

CASE OF THE MONTH (January 2012)

Signalment and History: A three year old neutered male Lab X was referred for extensive enamel defects affecting the majority of the teeth in the mouth. The affected teeth clearly demonstrated large areas of tooth surface with missing enamel, exposing the underlying dentin.





Procedures: The patient was placed under general anesthesia for a complete oral examination and full mouth radiographs: While under anesthesia we detected enamel defects on 32 of the 42 teeth in the mouth.

Full mouth radiographs were taken to search for evidence of endodontic involvement of these teeth. These findings were negative.

We elected to perform a dentinal bonding procedure on all of the affected teeth to seal them from bacterial invasion.

The first step in this procedure is acid etching of the teeth with a 37% phosphoric acid gel.



After acid etching, a bonding agent is applied to the defects.



Next a curing light is applied to the bonding agent.



Permaseal, an unfilled resin, is applied as a final layer and light cured.



Discussion: Being 96 % mineralized, enamel is the hardest tissue in the body. It lies on the surface of the tooth, protecting the dentin below. On the Mohs hardness scale, enamel ranks a grade of 5, while dentin comes in at 3-4.

Dentin is a porous material, filled with 30-40,000 dentinal tubules per mm². These tubules travel from the dentinal surface to the pulp of the tooth. Contained within these tubules are nerve endings connecting to the pulp, and if exposed by missing enamel, can cause tooth sensitivity. If dentin is exposed, these tubules also create an open avenue for bacterial invasion into the pulp, which can lead to pulpitis and pulp necrosis.

Enamel defects can occur as a result of trauma such as a fracture, or as a result of abnormal amelogenesis. Amelogenesis, the development of enamel on the tooth bud, can be affected by trauma, genetics, or systemic infection at the time this process is occurring in the unerupted tooth.

In this case, the enamel defects did not have the typical appearance of enamel fractures and they were widespread throughout the oral cavity, suggesting enamel hypoplasia, a developmental anomaly. If we see localized enamel hypoplasia in the mouth, we suspect localized trauma at the time of amelogenesis. With a widespread distribution of lesions such as we have here, we suspect systemic involvement such as an illness with a high fever. Those of you that are old enough to have seen cases of canine distemper will remember the extensive enamel hypoplasia often described as “distemper teeth.”

When treating enamel hypoplasia, we must first radiograph the affected teeth to be sure endodontic involvement has not already taken place. Radiographic evidence of endodontic infection would appear as a widened periodontal space, periapical lucency, or root resorption.

The first step in treatment is acid etching. This process removes debris from the dentinal tubules and can be thought of as “opening the pores” of the tooth. This process creates an optimal environment for placing the bonding agent.

The bonding agent is an acrylic, a liquid plastic. This liquid runs down into the open tubules. The chemical makeup of the bonding agent is a monomer. When using the curing light we are transforming the monomer liquid plastic into a polymer, which is a plastic in solid form, thus creating solid plugs to seal the openings of the dentinal tubules. This process prevents bacteria from invading the pulp of the tooth via the exposed dentinal tubules.

Permaseal is one brand of an unfilled resin, which is spread over the surface and light cured, creating the final layer of protection.

COMMUNITY ANIMAL HOSPITAL

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