Focus On: Dental Resorptive Lesions in Dogs.

For decades we have been all too familiar with dental resorptive lesions in cats. While there is lots we know about them (how frustratingly common they are, how challenging they are to deal with, how important intra-oral dental radiographs are for assessing them, what is actually going on at the cellular level) there is still lots we do not know about them (why they happen). I wrote about the feline version of the condition a few vears ago (see http://www.toothvet.ca/PDFfiles/Tooth resorption_in_cats.pdf)

Well, cats are not the only ones that can get external root resorption. As we take more intra-oral dental radiographs in dogs, we are finding this condition popping up more and more.

I am not talking about the inflammatory type of root resorption that can occur in the face of chronic endodontic and/or periodontal disease. In that instance, there is inflammatory destruction of both the root and the surrounding bone and the lucent area between the bone and root is filled with soft tissue. And it looks something like this.

Figure 1



Nor am I talking about internal root resorption which happens when chronic inflammation of the pulp leads to the development of odontoclasts inside the tooth eating away at the inside of the dentin wall. Figure 2 is a radiograph of an extracted incisor that had undergone some internal resorption in the region of the junction of the root and crown.

Figure 2



Before going into details about the condition, we need to review normal periodontal anatomy. More detailed discussion on this can be found and reviewed at

http://www.toothvet.ca/PDFfiles/PerioA nat&Physio.pdf.

The tooth is made of four tissues. A thin veneer of enamel covers the crown. A thinner veneer of cementum covers the root. Dentin (under the cementum and enamel) makes up the bulk of the mature tooth. The pulp tissue lies within the hollow chamber in the centre of the tooth. The pulp chamber is lined with a single layer of cells known as odontoblasts. Their job is to produce dentin on the inside of the tooth. They are the only cells capable of producing dentin.

Most of the root is embedded in a depression in the bone (the alveolus) but a portion of the root extends above the top of the alveolus.

There are four tissues that make up the periodontium: gingiva, alveolar bone, periodontal ligament and cementum. Note that cementum was mentioned in both the list of dental tissues and periodontal tissues. While it is physically part of the tooth, it is considered part of the periodontium and acts in a fashion similar to periosteum covering bone.





Of the four periodontal tissues, two are hard (alveolar bone and cementum) and two are soft (gingiva and periodontal ligament). The two soft tissues will only ever attach to those two hard tissues. Gingiva and periodontal ligament will not attach properly to enamel, dentin or restorative materials. Because of the presence of the periodontal ligament, the root should not be in contact with the bone – there should be a space between root and bone filled with the ligament fibers, blood vessels, nerves and various cell types including cementoblasts, which are the cells that produce cementum.

The gingival tissue, which in health is very dense and firm, should be solidly attached to the alveolar bone and then to the cementum on that part of the root that extends beyond the bony confines of the alveolus. It is this gingival attachment to the collar of cementum at the top of the root that prevents bacteria from getting at the deeper periodontal tissues.



Next digression is to ask, "why do the roots of adult teeth last a life time in most cases?" The primary tooth roots are supposed to resorb to allow the primary teeth to exfoliate, making room for the permanent teeth. The other hard tissue in the body (bone) is constantly being resorbed by osteoclasts and replaced by osteoblasts. There must be something peculiar about the adult tooth roots that normally protects them from resorption. Quite possibly it is the periodontal ligament acting as a soft tissue buffer between the root and the cells that might resorb them.

When the protective mechanism fails, multinucleated giant cells from the tissues around the outside of the root can evolve into odontoclasts, which are physically indistinguishable from osteoclasts. The difference is that instead of eating bone, they are eating dentin. When bone is remodeling. the osteoclasts eat bone and osteoblasts produce bone. When root is being resorbed, odontoclasts eat away at the outside of the tooth, but the only odontoblasts in the body are inside the tooth, lining the pulp chamber. So the

hard tissue that replaces the lost dental tissue is produced by osteoblasts and/or cementoblasts so the tissue has characteristics of bone and cementum and is often referred to as bonelike/cementum-like tissue or BCL tissue.

Time to look at some radiographs. Figure 5 is a film of a normal lower first molar tooth. On this film, you can see that the root is very definitely more radiodense than the surrounding bone and there is a thin, discrete lucent line between the root and the bone all the way around. This lucent line is the periodontal ligament space. The root surfaces are smooth and follow naturallooking contours. The endodontic chamber is also clearly visible as a lucent area within each root and the crown.

Figure 5



In figure 6, a radiograph of the right lower fourth premolar and first molar of an older dog (note how much smaller the endodontic chambers are due to dentin production inside by the layer of odontoblasts), we can still see a distinction between root and bone and there are some areas where the periodontal ligament space is still visible, but there are also areas where the ligament space is not visible. This indicates calcification of the ligament and fusion of root to bone, known as ankylosis.

This radiographic finding is significant for a few reasons. For one, without a periodontal ligament to act as a shock absorber, these teeth are more subject to fracture under stress. Also, due to the fusion of the root to the bone, extraction of these teeth would be much more challenging. Finally, the ankylosis may be a precursor to the development of external root resorption.

Next we have two films of similar presentations of external root resorption of the right lower 1st to 3rd premolars.

Figure 6



Figure 7

3



Figure 8



All of the teeth imaged show obvious loss of root structure, but a periodontal ligament space has been maintained between the disappearing root and the bone that is replacing it.

In figure 7, the mid portion of the distal root of the second premolar (arrows) has been partially resorbed and the area filled in with bone, but the apex of the root is unaffected. This means that the apical portion of this root is wider than the alveolus above it, making extraction of this root challenging. The apical portion of the root will not be able to pass through the small hole above it. To deliver this root from the socket, the socket would need to be surgically widened first. Another example to show why we always take pre-operative radiographs before any extraction.

In figure 8, the roots have been resorbed from the apex up and have maintained a tapered profile and so they would not be locked into their sockets.

Of the six teeth in these two images, there is only one that I would extract. In figure 7, all the lesions are deep in the socket, well below intact and healthy collars of gingival attachment and so are isolated from oral bacteria. There is sufficient root structure remaining to support the crowns and the teeth are functional. Based on histological studies and reports from human patients with similar lesions, there is likely no inflammation and no pain associated with these teeth. The same can be said for the second and third premolars in figure 8.

We cannot know by looking at these films if these lesions developed some years ago and have been sitting static ever since or if they just started last month and are progressing rapidly. So, for the three teeth in figure 7 and the two in figure 8, my plan would be to document and disclose the lesions to the owners and then re-radiograph in a year or sooner if there are any clinical signs of trouble. For the first premolar in figure 8, it has lost virtually all of its root, it is just floating in the gingiva and so I would remove it now and suture the wound.

Can we predict how rapidly these lesions will progress? Not in my experience. I have been able to follow a few patients with external root resorption for several years. I have seen some teeth remain completely static year after year while an adjacent tooth has gone from looking perfectly normal one year to needing extraction the next.

Below is a tougher presentation to deal with. Figure 9 is a radiograph of the left upper fourth premolar tooth with extensive external root resorption in a manner very similar to the radiographic appearance of feline dental resorptive lesions. Figure 9



There is loss of distinction between root and bone and the resorption is occurring in a diffuse manner with BCL tissue growing into and interdigitating with the dental tissue. The lesion has extended through the level of the gingival attachment to communicate with the oral cavity and become contaminated with oral bacteria. This tooth requires extraction of all three roots and it is not going to be easy. With the BCL tissue growing into the weakened root structure, the root is going to crumble and will be difficult to separate cleanly from the surrounding tissues. The apical quarter of the distal root still appears intact with periodontal ligament space visible, so if we can get down to it, that portion of the root should elevate out intact. This is a case that will require reflection of a mucogingival flap and aggressive surgical exposure of the roots to visualize them and excavate them. Intra-operative and post-operative radiographs will be needed to evaluate progress throughout the procedure. It is beyond the scope of this paper to go into greater detail on how to go about removing these challenging teeth, but basically I treat them just like cat teeth. The only difference is that they are much larger and there are potentially many more of them in a dog mouth to deal with.

Figure 10



Figure 10 is of the left lower first molar tooth. Again, the lesion has extended above the level of gingival attachment and so is contaminated by oral bacteria and the tooth requires surgical extraction. The mesial root (to the left) is intact but may be ankylosed whereas the distal root is in bits-and-pieces.

Figure 11 shows a left lower first molar. The distal root (to the right) looks pretty normal on this film. It is the mesial root that has issues. At the top of the socket, there is no periodontal ligament space visible and the bone around the root in this region appears denser than normal indicating ankylosis. Then on the mesial side of the apical half of the root, there is a lucent zone (arrows) indicating external resorption. When did this lesion start? How fast is it progressing? Is it progressing at all or is it static? We cannot know the answers to those questions based on a single set of films. They only tell us where the tooth is now but do not tell us anything about how fast it is moving.

Figure 11



In this case, we are dealing with a large and functionally important tooth. The ankvlosis would make extraction challenging. The resorptive lesion is deep in the socket well away from bacterial contamination. Therefore, my approach for this tooth would be to leave it in place and re-assess it clinically and radiographically on an annual basis. Extraction may eventually become necessary, but it is also possible that the tooth will last the rest of the dog's life.

I do find that some teeth are more commonly affected than others. In general, I find that the first premolars are the most commonly affected teeth. As they are not very important teeth and have but one simple root, I am quick to extract these teeth if they show any sign of resorption.

The next most common pattern, when dogs have more generalized external root resorption, is to find it affecting all of the premolars and the lower first molars. For some unknown reason, the upper molars, lower second and third molars, incisors and canines tend to be far less commonly affected. Having said that, here is a dog in which only the canine tooth was affected.





In figure 13 we see the lesion was largely affecting the root, but as the resorption moved up the tooth and extended into the dentin of the crown, it undermined the thin veneer of overlying enamel which then caved in revealing the coronal portion of the lesion.

Figure 13



Then there was Chloe. She is an 8-yearold Eurasier presented for concerns about external root resorption. She was already missing the crowns of many teeth and those that appeared to be present were all in trouble. Every single tooth in her head had some degree of resorption going on. Every single tooth and root remnant was removed.

One of the biggest questions about external root resorption is what the heck causes it. There are a few possibilities.

Use of active-force orthodontic appliances, especially if the forces are excessive, can lead to damage to the

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periodontal ligament and alveolar bone resulting in a cascade of events culminating in ankylosis and/or root resorption.

Figures 14, 15, 16



Chronic periodontal irritation such as from excessive occlusal forces (from inappropriate chewing or malocclusion) can lead to the same events as above.

When a single tooth or a few in one area are affected, trauma to that area could be the cause. If a tooth has been avulsed (traumatically removed from the socket) and placed back into the socket, we might get proper healing with reestablishment of a normal periodontal ligament or we may get ankylosis and eventual root resorption.

Hypervitaminosis D can lead to calcification of the periodontal ligament resulting in root resorption.

In humans, there are familial tendancies toward root resorption.

In most cases, however, we are left with 'idiopathic external replacement root resorption'.

Once again, the big take-home message is that you must be using intra-oral dental radiography on a regular basis to find and document this and so many other dental concerns.

Addendum January 2012

As I have mentioned, it is currently not possible to predict the rate of progression of tooth resorption. I would like to illustrate that with two contrasting cases.

First we have Luther, a male/neutered Labrador cross born around 2000. I first saw him in June of 2009 and took the following images.





These were the only teeth with radiographic evidence of resorption at that time. A few factors led me to leave these teeth in place:

- No other invasive procedures were being done to trigger post-op pain management.
- The lesions were still completely subgingival and uncontaminated and so likely asymptomatic.

As they are single rooted teeth, if their single root continued to resorb, the crown would have exfoliated and the wound would have healed quickly.

So I left 305 and 405 alone. Next time I saw Luther was in April of 2011 and I got these images.



Aside from the fact that the images were processed with different soft-ware, I can see no change over the 22 months between these studies. The rest of the whole-mouth study revealed no new lesions either. So by all accounts, nothing of significance changed between June of 2009 and April 2011.

Then there is Chelsea, a spayed female giant Schnauzer born in November of 2005. I did my first radiographic study of Chelsea in August of 2009 and the second one was done in June of 2010. In this instance I will post the 2009 image and the 2010 image of the same area.



Apparent progression of resorption of tooth 106.



No apparent progression of lesion of palatal root of 208.





Apparent development of lesion of tooth 308 but no progression of anykylosis of mesial root of 309.





Apparent development of new lesion affecting tooth 405.



Apparent new lesion affecting tooth 408.

So there you have it. Two dogs, two very different situations. In one dog, no radiographic evidence of progression of the affected teeth and no new lesions detected over a two-year span. In the other dog, some teeth progressed, some previously "normal" teeth now affected and some previously affected teeth apparently static a year later. There just is no predicting.