"TO ASSESS THE BONE REGENERATION IN EXTRACTION SOCKETS USING SIMVASTATIN IN COMPARISON TO ROSUVASTATIN: A RANDOMIZED CONTROL TRIAL"

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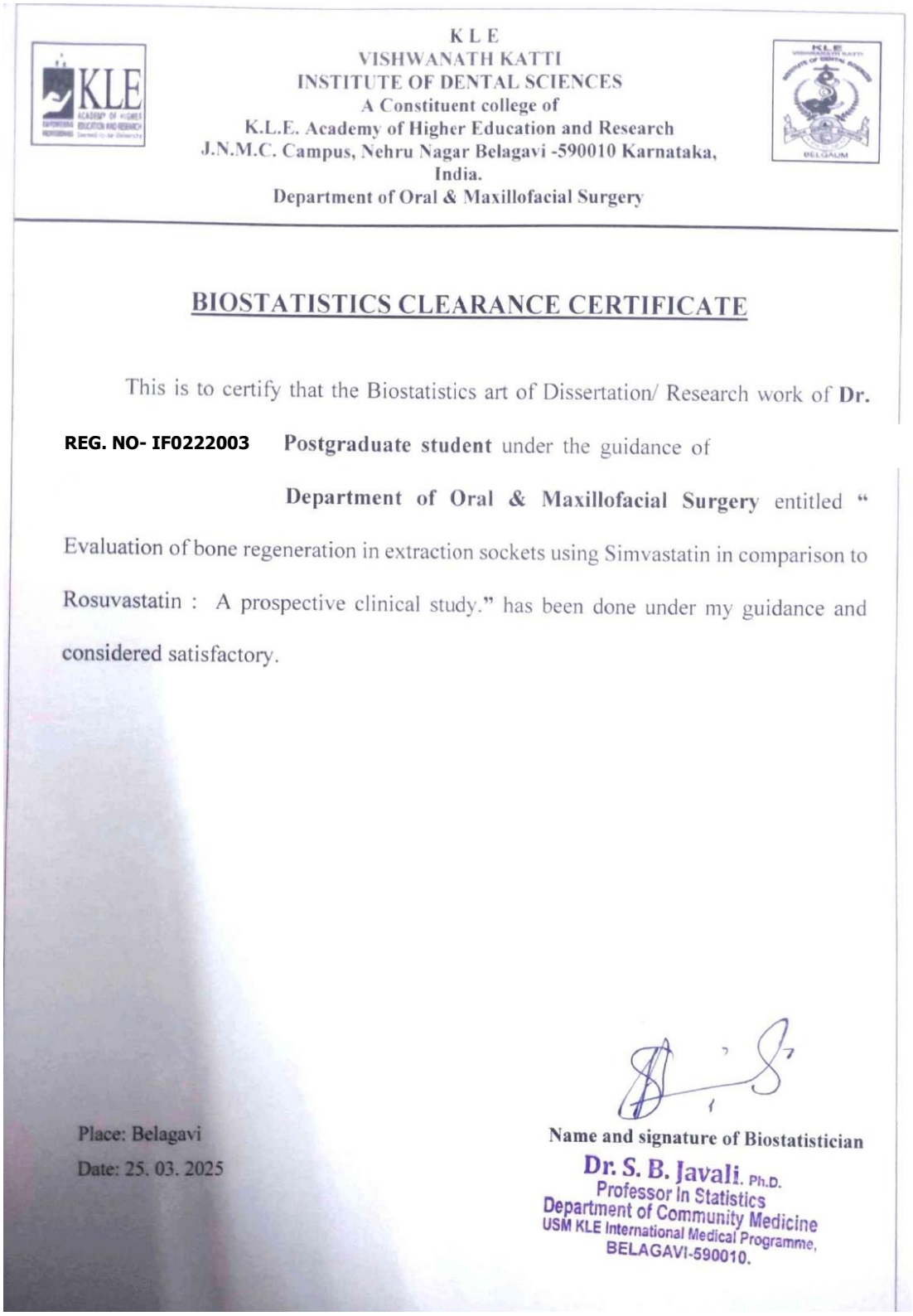

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LIST OF ABBREVIATIONS USED IN THE STUDY

ANOVA	Analysis of Variance (One-Way ANOVA; Kruskal-Wallis ANOVA)
AOBs	Alveolar Bone Osteoblasts
BMP-2	Bone Morphogenetic Protein-2
BMPs	Bone Morphogenetic Proteins
CBCT	Cone-Beam Computed Tomography
FGF-1	Fibroblast Growth Factor-1
FDBA	Freeze-Dried Bone Allograft
HA	Hydroxyapatite
HMG-CoA	3-Hydroxy-3-Methylglutaryl-Coenzyme A
ITB	Implant-Associated Trabecular Bone
MTT	(Tetrazolium) MTT Assay
PDL	Periodontal Ligament
PLA	Polylactic Acid (membrane)
PLGA	Poly (lactic-co-glycolic acid)
PRF	Platelet-Rich Fibrin
RSV	Rosuvastatin
SD	Standard Deviation
TCP	Tricalcium Phosphate
VEGF	Vascular Endothelial Growth Factor
mm	Millimeters

ABSTRACT

STATEMENT OF PROBLEM

Tooth extraction invariably leads to alveolar ridge resorption, compromising masticatory function, esthetics, and future implant placement. Traditional socket-preservation techniques—autografts, allografts, xenografts, and exogenous growth factors—are limited by donor-site morbidity, immunologic risks, infection potential, and inconsistent outcomes. Statins, beyond their lipid-lowering action via HMG-CoA reductase inhibition, have been shown to upregulate bone morphogenetic protein-2 (BMP-2) and vascular endothelial growth factor (VEGF), promoting osteoblast differentiation and angiogenesis. These properties position locally applied statins as promising pharmacologic graft substitutes for preserving post-extraction sockets.

PURPOSE

A randomized clinical trial was designed to assess and compare the efficacy of locally applied Simvastatin versus Rosuvastatin in enhancing alveolar bone regeneration following tooth extraction

METHODOLOGY

Patients requiring extractions were enrolled after informed consent. Extraction sites were randomized by Excel-generated numbers: the study site received Gelfoam impregnated with a crushed tablet of either Simvastatin or Rosuvastatin, while the control group was placed with plain Gelfoam. Atraumatic extractions were performed under 2% lignocaine with 1:100,000 adrenaline,

hemostasis achieved, and sockets closed with single black braided silk sutures. Postoperative care was standardized across all sites. Cone-beam computed tomography (CBCT) scans were obtained immediately post-extraction and at 3 months to measure bone height, coronal/middle/apical widths, and density (gray-value percentage). Postoperative pain was recorded via a 0–10 Visual Analogue Scale on days 1 and 3. One-Way ANOVA evaluated radiographic parameters, and Kruskal-Wallis ANOVA compared pain scores; significance was set at $p < 0.05$.

RESULTS

At 3 months, bone height reduction was significantly less in both statin groups (Simvastatin: 0.24 ± 0.22 mm; Rosuvastatin: 0.32 ± 0.12 mm) compared to control (0.59 ± 0.33 mm; $p=0.0005$), with no statin-to-statin difference ($p=0.597$) . Preservation of bone width and density was likewise superior in statin-treated sites at coronal, middle, and apical levels (all $p < 0.001$) . Postoperative pain scores declined similarly across all groups, with no statistically significant differences at any time point ($p > 0.05$) .

CONCLUSION

Local application of Simvastatin and Rosuvastatin via Gelfoam effectively preserves alveolar bone dimensions after extraction, demonstrating comparable efficacy and offering a low-morbidity alternative for socket preservation.

KEYWORDS

Alveolar bone regeneration; simvastatin; rosuvastatin; socket preservation; CBCT; randomized trial.

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INTRODUCTION

- Tooth loss is a significant dental issue since it impacts chewing ability, speech, appearance, and general well-being. Resultant alveolar bone resorption poses some challenges for later rehabilitation, requiring specific interventions that maintain the structural integrity of the extraction socket. Various regenerative approaches, including autografts, allografts, xenografts, and growth factors, have been used to address this issue. Each method has its advantages and limitations. Despite being the gold standard, autografts present several risks of donor site morbidity, risks of infection, and risks of increased surgical time. Likewise, xenografts still pose a risk for immunological reactions and infections. Thus, the pursuit of a perfect bone graft substitute continues.[1]

- Bone regeneration is a complex process involving cellular and molecular events, including osteoinduction, osteoconduction, and osseointegration. Osteoinduction is stimulated by growth factors like bone morphogenetic proteins (BMPs), and osteoconduction enables proliferation on a scaffold by osteoblasts. Statins are known for lipid-lowering properties. These have become possible agents in bone regeneration since they improve BMP-2 expression.[2]

- Statins exert their diverse effects by inhibiting HMG CoA reductase, the enzyme present in the pathway that biosynthesizes cholesterol. This inhibition results in some increase in BMP-2, as it performs a vital role in bone formation. It stimulates osteoblast differentiation in addition to inhibiting osteoclast activity.[3] Along with BMP-2 induction, statins provoke

angiogenesis through promotion of vascular endothelial growth factor (VEGF), which also plays a part in bone regeneration.[4]

- **Fisher et al. (2019)** showcased the role of osteoclast inhibition and an increase in osteoblast production in statin-mediated bone regeneration. Similarly, **Maeda et al.** discussed the induction of osteoblast differentiation through the initiation of the mevalonate pathway, a crucial step in bone metabolism. Recent reviews have consolidated these findings, emphasizing the need for further clinical trials to establish the efficacy and optimal dosing of statins for bone regeneration.[5]

- Additionally, **Da Hea Seo and Jeong et al.** provided an extensive review of statins and their impact on bone metabolism, underling their importance as promising therapeutic agents for osteoporosis and bone healing.[6]

- Despite the above findings, many challenges need to be addressed before statins can be widely adopted for bone regeneration. One concern is the optimal delivery method. Administration of statins through the systemic route might be effective in lowering cholesterol, but will not achieve sufficient local concentrations for bone regeneration. Local application via gel, coated scaffolds, or impregnated sponges appears to be a more viable approach.[7]

- Statins include readily available Lovastatin, Rosuvastatin and Simvastatin. Simvastatin has been useful in maxillofacial surgery for a decent period of time now.

- Simvastatin, a lipophilic statin, and Rosuvastatin, a hydrophilic statin, have been studied for their role in bone formation.[5]

- Comparative studies between lipophilic and hydrophilic statins have suggested that their differing pharmacokinetics and tissue penetration properties may influence their efficacy in bone regeneration.[8] According to a review conducted by **Luisetto G and Camozzi V**, comparing lipophilic and hydrophilic statins in bone regeneration and concluded that while both classes have osteogenic potential, lipophilic statins like Simvastatin may exhibit superior bioavailability in osseous tissues.[9] However, further controlled trials are required to confirm these findings.

- While Simvastatin has been extensively studied, Rosuvastatin's hydrophilic nature and potential bone regenerative properties warrant further investigation.

- This current study intends to evaluate and contrast the effectiveness of Simvastatin and Rosuvastatin in alveolar bone regeneration following tooth extraction.

AIMS AND OBJECTIVES

- To assess efficacy of Simvastatin and Rosuvastatin for bone regeneration following extraction.

OBJECTIVES:

- To assess pain in the Simvastatin and Rosuvastatin groups
- To evaluate the efficacy of Rosuvastatin and Simvastatin in the bone regeneration in extraction socket radiographically at different times during healing.

REVIEW OF LITERATURE

1. **Mundy et al. (1999)** conducted a study which played a central role in concluding that statins enhance expression of bone morphogenetic protein-2 (BMP-2) that is a key regulator of osteoblast differentiation. Lab studies and animal studies in rodents showed increased bone formation upon administration of statins like simvastatin and lovastatin. These effects were comparable to traditional bone growth stimulators such as BMP-2 and FGF-1. The findings suggest that statins could be used as therapeutic agents for osteoporosis, offering a promising approach to stimulate bone regeneration and increase bone mass.[2]
2. **Bjorn Skoglund et al. (2002)** studied the effects of simvastatin on fracture healing in 81 male mice with standardized femur fractures stabilized via marrow nailing. participating mice were distributed into following groups: one received simvastatin-supplemented diets, while the control group did not. On day 8, calluses in treated mice remained soft, indicating early healing. By day 14, treated mice showed a 53% increase in callus area, 63% higher breaking force, and a 150% increase in energy absorption compared to controls. Histological analysis on day 21 confirmed complete healing. The study concluded that systemic simvastatin significantly enhanced fracture healing by improving both bone strength and structural integrity, indicating its potential anabolic effects on bone regeneration.[10]
3. **Michael R. Thylin et al. (2002)** estimated the osteogenic potential of simvastatin gel in murine calvarial bone, exploring its application in periodontal ligament therapy via two single-dose delivery systems. Mice were divided into five groups: untreated control, methylcellulose gel alone, simvastatin gel, PLA

membrane alone, and PLA membrane with simvastatin gel. Calvarial bones were analyzed histologically to assess new bone formation. Simvastatin gel resulted in a 53% greater bone thickness compared to the group treated with gel alone. When delivered under an occlusive PLA membrane, bone thickness increased by 159–172% and bone area by 144–180%. The study concluded that a single high dose of simvastatin gel, especially under a membrane, significantly enhances localized bone regeneration.[11]

4. **Lars Rejnmark et al. (2004)** also performed a randomized controlled trial for 1 year with the aim of studying the effect of simvastatin on postmenopausal osteopenia bone turnover. The study subjects were 82 healthy postmenopausal women, who were randomly assigned to two groups and were treated with 40 mg/day of simvastatin or placebo for 12 months. Both groups of volunteers were given calcium supplementation (400 mg/day) during the course of the 1.5-year study. Bone mineral density (BDM),plasma lipids, parathyroid hormone (PTH), and biochemical markers of bone remodeling were measured at baseline, after treatment for 12 months, and subsequently again 26 weeks following termination of therapy (week 78). Though effective in lowering cholesterol, simvastatin had no significant effect on bone remodeling or BMD when compared with placebo. The findings were not consistent with even any bone-favorable effect of simvastatin in these patients, suggesting very little therapeutic effect of systemicsimvastatin in the improvement of bone status in osteopenic postmenopausal women.[12]

5. **Jun-Beom Park et al. (2009)** executed a systematic review with the objective to judge the impact of simvastatin across administration forms, dosing, and vehicles by both in vitro and in vivo models. The data showed that simvastatin has a bivalent role in bone metabolism to increase osteoblast activity as well as

to suppress osteoclast activity. In vivo experiments showed that effective promotion of bone formation with simvastatin depends mainly on delivering a maximum concentration locally and on possessing an ideal delivery system. Simvastatin notably enhanced cancellous bone volume and accelerated the rate of bone formation. Moreover, various animal studies reported increased compressive strength of cancellous bone, reinforcing its potential role in promoting bone regeneration. The study emphasized that tailored delivery approaches are essential for maximizing simvastatin's osteogenic effects in clinical applications.[13]

6. **Yongsheng Zhou et al. (2010)** performed an experiment to determine the therapeutic value of simvastatin as an anabolic bone agent for the treatment of trabecular bone induced by implants. The optimal simvastatin concentration was 1 μM as identified by the researchers, citing its effectiveness to a significant rise in secretion of osteocalcin—a critical marker of osteoblastic activity. When applied at this concentration, simvastatin effectively restored critical-size calvarial defects in mice, significantly enhancing bone formation in the defect region. The study concluded that simvastatin, when administered at an optimal dosage, could accelerate osteogenesis in ITB, highlighting its promise as a therapeutic agent for promoting bone regeneration in implant-associated or defect-related scenarios.[14]
7. **Newton Maciel-Oliviera et al. (2011)** have performed an ultrastructural investigation to ascertain the influence of simvastatin on mouse mandible bone healing. It was a standardized surgical defect measuring 0.8 mm in width on the buccal surface of the first lower molar. In the test group, defect received one dose of 2.5% topically applied simvastatin, and the control group was allowed to heal naturally without any intervention. The results demonstrated that the

simvastatin-treated group exhibited enhanced bone quality and a noticeable reduction in bone resorption compared to controls. These findings suggested that a single localized application of simvastatin can positively influence early bone healing and may serve as a promising therapeutic agent for improving bone regeneration in oral surgical sites.[15]

8. **Shutai Liu et al. (2012**) performed an experiment to assess the effect of simvastatin on the osteogenic activity of AOBs and PDL cells. The cells were incubated with different concentrations of simvastatin (1–100 nM) and their viability and proliferation assessed at 24, 48, and 72 hours through the MTT assay. Also, the level of alkaline phosphatase (ALP) activity and mRNA expression level of osteocalcin (OH) and osteoprotegerin were employed to study osteogenic differentiation. Findings suggested that simvastatin dose dependently enhanced the expression of OC mRNA, especially at 1, 10, and 100 nM doses. Moreover, expression of the osteogenic markers in AOBs and PDL cells significantly increased following treatment with simvastatin. It concluded that simvastatin could enhance alveolar bone formation and periodontal tissue regeneration.[16]
9. **Montero J, Manzano G, and Albaladejo A. (2014)** published a systematic review in order to assess the impact of topical simvastatin on bone healing. Comparison of the existing in vivo and in vitro examinations of the osteogenic action of simvastatin when applied locally. The authors found consistent evidence that topical simvastatin enhances bone formation by stimulating osteoblastic activity and suppressing osteoclastic function. The review highlighted that the efficacy of simvastatin in promoting bone regeneration largely depends on the dosage, the method of delivery, and the presence of suitable carriers. Although results varied among studies, the overall conclusion

supported the promising role of locally applied simvastatin as a cost-effective and biocompatible agent for enhancing bone regeneration, especially in periodontal and maxillofacial applications. However, the authors also emphasized the need for further standardized clinical trials to establish optimal dosing protocols and long-term outcomes.[17]

10. **Chauhan et al. (2015)** conducted a clinical trial to assess the efficacy of simvastatin in aids alveolar bone healing after surgical extraction of mandibular third molars. In the present study, the test patients had simvastatin implanted in their extraction sockets group, and the control group received none of these interventions. A total of 30 patients participated, and clinical evaluations were performed over a 3-month follow-up period to monitor healing and bone regeneration. The results demonstrated that the sites treated with simvastatin showed significantly improved bone regeneration compared to the control group. The study concluded that local application of simvastatin enhances bone healing in extraction sockets, suggesting its potential as a therapeutic adjunct in oral surgical procedures to promote faster and more effective alveolar bone regeneration.[18]
11. **A. Pradeep et al. (2016)** performed a double-masked randomized trial to evaluate the effectiveness of a1.2 mg Rosuvastatin in situ gel and a 1:1 combination of autologous Plasma Rich Fibrin (PRF) and Hydroxyapatite bone graft for the treatment of mandibular Class II furcation defects. One hundred five defects were treated with three different modalities: Open Flap Debridement + placebo gel (group 1), PRF + Hydroxyapatite with Open Flap Debridement (group 2), and 1.2 mg Rosuvastatin gel + PRF + Hydroxyapatite with Open Flap Debridement (group 3). The investigation concluded in the fact that use of Rosuvastatin gel, PRF, and Hydroxyapatite combination provided

better clinical and radiographic outcomes when compared to application of Open Flap Debridement alone.[4]

12. **Ibrahim and Fahmy (2016)** developed implantable bioerodible sponges for localized delivery of rosuvastatin to enhance bone healing. Using a factorial design, they prepared twelve lyophilized sponge formulations combining chitosan with various anionic polymers. Characterization techniques confirmed polyelectrolyte complex formation in most combinations. Optimized sponges showed controlled drug release and significantly advanced bone healing in fractured rat femora. Histopathological analysis revealed enhanced bone regeneration in treated specimens. The study concluded that biodegradable sponges delivering rosuvastatin locally could effectively augment bone repair processes.[19]
13. **Türer et al. (2016**investigated the effect of locally administered rosuvastatin (RSV) on mandibular fracture healing in rats. Thirty-two rats were divided into four groups: control and RSV-treated groups at 14- and 28-days post-operation. The RSV-treated groups showed significantly more new bone formation at 2 weeks, but no differences were observed at 4 weeks. The study concluded that Rosuvastatin local application induces bone regeneration in mandibular fracture.[20]
14. **Saifi and Giraddi et al. (2017)** used split-mouth design to analyze the effect of simvastatin on extraction socket bone healing. Fifteen patients received simvastatin mixed with a gelatin sponge inserted into test sites, while control sites had the sponge alone. Radiographs at 2 and 4 months revealed much greater bone density at test sites. The research found that simvastatin accelerates bone healing following extraction.[3]

15. **Noronha Oliveira M (2017)** in a study investigated ridge preservation in maxillary third molar sockets using a composite graft material composed of 30% porosity PLGA/HA/TCP loaded with Simvastatin. The study aimed to assess the synergistic effects of this biodegradable scaffold and Simvastatin on bone regeneration. The results revealed significantly improved bone preservation and regeneration in the experimental group compared to controls. Histologic analysis showed increased new bone formation, enhanced vascularization, and greater osteoblastic activity in the Simvastatin-loaded scaffold group. Radiographic evaluations confirmed better maintenance of ridge height and width, with increased bone density over time. The porous structure of the PLGA/HA/TCP matrix provided a suitable environment for cellular infiltration, while Simvastatin promoted osteoinduction.[15]
16. **Degala and Bathija et al. (2018)** also carried out a split-mouth randomized clinical trial among 30 patients between the age group of 18-40 years to compare the effect of simvastatin on bone regeneration following bilateral impacted mandibular third molar extraction. One socket was treated with 10 mg of simvastatin on Gelfoam soaked in saline, while the other received saline-only Gelfoam as the control. Bone regeneration was assessed through intraoral periapical radiographs and CBCT scans at various time points. The results showed significantly higher bone density in simvastatin-treated sockets compared to controls at all time points. The study concluded that simvastatin enhances osseous regeneration, offering a cost-effective method for post-extraction bone healing.[26]
17. **Sezavar M (2018),** in a study published a study that evaluated the effects of Simvastatin on the quality of dental socket healing following tooth extraction. The primary objective was to determine whether local application of

Simvastatin could enhance bone regeneration and improve the structural integrity of the healing socket. The results showed that sockets treated with Simvastatin exhibited significantly better bone quality, with increased trabecular thickness, bone density, and organized bone formation compared to control sites. Histological assessment confirmed enhanced osteoblastic activity and reduced inflammation in the Simvastatin group.[22]

18. **Pankaj et al. (2018)** carried out a clinical trial of 90 patients to evaluate the effectiveness of 1.2% rosuvastatin (RSV) gel and 1% metformin (MF) gel as adjuncts to scaling and root planing (SRP) in chronic periodontitis management. The patients were divided into three groups: SRP + placebo, SRP + 1.2% RSV gel, and SRP + 1% MF gel. The RSV and MF treatment groups had greater probing depth reduction, clinical attachment level gain, and bone fill than placebo, with the improvement being more with RSV. The research states that RSV gel is superior for periodontal regeneration.[23]
19. **Gouda A (2018)** investigated the efficacy of Simvastatin combined with tricalcium phosphate (TCP) in maxillary sinus lift procedures to enhance bone regeneration. Participants received a combination of Simvastatin and TCP as a scaffold, and results showed significantly improved bone formation compared to TCP alone. Histological analysis revealed increased trabecular bone density, mature bone deposition, and osteoblastic activity. Radiographs confirmed enhanced vertical bone height and volume. The study concluded that Simvastatin in combination with TCP promotes predictable and accelerated bone healing, making it a promising adjunct for maxillary sinus lift surgeries.[24]
20. **Roca-Millan et al. (2019)** created a systematic review and meta-analysis to assess the efficacy of locally applied statins in regenerating non-periodontal bone defects. Analyzing 15 in vivo animal studies involving 546 subjects, the

review found that local statin application significantly enhanced new bone formation, bone density, and healing. Notably, meta-analyses revealed a mean increase in new bone formation of 39.5% at 4–6 weeks and 43.3% at 12 weeks post-application compared to controls. The studies also reported elevated levels of bone morphogenetic protein-2 (BMP-2), vascular endothelial growth factor (VEGF), progenitor endothelial cells, and osteocalcin, indicating both osteogenic and angiogenic benefits. However, the authors cautioned that due to heterogeneity among studies and limited sample sizes, these findings should be interpreted carefully. They emphasized the need for further well-designed clinical trials to validate the potential of statins in bone regeneration.[25]

21. **Gupta, Del Fabbro, and Chang (2019)** created a meta-analysis to investigate the impact of simvastatin on hard tissue, soft tissue, and TM joint cartilage healing in dental therapy. Reviewing ten animal studies and six human clinical trials, they reported that simvastatin treatment was positive in different dental therapies, such as periodontal infection control, bone regeneration, soft tissue grafting, and TMJ cartilage healing. The mechanisms identified include stimulation of bone formation, promotion of soft tissue healing, increased thickness of articular and condylar cartilage, and reduction of inflammation at surgical sites in TMJ disorders. The study concluded that simvastatin is beneficial for healing oral bone and cartilage, but further research is needed to fully understand its potential in soft tissue healing.[26]

22. **Yagobhee.S et al (2020)** carried out a randomized control trial on 12 patients for maxillary sinus augmentation. The study groups were divided into 12 sinuses in each group in which the study group was treated with bovine bone mineral and simvastatin, and the control group was treated with bovine bone mineral. The study was done with a histologic and histomorphometric analysis

to evaluate the effect of Simvastatin on bone healing. Their results indicated that Simvastatin, when used topically, greatly improved new bone formation and quality of reconstructed bone. The test showed higher osteoblastic activity and higher volume of bone in the test group than in controls and indicates that Simvastatin hastens bone remodeling process. This healing process is credited to the drug's capacity to enhance bone morphogenetic protein-2 (BMP-2) expression and stimulate angiogenesis, both essential processes for successful bone healing.[27]

23. **Gupta et al. (2020**) carried out a clinical bone regeneration trial to treat periapical defects. Patients were separated into three groups: one group was given hydroxyapatite, the second group was given platelet-rich fibrin, and the third group was given simvastatin.. In the radiographic analysis done after 12 months, it was noted that the group receiving simvastatin performed significantly better on the periapical index than the other two groups. This signifies that simvastatin has superior regenerative potential in promoting periapical bone healing, likely due to its osteoinductive properties, ability to stimulate BMP-2 expression, and anti-inflammatory effects. These findings support its potential as an effective and non-invasive therapeutic agent in endodontic regenerative procedures.[28]
24. **Özer et al. (2021)** studied locally administered rosuvastatin (RSV) effects on bone formation in a three-dimensional reconstruction rabbit xenograft model. In this study, 16 rabbits underwent calvarial decortication, and titanium caps were placed over the defects. One side received a xenograft combined with RSV (1 mg), while the contralateral side received a xenograft alone, serving as the control. Histological and radiological evaluations at 6 and 12 weeks assessed new bone formation, remaining graft material, and total bone volume. By 12

weeks, sites treated with RSV showed a significantly higher amount of new bone and total bone volume compared to the control group ($p < 0.05$), while no notable differences were observed at the 6-week mark. These results indicate that localized RSV application beneath a titanium barrier progressively enhances bone regeneration.[42]

25. **Abdi et al. (2021)** performed an experiment to compare the efficacy of freeze-dried bone allograft (FDBA) with simvastatin in socket preservation in rats. The study sought to identify whether simvastatin influences bone healing and graft integration. The researchers divided rats into two groups: one group was treated with FDBA alone, while another group received the FDBA and simvastatin treatment. Over a specified healing period, the rats were observed, and outcomes such as bone formation, graft incorporation, and histological changes were assessed. The results demonstrated that the group treated with FDBA and simvastatin exhibited superior bone regeneration and graft integration compared to the control group. This suggests that simvastatin may potentiate the osteogenic properties of bone grafts, offering a promising approach for socket preservation in dental procedures. However, further studies, including clinical trials, are necessary to confirm these findings and establish optimal protocols for simvastatin use in dental applications.[30]

26. Cruz et al. (2021) performed a randomized clinical trial to evaluate the effect of 1.2% simvastatin gel treated with a polypropylene membrane (PPPM) on extraction socket healing. Twenty-six posterosuperior extraction sites were allocated into test group (simvastatin + PPPM) and control group (placebo + PPPM). Both preoperative and 90 days post-extraction CBCT scans were characterized by bone changes of different depths. The simvastatin group demonstrated much less vertical and horizontal bone loss (p =.044,.036,.048,

<.0001). Soft tissue healing, pain, and analgesic consumption were free of significant difference. The combination of simvastatin gel and PPPM was safe in the prevention of bone loss without disturbing patient comfort or healing.[31]

27. **Abu Sheehah HA (2022)** performed a trial to assess the effectiveness of simvastatin in socket preservation following tooth extraction. While the precise number of samples was not stated, patients were allocated to control and test groups, the latter receiving local placement of simvastatin within extraction sockets. Histological and radiographic assessment was done to ascertain healing outcomes. The outcome revealed impressive enhancement of bone regeneration in the sites where simvastatin was applied, as demonstrated by greater new bone formation, greater trabecular architecture, and greater osteoblastic activity. Radiographs revealed less bone resorption and greater socket preservation. These were due to the ability of simvastatin to induce osteoblastic differentiation, angiogenesis, and BMP expression. However, larger-scale clinical trials are required to determine its long-term efficacy and potential comparison with standard therapy.[32]
28. **Diniz et al. (2022)** also carried out a randomized controlled experiment on 29 patients to determine the impact of topical simvastatin on healing of alveolar bone following mandibular third molar removal. Patients received a simvastatin-soaked collagen in one socket and control in the other socket. Significant improvements in trabecular thickness and bone volume were seen in the test group, suggesting enhanced bone regeneration. Inflammatory responses like pain and swelling were higher initially but resolved without complications. The study concluded that topical simvastatin improves bone healing post-extraction. However, further research is needed to explore long-term outcomes and optimal application methods for clinical use.[33]

29. **Harsha G (2023)** assessed the effects of Simvastatin on bone healing after impaction surgery, focusing on its osteoinductive property. Locally applied Simvastatin showed significantly greater bone fill, increased trabecular bone formation, and enhanced bone density compared to controls. These improvements were attributed to Simvastatin's promotion of osteoblastic activity and angiogenesis through the upregulation of bone morphogenetic proteins.[34]
30. **Deepanjali (2023)** evaluated the effectiveness of locally applied Simvastatin in promoting bone reformation after exodontia of mandibular third molar. The study showed that Simvastatin-treated sockets had greater bone fill, higher bone density, and earlier trabecular bone formation. Additionally, these sites exhibited reduced inflammation and faster soft tissue healing. The enhanced regeneration was attributed to Simvastatin's ability to stimulate osteoblast activity, and angiogenesis, and upregulate BMPs and VEGF.[35]
31. **El-Salamouni et al. (2024)** developed a rosuvastatin/calcium carbonate (Ru/CC) nanoparticle formulation to enhance bone regeneration in osteoporotic conditions. The nanoparticles, optimized to 106 nm, were incorporated into a thermosensitive Pluronic gel for sustained drug release. In vivo studies on osteoporotic rats showed significant improvement in bone healing, with enhanced microstructure of bone, increased area of trabecular bone, upregulated osteogenic expression of gene, and reduced osteoclast activity. These findings suggest the Ru/CC gel is a very much able therapeutic approach for osteoporotic bone regeneration.[36]
32. **Diniz et al. (2024)** undertook a scoping review to assess the effect of topical simvastatin on healing of alveolar bone after exodontia, analyzing multiple randomized controlled trials (RCTs). The review found that intra-alveolar

application of simvastatin, using different concentrations and carriers, effectively preserved alveolar bone without significant adverse effects. While seven RCTs evaluated pain outcomes, six reported no significant impact from simvastatin. Among four studies on postoperative swelling, two noted a significant increase in the simvastatin group. Overall, the studies showed positive bone formation and no direct adverse effects linked to topical simvastatin, suggesting its safety and efficacy for enhancing alveolar bone regeneration post-extraction.[37]

33. **Tidke et al. (2025)** conducted a randomized, split mouth, blinded trial to compare the efficacy of simvastatin powder and gel in promoting bone regeneration following third molar extraction. Ten patients received 10 mg of simvastatin powder on one site and gel on the contralateral site. Bone healing was assessed with radiovisiography at 1, 4, 8, and 12 weeks. Both formulations promoted significant bone regeneration, with the highest bone density at 12 weeks. While both were effective, the study highlighted the need for fresh preparation of simvastatin gel and suggested further research into sustained-release systems for improved clinical application.[38]

MATERIALS AND METHODS

Aquisition of data:

The current study was conducted with the institutional ethical committee's consent at the "Department of Oral and Maxillofacial Surgery at KLE Academy of Higher Education's KLE VK Institute of Dental Sciences and Dr. Prabhakar Kore's Basic Science Research Centre, Belagavi, Karnataka, India." Treatment consent was acquired after informing the patients.

INCLUSION CRITERIA:

- Patients between 18 to 65 years requiring tooth extraction.
- Patients willing to give informed and written consent.

EXCLUSION CRITERIA:

- Patients who are medically compromised
- Patients allergic to the drug.

ARMAMENTARIUM:

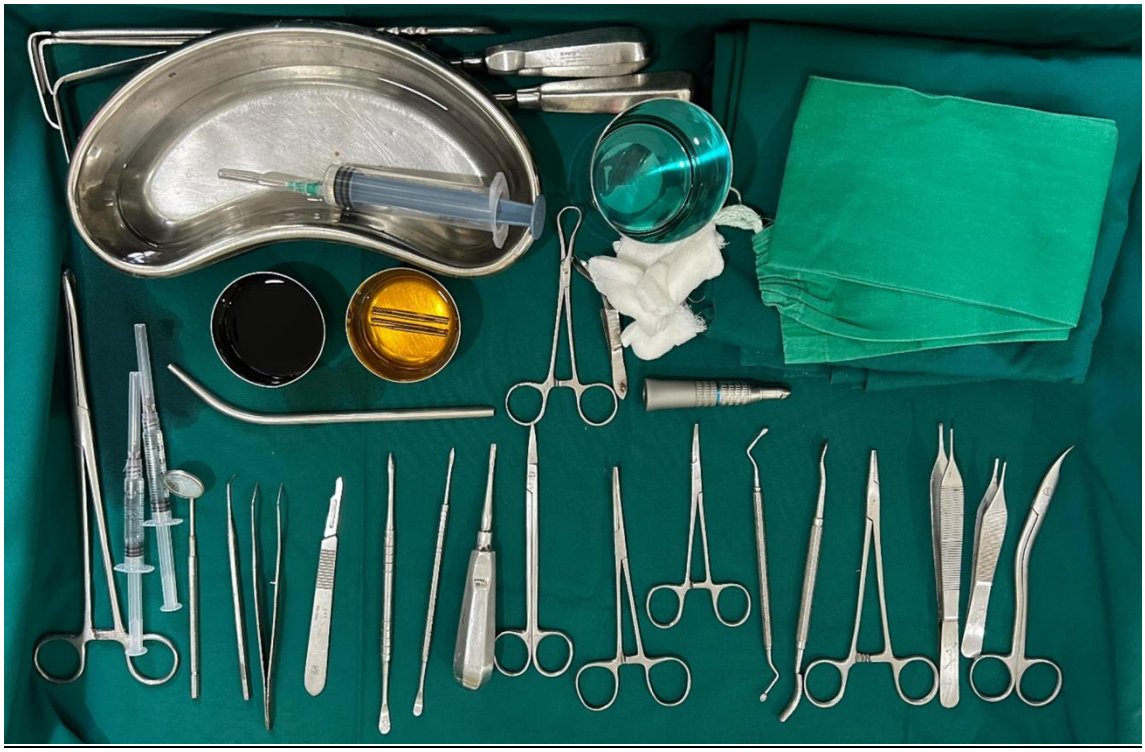


Figure 1: Armamentarium

Materials:

1. Simvastatin 10 mg Tablet (SIMVOTIN 10)
2. Rosuvastatin 10 mg tablet
3. NORMAL SALINE 2 ml
4. ABGEL
5. Instruments for exodontia

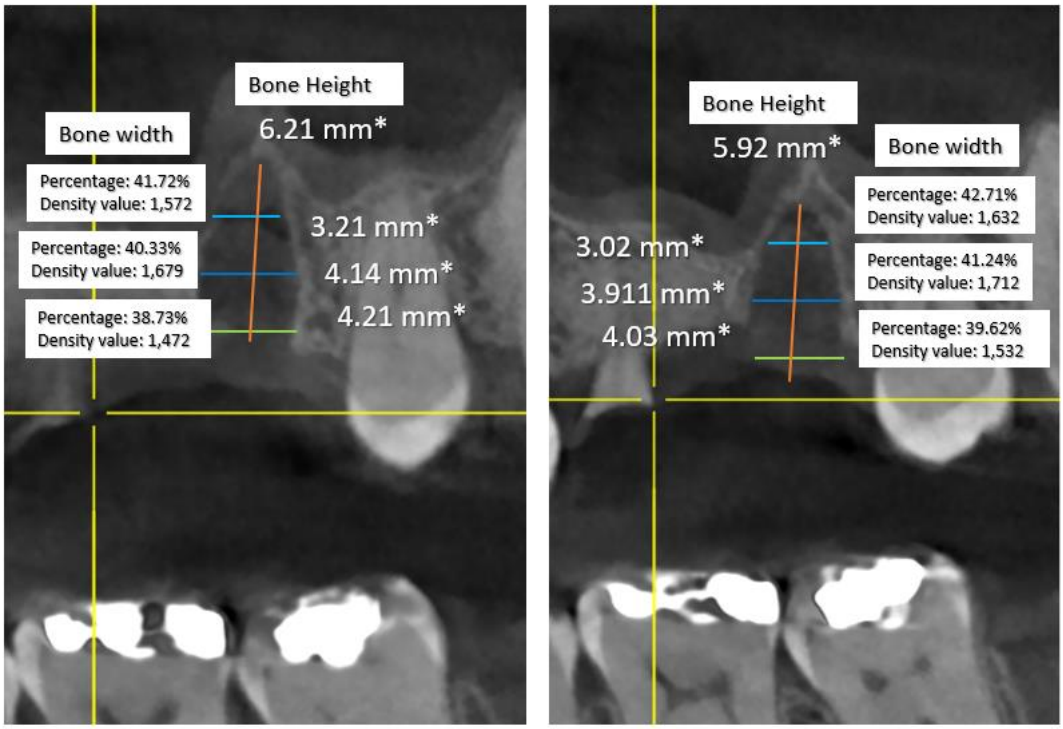


Figure 2. Simvastatin group (pre op and post op bone height, bone width and bone density of the extracted socket of tooth 24)

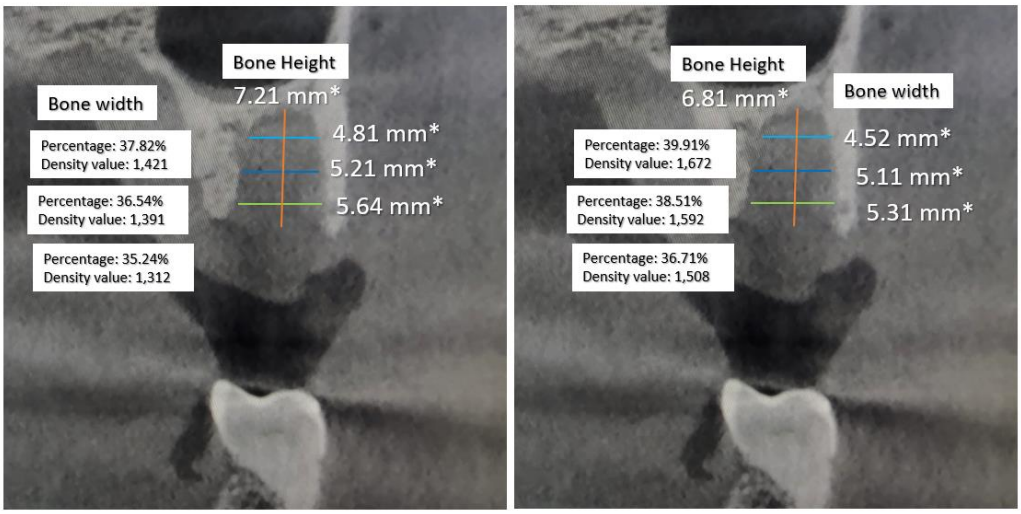


Figure 3. Rosuvastatin group (pre op and post op bone height, bone width and bone density of the extracted socket of tooth 14)

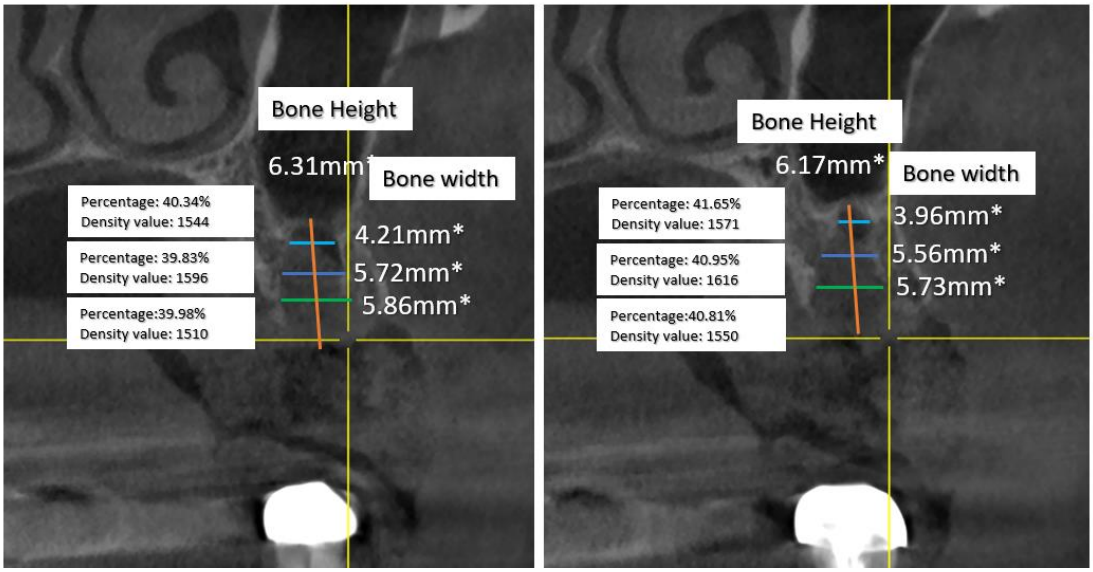


Figure 4. Control group (pre op and post op bone height, bone width and bone density of the extracted socket of tooth 24)

Figure 5. A) Rosuvastatin Tablets, B) Simvastatin Tablets, C) Mortar Pestle

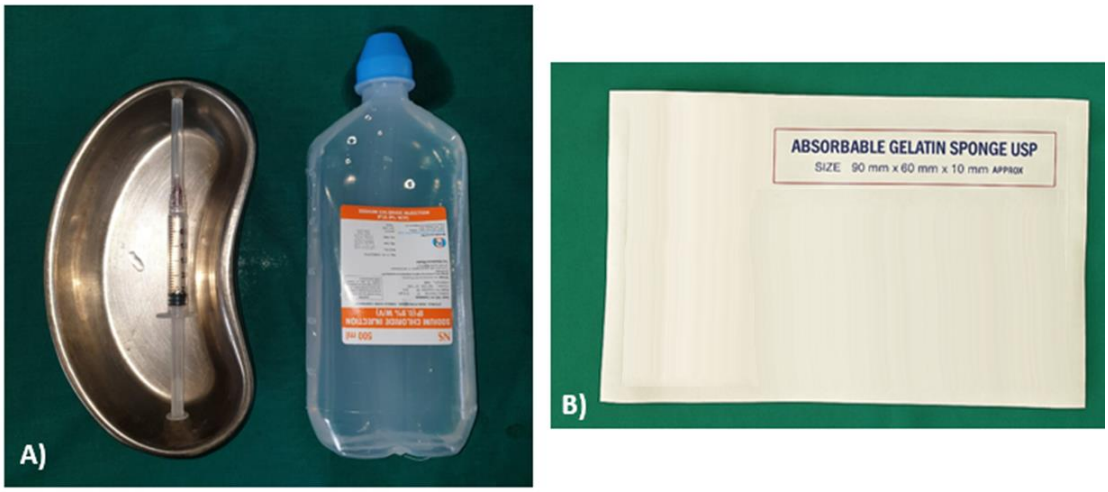


Figure 6. A) 2ml Saline, B) AbGel

METHODOLOGY

All patients underwent a thorough preoperative work up comprising of:

1. Case history
2. Clinical examination
3. Radiographic examination
4. Necessary blood investigations

All patients requiring extractions of advised teeth were identified irrespective of sex. Procedure was explained to every patient and informed consent was also taken from them. Patients were also explained about possible complications.

Study side (simvastatin and rosuvastatin) and control side (Plain Gelfoam) were selected by randomization by number allotment on MS Excel and blinded about the study side and control side.

Extraction of teeth recommended was carried out under local anaesthesia (2% Lignocaine with 1: 1,00,000 adrenaline). Pressure pack was temporarily inserted in extraction socket. One tablet of 'simvastatin' or 'rosuvastatin' was taken out from pack and crushed into powder form of nearly equal grain size by mortar and pestle. Gelfoam was cut as per size required and soaked in 2 ml saline. Water soaked gelfoam was then compressed to dispose of surplus saline. Gelfoam was then charged with powdered tablet. Charged gelfoam was then placed in study site socket. Sockets were checked for hemostasis, then a single suture was tied with black braided silk. Pressure pack was administered and post operative instruction and medications were administered. On the control side normal extraction was done and Gelfoam was placed. Black braided silk suture was placed and pressure pack was given.

EVALUATION CRITERIA

POSTOPERATIVE PAIN:

SCORE	INTENSITY OF PAIN
Range 0	No pain
Range 1-3	Mild pain
Range 4-7	Moderate pain
Range 8-10	Severe pain

Patients were provided with a Visual Analogue Scale with a score of 0-10.

Postoperative pain was assessed at 1st day and 3rd day after tooth extraction.

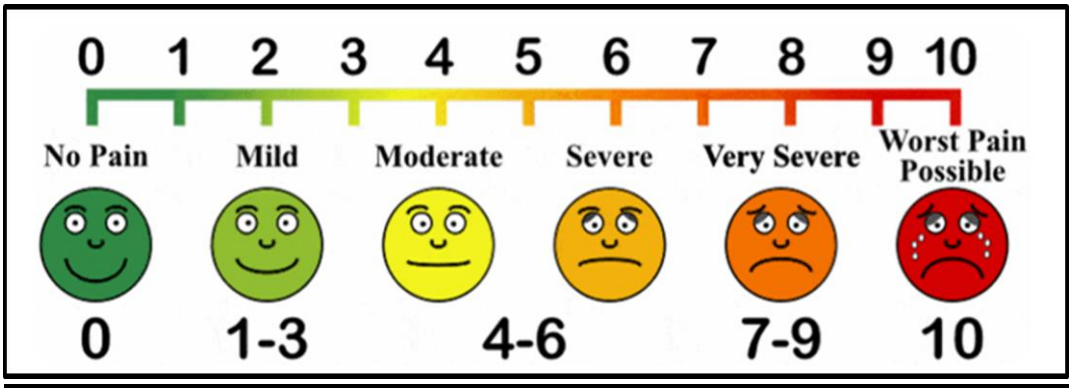


Figure 7:Visual analog scale used to measure the pain

CBCT scanning protocol

1. A cone beam computed tomography (CBCT) scan was performed on each subject to evaluate bone regrowth.

2. CBCT was done soon after graft placement and at the 3-month mark in both study groups 1 and 2. CBCT will be obtained in the control group after the extraction and after the 3rd month.

3. A standardized scanning protocol was followed with all patients.

4. A standardized area was marked in the cone-beam computed tomography of the extracted socket and in sockets placed with the Simvastatin or Rosuvastatin graft. This area was recorded, and its gray value percentage was noted.

5. The gray value percentage of the image denotes the density of that specific area.

6. The CBCT was performed again af ter 3 months from the time of graft placement.

RESULTS

The analysis aimed to assess differences in bone regeneration and post-operative pain across three groups: Control, Rosuvastatin, and Simvastatin.

The data was recorded and then made into a data sheet of 3 different groups on Microsoft Excel.

To compare the three groups, the One-way ANOVA test was used to measure alveolar height, width (coronal, middle, and apical), along with bone density (coronal, middle, and apical). It aided in identifying any statistically significant variations in the groups' mean values. There is a statistically significant difference among the groups if $p < 0.05$.

Kruskal-Wallis ANOVA test was applied to analyze postoperative pain scores over various time intervals. Time Intervals Assessed were 2 hours, 4 hours, 8 hours, 12 hours, 24 hours, 48 hours, and 72 hours post-operatively. A statistically significant H-value with p-value < 0.05 indicates that at least one group differs in median pain scores.

Table 1: Comparison of three groups with pretest and posttest bone height by one way ANOVA

Time point	Control		Rosuvastatin		Simvastatin		F-value	p-value	Comparison of pairs using Tukey's posthoc		
	Mean	SD	Mean	SD	Mean	SD			Control vs Rosuvastatin (P=)	Control vs Simvastatin (P=)	Rosuvastatin vs Simvastatin (P=)
Pretest	6.66	3.30	8.87	1.17	9.24	0.94	6.6838	0.0030*	0.0160*	0.0040*	0.8740
Posttest	6.06	3.05	8.54	1.22	9.00	0.83	9.8025	0.0003*	0.0030*	0.0010*	0.7940
Difference	0.59	0.33	0.32	0.12	0.24	0.22	9.0917	0.0005*	0.0090*	0.0010*	0.5970

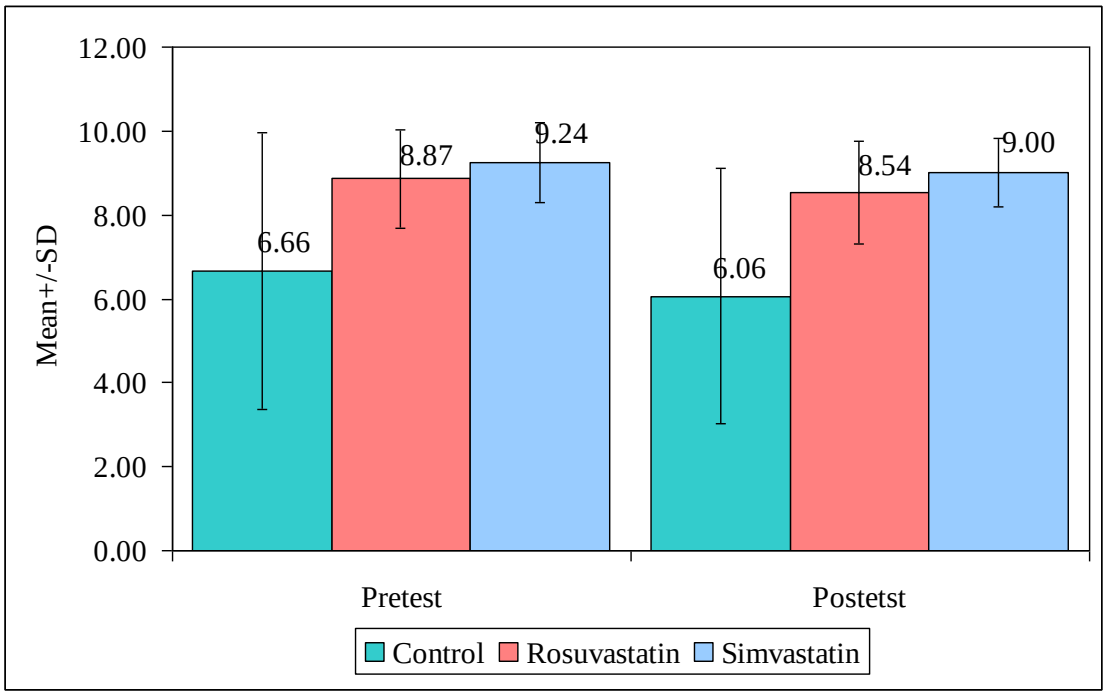
*p<0.05

The comparison of pretest and posttest bone height among the three study groups — Control, Rosuvastatin, and Simvastatin — using One-Way ANOVA revealed statistically significant differences. At the pretest stage, the mean bone height was lowest in the Control group (6.66 ± 3.30 mm) compared to the Rosuvastatin (8.87 ± 1.17 mm) and Simvastatin (9.24 ± 0.94 mm) groups. Tukey's post-hoc analysis revealed significant differences among the Control group and both the Simvastatin (p=0.0040) and Rosuvastatin (p=0.0160) groups, but no significant difference among the Simvastatin and Rosuvastatin groups (p=0.8740). The difference between the groups proved statistically significant (p=0.0030). Additionally, there was a notable difference in bone height across the groups in the posttest evaluation. (p=0.0003). The Control group continued to have the least bone

height (6.06 ± 3.05 mm), whereas the Rosuvastatin and Simvastatin groups maintained higher bone height values of 8.54 ± 1.22 mm and 9.00 ± 0.83 mm, respectively. Pairwise comparison revealed statistically significant differences between the Control group and both statin groups (Rosuvastatin: p=0.0030; Simvastatin: p=0.0010), confirming the positive effect of statins on preserving bone height post-extraction.

Furthermore, when evaluating the difference in bone height from pretest to posttest, the Control group exhibited the greatest reduction (0.59 ± 0.33 mm), while the Rosuvastatin (0.32 ± 0.12 mm) and Simvastatin (0.24 ± 0.22 mm) groups showed minimal bone loss. Pairwise analysis subsequently revealed significant differences between the statin groups and the control group (Simvastatin: p=0.0010; Rosuvastatin: p=0.0090), and this variation was statistically significant (p=0.0005). The Simvastatin and Rosuvastatin groups did not differ significantly (p=0.5970), suggesting that both statins are equally effective at maintaining bone height.

Graph 1: Comparison of three groups with pretest and posttest bone height scores



The graph presents the pretest bone height measurements for the three groups. The Control group exhibited a mean bone height of 6.66 mm (SD = 3.30), which is considerably lower than the values obtained for the rosuvastatin group (8.87 mm, SD = 1.17) and the simvastatin group (9.24 mm, SD = 0.94).Tukey's post-hoc analysis displayed that the rosuvastatin (p = 0.0160) as well as simvastatin (p = 0.0040) groups were different substantially from the Control group, but there was no significant difference among both of the statin groups (p = 0.8740). A one-way ANOVA showed a statistically significant variance between the groups (F = 6.68, p = 0.0030).

The Graph further displays the posttest bone height scores after the intervention. Here, the Control group's mean bone height decreased to 6.06 mm (SD = 3.05), whereas the rosuvastatin and simvastatin groups maintained higher values of 8.54 mm (SD = 1.22) and 9.00 mm (SD = 0.83), respectively. The overall posttest

comparison was highly significant ($F = 9.80$, $p = 0.0003$), with pairwise comparisons confirming that both the rosuvastatin ($p = 0.0030$) and simvastatin ($p = 0.0010$) groups differed significantly from the Control group. Again, no significant difference was observed between the rosuvastatin and simvastatin groups ($p = 0.7940$).

Additionally, the graph shows how bone height changed from the pretest to the posttest. Smaller decreases of 0.32 mm (SD = 0.12) and 0.24 mm (SD = 0.22) were observed in the rosuvastatin and simvastatin groups, respectively, compared to 0.59 mm (SD = 0.33) in the control group. Tukey's analysis showed that the Control group experienced significantly more bone loss than both the rosuvastatin ($p = 0.0090$) as well as simvastatin ($p = 0.0010$) groups, and there was no significant difference between the two statin groups ($p = 0.5970$). The intergroup variation in the alteration was statistically significant ($F = 9.09$, $p = 0.0005$).

Table no. 2: Comparison of three groups with pre-test and post-test Bone width-coronal by one way ANOVA

Time point	Control		Rosuvastatin		Simvastatin		F-value	p-value	Comparison of pairs using Tukey's posthoc		
	Mean	SD	Mean	SD	Mean	SD			Control vs Rosuvastatin (P=)	Control vs Simvastatin (P=)	Rosuvastatin vs Simvastatin (p=)
Pretest	4.90	1.00	4.79	0.51	4.94	0.34	0.2032	0.8169	0.8880	0.9880	0.8150
Posttest	4.53	1.03	4.89	1.02	4.77	0.33	0.6662	0.5190	0.4990	0.7320	0.9240
Difference	0.37	0.25	-0.10	0.85	0.17	0.10	3.1864	0.0500*	0.0440*	0.5430	0.3370

*p<0.05

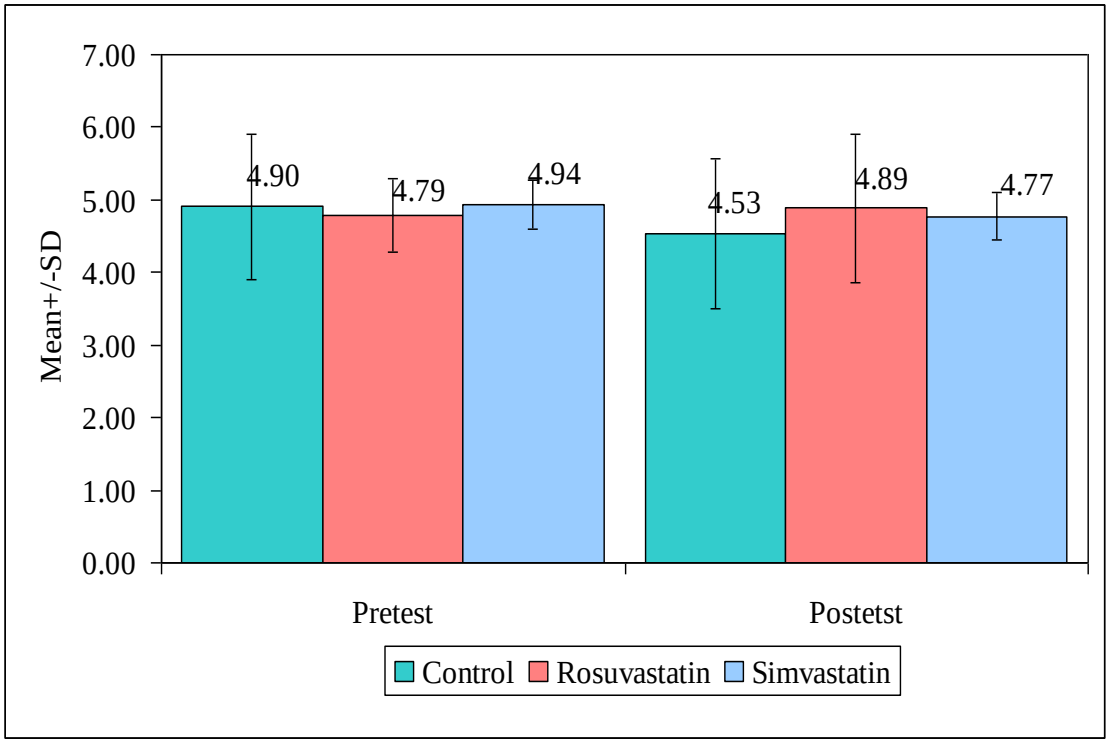
The comparison of coronal bone width among the Control, Rosuvastatin, and Simvastatin groups at both pretest and posttest time points was carried out using one-way ANOVA. In the pretest assessment, the mean bone width at the coronal level was found to be similar across all three groups. The average width of the Simvastatin group was 4.94 ± 0.34 mm, the Rosuvastatin group was 4.79 ± 0.51 mm, and the Control group was 4.90 ± 1.00 mm. Tukey's post-hoc test pairwise comparison failed to indicate statistically significant differences among any of the groups, and the statistical comparison showed no significant distinction between the groups (p=0.8169).

At the post-test evaluation, the Control group exhibited an average coronal bone width of 4.53 ± 1.03 mm, whereas the Rosuvastatin and Simvastatin groups showed mean values of 4.89 ± 1.02 mm and 4.77 ± 0.33 mm, respectively. The group

differences were still statistically non-significant (p = 0.5190), and pairwise comparisons did not reveal any significant differences between the groups.

However, if the pretest to post=test difference in bone width was compared, a statistically significant difference among the groups was present (p = 0.0500). The Control group measured the greatest loss of coronal bone width (0.37 ± 0.25 mm), which showed a higher loss of bone than in the Rosuvastatin group where there was an increase by a mean of 0.10 ± 0.85 mm and in the Simvastatin group where there was a slight decrease of 0.17 ± 0.10 mm. According to a pairwise comparison, the difference among the Control as well as Rosuvastatin groups was statistically significant (p = 0.0440), suggesting that Rosuvastatin was superior to the Control group in terms of retaining or expanding coronal bone width. However, neither the Control nor Simvastatin groups differed significantly (p = 0.5430) nor did the Rosuvastatin and Simvastatin groups (p = 0.3370).

Graph 2: Comparison of three groups with pre-test and post-test Bone width-coronal



The Graph presents the pretest coronal bone width measurements for the three groups. At baseline, the Control group showed an average coronal width of 4.90 mm (SD = 1.00), while the rosuvastatin and simvastatin groups recorded mean values of 4.79 mm (SD = 0.51) and 4.94 mm (SD = 0.34), respectively. One-way ANOVA also failed to show a statistically significant variance among the groups at the second stage of the pretest ($F = 0.2032$, $p = 0.8169$), and the pairwise Tukey's post-hoc comparisons showed that the differences among the groups were not significant (Control vs. Rosuvastatin, p=0.8880; Control vs. Simvastatin, $p = 0.9880$; Rosuvastatin vs. Simvastatin, $p = 0.8150$).

The Graph further displays the post-test measurements for coronal bone width. In this phase, the Control group's mean width reduced to 4.53 mm (SD = 1.03),

whereas the rosuvastatin and simvastatin groups exhibited mean widths of 4.89 mm (SD = 1.02) and 4.77 mm (SD = 0.33), respectively. The differences at post-test also were not of any statistical significance (p = 0.5190), as indicated by the overall ANOVA results and confirmed by the pairwise comparisons (Control vs. Rosuvastatin, p = 0.6662; Control vs. Simvastatin, p = 0.5190; Rosuvastatin vs. Simvastatin, p = 0.4990).

The Graph also illustrates the change in coronal bone width from pretest to post-test. The Control group demonstrated a reduction of 0.37 mm (SD = 0.25), while the rosuvastatin group showed a slight increase of 0.10 mm (indicated by a negative difference of -0.10 mm) and the simvastatin group manifested a reduction of 0.17 mm (SD = 0.10). Analysis of the group differences revealed statistically significant differences (F = 3.1864, p = 0.0500). While the differences among the Control as well as simvastatin groups (p = 0.5430) and among the rosuvastatin and simvastatin groups (p = 0.3370) did not prove statistically significant, Tukey's post-hoc test showed that the determining factor among the Control and rosuvastatin groups proved (p = 0.0440).

Table 3: Comparison of three groups with pre-test and post-test Bone width-middle by one way ANOVA

Time point	Control		Rosuvastatin		Simvastatin		F-value	p-value	Pair wise comparison by Tukeys posthoc		
	Mean	SD	Mean	SD	Mean	SD			Control vs Rosuvastatin (p=)	Control vs Simvastatin (p=)	Rosuvastatin vs Simvastatin (p=)
Pretest	4.10	1.15	4.31	0.46	4.62	0.26	1.9065	0.1612	0.7120	0.1400	0.4880
Posttest	3.78	1.21	4.13	0.48	4.53	0.35	3.5267	0.0384*	0.4410	0.0300*	0.3400
Difference	0.32	0.27	0.18	0.08	0.09	0.12	6.9679	0.0024*	0.0830	0.0020*	0.2960

*$p<0.05$

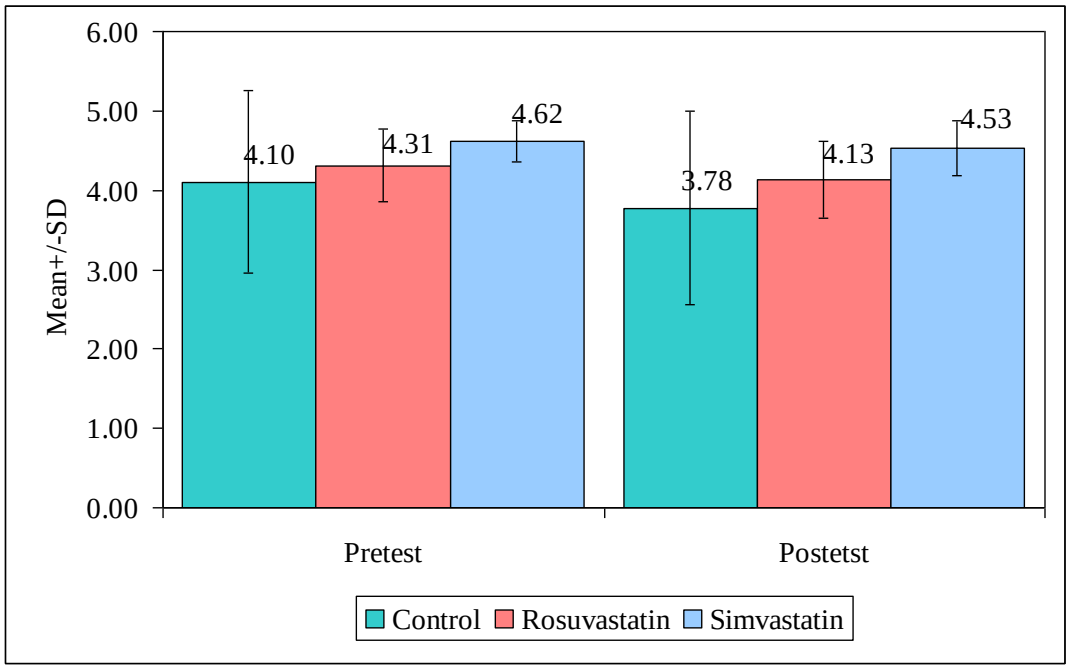
The comparison of bone width at the middle third of the extraction socket among the Control, Rosuvastatin, and Simvastatin groups was analyzed at both pretest and post-test intervals using one-way ANOVA. In the pretest evaluation, the Control group exhibited a mean middle bone width of 4.10 ± 1.15 mm, the Rosuvastatin group showed an average of 4.31 ± 0.46 mm, and the Simvastatin group recorded a mean of 4.62 ± 0.26 mm. The examination revealed that the distinguishing factor between the groups were not of statistical significance (p=0.1612), indicating comparable baseline bone width at the middle level among the three groups.

Following the intervention, the post-test results exhibited a mean bone width of 3.78 ± 1.21 mm in the Control group, while the Rosuvastatin and Simvastatin groups exhibited higher mean values of 4.13 ± 0.48 mm and 4.53 ± 0.35 mm, respectively. The difference in bone width among the three groups at the post-test stage was found of statistical significance (p=0.0384). Tukey's post-hoc analysis of pairwise comparisons between the Simvastatin and Control groups showed a

statistically significant difference (p=0.0300), indicating that Simvastatin was superior to the Control group in preserving alveolar width at the middle level. Nevertheless, there was no discernible difference between the Simvastatin and Rosuvastatin groups (p = 0.3400) or among the Control as well as Rosuvastatin groups (p = 0.4410).

After the consideration of bone width reduction from pretest to post-test, the Control group suffered the largest bone width reduction at the middle third (0.32 ± 0.27 mm), the Rosuvastatin group (0.18 ± 0.08 mm), and the Simvastatin group (0.09 ± 0.12 mm) showed the least reduction. This reduction was statistically significant (p=0.0024). There was no statistically significant difference among the Control as well Rosuvastatin groups (p=0.0830) or among the Rosuvastatin and Simvastatin groups (p=0.2960.00), but a pairwise comparison revealed a statistically significant difference among the Control and Simvastatin groups (p=0.0020), indicating the greater efficacy of Simvastatin in maintaining bone width at the middle third of the socket.

Graph 3: Comparison of three groups with pretest and posttest Bone width-middle



The three groups' pre-test middle bone width values are displayed in the graph. The Control group's average middle bone width at baseline was 4.10 mm (SD = 1.15), while the rosuvastatin as well as simvastatin groups had mean values of 4.31 mm (SD = 0.46) and 4.62 mm (SD = 0.26), respectively. Tukey post-hoc tests revealed no statistically significant group disparities (Control in comparison to Rosuvastatin, p = 0.7120; Control compared to Simvastatin, p = 0.1400; Rosuvastatin compared to Simvastatin, p = 0.4880), and One-way ANOVA did not find any differences of statistical significance between groups (F = 1.9065, p = 0.1162).

The Graph further displays the post-test measurements for middle bone width. Following the intervention, the Control group's mean value decreased to 3.78 mm (SD = 1.21), while the rosuvastatin and simvastatin groups showed means of 4.13 mm (SD=0.48) and 4.53 mm (SD = 0.35), respectively. Here, the overall comparison

became statistically significant (F = 3.5267, p = 0.0384).Pairwise comparisons showed that while there was no significant difference among the rosuvastatin as well as simvastatin groups (p = 0.3400), the disparity between the Control as well as simvastatin groups attained significance (p = 0.0300), while the determining factor among the Control and rosuvastatin groups did not prove statistically significant (p = 0.4410).

The graph also shows how the breadth of the middle bone changed from the pretest to the posttest. The simvastatin group had the least reduction at 0.09 mm (SD = 0.12), the rosuvastatin group showed a drop of 0.18 mm (SD = 0.08), and the control group saw a reduction of 0.32 mm (SD = 0.27). Tukey's post-hoc analysis showed a significant variance between the Control as well as simvastatin groups (p=0.0020), a difference among the Control and rosuvastatin groups that was close to significance (p=0.0830), and a significant indifference between the two statin groups (p=0.2960). This difference in the degree of change between the groups was of statistical significance (F = 6.9679, p = 0.0024).

Table no. 4: Comparison of three groups with pre-test and post-test Bone width-apical by one-way ANOVA

Time point	Control		Rosuvastatin		Simvastatin		F-value	p-value	Pair wise comparison by Tukeys posthoc		
	Mean	SD	Mean	SD	Mean	SD			Control vs Rosuvastatin (p=)	Control vs Simvastatin (p=)	Rosuvastatin vs Simvastatin (p=)
Pretest	3.39	1.31	4.06	0.42	4.30	0.28	5.0593	0.0107*	0.0730	0.0100*	0.6980
Posttest	3.07	1.28	3.85	0.42	4.06	0.29	6.4513	0.0036*	0.0280	0.0040*	0.7450
Difference	0.32	0.23	0.21	0.13	0.24	0.14	1.5625	0.2216	0.2170	0.4200	0.9030

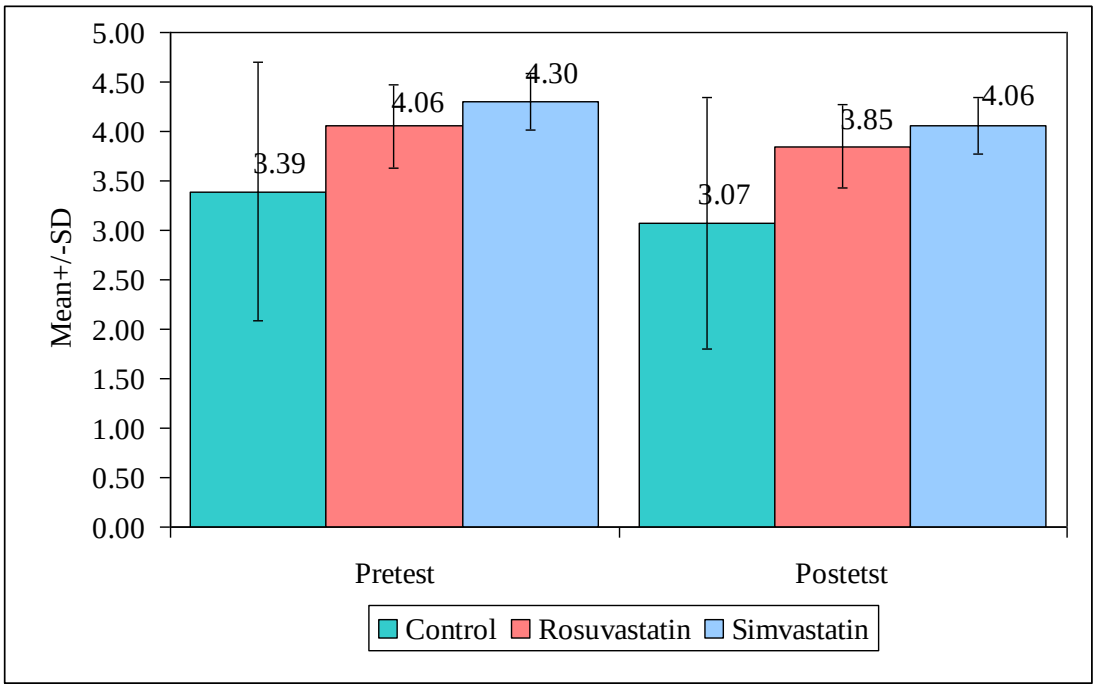
*p<0.05

The comparison of bone width at the apical third of the extraction socket among the Control, Rosuvastatin, and Simvastatin groups was assessed at pretest and post-test intervals using one-way ANOVA. In the pretest evaluation, the mean apical bone width in the Control group was 3.39 ± 1.31 mm, while the Rosuvastatin group showed a mean of 4.06 ± 0.42 mm, and the Simvastatin group exhibited the highest mean bone width of 4.30 ± 0.28 mm. The comparison showed a statistically significant difference between the three groups (p=0.0107). Pairwise comparison employing Tukey's post-hoc test demonstrated a big difference among the Control as well as Simvastatin groups (p-value = 0.0100), indicating a higher bone width in the Simvastatin group. However, significant indifference was observed among the Control as well as Rosuvastatin groups (p=0.0730) or between the Rosuvastatin and Simvastatin groups (p value=0.6980).The Control group's post-test apical bone width mean was 3.07 ± 1.28 mm, whereas the Simvastatin and Rosuvastatin groups maintained higher values of 4.06 ± 0.29 mm and 3.85 ± 0.42 mm, respectively. The three groups' differences were nonetheless statistically significant (p=0.0036). Both

Rosuvastatin and Simvastatin were effective in maintaining apical bone width in contrast to the Control group, as evidenced by the statistically significant disparity among the Control and Simvastatin groups (p-value=0.0040) as well as between the Control along with Rosuvastatin groups (p=0.0280) found by pairwise comparison. The Simvastatin and Rosuvastatin groups did not differ significantly, though (p=0.7450).

The Control group had the largest mean reduction in bone width from the pretest to the posttest, measuring 0.32 ± 0.23 mm. The Simvastatin group came in second with a decrease of 0.24 ± 0.14 mm, whereas the Rosuvastatin group came in third with a decrease of 0.21 ± 0.13 mm. According to the one-way ANOVA, the disparities were not statistically significant (p=0.2216), even though the Control group had more bone loss at the apical third than the other groups. Pairwise comparisons also verified that there were no appreciable variations in the groups' reductions in apical bone width.

Graph4: Comparison of three groups with pre-test and post-test Bone width-apical



The Graph presents the pretest bone width-apical measurements for the three groups. The Control group exhibited an average apical width of 3.39 mm (SD = 1.31), which was lower than 4.06 mm (SD = 0.42) for the rosuvastatin group and 4.30 mm (SD = 0.28) for the simvastatin group. These variations were statistically significant, according to the one-way ANOVA (F = 5.0593, p = 0.0107). The differences among the Control and simvastatin groups were statistically significant (p = 0.0100) in the following pairwise comparisons, but not among the Control and rosuvastatin groups (p = 0.0730), whereas there was no statistically significant difference among the rosuvastatin along with simvastatin groups (p = 0.6980).

The Graph further displays the post-test apical bone width scores. At this stage, the Control group's mean apical width decreased to 3.07 mm (SD = 1.28), whereas the rosuvastatin and simvastatin groups exhibited higher post-test values of

3.85 mm (SD = 0.42) and 4.06 mm (SD = 0.29), respectively. The overall one-way ANOVA remained highly significant (F = 6.4513, p = 0.0036). According to pairwise comparisons, the Control group was substantially different from the simvastatin group (p = 0.0040) and the rosuvastatin group (p = 0.0280). Nevertheless, there was no statistically significant difference between the simvastatin and rosuvastatin groups (p = 0.7450).

Additionally, the graph shows how the apical bone width changed from the pretest to the post-test. The average reduction for the Control group was 0.32 mm (SD = 0.23), 0.21 mm (SD = 0.13), and 0.24 mm (SD = 0.14), respectively, for the rosuvastatin and simvastatin groups. The one-way ANOVA (F = 1.5625, p = 0.2216) with comparisons of pairs (Control compared. rosuvastatin, p = 0.2170; Control compared. simvastatin, p = 0.4200; rosuvastatin compared. simvastatin, p = 0.9030) showed that the overall disparity in the effect was not statistically significant.

Table no. 5: Comparison of three groups with pre-test and posttest Bone density-coronal by one way ANOVA

Time point	Control		Rosuvastatin		Simvastatin		F-value	p-value	Pair wise comparison by Tukeys posthoc		
	Mean	SD	Mean	SD	Mean	SD			Control vs Rosuvastatin (p=)	Control vs Simvastatin (p=)	Rosuvastatin vs Simvastatin (p=)
Pretest	37.78	6.57	51.29	5.66	53.76	2.32	41.2919	0.0001*	0.0001*	0.0001*	0.4000
Posttest	34.46	9.85	49.96	5.47	52.03	2.59	31.0286	0.0001*	0.0001*	0.0001*	0.6760
Difference	3.33	6.38	1.33	0.54	1.73	0.88	1.2095	0.3085	0.3150	0.4750	0.9530

*p<0.05

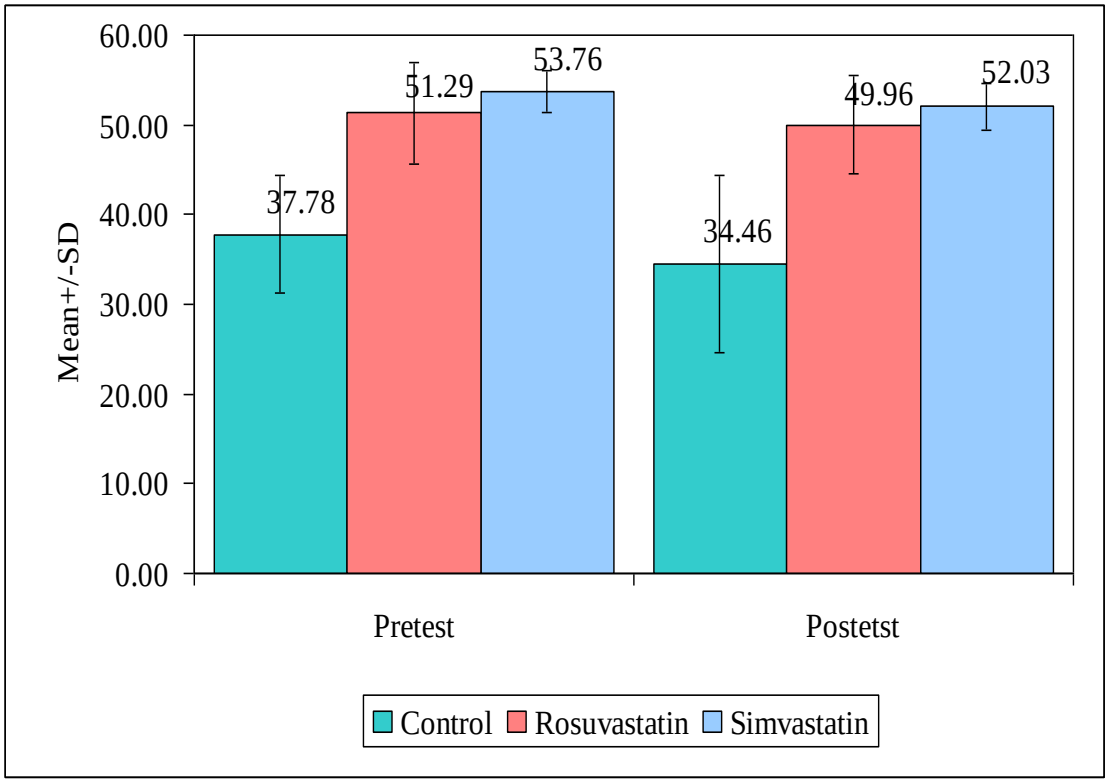
The comparison of bone density at the coronal third of the extraction socket among the Control, Rosuvastatin, and Simvastatin groups was analyzed at both pretest and post-test intervals using one-way ANOVA. In the pretest evaluation, the mean bone density of the Control group was recorded as 37.78 ± 6.57 %, which was considerably lower compared to the Rosuvastatin group (51.29 ± 5.66 %) and the Simvastatin group (53.76 ± 2.32 %). The statistical analysis revealed an extremely significant difference between the three groups (p=0.0001). Tukey's post-hoc pairwise contrast also showed statistically significant difference between Control and Rosuvastatin groups (p=0.0001) and between Control and Simvastatin groups (p=0.0001), determining that both statin groups had higher bone density values compared to the Control group. However, no significant difference was found between Rosuvastatin and Simvastatin groups (p=0.4000).

Post-test evaluation showed a similar trend, with the Control group demonstrating a reduced mean bone density of 34.46 ± 9.85 %, whereas the

Rosuvastatin and Simvastatin groups maintained higher bone density values of 49.96 ± 5.47 % and 52.03 ± 2.59 %, respectively. Differences between groups were statistically significant (p=0.0001). The superior performance of both statins in preserving coronal third bone density was once again confirmed by pairwise comparison, which showed statistically significant differences among the Control group along with the Simvastatin group (p=0.0001) and among the Control group as well as the Rosuvastatin group (p=0.0001). The Simvastatin and Rosuvastatin groups did not vary statistically significantly (p=0.6760).

The Control group had the largest variation in bone density from the pretest to the posttest (mean reduction of 3.33 ± 6.38 percent), followed by that of the Simvastatin group (1.73 ± 0.88 percent) along with the Rosuvastatin group (1.33 ± 0.54%). The intergroup assessment of these variations, however, revealed a lack of statistical significance (p=0.3085), indicating that while the Simvastatin and Rosuvastatin groups showed less bone density loss than the Control group, the three groups' levels of change were not significantly different from one another.

Graph 5: Comparison of three groups with pretest and posttest Bone density-coronal



The Graph represents the pretest bone density scores at coronal level among the three groups. The mean value of 37.78% (SD = 6.57) of the Control group was found significantly lower when compared to rosuvastatin with a mean value of 51.29% (SD = 5.66) and simvastatin with a mean value of 53.76% (SD = 2.32). One-way ANOVA concluded that the groups were different significantly (F = 41.29, p = 0.0001), and using Tukey's post-test, it was concluded that the statin as well as Simvastatin and the statin groups did not differ significantly from one another (p = 0.4000), although they were both substantially distinct from the control group (both p = 0.0001).

The Graph further displays the post-test bone density values at the coronal level. At this stage, the Control group's mean density decreased to 34.46% (SD =

9.85), whereas the rosuvastatin and simvastatin groups maintained higher values, with means of 49.96% (SD = 5.47) and 52.03% (SD = 2.59), respectively. The overall comparison remained highly significant with one-way ANOVA (F = 31.03, p = 0.0001). Pairwise comparisons confirmed that both the rosuvastatin (p = 0.0001) and simvastatin (p = 0.0001) groups differed significantly from the Control group, while the difference between the rosuvastatin and simvastatin groups was not statistically significant (p = 0.6760).

The Graph also illustrates the change in bone density from pretest to posttest. The reduction observed in the Control group was 3.33% (SD = 6.38), which was notably greater than the reductions in the rosuvastatin group (1.33%, SD = 0.54) and the simvastatin group (1.73%, SD = 0.88). However, the overall difference in the change among the groups did not reach statistical significance, as indicated by the one-way ANOVA (F = 1.21, p = 0.3085).

Table 6: Comparison of three groups with pretest and posttest Bone density-middle by one way ANOVA

Time point	Control		Rosuvastatin		Simvastatin		F-value	p-value	Pair wise comparison by Tukeys posthoc		
	Mean	SD	Mean	SD	Mean	SD			Control vs Rosuvastatin (p=)	Control vs Simvastatin (p=)	Rosuvastatin vs Simvastatin (p=)
Pretest	39.43	7.83	50.58	5.65	54.35	1.68	28.1766	0.0001*	0.0001*	0.0001*	0.1740
Posttest	35.62	11.42	49.16	5.54	53.20	1.79	23.2383	0.0001*	0.0001*	0.0001*	0.3040
Difference	3.81	9.54	1.41	0.52	1.14	0.46	1.0566	0.3567	0.4670	0.3920	0.9900

*p< 0.05

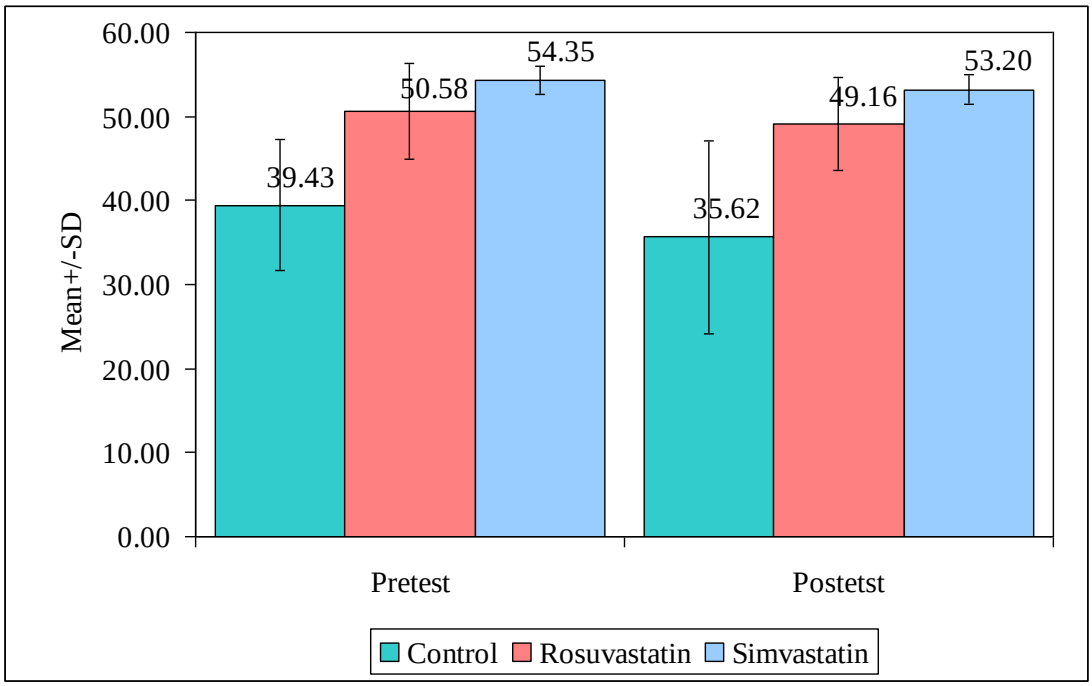
The comparison of the bone density at the middle third of the extraction socket among the Control, Rosuvastatin, and Simvastatin groups was performed at both pretest and posttest intervals using one-way ANOVA. During pretest measurement, the mean bone density of the Control group was 39.43 ± 7.83 %, which is much lower than Rosuvastatin group (50.58 ± 5.65 %) and Simvastatin group (54.35 ± 1.68 %). The three groups' differences were statistically significantly different, according to the one-way ANOVA (p=0.0001). Additionally, statistically significant variations in bone density were found between the Control group and the Simvastatin group (p=0.0001) as well as the Control group and the Rosuvastatin group (p=0.0001) using Tukey's pairwise post-hoc comparison. However, the Simvastatin and Rosuvastatin groups did not vary statistically significantly (p=0.1740).

At post-test testing, the control group had a mean bone density loss of 35.62 ± 11.42 percent, the Rosuvastatin group had a mean of 49.16 ± 5.54 percent, and the

Simvastatin group had the highest average of 53.20 ± 1.79 percent. The three groups' differences were statistically very significant, according to the between-group test (p=0.0001). The statistical difference between the Simvastatin and Control (p=0.0001) and Rosuvastatin and Control (p=0.0001) groups was demonstrated by the Tukey post-hoc test, suggesting that both statins had a superior ability to maintain bone density in the middle portion of the socket. However, the difference between the Simvastatin and Rosuvastatin groups was not statistically significant (p=0.3040).

Mean reduction in bone density from pretest to posttest was greatest in the Control group (3.81 ± 9.54 %), whereas the Rosuvastatin group showed a smaller reduction (1.41 ± 0.52 %), and the Simvastatin group exhibited the least reduction (1.14 ± 0.46 %). However, statistical comparison of the differences between the three groups was not significant (p=0.3567), i.e., even if bone density loss was less in the two statin groups when compared to the Control group, the difference was not significant.

Graph 6: Comparison of three groups with pretest and posttest Bone density-middle



The graph presents the pretest bone density measurements at the middle level for the three groups. At baseline, the Control group had a mean density of 39.43% (SD = 7.83), significantly lower than the mean of 50.58% (SD = 5.65) for the rosuvastatin group and 54.35% (SD = 1.68) for the simvastatin group. Tukey's post-hoc tests showed that the rosuvastatin and simvastatin groups had been significantly different compared to the Control group (p = 0.0001 for either), however there was not a statistically significant distinction between the two statin groups. One-way ANOVA reiterated a highly significant difference among the groups (F = 28.18, p = 0.0001).

The graph further displays the posttest bone density measurements at the middle level. Following the intervention, the Control group's mean density decreased to 35.62% (SD = 11.42), while the rosuvastatin and simvastatin groups maintained

higher values of 49.16% (SD = 5.54) and 53.20% (SD = 1.79), respectively. At this stage, the total one-way ANOVA was highly significant (F = 23.24, p = 0.0001), while pair analysis revealed that neither the simvastatin nor the rosuvastatin groups differed significantly compared to the Control group (p = 0.0001 for either), but again, there was no significant difference among the two statin groups (p = 0.3040).

The graph also displays the alteration in bone density from the pretest to the posttest. The rosuvastatin and simvastatin groups experienced smaller declines of 1.41% (SD = 0.52) and 1.14% (SD = 0.46), respectively, while the control group experienced a mean decline of 3.81% (SD = 9.54). However, despite significant disparities at baseline and posttest, the statistical difference in the alteration among the groups did not prove significant (F = 1.06, p = 0.3567), indicating that the intermediate level of bone density loss was the same for all groups.

Table 7: Comparison of three groups with pretest and posttest Bone density-apical by one way ANOVA

Time point	Control		Rosuvastatin		Simvastatin		F-value	p-value	Pair wise comparison by Tukeys posthoc		
	Mean	SD	Mean	SD	Mean	SD			Control vs Rosuvastatin (p=)	Control vs Simvastatin (p=)	Rosuvastatin vs Simvastatin (p=)
Pretest	37.84	10.31	49.75	5.10	54.18	1.86	23.6653	0.0001*	0.0001*	0.0001*	0.1810
Posttest	35.18	9.53	48.54	5.05	52.14	1.81	30.0190	0.0001*	0.0001*	0.0001*	0.2740
Difference	2.65	3.10	1.21	0.42	2.04	0.82	2.2571	0.1172	0.0990	0.6470	0.4470

*p<0.05

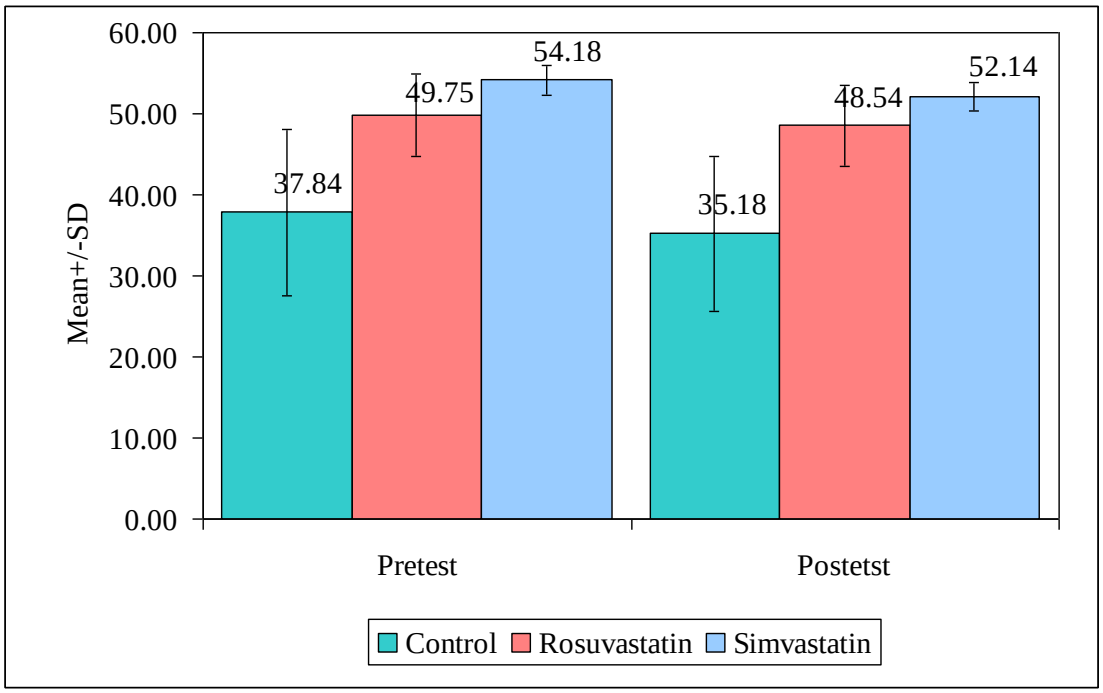
Comparison between pretest and posttest bone density values at the apical site in the three groups — Control, Rosuvastatin, and Simvastatin — was done by One Way ANOVA, pair wise comparison by Tukey's post hoc test.

The lowest mean bone density at the pretest time point was in the Control group (37.84 ± 10.31), followed by the Rosuvastatin group (49.75 ± 5.10) and highest in the Simvastatin group (54.18 ± 1.86). There was a statistically significant difference in the mean bone density between the three groups (F=23.6653, p=0.0001). A statistically significant variance between the Control and Simvastatin (p=0.0001) and Control and Rosuvastatin (p=0.0001) groups was found by pairwise comparison, indicating that both statin groups had higher bone density than the Control. However, there was no statistically significant variance between the Simvastatin and Rosuvastatin groups (p=0.1810), and baseline bone density measures were comparable between the two groups.

In the same way, the Control group, posttest interval, had the lowest mean bone density (35.18 ± 9.53), whereas in the Rosuvastatin group (48.54 ± 5.05) and above all in the Simvastatin group (52.14 ± 1.81) greater values were noted. The statistical highly significant difference between the bone density groups remained (F=30.0190, p=0.0001). Pairwise comparison demonstrated that the Control and Rosuvastatin (p=0.0001) as well as the Control and Simvastatin groups (p=0.0001) had significant difference while not having any difference (p=0.2740) between the Rosuvastatin and Simvastatin groups can imply both statins acting with an equally beneficial effect towards increased bone density.

When comparing the decrease in bone density (posttest-pretest) between the groups, the greatest decrease was in the Control group (2.65 ± 3.10), then the Simvastatin group (2.04 ± 0.82), and the least in the Rosuvastatin group (1.21 ± 0.42). Nevertheless, there was no statistically significant difference in the three groups' reductions in bone density (F=2.2571, p=0.1172). Control vs. Simvastatin (p=0.6470), Rosuvastatin vs. Simvastatin (p=0.4470), and Control vs. Rosuvastatin (p=0.0990) did not vary statistically significantly, according to additional pairwise comparisons.

Graph 7: Comparison of three groups with pretest and posttest Bone density-apical



The graph indicates pretest bone density measurements at the apical site for the three treatment groups. At baseline, the mean bone density of the Control group was 37.84% (SD = 10.31), which was substantially lower than the rosuvastatin group's 49.75% (SD = 5.10) and simvastatin group's 54.18% (SD = 1.86). Tukey's post-hoc test demonstrated that the Control vs. rosuvastatin (p = 0.0001) and Control vs. simvastatin (p = 0.0001) comparisons were all significantly different from one another, while the rosuvastatin and simvastatin groups did not vary significantly different from one another (p = 0.1810). One-way ANOVA revealed a highly significant distinction between groups (F = 23.6653, p = 0.0001).

Posttest bone density values at the apical level are also depicted in the graph. The mean density of the Control group dropped to 35.18% (SD = 9.53) post intervention, whereas the rosuvastatin and simvastatin groups observed higher

densities, at 48.54% (SD = 5.05) and 52.14% (SD = 1.81) respectively. The overall difference was significantly different (F = 30.0190, p = 0.0001), and pairwise tests revealed further that both the rosuvastatin (p = 0.0001) and simvastatin (p = 0.0001) groups were significantly different from the Control group, but not between the two statin groups (p = 0.2740).

The graph also shows the bone density difference between pretest and posttest. For the Control group, the mean loss was 2.65% (SD = 3.10) but for the rosuvastatin and simvastatin groups, it was lower at 1.21% (SD = 0.42) and 2.04% (SD = 0.82), respectively. Although these absolute changes indicate comparably less loss of bone density in the rosuvastatin group, overall intergroup difference in change of bone density was not significant (F = 2.2571, p = 0.1172), and there were no significant pairwise differences (Control vs. rosuvastatin, p = 0.0990; Control vs. simvastatin, p = 0.6470; rosuvastatin vs. simvastatin, p = 0.4470).

Table 8: Comparison of three groups with pain scores at different treatment time points by Kruskal Wallis ANOVA

Time point	Control			Rosuvastatin			Simvastatin			H-value	p-value
	Mean	SD.	Mean rank	Mean	SD.	Mean rank	Mean	SD.	Mean-rank		
2- hours	3.53	0.83	18.17	4.13	0.64	27.67	3.87	0.74	23.17	4.5630	0.1020
4- hours	2.60	0.74	18.67	2.93	0.59	25.17	2.93	0.59	25.17	3.0750	0.2150
8- hours	1.80	0.41	18.1	2.13	0.35	24.73	2.20	0.56	26.17	6.1160	0.0470
12- hours	1.67	0.49	22.67	1.67	0.49	22.67	1.73	0.59	23.67	0.0830	0.9590
24- hours	1.33	0.49	23.5	1.33	0.49	23.5	1.27	0.46	22	0.2030	0.9040
48- hours	1.00	0.53	22.13	1.07	0.26	23.4	1.07	0.46	23.47	0.2220	0.8950
72- hours	0.87	0.52	25.2	0.73	0.46	22.63	0.67	0.49	21.17	1.1660	0.5580

In the present study, comparison of post-operative pain scores was done among the three groups — Control, Rosuvastatin, and Simvastatin — at various treatment time intervals using the Kruskal-Wallis ANOVA test.

At the 2-hour interval, the mean pain score was found to be highest in the Rosuvastatin group (4.13 ± 0.64), followed by the Simvastatin group (3.87 ± 0.74), and the least in the Control group (3.53 ± 0.83). However, the difference was statistically insignificant (p=0.1020).

At 4-hour interval, Rosuvastatin and Simvastatin groups recorded an identical mean pain score (2.93 ± 0.59), whereas Control group had slightly lower mean pain

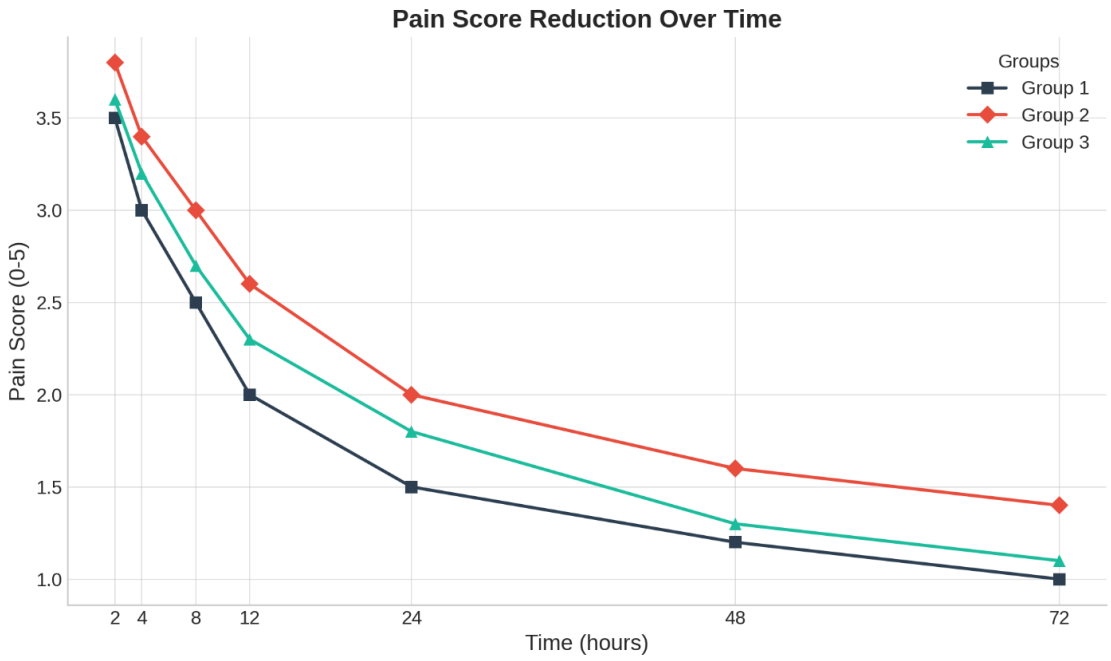
score- 2.60 ± 0.74. The intergroup difference remained statistically insignificant (p=0.2150).

At 8-hour interval, the highest pain score was observed in the Simvastatin group (2.20 ± 0.56), followed by the Rosuvastatin group (2.13 ± 0.35), and the least in the Control group (1.80 ± 0.41). Difference was statistically significant (p=0.0470), indicating that Control group experienced significantly less pain at this time point compared to the statin groups.

At the 12-hour interval, the pain scores among the three groups were almost similar, with mean values of 1.67 ± 0.49 in the Control and Rosuvastatin groups, and 1.73 ± 0.59 in the Simvastatin group, showing statistically insignificant difference (p=0.9590).

Similarly, at 24-hour, 48-hour, and 72-hour intervals, the pain scores continued to decrease in all three groups. Statistically insignificant differences were observed among the groups at these time-points (p> 0.05). At the 72-hour interval, the least mean pain score was recorded in the Simvastatin group (0.67 ± 0.49), followed by Rosuvastatin group (0.73 ± 0.46), and the Control group (0.87 ± 0.52).

Graph 8: Comparison of three groups with pain scores at different treatment time points



The Graph presents the pain scores measured at different time intervals after treatment across the three groups: Control, Rosuvastatin, and Simvastatin.

At the 2-hour time point, the Control group recorded mean pain score- 3.53 (SD= 0.83) with a mean rank of 18.17. In comparison, the Rosuvastatin group reported higher average score of 4.13 (SD= 0.64) with a mean rank of 27.67, while the Simvastatin group had a mean- 3.87 (SD = 0.74) with a mean rank of 23.17. The Kruskal-Wallis test yielded an H-value of 4.5630 with a 'p'value of 0.1020, indicating that the differences in pain scores at 2 hours were statistically insignificant.

At 4 hours post-treatment, the Control group's mean pain score decreased to 2.60 (SD = 0.74) with a mean rank of 18.67. Both the Rosuvastatin and Simvastatin groups exhibited identical mean pain scores of 2.93 (SD = 0.59 each) with mean ranks

of 25.17. With an H-value of 3.0750 and a ‘p’value of 0.2150, significant indifferences were observed among the groups at 4 hours.

At 8-hour time-point, a significant difference emerged. The Control group’s pain score further lowered to a mean of 1.80 (SD = 0.41) with a mean rank of 18.1, whereas the Rosuvastatin group registered a mean of 2.13 (SD = 0.35) with mean rank- 24.73, and the Simvastatin group had mean- 2.20 (SD = 0.56) with a mean rank- 26.17. The overall difference at this time was statistically significant (H = 6.1160, p = 0.0470), suggesting that the Control group experienced significantly less pain compared to the statin groups.

By the 12-hour mark, the pain scores among the groups converged, with both the Control and Rosuvastatin groups showing a mean of 1.67 (SD = 0.49) and the Simvastatin group showing 1.73 (SD = 0.59). The p-value of 0.9590 indicated no significant differences at this interval.

At later time points, the pain scores continued to diminish. At 24 hours, the Control and Rosuvastatin groups both recorded a mean pain score of 1.33 (SD = 0.49), while the Simvastatin group scored slightly lower at 1.27 (SD = 0.46); the overall difference was not significant (p = 0.2030). Likewise, for 48 hours, pain scores were 1.00 (SD = 0.53) for the Control group and 1.07 (SD was reported equally for both statin groups), with no statistical significance (p = 0.2220). Lyastly, in 72 hours, the Control group had a mean of 0.87 (SD = 0.52, mean rank = 25.2), the Rosuvastatin group had 0.73 (SD = 0.46, mean rank = 22.63), while the Simvastatin group had the lowest mean of 0.67 (SD = 0.49, mean rank = 21.17) with the differences remaining not significant (H = 1.1660, p = 0.5580).

DISCUSSION

Tooth extraction results in change of alveolar bone dimensions, which includes loss of bone width (29-63%) and loss of bone height (11-22%) in first six month of tooth removal.[39]

Various strategies have been utilized for the induction of the bone growth to decrease the prosthetic complication which include autografts, allografts, xenografts etc.[2] Each one of these has its own disadvantages which has led to introduction of Pharmacological substitutes like Statins. Statins are the lipid-lowering agents and act by the mechanism of HMG CoA reductase inhibition. Additionally, statins exert osteogenic effects through acceleration of BMP-2 expression, stimulation of vascular endothelial growth factor , enhancement of osteoblastic differentiation and inhibition of osteoclastic activity.[2,10] Hence, statins have emerged as a novel class of agents with significant bone regenerative capabilities.

The effect of statins when given systematically is minimized due to clearance from the liver, but excessively high doses can cause liver and kidney diseases, and this has led to simvastatin to be used locally with no side effects.[40]

Local application of Statins has been advocated for preservation of bone post-extraction.

Statins like Simvastatin, Rosuvastatin, atorvastatin have gained popularity for bone regeneration post tooth extraction, in periodontal defects etc.

In the current research the efficacy of Simvastatin and Rosuvastatin in tooth socket extracted bone regeneration was compared. Alveolar bone height was measured on CBCT. Control group had maximum bone height loss (0.59 ± 0.33 mm),

followed by Rosuvastatin (0.32 ± 0.12 mm) and Simvastatin (0.24 ± 0.22 mm). However no significant difference in efficacy between Rosuvastatin and Simvastatin was found statitically.

Chauhan et al (2015) piloted a study to evaluate the efficiency of simvastatin for bone regeneration in third molar socket post-extraction in 30 patients. Accelerated bone formation was noted in extracted tooth sockets treated with simvastatin and gelfoam in comparison to control site treated only with gelfoam.[18]

Bone width assessment at the coronal level showed the greatest reduction in the control group (0.37 ± 0.25 mm), indicating greater bone loss in comparison to the Rosuvastatin group, which showed a mean gain of 0.10 ± 0.85 mm, and the Simvastatin group, which demonstrated a negligible loss of 0.17 ± 0.10 mm. This research indicates that Rosuvastatin is meaningful in its impact for the preservation of the width of alveolar bone on the coronal level.

Bone width assessment at the middle third level, the Simvastatin exhibited better efficacy in the preservation of the bone width at the middle third of the socket (0.09 ± 0.12 mm).

At the apical third region, the Rosuvastatin (0.21 ± 0.13 mm) exhibited better efficacy in maintaining the bone width, followed by the simvastatin. The Control group (0.32 ± 0.23 mm) exhibited the highest mean reduction which is followed by the Simvastatin group (0.24 ± 0.14 mm).

Therefore, the study draws the inference that the bone width at the respective levels of socket can be preserved with the use of Rosuvastatin and Simvastatin.

In a clinical trial by **Cruz et al**, the effect of 1.2% simvastatin gel with polypropylene membranes (PPPM) was tested for socket preservation. According to the findings of the study, those groups receiving simvastatin gel and PPPM were better for horizontal bone preservation, i.e., bone width at 3 socket depth levels from the crest. And vertical bone loss was arrested in these patients.[31]

Abu Sheehah HA carried out research in tooth socket extracts to determine the effectiveness of simvastatin in bone preservation. In radiographic assessments done using CBCT, after four months, it was noticed that the group that received simvastatin performed better in the preservation of the bone width. But no significant effect could be observed for the alveolar bone height preservation.[32]

The bone density was also measured at coronal, middle, and apical third levels of the extraction socket, respectively. The bone density at the coronal level was as follows in control group (mean reduction of 3.33 ± 6.38 %), followed by the Simvastatin group (mean reduction of 1.73 ± 0.88 %), and the Rosuvastatin group (mean reduction of 1.33 ± 0.54 %), respectively, can be elicited. However, no significant difference can be noted at this level amongst the three. At the middle third level, the Control group shows (3.81 ± 9.54 %) reduction, whereas the Rosuvastatin group shows a smaller reduction (1.41 ± 0.52 %), and the Simvastatin group exhibits the least reduction (1.14 ± 0.46 %). In the apical region, maximum reduction was noted in the Control group (2.65 ± 3.10), followed by the Simvastatin group (2.04 ± 0.82), while the least reduction was seen in the Rosuvastatin group (1.21 ± 0.42). But statistically, the results are not significant, still Rosuvastatin and Simvastatin have shown better efficiency than the control.

Ayman Gouda et al organized a study in patients who had to undergo a sinus lift procedure. It was noted that there was significant bone formation in patients with sinus lift procedures in the group that received beta tricalcium phosphate along with simvastatin in comparison to the group which received only beta tricalcium phosphate on histomorphic analysis done at 9 months.[24]

Increase in bone contact ratio and 'bone density', with very little post-operative swelling was noted in patients treated with Simvastatin around implants. [41]

In a study conducted by **Özer T** and **Aktaş A**, on 16 decalvarized rabbits 2 titanium occlusive splints were used respectively for each rabbit. 1 mg Rosuvastatin was used with the xenograft in study group and after 12 weeks evaluation was done. The histological analysis done at 12 weeks revealed that Rosuvastatin with xenograft has performed superior than the xenograft group alone. Radiological findings also favoured the Rosuvastatin as it has shown to new bone volume and bone dimensions.[42]

In a research conducted with Rosuvastatin gel and atorvastatin gel in concentration of 1.2% in intra-bony defects of chronic periodontitis patients, it was observed that in the Rosuvastatin and atorvastatin group (30 patients each) had better clinical attachment gain and decrease in the intra-bony defect depth against the placebo group of 30 patients. In this study, it was also noted that Rosuvastatin performed better than the atorvastatin gel at the 6^{th} and 9^{th} months respectively.[4]

Rosuvastatin has also proved its efficacy in the treatment of mandibular fractures in the study done by **Türer Akif** and **Durmuşlar Mustafa Cenk**. In this study, the fracture was induced by vertical osteotomy and the healing was noted on the 14^{th} and 28^{th} day. The stereological analysis proved that at day 14, Rosuvastatin

administered group of rats had better formation of bone and connective tissue than the control group of rats.[20]

In our research it was observed that sockets which were loaded with Simvastatin and Rosuvastatin showed progressive increase in bone density compared to control side but the rate gradually improved until the end of 3rd month.

The performance of Rosuvastatin in increasing the bone level or bone fill in the socket could be explained based on its pharmacological properties. Rosuvastatin is a hydrophilic statin with higher systemic bioavailability and lower hepatic metabolism compared to the lipophilic Simvastatin.[43] Due to this, Rosuvastatin tends to remain longer at the site of application. This tendency also leads to sustained drug release and a prolonged osteogenic effect. Rosuvastatin also tends to possess better antioxidant properties, which help in reducing oxidative stress at the surgical site which in turn promotes favourable healing conditions.

Various carriers for the local delivery of simvastatin (**Iker Ozec et al, Chauhan et al & Aamir Malik et al.**) like gelatin sponge to carry Simvastatin have been found it to be effective and convenient from a surgical viewpoint.[3,18,29] **Myat Nyan et al** used calcium sulphate as the carrier, in whose study, rat calvaria were treated with calcium sulphate or a combination of Simvastatin and calcium sulphate, and concluded that the combination of said drugs stimulated bone regeneration despite of inflammatory response.[44]

The Visual Analogue Scale was utilized in order to record the pain score at 2, 4, 8, 12, 24, 48, and 72 hours, respectively. No variation was induced in the 3 groups at all the hours of recording. Statins have also been found to reduce the activity of the proinflammatory cytokines like tumour necrosis factor alpha and interleukin-6, which

further result in the regeneration of the tissue. The effect of statins in pain reduction should be explored further and backed up with evidence using a more extensive population.

Numerous research studies have determined the role of simvastatin on postoperative pain control in the context of dental procedures. **Cruz et al. (2021)** assessed the application of simvastatin gel with polypropylene membranes to extraction sockets within a triple-blind randomized controlled study. No comparison between the simvastatin and control groups regarding pain was found ($p = 0.23$), which means the application of simvastatin did not add to postoperative pain.[31]

Similarly, a randomized clinical trial conducted by **Diniz et al**. (2022) assessed the effect of topical simvastatin on post-mandibular third molar extraction-induced pain, swelling, and alveolar bone microarchitecture. The outcome concluded that the pain in the postoperative period was not significantly changed in the presence of simvastatin than that of the control group.[37]

This suggests that while simvastatin is effective in promoting bone regeneration, it does not adversely affect postoperative pain management.

These findings collectively indicate that the local application of simvastatin in dental procedures is safe and does not increase postoperative pain, making it a viable option for enhancing bone regeneration without compromising patient comfort.

From a clinical perspective, the use of Rosuvastatin as a topical drug delivery agent for enhancing bone regeneration in extraction sockets appears promising. Its superior performance in promoting bone density and bone fill, along with excellent patient compliance and safety profile, makes it a potential therapeutic agent in

regenerative dentistry. Furthermore, combining statins with other osteogenic biomaterials such as bone grafts or platelet concentrates could further enhance their regenerative efficacy.

In summary, the current research proved that Simvastatin and Rosuvastatin are both effectively able to induce bone regeneration in extraction sockets when used locally. However, Rosuvastatin exhibited superior outcomes in terms of bone density enhancement, but the results were comparable for bone fill and pain perception compared to Simvastatin. The results of this study uphold the use of Simvastatin and Rosuvastatin as a potent osteopromotive mediator in clinical scenarios requiring enhanced bone regeneration.

LIMITATION

Despite its strengths, this study has some limitations:

- The sample size is comparatively small, which may lead to a limitation for the generalization of the findings.
- The comparatively short follow-up duration of up to 12 weeks, since longer follow-up durations could provide data on the consolidation and stability of the newly developed bone.
- Histologic examination should be performed to evaluate quality of new bone formation.

CONCLUSION

To conclude, topical application of Simvastatin and Rosuvastatin initiates 'bone regeneration' in extraction sockets. The use is not technique sensitive. It provides better way for faster regeneration of bone after tooth extraction. Limited sample size could be considered a limitation. It is recommended to have a larger samples and recall time to further emphasize these claims. More studies need to determine difference in bone growth, mode of application of 'simvastatin' and 'rosuvastatin' its effectiveness for bone regeneration.

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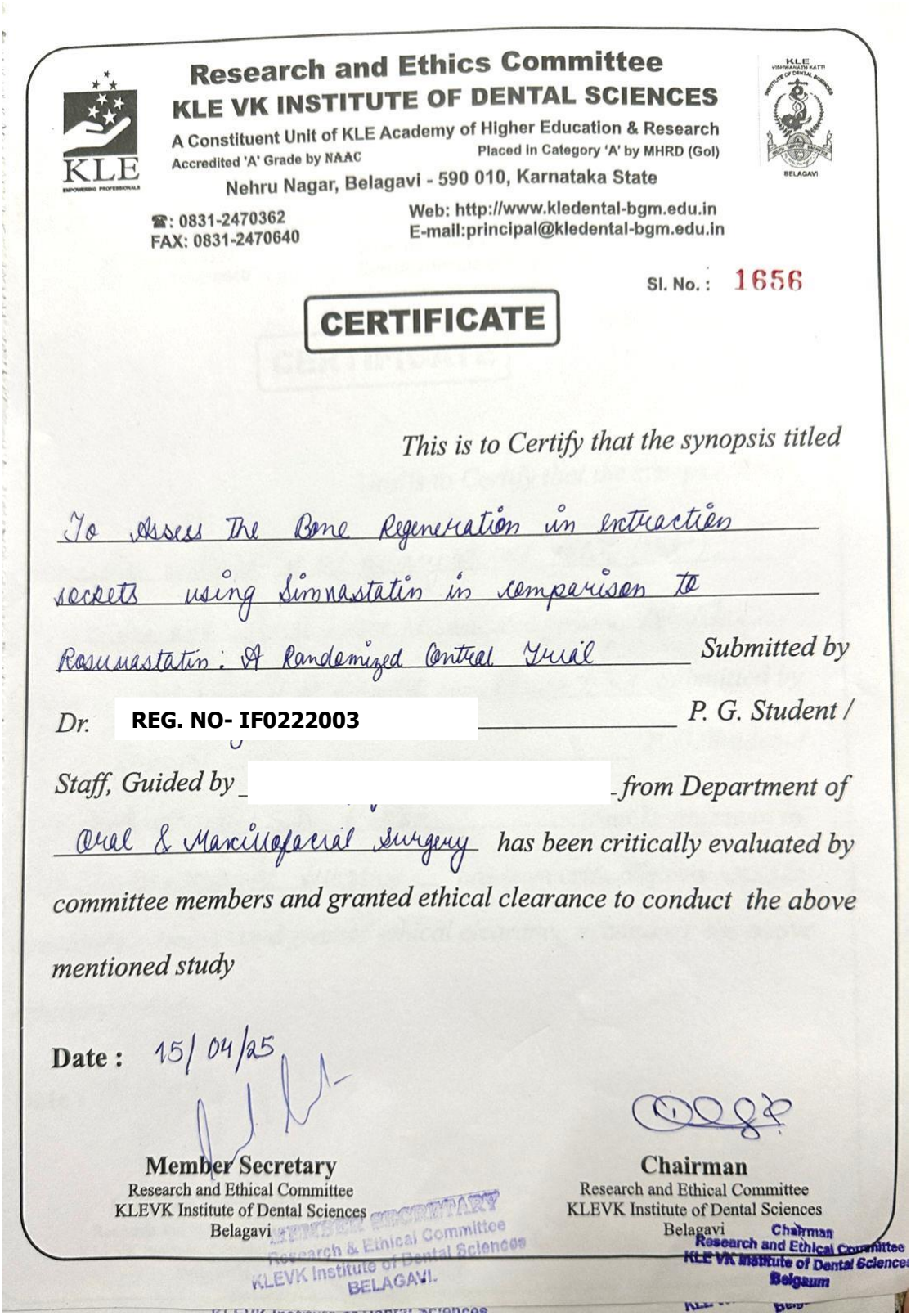
ANNEXURES

ANNEXURE-I-ETHICAL CLEARANCE CERTIFICATE



Research and Ethics Committee
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Sl. No. : 1656

CERTIFICATE

This is to Certify that the synopsis titled

To Assess The Bone Regeneration in extraction sockets using Simvastatin in comparison to Rosuvastatin: A Randomized Control Trial *Submitted by*

Dr. **REG. NO- IF0222003** *P. G. Student /*

Staff, Guided by _____ *from Department of*

Oral & Maxillofacial Surgery *has been critically evaluated by committee members and granted ethical clearance to conduct the above mentioned study*

Date : 15/04/25

Member Secretary
Research and Ethical Committee
KLEVK Institute of Dental Sciences
Belagavi

Chairman
Research and Ethical Committee
KLEVK Institute of Dental Sciences
Belagavi

MEMBER SECRETARY Research & Ethical Committee KLEVK Institute of Dental Sciences BELAGAVI.

Chairman Research and Ethical Committee KLE VK Institute of Dental Sciences Belgaum

ANNEXURE II-CONSENT FORM

K.L.E.'s V.K. Institute of Dental Sciences

Department of Oral and Maxillofacial Surgery,

Belagavi

PATIENT CONSENT FORM

Date:

Time: a.m./p.m.

1. I, aged years have been informed about my involvement in the study.
2. I agree to give my personal details like Name, Age, Sex, Address, and any other details required for the study to the best of my knowledge.
3. I will cooperate with the surgeon for examination and also for the investigation.
4. I permit the operator to utilise the information given by me and the results obtained from this study for presentation and publication purposes.
5. I permit the surgeon to take my photographs to utilize them for presentation and publication purpose.
6. I am participating in this study with my own wish and will and the surgeon has explained the nature and the effect of procedure which includes conducting the ultrasonography and its advantage in arriving at a diagnosis in my vernacular language.
7. The nature and purpose of the procedure and the materials being used, possible alternative methods of treatment, the risk involved and the possibility of complications have been fully explained to me in my vernacular language. No guarantee or assurance has been given by anyone as to the results that may be obtained.
8. I have been informed about the follow up after 6 months and I agree to visit for the same.
9. I have read and understood the above information given by surgeon about the study and willingly agree to participate in the study.

Name: Date:

Signature: Mob. No:

KLE च्या VK इन्स्टिट्यूट ऑफ डेंटल सायन्सेस विभाग

तोंडी आणि मॅक्सिलोफेशियल शस्त्रक्रिया, बेलागावी

रुग्ण संमती फॉर्म

तारीख: वेळ: am/pm

1. मी, वय वर्षे माहिती दिली आहे बद्दल माझे मध्ये सहभाग अभ्यास.

2. मी माझे वैयक्तिक तपशील जसे की नाव, लिंग, पत्ता आणि इतर तपशील देण्यास सहमत आहे आवश्यक साठी अभ्यास सर्वोत्तम करण्यासाठी च्या माझे ज्ञान

3. आय सहकार्य करेल सह साठी सर्जन परीक्षा आणि साठी देखील द तपास.

4. आय परवानगी द ऑपरेटर करण्यासाठी वापरणे द माहिती दिले द्वारे मी आणि द परिणाम प्राप्त पासून हे अभ्यास च्या साठी सादरीकरण आणि प्रकाशनाचा उद्देश.

5. आय परवानगी द सर्जन माझे घेणे करण्यासाठी छायाचित्रे वापरणे त्यांना सादरीकरण आणि प्रकाशन हेतूसाठी.

6. मी माझ्या स्वतःच्या इच्छेने आणि इच्छेने या अभ्यासात सहभागी होत आहे आणि सर्जनने त्याचे स्वरूप स्पष्ट केले आहे आणि प्रक्रियेचा परिणाम ज्यामध्ये अल्ट्रासोनोग्राफी आयोजित करणे आणि माझ्या निदानावर पोहोचण्याचा त्याचा फायदा समाविष्ट आहे स्थानिक भाषा.

7. प्रक्रियेचे स्वरूप आणि उद्देश आणि वापरलेली सामग्री, संभाव्य पर्यायी पद्धती उपचार, त्यात समाविष्ट असलेला धोका आणि गुंतागुंत होण्याची शक्यता मला पूर्णपणे समजावून सांगितली आहे स्थानिक भाषा. परिणामांबाबत कोणतीही हमी किंवा आश्वासन कोणीही दिलेले नाही प्राप्त.

8. आय आहे पाठपुरावा करण्याबाबत माहिती दिली नंतर 6 महिने आणि आय भेट देण्यास सहमत आहे त्याच साठी.

9. मी अभ्यासाबद्दल सर्जनने दिलेली वरील माहिती वाचली आणि समजली आहे आणि स्वेच्छेने सहमत आहे करण्यासाठी अभ्यासात सहभागी व्हा.

नाव: तारीख:

स्वाक्षरी:

डॉक्टरांचे नाव: _____

संपर्क क्रमांक: _____

हॉस्पिटल संपर्क: _____

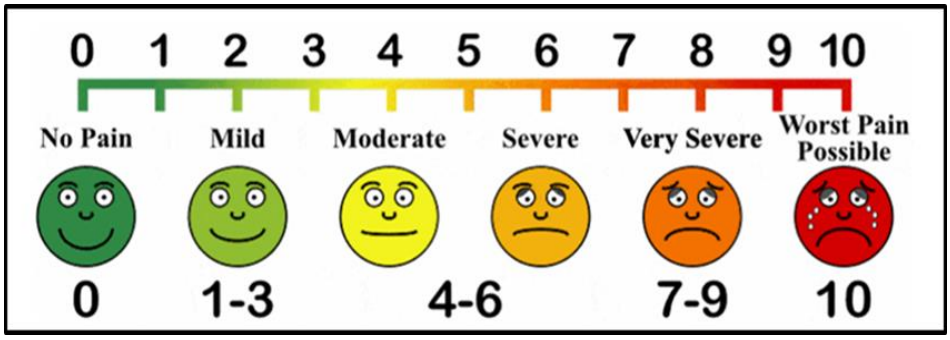
ANNEXURE III – PATIENT PROFORMA FOR PAIN ASSESSMENT

PATIENT PROFORMA

DATE:

NAME:

AGE: SEX: OPD NO:



SCORE	INTENSITY OF PAIN
0	No pain
1-3	Mild pain
4-7	Moderate pain
8-10	Severe pain

TIME	2 HOUR	4 HOUR	8 HOUR	12 HOUR	24 HOUR	48 HOUR	72 HOUR
PAIN							

PATIENT SIGNATURE: _____