Diagnosis of Secondary Caries

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Abstract:

A systematic review of the diagnosis of dental caries was produced before the conference. It did not include the diagnosis of secondary or recurrent caries. This was a wise decision because what little literature exists on the subject potentially clouds the issue. Diagnosis is a mental resting place on the way to a treatment decision. A vital part of caries diagnosis is to decide whether a lesion is active and rapidly progressing or already arrested. This information is essential to plan logical management. However, lesion activity should be judged in the patient. Thus, research on the diagnosis of secondary caries must be carried out *in vivo* and this usually precludes histological validation. Even if such validation is possible it has its own problems, particularly distinguishing recurrent from residual caries. The diagnosis of secondary caries is very important since so many restorations are replaced because dentists think there is a new decay. It will be important to establish valid criteria for the diagnosis of active secondary caries, which will be facilitated by the suggestion that secondary caries is no different from primary caries except that it occurs next to a filling. This implies that it can be seen clinically and on a radiograph, next to a restoration.

Keywords: secondary caries, recurrent caries, diagnosis

The given assignment for this discussion paper was to address the RTI/UNC findings regarding the Diagnosis of Secondary Caries and translate the report into recommendations for research, clinical practice and education. While the report addressed the diagnosis of primary caries, it did not investigate the diagnosis of secondary caries. Thus there are no findings. It was sensible to exclude secondary caries because:

- the minimal literature on the subject potentially clouds the issue;
- the definition of secondary caries is in doubt; and
- there is no appropriate way to validate the diagnosis.

DEFINITIONS OF DENTAL CARIES AND DIAGNOSIS

Before justifying these statements it is sensible to step back to define what is meant by dental caries and by diagnosis. Dental caries is a process resulting from the microbial deposits covering the tooth surface at any given site. The metabolic processes in the biofilm are a physiological phenomenon and, at the crystal level, caries is an ubiquitous, natural, phenomenon. Mineral loss and subsequent cavity formation is a result of imbalance in the dynamic equilibrium between tooth mineral and plaque fluid. The carious lesion reflects the activity of the biofilm and lesion progression can be controlled.[1]

Diagnosis is a mental resting place on the way to a treatment decision; as such caries diagnosis implies deciding whether a lesion is active, progressing rapidly or slowly, or already arrested. Without this information a logical decision about treatment is impossible.

The report produced on the diagnosis of primary caries concerns mainly the detection of demineralization; there is little mention of lesion activity. However, as Featherstone made

clear in 1996,[2] both detection of demineralization and an appreciation of lesion activity are required for caries diagnosis. The RTI/UNC report applied histological validation as the appropriate gold standard for diagnostic studies. Inevitably most of these studies were done in the laboratory on extracted teeth of unknown clinical history. However, it is difficult to judge lesion activity histologically and unwise to attempt diagnosis (as opposed to lesion detection) in a laboratory simulation of a clinical setting. Diagnosis requires the warm human being and a clinical nose!

QUESTIONS RELEVANT TO SECONDARY CARIES DIAGNOSIS

The following questions are important:

- What is secondary caries?
- Why is it important?
- Where does it occur and why?
- What does it look like?
- What does it not look like?
- What are the problems in validating the diagnosis?

What is Secondary Caries?

Secondary caries is the lesion at the margin of an existing restoration. It is primary caries at the margin of an existing filling.[3] This definition has been confused for many years by those working only in the laboratory.[4] In this setting histological examination of artificial, caries-like lesions and natural lesions around restorations may show lines of demineralized tissue running along the cavity wall. These are called “wall lesions” and they are the result of microleakage. They are very commonly seen on histological examination of natural teeth with occlusal amalgam restorations. The “wall lesions” probably represent initial leakage that

occurred prior to sealing of the filling margins with corrosion products.[5] Secondary caries (i.e. primary caries next to the filling) is rare on this occlusal surface because the margin of the filling is cleansable.

As well as not confusing secondary caries with histological signs of microleakage, it is also important not to confuse secondary caries with residual caries. This is residual demineralized tissue left during cavity preparation. Our thoughts on how much demineralized tissue may be left during cavity preparation should have been profoundly shaken by the careful clinical studies of the Mertz-Fairhurst group.[6] This group simply removed the enamel lid from large occlusal lesions leaving extensively demineralized dentin. The cavities were then sealed with acid-etch composite restorations. Ten year results showed these restorations were satisfactory provided the patients did not escape to new dentists who took radiographs, noted demineralization (residual caries) and replaced the fillings. This study runs totally contrary to our conventional teaching in operative dentistry. We currently assume that the infected, demineralized dentin that is part of the carious lesion must be removed in order to arrest the caries process. Yet we now have this remarkable, controlled, ten-year study showing no deleterious effect in leaving the infected tissue in place. However, the results make sense if it is accepted that dental caries is the tissue destruction caused by bacterial metabolism in the biofilm. If the process is arrestable by simply removing the biofilm, then why do the symptoms of the process (demineralized dentin) have to be removed at all? Why not just remove the biofilm and seal the hole in the tooth so that the patient can clean? This study and this argument have profound implications for operative dentistry and for the validation of a diagnosis of secondary caries. Imagine extracting these teeth and examining them histologically. A microscopist who did not know the clinical history of the

tooth could erroneously assume the carious tissue was active, secondary caries. It would in fact be inactive, residual caries.

Why is the Diagnosis of Secondary Caries Important?

This diagnosis is the main reason given by dentists for replacing fillings; 50-60 percent of restorations are replaced because dentists diagnose secondary caries.[3] Are they correct? It is thought provoking that this high prevalence is not found in controlled clinical trials where 1-4 percent of secondary caries has been reported.[3] Incidentally only these latter trials might survive the scrutiny of a systematic review on the causes of failure of restorations if the parameter for inclusion of the study were a randomized, controlled clinical trial. Why are there huge differences between secondary caries diagnosis in a general practice setting and in a clinical trial? Are the general practitioners poorly trained, idiosyncratic and ignorant about this diagnosis? This explanation seems dangerously facile and yet it is obvious that dentists need reliable and valid criteria with which to diagnose secondary caries.

Where Does Secondary Caries Occur and Why?

Secondary caries occurs in areas of plaque stagnation. For this reason the cervical margins of restorations are commonly affected.

What Does it Look Like?

If it is accepted that secondary caries is primary caries at the margin of a filling, it looks clinically and radiographically like primary caries.

What Does it Not Look Like?

There is some evidence from combined clinical and microbiological studies that ditching and staining around amalgam fillings[7] and staining around tooth colored restorations[8] are all poor predictors of active secondary caries. This, too, can be explained if it is accepted that secondary caries is primary caries at the margin of a filling and not microleakage (seen as a line of stain around a tooth-coloured filling) or residual caries (which may present as a grey, undermining discolouration next to the restoration). As for when a ditching around an amalgam is concerned, it should be remembered that this phenomenon is a feature of occlusal restorations. This surface is not where secondary caries usually occurs because once the filling has been placed, this is not generally a plaque stagnation area. In other words toothbrushing cleans plaque out of the ditch.[5]

What are the Problems in Validating the Diagnosis?

There are major difficulties validating the diagnosis of secondary caries. One study has examined freshly extracted teeth histologically and related lesions at the margins of fillings to the overlying plaque.[9] This work showed the carious lesion developing beneath the biofilm at the tooth surface. As has been pointed out, a pure laboratory study, without an in vivo component or examination of the plaque over the lesion, is inappropriate because it would be easy to confuse active secondary caries with old microleakage or residual caries.[10]

A clinical study, where a diagnosis is made and the restoration dissected out to allow clinical examination of the cavity beneath for soft, demineralized dentin, may be similarly fraught with dangers.[7,8] It would be too easy to confuse residual caries with secondary caries. Imagine dissecting out a Mertz-Fairhurst type restoration.[6] Soft, demineralized dentine would

be present beneath the filling, but this is residual caries, not primary caries at the margin of the restoration.

Similarly the clinical and microbiological studies referred to may oversimplify the problem.[7,8] There are now many studies showing that the microbiological load in infected dentin is reduced when it is sealed off from the oral environment.[11-16] However, it is not eliminated. The relevance of these residual organisms is not clear. If Mertz-Fairhurst's work[6] is to be believed they have no relevance.

The only valid test is the visual appearance of lesions in teeth of patients. However, these appearances are open to interpretation and the authors of the RTI/UNC report would probably have dismissed this as poor and insufficient evidence.

RECOMMENDATIONS FOR RESEARCH, CLINICAL PRACTICE AND EDUCATION

Further clinical studies on the diagnosis of secondary caries are required. The working hypothesis should be that secondary caries is primary caries at the margin of a restoration. If this hypothesis is valid, the process should be arrestable by plaque control with a fluoridated dentifrice. This hypothesis should be tested. The work of Mertz-Fairhurst et al[6] should be repeated extending the study to approximal lesions. Only by conducting long-term, randomized, carefully controlled, clinical trials can the relevance of leaving infected dentin be assessed.

Clear guidelines relating to the clinical and radiographic appearances of secondary caries should be drawn up. Research studies should be initiated to see whether, using these

guidelines, dentists could become reproducible in their diagnostic decisions both with themselves (intra examiner reproducibility) and with other dentists (inter-examiner reproducibility).

These clinical guidelines should be introduced into undergraduate education. The students and their teachers should strive for consistency of diagnosis, remembering that diagnosis implies both lesion detection and assessment of activity.

Textbooks of operative dentistry should discuss what secondary caries is and how it should be diagnosed. Currently very little guidance is given, which simply is not good enough when dentists are spending so much time replacing fillings, often because they consider secondary caries to be the reason for the replacement.

Finally, an understanding of the disease process for dental caries should be the bedrock of all teaching in operative dentistry. The mechanics of filling and refilling teeth are but a part of the story!

REFERENCES

1. Fejerskov O. Concepts of dental caries and their consequences for understanding the disease. Community Dent Oral Epidemiol 1997;25:5-12.

2. Featherstone JDB. Clinical implications: new strategies for caries prevention. In: Proceedings of the 1st Annual Indianna Conference. Early Detection of Dental Caries. Ed Stookey GK. p287-295. Indianna University, 1996.

3. Mjör IA, Toffenetti F. Secondary caries: a literature review with case reports. Quintessence Int 2000;31:165-79.

4. Kidd EAM, Toffenetti F, Mjör IA. Secondary caries. Int Dent J 1992;42:127 -38.

5. Kidd EAM, O'Hara JW. The caries status of occlusal amalgam restorations with marginal defects. J Dent Res 1990;69:1275-7.

6. Mertz-Fairhurst E, Curtis JW, Engle JW, Rueggeberg FA. Ultra-conservative and cariostatic sealed restorations: results at year 10. J Am Dent Assoc 1998;129:55-66.

7. Kidd EAM, Joyston-Bechal S, Beighton D. Marginal ditching and staining as a predictor of secondary caries around amalgam restorations: a clinical and microbiological study. J Dent Res 1995;75:1206-11.

8. Kidd EAM, Beighton D. Prediction of secondary caries around tooth-coloured restorations: a clinical and microbiological study. J Dent Res 1996;75:1942-6.

9. Özer L. The relationship between gap size, microbial accumulation and the structural features of natural caries in extracted teeth with class II amalgam restorations (thesis). University of Copenhagen, 1997.

10. Merrett MCW, Elderton RJ. An in vitro study of restorative dental treatment decisions and dental caries. Br Dent J 1984;157:128-33.

11. Schouboe T, MacDonald JB. Prolonged viability of organisms sealed in dentinal caries. Arch Oral Biol 1962;7:525-6.

12. King JB, Crawford JJ, Lindahl RL. Indirect pulp capping: a bacteriologic study of deep carious dentine in human teeth. Oral Surg Oral Med Oral Pathol 1965;20:663-71.

13. Mertz-Fairhurst EJ, Schuster GS, Williams JE, Fairhurst CW. Clinical progress of sealed and unsealed caries. Part 1: Depth changes and bacterial counts. J Prosthet Dent 1979;42:521-6.

14. Handelman SL. Therapeutic use of sealants for incipient or early carious lesions in young adults. Proc Finn Dent Soc 1991;87:467-75.

15. Björndal L, Larsen T, Thylstrup A. A clinical and microbiological study of deep carious lesions during stepwise excavation using long treatment intervals. Caries Res 1997;31:411-7.

16. Weerheijm KL, Kreulen CM, de Soet JJ, Groen HJ, van Amerongen WE. Bacterial counts in carious dentine under restorations; 2-year in vivo effects. Caries Res 1999;33:130-4.