ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11,Iss 6, Sep 2022

Review: Calcium Hydroxide In Dental Restoration

Dr. Shubhra Malik[1*], Dr. Sumita Giri Nishad[2], Dr. Chetna Arora[3]

1. Professor, Department of Conservative Dentistry & Endodontics, Santosh Dental College & Hospital, Santosh Deemed to be University, Ghaziabad.
2. Professor, Department of Conservative Dentistry & Endodontics, Santosh Dental College & Hospital, Santosh Deemed to be University, Ghaziabad.
3. Professor, Department of Conservative Dentistry & Endodontics, Santosh Dental College & Hospital, Santosh Deemed to be University, Ghaziabad.

*** Dr. Subhra Malik – Corresponding Author**

ABSTRACT

This research examines the ingredients of the various calcium hydroxide preparations used in restorative dentistry. The significance of specific elements in regard to the qualities of such substances and their mode of therapeutic action in relation to the dentine pulpal response and antibacterial activity is explored. Also covered are the use of calcium hydroxide in restorative dentistry.

Keywords: Calcium hydroxide, Restorative dentistry, restorative dentistry

INTRODUCTION

Calcium hydroxide preparations are widely utilised in restorative dentistry as therapeutic cavity liners and as temporary root canal dressings to stimulate hard tissue development. In a variety of endodontic circumstances and as the foundation for permanent root canal sealers.

This widespread use, however, is not matched by a clear understanding of how calcium hydroxide promotes osteodentine bridge formation or its putative antibacterial activity, despite widespread reports of hard tissue repair in pulp capping and in the periapical tissues after calcium hydroxide treatment (Glass and Zander, 1949; Berman and Massler, 1958; Stanley and Lundy, 1972; Schroder, 1973; Tronstad, 1974; Hendry et al., 1982; Holland

The objective of the first section of this paper is to review the tissue responses and therapeutic activity of calcium hydroxide and to detect any remaining uncertainty. Calcium hydroxide's applications in restorative dentistry have expanded thanks to the vast array of available formulations. In the second half of this work, various applications are discussed. Given that this alkaline substance is intrinsically non-biocompatible (Granath, 1982), its long history and widespread use are even more remarkable.There is a vast selection of commercially available products, but none for calcium hydroxide-based cements, dressings, or sealers.

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11,Iss 6, Sep 2022

Tissue reactions and therapeutic activity of calcium hydroxide

Material constituents and effect on solubility

The ingredients and amounts of calcium hydroxide cements available on the market vary from product to product. De Freitas (1982) and Prosser et al. (1982) have calcium hydroxide preparations were described, and Table I is based on their results. However, like all other dental cements, calcium hydroxide cements harden through an acid-base interaction, with the phenolic group in the alkyl salicylate ester serving as the acid (Prosser et al., 1979). Once the material has hardened, its therapeutic activity will depend on the release of CaZ+ and OH-, which can only occur if the cement is water soluble.

This solubility is a result of the plasticizer's inherent characteristics. Currently, Ca(OH) reacts with a salicylate ester chelating agent in the presence of a toluene sulphonamide plasticizer to set the majority of cements. The latter is soluble and hydrophilic. The hardened cement is composed of a matrix of calcium-a&y1 salicylate chelate and unreacted calcium hydroxide.

Physical properties

Compressive tensile force

Set calcium hydroxide lining materials are fragile, brittle materials utilised in thin section at the deepest part of a mine.the cavity's interior. Ray (1982) reported average 24-hour compressive strengths of 7.8 N.mm-z and 8.2 N*mmM2 for Dycal and Life, respectively, concluding that these fully cured cements were unable to withstand the average amalgam condensation pressure of 10.5 N*mme2 (Basker and Wilson, 1968). In contrast to these findings, Draheim et al. (1988) determined that the compressive strength of Dycal and Life at 24 hours was on the order of 30 N.mmw2 and that in 7 minutes, around 10 Nemm-2 was achieved. However, it seems prudent to apply a structural lining before amalgam insertion.

Acid dissolution

Any liner accidentally exposed to an acid etchant must be impermeable and unmodified. McComb (1983) discovered that Procal is more acid-sensitive than Life. According to Burke and Watts (1986), Dycal lost a much higher proportion of its mass than Life, MPC, or Procal. That MPC was most resistant to acid assault was a result of the paraffin oil plasticizer, which also explains the material's resilience to water dissolution (Prosser et al., 1982). The difference in plasticizer between Dycal and Life explains similar variations in behaviour.

Therapeutic mode of action calcific bridge development

Despite voluminous literature, the mechanism of calcium hydroxide's therapeutic effect for either calcitic bridge building or antibacterial action is unclear. Pulpal and periapical connective tissue reactions to calcium hydroxide will not be identical due to the presence of pulp-specific odontoblast precursors and the use of essentially distinct calcium hydroxide preparations in root canals and pulp capping agents. Calcific bridge formation is a generic term for the restoration of

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11,Iss 6, Sep 2022

pulpal exposure by osteodentine bridge creation and cementum-like substance or cementoid formation in periodontal or periapical tissues.

The pulpal response adjacent to calcium hydroxide has been recorded, but the actual sequence of histological alterations depends on whether a proprietary hard-setting cement or a laboratory-prepared calcium hydroxide paste was utilised. In 35 teeth from 10 individuals, Stanley and Lundy (1972) documented a Dycal-induced mummified layer that was phagocytosed and replaced by mineralizing granulation tissue. Most specimens had hard tissue bridges next to the Dycal by 23 days, but with varying thickness.

Tronstad (1974) and Fitzgerald (1979) noticed a l-3 cell thick layer of tibroblasts right on Life, followed by a lo-15 pm thick zone of osteodentine after 9 days, confirming this observation. However, Schroder and Granath consistently observed a 1.5-millimeter-thick necrotic zone following calcium hydroxide paste administration (1971).[1-2] After four days, cellular organisation and collagen production occurred near the edge of necrosis, followed by mineralization three days later. Necrosis may have been caused by the paste's higher dissociation and, consequently, alkalinity. Pitt Ford (1985) in dogs and Tagger and Tagger (1985) in monkeys demonstrated additional histological evidence of bridging by Dycal and Life, but not by Reolit (Ivoclar Vivadent Ltd, Schann, Liechtenstein). These studies typically reveal a mineralized collagenous matrix with cellular and vascular inclusions on histology.

After 28 days, seven out of twelve rat teeth treated with Analar grade calcium hydroxide and five out of eleven rat teeth treated with zinc oxide/purified eugenol formed dentine bridges, respectively (Watts and Paterson, 1987).However, with both calcium hydroxide and zinc oxide/eugenol, the pulp of the majority of teeth exhibited additional localised dystrophic calcification.

Apexification

Children's upper central incisors are the teeth most frequently affected by trauma (Todd and Dodd, 1985). Immature Roots with open apices may be treated with calcium hydroxide in a pulpotomy treatment with placement of calcium hydroxide over vital radicular pulp, or as a temporary root filling material in situations of non-vital young teeth with or without periapical disease (Frank, 1966; Heithersay, 1970, 1975; Cvek et aI., 1976).

The former condition is considered to be real apexitication, whilst the latter is best characterised as the induction of root end closure by the creation of a hard barrier that serves as a barrier for following traditional endodontic filling procedures.[2] The induction of root end closure is not limited to calcium hydroxide alone, although this substance has the highest documented success rate, ranging from 74% to 100% (Hallett and Porteous, 1963; Cvek, 1978; Chawla, 1986; Ghose et al., 1987).

The apical hard tissue barrier of human teeth has been described histologically as consisting of cementum, dentine, and pulp (Heithersay, 1970), cementum-like tissue with loose vital connective tissue inclusions (Cvek and Sundstrom, 1974), or cementum alone of both acellular

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11,Iss 6, Sep 2022

and cellular types (Klein and Levy, 1974).[3-4] However, Heithersay (1970) and Klein and Levy (1974) only documented the histological characteristics of a single tooth. Cvek and Sundstrom (1974) reported on 12 teeth that had all had effective calcium hydroxide treatment as determined radiographically, but had subsequently sustained root fractures (eight) or required extraction for other reasons (four). They discovered new cementum-like tissue and calcifications of various morphologies on the root canal walls in numerous sections, but only three teeth have complete apical closure.

It has been demonstrated in monkeys that the hard tissue barrier is heterogeneous (Dylewski, 1971; Ham et al., 1972; Torneck et al., 1973) and that apical closure is partial (Dylewski, 1971; Ham et al., 1972; Torneck et al., 1973). (Steiner and Van Hassel, 1971). Torneck et al. (1973) discovered prolonged periapical inflammation despite what appeared to be effective apical closure. They attributed this to necrotic debris trapped within the barrier and root canal. Presumably, the necrotic material-filled cavities were in contact with important periapical tissue, or the barrier is permeable.[4-6] They determined that the presence of a calcitic barrier is not a sufficient factor for measuring success. Although the experimenters of induction investigations attempted to prevent calcium hydroxide from entering the periapical tissues, it cannot be concluded that this did not occur based on the results of these studies. As described in the section entitled 'Method of therapeutic action,' the tissue response to calcium hydroxide overdosing is unpredictable. In addition, direct comparison of barrier induction results is difficult due to the use of numerous calcium hydroxide preparations, including Pulpdent (Heithersay, 1970), calcium hydroxide with sterile saline (Cvek and Sundstrom, 1974), calcium hydroxide with CPCP (Torneck et al., 1973; Javelet et al., 1985), calcium hydroxide with CMCP (Steiner and Van Hassel, 1971), and calcium hydrox (Hollandet al., 1979a,b).[7-9] Comparative investigations have rarely been conducted. As a result, it is difficult to determine which procedure for root end induction is the most predictable and effective, and the existing clinical criteria for success are quite rudimentary.

Mackie et al. (1988) reported that Reogan Rapid (Ivoclar Vivadent Ltd, Schann, Liechtenstein) was used for root end closure in 11-15 year olds for a mean treatment time of 5 months, but for twice as long in 6-8 and 9-10 year olds.[10]

It took root apices 2 mm in diameter only 6.2 months to shut, compared to 11 months for those > 2 mm in diameter. The clinical procedure may be prolonged, requiring monthly visits during which the calcium hydroxide dressing is removed and a check for wound closure is performed radiographically or clinically by tapping the wound with a paper tip. Apical repair may be finished six months after the placement of calcium hydroxide, but it may take two to three years (Kennedy, 1986). Radiographic proof of root enclosure alone should not lull the operator into a false feeling of security, and an open-ended follow-up approach is prudent.

Apical plug

In cases with an open apex or normal apical anatomy, the dentine chip plug in the periapical tissue has been recommended as an artificial yet biological apical stop against which to compact gutta percha (Tronstad, 1978; El Deeb et al., 1983). The deliberate extrusion of calcium

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11,Iss 6, Sep 2022

hydroxide powder to act as an apical stop itself, allowing gutta percha condensation, has also been recommended with positive clinical outcomes (Coviello and Brilliant, 1979; Pitts et al., 1984).[11-12]Calcium hydroxide packed as a mechanical plug into the apical 2 mm of the tooth followed by laterally condensed gutta percha led to less dye penetration in vitro than gutta percha tilled apices without the plug (Weisenseel et al., 1987).

Root canal sealer

Recently, two commercially developed cilcium hydroxide products have been introduced to the marketplace. Corporation, Akron, Ohio, United States) and Sealapex (Kerr/ Sybron, Romulus, Michigan, United States). Cohenet al. (1985) compared the sealing performance of CRCS to that of Procosol (Star Dental Manufacturing Co., Conshohocken, PA, USA), a zinc oxide-eugenol sealer, and discovered similar clinically acceptable leakage patterns with both materials, although CRCS leakage decreased over time.[14-16] Hovland and Dumsha (1985) reported acceptable and comparable leakage over 30 days for Sealapex, Tubliseal, and Procosol.

Jacobsen et al. (1987) determined that there were no significant variations in leakage between CRCS, Sealapex, and Roth Root Canal Cement (a zinc oxide-eugenol material). Zmener (1987) documented increased but not statistically significant differences in leakage between CRC& Sealapex and Tubliseal with time.[17-19] However, he concluded that this amount of leakage was clinically unsatisfactory. Different approaches for root canal preparation, obturation, and evaluation of results may account for these divergent judgements. In addition, the stability of the apical seal over time is questionable. It has not been determined whether these root canal sealers promote faster healing or a more predictable tissue response than non-calcium hydroxide sealers.

Microleakage demonstrator

Leinfelder et al. proposed a rather innovative application for calcium hydroxide as a microleakage demonstration (1986).[20-21] Based on the solubility and OHrelease, this conclusion was reached. pertaining to these cements In vitro Class V cavities filled with amalgam or Sevriton (De Trey Dentsply, Weymouth, UK) and lined with Dycal were syringed with ice water (pH 7). Detection of subsequent microleakage was accomplished by placing pH paper over the fillings and observing a change in colour. They concluded that the approach was straightforward, biocompatible, rapid, and applicable in vivo. [23-25]This was validated by a subsequent in vivo research (Isenberg et al., 1987).

CONCLUSION

Numerous years of intensive usage of calcium hydroxide in restorative dentistry have spawned extensive study. Despite this, the actual mechanisms involved in calcium transport remain unknown. Hydroxide-induced hard tissue repair is still poorly understood, which may explain for the unpredictability of the results when utilising this substance in any form. Numerous other variables, both local and systemic, may impact the formation of the osteodentine bridge and cementoid. For instance, the amount of a dentinal or periapical infection, the microbial species involved, the degree and kind of pulpal or apical inflammation, and the

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11,Iss 6, Sep 2022

microbial species involved could all affect calcium hydroxide responses. Changes in tissue due to ageing could further modify the healing response to calcium hydroxide.

In addition, antibacterial activity implies a level of cytotoxicity, such that substantial antibacterial activity is typically linked with unpleasant responses during and after therapy (Orstavik and Mjor, 1985). Regarding endodontics, Orstavik (1988) stated that asepsis, interappointment disinfection, and relatively inert filling materials are superior than antibacterial-based techniques. The antibacterial efficacy of calcium hydroxide has been called into question and varies depending on the formulation.

The chemistry, biocompatibility, and antibacterial action of calcium hydroxide formulations appear to require more exploration.

Acknowledgements

REFERENCES

1. Cox C. F. and Bergenholtz F. (1986) Healing sequence in capped inflamed dental pulps of Rhesus monkeys (Macaca mulatta). Znt. Endod. .Z. 19, 113-120.

2. Cvek M. (1974) Treatment of non vital permanent incisors with calcium hydroxide. IV, Periodontal Healing and Closure of the Root Canal in the coronal fragment of teeth with intra alveolar fracture and vital apical fragment. A follow-up. Odontol. Rev. 25,239-246.

3.Cvek M. (1978) A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated crown fracture. J Endod. 4,232-237.

4.Cvek M. (1981) Endodontic treatment of traumatized teeth. In: Andreasen J. 0. (ed.), Traumatic Injuries of the Teeth, 2nd edn. Copenhagen, Munksgaard, chap. 10, pp. 361-363.

5.Cvek M. and Sundstrom B. (1974) Treatment of non-vital permanent incisors with calcium hydroxide. V, Histologic appearance of roentogenographically demonstrable apical closure of immature roots. Odontol. Rev. 25, 379-392.

6.Cvek M., Nord C. E. and Hollender L. (1976) Treatment of non-vital permanent incisors with calcium hydroxide. A clinical, microbiological and radiological evaluation of treatment in one sitting of teeth with mature or immature roots. Odontol. Rev. 27, 93-108.

7.Cwikla J. R. (1972) The vaporisation and capillarity effect of endodontic medicaments. Oral Surg. Oral Med. Oral Pathol. 34, 117-121.

8.Fisher F. J. and McCabe J. F. (1978) Calcium hydroxide base materials. An investigation into the relationship between chemical structure and antibacterial properties. Br, Dent. J. 144, 341-344.

9. Fisher F. J. and Shortall A. C. (1984) Setting calcium hydroxide base materials. Studies on their antibacterial effects in vitro. Br. Dent. .I 157, 133-135.

10. Fitzgerald M. (1979) Cellular mechanics of dentinal bridge repair using 3H-Thymidine. J. Dent. Res. 58, 2198-2206.

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11,Iss 6, Sep 2022

11.Frank A L. (1966) Therapy for the divergent pulpless tooth by continued apical formation. J Am. Dent. Assoc. 72, 87-92.

12.Frank A. L. and Weine F. S. (1973) Nonsurgical therapy for the perforative defect of internal resorption. J. Am. Dent, Assoc. 87, 863-868.

13. Fusayama T., Okuse K. and Hosada H. (1966) The relationships between hardness, discolouration and microbial invasion of human dentine. J Dent. Res. 45, 1033-1046.

14. Fuss Z., Szajkis S. and Tagger M. (1989) Tubular permeability to calcium hydroxide and to bleaching agents. J Endod. 15,362-364.

15.Ghose L. J., Baghdady V. S. and Hikmat B. Y. M. (1987) Apexitication of immature apices of pulpless permanent anterior teeth with calcium hydroxide. J. Endod. 13, 285-290.

16.Glass R. L. and Zander H. A (1949) Pulp healing. J. Dent. Res. 28, 97-107.

17.Goldberg F. and Gurtinkel J. (1979) Analysis of the use of Dycal with gutta-percha points as an endodontic tilling technique. Oral Surg. Oral Med. Oral Pathol. 47,78-82.

18.De Freitas J. F. (1982) Characterisation and aqueous extraction of calcium hydroxide materials. Aust. Dent. .Z. 27, 352-356.

19.Gordon T. M., Ranly D. M. and Boyan B. D. (1985) The effects of calcium hydroxide on bovine pulp tissue: variations in pH and calcium concentration. J. Ended. 11, 156-160.

20. DiFiore P. M., Setterstrom J. A. and Lorton L. (1983) The antibacterial effects of calcium hydroxide apexification pastes on Streptococcus sanguis. Oral Surg. Oral Med.. Oral Pathol. 55, 91-94.

21. Draheim R. N., Titus H. W. and Murrey A J. (1988) A comparison of compressive strength of six cavity base materials. J. Prosthet. Dent. 60, 317-319.

22.Dylewski J. (1971) Apical closure of non vital teeth. Oral Surg. Oral Med. Oral Pathol. 32, 82-89. Eidelman E., Finn S. B. and Koulourides T. (1965) Remineralisation of carious dentine treated with calcium hydroxide. ASDC J. Dent. Child. 32,218-225.

23. El Deeb M. E., El Deeb M., Tabibi A. et al. (1982) An evaluation of the use of amalgam, Cavit, and calcium hydroxide in the repair of furcation perforations. J. Endod. 8,459-463.

24. El Deeb M. E., Thuc-Quyen N. T. and Jensen J. R (1983) The dentinal plug: its effect on confining substances to the canal and on the apical seal. J. Endod. 9, 355-359.

25. Ellerbruch E. S. and Murphy R. A. (1977) Antimicrobial activity of root canal medicament vapours. J. Endod. 3, 189-193. Granath L.