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Evaluation and comparison of changes in microhardness of Infected and Affected Dentin following application of 38% Silver Diamine Fluoride.

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Abstract

Background: Dental caries is one of the most prevalent infectious diseases in children. Caries treatment in children is not easy, as it is difficult for less cooperative children to receive the proper dental treatment. SDF is a material that can reduce the tooth demineralization that occurs during the formation of caries, inhibit the growth of cariogenic multispecies biofilm and promote remineralisation. Measuring microhardness has indeed been considered to be a reasonable method of examining the mineral content of teeth.

Material & Method: The study will comprise of 20 carious deciduous teeth indicated for extraction. In Group I (10), SDF will be applied directly onto infected dentin.

In Group II (10), SDF will be applied onto affected dentin after removal of infected dentin using a hand instrument. Patients will be recalled after 24 hours for extraction. Microhardness of infected and affected dentin will be evaluated and compared for both groups.

Result: The average microhardness of the affected dentin group (38.8±3.2) was higher than the average microhardness of the infected dentin group (11±1.5). There was no significant difference in the microhardness between the *in vivo* and *in vitro* subgroups.

Conclusion: The results of this study indicates that 38% Silver Diamine Fluoride is effective in increasing the microhardness of the affected and infected carious dentin thereby arresting the caries progression.

Keywords: Silver Diamine Fluoride, Microhardness, Infected Dentin, Affected Dentin.

Introduction

Dental caries can be classified as a bacterial disease of the calcified tissues of the teeth. It is characterized by the demineralization of tooth's inorganic content and dissolution of its organic content[1].

Histologically, dentinal caries is classically described as consisting of two main layers. In the outer or ''infected'' layer, the dentin is heavily infected with bacteria, both organic matrix and mineral have been lost and the dentin is beyond repair. In the deeper or ''affected'' layer, the dentin is demineralized by plaque acids, but there is no bacterial invasion[1].

Measuring microhardness has indeed been considered to be a reasonable method of examining the mineral content of teeth and several studies of caries or arrested caries have shown that changes in the microhardness of dentin is directly related to its mineral content.[1]

High-concentration topical fluoride agents, such as 38% silver diamine fluoride (SDF) with 44,800 ppm fluoride, 5% sodium fluoride (NaF) varnish with 22,600 ppm fluoride and have been used to arrest caries[1].

Silver diamine fluoride, a chemical that is claimed to be more stable and can be kept in a constant concentration has been in use in many countries including China and Japan, to arrest dental caries for many years. SDF is not as alkaline (pH = 8-9) as AgF and also it does not require a reducing agent[2].

This study aims to measure and compare the changes in microhardness of infected and affected dentin of primary teeth after SDF application.

Aim: To evaluate and compare microhardness of infected and affected dentin following application of 38% Silver Diamine Fluoride.

Objective: Effect of SDF on microhardness of infected and affected dentin.

1. To evaluate the effect of saliva in increasing the microhardness of affected and infected dentin along with SDF.

Materials and Method: The study comprised of total 20 carious deciduous teeth that were indicated for extraction. These teeth were randomly divided into two groups containing 10 teeth each which were further divided into 2 subgroups.

Group I (Infected dentin group): This group consisted of 10 teeth in which SDF was applied on the infected dentin using a microbrush. Before application the tooth surface was dried with cotton roll and gentle flow of compressed air was used to dry the silver diamine fluoride liquid after application. Petroleum jelly was applied onto the surrounding gingival tissue and mucosa to avoid contact with SDF. Group I was further divided into 2 subgroups (in vivo and in vitro) containing 5 teeth in each.

- **In vivo group:** In this group SDF was applied directly on to the infected dentin soon after flushing of food debris with a water jet. The patient was recalled after

24 hours for extraction and the extracted tooth was placed in deionised water.

- **In vitro group:** In this group SDF was applied after extraction and the extracted tooth was placed in deionised water.

GROUP II (Affected dentin group): This group consisted of 10 teeth in which SDF was applied onto the affected dentin after excavation of infected dentin with hand instruments leaving 1 mm of sound dentin. Group II was further divided into 2 subgroups (in vivo and in vitro) containing 5 teeth in each:

- **In vivo:** Excavation of the infected dentin was done using hand instruments and burs with slow speed hand piece and SDF was applied on to the remaining affected dentin. The patient was recalled after 24 hours for extraction.
- **In vitro:** SDF was applied onto the affected dentin after extraction of the tooth and placed in deionised water.

Each tooth was then sectioned buccolingually and mounted on an acrylic base and was subjected to Vickers hardness tester. The mounted sample were placed under the Vickers indenter and microhardness was measured for each group.

Result

Data was analysed with SPSS version 22.0 for Windows. Nonparametric tests were used because, the samples in the affected and infected dentin group were small.

Mann–Whitney U-test was used to assess the microhardness between in vivo and in vitro groups of the affected and infected dentin which was statistically not significant with a p value of 0.66 and 0.11.(Table 1)

Student t test was used to assess the microhardness of infected and affected dentin which was statistically significant at 50µm and 100µm with a p value of 0.00 for each group. At 250µm p value for both the groups was statistically not significant ($p< 0.33$).(Table 2)

The average microhardness of the affected dentin group (38.8 ± 3.2) was higher than the average microhardness of the infected dentin group (11 ± 1.5). There was no significant difference in the microhardness between the *in vivo* and *in vitro* subgroups.

Discussion

Caries is a dynamic process of demineralization and remineralization that can lead to lesion formation in enamel and dentin. Fusayama et al. (1966)[3] classified carious dentin in to two layers: (1) the outer carious dentin (or caries-infected dentin), which is contaminated with bacteria and in which the collagen fibers are degraded and cannot be remineralized; and (2) the inner carious dentin (socalled caries-affected dentin), which is bacteria-free with limited denaturation of the collagen and which can be remineralized.

Caries treatment in children is not easy, as it is difficult for less cooperative children to receive the proper dental treatment.[4] The traditional conservative treatment of dental caries involves mechanical cavity preparation and restoration with suitable material. However, this kind of treatment requires clinical skills, costly instruments, materials, and patient's cooperation. In young children, behavioral issues or lack of cooperation often complicates this kind of traditional restorative treatment of carious tooth and often leads to disease progression and subsequent loss of tooth.[5]

Again, when a child is pre-co-operative or when care for a senior (such as one who may be in a long-term facility) is too difficult, or if psychological problems preclude a patient from tolerating more invasive care, SDF can arrest caries progression while keeping the patient comfortable. Similarly, if a patient's medical status precludes

comprehensive clinical dental intervention, SDF can dramatically improve quality of life.[6]

The usage of silver diamine fluoride to prevent tooth decay seems to be the most effective way of tackling the rising epidemic of dental caries. Topical application of silver diamine fluoride on exposed dentinal surface results in the formation of a squamous layer, partially plugging the dentinal tubules. Silver in silver diamine fluoride interacts with sulfhydryl groups of proteins and with deoxyribonucleic acid (DNA), altering hydrogen bonding and inhibiting respiratory processes, DNA unwinding, cell wall synthesis, and cell division. It has also been shown that silver diamine fluoride can prevent biofilm formation and this inhibition is quite prominent in the first 7 days after application. Silver diamine fluoride has also shown to have an inhibitory effect on matrix metalloproteinase and thus reduces the degradation of organic collagen matrix

It reacts with hydroxyapatite cystals to release calcium fluoride and silver phosphate, which are responsible for the prevention and hardening of dental caries. Ag_3PO_4 precipitates on tooth surface are insoluble and the CaF_2 acts as reservoir of fluoride for the formation of fluorapatite, which is more resistant to dissolution caused by various organic acids in oral environment than hydroxyapatite.[5]

Mei et al. (2013)[7], investigated the inhibitory effect of 38% silver diamine fluoride on demineralized dentin and found that it inhibited demineralization and preserved collagen from degradation in demineralized collagen in carious dentin thus positively promote dentin remineralisation and finally arrest dentin carious lesion.

Hardness is one of the parameters often used in the evaluation of demineralization and remineralization. Hardness has been used to reflect loss and gain of mineral.[8]

Yamaga et al. (1972)[9] in his study on Silver Diamine Fluoride and its Clinical Application attributed the increased hardness of dentin due to deposition of silver phosphate in SDF.

Firouzmandi M et al. (2019)[10] attributed that SDF treatment resulted in the greatest improvement in mechanical properties of carious dentin compared to other groups. This can be associated with increased mineral content. Increased hardness and increased elastic modulus (quantity that measures an object or substance's resistance to being deformed elastically when a stress is applied to it) were evident in the SDF group.

In the present study also microhardness of primary teeth was shown to be increased after application of silver diamine fluoride. The lesions showed areas of arrested caries and the increase in hardness compared to caries lesion. Micro-hardness measurement was done in the affected and the infected dentin which showed higher VHN values in the affected dentin group with a mean of 38.3 ± 2.9.

Aljabo A et al. (2016)[11] in his study, Demineralization–remineralization dynamics in teeth and bone stated that saliva acts as a source of calcium and phosphate ions that are required for remineralization of decalcified tooth.

In the present study, there was no significant difference observed between the in vivo and in vitro subgroups which suggests that the saliva does not provides any additional contribution in increasing the dentinal microhardness following 38% SDF application.

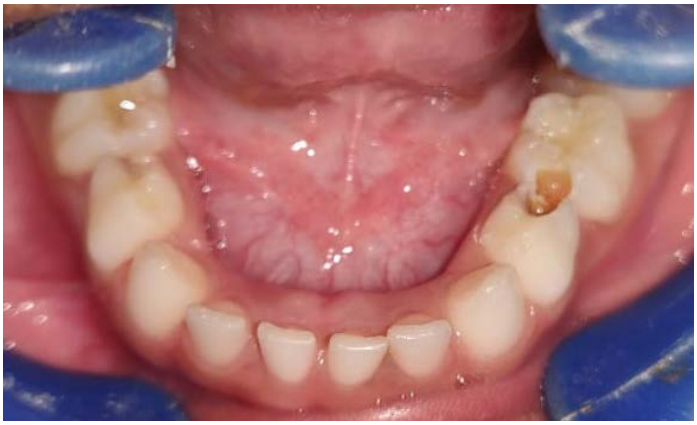


Figure 1: Excavation of the infected dentin

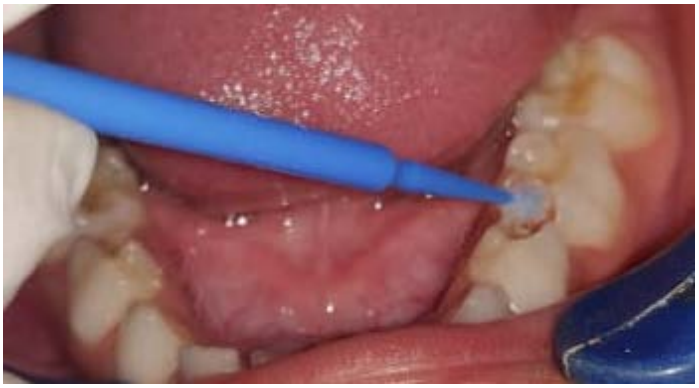


Figure 2: SDF applied on to the remaining affected dentin

Figure 3: SDF applied directly on to the infected dentin

Figure 4: Vickers hardness tester

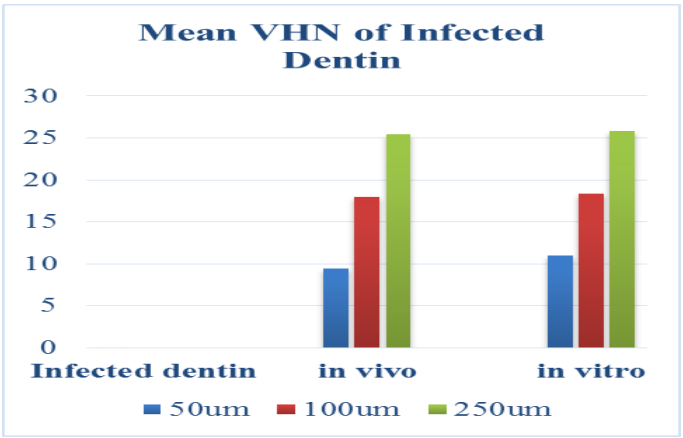
Table 1: Comparison of Microhardness in vivo and in vitro Infected dentin and Affected dentin

Variables	n(%)	Mean	p value
Infected dentin in vivo	5(25%)	9.4±1.14	0.11
In vitro	5(25%)	11±1.5	
Affected dentin in vivo	5(25%)	37.8±2.7	0.66
In vitro	5(25%)	38.8±3.2	

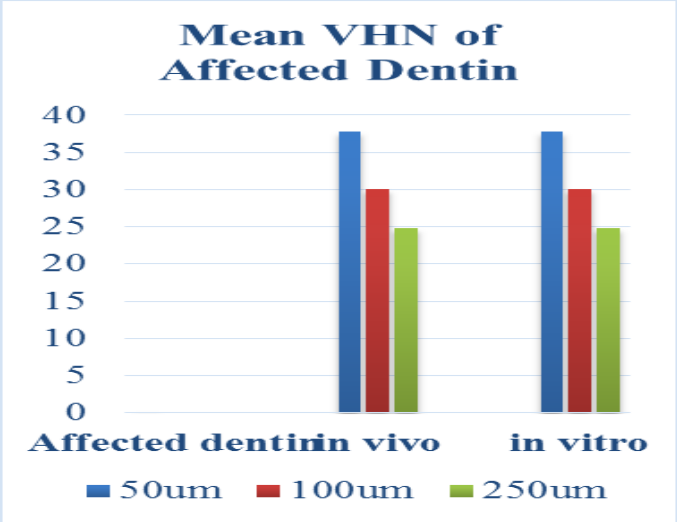
Table 2: Comparison of Microhardness of Infected and Affected dentin

Variables	n (%) In vivo and In vitro	Mean	p value
Infected dentin 50µm	10(50%)	10.2±15	0.00
affected dentin 50µm	10(50%)	38.3±2.9	
Infected dentin 100µm	10(50%)	18.2±0.9	0.00
affected dentin 100µm	10(50%)	29.9±1.4	
Infected dentin 250µm	10(50%)	25.6±1.6	0.33
affected dentin 250µm	10(50%)	24.9±1.5	

Graph 1



Graph 2



Conclusion

The results of this study indicates that 38% Silver Diamine Fluoride is effective in increasing the microhardness of the affected and infected carious dentin thereby arresting the caries progression. There was no significant difference in the microhardness between the *in vivo* and *in vitro* subgroups.

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