Current therapeutic options for treating deep carious lesions: a review

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Abstract

This review analyzes how to treat deep carious lesions taking into consideration histophysiologic and biomolecular events of the dentin-pulp complex in permanent teeth. We focus on clinical resources to assess the degree of lesion progression and to guide the removal of carious lesions.

Indirect Pulp Treatment, Stepwise Excavation and Partial Caries Removal are described by presenting clinical cases, and their follow-ups, led by students of Integrated Clinic II of the School of Dentistry, Universidad de la República, Uruguay.

These simple and inexpensive treatments are available to all clinicians and significantly decrease the number of pulp exposures.

The success of these therapeutic options depends on the proper selection of cases and on the integrity of the restoration within a comprehensive preventive plan.

Keywords: dentin physiology, dental caries, dental therapy, tooth remineralization.

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Introduction

Despite the application of preventive strategies, caries incidence remains high in Latin America[1]. For some decades now, the treatment of deep carious lesions has considered the biology of the dentin-pulp complex, its defense mechanisms and the etiopathogenesis of dental caries, applying therapeutic procedures that are increasingly less invasive. Exposures decrease significantly if a comprehensive and preventive plan is implemented, according to each patient's risk factors.

The aim of this review is to analyze the histophysiologic, biomolecular and clinical events, and to convey the importance of current therapeutic options of wide coverage, low cost and high biological value.

Review

A literature search was conducted in the following databases: Pubmed, Scopus, Odont (School of Dentistry Library, UdelaR), Biblioteca Virtual de Salud, PortalTimbo, Cochrane Library.

The mastery of histophysiology, molecular biology and defense mechanisms will help clinicians select the best therapeutic options and understand events relative to repair.

Young pulp with cellular abundance and few fibers have a better defense capacity.

Several factors can accelerate the aging process, whereby a young tooth may present aged pulp and an adult tooth may have active pulp if its structures have remained normal. It is not important to determine a chronological age limit for these treatments, but rather clinicians should assess pulp age, conduct a clinical examination and evaluate radiographs[2].

Intrinsic protection of the dentin-pulp complex

Dentinal fluid has a major protective role. It is considered an ultrafiltrate of blood from the pulp capillaries. It contains glycosaminoglycans, dental matrix proteins, plasma proteins such as fibrinogen, and it is saturated with calcium and phosphorus. It has an immunologic role as it contains immunoglobulins[3]. It has beta defensins with antimicrobial properties[4]. We can find cytokines, chemokines and an α tumor necrosis factor (TNFα). The substances found do not fully correlate to those in plasma; therefore, the fluid's composition seems to be regulated

by odontoblasts[5]. Odontoblasts form a layer that protects the pulp as they communicate through junction complexes. This restricts the diffusion of toxic components towards the pulp tissue, and the subodontoblastic capillary plexus helps dilute toxins. We must remember that the vasoconstrictor in anesthesia reduces circulation in the pulp and the dentinal fluid, slowing down toxin removal and reducing the defense capacity of the dentin-pulp complex[3]. Therefore, if terminal anesthesia is applied, it is better to select one without vasoconstrictor.

Defense mechanisms

Dentin matrix is considered a reservoir for bioactive molecules, among them transforming growth factor β (TGF-β) as the main element in the formation of dentin sclerosis. This happens as it interacts with the membrane receptors of odontoblasts, thus reducing dentin permeability when facing an aggression[6].

Current evidence suggests that the low pH of the acids released by cariogenic bacteria such as acetic acid or lactic acid not only demineralizes hard tissues but also activates metalloproteinases (endogenous dentin proteinases). This degrades the dentin matrix, thus releasing the bioactive molecules sequestered during dentinogenesis[7]. Once released, they send molecular signals, thus stimulating the formation of tertiary dentin, which can be reactionary or reparative.

If the injury is moderate, odontoblasts survive and release reactionary dentin matrix underneath the injury site[8]. The resulting dentin is similar to physiologic dentin. Its only difference is the change in direction of the new dentinal tubules[3]. The fibronectin deposited by odontoblasts regulates the formation of reactionary tertiary dentin.

Growth factors act as signaling molecules as they activate the surface receptors of odontoblasts. These acquire enzymatic activity and trigger signal transduction pathways, causing the phosphorylation of transcription factors in the cytoplasm or in the nucleus, which leads to the hyper-regulation of gene activity. Much interest has been given recently to the regulation of the secretory activity of odontoblasts to identify the mechanisms involved in the formation of tertiary dentin. This activity is linked to genes and regulation pathways[9].

When the injury is more severe, some odontoblasts are destroyed. Reparative dentin is formed, with fewer and more irregular tubules. This dentin is deposited by the new odontoblasts derived from Höhl cells, which are considered pulp stem cells. In the last mesenchymal cell mitosis, the cell in contact with the basement membrane of the inner epithelium differentiates into odontoblast. The underlying cell remains a Höhl cell with the potential to differentiate into odontoblast-like cells (Fig.1)[10,11].

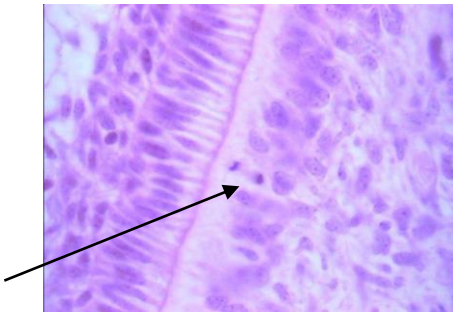

Fig 1. Histology Department. School of Dentistry.

The quantity and quality of tertiary dentin produced depends on the duration and intensity of the stimulus. The stronger the factors, the faster and more irregular their apposition. In these cases, up to 3.5 µm of dentin is deposited daily[10.]

From the onset of the injury, an immune and inflammatory defense mechanism is triggered in the pulp. Odontoblasts are the first to meet the antigens and to trigger an innate immune response. They have Toll-like receptors that recognize the molecular patterns of bacterial antigens. Once these receptors have been activated, odontoblasts release substances that regulate the immune and inflammatory response such as proinflammatory cytokines, chemokines and antimicrobial peptides[3,4,12]. In this way they recruit and stimulate immune cells, and they also destroy bacteria. The chemokines released include Interleukin (IL-8), which acts with the TGF-β1 released from the dentin matrix. This increases the number of dendritic cells with release of chemotactic mediators[3]. The subsequent flow of cells in the immune system contains macrophages, lymphocytes and plasma cells. As the carious lesion progresses, the density of dendritic cells increases. They tend to appear in the perivascular region of the central pulp and in the subodontoblastic region. They then spread within the odontoblast layer, and some have extensions inside the tubules. They capture the antigens

and then take them to the T lymphocytes[13]. The close link between odontoblasts and dendritic cells underneath the caries indicates that they may have a role in odontoblast differentiation, and/or have secretory activity in dentinogenesis and the immune system[3].

The injury of the carious process presents several mechanisms that regulate pulp microcirculation, reducing intrapulpal pressure and restoring blood flow.

Additionally, with the action of bacterial antigens, nerve endings associated with blood vessels release vasoactive neuropeptides such as substance P (SP) calcitonin gene-related peptide (CGRP), vasoactive intestinal peptide (VIP), which triggers neurogenic inflammation. This is part of the immune defense mechanism. These neuropeptides regulate blood flow, increasing the volume and vascular permeability in the affected region. They modulate the pulp immune response by recruiting immune cells, thus enabling tissue repair processes[13]. It has been shown that SP acts as a chemotactic and stimulating agent for macrophages and T lymphocytes[14].

Advances in molecular biology and immunology set the scientific bases for the new therapeutic strategies when treating deep carious lesions.

What to asses in the diagnosis of a deep carious lesion

It is important to assess the progression rate of the lesion, if the progress is quick or slow, if it is open or closed, in order to guide the removal of carious lesions[8].

In a closed ecosystem, bacteria are protected by the enamel, therefore this is an active lesion of rapid progression. If the enamel collapses, the environment might change and the cariogenic plaque is more vulnerable to brushing, mastication forces and other self-cleaning phenomena. Therefore, microbial ecology changes positively, leading to dentin remineralization, which turns dentin harder, darker and resistant to acids.

Poor hygienic habits might lead to caries that progress rapidly in open ecosystems. Light color and soft texture point to very active caries, where swift action is needed.

The analysis of color, consistency and texture reflects differences in bioactive molecules in the carious dentin, and in the potential for pulp repair.

The condition of the pulp must be evaluated. Therapeutic options that avoid pulp exposure in deep carious lesions are indicated for reversible pulpitis[15], such as in the case of deep carious lesions with asymptomatic pulp, and hyperemia[16]. The radiograph shows a deep carious lesion with wide pulp, which indicates a good repair potential.

Criteria in the treatment of deep carious lesions

Removing deep carious lesions always presents the risk of removing healthy dentin tissue and exposing pulp unnecessarily. There is still no agreement, as there are several criteria on how to determine the boundary between carious tissue to remove and tissue to conserve.

Fusayama[17] describes two zones in carious dentin: the external zone or infected dentin which cannot be remineralized, and the internal zone or affected dentin, which can be remineralized.

Although currently used, physical diagnostic methods, that is to say, assessing tissue color and hardness, are very subjective[18]. Dentin hardness varies according to the zone and is lower in deeper areas. Therefore, healthy circumpulpal dentin can be softer than some carious dentin values. In acute caries, soft dentin precedes bacterial invasion, which might cause the unnecessary wear of healthy tissue.

Regarding color, there is no clear correlation to the degree of infection. Dark dentin might indicate an arrested infection with non-viable bacteria. Demineralized dentin might turn dark on account of the extrinsic action of the patient's diet[18].

Chemical methods are questioned because they lack specificity. In 1963, Turell[19] suggests the use of basic fuchsin in a hydroalcoholic solution. Given the threat of carcinogenicity posed by fuchsin, Fusayama reformulates the caries detector by using 1% acid red in propylene glycol[20]. Fusayama shows that staining can expand to healthy dentin in acute carious lesions, as the stained area is deeper than that of bacterial invasion. In chronic lesions, staining is superficial compared to the bacterial invasion area, as infected tissue remains without staining[17].

Yip and Kidd have shown that colorimetric tests tend to overextend the cavity, specially near the amelodentinal junction and the circumpulpal dentin, which are areas with lower mineralization[21,22]. The terminal ramifications that form the Fish plexus at the amelodentinal junction, and the larger diameter of the circumpulpal dentin tubules, jointly with the presence of interglobular dentin (Czermak interglobular spaces) make this dentin more permeable and less demineralized[10]. A new colorimetric test was developed in the Japanese market using polypropylene glycol (PM=300) instead of propylene glycol (PM=76) to prevent excessive dentin removal, as the higher the molecular weight, the lower diffusion in porous tissues[23].

Nowadays, at the School of Dentistry of UdelaR, organic products are used, such as acid red 52 at 1%, with a careful interpretation and short exposure time. It is applied and then immediately removed with water.

When we consider the limitations of physical and chemical methods, and based on multiple microbiological studies that have shown that sealed carious lesions present fewer bacteria, inactivation and progression arrest, there is a change in the idea of how dentin caries should be treated[24,25].

Therapeutic options for treating deep carious lesions:

Below we describe different strategies to treat deep carious lesions, considering the paradigm shift in this area.

1. Indirect pulp treatment

It is the protection of dentin after deep excavation, which entails leaving a thin layer of carious dentin to prevent exposure. It has received different names. It is known as Indirect Pulp protection[26], Indirect pulp-capping[27], Indirect pulp therapy[28], Expectant treatment[29], and Indirect pulp treatment[30,31]. Indirect pulp protection can also be implemented on a thin layer of healthy dentin which was exposed by trauma. Indirect pulp protection of a thin residual layer of carious dentin can be performed in one session without reopening, or in two sessions reopening in six to eight weeks' time[26].

Petrou[31] calls it one-step Indirect pulp treatment or two-step indirect pulp treatment. Bjørndal calls the treatment traditional Stepwise when a thin layer of carious dentin is protected and reopened[32].

In brief, Indirect pulp protection and indirect pulp treatment are synonymous, and they can be performed in one or two sessions.

2. Stepwise excavation technique

Several authors have asked themselves which is the boundary to eliminate caries as close as possible to the pulp, leaving a thin layer of infected tissue without risking pulp exposure[32]. This is how the Stepwise excavation technique appears. It does not aim to remove as much tissue as possible in the first session, but rather to change the lesion's cariogenic environment and activity[32]. We find it in literature as Serial excavation[30], Stepwise excavation[33] and Gradual caries excavation[34].

It is done in two clinical stages: in the first stage, the superficial necrotic dentin layer is removed, the caries is completely removed from peripheral dentin without acting on the pulp wall, which is covered with soft, wet and highly infected dentin. A calcium hydroxide base material is applied and the cavity is sealed.

In the second session, two to six months later, dentin is reassessed, caries is completely removed and final reconstruction is done, with the corresponding follow-ups.

3. Partial caries removal

Some authors question the need for a second stage[25] when considering the high success rate of these treatments according to the studies that show that sealing the cavity results in the arrest of the carious process. They state that a two-session treatment increases the risk of pulp exposure, the cost of the treatment and it is less comfortable. Extended periods might lead to microfiltration, therefore the patient might not return to complete the treatment or there might be dental fracture, which may result in treatment failure[35].

Clinical examples

We provide contexts for the therapeutic options described above through cases led by students of the Integrated Clinic II, adults, of the School of Dentistry, Universidad de la República (UdelaR), under the direct supervision of Endodontics and Operative Dentistry tutors.

Clinical case I

In 2013, a 37-year old female patient attended the Integrated Clinic II, Universidad de la República, with a deep carious lesion in tooth 37 (Fig. 2a). A deep carious lesion with asymptomatic pulp was diagnosed, as the cavity test was positive. Complete caries removal from the lateral sides with full isolation was planned. The most recent colorimetric test with acid red 52 in propylene glycol was observed (Detector, Pharma Dent, Uruguay), leaving a thin layer of infected dentin on the axial wall (Fig. 2b), which was protected with a mixture of pure calcium hydroxide ($Ca(OH)_2$) with saline solution, and then settable $Ca(OH)_2$ (Life, Kerr, USA) (Fig. 2c). It was sealed with glass ionomer (Gold Label Luting & Lining Cement, Tokyo, Japan).

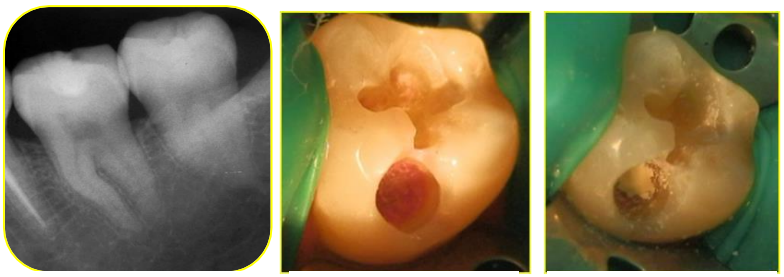

Fig. 2a - 3/Oct/2013 Fig. 2b - 3/Oct/2013 Fig. 2c - 3/Oct/2013

Although the second session was scheduled for two months later, as it was considered the time needed to obtain a dentinogenic response, the patient returned after a year. On clinical examination, the complete sealing was intact and the X-ray showed good periradicular health (Fig. 2d).

The cold test (Miracold Plus Hager Werken spray, Germany) was positive. When the seal was removed, there was no staining left, and the dentin was darker, hard and dry (Fig. 2e). The colorimetric test was conducted again to remove the stained dentin, before placing a glass ionomer cavity base and restoring with resin (TPH, Dentsply, Brazil) (Fig. 2f). Two

years after the X-ray evaluation, tooth 37 responded normally to the cold test and there was good periradicular health (Fig. 2g).

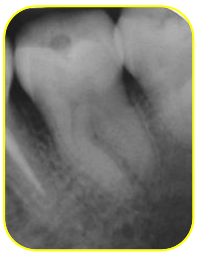

Fig. 2d – 31/Jul/2014

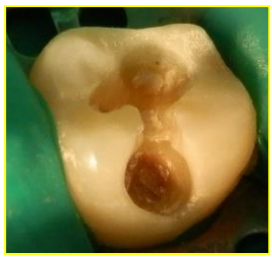

Fig. 2e – 7/Aug/2014

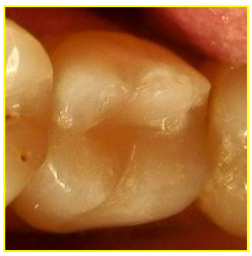

Fig. 2f – 7/Aug/2014

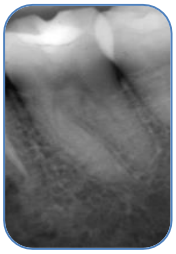

Fig. 2g – 7/Aug/2014

Clinical case 2

In 2011, a 22-year old male patient attended the clinic with a deep carious lesion in tooth 38. On the radiograph we noticed that the carious cavity was close to the large pulp chamber (Fig. 3a). A deep carious lesion with asymptomatic pulp was diagnosed after removing the superficial soft necrotic dentin with a dentin spoon and checking its vitality with the cavity test. Dentin assessment showed brown, soft and wet dentin. Stepwise excavation was planned. Fig.3b shows complete caries removal of lateral walls with colorimetric control without working on the pulp wall.

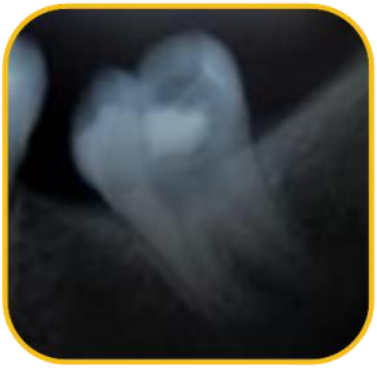

Fig. 3a – 27/Jun/2011

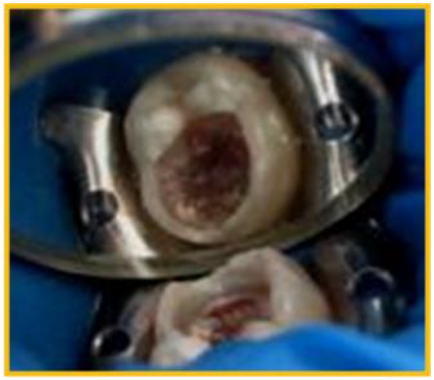

Fig. 3b – 27/Jun/2011

As indicated by Hasse et al.[26], a mixture of pure $Ca(OH)_2$ with saline solution was placed as medicinal dressing to inactivate the bacteria on the pulp wall. Settable $Ca(OH)_2$ was placed over it. This was done to prevent the pH of the glass ionomer used to seal the cavity from neutralizing the beneficial action of $Ca(OH)_2$.

Fig. 3c shows dentin deposition in the radiograph after a year. The tooth was asymptomatic and had a positive response to the electric test. The cavity floor was reevaluated in the

second clinical session. The dentin was brownish, hard and dry, and there was no staining left (Fig. 3d).

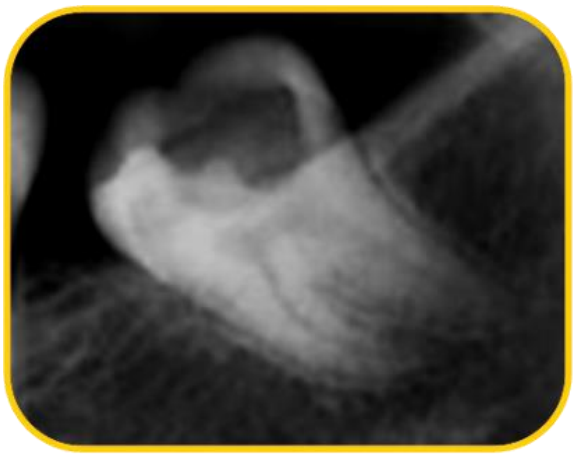

Fig. 3c – 20/Oct/2012

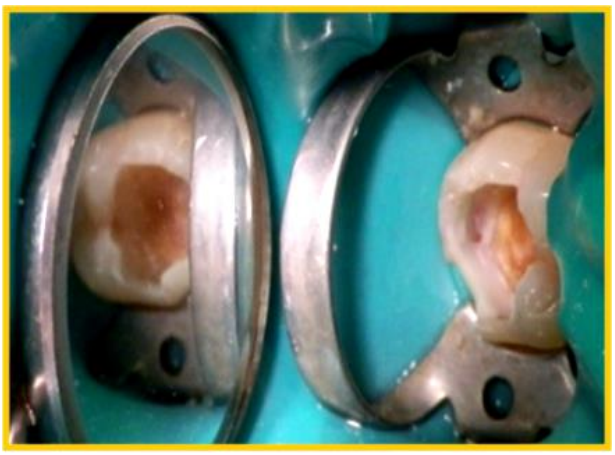

Fig. 3d – 20/Oct/2012

The colorimetric test was repeated with acid red; the stained dentin was removed (Fig. 3e) and the cavity was sealed with glass ionomer (Fuji Plus, Tokyo, Japan). In 2013, after the X-ray evaluation, indirect reconstruction was done (Fig. 3f).

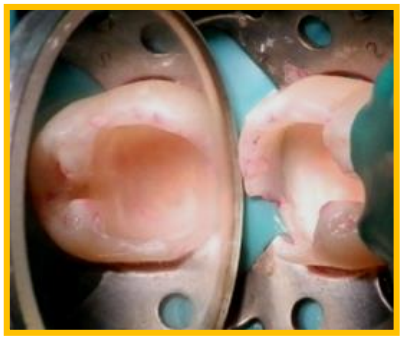

Fig. 3e – 20/Oct/2012

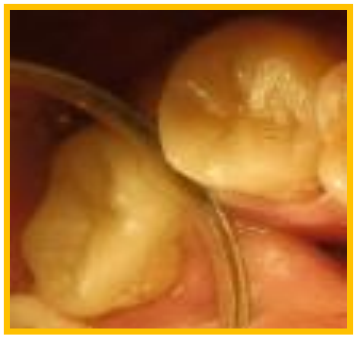

Fig. 3f – 20/Oct/2012

The 2014 and 2015 evaluations show a positive response to the electric test and there is good periradicular health (Figs. 3g,h,i).

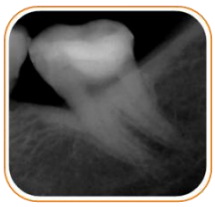

Fig. 3g -25/Sep/2014

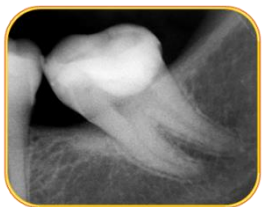

Fig. 3h – 25/Sep/201

Fig. 3i -19/Aug/2015

Clinical case 3

A 23-year old male patient attended the clinic with deep carious lesions in teeth 27 and 28. The radiograph showed a closed ecosystem and large chambers (Figs. 4a,b). Conservative

treatment was suggested for tooth 27 and extraction of tooth 28, as it did not come into occlusion with its antagonist. The undermined enamel of tooth 27 was opened and the superficial soft necrotic dentin was removed with a dentin spoon. A slight sensitivity was noted. Dentin analysis showed yellow, soft and wet dentin: all features of highly active caries (Fig. 4c).

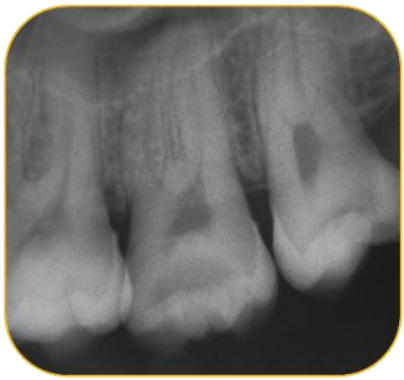

Fig. 4a – 20/Oct/2012

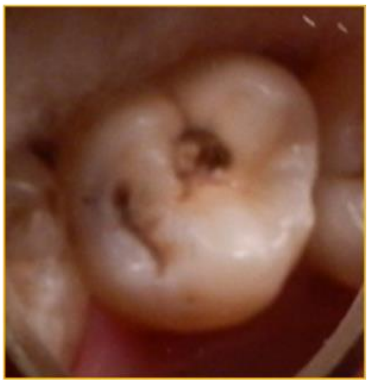

Fig. 4b – 20/Oct/2012

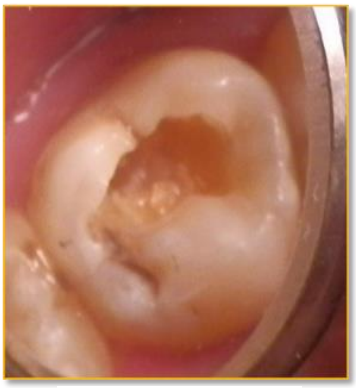

Fig. 4c – 20/Oct/2012

This partial caries removal from the whole carious cavity was lined with a mixture of $Ca(OH)_2$ and saline, settable $Ca(OH)_2$, and sealed with Fuji Plus glass ionomer until the following session. However, the patient returned two years later. The radiograph showed caries progression in tooth 28, while tooth 27 showed an arrested carious process (Fig. 4d) and there was a clinically positive response to the electric test. On reevaluation, the dentin was brown, semi-hard and dry, which showed that the carious process was inactive. The caries was completely removed from the lateral walls with a colorimetric test (Fig. 4e), and the pulp wall was protected with $Ca(OH)_2$, finally sealing with light-curing glass ionomer (Gold Label Light Cured, Tokyo, Japan).

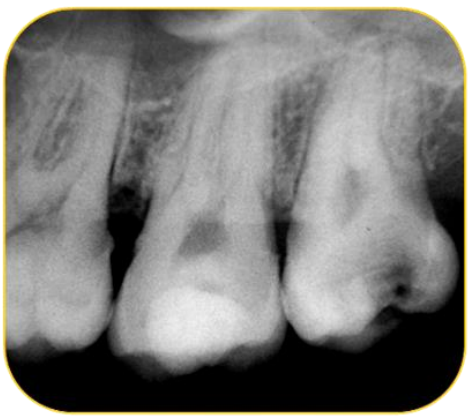

Fig. 4d – 28/Sep/2014

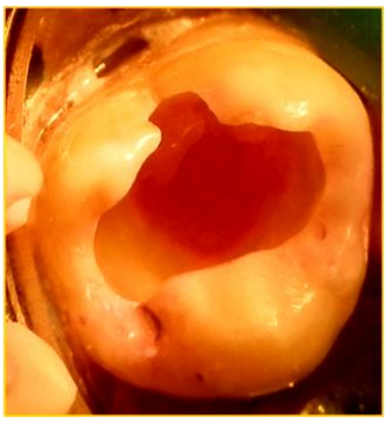

Fig. 4e –

A year later the patients sought assistance, reporting fractured vestibular enamel in tooth 27. However, the ionomer adhesion had conserved the tooth sealed (Fig. 4f). The cold spray test was positive. Dentin reevaluation showed hard tissue, with no traces of staining from the previous session (Fig. 4g).

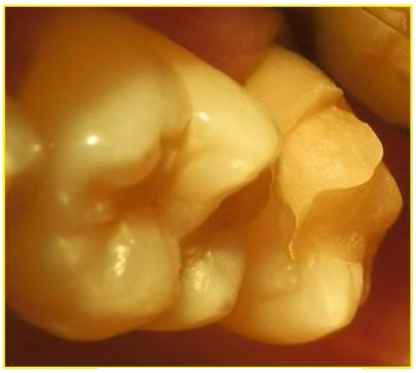

Fig. 4f -20/Aug/2015

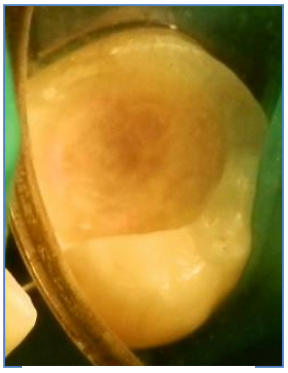

Fig. 4g – 20/Aug/2015

Colorimetric test with acid red yielded a pale rose stain (Fig. 4h). Considering the internal anatomy of the chamber, some dye was left in the mesiovestibular area so as not to expose the pulp horn. It was protected with settable $Ca(OH)_2$ (Fig. 4i) and lined with light-curing glass ionomer. Fig. 4j shows the final restoration with a zirconium inlay.

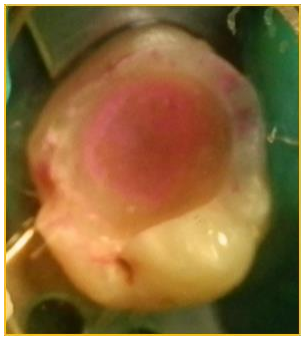

Fig. 4h -20/Aug/2015

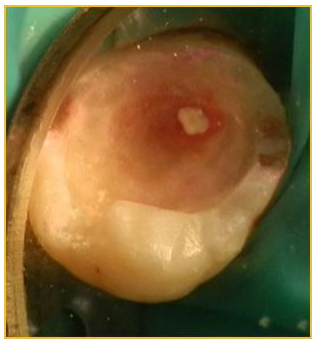

Fig. 4i -20/Aug/2015

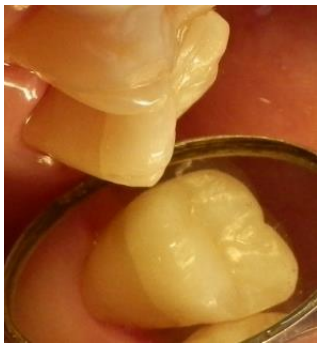

Fig. 4j -03/Nov/2016

Discussion

Based on the literature on conservative treatments, the histophysiologic and clinical events analyzed confirmed the preference for caries removal through more biological approaches.

The lack of specificity of physical[18] and chemical[21,22] methods to identify the boundary of carious tissue to remove, the defense mechanisms of the dentin pulp complex, as well as the arrest of the progression of sealed carious lesions[24,25] allow us to treat deep carious lesions

with techniques that are less invasive, such as Indirect protection, Stepwise excavation and Partial caries removal.

Different authors agree on complete caries removal from lateral walls. This is how adhesives act most efficiently, ensuring good sealing to prevent the penetration of nutrients to the residual bacteria, thus arresting the lesion[24,25,35].

There are differences regarding: amount of dentin infected left on the pulp or axial wall, protector used, waiting time before reopening and need to reopen.

Indirect pulp protection removes the largest possible amount of infected dentin on pulp or axial wall[26], whereas Stepwise excavation only removes superficial necrotic dentin[32]. The literature review includes various protectors when treating deep carious lesions. The most recommended one in two-session therapeutic strategies has been calcium hydroxide in different formulations[36].

In the clinical cases presented here, just as Hasse [(26)], medicinal dressing was placed at the end of the first session, with a mixture of pure $Ca(OH)_2$ and saline solution, covered with settable $Ca(OH)_2$.

On reassessing the cavity floor in the second session, dentin was always darker, harder and dryer, which agrees with several published studies[27,32,33,34].

When the layer of infected dentin is sealed, nutrients are removed from outside, leaving the serum glycoproteins of the dentinal fluid. They decrease with the formation of sclerotic and tertiary dentin, therefore, nutrients are also removed from the inside[37].

According to Bjørndal[24] and Maltz[25], the microbiological analysis conducted after the first session shows a reduction in the cariogenic flora of Lactobacilli, Streptococcus, and the prevalence of Streptococcus oralis and Actinomyces Naeslundii, which are not linked to active lesions, confirming the arrest of the carious process.

The waiting period before reintervention is between 1 and 12 months. Those who advocate for a longer waiting period (six months or longer) believe that this induces more tertiary dentin and thus reduces the risk of pulp exposure[38]. In their clinical study, Leksel et al.[39] found no differences in exposure frequency between a group of lesions reopened after two

months and another reopened after six months. The success of Stepwise excavation technique depends on the sealing: follow-up is essential. If the restoration fails and is not detected on time, the lesion reactivates and can reach an advanced state[40]. This is why the lesions are reopened after two or three months, time which is necessary for the $Ca(OH)_2$ to have its dentinogenic effect on the dentin-pulp complex.

In 2002, Maltz[25] publishes a study of 32 teeth with deep carious lesions after Partial caries removal. It questions the need for reintervention when the clinical, microbiological and radiographic carious process is halted. Two-session treatment increases the risk of: pulp exposure, microfiltration, dental fracture, the patient not returning and a higher cost[25,35].

Some authors describe the advantages of reintervention:

- Being able to clinically monitor the response of the dentin-pulp complex, verifying the arrest of the lesion[27,38].
- Being able to remove the slow-progressing caries that is still infected before placing the permanent restoration[27,38].
- Taking into account the gap described by Ricketts in 2001[40], which appears beneath the restoration because of dentin contraction (due to the arrest of the carious process). However, in 2006, Ricketts states that there is no clear evidence in favor of reintervention[41].

In a clinical study including 299 treatments, Maltz et al. compare Partial caries removal and Stepwise excavation. After a three-year follow-up, pulp survival is lower in the second technique, possibly because patients did not return to the second session[42].

In 2011, Maltz et al.[35] publish the 10-year follow-up on 32 posterior teeth with Partial caries removal. After three years of monitoring, the survival rate is 90%. After 5 years, it decreases to 82%, and between 5 and 10 years of monitoring, it decreases to 63%. Most failures occurred in teeth with multiple restored surfaces.

There are doubts regarding the reduced elastic modulus of the remaining carious dentin and its influence on the integrity of the restoration.

In an in-vitro study of 62 upper premolars, Schwendicke et al.[43] show that preserving a thin layer of infected dentin may affect the resistance to fractures. Adhesive systems that are efficient for teeth with complete caries removal also work with teeth with partial caries removal. Fiber-reinforced resins such as Ever X can increase resistance to fractures.

There are no definitive conclusions regarding the suitability of having one-session or two-session treatments.

Although the clinical cases presented here included two sessions, their success and the success of several other cases from Integrated Clinic II indicate that in some cases it might be unnecessary to reopen the lesion. The factors influencing this decision are: absence of pain (due to hyperemia), dentin assessment and assessment of dental remains. It might not be necessary to reopen if most of the carious dentin was removed, leaving a thin layer of infected dentin whose cavity edges are on the enamel. If there are doubts regarding the lesion's activity, the surfaces included and or the presence of gingival margin on the dentin that might affect the sealing in the long term, which might lead to the lesion reactivating, then it is better to reopen.

Bioactive materials such as glass ionomer, MTA and Biodentine, are suggested for one-session treatments. The bioactivity of these materials leads to remineralization with the underlying dentin substrate[44], and to excellent sealing. The dissolution that occurs when placing $Ca(OH)_2$ and its lack of adherence can be avoided.

In a multicentric study conducted with 314 patients, Bjørnal et al.[45] conclude that Stepwise excavation significantly reduces the number of pulp exposures, and that pulp survival is higher than in superficial pulpotomy in caries-related exposure.

Conclusions

- The main aim of conservative treatments of deep carious lesions is to avoid removing all the infected tissue, and to inactivate or arrest the lesion on account of changes in

the cariogenic environment, in turn enhancing the defense mechanisms of the dentin-pulp complex.

- Professionals still have to determine if it is better to treat in one or two sessions. This depends on the analysis of pulp health, its response capacity, dentin assessment and the characteristics of the dental remains.
- If professionals choose the one-session treatment, they should use materials with good mechanical qualities, suitable sealing and biostimulating characteristics.
- The success of these treatments hinges on the integrity of the restoration and on the follow-up within a comprehensive preventive plan according to the patient's risk factors.
- As the success rate is lower in long-term follow-ups, professionals should always clinically monitor patients and take X-rays regularly.
- We can conclude that in selected cases that have a good diagnosis, the treatments described above significantly reduce pulp exposures and subsequent complications.

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