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Histological comparison of pulpal inflammation in primary teeth with occlusal or proximal caries

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Objective. A number of clinical and histological studies have investigated caries-related changes in the primary tooth pulp, but the effect of caries site, as a clinical variable, has not been previously considered. This study sought to compare inflammatory changes within the pulp of primary molars according to the location of the caries lesion (occlusal or proximal).

Methods. Eighty-three primary molars were extracted under general anaesthesia for caries and/or orthodontic reasons, and were split immediately after removal and fixed in 10% formalin. Teeth were then decalcified, sectioned, and stained with haematoxylin and eosin for histological examination using light microscopy. Caries depth was measured using a graticle, and the site of the caries lesion noted as occlusal or proximal. Samples were further classified into one of five subgroups according to the observed degree of pulpal inflammation.

Results. Key findings were that where caries depth was less than 50% of the total dentine thickness, there were no significant differences in inflammatory status according to caries site. In contrast, marked inflammatory changes were significantly more likely throughout the coronal pulp of teeth with proximal caries compared to teeth with occlusal caries where caries depth was equal to, or greater than, 50% of the total dentine thickness ($P = 0.017$, Fisher's exact test).

Conclusion. Primary teeth with proximal carious lesions extending more than 50% through the dentine thickness appear to have more extensive inflammatory pulpal changes than teeth with occlusal caries of a similar depth. This finding has clinical implications and may help inform treatment decisions in the management of primary teeth with deep carious lesions.

Introduction

Dental caries remains one of the most prevalent health problems for children in most industrialized countries[1]. It is increasingly acknowledged that the disease, and its related treatment, may place a considerable burden on the child and parents, with an overall negative effect on quality of life[2].

Managing dental decay, especially in young children, presents unique challenges to the clinician who has a responsibility to provide effective and evidence-based treatment for their paediatric patients. In clinical practice, a diverse range of therapeutic strategies and materials are available for treating carious primary teeth. There is, however, a paucity of high-quality evidence to support any particular intervention[3]. Treatment options such as indirect pulp capping, pulpotomy, pulpectomy, or extraction may be considered for primary molars with deep carious lesions[4]. The success of vital pulp treatment is largely dependent on an accurate assessment of pulp status. Unfortunately, diagnosis of pulp pathology can be difficult, especially in very young children where a detailed pain history and response to vitality testing may be less reliable[5]. Moreover, studies have failed to prove a strong correlation between histological inflammatory changes within the pulp and reported pain history, reactions to sensibility tests and percussion, or even radiographs[6–9]. Thus, findings from clinical tests should be interpreted with caution.

A few investigators have attempted to correlate the extent of the carious lesion with histopathological changes within the tooth pulp as a more reliable indicator of pulp status. It is generally acknowledged that advanced caries progression,

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involving more than half the dentine thickness, is associated with a variable degree of pulpal inflammation[8,10–12]. Furthermore, widespread inflammatory changes have been reported in carious primary molars with marginal ridge breakdown[7,11,13]. Duggal and colleagues demonstrated that pulp inflammation may actually occur at an early stage of proximal caries attack, and once proximal caries involvement presents clinically with marginal ridge breakdown, pulp inflammation is usually extensive[13].

To date, it would appear that only caries depth, and not site, has been explored as a predictor of pulpal reactions. Previous investigators have preferentially included either teeth with occlusal caries or teeth with proximal caries, but not both. Furthermore, in some cases, the location of the lesion has not actually been reported[8,10–12]. Clinical studies comparing outcomes of different pulp therapies have also been restricted to either teeth with occlusal lesions or teeth with proximal lesions: no doubt in an attempt to limit the number of clinical variables[14,15]. Clinical decision-making for the compromised primary dentition should incorporate a range of patient- and tooth-related variables. Knowledge of any potential differences in caries-induced pulpal inflammation in teeth with occlusal or proximal lesions would be helpful in prescribing the most appropriate treatment strategy.

The aim of this study, therefore, was to determine whether there are any differences in pulpal inflammation between primary teeth with occlusal or proximal carious lesions.

Materials and methods

The study was conducted within the Departments of Paediatric Dentistry and of Oral Biology, Leeds Dental Institute, UK. Ethical approval was granted by Leeds (West) Research Ethics Committee, and informed consent was obtained from legal guardians to allow the use of their child's extracted teeth for the specific purposes of this research.

Experimental material

Maxillary and mandibular first and second primary molars comprised the experimental material for the study. Teeth were obtained from fit and healthy children who required routine dental extractions under general anaesthesia (GA) at the day-care unit of Leeds Dental Institute, UK. Treatment plans were prescribed at a pre-GA assessment by a consultant paediatric dentist. Inclusion criteria were as follows: (i) teeth were from healthy children with no relevant medical condition that could affect pulp histology[13]; (ii) teeth had either an occlusal lesion or a proximal lesion (mesial or distal surface) involving only one proximal ridge; (iii) the tooth was restorable; (iv) there was no clinical or radiographic evidence of irreversible pulpitis or periapical pathology; and (v) teeth were from children under the age of 6 years to ensure that there was no appreciable physiological root resorption in view of previous reports of inflammatory change with exfoliation[16].

Tissue preparation

Immediately following forceps extraction, teeth were split longitudinally through the carious lesion, either mesio-distally for teeth with an occlusal lesion or bucco-lingually for teeth with a proximal lesion (Fig. 1). Visual inspection of the carious lesion was undertaken to confirm that the inclusion criteria were satisfied. Tooth halves were then fixed in 10% formalin for at least 10 days. The teeth were subsequently

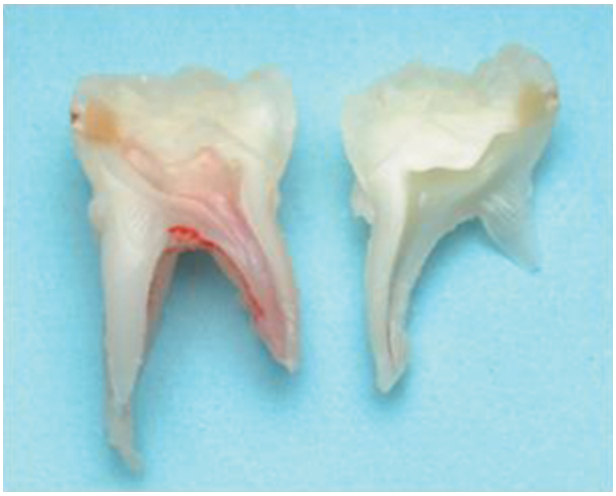

Fig. 1. Primary molar following longitudinal split through proximal caries lesion and pulp tissue.

immersed in 10% ethylenediaminetetraacetic acid solution for approximately 4 weeks until full decalcification, confirmed radiographically, had taken place.

Following decalcification, teeth were subjected to alcohol dehydration and embedded in paraffin wax. Serial sections, of 3–5 μm thickness, were then cut on a microtome through the deepest part of the carious lesion. Sections were stained with haematoxylin and eosin for histological examination under the light microscope.

Caries depth

Caries depth was measured as percentage of the whole dentine thickness. The measurements were done with graticule under light microscope using the ×20 objective. Firstly, the distance from the outer surface of the tooth to the deepest aspect of the carious lesion was measured in millimetres and recorded as d^1. Secondly, the distance from the deepest aspect of the carious lesion to the outer aspect of the pulp chamber was measured and recorded as d^2. Caries depth was then calculated as a percentage of whole dentine thickness $[= 100 \times (d^1 \div \{d^1 + d^2)]$. Caries depth measurements were made by one examiner (D.K.), and were repeated on 10% of the sample 4 weeks after the first examination to determine intra-examiner reproducibility.

Degree of inflammation

Each tooth was classified into one of five subgroups, according to the most severe inflammatory changes observed within any of the pulp sections[13].

1) Class I – normal pulp architecture with no inflammatory changes: In this category, no inflammatory changes were observed in the dentine–pulp complex, evidenced by the absence of reparative dentine, a normal odontoblast layer, and cell-free zone.

2) Class II – inflammatory changes limited to the odontoblast layer: Localized changes were only observed within the odontoblast layer and did not extend to the cell-free zone. Key characteristics included a thin layer of reparative dentine and a normal, or slightly disturbed, odontoblast cell layer.

3) Class III – inflammatory changes limited to pulp region sub-adjacent to the carious lesion: Inflammatory changes extended beyond the odontoblast layer and the cell-free zone, but were confined to the pulp region sub-adjacent to the carious lesion. Features here included: thick or discontinuous reparative dentine, absent or necrotic odontoblast cells, a poorly defined cell-free zone with signs of hyperaemia, fibrosis, or mild to moderate inflammatory cell infiltrate.

4) Class IV – inflammatory changes limited to the coronal pulp: In this category, changes described earlier extended into the coronal pulp but spared the radicular pulp. A profound inflammatory cell infiltrate was observed within the coronal pulp tissue.

5) Class V – inflammatory changes within both the coronal and radicular pulp: This category reflected the most advanced degree of pulpal inflammation: all the above changes were seen in addition to the presence of an inflammatory reaction within the radicular pulp tissue.

The assessment of inflammation was made by one examiner (D.K.) and was repeated on 10% of the sample 4 weeks after the first examination to determine intra-examiner reproducibility.

Statistical analysis

Fisher's exact test was used to determine whether there was any difference in the frequency distribution of inflammation subgroups (1-V) according to caries site (occlusal and proximal). The level of significance was set at $P < 0.05$.

Results

A total of 83 upper and lower primary molars, collected from 52 children, were subject to analysis. Forty-eight teeth had occlusal caries and 35 had proximal caries.

Caries depth

Mean caries depth, as a percentage of the total hard tissue thickness, was 59.5% (SD = 20.69, 9–96.8) for teeth with occlusal lesions and 63.2% (SD = 24.14, range = 9–95) for teeth

Table 1. Distribution of teeth in each inflammatory class according to caries site (occlusal or proximal).

Inflammatory class	Caries site	
	Occlusal n (%)	Proximal n (%)
Class I: normal pulp	3 (6.3)	1 (3.0)
Class II: inflammatory changes limited to the odontoblast layer	20 (41.7)	8 (22.8)
Class III: inflammatory changes limited to pulp horn at caries site	18 (37.5)	13 (37.1)
Class IV: inflammatory changes limited to coronal pulp	6 (12.5)	13 (37.1)
Class V: widespread inflammatory changes extending to radicular pulp	1 (2.0)	0 (0.0)
Total	**48**	**35**

with proximal lesions. An independent *t*-test confirmed that there was no statistically significant difference in caries depth between the two experimental groups ($P < 0.05$). There was good intra-examiner repeatability for caries depth measurement: the mean difference (bias) found between the initial and repeat caries depth measurements was 1.25% with a 95% confidence between –3.35 and 5.85.

Inflammatory change according to caries site

Table 1 shows the overall distribution of the 83 samples within the five different inflammation classes according to the presence of occlusal or proximal caries. It can be readily seen that the greatest proportion of teeth with occlusal caries (41.7%) was categorized as class II, having inflammatory changes limited to the odontoblast cell layer. This was in marked contrast to the proximal caries group, where the majority of teeth (31.7%) were found to have inflammatory changes within the pulp horn (class III) or the coronal pulp tissue (class IV). Only one tooth sample (with occlusal caries) had evidence of radicular inflammation, thus no further statistical analysis could be undertaken for this category (see Fig. 2 for histological findings).

For each inflammation subgroup, Fisher's exact test and an odds ratio (OR) were calculated to determine any statistically significant differences according to caries site. For samples with a normal pulp (class I), inflammatory changes extending to the odontoblast layer (class II) and inflammatory changes extending to the pulp tissue directly below the caries lesion (class III), there were no statistically significant differences according to caries site ($P = 0.64$, 0.10, and 1.0, respectively). Teeth assessed as having class I, II, or III inflammation were, however, more likely to have occlusal than proximal caries (OR = 2.27, 2.4, and 1.02, respectively).

There was, however, a statistically significant difference in the proportion of teeth with inflammatory changes extending into the coronal pulp (class IV) according to caries site ($P = 0.016$, Fisher's test). Teeth with generalized coronal pulp inflammation were four times as likely to have proximal caries than occlusal caries (OR = 4.13).

There was a 100% agreement in categorization of inflammation class at the initial and repeat assessment, which demonstrated optimal intra-examiner reliability.

Inflammatory change according to caries site and depth

In order to gain further insight into the relationship between specific characteristics of the caries lesion and the associated degree of pulpal inflammation, occlusal and proximal samples were further subdivided according to caries depth Teeth were categorized as having a caries depth of: (i) less than; or (ii) greater or equal to 50%, of the total dentine thickness. Interestingly, when caries depth was less than 50% of the total dentine thickness, there were no statistically significant differences in the distribution of inflammatory subgroups between teeth with occlusal and proximal caries ($P = 0.824$, Fisher's test). In contrast, when caries depth was greater or equal to 50% of the total dentine thickness, there was a statistically significant difference in the categorization of inflammation between teeth with occlusal and proximal caries ($P = 0.017$, Fisher's test). Notably,

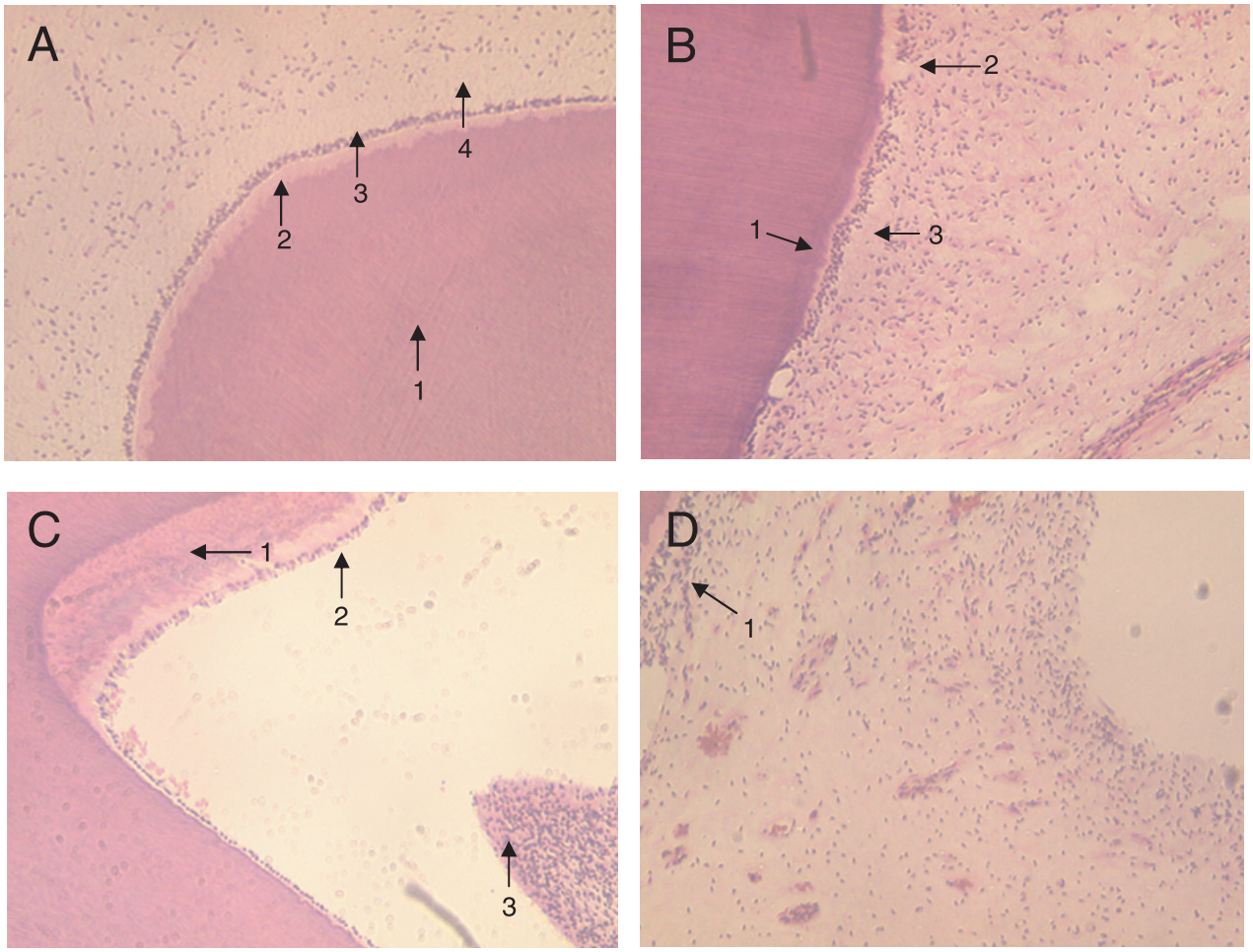

Fig. 2. Haematoxylin and eosin stained demineralised sections of extracted carious primary molars showing different degrees of inflammatory change. (A) Normal dentine-pulp (class I) showing: 1. dentine; 2. predentin layer; 3. odontoblast cell layer; 4. cell free zone (×25). (B) Histological features and characteristics of class II inflammatory changes in the area adjacent the caries site: 1. thin reparative dentine; 2. abnormal odontoblast cell layer; 3. normal cell free zone (×25). (C) Histological features near the carious lesion in tooth section with class III inflammatory changes: 1. thick layer of reparative dentine; 2. abnormal odontoblast cell layer; 3. undistinguished cell free zone layer with signs of fibrosis and moderate inflammatory cell infiltration (×25). (D) Histological features of Class IV inflammatory changes showing: 1. poorly distinguished cell free zone layer with signs of hyperaemia, fibrosis, and moderate to severe inflammatory cell infiltration at some distance from the caries site (×25).

a significantly greater proportion of teeth with proximal caries had generalized inflammation in the coronal pulp (class IV) than was the case for samples with occlusal caries.

Discussion

Strict inclusion criteria were adopted for the experimental material to ensure that findings would be as clinically relevant as possible. Teeth with a history of spontaneous or persistent pain, an associated sinus/swelling, or furcation radiolucency were excluded, as these teeth would be expected to have an irreversibly inflamed or non-vital pulp, and hence a different treatment approach (pulpectomy or extraction) would be indicated. It is worth noting that only one sample in this study was found to have inflammatory changes within the radicular pulp, indicative of an irreversible pulpitis, thus validating the study's clinical and radiological selection criteria.

The purpose of this study was to gain an appreciation of the likely degree of pulpal inflammation in potentially restorable teeth with either occlusal or proximal caries, where

an indirect pulp capping or vital pulpotomy would normally be considered. It is acknowledged that the study design was limited by only including teeth with caries involving a single surface (occlusal or proximal). This was to ensure that pulpal histology could be reliably related to a specific carious site. Further research would be warranted to explore caries-induced pulpal reactions in response to multisurface lesions. In addition, it is recognized that, although this study considered both caries site and depth, other caries-related variables may also have a profound effect on overall pulpal response. The activity of the caries lesion, for example, may be a very important factor, which has not been previously correlated with the degree of pulpal response in the primary dentition.

At this point, the availability of asymptomatic and potentially restorable primary teeth for the purposes of this study needs explanation. Firstly, for ethical reasons, treatment plans were prescribed at a pre-GA assessment by paediatric dentists not involved in the study itself. Secondly, children receiving dental care under GA at Leeds Dental Institute are typically young, uncooperative, irregular attenders, with a high caries risk. For this patient group, the avoidance of a repeat GA for further dental treatment is of paramount importance in treatment planning. Thus, on an extraction-only list, all carious primary teeth may be extracted. In addition, minimally carious, or indeed intact, first primary molars may be extracted to balance the extraction of a contralateral grossly carious molar, to prevent future centre line shift[17]. Therefore, extracted teeth with moderate caries lesions were readily available for laboratory investigation.

Duggal and colleagues have previously investigated caries-related pulpal inflammation in the primary tooth, and have suggested a classification in which inflammatory changes in each layer of the pulp are studied[13]. This approach was also adopted by this study as it is based on objective and reproducible observations and, importantly, provides clinically relevant data with respect to the extent of inflammatory spread throughout the entire pulp. It should be pointed out that the current methodology is unable to identify whether the observed pulpal inflammation is of a reversible or irreversible nature.

A fundamental step in this study was to ensure there was no significant difference in mean caries depth between teeth with occlusal and proximal caries as it has been well-established that pulpal inflammation increases with increasing caries depth[18–20]. As no significant difference was found in mean caries depth between the two experimental groups, differences in inflammation could be more reliably attributed to the effect of the caries site itself. This study found that when caries extended 50% or more within the total dentine thickness, a greater proportion of teeth with proximal lesions showed severe inflammatory changes than was found for teeth with occlusal lesions. This was not the case for teeth with < 50% caries progression, where there was no difference in inflammatory response according to the caries site. There may be a number of explanations for this fascinating difference. Firstly, overall dentine thickness is less over the pulp horn region than on the occlusal surface, as primary teeth have very prominent pulp horns. Thus, for a proximal lesion extending 50% through the hard tissue thickness, potential irritants are actually closer to the pulp tissue than is the case for an occlusal lesion which has involved 50% of the total dentine thickness[5,11]. Numerous previous studies have considered pulpal responses in relation to the percentage caries involvement of the total hard tissue thickness[7,8,11,20]. There is, however, growing consensus that the residual dentine thickness (the depth of unaffected hard tissue between the base of the caries lesion and the pulp periphery) may be the most predictive measure of likely pulpal reactions[21]. Thus, future studies may need to explore residual dentine thickness, rather than the proportion of total dentine thickness invloved, in relation to pulpal inflammation in primary teeth.

A number of different treatment strategies have been proposed for primary teeth with deep carious lesions. Indirect pulp capping and pulpotomy are considered the two main therapeutic options for asymptomatic primary molars with extensive caries but no clinical or radiographic evidence of an irreversible pulpitis[22,23]. Findings from previous histological investigations of carious primary teeth have been interpreted as supporting the indirect

pulp cap in preference to vital pulpotomy[10,24]. It should be noted, however, that these studies have involved either primary incisors[24] or primary molars with occlusal caries only[10]. The clinical implications of treating teeth with proximal lesions, which appear to undergo widespread inflammation at an earlier stage of caries progression, do not appear to have been fully appreciated. It is speculated that, in light of this study, teeth with proximal lesions of greater than more than half the dentine thickness may respond better to a vital pulp therapy approach than indirect pulp capping in view of their more extensive inflammatory reaction. This approach would ensure the removal of inflamed coronal pulp tissue leaving vital, and uninflamed radicular tissue, which would have the potential for healing and repair[23,25,26]. This theory would, however, require validation through appropriate clinical investigation.

Multiple studies have investigated the success of different restorative materials for occlusal or proximal caries in primary molars[27,28]. Although rarely do studies compare success of the same material at different sites. When comparison between studies are made, proximal restorations almost always show poorer outcomes[29,30]. Clinicians have frequently assumed this difference relates to the more technically demanding cavity preparation and restoration required for proximal lesions. This assumption may have been overly simplistic because it did not take into account any potential differences in the underlying pulpal status of teeth with similar caries penetration. This study has reinforced the need to use a randomized controlled trial design for restorative material studies or pulp therapy interventions, as only this methodology is able to randomly allocate subjects to different groups even when potential variables are unknown.

Conclusion

This study is the first to explore the effect of both caries site and depth on pulpal inflammation. Findings suggest that inflammatory reactions are more likely to extend into the coronal pulp tissue of teeth with proximal lesions compared to teeth with occlusal lesions, where the caries depth is greater than, or equal to, half the dentine thickness. Clinicians should therefore consider the effect of caries depth in treatment decisions for the compromised primary tooth. Furthermore, investigators should also consider caries site as a variable factor in future laboratory and clinical studies.

What this paper adds

- This study has provided a new insight into the effect of caries site on the degree of pulpal inflammation in carious primary molars.
- It has shown that teeth with a proximal caries lesion may experience more extensive pulpal inflammation than teeth with an occlusal lesion, when caries involvement is greater than 50% of the dentine thickness.

Why this paper is important to paediatric dentists

- Paediatric dentists routinely provide pulp therapies for primary teeth with different caries lesions. It is important that clinicians consider the potential effect of caries site, as well as depth, on pulpal status, in order to choose the most appropriate treatment approach.

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