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A Prospective Study of the Prevalence of Periapical Pathology in Severely Worn Teeth

Abstract: The aim of this article was to carry out a prospective audit on a convenience sample of all new patients referred to the toothwear clinic at Cardiff University Dental Hospital to establish the prevalence of undiagnosed periapical pathology.

Clinical Relevance: The low prevalence value for undiagnosed periapical pathology in patients with advanced toothwear suggests that, for many patients, toothwear is a slow process which allows the defence mechanisms of the pulp to counteract the effects of wear. It also questions the necessity of taking routine radiographs of teeth with wear into dentine, in the absence of clinical symptoms. This would reduce the total radiation dose delivered to the patient and preserve valuable healthcare resources.

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Toothwear is a clinical problem that is frequently encountered in dental practice and prevalence values in adult patients of up to 82% have been reported.[1] The most recent Adult Dental Health Survey (1998) reported that 11% of adults had anterior toothwear with extensive involvement of dentine.[2] Toothwear is thought to be multifactorial in nature and may be caused by a combination of attrition, abrasion and erosion.[3] Attrition is caused by tooth to tooth contact, both occlusally and interproximally, and is often associated with tooth clenching and grinding habits. Abrasion is caused by abrasive particles introduced into the mouth as part of the diet. A good example is the abrasive particles found in toothpaste.

Dental erosion may be defined as an irreversible loss of dental hard tissues due to a chemical process without the involvement of micro-organisms.[4] This process may be caused by either extrinsic or intrinsic agents. Extrinsic agents include:

- Acidic substances;
- Beverages (Figure 1);
- Snacks; or
- Environmental exposure to acidic agents.[5,6]

Intrinsic causes of erosion include:

- Recurrent vomiting as part of anorexia or bulimia; or
- The regurgitation of the gastric contents.[7]

Once the outer protective enamel is lost, teeth may become hypersensitive as the dentine is connected to pulpal nociceptors via tubular fluid.[8] It is also known that dentine wears at least twice as fast as enamel owing to its lower mineral content.[3] Following tooth eruption, the pulp dentine complex will lay down secondary dentine as an ongoing process.

Figure 1. Palatal tooth surface loss caused by 'swishing' carbonated drinks in a 21-year-old male. Note near pulp exposures.

Dentine that becomes exposed as part of the wear process will initiate a reactive response, causing the laying down of tertiary (reparative or reactionary) dentine and the sealing of dentinal tubules and the formation of dead tracts. However, if the wear process is aggressive enough, the reactive responses of the pulp may be overwhelmed, leading to frank pulpal exposure or necrosis, necessitating root canal treatment. Indeed, pulpal exposure beneath labial cervical erosion lesions

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Score	Surface	Clinical criteria
0	B/L/O/I	No loss of enamel surface characteristics
	C	No change in contour
1	B/L/O/I	Loss of enamel surface characteristics
	C	Minimal loss of contour
2	B/L/O	Loss of enamel exposing dentine for less than 1/3 of the surface
	I	Loss of enamel, just exposing dentine
	C	Defect less than 1mm deep
3	B/L/O	Loss of enamel exposing dentine for more than 1/3 of the surface
	I	Loss of enamel and substantial loss of dentine, but not exposing the pulp or secondary dentine
	C	Defect 1-2 mm deep
4	B/L/O	Complete loss of enamel, pulpal exposure or exposure of secondary dentine
	I	Pulp exposure or exposure of secondary dentine
	C	Defect more than 2mm deep, pulpal exposure or exposure of secondary dentine
B = buccal	L = lingual	O = occlusal	I = incisal	C = cervical

Table 1. Smith and Knight toothwear index.[12]

	Number	Mean age (years)	SD (years)
Total sample	54	48.2	15.48
Male	34	49.1	14.60
Female	20	46.7	16.69

Table 2. Sample demographics.

Sex	Age	Tooth	TWI
Female	40	UL5	4
Female	48	UL2	3
Female	52	UR2	3
Male	64	UR2	4
Male	68	LL6	4
Mean age = 54.4 years (11.52)			
Mean TWI = 3.60 (0.55)			

Table 3: Untreated apical pathology (n = 5).

has been reported in a case series of 14 patients.[9]

Furthermore, dentine has a tubular structure, with the tubules passing from the external tooth surface to the pulp. As the tubules have a small diameter of around 1 micron, each square millimeter of exposed dentine contains around 15,000 to 65,000 tubules. Once exposed, oral bacteria may infiltrate the exposed tubules and gradually migrate to the pulp. If they do so in sufficient numbers, they will overwhelm the pulp, eventually causing necrosis. Adriaens *et al*[10] have shown that this type of bacterial invasion occurs when root dentine becomes exposed as a result of periodontal disease. It seems reasonable to suggest that this may also happen in toothwear cases where dentine is exposed occlusally and cervically.

Owing to the possibility of pulpal necrosis in toothwear cases with exposed dentine, many clinicians routinely take periapical radiographs of worn teeth with exposed dentine to establish the apical status and assess bone levels.[11] This can be readily justified as it is known that the early diagnosis of an apical area, while it is still relatively small, leads to better long term clinical outcomes.[12]

The aim of this prospective clinical audit was to establish the prevalence of undiagnosed apical pathology in a group of patients with severe toothwear referred to a University Dental Hospital.

Methods

A convenience sample of all consecutive patients referred to the toothwear clinic at Cardiff University Dental Hospital between October 2008 and March 2009 was examined carefully for exposed dentine. The extent of the wear was scored by the same clinician (JSR) using the toothwear index (TWI) of Smith and Knight, which is summarized in Table 1. All teeth with wear extending into dentine had a periapical radiograph taken using the paralleling technique by a qualified radiographer. Digital radiographs were taken with a Heliodent DS digital x-ray set (Siemens, Sir William Siemens Square, Frimley, Camberley, Surrey, UK) using a digital phosphor plate and a matching phosphor plate sensor holder and centering device (KerrHawe SA, Via Strecce 4, PO Box 268, 6934 Bioggio, Switzerland). These radiographs were then examined using IMPAX Web 1000 (Agfa HealthCare UK Limited, Vantage West, Great West Road, Brentford, Middlesex, UK) on a 19 inch LCD monitor at x5 magnification. Any teeth with a periapical radiolucency present were vitality tested using electric pulp testing and ethyl chloride was used to confirm the diagnosis of apical periodontitis.

A total of 54 sequential patients (34 males and 20 females), with a mean age of 48 years, were examined between October 2008 and March 2009, and 523 teeth in total were examined as part of this audit. The demographics of the sample group are shown in Table 2.

Results

The results of the audit are

shown in Tables 3–5. Diagnosis of the precise aetiology of toothwear is often difficult as toothwear is usually multifactorial.[11] Within our small sample, the majority of patients under the age of 30 seemed to have wear caused predominantly by erosion, whereas in the older patients this was predominantly due to attrition. Surprisingly, none of the patients complained of pain or sensitivity.

Overall, out of 523 teeth radiographed, only five of the teeth showed undiagnosed apical pathology (Table 3). Three of these teeth were upper anterior teeth, one was a premolar and one a molar. This gave an overall prevalence figure of 0.96% for untreated periapical pathology.

Within this sample, four of the teeth with existing root fillings also showed evidence of apical pathology (Table 4). These were two anterior teeth, one premolar and one molar. One of these teeth had been crowned, but the remaining teeth all had TWI scores of 4. This gave an overall prevalence value of 0.76% for apical pathology associated with root-treated teeth.

In addition to these teeth with apical pathology, 20 teeth were found to be root-filled, with no evidence of apical pathology, giving an overall prevalence value of 3.8%. Of these teeth, the majority (16 out of 20) were anterior teeth, one was a premolar and one a molar tooth.

As far as the toothwear index (TWI) scores were concerned, 60 teeth had a TWI of 2, 277 had a TWI score of 3 and 149 had a TWI score of 4. The overall distribution of the TWI scores of 2, 3 and 4 are given in Figures 2–4. This clearly shows that most of the patients had scores of 3 or above and that most of the toothwear was concentrated in the anterior dentition.

Sex	Age	Tooth	TWI
Female	29	UR2	4
Male	53	LL6	4
Male	63	LR2	4
Male	63	LR4	Crown
Mean age = 52.0 years (16.04)			
Mean TWI = 4.0 (0.00)			

Table 4. Apical pathology associated with existing root filling.

Sex	Age	Tooth	TWI
Male	25	UR1, UL1	4, 4
Male	35	UL1	Crown
Male	37	UL4	2
Male	37	LL6	3
Female	39	UL3	2
Female	43	UR3, UL4	2, 2
Male	59	UL1	3
Male	59	LL3	3
Male	64	UR3, UL3, LL4	3, 3, 3
Female	65	UR3, UR2, UR1, UL1	4, 4, 4, 4
Female	69	UR2	2
Male	70	UR3, UL1	3, 3
Mean age = 50.2 years (15.67)			
Mean TWI = 3.05 (0.78)			

Table 5. Existing root filling with no apical pathology.

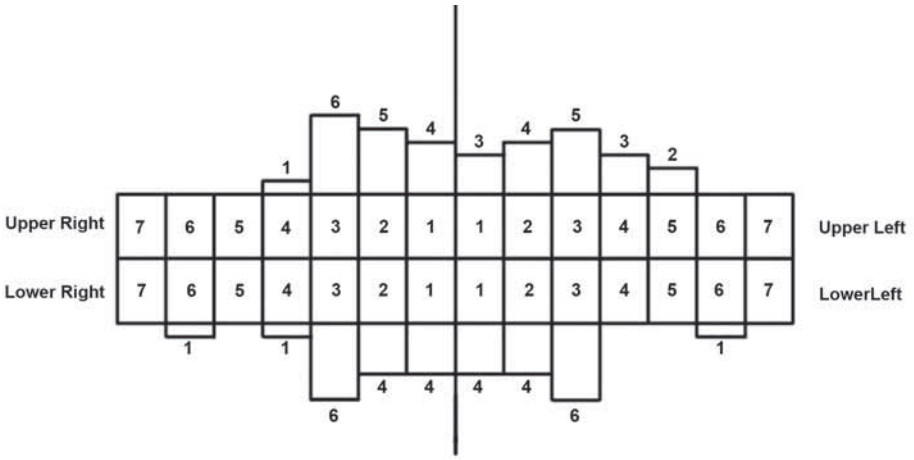

Figure 2. Intra-oral distribution of teeth with a score of 2.

Discussion

In severely worn teeth, the continued loss of hard tooth tissue may eventually result in pulpal involvement, either from direct pulpal exposure or by bacterial invasion of the dentinal tubules.[10] Meister *et al*[13] reported that reparative dentine is seen in most worn teeth, with evidence of calcification of the pulp chambers and a potentially increased risk of periapical pathology. The aim of this prospective clinical audit was to estimate the prevalence of undiagnosed periapical pathology in a cohort of patients referred for the management of severe toothwear.

Overall, five teeth in this sample had undiagnosed apical pathology, giving a prevalence rate of 0.96%. The only other

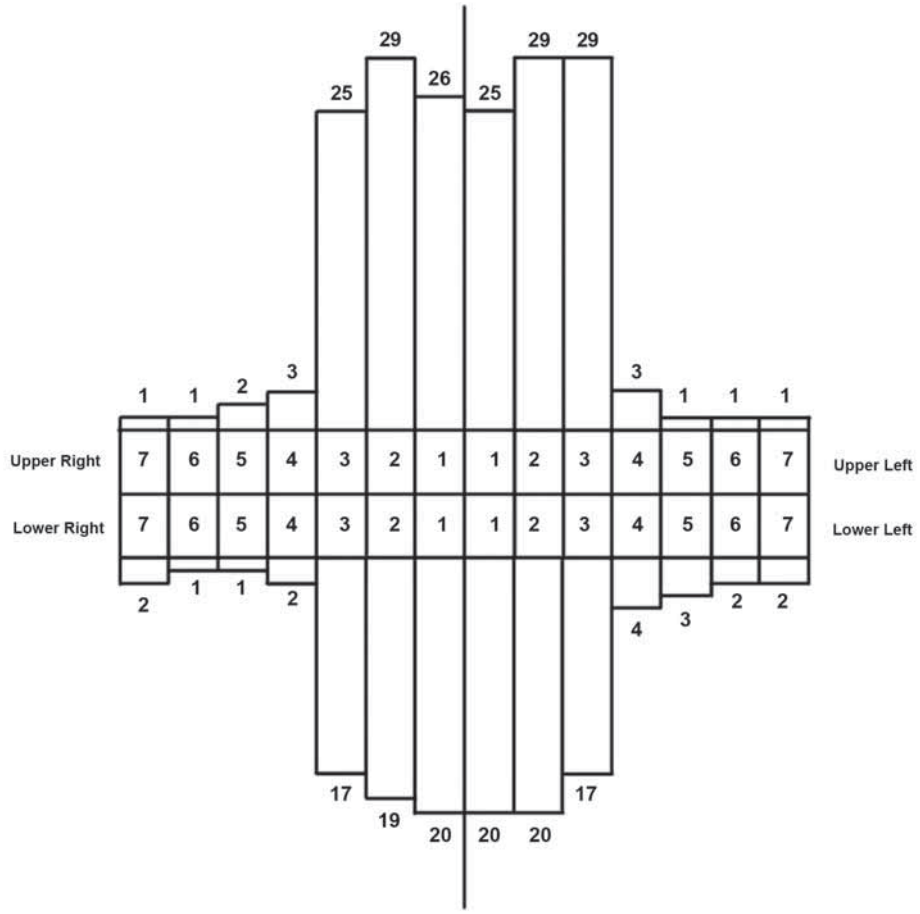

Figure 3. Intra-oral distribution of teeth with a score of 3.

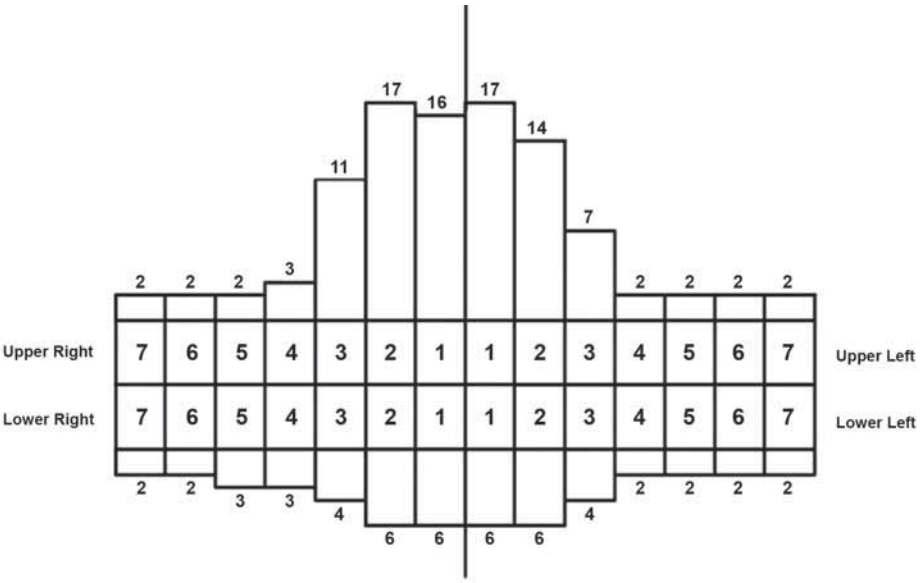

Figure 4. Intra-oral distribution of teeth with a score of 4.

study to examine this problem is that of Sivasithamparam *et al* [14] who reported a prevalence value of 11.6%. Unfortunately, they combined teeth with a near pulpal exposure, a frank pulpal exposure and root-filled teeth, rather than reporting separate prevalence values. The majority of the teeth with undiagnosed apical pathology in the present study were anterior teeth, which is similar to the study of Sivasithamparam *et al,*[14] who reported that mainly maxillary anterior teeth were affected. It seems likely that this finding relates to the site specificity of erosion. The protective effects of the saliva and pellicle in dental erosion are well reported.[3,6] Saliva, owing to its buffering and flushing action, is considered to be the most important protective factor.[15] The palatal surfaces of the maxillary anterior teeth are more severely affected by acidic attack (Figure 1), since there are no salivary glands in the anterior hard palate and these sites are relatively unprotected by saliva. Furthermore, a recent study has shown that erosion-abrasion caused by the dorsum of the tongue may also contribute to this pattern of tooth surface loss.[16]

This particularly low prevalence value of 0.96% reported here was much lower than we initially suspected. However, some recent studies have confirmed that toothwear for many patients is generally a slow process.[17] Therefore, for the majority of patients, the protective mechanisms within the dentine-pulp complex, particularly the laying down of secondary or tertiary dentine, is able to exceed the rate of dentine loss caused by toothwear, which results in few cases of total pulpal necrosis reflected by the low prevalence value reported here. Furthermore, toothwear does not usually occur in isolation and it is quite likely that, in some patients, previous caries may have contributed to pulpal necrosis.

The low prevalence value of undiagnosed apical pathology also calls into question the need to take periapical radiographs routinely for all worn teeth in the absence of clinical signs or symptoms. However, if worn teeth are to be restored with full coverage crowns, many guidelines suggest that a pre-operative radiograph is undertaken.[18-20]

The total number of teeth in this sample which were root-filled (Tables 4 and 5) was also small, with 24 out of 523 teeth (4.6%) having been

root-filled. This was particularly surprising, as the majority of the patients had Smith and Knight toothwear scores of 3 or 4, indicating that many of the patients had significant amounts of dentine exposed to the oral environment. One explanation for this could be that the dentine-pulp complex is particularly efficient in sealing off exposed dentinal tubules, thereby denying access to oral plaque bacteria. It has been known for many years that bacteria are the key aetiological agent in pulpitis, pulpal necrosis and apical periodontitis.[21]

The number of root-filled teeth with persistent periapical periodontitis was also examined. Overall, 16.7% had evidence of periapical pathology associated with a root-filled tooth, although the absolute number was small. This finding is consistent with many cross-sectional studies on the periapical status of endodontically treated teeth that show, on average, 30% of root-filled teeth have co-existing apical pathology.[22] In a more recent review of the literature, Wu *et al*[23] reported that radiographic evidence of periapical pathology was present in 40–50% of teeth. Most of these referred patients had at least one tooth surface with Smith and Knight TWI scores of 3 or 4 (Figures 3, 4), meaning that significant amounts of dentine had been exposed. Bartlett,[17] in his retrospective study, found that 5% of surfaces had a TWI score of 3 or 4. In the present study, the values were slightly higher; 11.7% of surfaces had a TWI score of 3 and 5.6% of surfaces had a TWI score of 4. Therefore, the sample examined here may well represent a 'worst case scenario', which is not too surprising as they were patients with severe wear referred to a special regional clinic.

Conclusions

This study found that 99% of teeth with severe toothwear did not have apical pathology. This low prevalence value suggests that, for many patients, toothwear is a slow process, which allows the defence mechanisms of the pulp to counteract the effects of wear. It also questions the necessity of taking routine radiographs of teeth with wear into dentine in the absence of clinical symptoms. This will reduce the total radiation dose delivered to the patient and preserve valuable healthcare resources.

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CPD ANSWERS

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1. A, B, C
2. D
3. A, B, D
4. A, C
5. A, B, D
6. A, C
7. B, C
8. B, D
9. A, B, D
10. A, C, D