REVIEW ARTICLE

CALCIUM HYDROXIDE IN DENTISTRY: A REVIEW OF LITERATURE.

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ABSTRACT

Calcium hydroxide is one of the most widely used medicaments in dentistry. This article aims to review the material based on its history, properties, mode of action, clinical use of its different formulations, significance of the vehicles used in $Ca(OH)_2$ pastes, and the role $Ca(OH)_2$ plays in various clinical procedures. The review of literature enabled us to conclude that its application chiefly related to its ability to stimulate dentin bridge formation and its antimicrobial properties. The review will also enable clinicians to make informed judgements about which formulations of calcium hydroxide should be used for specific clinical procedures.

Key words: calcium hydroxide, mode of action, clinical applications

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INTRODUCTION

Calcium hydroxide has played a major role in dentistry, being one of the most widely used medicaments, ever since its use was popularized by the pioneering studies of Hermann.[(1)] Well conducted research about the properties of calcium hydroxide such as its biocompatibility, antimicrobial potential, hard tissue deposition, physical-chemical aspects, give credibility to the choice of this materials in several clinical procedures.[(2)] This retrospective of literature aims to review the material under the following general headings: history, physical & chemical properties, its mechanism of action, the different formulations of $Ca(OH)_2$, vehicles used in $Ca(OH)_2$ pastes, and the role of $Ca(OH)_2$ in various clinical procedures where its use is indicated.

HISTORY

The initial reference to the use of calcium hydroxide in dentistry has been attributed to Nygren (1838)[3] for the treatment of a dental fistula, whilst Codman (1851)[4] was the first to use the material to preserve involved pulp. It was however through the studies of Hermann (1930)[1] that calcium hydroxide was introduced to dentistry as a successful pulp capping agent. Before this pulp therapy consisted of devitalization with arsenic and other fixative agents. Hermann demonstrated the formation of secondary dentin over the amputation sites of vital pulps capped with calcium hydroxide. Hermann's introduction of calcium hydroxide to dentistry had begun a new era of successful vital pulp therapy.[5]

The first reports dealing with successful pulp capping using calcium hydroxide appeared in literature between 1934 and 1941, since then the clinical indications for its use have expanded greatly. In 1938, Teuscher and Zander introduced calcium hydroxide in the United States and they histologically confirmed complete dentinal bridging with healthy radicular pulp under the calcium hydroxide medicament.[6] $Ca(OH)_2$ was most favoured as a pulpotomy agent in the 1940s and mid-1950s because it was thought to be more biologically acceptable owing to the fact that it promoted reparative dentin bridge formation and pulp vitality was maintained.

PHYSICAL PROPERTIES

· $Ca(OH)_2$ is a white odourless powder with a molecular weight of 74.08

· It has low solubility in water (1.2 g/l at 25°c), which further decreases as temperature rises. This low solubility is a good clinical characteristic, because a long period is necessary before it becomes soluble in tissue fluids, prolonging its duration of action when in contact with vital tissues.

· $Ca(OH)_2$ has been classified as a strong base with a pH of 12.5-12.8.[7,8,9] The high pH of calcium hydroxide plays a vital role in the various actions of calcium hydroxide.

CHEMICAL PROPERTIES

The main actions of calcium hydroxide come from its dissociation into Ca^{2+} ions and OH^- ions. The percentage of these ions is about 45.89% Ca^{2+} and 54.11% OH^- ions. The actions of these ions on vital tissues and bacteria induce hard tissue deposition and its antibacterial effect.[9]

Changes in the biological properties of $Ca(OH)_2$ can also be understood through its chemical reactions. For example, $Ca(OH)_2$ in the presence of carbon dioxide (from tissues or atmosphere) forms calcium carbonate (a weak acid oxide), and this product does not have $Ca(OH)_2$ hard tissue depositing and antibacterial properties.

Thus a chemical analysis of the essential aspects of calcium hydroxide is important in order to use it correctly. Factors such as the influence of the vehicle on its rate of ionic dissociation, time necessary for reaching appropriate pH for microbial control, the action of carbon dioxide that interferes with its properties etc., have to be taken into account when using the material in different clinical situations.[2]

MECHANISM OF ACTION

The mechanism by which calcium hydroxide brings about its various actions has been broadly divided into two categories:[2]

1. Biological Effects
2. Antibacterial Effects

1) Biological Effects:

When calcium hydroxide is applied directly to the pulp (for example in a direct pulp cap or pulpotomy procedure), the tissue immediately adjacent to the medicament is necrotized by the high pH of calcium hydroxide. The necrosis is accompanied by acute inflammatory changes in the underlying tissue. In 4-9 days, three distinct histological zones can be identified under the calcium hydroxide medicament-

1. Zone of coagulation necrosis.
2. Zone of variable osteodentin.
3. Zone of relatively normal pulp under a hyperemic odontoblastic layer.

Holland et al. have showed that formation of mineralized dentin starts between 7-10 days of applica-

tion, at the junction of the necrotic zone and vital inflamed tissue.[10]

Calcium Hydroxide Induced Mineralization

It seems that calcium hydroxide has the unique potential to induce mineralization even in tissues which are not programmed to mineralize. [11] Although calcium hydroxide works effectively in forming a dentin bridge, its exact mechanism is not fully understood. The following routes have been suggested as to how calcium hydroxide induces the reparative process:

- Calcium hydroxide is believed to maintain a local state of high alkalinity and thereby has the capability of activating tissue enzymes such as alkaline phosphatase, which is postulated to play an important role in hard tissue formation.[12] Alkaline phosphatase, which acts best in an alkaline pH of 8.6 to 10.3, is associated with providing phosphate ions at the mineralization sites. It does this by hydrolyzing phosphate ions from organic radicals at an alkaline pH.[13] These phosphate ions react with Ca^{2+} ions from the circulating blood, creating a sediment of calcium phosphate on the organic matrix, and indeed this sediment is the molecular unit of hydroxyapatite, starting the process of dentin bridge formation.

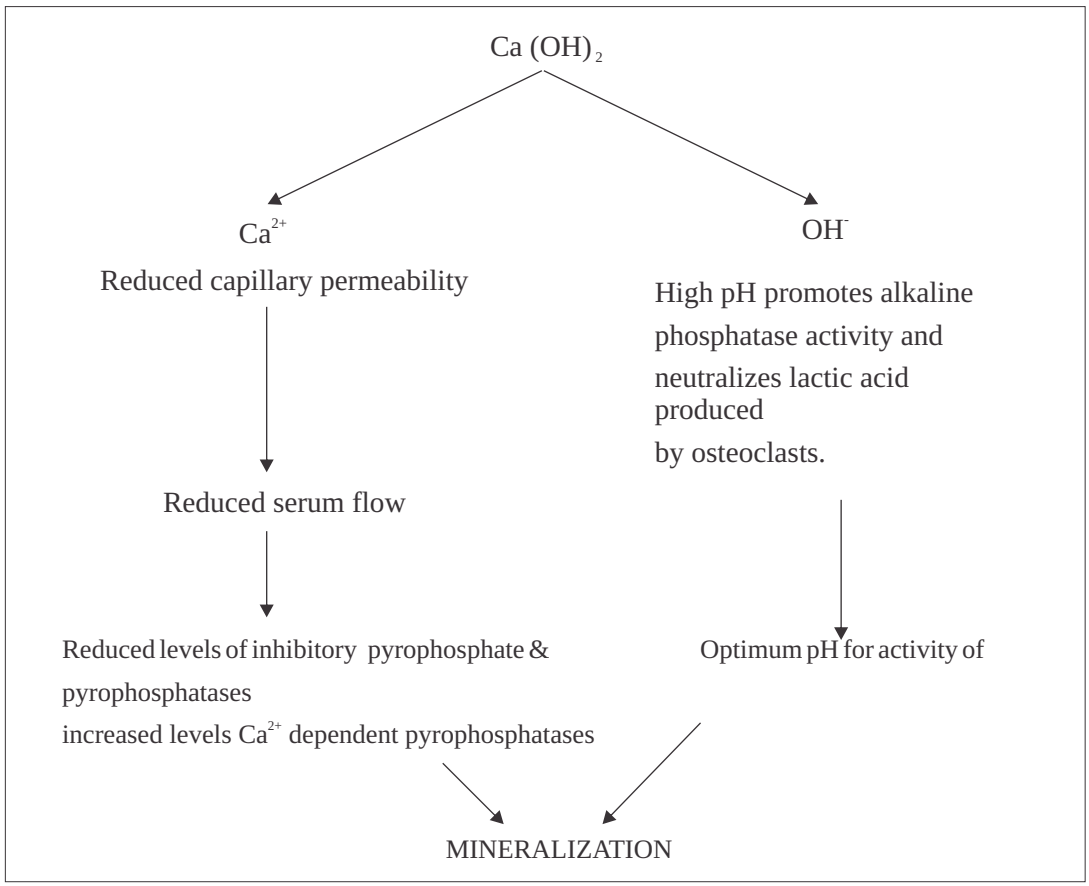
- Heithersay suggested that calcium ions may reduce the permeability of capillaries, so that less intercellular serum is produced, thus increasing concentration of Ca^{2+} ions at the mineralization site.[13] The reduced capillary permeability and the increase in the number of Ca^{2+} ions, could further reduce serum flow within the dental pulp and consequently the concentration of the inhibitory pyrophosphate ion would be reduced. The pyrophosphate ions play a role in inhibiting the mineralization process. Not only is its concentration reduced, but it is also metabolized at the mineralization sites, due to an increase in the levels of calcium dependent pyrophosphatases, and this could result in uncontrolled mineralization of pulp tissues. This could also possibly explain the high incidence of mineralized canals following pulpotomy or direct pulp capping with $Ca(OH)_2$.[14] Uncontrolled mineralization of pulp is therefore dependent on a reduced blood supply to the remaining vital tissue and not necessarily on the amount of reparative dentin formed with time.[15]

- It has been suggested that a rise in pH, as a result of the free OH^- ions, may favour mineralization.[16] An alkaline pH may neutralize the lactic acid secreted by osteoclasts and this may help to prevent further destruction of mineralized tissue. The high pH is also optimum for pyrophosphatase activity, thereby reducing levels of inhibitory pyrophosphate; leading to or favouring mineralization. There is however a problem in accepting the hydroxyl ions as the sole initiator of the mineralization process, as it has been shown that other highly alkaline compounds such as barium hydroxide and calcium phosphate fail to initiate mineralization.[11]

It has been speculated that $Ca(OH)_2$ exerts a mitogenic and osteogenic effect. The high pH combined with the availability of Ca^{2+} and OH^- ions have an effect on enzymatic pathways and hence mineralization.[17]

Yoshiba et al. provided immunofluoroscence evidence of the possible contribution of calcium hydroxide to odontoblastic differentiation. They found increased amounts of fibronectin (an extracellular glycoprotein linked to cell differentiation), among migrating fibroblasts and newly formed odontoblasts, in areas of initial calcium hydroxide induced calcification. They noted that while calcium hydroxide was not unique in initiating reparative dentinogenesis, it demonstrated the most rapid tubular dentin formation in comparison to calcium phosphate ceramics and tricalcium phosphate.[18]

Summary of Ca (OH)$_2$ induced mineralization



The Dentin Bridge

A mineralized barrier or 'dentin bridge' is usually produced following the application of calcium hydroxide to vital pulp. This repair material appears to be the product of odontoblasts and connective tissue cells. A discussion on this critical dentin bridge, which is the product of the repair process induced by calcium hydroxide, is important to understand the result of the mineralization process induced by $Ca(OH)_2$.

Source of Ca^{2+} ions in the Dentin Bridge

There is some controversy as to whether the calcium ions necessary for dentinal bridge formation comes exclusively from the circulating blood or whether the calcium hydroxide medicament also makes a contribution. Radioisotope studies have established that Ca^{2+} ions present in the applied $Ca(OH)_2$ do not become incorporated in the mineralized repair tissue.[19,20] Rather the dentin bridge derives its mineral content solely from the dental pulp, via the blood supply. These studies indicate that calcium hydroxide is an initiator rather than a substance for repair and its role in dentin bridge formation is limited to maintaining a low pH and producing a low grade irritation and inflammation.

However other studies showed that the Ca^{2+} ions from the medicament may also enter into the dentin bridge by forming calcium salts and calcium protein complexes.[21,22] What these investigators did was to use pastes such as barium hydroxide and strontium hydroxide, instead of calcium hydroxide. When they evaluated the bridge formed later, it showed deposits of barium carbonate and strontium carbonate grains, resembling graining observed with calcium hydroxide. Since barium and strontium are not normally present within the circulating blood, they concluded that these deposits could only come from the capping medicament used.

What can be tentatively concluded is that, it is the circulating calcium from the blood that is mainly responsible for the calcium phosphate dentin bridge, while other calcium complexes and salts, such as calcium carbonate in the dentin bridge, may be formed by calcium coming from the medicament. What is undisputed is the role calcium hydroxide plays in maintaining a localized state of alkalinity, stimulating alkaline phosphatase activity, and reducing capillary permeability, all factors that favour mineralization and pulpal repair.

2.) Antibacterial effects

Along with its biological effects on tissues, calcium hydroxide has an important antimicrobial action, and this property is important in many of the clinical procedures. These antibacterial effects are thought to be related to pH and directly proportional to the rate of release of OH^- ions from $Ca(OH)_2$. The various ways by which $Ca(OH)_2$ brings about its antimicrobial action are:

- There is liberation of ions OH^- from the alkaline $Ca(OH)_2$ and these ions act on the enzymatic sites, proteins and phospholipids located on the bacterial cytoplasmic membrane. The effect of high pH alters the integrity of the cytoplasmic membrane by interfering with enzymatic activity, chemical injury to organic components and transport of nutrients. The extreme values of pH cause the uncoiling of many bacterial proteins with loss of their biological activities. Estrela et al. suggested the hypothesis of an irreversible bacterial enzymatic inactivation if extreme pH conditions are maintained for a long period of time and also a temporary bacterial enzymatic inactivation, with the restoration of normal activity when the pH returns to the ideal level for enzymatic activity.[23] This suggests the importance of maintaining a high pH of the $Ca(OH)_2$ dressing used as an intracanal medicament so has to give it enough time to manifest its potential of action on microorganisms present in endodontic infections.

- Another essential aspect in the antimicrobial action of $Ca(OH)_2$ is its neutralization of bacterial toxins. Buck et al. evaluated the detoxification of endotoxin by different endodontic irrigants and calcium hydroxide. The results showed that the biologically active portion of endotoxin, Lipid A, is hydrolyzed by highly alkaline chemicals like $Ca(OH)_2$.[24]

- Safavi and Nichols showed that $Ca(OH)_2$ is capable of hydrolyzing the lipid moiety of the bacterial lipopolysaccharide cell wall (LPS), resulting in the release of free hydroxyl fatty acids. They suggested that the calcium hydroxide mediated degradation of LPS may be an important reason for the beneficial effects obtained with calcium hydroxide used in clinical endodontics.[25]

- Estrela et al. suggested that $Ca(OH)_2$ is effective against anaerobic organisms, which are the most abundant organisms in the infected root canals, through its ability to absorb carbon dioxide, essential for the survival of these organisms.[26]

What can be concluded in the search for explaining the antimicrobial mechanism of calcium hydroxide is that the enzymes in the cytoplasmic membrane were the primary targets of pH changes, which can lead to reversible or irreversible inactivation of a wide range of microorganisms. As the site of action of OH^- ions of calcium hydroxide is the bacterial cytoplasmic membrane, and since in the microbial world, cytoplasmic membranes are similar irrespective of the microorganisms other characteristics, calcium hydroxide has a wide range of action with similar effect on aerobic, anaerobic, Gram+ve and Gram-ve bacteria. This is clinically significant as in an infected canal system all these organisms are encountered.[2] It is to be noted that calcium hydroxide kills only the bacterial on the surface of the pulp and not those that have penetrated necrotic tissue.[27] Thus the material has no beneficial effect on the healing of an inflamed pulp and its use would appear to be indicated for the treatment of healthy or superficially contaminated pulps where bacteria have not penetrated into the deeper parts.[28] The main issue is not 'how bacteria are killed' but 'how vital tissues can be protected from the toxicity of calcium hydroxide'. The separation of the material from the vital tissues by a zone of necrosis is probably what prevents gross tissue damage.[14]

DENTAL FORMULATIONS OF $Ca(OH)_2$:
Setting vs. Non-setting pastes

Several calcium hydroxide containing pastes are currently being used in dentistry. These can broadly be classified according to whether they are setting (e.g. Dycal) or non-setting materials (e.g. Pulpdent). Clinicians tend to develop their own preferences according to ease of manipulation and apparent clinical success, however there are some procedures where one formulation is recommended over the other.

Non-setting pastes have a higher pH than hard setting pastes, and thus have a better antibacterial effect. However non-setting calcium hydroxide pastes tend to undergo dissolution at a much faster rate and are likely to leach out from beneath the restoration. Therefore it is essential to establish a balance between a material that is sufficiently soluble to exert a therapeutic effect, yet which is not so soluble that it dissolves away, thus vitiating its desired use. Thus non-setting $Ca(OH)_2$ pastes, like ApexCal (Ivoclar, Schaan, Liechtenstein.), are generally used for root canal dressings, where as setting $Ca(OH)_2$ pastes are used as root canal sealers, e.g. Apexit (Ivoclar, Schaan, Liechtenstein).[29] In an IPC procedure, where the antimicrobial action of $Ca(OH)_2$ is more important, a non-setting calcium hydroxide paste is recommended. British researchers have confirmed this, by finding significantly more bacteria under the hard setting version versus the non-setting pastes.[30] In a DPC or pulpotomy procedure both pastes are equally effective in inducing reparative dentin formation. The advantage a paste like Dycal (Dentsply, Milford, USA) has is that being less alkaline, it is less caustic to the remaining vital pulp, and being stronger, condensation of the permanent filling can be done directly over it. The advantage a non-setting paste like Pulpdent (Pulpdent Corp., Brookline, USA) has is that the dentin bridge it forms is more visible radiographically, allowing better clinical judgment of whether the procedure is a success or not. The reason for the better radiographic contrast under a non-setting paste, is that a necrotic zone of chemically altered tissue is formed between the material and the dentin bridge, as a result there is a gap between the bridge and the medicament, making it easier to distinguish the dentin bridge. Although the dentin bridge formed under the setting pastes are histologically similar, the necrotic zone is resorbed prior to the formation of the dentin bridge, which then comes to lie directly under the capping material making it difficult to distinguish radiographically.

VEHICLES USED IN CALCIUM HYDROXIDE PASTES

Many substances have been added to calcium hydroxide to improve it antibacterial action, radiopacity, flow, consistency, and to maintain its high pH.[31] An ideal vehicle should allow slow diffusion of Ca^{2+} and OH^- ions, have low solubility in tissue fluids, and permit hard tissue deposition.[32] The differences in the velocity of ionic dissociation of $Ca(OH)_2$ are directly related to the vehicle used in the paste, the lower the viscosity of the paste the higher will be the ionic dissociation.[23] In general 3 types of vehicles are used - aqueous, viscous and oily.[32]

Aqueous Group Vehicles: This group is represented by water-soluble vehicles including water, saline, dental anaesthetics, and methylcellulose. These types of vehicles promote rapid release of Ca^{2+} and OH^- ions and a high degree of solubility, causing $Ca(OH)_2$ to be rapidly solubilized and resorbed. Examples: Calxyl (Otto & Co., Frankfurt, Germany) - oldest manufactured paste introduced by Hermann and is basically a solution of $Ca(OH)_2$ in water, Pulpdent (Pulpdent Corp., Brookline, USA) - $Ca(OH)_2$ in an aqueous suspension of methylcellulose,& Calcipulpe (Septodont, Saint-Maur, France) - vehicle used is carboxymethylcellulose.

Viscous Group Vehicles: The pastes in this category use viscous vehicles such as glycerin, polyethylene glycol, and propylene glycol. The viscous vehicles cause a much slower release of Ca^{2+} and OH^- ions over extended periods. The higher molecular weight of these vehicles minimizes the solubility of $Ca(OH)_2$ into the tissues and maintains the paste in the required areas for longer intervals, prolonging the action of the paste. Example: Calen (S.S. White-Artigos Denta´rios, Rio de Janeiro, Brazil) - polyethylene glycol is the viscous vehicle in which $Ca(OH)_2$ is dispersed.

Oily Group Vehicles: The oily vehicles like silicone oil, camphor, & eugenol are non-water soluble substances that promote the lowest solubility and diffusion of $Ca(OH)_2$ within the tissues. Pastes containing this type of vehicle remain in the root canal for a longer time as compared to aqueous or viscous vehicles. Example: Vitapex (Neo Dental Chemical Products Co., Tokyo, Japan) - composed of calcium hydroxide, iodoform, and silicone oil as the oily vehicle.

Clinical situations that require a rapid liberation Ca^{2+} and OH^- ions at the beginning of treatment require an aqueous vehicle containing $Ca(OH)_2$ paste, whilst in clinical situations that require gradual and more uniform liberation of ions, a viscous vehicle containing paste should be used. Pastes containing oily vehicles have restricted use and are employed in those clinical situations that require a very slow ionic dissociation.[32] For example, in cases of dental replantation, as soon as treatment is performed, a paste with an aqueous vehicle should be used, because of the need for rapid ionic release. Subsequently a $Ca(OH)_2$ paste with a viscous vehicle should be used in the following dressings, because the paste must remain in the root canal for a longer period, during which high pH will be maintained and slow ionic release will occur. Pastes containing oily vehicles, like Vitapex, are used for apexification procedures, where the $Ca(OH)_2$ dressings have to last for 6-24 months.

ROLE OF CALCIUM HYDROXIDE IN CLINICAL PROCEDURES

1) Lining of cavities

The setting calcium hydroxide pastes are now in general use as lining materials. Their perceived advantages in addition to their therapeutic effects are as follows:[30]

i) They have a rapid initial set in the cavity, under the accelerating effect of moisture in the ambient air of the oral cavity.

ii) They do not interfere with the setting reaction of BIS-GMA resins, and are therefore the lining material of choice under composite resin materials.

iii) It is generally considered that the initial set of the material is sufficiently hard to resist the applied condensation pressure required for amalgam alloys.

2) Indirect Pulp Capping (IPC)

There is evidence that $Ca(OH)_2$ stimulates an increase in mineralization, within the affected dentin that remains at the base of the cavity.[29] The mechanisms by which mineralized dentin formation is initiated by calcium hydroxide, cannot hold true in an IPC procedure, as the medicament is not in direct contact with the pulp (as occurs is an direct pulp cap or pulpotomy procedure). The dentin bridging effects of calcium hydroxide occur only when the agent is in direct contact with healthy pulp tissue.[33] So the role of $Ca(OH)_2$, in an IPC procedure is limited to acting as an antimicrobial agent and a mild pulp stimulant with the pulp responding physiologically to the carious process to produce reparative dentin.[10]

Clinical studies have shown no significant differences in the ultimate success of this procedure, regardless of whether $Ca(OH)_2$ or some other medicament like Zinc oxide-eugenol is used over the residual carious dentin.[34,35] So while some clinicians recommend Zinc oxide-eugenol because of its better sealing and obtudent properties, others recommended calcium hydroxide because of its ability to stimulate a more rapid formation of reparative dentin and also it can stimulate healing of minute pulpal exposure which may have been overlooked.

3) Direct Pulp Capping (DPC)

DPC is undertaken in an attempt to maintain the health of an exposed vital pulp. Calcium hydroxide works very effectively in inducing the formation of a mineralized dentin bridge over the vital pulp. If bacterial contamination is minimal, calcium hydroxide pulp capping procedures can produce successful results in 96% of cases.[36] Just like in the IPC procedure, the importance of preventing bacterial microleakage and maintaining a proper apical seal for the success of a direct pulp capping procedure is vital.

4) Pulpotomy

This is a standard clinical procedure, which differs from DPC in that, surgical removal of the coronal

pulp is undertaken, prior to application of a medicament like $Ca(OH)_2$ The healing process is similar to that which occurs in direct pulp capping, i.e. a mineralized dentin bridge, is formed over the amputated pulp.

Calcium hydroxide was most favoured as a pulpotomy agent in the 1940s and mid-1950s because it was thought to be more biologically acceptable owing to the fact that it promoted reparative dentin bridge formation and pulp vitality was maintained.[6] Inspite of the great success of $Ca(OH)_2$ in forming a dentin bridge, a calcium hydroxide pulpotomy technique is generally not recommended for primary teeth owing to its low clinical success rate. Most of the long-term clinical studies have suggested this failure was the result of chronic inflammation and internal resorption calcium hydroxide causes.[37,38] At present calcium hydroxide pulpotomy cannot be recommended for primary teeth owing its low clinical success rate.[39]

Because of improved clinical outcomes, $Ca(OH)_2$ is the recommended pulpotomy agent for carious and traumatic exposures of young permanent teeth, particularly for the apexogenesis procedure. It is also recommended that once the apex has formed, the $Ca(OH)_2$ dressing be removed and conventional root canal obturation be done so has to prevent the long term canal calcification and obliteration it causes.[40]

***5)* Primary root canal filling material**

A study conducted by Mani & Chawala found $Ca(OH)_2$ mixed with lodoform to be a near ideal primary root canal filling material. The main advantages were its biocompatibility, anti-microbial actions, any over fill resorbs quickly, and it can be more easily applied or if necessary removed from the roots as compared to Zinc oxide-Eugenol.[42] One disadvantage with the use of $Ca(OH)_2$ as a root canal filling material was that it resorbs at a slightly faster rate that the deciduous root.

6) Intra canal medicament

Calcium hydroxide has been found to be the most effective intra canal medicament to obtain canal sterilization between endodontic appointments.[43] Although the cleaning, debridement and shaping of the root canal is the most important step of eliminating bacteria from the canal system, use of an intra canal medicament like $Ca(OH)_2$ can reduce treatment time and number of appointments. It is important to maintain a high pH as well as to allow enough time for the medicament to manifest its antimicrobial potential on the microorganisms present in endodontic infections. Heithersay recommends that $Ca(OH)_2$ be used as a root canal dressing in teeth with large periapical lesions and in cases where it is necessary to control the seepage of periapical exudates into the canal.[13] The mechanism whereby reduction of the seepage occurs is probably due to the fibrous barrier that is formed when $Ca(OH)_2$ is placed in direct contact with host tissues[44] or due to the constriction of capillaries.[13] $Ca(OH)_2$ is also believed to accelerate the natural healing of the periapical lesions regardless of the bacterial status of the root canal system.[45] The ability of $Ca(OH)_2$ to dissolve necrotic tissue is also useful, as anatomical problems may make it difficult for irrigating solutions to reach all areas of the root canal.[46]

7) Apexification

When $Ca(OH)_2$ is used for the apical closure of a non-vital young permanent tooth it forms a calcific

$Ca(OH)_2$ induced internal resorption

Calcium hydroxide induced internal resorption has been a subject of interest because of the implications it may have when used in different procedures like DPC or pulpotomy. Internal resorption occurs because the highly alkaline $Ca(OH)_2$ causes an over stimulation of the remaining vital pulp tissue, which results in undifferentiated mesenchymal cells giving rise to odontoclasts, that cause the resorption.[41] Internal resorption usually occurs when $Ca(OH)_2$ is in contact with the highly cellular vital primary pulp, and it is for this reason that DPC or $Ca(OH)_2$ pulpotomies are not recommended in deciduous teeth. Internal resorption does not appear to be a problem in permanent teeth, probably because of its lower cellular content and decreased inflammatory response of the permanent tooth pulp.

barrier of mineralized scar tissue across the apical foramen, in a manner similar to how it brings about dentinal bridging in DPC or pulpotomy. The difference here being that the hard tissue barrier it forms consists mainly of osteoid tissue (as there is no vital pulp and odontoblasts to differentiate and form reparative dentin). Because $Ca(OH)_2$ is inherently soluble, it must be replaced at 3-6 month intervals, until closure of the apex has occurred. It has also been suggested that role of $Ca(OH)_2$ in apexification is secondary, and what is more important in the success of this procedure is complete debridement and asepsis of the canal system, as apical closure has been found to occur even without a root-end filling material.

CONCLUSION

Ever since the introduction of calcium hydroxide as a pulp capping agent by Hermann, its role in dentistry has been almost unparalleled, and the indications for its use has expanded greatly. This review of literature enables us to conclude that its application in various dental procedures chiefly relate to its ability to stimulate dentin bridge formation and its antimicrobial properties. Considering the immense potential of the medicament it is truly a 'panacea in dentistry'.

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