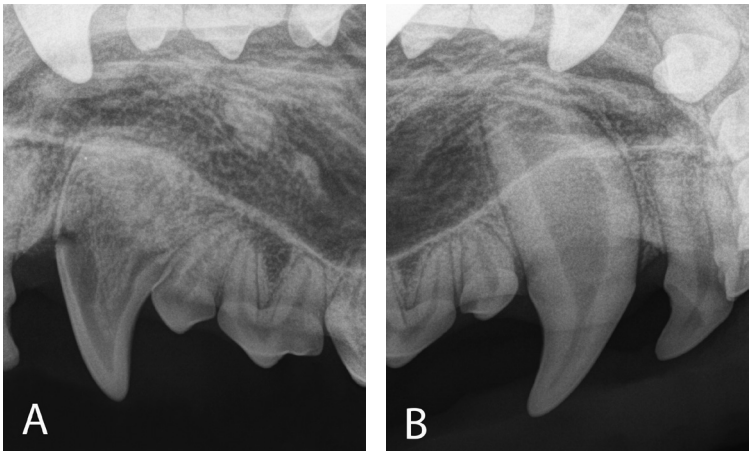

Figure 2—Lateral bisecting-angle radiographs of the left (A) and right (B) maxillary canine teeth of the same dog as in Figure 1.

History and Physical Examination Findings

A 1-year-old 16.7-kg (36.7-lb) sexually intact female Bull Terrier was referred to the dentistry and oral surgery service of a veterinary teaching hospital for evaluation of a left maxillary canine tooth with discoloration of unknown duration. The owner reported that the dog was adopted at 4 months of age; since that time, the medical history was unremarkable, with no known history of trauma.

A general physical examination did not reveal any clinically important abnormalities, and results of a CBC and serum biochemical analysis were within the respective reference ranges. Examination of the oral cavity revealed gray-brown intrinsic discoloration of the crown of the left maxillary canine tooth. The discolored area was examined with a dental explorer, and the enamel appeared to be of normal contour and hardness. The tooth had not erupted as much as its contralateral counterpart and thus appeared smaller. Focal gingivitis was also noted at the mesial aspect of the affected tooth. Enamel fractures of the left maxillary second and third incisor teeth were also noted **(Figure 1)**.

The dog was premedicated by IM administration of methadone (0.2 mg/kg [0.09 mg/lb]) and dexmedetomidine (6 µg/kg [2.7 µg/lb]). General anesthesia was induced with propofol (3.6 mg/kg [1.6 mg/lb], IV) and maintained with isoflurane (delivered at a variable rate) in 100% oxygen. Intraoral radiographs of the anesthetized patient were obtained. A lateral bisecting-angle radiograph[1] of the left maxillary canine tooth and a radiograph of the contralateral tooth are provided **(Figure 2)**.

Determine whether additional studies are required, or make your diagnosis, then turn the page →

This report was submitted by Kevin K. Ng, BVMS; Nadine Fiani, BVSc; and Santiago Peralta, DVM; from the Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853.

Address correspondence to Dr. Peralta (sp888@cornell.edu).

Diagnostic Imaging Findings and Interpretation

On intraoral radiographs, the pulp cavity of the left maxillary canine tooth appeared relatively wide, compared with that of the contralateral canine tooth. The affected tooth also had abnormally thin dentin with a shorter root than its counterpart and an open apex. The enamel was of a thickness similar to that of the contralateral tooth. These findings were consistent with arrested tooth development.[2] A complicated crown fracture was present at the mesial aspect of the undererupted tooth, along with secondary focal periodontitis **(Figure 3)**. Additionally, heterogeneous material of mineral opacity, resembling trabecular bone, was present within the pulp cavity, consistent with pulp mineralization.[3] Distoapical to the left maxillary canine tooth, 2 radiographically quiescent objects of mineral opacity were observed. The nature of the mineral opacities was uncertain, but their appearance was considered suggestive of foci of sclerotic bone.[4]

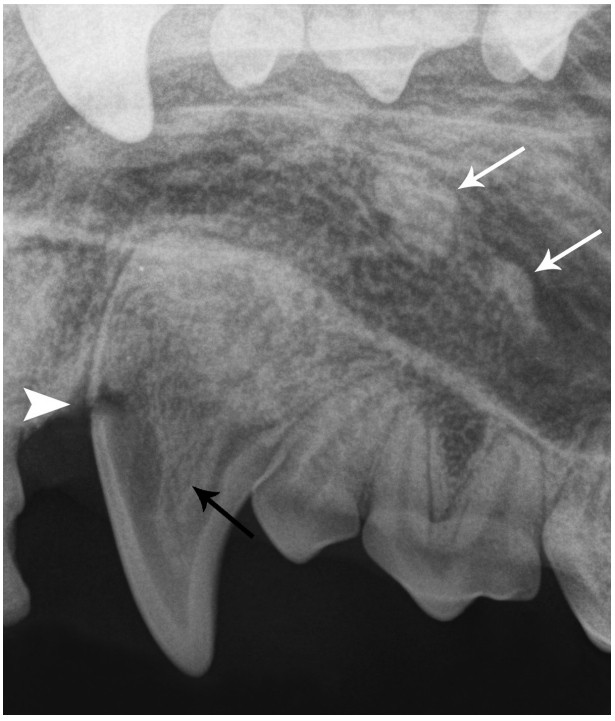

Figure 3—Same radiographic image of the left maxillary canine tooth as in Figure 2. The affected tooth has a relatively wide pulp cavity, with thin dentin, although the enamel width is similar to that of the contralateral maxillary canine tooth. The findings are consistent with arrested tooth development. The pulp cavity is filled with heterogeneous material of mineral opacity, indicating pulp mineralization (black arrow). Two dense objects of mineral opacity are located distoapical to the left maxillary canine tooth (white arrows). The bone surrounding these objects appears radiographically normal. The crown structure at the mesial aspect of the undererupted left maxillary canine tooth is discontinuous, consistent with a complicated crown fracture (arrowhead), and there is localized widening of the periodontal ligament, consistent with focal periodontitis.

Treatment and Outcome

Under the same anesthetic episode, the oral cavity was rinsed with 0.12% chlorhexidine gluconate solution, and all teeth were supragingivally and subgingivally ultrasonically scaled. Nerve blocks were performed with bupivacaine solution (0.5 mL; 5 mg/mL) containing epinephrine (1:200,000).

The thin dentin, open apex, and extensive pulp mineralization precluded root canal treatment of the left maxillary canine tooth, which was surgically extracted. Briefly, a triangular mucogingival flap was elevated before labial alveolar bone was removed with tungsten-carbide burs. Luxators were used to facilitate removal of identifiable tooth material, and the extraction site was closed with 4-0 poliglecaprone 25 suture material in a simple interrupted pattern. Grossly, the mineralized pulp contents could not be differentiated from alveolar bone. Surgical removal of the mineralized objects distoapical to the affected tooth was not attempted because of their benign radiographic appearance.

The dog was discharged from the hospital, and the owners were instructed to administer tramadol (4.5 mg/kg [2.04 mg/lb], PO, q 8 to 12 h) and meloxicam (0.1 mg/kg [0.05 mg/lb], PO, q 24 h) for analgesia for 7 days. The owners were also advised to feed the dog soft food for a period of 2 weeks and to prevent the dog from engaging in activities involving the oral cavity such as chewing or playing with toys for the same period. An oral examination performed 5 months later revealed gross healing of the left maxillary canine tooth extraction site. Annual radiographic follow-up of the objects with mineral opacity was recommended.

Comments

Tooth discoloration can be intrinsic, with the discoloration incorporated into the enamel, dentin, or both; it can also be extrinsic, with the discoloration arising from the accumulation of exogenous pigments on the surface of the tooth.[5] Intrinsic discoloration can result from a wide variety of causes, including tetracycline administration, amelogenesis imperfecta, dentinogenesis imperfecta, trauma, and hyperbilirubinemia.[5,6] In the dog of the present report, the discoloration was associated with arrested tooth development and mineralization of the pulp cavity in a fractured left maxillary canine tooth. This was suggestive of a previous traumatic episode. Moreover, the enamel fractures on the adjacent left maxillary second and third incisor teeth were likely the result of trauma. Even if trauma does not result in pulp exposure and direct bacterial contamination of the pulp, pulpitis can result. Pulpitis can lead to intrinsic discoloration due to localized vascular damage or diffusion of hemoglobin breakdown products into dentinal tubules vacated by the death of odontoblasts. Initially, this appears as purplish or pinkish discoloration, which may gradually fade or resolve

in cases of reversible pulpitis. In cases of irreversible pulpitis resulting from substantial pulp damage, the discoloration can take on a bluish or grayish tinge (as observed in this dog) as the hemoglobin components in the tubules degrade.[5,6]

Eruption of the canine teeth in dogs is reported to occur between 5 and 6 months of age.[7] While a distinct episode of trauma was not reported by the owners of the dog of this report, the incomplete eruption of the left maxillary canine tooth, along with the presence of a relatively wide pulp cavity, thin dentin walls, and an open apex, suggested that a traumatic episode occurred just prior to complete eruption. Disruption of the apical blood supply, irreversible pulpitis, and bacterial infection of the pulp cavity (alone or in combination) typically lead to loss of pulp vitality and death of odontoblasts, which are specialized cells located on the periphery of the pulp cavity that produce dentin. This leads to cessation of dentin formation and arrested tooth development. When loss of pulp vitality occurs in an immature tooth, the characteristic features of a wide pulp cavity, thin dentin walls, incomplete root development (evidenced by a shorter root, compared with that of the contralateral tooth), and an open apex frequently persist. If identified substantially later than the traumatic episode, these features appear inconsistent with the patient's age, as was the case in the dog described here.

Other radiographic features described in this case could also be attributed to trauma. Trauma to developing teeth can result in eruption disturbances,[8] and this could explain the incomplete eruption of the affected tooth in this dog. Likewise, the radiodense objects distoapical to the left maxillary canine tooth were suggestive of foci of sclerotic bone secondary to trauma. An episode of trauma can be overlooked if the affected teeth are not grossly displaced or obviously fractured, as was the case in the dog of this report.

Pulp mineralization was another feature observed radiographically. Although histologic evaluation is required to determine the exact nature of pulp mineralization, dentin-like, bone-like, or fibrotic-type mineralization has been reported in the pulp cavities of human teeth following trauma,[9] and bone-like pulp mineralization has been experimentally induced in dogs.[10] In most cases, the mineralized tissue originates from the pulp cavity walls and is dentin-like, but it occasionally may be bone-like and not associated with the pulp cavity walls.[3,9,10] Pulp mineralization has been most frequently reported in avulsed or luxated teeth with immature apices following treatment, but has also been reported secondary to crown fractures or concussion.[11] Although the exact etiopathogenesis of pulp mineralization is unclear, the open apex is thought to allow recruitment of surrounding progenitor cells to replace the devitalized pulp with mineralized tissue, as long as the pulp remains uninfected.[12] Excessive odontoblastic activity following trauma has also been proposed as a cause of pulp mineralization.[13] Lack of endodontic infection seems to be an important determinant in the formation of mineralized pulp secondary to trauma. The nature of the tooth fracture in this case and its subgingival location might have delayed bacterial infection of the pulp cavity and allowed pulp mineralization to develop.

The case reported here highlights the importance of intraoral radiography for the evaluation of discolored teeth. Dental radiographs can enable detection of teeth that have been injured by previous trauma even if a history of trauma is not elucidated. The radiographic appearance of the affected tooth can also provide clues to the timing and nature of the traumatic event.

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