Influence of insulin on the healing of exposed dental pulp after pulp capping: An experimental study in a dog model

Mokhtar A. Al-Anesi | Ashraf M. Abu-Seida | Salma H. El Ashry | Abeer H. Mahran | Ehab S. Abd-Elhamid

1 Department of Endodontic, Faculty of Dentistry, Thamar University, Dhamar, Yemen
2 Department of Surgery, Anesthesiology and Radiology, Faculty of Veterinary Medicine, Cairo University, Giza, Egypt
3 Department of Endodontic, Faculty of Dentistry, Ain Shams University, Cairo, Egypt

Correspondence
Ashraf M. Abu-Seida, Department of Surgery, Anesthesiology and Radiology, Faculty of Veterinary Medicine, Cairo University, Giza - Giza Square. PO: 12211- Egypt.
Email: ashraseida@cu.edu.eg; ashraseida@yahoo.com

Abstract

Background: This study investigates the influence of insulin on pulp tissue healing after pulp capping in diabetic dogs.

Methods: Diabetes mellitus was induced in four dogs, and their blood glucose levels were adjusted by insulin to normoglycemic level in two dogs (normoglycemic group) and to hyperglycemic level in two dogs (hyperglycemic group). Class V buccal cavities were performed in 15 teeth in each diabetic dog and two normal dogs (control group). The three groups (30 teeth each) were divided according to the capping materials into three subgroups (10 teeth each) including: subgroup A - mineral trioxide aggregate (MTA), subgroup B - bioaggregate (BA), and subgroup C - calcium hydroxide (Ca(OH)₂). Each subgroup was divided into two subdivisions according to the evaluation period, subdivision 1: 1 month and subdivision 2: 2 months. Qualitative and quantitative evaluations of the inflammation and dentine bridge formation were assessed histologically.

Results: The hyperglycemic diabetic group exhibited significant higher inflammatory cell count and scores and lower dentine bridge thickness than those of the normoglycemic diabetic and control groups (P < .05). There were no significant differences in these parameters between the normoglycemic diabetic and control groups (P > .05).

Conclusion: Insulin has favourable effects on the pulp tissue healing after pulp capping in diabetic dogs.

KEYWORDS
blood glucose, dental pulp, dentine bridge, diabetes mellitus, hyperglycemia

1 INTRODUCTION

Pulp capping is essential to maintain the function and vitality of the previously exposed pulp-dentine complex. The pulp exposure is usually resulted from a trauma or dental caries. The ideal pulp capping materials should have the ability of preservation of viability and function of the dental pulp, adhesion to the dentine and formation of a reparative dentine bridge, easy applicability and good mechanical properties. Various pulp capping materials have been used such as, calcium hydroxide (Ca(OH)₂), calcium silicate-based...
Calcium hydroxide is one of the most popular capping materials. It has some drawbacks such as its degradation over time, tunnel defects in the reparative dentine bridges, and poor sealing. Other capping materials with excellent chemophysical and biological characteristics such as mineral trioxide aggregate (MTA) and bioaggregate (BA) gain popularity due to their remarkable success compared with Ca(OH)\textsubscript{2}.\textsuperscript{9,10}

Diabetes mellitus (DM) is a metabolic disease resulting from a defect in insulin production, insulin function, or both and leading to a chronic hyperglycemia with disturbances of carbohydrate, fat, and protein metabolism.\textsuperscript{11} These disturbances have negative effects on most of the body’s organs and tissues including the dental pulp. DM leads to many oral signs such as: xerostomia, taste impairment, sialosis, dental caries, periodontal disease, soft-tissue lesions, and fungal infections.\textsuperscript{12} Dental pulp is at high risk for infection because it has limited or no collateral circulation. The impaired vasculature caused by diabetes could interfere in tissue nutrition and pulp repair and could create a microaerophilic state for the development of anaerobic microorganisms. Moreover, diabetic patients present circadian rhythms of pulp sensitivity that differ from those of healthy people.\textsuperscript{11}

This study evaluates the influence of insulin as a treatment of DM on the pulpal tissue healing after direct pulp capping with either MTA, BA, or Ca(OH)\textsubscript{2}.

2 | METHODS

2.1 | Ethical approval

This study was approved by the ethical committee at Faculty of Dentistry, Ain Shams University, Egypt (Protocol No: 17-04-2011-Endo). All international and institutional guidelines for animal use and care were followed up.

2.2 | Animals

The present study was conducted on six healthy mongrel dogs. The dogs were of both sexes, and their age ranged from 2 to 3 years. In each dog, 15 teeth were enrolled in this study. These dogs were randomly divided into three main groups (two dogs, 30 teeth each): group I: normoglycemic diabetic group, group II: hyperglycemic diabetic group, and group III: control group. Each group was randomly subdivided into three subgroups (10 teeth each) according to the capping materials used: subgroup A - treated with MTA, subgroup B - treated with BA, and subgroup C - treated with Ca(OH)\textsubscript{2}. Each subgroup was further subdivided into two subdivisions (five teeth each) according to the observation period: subdivision 1 - 1 month and subdivision 2 - 2 months.

2.3 | Induction of DM

It was induced in groups I and II (four dogs) by slow intravenous injection of a single dose of 100 mg/kg of 5% alloxan monohydrate solution (Alloxan SD Fine-Chem Limited, India).

The occurrence of DM was confirmed on the next day by blood glucose monitoring system (ACCU-CHEK; Roche, Germany). The blood samples were collected from the cephalic vein. Blood glucose level (BGL) >200 mg/dL was used as a threshold level of hyperglycemia. The dogs were daily monitored for their BGL in the morning. The BGL was controlled by adjusting the amount of insulin (Humulin N; Lilly, IN) required to maintain the BGL of the dogs as follows:

- Group I (normoglycemic diabetic group) had normal BGL (less than 180 mg/dL).
- Group II (hyperglycemic diabetic group) had a high BGL (More than 240 mg/dL).

The insulin was injected subcutaneously daily before the operative procedure and throughout the experimental periods. After 2 weeks of DM induction, the operative procedure was conducted.

In group III (control group), the two dogs of this group received no treatment before and after the surgical procedure.

2.4 | Operative procedure

Each dog was anesthetized by subcutaneous injection of atropine sulphate at a dose of 0.05 mg/kg, intravenous injection of xylazine HCl at a dose of 1 mg/kg and intravenous ketamine HCl at a dose of 5 mg/kg. The anesthesia was maintained during the operation by 25 mg/kg incremental doses of 2.5% solution of thiopental sodium.

The teeth were disinfected by 0.5% povidone iodine solution. A class V buccal cavity was prepared approximately 1 mm coronal to the gingival margin with inverted cone carbide (#2) at a high speed and under copious normal saline irrigation until the appearance of pulpal shadow.\textsuperscript{4} Pulp exposure was conducted by a sterile sharp probe, and hemostasis was done by rinsing with sterile saline solution until the physiological hemostasis occurred. The capping materials (MTA, BA, and Ca(OH)\textsubscript{2}) were mixed and
applied as indicated by the manufacturer, and the cavities were closed with glass ionomer. The animals were fed on soft diet along the time of experiment.

2.5 Histopathological examination

The animals were sacrificed using anesthetic overdose after each observation period by using 20 mL of 5% thiopental sodium solution given by rapid intravenous injection. Each capped tooth with its surrounding bone was obtained and fixed in 10% neutral buffered formalin solution for 72 hours. Then, the specimens were decalcified in formic acid 5%. The decalcified specimens were prepared as usual and cut in the buccolingual plane of the tooth main vertical axis through the capping site and the pulp into sections of 5 μm thickness. These sections were stained with hematoxylin and eosin for interpretation of the inflammatory cell response and dentine bridge formation as follows:

2.5.1 Qualitative evaluation of the inflammatory cells

For each slide, four representative fields were analyzed at X40. The selected fields had well observed tissue with good architecture, no artifacts, and highest infiltration of inflammatory cells. The inflammatory reactions were graded according to Accorinte et al. Briefly, score 0: represents normal tissue with none or few scattered inflammatory cells present in the pulp area corresponding to the pulp exposure, score 1: slight inflammatory cell infiltrate, score 2: moderate inflammatory cell infiltrate involving the coronal pulp, and score 3: severe inflammatory cell infiltrate involving the coronal pulp or abscess formation.

2.5.2 Quantitative evaluation of the inflammatory cells

The total number of inflammatory cells of each microscopic field was counted using the image analysis software (Image J; 1.41.NIH, MD). The colour threshold for the inflammatory cells ranged from 38 to 120 pixels.

2.5.3 Dentine bridge formation

The dentine bridge formation was graded as follows: scores 0, 1, and 2 represent no, partial, and complete dentine bridge, respectively.

The dentine bridge thickness was assessed using image analysis software (Image J). A line was drawn across the dentine bridge at the highest thickness and measured.

2.6 Statistical analysis

The data were collected and statistically analyzed using the Statistical Package for Scientific Studies (16.0 software, IBM, NY). The data were presented as mean and standard deviation (SD) values. The Kruskal Wallis test was used to compare between the groups and the materials within the group. Mann-Whitney U test was used to study the changes of time in each group. The Tukey HSD test was used for multiple comparisons between the means. The P-value ≤ .05 was considered significant.

3 RESULTS

3.1 Qualitative findings

The hyperglycemic diabetic group had significant higher mean values of the inflammatory scores than those of the normoglycemic diabetic and control groups after 1 month and 2 months (P < .05).

3.1.1 Group I (normoglycemic diabetic group)

Subdivision 1 (after 1 month)

The mean score values of inflammation were 0.40, 0.40, and 1 in subgroups A, B, and C, respectively. In subgroup A1 (MTA after 1 month), the superficial portion of the pulp exhibited an irregular architecture in the area opposite to the exposure site. Numerous fibroblasts and few inflammatory cell infiltrations were seen opposite to the completely formed dentine bridge (Figure 1A). Abundant and dilated blood vessels were noted in the middle part of the pulp (Figure 1B).

The subgroup B1 (BA after 1 month) exhibited an irregular odontoblastic layer opposite to the exposure site with a complete dentine bridge formation (Figure 1C). There were few inflammatory cell infiltrations in the middle part of pulp (Figure 1D).

In subgroup C1 (Ca(OH)2 after 1 month), a destructed odontoblastic layer opposite to the exposure site with a partial dentine bridge formation was noted (Figure 1E). The pulp had heavily inflammatory cell infiltrates and congested blood vessels. Areas of degenerations and inflammatory cell infiltrates could be seen (Figure 1F).
FIGURE 1  Photomicrographs of the normoglycemic group after 1 month. The MTA subgroup showing the exposure site with a complete dentine bridge formation (A) and several dilated blood vessels (B). The bioaggregate subgroup showing the exposure site with a complete dentine bridge formation (C) and few inflammatory cells (D). The Ca(OH)2 subgroup showing the exposure site with a partial dentine bridge formation (E) and dilated, congested blood vessels and areas of degeneration (F). The images A, C, and E: H&E, X10 while B, D, and F: H&E, X40

Subdivision 2 (after 2 months)
The mean score values of inflammation were 0.20, 0.20, and 0.40 in subgroups A (MTA), B (BA), and C (Ca(OH)2), respectively.

In subgroup A2 (MTA after 2 months), the connective tissue near the exposure site appeared to regain its integrity. Abundant and dilated blood vessels were seen with regeneration of the odontoblastic layer opposite to the formed complete dentine bridge (Figure 2A). Numerous blood vessels were noted with few inflammatory cell infiltrates (Figure 2B).

The subgroup B2 (BA after 2 months) exhibited a complete reparative dentine bridge (Figure 2C), dilated blood vessels and absence of the inflammatory cells (Figure 2D).

The subgroup C2 (Ca(OH)2 after 2 months) exhibited an organized connective tissue, regenerated odontoblastic layer and a complete dentine bridge (Figure 2E). Dilated and congested blood vessels were seen with minimal inflammatory cell infiltrates (Figure 2F).

3.1.2  |  Group II (hyperglycemic diabetic group)

Subdivision 1 (after 1 month)
The mean score values of inflammation were 2, 2.20, and 2.6 in subgroups A, B, and C, respectively.

The subgroup A1 (MTA after 1 month) exhibited a destructed odontoblastic layer opposite to the exposure...
site, heavy inflammatory cell infiltrates, and no dentine bridge formation (Figure 3A). Dilatation and congestion of blood vessels and areas of vacuolar degeneration were also noted (Figure 3B).

The subgroup B1 (BA after 1 month) had a destructed odontoblastic layer without dentine bridge formation (Figure 3C). Heavy inflammatory cell infiltrates, loss of normal structure of the pulp, and vacuolar degenerations were observed (Figure 3D).

The subgroup C1 (Ca(OH)₂ after 1 month) exhibited a destructed odontoblastic layer, pulp degeneration opposite to the exposure site, heavily inflammatory cell infiltrates, and no dentine bridge formation (Figure 3E). Abundant and dilated blood vessels with inflammatory cell infiltrates were observed (Figure 3F).

**F I G U R E 2** Photomicrographs of the normoglycemic group after 2 months. The MTA subgroup showing complete dentine bridge formation (A) and numerous blood vessels with few inflammatory cells (B). The bioaggregate subgroup showing complete dentine bridge formation (C) and dilated blood vessels (D). The Ca(OH)₂ subgroup showing complete dentine bridge formation (E), dilated and congested blood vessels with few inflammatory cell infiltration (F). The images A, C, and E: H&E, X10 while B, D, and F: H&E, X40

**Subdivision 2 (after 2 months)**

The mean score values of inflammation were 1.6, 1.8, and 2.2 in subgroups A, B, and C, respectively.

The subgroup A2 (MTA after 2 months) exhibited an irregular odontoblastic layer, minimal pulp destruction, few inflammatory cell infiltrates, and areas of partial dentine bridge formation (Figure 4A). The pulp appeared to regain its integrity with few inflammatory cell infiltrates (Figure 4B).
Figure 3 Photomicrographs of the hyperglycemic group after 1 month. The MTA subgroup showing the exposure site without dentine bridge formation, heavy inflammatory cell infiltration (A), dilated and congested blood vessels and vacuolar degenerations (B). The bioaggregate subgroup showing the exposure site without dentine bridge formation (C), inflammatory cell infiltration and vacuolar degeneration (D). The Ca(OH)2 subgroup showing the exposure site without dentine bridge formation (E), heavy inflammatory cell infiltration and dilated blood vessels (F). The images A, B, D, and F: H&E, X40 while C and E: H&E, X10

In subgroup B2 (BA after 2 months), destruction of the odontoblastic layer with areas of degeneration was observed opposite to the partially formed dentine bridge (Figure 4C). Inflammatory cell infiltrates and vacuolar degenerations were also noted in the pulp (Figure 4D).

In subgroup C2 (Ca(OH)2 after 2 months), a destructed odontoblastic layer was seen with heavy inflammatory cell infiltrates and no dentine bridge formation (Figure 4E). The pulp had dilated blood vessels and heavily inflammatory cell infiltrates (Figure 4F).

3.1.3 Group III (control group)

Subdivision 1 (after 1 month)
The mean score values of inflammation were 0.0, 0.20, and 0.40 in subgroups A, B, and C, respectively.

In subgroup A1 (MTA after 1 month), regular pulp architecture was seen in the area opposite to the exposure site, a partial degenerated odontoblastic layer and a complete dentine bridge formation (Figure 5A). Dilated and congested blood vessels were noted with few inflammatory cell infiltrations (Figure 5B).

The subgroup B1 (BA after 1 month) had an irregular odontoblastic layer, dilated and congested blood vessels, and a complete bridge formation (Figure 5C). The pulp tissue appeared to regain its integrity with absence of inflammatory cells in the pulp (Figure 5D).

In subgroup C1 (Ca(OH)2 after 1 month), a destructed odontoblastic layer was noted opposite to the exposure site with partial dentine bridge formation (Figure 5E). Dilated blood vessels and few inflammatory cell infiltrates could be noted in the pulp (Figure 5F).

Subdivision 2 (after 2 months)
The mean score values of inflammation were 0.0, 0.0, and 0.2 in subgroups A, B, and C, respectively.

In subgroup A2 (MTA after 2 months), the superficial portion of the pulp appeared to regain its integrity with a well-organized odontoblastic layer. Abundant and dilated blood vessels were noticed opposite to the completely formed dentine bridge (Figure 6A). No
inflammatory cell infiltrates were noted in the pulp (Figure 6B).

The subgroup B2 (BA after 2 months) had an irregular odontoblastic layer, areas of degenerations, and a complete dentine bridge formation (Figure 6C). Few inflammatory cell infiltrates were noted in the pulp (Figure 6D).

The subgroup C2 (Ca(OH)2 after 2 months) had a well-organized and regular odontoblastic layer, and areas of degeneration opposite of the completely formed dentine bridge (Figure 6E). The connective tissue resembled that of the normal pulp. High vasculature with some dilated blood vessels and no inflammatory cells were noticed in the pulp (Figure 6F).

### 3.2 Quantitative findings

#### 3.2.1 Inflammatory cell count

The hyperglycemic diabetic group had significant higher mean values of the inflammatory cell count than those of the normoglycemic diabetic and control groups after 1 month and 2 months ($P < .05$).

The means, SD, and statistical analyses of the inflammatory cell count are presented in Table 1.

There was a significant difference in the mean inflammatory cell count between MTA, BA, and Ca(OH)2 after 1 month and 2 months ($P < .05$). The maximum mean inflammatory cell count was recorded in the subgroup C (Ca(OH)2), followed by that of the subgroup B (BA), then the subgroup A (MTA).

In all groups, all tested subgroups exhibited a decrease in the mean inflammatory cell count by the time. The decrease in the mean inflammatory cell count was significant in all groups after 2 months ($P < .05$).

In group I, the difference in the mean inflammatory cell count between the MTA and Ca(OH)2 was statistically significant ($P = .037$), while the difference between BA and Ca(OH)2 and between MTA and BA was not significant ($P > .05$) after 1 month. After 2 months, there was no significant difference between the tested materials ($P > .05$).
Figure 5  Photomicrographs of the control group after 2 months. The MTA subgroup showing complete dentine bridge formation (A), normal pulp tissue with no inflammatory cell infiltrate (B). The bioaggregate subgroup showing irregularity in the odontoblastic layer, complete bridge formation (C) and few inflammatory cell infiltrations (D). The Ca(OH)₂ showing complete dentine bridge formation (E), dilated blood vessels and no inflammatory cell infiltrate (F). The images A and E: H&E, X10 while B, C, D and F: H&E, X40

In group II, the difference in the mean inflammatory cell count between the MTA and BA subgroups was not significant ($P > 0.05$) after 1 month and 2 months while the difference was significant between the MTA and Ca(OH)₂ and BA and Ca(OH)₂ after 1 month and 2 months ($P < .05$).

In group III, there was no significant difference between all tested materials after 1 and 2 months ($P > .05$).

3.2.2  |  Dentine bridge formation

The hyperglycemic diabetic group had significant lower mean values of the dentine bridge formation than those of the normoglycemic diabetic and control groups after 1 and 2 months ($P < .05$).

In group I, the mean scores of the dentine bridge formation were 1.80, 1.60, and 1.60 in the MTA, BA, and Ca(OH)₂ subgroups after 1 month, respectively. After 2 months, the scores were 2.00, 2.00, and 1.60 in the MTA, BA, and Ca(OH)₂ subgroups, respectively.

In group II, the mean scores of the dentine bridge formation were 0.40, 0.40, and 0.00 in the MTA, BA, and Ca(OH)₂ subgroups after 1 month, respectively. After 2 months, the scores were 1.00, 0.60, and 0.40 in the MTA, BA, and Ca(OH)₂ subgroups, respectively.

In group III, the mean scores of the dentine bridge formation were 2.00, 1.80, and 1.60 in the MTA, BA, and Ca(OH)₂ subgroups after 1 month, respectively. After 2 months, the scores were 2.00, 2.00, and 1.80 in the MTA, BA, and Ca(OH)₂ subgroups, respectively.

The mean values of dentine thickness in micrometers are presented in Table 2. There was a significant difference between the three materials after 2 months in the normoglycemic diabetic group and after 1 month and 2 months in the control group ($P < .05$).

There was a significant difference between 1 month and 2 months subdivisions in the MTA and BA subgroups in both normoglycemic and control groups ($P < .05$).

4  |  DISCUSSION

About 6.4% of the whole population worldwide suffer from DM. This devastating disease leads to tissue or vascular damages that induce several diabetic complications. However, the literature on the pathogenesis, progression,
Photomicrographs of the control group after 2 months. The MTA subgroup showing complete dentine bridge formation (A), normal pulp tissue with no inflammatory cell infiltrate (B). The bioaggregate subgroup showing irregularity in the odontoblastic layer, complete bridge formation (C) and few inflammatory cell infiltrations (D). The Ca(OH)2 showing complete dentine bridge formation (E), dilated blood vessels and no inflammatory cell infiltrate (F). The images A and E: H&E, X10 while B, C, D and F: H&E, X40

and healing after pulp capping in diabetic patients is relatively scarce.\textsuperscript{13} Therefore, this study described the influence of insulin as a treatment of DM on the healing of exposed dogs’ dental pulp capped with three popular capping materials. The dental pulp is a well-vascularized connective tissue that is able to heal after damage, however the diabetic patients are usually susceptible to bacterial or opportunistic infections. Therefore, DM may be a modulating factor of endodontic infections and may interfere the healing of pulpal tissue, particularly in poorly controlled diabetics.\textsuperscript{13}

In the present study, we selected three of the commonly used pulp capping materials in the clinical situations. The mechanism of action of Ca(OH)\textsubscript{2} is attributed directly to its ability of releasing the calcium and hydroxyl ions and alkalinizing of the cavity, which is not suitable for the bacterial growth.\textsuperscript{14} The MTA cement exhibits an acceptable biocompatibility; however, the BA has excellent biocompatibility due to the presence of tantalum oxide as radiopaqueifier.\textsuperscript{15}

The dog was the animal model selected in the present study because the mechanisms of induction and production of dentine are similar to those of the humans but in a short time.\textsuperscript{4,6} Furthermore, the pulp size of dogs allows suitable samples for histopathological evaluation, and the number of teeth allows the comparison of more than one material in the same dog.\textsuperscript{16,17} The animals were fed on soft diet after the pulp capping procedure in order to avoid the loss of restorative material and to minimize the risk of bacterial microleakage.

Pulp exposure was performed by a probe to avoid the extensive pulp damage and to standardize the created exposure sites as mentioned before.\textsuperscript{4,6} The dentine fragments pushed into the pulp induce no inflammatory pulp response.\textsuperscript{17} In addition, the auto-induction of reparative dentinogenesis could be observed on the surface of these fragments.\textsuperscript{18}

Like many previous studies, two evaluation periods were selected in order to assess the primary and late responses of the tissues in this study.\textsuperscript{3,4,6}

The inflammation of the dental pulp in the hyperglycemic diabetic group was higher after pulp capping than that in the normoglycemic and control groups. This could be attributed to the systemic effect of hyperglycemia. The hyperglycemia results in limited collateral circulation that may interfere in tissue nutrition and pulp repair and may
create a microaerophilic state for the growth of anaerobic microorganisms. Moreover, the hyperglycemia increases the catalase activity, decreases the concentration of the anti-oxidant agent (sialic acid), and produces an unsuitable condition for angiogenesis and cellular proliferation. In the normoglycemic diabetic group, the dental pulps responded to the pulp capping procedure as the control group. In this group, the diabetes was controlled by the insulin and consequently better tissue healing and lower inflammation than those of the hyperglycemic diabetic group.

In all groups, the inflammation in both MTA and BA subgroups was lower than that in the Ca(OH)\(_2\) subgroup. This could be attributed to the porosity of the produced dentine bridge, the poor adherence to dentine, and microleakage in the Ca(OH)\(_2\) subgroup. Moreover, there is a minimum chemical difference between the MTA and BA. This could interpret the comparative results of the MTA and BA subgroups in the present study.

In normoglycemic and control groups, most of the samples exhibited dentine bridge formation after 1 month. This is due to the presence of a good environment for healing of the pulp tissue and formation of the dentine bridge. Similar findings were recorded in a clinical study that evaluated the use of pulp capping in controlled diabetics.

The insulin has anabolic effects on the different tissues, enhances the healing by stimulating proliferation, decreases the hypoxia, and helps the angiogenesis in the damaged tissues. This may explain the differences in the histopathological results between the normoglycemic and hyperglycemic diabetic groups in this study. In addition, insulin-like growth factors may play a crucial role in the growth, differentiation, function, and protection of dental pulp cells.

After 2 months, a resolution of inflammation with formation of a dentine bridge in all samples was seen. This could be attributed to the time that allowed the pulp tissue healing and inducing the hard tissue formation. These results are in agreement with a previous study. In contrast, Nair et al postulated that the pulp tissue capped with Ca(OH)\(_2\) still had a degree of inflammation with variable degrees of dentine bridge formation after 3 months. This could be explained by the difference in the study design, where they used human teeth while animal teeth were used in the present study.

Finally, the normoglycemic diabetic group and control group exhibited comparable histopathological findings confirming the ability of the body to heal and repair when favourable conditions and healthy environment are available. The applicability of these findings to clinical situations is that the controlled diabetics are favorable...
candidates for pulp capping than those of uncontrolled conditions.

5 | CONCLUSIONS

Insulin has favourable effects on the pulp tissue healing after pulp capping using MTA, BA, and Ca(OH)₂ regarding both inflammation and dentine bridge formation in the diabetic dogs.

ETHICAL APPROVAL

This study was approved by the ethical committee at Faculty of Dentistry, Ain Shams University, Egypt (Protocol No: 170411-Endo). All international and institutional guidelines for animal use and care were followed up.

CONFICT OF INTEREST

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

ORCID

Ashraf M. Abu-Seida https://orcid.org/0000-0001-5466-2016

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