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EDITOR'S MESSAGE

As I share this final issue of the IDA Thiruvalla Journal, I am grateful for the journey of learning, collaboration, and celebration of our dental community. Beyond clinical excellence, this edition reminds us of the importance of mental well-being.

As dental professionals, we face daily challenges and pressures — taking care of our minds is as vital as caring for our patients. Let us support one another, stay mindful, and continue to grow with compassion, both professionally and personally.

Thank you to all contributors and readers who made this journey meaningful. May the spirit of knowledge, empathy, and wellness continue to guide our fraternity.

Dr. Prameetha George Ittycheria
Editor IDA Thiruvalla

ASSISTANT EDITOR'S MESSAGE

Greetings to all dear IDA Thiruvalla members!

A healthy smile is more than just beautiful- it's a reflection of overall well-being. In this issue our journal "Taper" we bring about thought-provoking articles of ever-changing landscape of dental science. As professionals, we have the privilege and responsibility to stay informed, adapt and share our knowledge for the betterment of oral health in our communities. Let us continue to learn, grow and smile together as every smile we create reflects our passion for excellence.
Warm Regards,

Dr. Merlin Thomas
Assistant Journal Editor

PRESIDENT'S MESSAGE

Dear Members,

It is with great pleasure that I extend my greetings through the final edition of TAPER, the official journal of IDA Thiruvalla Branch. This publication continues to reflect the academic spirit, professional dedication and unity that define our branch.

The past year has been a period of growth and meaningful engagement. Through a series of CDE programmes, table clinics, and scientific discussions, we have strengthened our commitment to excellence in dental practice.

Our Community Dental Health activities have further extended our reach to the public, reaffirming our mission of service beyond the clinic.

A proud moment for our branch this year was the inauguration of the renovated Community Dental Clinic at Gilgal Ashwasa Bhavan, inaugurated by Dr. Nithin Joseph, State CDH Chairman, IDA Kerala State. This initiative truly embodies the compassionate spirit of IDA Thiruvalla.

Alongside our academic and service-oriented initiatives, the fellowship and friendship among our members have been equally inspiring. Family tours, social gatherings, and wellness programmes have helped strengthen our sense of togetherness and belonging.

I take this opportunity to convey my heartfelt appreciation to the Editorial Board of TAPER headed by Dr. Prameetha George Ittycheria and Dr. Merlin Thomas for their meticulous effort in bringing out this fine edition. Heartfelt gratitude to Dr. Sandra Arun for the beautiful designing and compiling.

My sincere thanks also to all members who have contributed articles, adding immense value and richness to this publication.

As we conclude another successful term, let us continue to uphold the values of professionalism, service, and unity that guide our association. I thank each member for your cooperation, enthusiasm, and unwavering support that make IDA Thiruvalla a model of collective strength and excellence.

With warm regards

Dr. Maya Mathai
President, IDA Thiruvalla Branch

EVALUATION OF TIME TAKEN BY ROTARY NITI INSTRUMENTS IN THE REMOVAL OF GUTTA-PERCHA DURING ROOT CANAL RETREATMENT IN COMPARISON WITH HAND INSTRUMENTS: A SYSTEMATIC REVIEW AND META-ANALYSIS

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ABSTRACT

Introduction: The main objective of the root canal retreatment technique is to remove the obturation material completely from the root canal system, thereby promoting adequate shaping, cleaning, and three-dimensional obturation. This systematic review aims to discuss the difference in time taken by the Protaper retreatment files and Hedstrom files to remove gutta-percha from the root canal during endodontic retreatment.
Methodology: This systematic review and meta-analysis were reported and conducted according to the PRISMA guidelines. An electronic search was performed on MedLine (from 1948 onwards), Scopus, and Google Scholar databases. Publications of in vitro and ex vivo studies in the English language up to 31st March 2021 were included in the review. A customized tool was used to assess the risk of bias. Covidence software was used to record the decisions and RevMan 5.4.1 was used to perform the meta-analysis. The effect was estimated using a standardized mean difference (SMD), with 95% confidence intervals.
Results: 26 articles were selected for the systematic review, and 16 articles were included for the meta-analysis. A sub-group analysis was conducted among Protaper retreatment (PTR) files versus Hedstrom file (H-file) variants and Protaper + Solvent versus Hedstrom file variants.The meta-analysis demonstrated that the time taken for removal of gutta-percha
(GP) was significantly lower in the PTR group when compared with H-file variants [SMD: -4.12 (95% CI: -5.32, -2.92)]. It was found that time taken for removal of gutta-percha in PTR + Solvent Vs H-file variants, showed a significant difference favoring PTR + Solvent group [SMD:-4.88 (95% CI: -6.91, -2.84)].

Conclusion: During the endodontic retreatment procedure, current evidence suggests that Protaper retreatment files reduce operative time in the removal of gutta-percha from the root canals when compared with Hedstrom files.

Keywords: Endodontic Retreatment, Gutta-percha, Protaper Retreatment Files, H-Files, Solvents

INTRODUCTION

An endodontic treatment aims to completely shape, disinfect and three dimensionally obturate the canal space in order to obtain a hermetic seal(1). However, root canal failures are observed ,despite a proper chemo-mechanical preparation, which could be due to the existence of microbes within the complexities of the root canal system(2). Retreatment in endodontics can be carried out either by non-surgical or surgical procedures. With a success rate of 74 to 98%, non-surgical retreatment is the preferred choice for managing endodontic failures(3). Thus, the purpose of nonsurgical retreatment is to effectively remove the root canal filling material, and to achieve patency so that a thorough debridement of the canal system is accomplished(4). Gutta-percha is the most oftently used filling material and different methods employed for its removal includes chemical (chloroform, xylene, eucalyptol, orange oil, tetrachloroethylene, methoxyflurane, cinnamon oil, halothane, isoflurane, anise oil, almond oil, and turpentine oil), mechanical (hand and rotary endodontic files), physical (ultrasonic, heat, and laser), and finally, combination techniques (heat and endodontic files, endodontic files and chemicals)(5).

cinnamon oil, halothane, isoflurane, anise oil, almond oil, and turpentine oil), mechanical (hand and rotary endodontic files), physical (ultrasonic, heat, and laser), and finally, combination techniques (heat and endodontic files, endodontic files and chemicals)(5). Removal of GP using Hedstrom files (H-files) with and without solvents, or in combination with Gates-Glidden (GG) drills is a commonly used retreatment technique. However, this procedure can be time-consuming, especially when the material is tightly condensed inside the root canal(6). Yet, more efficient and faster nickel-titanium (NiTi) retreatment files are available of which Protaper retreatment files(PTR) are widely used(5), which consists of three instruments (D1, D2, and D3) with variable tapers and diameters at the tip (D1 is size 30, 0.09 taper and 16 mm in length, D2 size 25, 0.08 taper,18 mm in length, and D3 size 20, 0.07 taper and 22 mm in length). Removal of GP using Hedstrom files(H- files) with and without solvents, or in combination with Gates-Glidden (GG) drills is a commonly used retreatment technique. However, this procedure can be time-consuming, especially when the material is tightly condensed inside the root canal(6).

Methods

This systematic review was registered in the PROSPERO database (PROSPERO registration number CRD 42021243110) and followed the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines.

This review included in-vitro and ex-vivo studies done on permanent teeth with a single root and a single root canal with a closed apex. Articles reporting literature reviews, case reports, and studies in which teeth with open apex and fractures, teeth with more than one root canal, presence of resorption, and internal calcifications were excluded. The intervention group comprised of teeth in which retreatment was done using Protaper Retreatment files. Teeth that had been retreated with Hedstrom files formed the control group. The outcome measured was the assessment of time taken (in minutes) from the start of the retreatment procedure (file insertion) to the complete removal of GP from the root canals.

A comprehensive search was performed using electronic databases: MedLine (from 1948 onwards), Scopus, and Google Scholar for all the articles published until the end of 31st March 2021. There were no limitations on the date and country of publication, but only studies published in English were included in this review. The full search strategy employed was as follows: ((endodontic retreatment) OR (root canal retreatment) OR (GP removal) OR (root canal filling removal) OR (gutta percha removal)) AND ((H file) OR (H-file) OR (Hedstrom file)OR (hand files)) AND ((protaper) OR (pro taper) OR (rotary) OR (ProTaper)). Search management was done using Covidence software. After duplicates were removed, two authors (AAP and PS) independently assessed the remaining articles based on title and abstract. Any disagreement regarding the inclusion of a given study was resolved by a third author (AV). The full texts were then obtained and analyzed for inclusion/exclusion criteria. After the title and abstract were screened, the full text of the articles was identified. The selected articles were included in this systematic review after full-text assessments of the potentially relevant research. For all the databases specified, a search was conducted in March 2021.

Using a Microsoft Excel sheet, the following descriptive and quantitative data was extracted for each included study: authors and year of publication, country of origin, type of study design (In vitro or Ex vivo), obturation method, type and category of sealer, type of solvent used, sample size in each group, definition of intervention and comparator, and details of the outcomes reported. The data collection process were completed by two independent reviewers (AAP and RV).

Two review authors independently assessed the risk of bias. There was no standardized tool available for assessing the risk of bias for in vitro studies. Previous studies have used customized tools. So, the present study also used a customized tool adapted from the study by AlShwaimi et al(7).The following parameters were assessed and graded for calculating the risk of bias: presence of control group, description of sample size calculation, randomization, gutta-percha removal for root canal retreatment performed by a single operator, use of

retreatment files according to manufacturer's instructions and the evaluation of quality of RCT using any method. In case of missing data, the authors were contacted. If no response was obtained, the study was excluded from the review.

Since the measurement end-points were different (in some studies, time taken for the change of instruments and irrigation performed during retreatment procedure were included, whereas in some studies these parameters have been excluded and then measured the total retreatment time. Also, the measurement of the final length of the teeth used during retreatment procedure was different) the treatment effect for each sample was summarised using standardized weighted-mean differences (SMD). The data was examined using RevMan

5.4.1 version software from the Cochrane organisation, and a meta-analysis was done. To

analyse the data's heterogeneity, Cochran's Q statistic, a chi-square test, and a threshold p- value of less than 0.10 were used (8). The I^2 statistic and forest plots were used to assess the consistency of the results (9).

Based on the variations in the file system (H-file, rotary), use of Gates-glidden [(GG) (yes/no)], and use of a solvent (yes/no) in the procedure, a subgroup analysis was done. The Protaper versus H-file variants were further grouped into four: PTR Vs H-file, PTR Vs H-file + GG, PTR Vs H-file + Solvent and PTR Vs H-file + GG + Solvent while the Protaper + solvent versus H-file variants was grouped into two: PTR + Solvent Vs H-file + Solvent and PTR + Solvent Vs H-file+GG+Solvent.

The search strategy of this systematic review identified a total of 251 studies (MedLine – 103, Scopus – 115, and Google Scholar – 33). From the retrieved 251 studies, 105 were excluded after the removal of the duplicates. A total of 146 studies were screened based on the title and abstract, from which 106 studies were excluded, resulting in 40 full-text studies that were assessed for eligibility. Among the 40 studies for full-text screening, 26 studies were included for qualitative synthesis, and 16 studies were selected for quantitative synthesis (Figure 1.).

Amongst these 26 studies, 12 studies were conducted in India(1,5,8–17), four from Turkey(18–21), two from Brazil(22,23) and one study each from Malaysia(24), Israel(25), Saudi Arabia(26), Greece(27), China(2), Iran(28), Italy(29) and Spain(30). Most of the studies were carried out in vitro(1,2,5,8–21,24–28,30) and only three were ex vivo studies(22,23,29), with sample sizes ranging from 10 to 36. Obturation was performed using lateral compaction (LC) technique in 20 studies(1,2,5,8–12,14,15,18–21,23–25,27–29),

thermoplasticized obturation (TPO) in five studies(13,16,17,22,30) and one study used single cone obturation(26). Also, the sealer used for obturation were resin-based sealer in 18 studies(1,2,5,9,11,12,15–22,25,26,28,30),

zinc oxide eugenol (ZnOE) based sealer in four studies(8,10,23,24), calcium silicate-based sealer in one study(27) and three studies used both resin-based and zinc oxide eugenol based sealers(13,14,29).

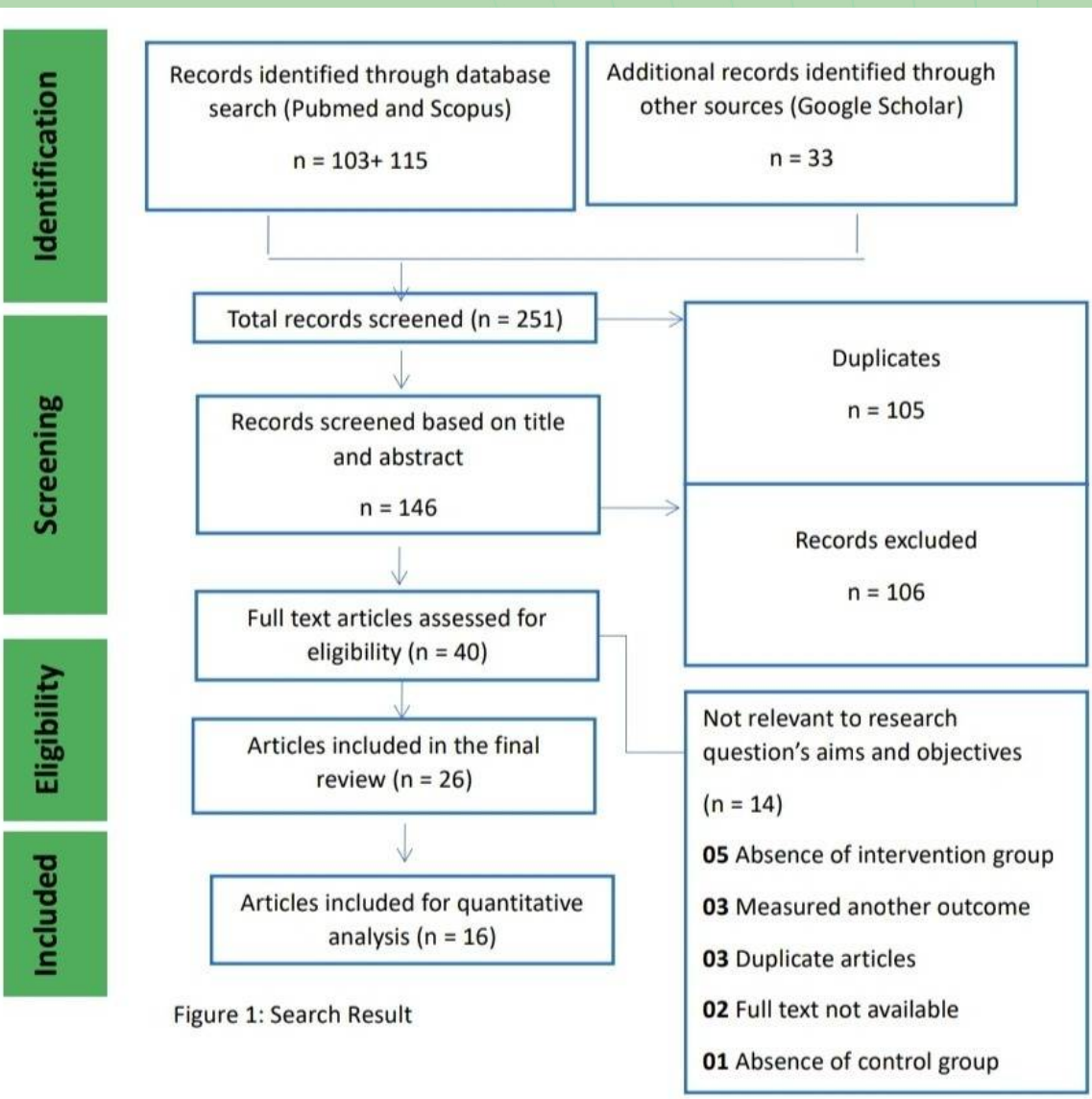

Figure 1: Search Result

Solvents used during the endodontic retreatment to remove the obturating material were chloroform in five studies(2,25,26,28,29), orange oil in five studies(5,8,12–14) and four studies used xylene(9,10,23,24), and eucalyptol oil(16,17,19,22). The intervention group was Protaper Retreatment files (D1, D2, D3) and Hedstrom files of sizes varying from 15 to 70 were used as the control group. The outcome measured was the time taken from the start of the procedure (insertion of file) to complete gutta-percha removal (t2) in 23 studies and the time taken from the start of the procedure till repreparation of the canal, that is, the total retreatment time (t3) in three studies. A study conducted by Somma compared Protaper retreatment and H- files when used with three different types of sealers.The three groups in this study have been considered as three individual studies for this review (Somma a, b, c)(29).

Similarly, the studies by Amal and Jayasenthil, have been considered as two individual studies[(Amal a, b)(13) and (Jayasenthil a, b)(14)] respectively based on the different sealers used. Also, the study by Khalilak, has been considered as two individual studies (Khalilak a, b)(28), because

here groups were divided as Protaper retreatment and H-files with and without solvent (Table 1.)

14 studies were excluded which did not meet the inclusion criteria [absence of intervention group (5 studies), measured other outcomes (3 studies), duplicate articles (3 studies), absence of control group (1 study) and the full text was not available for two studies].

A total of 26 articles were assessed for methodological quality.

Out of the total, five studies showed a low risk of bias and seven studies had a high risk of bias. Overall, the presence of the control group and randomization had a low risk of bias, whereas, high risk of bias was shown for use of retreatment files according to the manufacturer's instructions and for description of sample size calculation. Analyzing the risk of bias, it was found that in 24 out of 26 studies, randomization was done and 17 out of 26 studies, evaluated the quality of root canal treatment using any method. Sample size calculation was not mentioned in any of the studies, excluding one study (Table 2.)

Among the 26 articles included, 10 were excluded from quantitative synthesis due to the following reasons: three studies(16,19,20) measured the time taken from the start of the procedure till repreparation of the canal,two studies (22,27) did not include the mean and standard deviation values and in five studies(2,11,14,26,28), standard deviation values were not mentioned. GP removal was significantly lower in PTR group [SMD: -8.08 (95% CI: -12, - 4.16)]. The heterogeneity observed was 84%. The time taken to remove GP in PTR Vs H-file + GG group which included four studies(15,18,21,30) with 78 teeth each, showed a significant difference favoring PTR group [SMD: -3.28 (95% CI: -4.84, -1.71)]. PTR Vs H-file + Solvent was examined in three studies(5,24,25) with a total of 30 teeth. The time taken to remove GP was significantly shorter in PTR group [SMD: -5.61 (95% CI: -9.69, -1.53)]. When PTR was compared with H-file + GG + Solvent, the time taken for GP removal was favoring PTR variant in four studies(8,29) which involved 40 teeth, and one study(23) involving 20 teeth showed no significant difference in the time taken [SMD: -2.85 (95% CI: -4.76, -0.93), five studies, 60 teeth]. Overall, in comparison of PTR with H-file variants in 14 studies involving 213 teeth, it was noted that the time taken for removal of GP was significantly lower in PTR group [SMD: - 4.12 (95% CI: -5.32, -2.92)], but there was substantial heterogeneity (93%) across studies.The following sub-group analysis was conducted among PTR + Solvent Vs H-file variants (Figure 3.). Five studies(9,10,12,17,25) with a total of 67 teeth compared PTR + Solvent Vs H- file + Solvent. The PTR + Solvent group took significantly less time for the removal of gutta- percha [SMD: -4.92 (95% CI: -7.80, -2.04)]. When comparing the time taken to remove GP in the PTR + Solvent Vs H-file + GG + Solvent group, including three

studies(8,13) with 40 teeth each, PTR + Solvent group demonstrated significant difference which shows a substantial advantage over H-file + GG + Solvent [SMD: -4.89 (95% CI: -8.42, -1.35)]. So, in general, time taken for removal of GP in PTR + Solvent Vs H-file variants, which included eight studies with 107 teeth each, showed significant difference favoring PTR + Solvent group [SMD: -4.88 (95% CI: -6.91, -2.84)].

DISCUSSION

The success of endodontic retreatment depends on several factors that influence the procedure's final quality such as the type of obturation material used, the method used for removal, and the time taken to achieve satisfactory results(31). Various methods employed to remove gutta-percha include conventional hand instruments, rotary Ni-Ti instruments, gates-glidden drills, solvents, etc. Among the methods used, removal using Hedstrom files falls under the category of conventional technique. Being still one of the commonly used methods, it could be time-consuming when the material used for filling is thoroughly condensed inside the root canal. The introduction of rotary retreatment files has proven to be faster thereby simplifying the clinical steps(32). The present review was able to cumulate the difference in time taken for gutta-percha removal seen by Protaper retreatment files and Hedstrom files during retreatment procedure. The meta-analysis performed was suggestive of favouring Protaper retreatment files with and without use of solvents over H-file variants.

Because of the methodological heterogeneity observed during the study (inclusion and exclusion of time taken for change of instruments and irrigation done during retreatment procedure; variation in size of H-files; use of gates-glidden drills and measurement of final length of the teeth used for retreatment procedure), a subgroup analysis and standard mean difference were performed.

internal core and area, convex triangular cross section, variable taper, and a continuously changing helical pitch. All these factors contribute to successful gutta-percha cutting and coronal extrusion from the canal(33). Amal et al, in their study mentioned that D1, D2, and D3 files for retreatment have a gradual taper that allows them to shape certain areas of a root canal with only one file and a variable tip diameter, allowing them to cut in a predetermined area of the canal without stressing the instrument in other sections(13). Gu et al, reported improved performance of the PTR files is due to their unique flute design.During root canal filing removal, the file cut the gutta-percha along with the outermost layer of dentin.

Moreover, the flutes' specific design and the rotatory movements of the files, cut a significant amount of gutta-percha in a spiral around the instrument, directing it towards the orifice and therefore contributing to the removal of the obturating materials. In addition, the frictional heat produced by engine-driven equipment plasticizes gutta-percha and this plasticized GP would present less resistance and aids in easy removal(2). According to Hulsmann and Bluhm, the cross-sectional shape of PTR files enabled removal of large amounts of GP, while H-files removed them only in small amounts, which explains why H-files take more time for retreatment(34). Another reason for H-files taking more retreatment time is that these files were used in push-pull filing action(35).In one of the studies by Bramante et al, comparing Protaper retreatment files and H-files in combination with GG and use of solvent, no significant difference were found in time for removing the filling material(23). This may be attributed to the fact that, in hand instrumented specimens, the use of GG drills in the coronal and middle third serves as a reservoir for the chemical solvent that acts by softening the gutta-percha(10).The oval shaped design of the GG drill also favoured cutting and reduced the resistance of the filling material by the heat generated by friction that plasticizes the gutta-percha(36) thereby facilitating easy removal by pushing out the material from root canal(23).

Considering the usage of solvents, chloroform was commonly used and well known as the most efficient in disintegrating gutta-percha(37), but due to the potential cytotoxic effects, its usage has been restricted(38). Furthermore, such solvents may result in the production of a thin film of softened gutta-percha layer on the root dentin walls, resulting in decreased cleanliness(39). It has been observed that eucalyptol oil, when employed as a softening solvent, is a safe and effective non-carcinogenic alternative for chloroform(40,41). Xylene can be used as a solvent and has a higher capability for gutta-percha disintegration(Filho et al)(42). In some studies, the solvent used was orange oil which has been reported effective and less cytotoxic than eucalyptol, xylol and chloroform(14). The combined use of solvents along with hand or rotary files complicates debridement, because these solvents dissolve, flow into, or penetrate the peri-radicular tissues(43). Moreover, the chemically softened gutta-percha can easily be driven into the complex canal morphology (isthmuses, lateral canals and irregularities) where the instruments have not touched. This increases the difficulty in removal of the filling and necessitates additional time(25). However, other studies stated that rotary files without solvent speeded up the retreatment time(2). Methodological quality assessment of the article revealed that almost all of the included studies (except for one) did not specify the sample size calculation and several studies did not report to follow the use of retreatment files according to manufacturer's recommendations. While calculating the minimum required sample size would be essential to ensure the validity of the results, non-reporting of use of files according to the manufacturer's instructions could be an inadvertent omission.

Furthermore, other factors that could influence the final outcome include the length of the teeth and the ability of the individual clinician to remove gutta-percha, where absolute standardization was not attainable. Also, there were variations in the measurement of the time taken. Some studies measured time from the initial start of the procedure (file insertion) to complete removal of GP, whereas others measured time from the start of the procedure, GP removal and till repreparation of the canal. In this review for quantitative synthesis, we have taken only the time from the start of procedure to complete removal of GP. Evaluation of root canal treatment by using radiographs has shown baseline characters equally standardized.

This is probably the first systematic review to compare the time taken for GP removal using PTR files and H-files during endodontic retreatment. The present review favours Protaper retreatment files over H-files in gutta-percha removal. This information can provide a better idea to the clinicians while selecting cases to be used by PTR files and H-files in combination with or without solvents, where the difference in time for retreatment plays an important role.

CONCLUSION

Within the limitations of the study, with recent technological advances in the area of endodontics, we can affirm that the system of instrumentation of root canals during retreatment with rotary files reduces the operative time than with hand files.

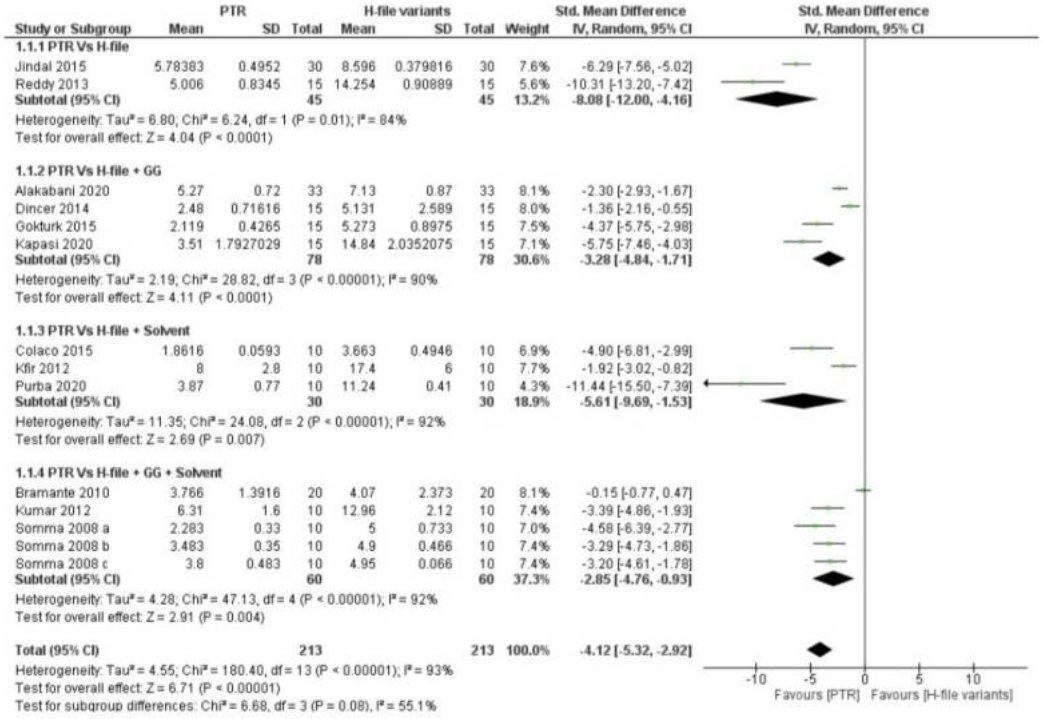

Figure 2: Overall and subgroup analysis of time taken by PTR Vs H-file variants in removal of gutta-percha

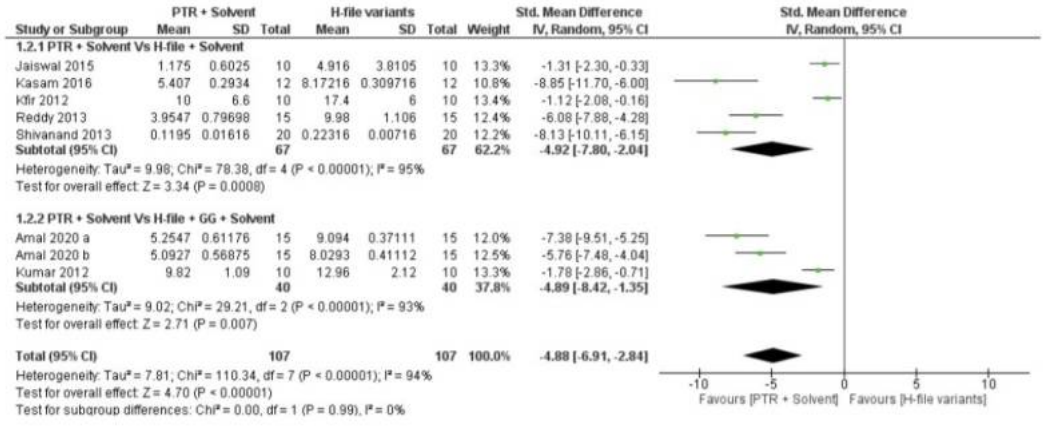

Figure 3: Overall and subgroup analysis of time taken by PTR + Solvent Vs H-file variants in removal of gutta-percha

Study ID	Country	Study Design	Sample Size (for each group)	Obturation method	Type of Sealer	Category of sealer	Type of Solvent	Intervention group	Details of Intervention group	Type of solvent	Control group.	Details of control group	Time
Colaco 2015	Malaysia	in vitro	10	LC	ZnOE	ZnOE based sealer		PTR	D1,D2, D3; 300 rpm speed; 2N/cm torque	xylene	H + solvent	H(20, 25,30)	t2
Kfir 2012	Israel	in vitro	10	LC	AH 26	Resin based sealer		PTR	D1,D2, D3; 400 rpm speed; 3 N/cm torque	chlorofo rm	H + solvent	H(60, 55,50 ,45,4 0)	t2

Purba 2020	India	in vitro	10	LC	AH Plus	Resin based sealer		PTR	D1,D2, D3; 500 rpm speed; 2N/cm torque	2 ml Wonder orange solvent	H + solvent	H(15, 20); 25,30 K file	t2
Jindal 2015	India	in vitro	30	LC	AH Plus	Resin based sealer		PTR	D1,D2, D3		H file	H(70, 60,55 ,50,40)	t2
Reddy 2013	India	in vitro	15	LC	AH Plus	Resin based sealer		PTR	D1,D2, D3; 300 rpm speed		H file	H file	t2
Alakabani 2020	Spain	in vitro	33	TPO	AH Plus	Resin based sealer		PTR	D1,D2, D3; 500 rpm speed; 4N/cm torque		H-GG	H(35, 30,25); GG 3,2	t2
Dincer 2015	Turkey	in vitro	15	LC	AH Plus	Resin based sealer		PTR	D1,D2, D3; Apical preparation F3,F4		H-GG	H(35, 30,25); GG 1,2,3; Apical 40 H file	t2
Goktur k 2015	Turkey	in vitro	15	LC	AH Plus	Resin based sealer		PTR	D1,D2, D3; 500rpm speed		H-GG	H(25, 20); GG 3,2	t2
Kapasi 2020	India	in vitro	15	LC	AH Plus	Resin based sealer		PTR	D1,D2, D3; 500 rpm speed; 3 N/cm torque		H-GG	H(40, 35,30); GG 3,2	t2
Bramante 2010	Brazil	ex vivo	20	LC	ZnOE	ZnOE based sealer		PTR	D1,D2, D3; 500 rpm speed; 2N/cm torque	Xylol	H-GG + solvent	GG 1,2,3; 30 H file; 30 K file	t2
Kumar 2012	India	in vitro	10	LC	ZnOE	ZnOE based sealer		PTR	D1,D2, D3; 500,	0.5 ml RC Solve	H-GG +	H(35-20);	t2

									400 rpm speed; 3N/cm torque		solvent	GG 3,2	
Somma 2008 a	Italy	ex vivo	10	LC	Realseal sealer	Resin based sealer		PTR	D1,D2, D3; 600 rpm speed	chlorofo rm	H-GG + solvent	H(20, 25,30 ,35,40); GG 2,3	t2
Somma 2008 b	Italy	ex vivo	10	LC	endorez sealer	Resin based sealer		PTR	D1,D2, D3; 600 rpm speed	chlorofo rm	H-GG + solvent	H(20, 25,30 ,35,40); GG 2,3	t2
Somma 2008 c	Italy	ex vivo	10	LC	Kerr pulp canal sealer	ZnOE based sealer		PTR	D1,D2, D3; 600 rpm speed	chlorofo rm	H-GG + solvent	H(20, 25,30 ,35,40); GG 2,3	t2
Jaiswal 2015	India	in vitro	10	LC	AH Plus	Resin based sealer	RC Solve	PTR + solvent	D1,D2, D3	RC Solve	H + solvent	H(20, 25,30)	t2
Kasam 2016	India	in vitro	12	LC	ZnOE	ZnOE based sealer	xylene	PTR + solvent	D1,D2, D3; 300 rpm speed	xylene	H + solvent	H(15 - 40)	t2
Kfir 2012	Israel	in vitro	10	LC	AH 26	Resin based sealer	chloroform	PTR + solvent	D1,D2, D3; 400 rpm speed; 3 N/cm torque	chlorofo rm	H + solvent	H(60, 55,50 ,45,40)	t2
Reddy 2013	India	in vitro	15	LC	AH Plus	Resin based sealer	xylene	PTR + solvent	D1,D2, D3; 300 rpm speed	xylene	H + solvent	H file	t2
Shivanand 2013	India	in vitro	20	TPO	AH Plus	Resin based sealer	eucalyptol oil	PTR + solvent	D1,D2, D3; 500 rpm speed	eucalypt ol oil	H + solvent	H(60-30)	t2

Amal 2020 a	India	in vitro	15	TPO	AH Plus	Resin based sealer	0.1 ml RC Solve	PTR + solvent	D1,D2, D3	0.1 ml RC Solve	H-GG + solvent	H(35, 30,25); GG 3,2	t2
Amal 2020 b	India	in vitro	15	TPO	ZnOE	ZnOE based sealer	0.1 ml RC Solve	PTR + solvent	D1,D2, D3	0.1 ml RC Solve	H-GG + solvent	H(35, 30,25); GG 3,2	t2
Kumar 2012	India	in vitro	10	LC	ZnOE	ZnOE based sealer	0.5 ml RC Solve	PTR + solvent	D1,D2, D3; 500, 400 rpm speed; 3N/cm torque	0.5 ml RC Solve	H-GG + solvent	H(35-20); GG 3,2	t2
Akbulut 2016	Turkey	in vitro	15	LC	AH Plus	Resin based sealer		PTR	D1,D2, D3; Apical preparation F4,F5; 300 rpm speed		H file	H(40, 35,30) Apical prep n K file 50	t3
Alkharboush 2020	Saudi Arabia	in vitro	12	single cone obturation	AH26 silver free	Resin based sealer	chloroform	PTR + solvent	PTR files; chloroform	chlorofo rm	H-GG + solvent	H files, GG drills	t2
Fariniuk 2017	Brazil	ex vivo	12	TPO	AH Plus	Resin based sealer		PTR	D1,D2, D3; Apical preparation F2,F3,F4,F5; 350 rpm speed	0.1 ml eucalyptol oil	H-GG + solvent	H file; GG 3,2,1; Apical 50 H file	t2
Gkampesi 2016	Greece	in vitro	15	LC	MTA Fillapex	Calcium silicate based sealer		PTR	D1,D2, D3; final canal preparation		H-GG	H file(30-15); GG 3; final	t2

									F3,F4; 500 rpm speed; 2N/cm torque			canal preparation 20,25,30,35,40	
Gu 2008	China	in vitro	20	LC	AH Plus	Resin based sealer		PTR	D1,D2, D3;500 rpm speed; Canal preparation S1,S2,FI,F2,F3; 300 rpm speed	chloroform	H-GG + solvent	H file (30,25,20); GG 3,2,1; Canal preparation K flex file(35,40, 45,50)	t2
Helvacioglu 2014	Turkey	in vitro	10	LC	AH Plus	Resin based sealer	0.5 ml eucalyptol oil	PTR + solvent	D1,D2, D3; Apical preparation F2	0.5 ml eucalyptol oil	H + solvent	H(15, 20,25); Apical preparation 40 H file	t3
Jayasenthil 2012 a	India	in vitro	10	LC	ZnOE	ZnOE based sealer	0.1 ml RC Solve	PTR + solvent	D1,D2, D3; 300 rpm speed	0.1 ml RC Solve	H-GG + solvent	H file(30,25, 20,15); GG 3,2	t2
Jayasenthil 2012 b	India	in vitro	10	LC	AH Plus	Resin based sealer	0.1 ml RC Solve	PTR + solvent	D1,D2, D3; 300 rpm speed	0.1 ml RC Solve	H-GG + solvent	H file(30,25, 20,15); GG 3,2	t2
Joseph 2016	India	in vitro	15	TPO	AH Plus	Resin based sealer	eucalyptol oil	PTR + solvent	D1,D2, D3; Apical preparation	eucalyptol oil	H-GG + solvent	H(35, 30,25); GG 2,3; Apica	t3

									F2,F3F4 ; 600 rpm speed			I prep arati on 40 H file	
Khalila k 2013 a	Iran	in vitro	15	LC	AH 26	Resin based sealer		PTR	D1,D2, D3		H-GG	H file(1 5-40); GG 3,2	t2
Khalila k 2013 b	Iran	in vitro	15	LC	AH 26	Resin based sealer	chl oro for m	PTR + solv ent	D1,D2, D3	chlorofo rm	H-GG + solven t	H file(1 5-40); GG 3,2	t2
Patil 2018	India	in vitro	20	LC	AH Plus	Resin based sealer		PTR	PTR files		H file + solven t	H file with solve nt	t2

Table 1: Characteristics of included studies

Study ID	Presence of control group	Descrip tion of sample size calculat ion	Rand omiz ation	GP removal for RCT performed by a single operator	Use of retreatment files according to manufacturer 's instructions	Eval uatio n of quali ty of RCT usin g any met hod	Total risk score	Interpretation
Akbul ut 2016	Yes	No	Yes	No	No	Yes	3	Medium
Alakab ani 2020	Yes	Yes	Yes	Yes	Yes	Yes	6	Low

Alkharboush 2020	Yes	No	Yes	No	No	Yes	3	Medium
Amal 2020	Yes	No	Yes	No	No	No	2	High
Bramante 2010	Yes	No	Yes	Yes	Yes	Yes	5	Low
Colaco 2015	Yes	No	Yes	No	Yes	No	3	Medium
Dincer 2014	Yes	No	Yes	No	No	No	2	High
Fariniuk 2017	Yes	No	Yes	Yes	No	Yes	4	Medium
Gkampesi 2016	Yes	No	Yes	Yes	Yes	Yes	5	Low
Gokturk 2015	Yes	No	Yes	Yes	No	Yes	4	Medium
Gu 2008	Yes	No	Yes	Yes	Yes	Yes	5	Low
Helvacioglu 2014	Yes	No	Yes	Yes	No	Yes	4	Medium
Jaiswal 2015	Yes	No	Yes	No	No	Yes	3	Medium
Jayasenthil 2012	Yes	No	No	No	No	Yes	2	High
Jindal 2015	Yes	No	Yes	No	Yes	Yes	4	Medium
Joseph 2016	Yes	No	Yes	Yes	No	Yes	4	Medium
Kapasi 2020	Yes	No	Yes	Yes	No	Yes	4	Medium

Kasam 2016	Yes	No	Yes	No	No	No	2	High
Kfir 2012	Yes	No	Yes	Yes	Yes	Yes	5	Low
Khalilak 2013	Yes	No	Yes	No	Yes	No	3	Medium
Kumar 2012	Yes	No	Yes	No	No	Yes	3	Medium
Patil 2018	Yes	No	Yes	No	No	No	2	High
Purba 2020	Yes	No	Yes	Yes	No	Yes	4	Medium
Reddy 2013	Yes	No	No	No	No	No	1	High
Shivanand 2013	Yes	No	Yes	No	No	No	2	High
Somma 2008	Yes	No	Yes	No	Yes	No	3	Medium

Table 2: Risk of bias of included studies

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ANTIHYPERTENSIVE DRUG–INDUCED GINGIVAL ENLARGEMENT: PATHOGENESIS, CLINICAL MANIFESTATIONS, AND MANAGEMENT STRATEGIES

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ABSTRACT

Drug-induced gingival enlargement (DIGE) is a clinically significant adverse effect predominantly linked with certain antihypertensive agents, particularly calcium channel blockers (CCBs). Characterized by the abnormal proliferation of gingival connective tissue, DIGE can adversely impact oral function and aesthetics, thereby reducing patients' quality of life.1 Antihypertensive drug-induced gingival enlargement represents a clinically significant adverse effect associated predominantly with calcium channel blockers (CCBs), widely prescribed for the management of hypertension and cardiovascular disorders. This condition is characterized by an abnormal and often progressive increase in the volume of gingival connective tissue, which can result in functional impairment, aesthetic concerns, and increased susceptibility to periodontal disease. The pathogenesis is complex and multifactorial, involving altered fibroblast function, disrupted collagen metabolism, and an inflammatory response exacerbated by bacterial plaque accumulation.2,3,4 This review comprehensively examines the epidemiology, molecular and cellular mechanisms, clinical presentation, diagnostic considerations, and current management strategies of antihypertensive drug-induced gingival enlargement.

Keywords: Drug induced gingival enlargements, Antihypertensive drugs, calcium channel blocker, Pathogenesis

INTRODUCTION

Gingival enlargement, also known as gingival overgrowth or hyperplasia, is a pathological increase in the size of the gingival tissues that can result from a variety of etiological factors, including inflammatory conditions, systemic diseases, and adverse drug reactions.

Among drug-induced causes, antihypertensive medications—particularly calcium channel blockers (CCBs)—have been widely implicated as significant contributors to gingival overgrowth.

However, their use has been associated with the undesirable side effect of gingival enlargement, which poses challenges to oral health maintenance and patient quality of life.[3,4]

Antihypertensive drug-induced gingival enlargement typically manifests as a progressive, painless enlargement of the gingiva, often beginning within weeks to months after initiation of therapy. While initially asymptomatic, this gingival overgrowth can interfere with mastication, phonation, and oral hygiene practices, leading to secondary complications such as increased plaque accumulation, periodontal inflammation, and heightened risk of periodontal disease. The condition is of particular clinical importance because it not only affects oral function but also has significant psychosocial impacts due to its aesthetic consequences.[5,6]

The pathophysiology of antihypertensive drug-induced gingival enlargement is complex and multifactorial, involving the interplay of drug effects on gingival fibroblast activity, collagen metabolism, inflammatory responses, and individual genetic predispositions. Understanding these mechanisms is critical for the development of effective preventive and therapeutic strategies.[7] Moreover, comprehensive patient management requires interdisciplinary collaboration between medical and dental practitioners to balance systemic disease control with the mitigation of oral adverse effects.

PATHOGENESIS [10,11,12]

The pathogenesis of antihypertensive drug-induced gingival enlargement (DIGE) is complex and multifactorial, involving a combination of cellular, molecular, and environmental factors that contribute to the abnormal proliferation of gingival connective tissue. Although the precise mechanisms remain incompletely understood, current evidence supports an interplay between drug-specific effects on gingival fibroblasts, alterations in collagen metabolism, inflammatory processes, and genetic predisposition.

1. ROLE OF GINGIVAL FIBROBLASTS

Central to the development of gingival overgrowth is the altered function of gingival fibroblasts—the primary cells responsible for the synthesis and turnover of the extracellular matrix (ECM) in gingival connective tissue. Calcium channel blockers (CCBs), particularly nifedipine and amlodipine, interfere with calcium ion influx into fibroblasts, which is essential for cellular signaling and homeostasis. This disruption leads to an imbalance favoring fibroblast proliferation and increased synthesis of collagen and other ECM components, resulting in excessive accumulation of connective tissue.

Several in vitro studies have demonstrated that gingival fibroblasts from susceptible individuals exhibit heightened responsiveness to CCBs, with increased collagen production and reduced degradation, highlighting the variability in cellular response that may underlie individual susceptibility to DIGE.

2. ALTERED COLLAGEN METABOLISM

An imbalance between collagen synthesis and degradation is a hallmark of DIGE.

CLASSIFICATION OF DRUG INDUCED GINGIVAL ENLARGEMENT[8,9](TABLE :1)

Drug Class	Common Drugs	Primary Indication	Incidence of DIGE	Typical Onset of Gingival Enlargement	Duration/Progression	Proposed Mechanism
Anticonvulsants	Phenytoin, Valproic acid, Carbamazepine	Epilepsy, seizure disorders	High (esp. Phenytoin: up to 70%)	1–3 months after initiation	Progressive enlargement may stabilize or regress after drug withdrawal	Increased fibroblast proliferation; reduced collagenase activity
Immunosuppressants	Cyclosporine A Tacrolimus Sirolimus	Organ transplantation, autoimmune diseases	Moderate to High (Cyclosporine: up to 81%)	1–6 months	Can persist or worsen without intervention; partial regression after discontinuation	Stimulates fibroblast proliferation; alters cytokine expression; inhibits apoptosis
Calcium Channel Blockers (CCBs)	Nifedipine Amlodipine Verapamil Diltiazem	Hypertension, angina	Low to High (Nifedipine: up to 85%)	1–8 weeks (Nifedipine); 1–3 months (Amlodipine)	May progress with continued use; partial regression possible after dose adjustment or cessation	Inhibits calcium influx; impairs collagen degradation; ECM accumulation
Other Antihypertensives	Enalapril (ACE inhibitor) Propranolol (β-b locker)	Hypertension, cardiac condition	Rare	Variable; often delayed	Usually transient or mild	Poorly defined; possibly inflammatory or idiosyncratic responses
Others (miscellaneous)	Erythromycin Oral contraceptive	Infection, birth control	Very Rare	Variable	Typically transient or mild	Hormonal or inflammatory modulation (in case reports)

Normally, matrix metalloproteinases (MMPs) regulate collagen breakdown, maintaining tissue homeostasis. Antihypertensive drugs may reduce the activity of collagenase enzymes, either directly or indirectly via modulation of inflammatory mediators, leading to diminished collagen degradation and accumulation of fibrotic tissue.

Furthermore, drugs may stimulate the production of transforming growth factor-beta (TGF-β), a cytokine known to promote fibroblast proliferation and collagen deposition, further exacerbating gingival enlargement.

3. INFLAMMATORY MEDIATORS AND PLAQUE-INDUCED INFLAMMATION

Although drug effects are primary, bacterial plaque accumulation acts as a critical cofactor in the pathogenesis of DIGE. Plaque-induced inflammation leads to increased levels of pro-inflammatory cytokines such as interleukin-1 beta (IL-1β), interleukin-6 (IL-6), and prostaglandin E2 (PGE2), which synergize with drug-induced fibroblast stimulation to enhance tissue overgrowth.

Clinically, poor oral hygiene has been consistently identified as a risk factor for increased severity of gingival enlargement, underscoring the role of inflammation as a potentiating mechanism.

4. GENETIC SUSCEPTIBILITY

Individual variation in susceptibility to antihypertensive drug-induced gingival enlargement is well recognized.

Genetic polymorphisms affecting collagen metabolism, fibroblast function, and immune response likely contribute to this variability. For example, differences in the expression of fibroblast receptors or variations in MMP genes may influence the extent of gingival overgrowth in response to CCBs.

Research into genetic markers and fibroblast phenotypes is ongoing and may pave the way for personalized risk assessment and tailored management strategies in the future.

5. EPITHELIAL-MESENCHYMAL INTERACTIONS

Emerging evidence suggests that drug-induced changes in the gingival epithelium also contribute to DIGE. Altered epithelial cell signaling and proliferation may affect the underlying connective tissue by modulating fibroblast activity and inflammatory responses. The complex epithelial-mesenchymal crosstalk thus plays an integral role in the pathogenesis of gingival enlargement.

CLINICAL FEATURES

Antihypertensive drug-induced gingival enlargement (DIGE) typically presents as a progressive and painless increase in the size of the gingival tissues. The clinical manifestations vary widely depending on the duration of drug exposure, individual susceptibility, oral hygiene status, and concurrent inflammatory conditions. Understanding these features is critical for early diagnosis, differentiation from other causes of gingival enlargement, and guiding appropriate management.[13,14]

1. Onset and Progression[13]

• Onset: Gingival enlargement commonly develops within 1 to 3 months after initiation of antihypertensive therapy, particularly with calcium channel blockers such as nifedipine and amlodipine. However, onset can be delayed up to 6 months or more in some cases.

• Progression: The enlargement generally progresses gradually but may accelerate in the presence of poor plaque control or secondary inflammation. In some instances, the overgrowth stabilizes or regresses upon cessation or substitution of the offending drug.

2. Distribution and Location

• Predominant Sites: Enlargement primarily affects the anterior labial gingiva, more commonly involving the maxillary and mandibular anterior teeth. (Figure -1)

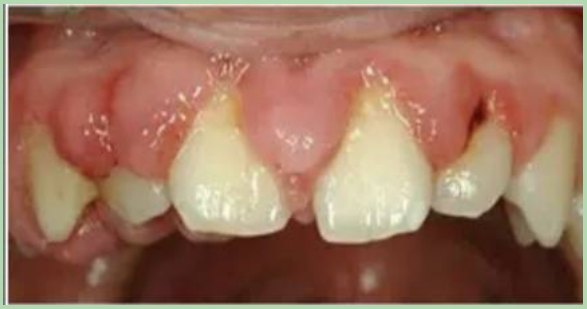


Figure 1: DIGO in anterior labial gingiva

• Extent: The enlargement may initially present as localized interdental papillae overgrowth but can extend to involve marginal and attached gingiva, sometimes covering substantial portions of the clinical crowns.

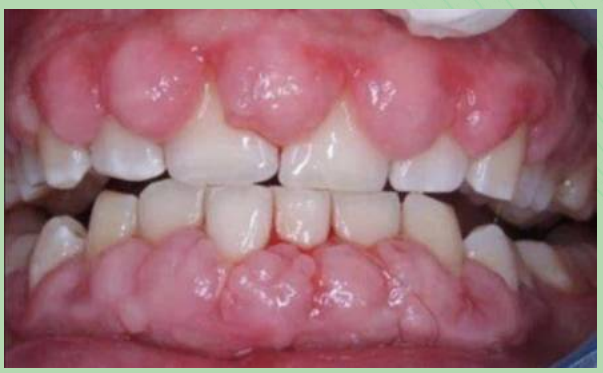
• Other Sites: Less frequently, the palatal or lingual gingiva may be involved.

3. Morphology and Appearance 15(Figure:2 &3)

• Color and Texture: The gingiva often appears firm and pale pink, consistent with fibrotic tissue proliferation. In early stages or with secondary inflammation, erythema and edema may be evident.

• Surface Characteristics: The surface may be smooth or exhibit a lobulated, nodular contour, with exaggerated stippling often absent in severe cases.

• Consistency: Lesions typically demonstrate a resilient, fibrotic consistency on palpation, distinguishing them from purely inflammatory gingival swelling.



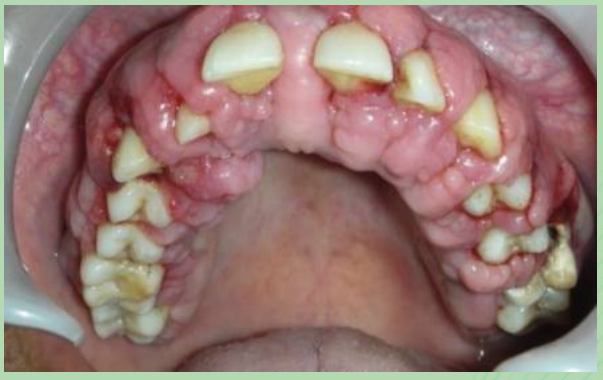


Figure:2&3 Morphology and appearance

4. Symptoms[16]

• Generally, gingival enlargement induced by antihypertensive drugs is asymptomatic in early stages.

• As the overgrowth progresses, patients may experience:

o Discomfort or tenderness, especially if secondary inflammation or ulceration occurs.

o Difficulty in maintaining oral hygiene, leading to increased plaque accumulation.

o Functional impairments such as difficulty in mastication, speech articulation, and toothbrushing.

o Aesthetic concerns due to the bulky appearance of gingiva.

5. Complications[17]

• Plaque Retention and Periodontal Disease: The enlarged gingiva creates niches conducive to plaque retention, exacerbating gingival inflammation and potentially leading to periodontal pocket formation, attachment loss, and tooth mobility.

• Tooth Displacement: Severe overgrowth can cause spacing or malalignment of teeth due to mechanical pressure.

• Interference with Prostheses: In patients with dentures or other prosthetic devices, gingival enlargement can impair fit and function.

Diagnosis[18,19]

The diagnosis of antihypertensive drug-induced gingival enlargement (DIGE) is primarily clinical but must be supported by a detailed medical and dental history, thorough examination, and, when necessary, histopathological evaluation. An accurate diagnosis is essential not only for differentiating DIGE from other forms of gingival overgrowth but also for formulating an effective treatment plan that balances systemic disease control with oral health management.

1. Medical History

A meticulous review of the patient's medical history is the cornerstone of diagnosis.

• Drug History:

o Identify the use of antihypertensive drugs, particularly calcium channel blockers (CCBs) like nifedipine, amlodipine, or verapamil.

o Note the onset of symptoms in relation to the initiation, dosage, or duration of antihypertensive therapy.

o Assess for polypharmacy, especially the concurrent use of other gingival overgrowth–inducing drugs such as phenytoin or cyclosporine, which may have additive effects.

• Systemic Conditions:

o Rule out systemic diseases such as leukemia, which can cause gingival infiltration.

o Evaluate the patient's blood pressure control and any recent medication changes.

2. Dental and Oral History

• Investigate:

o Oral hygiene practices (frequency and effectiveness of brushing/flossing).

o History of gingival bleeding, discomfort, or previous episodes of overgrowth.

o Past dental treatments (e.g., restorations, prostheses) that may affect gingival health.

3. Clinical Examination

A comprehensive intraoral evaluation should be performed, with particular attention to:

• Site and Distribution:

o DIGE typically begins at the interdental papillae in the anterior regions, especially the labial surfaces.

o Enlargement may extend to marginal and attached gingiva, potentially covering tooth crowns in severe cases.

• Character of Tissue:

o Early stages: Soft, erythematous, and edematous.

o Chronic stages: Firm, fibrotic, pale pink with a lobulated or nodular appearance.

• Severity Assessment:

o Several clinical indices can help quantify the degree of gingival enlargement:

1. Angelopoulos & Goaz Index
2. Modified Miller and Damm Index
3. Bokenkamp Index (used in pediatric patients with cyclosporine use but adaptable)

• Associated Findings:

o Bleeding on probing

o Pseudopocket formation due to tissue overgrowth

o Poor access for oral hygiene

o Signs of secondary inflammation (ulceration, pain, halitosis)

4. DIFFERENTIAL DIAGNOSIS [20]

Condition	Distinguishing Features
Inflammatory gingival enlargement	Associated with poor oral hygiene; resolves with scaling and improved oral care
Hereditary gingival fibromatosis	Familial history; begins early in life; generalized fibrous overgrowth
Leukemic gingival infiltration	Accompanied by systemic symptoms (fever, fatigue); soft, spongy, and hemorrhagic gingiva
Hormonal gingival enlargement	Occurs during puberty or pregnancy; usually resolves postpartum or after hormonal changes
Other drug-induced enlargement	Requires careful drug history (phenytoin, cyclosporine); may present similar morphology

5. RADIOGRAPHIC EVALUATION

•In most cases, radiographs do not show bone involvement unless there is concurrent periodontal disease.

•Intraoral periapical and orthopantomograms (OPGs) help evaluate:

o Bone levels

o Root length and proximity

o Subgingival calculus

o Periodontal pockets or abscesses

6. HISTOPATHOLOGICAL EXAMINATION (FIGURE:4)

Biopsy is not routinely required but may be indicated when:

• Diagnosis is uncertain

• Lesions are atypical in presentation

• Malignancy or systemic disease is suspected

Typical Histological Features:

• Epithelium: Hyperplastic, with elongated rete pegs

• Connective Tissue: Dense collagen bundles, minimal inflammatory infiltrate in non-inflamed areas

• Fibroblasts: Increased number, with active morphology

• Blood Vessels: Reduced vascularity in chronic lesions

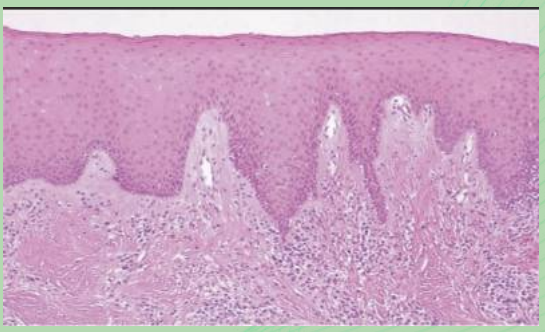


Figure :4 Histological features

7. Laboratory Investigations (if needed)

• Complete Blood Count (CBC): To rule out hematologic disorders (e.g., leukemia)

• Genetic testing (research setting): Investigational studies may evaluate polymorphisms related to collagen metabolism or fibroblast response.

MANAGEMENT[21,22,23]

The management of antihypertensive drug-induced gingival enlargement (DIGE) requires a multidisciplinary approach that integrates medical, dental, and sometimes surgical strategies. The primary objectives are to:

• Eliminate or reduce the gingival overgrowth

• Restore and maintain gingival health

• Preserve or improve the patient's oral function and aesthetics

• Prevent recurrence

Management is most effective when tailored to the individual patient's medical needs, drug regimen, severity of gingival involvement, and oral hygiene status.

1. Drug Modification (Medical Management)

a. Consultation with the Prescribing Physician

• The first step involves discussing the possibility of substituting or discontinuing the causative antihypertensive drug (usually a calcium channel blocker).

• For example, switching from nifedipine or amlodipine to an alternative antihypertensive such as:

o Angiotensin-converting enzyme (ACE) inhibitors

o Angiotensin receptor blockers (ARBs)

o Thiazide diuretics

o Beta-blockers

• Any change must be made under medical supervision to ensure blood pressure remains well controlled.

b. Dose Adjustment

• In some cases, reducing the dosage of the calcium channel blocker may result in partial regression of gingival enlargement.

2. Nonsurgical Dental Management

a. Plaque Control and Oral Hygiene Improvement

• Effective and meticulous oral hygiene is essential to reduce inflammation, which plays a synergistic role in DIGE.

• Patient education and motivation are critical:

o Brushing twice daily with a soft toothbrush

o Flossing or interdental cleaning

o Use of antimicrobial mouth rinses (e.g., 0.12% chlorhexidine)

b. Professional Dental Cleaning

• Scaling and root planing (SRP) to remove plaque and calculus deposits

• Frequent maintenance visits (every 3 months) are recommended to prevent recurrence

c. Adjunctive Therapies

• Local or systemic antibiotics (e.g., metronidazole) may be considered if there is secondary infection

• Anti-inflammatory agents (e.g., topical corticosteroids) have been used in some cases but with limited long-term benefit

3. Surgical Management

Surgical intervention is indicated when:

• Overgrowth is severe

• It interferes with function or aesthetics

• It is unresponsive to medical and nonsurgical therapy

a. Gingivectomy

- The traditional method involving excision of the overgrown tissue with a scalpel
- Provides immediate results and allows access for better oral hygiene
- May be associated with postoperative bleeding and discomfort

b. Flap Surgery

- In cases with underlying periodontal involvement, flap surgery allows access for root debridement and bone contouring

c. Laser Gingivectomy

- CO_2 or diode lasers can be used for precise tissue removal with minimal bleeding and discomfort
- Often preferred in aesthetic zones or for medically compromised patients

d. Electrosurgery

- Also used for tissue removal but may cause greater thermal damage and delayed healing

4. Postoperative Care and Long-Term Maintenance

- Patients should be kept on a strict recall schedule (every 3–6 months)
- Continuous reinforcement of oral hygiene is necessary
- Monitoring for recurrence is essential, especially if the causative drug could not be discontinued

Prognosis 24,25

- Mild to moderate enlargement often shows partial or complete regression after drug modification and plaque control.
- Severe or fibrotic overgrowth usually requires surgical excision.
- Recurrence is possible, particularly if oral hygiene is poor or if the offending drug cannot be discontinued.
- With proper management, functional and aesthetic outcomes are favorable, and patients can maintain gingival health over the long term.

CONCLUSION

Antihypertensive drug–induced gingival enlargement is a significant oral side effect, most commonly associated with calcium channel blockers such as nifedipine, amlodipine, and verapamil. Although the exact mechanism is not fully understood, it is believed to involve a complex interaction between the drug's effect on fibroblast activity, inflammation due to local plaque accumulation, and individual genetic susceptibility. Early recognition by dental professionals and physicians is essential to prevent functional and aesthetic complications.

Effective management involves improved oral hygiene, periodontal therapy, and in some cases, substitution or dose adjustment of the offending medication in consultation with the patients physician. Overall, a multidisciplinary approach between dentists and medical practitioners is vital to ensure optimal systemic and oral health outcomes for affected patients.

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ELASTOGRAPHY – A NEW NON-INVASIVE DIAGNOSTIC METHOD IN DENTAL PRACTICE

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ABSTRACT

Elastography is a modern, non-invasive technique for assessing tissue stiffness, traditionally used in liver disease diagnostics, with promising applications in dentistry now emerging. The core principle of elastography is based on tissue compression: softer tissues exhibit greater strain under compression, while harder tissues show less strain. This strain pattern is visualized as colour images for hard tissues, while soft tissues appear darker, effectively mapping tissue elasticity. Elastography aids in distinguishing benign from malignant tumours, differentiating lymph node types, evaluating masseter muscle stiffness, and assessing swellings in the parotid and submandibular glands. Key benefits include precise definition with clear margin delineation, low cost, minimal time requirements, reliability, and fewer side effects. However, challenges remain, such as uneven compression over bony areas, limited control over transducer compression depth, suboptimal strain imaging for large lymph nodes due to probe contact issues, and potential artifacts from nearby moving blood vessels or tissues. This review discusses elastography as an innovative, non-surgical diagnostic approach, its mechanism, and applications in dental practice.
Keywords: Elasticity, Ultrasound, Stiffness, Elastography

1. RELEVANCE

Change is the only constant, and to keep up with evolving trends and the world ahead, oral physicians must embrace advancements in dentistry. One such innovation is elastography. This review explores elastography, a modern non-surgical diagnostic method, its underlying mechanism, and its clinical applications.

2. ELASTOGRAPHY

Since the time of Hippocrates, physicians have palpated tissues to detect and assess variations in tissue stiffness, aiding in diagnosis.

Elastography builds on this principle as a non-surgical imaging technique that differentiates between healthy and diseased tissues by evaluating mechanical

properties at depths beyond the reach of traditional palpation. Defined as an imaging method that measures tissue elasticity in vivo, elastography was first introduced by Ophir et al. in 1991. [1,2] Initially used primarily for liver disease assessment and characterizing breast lesions. [3,4] Elastography is now gaining attention for its emerging potential in dental practice.

3. TECHNIQUES OF ELASTOGRAPHY

Elastographic techniques are employed in both ultrasound (US) and magnetic resonance (MR) imaging. Ultrasound elastography, also known as sonoelastography, relies on compression of tissues to generate distinct ultrasound signals that reflect variations in tissue stiffness. Two primary approaches are used in US elastography: quasi-static or strain-based (measuring tissue strain or deformation under force) and dynamic or shear wave-based (analysing the speed of shear wave propagation through tissues). [1,4] MRI-based elastography, on the other hand, is a quantitative method that operates on the principle of shear pressure waves and the tissue's stretchable movement. [1]

3.ULTRASOUND ELASTOGRAPHY

3.1.A.STRAIN IMAGING

Ultrasound elastography techniques primarily fall into two categories: strain imaging and shear wave imaging, each of which is further divided into specific variable schemes.

Strain imaging is a technique that qualitatively evaluates Young's modulus by applying normal stress and measuring the resulting normal strain. Additionally, strain imaging can be
categorized into two mechanisms: strain elastography (SE) and acoustic radiation force impulse (ARFI).[4]

ACOUSTIC RADIATION FORCE IMPULSE (ARFI)

Acoustic Radiation Force Impulse (ARFI) is an alternative method for measuring strain in situations where excitation or external stimulation is not feasible. This technique employs a high-intensity acoustic "pushing pulse" with a brief duration of 0.1 to 0.5 milliseconds. The spatial peak pulse average is 1400 W/cm², while the spatial peak temporal average is 0.7 W/cm². This pulse displaces tissue in the normal direction, perpendicular to the surface, resulting in tissue displacement of approximately 10 to 20 micrometers.[4,5]

The displacement within a defined region of interest (ROI) is measured using the same techniques as those used in strain elastography. Similar to strain elastography, the resulting displacements can be presented as an elastogram overlaid on a B-mode image. Although the authors did not emphasize this technique due to the limited amount of research conducted on it and its relatively recent development, it is noteworthy that ARFI imaging does not depend on transducer compression, unlike other quasi-static elastography methods. This characteristic makes it particularly useful for assessing and imaging deeper organs.[4,6]

STRAIN ELASTOGRAPHY

Strain Elastography (SE) is a qualitative or semi-quantitative imaging technique that evaluates and compares the relative stiffness of tissues when an external force is applied. Due to its reliance on external stimuli, SE is considered one of the more challenging elastography techniques. Strain elastography is categorized into two methods: excitation (manual compression) and the assessment of internal physiological motion.[4]

The manual compression method involves the operator applying pressure to the region of interest, making it particularly effective for evaluating superficial organs and their associated pathologies, such as those found in the thyroid or breast tissue. The second method, however, utilizes information from tissue displacement caused by internal physiological motion, which does not depend on superficial compression. This characteristic makes it advantageous for assessing deeper organs, including those in the cardiovascular and respiratory systems.[7]

3.1.B.SHEAR WAVE IMAGING

Shear wave imaging is an elastography technique that generates shear waves in either perpendicular or parallel directions. After dynamic stress is applied, researchers estimate tissue elasticity by measuring the speed of these shear waves both qualitatively and quantitatively. Currently, there are three primary techniques used for shear wave imaging: one-dimensional transient elastography (1D-TE), two-dimensional shear wave elastography (2D-SWE), and point shear wave elastography (pSWE).[8]

ONE-DIMENSIONAL TRANSIENT ELASTOGRAPHY (1D-TE)

One-dimensional transient elastography (1D-TE) is the first shear wave imaging technique developed and remains the most widely available and commonly used among the three methods. Its primary application is in assessing liver fibrosis, which led to the creation of Fibroscan® (Echosens, Paris, France), a non-invasive ultrasound-based imaging technique designed to evaluate fibrosis and other liver conditions. The Fibroscan® probe features a mechanical vibrating device along with an ultrasound transducer that generates shear waves through external vibration, allowing them to propagate through the tissue. While 1D-TE is ultrasound-based, it operates without the need for direct B-mode imaging; instead, it employs A-mode ultrasound to measure shear wave speed, which is then used to calculate Young's modulus.[9]

TWO-DIMENSIONAL SHEAR WAVE ELASTOGRAPHY (2D-SWE)

Two-dimensional shear wave elastography (2D-SWE) is the latest advancement in shear wave imaging techniques, utilizing dynamic stress applied via Acoustic Radiation Force Impulse (ARFI) as the excitation method. In 2D-SWE, the shear waves measured are perpendicular to the ARFI application, and the technique employs multiple focal zones that are interrogated rapidly, rather than focusing on a single location. This evolution allows for the visualization of a color-coded quantitative elastogram superimposed on a B-mode image, enabling the easy production of tissue stiffness information.[10]

POINT SHEAR WAVE ELASTOGRAPHY (pSWE)

Point shear wave elastography (pSWE), developed in 2008, also relies on dynamic stress from Acoustic Radiation Force Impulse (ARFI), but it concentrates on a single focal location, in contrast to 2D-SWE, which uses multiple focal points. While 1D-TE is mainly utilized for liver applications, pSWE presents several advantages that may enable it to replace 1D-TE in shear wave imaging. Unlike 1D-TE, which relies heavily on A-mode ultrasound, pSWE
employs B-mode ultrasound to directly visualize and select the region of interest. Additionally, pSWE can be easily performed on conventional ultrasound machines without the need for specialized probes or equipment; however, it does not provide images depicting tissue stiffness. [11]

3.2. MRI -BASED ELASTOGRAPHY

Magnetic Resonance Elastography (MRE) is a method developed to assess the propagation of shear waves in tissue utilizing magnetic resonance imaging (MRI) techniques. The initial demonstration of using MRI to record shear wave propagation was conducted in a collaborative study between the University of Michigan and Artann Laboratories from 1995 to 1996. In this study, an ultrasound transducer was mounted on rubber phantoms that were either homogeneous or contained two cylindrical inclusions. A single ultrasound pulse at 555 kHz, lasting 1.5 ms, achieved a remote displacement of 20 microns.

The displacement was measured using phase-sensitive MRI with a pair of opposite polarity gradient pulses. In the homogeneous phantom, the shear wave propagation is cylindrically symmetric, radiating outward from the point of initial displacement. In contrast, while the propagation in the phantom with the two inclusions begins similarly to that in the homogeneous phantom, the shear wave travels much faster upon reaching the inclusions, due to the differences in elastic modulus between the two media.[12,13]

4. THEORY OF ELASTOGRAPHY

Strain elastography assesses tissue elasticity by measuring displacement caused by compression; hard tissues show less displacement compared to soft tissues. This technique evaluates tissue movement along the axis of the applied force. In contrast, shear wave elastography analyzes elasticity based on the speed of transverse shear waves, which propagate faster in hard tissues, allowing differentiation between hard and soft tissues. Shear waves, generated by the tangential sliding of tissue particles, travel perpendicular to the direction of the applied force.[1]
Elastograms come in two types: grayscale and colour. In grayscale elastograms, hard tissues appear dark while soft tissues are bright. Colour elastograms use shades of red, yellow, green, and blue, representing tissue hardness in increasing order.

Tissue stiffness is quantified by Young's modulus, expressed in Pascals (Pa) or kilopascals (kPa), which defines the relationship between applied stress and induced strain.

Hard tissues have a higher Young's modulus compared to soft tissues. Elasticity varies across different tissues and within the same tissue during conditions like inflammation or malignancy, as stiffness generally increases with disease. Elastography, based on the principles of physical elasticity, tracks tissue motion and estimates induced strain distribution by applying pressure to the examined region. Young's modulus (E) relates to the shear modulus (G) of tissue through the formula E=3G.

Biological tissues can easily change shape when compressed due to their high-water content, while their volume remains relatively constant.[2,8]

TYPES OF SOFT TISSUE		YOUNG'S MODULUS (kPa)
BREAST	NORMAL FAT	18-24
	NORMAL GLANDULAR	28-66
	FIBROUS TISSUE	96-244
	CARCINOMA	22-560
PROSTATE	NORMAL	55-71
	BPH	36-41
	CARCINOMA	96-241
LIVER	NORMAL	0.4-6
	CIRRHOSIS	15-100

Table 1- Tissue stiffness

Different tissues exhibit varying elasticity, and pathological conditions, such as inflammation or malignancy, can alter the elasticity within the same tissue type (see Table 1). Tissue stiffness typically increases in the presence of disease, and this can be assessed by measuring tissue deformation under applied stress. Pathological changes can be visualized with high- contrast imaging.

5. HOW DOES THIS WORK??

Elastography allows for the assessment of the stretching effects in various tissues, capturing images before and after compression. Low-frequency vibrations are generated within the tissue to induce shear stress. By applying pressure to the examined medium, tissue motion can be tracked, enabling the estimation of induced strain distribution. The tissue is imaged with the objective of analyzing the resulting stress, from which a parameter related to tissue stiffness can be derived. The technique is considered measurable if Young's Modulus or elasticity can be directly obtained from the analysis. A cross-correlation technique is employed to compare data, focusing on movement within a small area of tissue identified by the compression applied by the ultrasound transducer. [14]

6. APPLICATIONS OF ELASTOGRAPHY

Elastography can be used to evaluate a variety of diseases across multiple organs. It is effective for assessing superficial masses in organs such as the breast, scrotum, neck, and thyroid, as well as deeper structures like the uterus, ovaries, and prostate gland. Additionally, it can be applied to tissues with physiological displacements, including arterial walls, liver, and brain. An intracavitary transducer facilitates access to deeper organs through applied pressure. Numerous studies have explored the application of elastography in these organs, yielding promising results. The first elastographic image was produced in 1988 using a compressed breast phantom made from silicone rubber gel within a rubber prosthesis, featuring two nylon ball swellings measuring 25 mm and 6 mm in diameter, along with a recorded stress pattern.[12]

7. APPLICATIONS OF ELASTOGRAPHY IN ORAL AND MAXILLOFACIAL REGION

Elastography serves as a valuable adjunctive tool in diagnosing pathologies affecting the maxillofacial region. Its applications include:

- Assessing lymph node involvement in cancer
- Detecting superficial neck masses
- Estimating muscle stiffness in Myofascial Pain Dysfunction Syndrome
- Evaluating major salivary gland masses

Cervical lymphnodes

Lymph nodes in the maxillofacial region are typically well-defined, fusiform, or kidney bean- shaped, featuring a medium to low reflective, homogeneous cortex and a reflective antral hilus. These characteristics make them excellent candidates for elastographic examination, as they are easily accessible and can be effectively compressed against the underlying structures. This technique is also valuable for guiding percutaneous biopsies and excisions of lymph nodes to detect cancer recurrence. [2]

Lyshchik et al. utilized sonoelastography in thyroid cancer patients to differentiate between benign and metastatic cervical lymph nodes, using histopathologic findings as the standard reference for comparison. They observed that the ultrasound elastograms were often unclear, with most benign nodes and surrounding anatomical tissues displaying similar brightness. In contrast, malignant nodes appeared darker and exhibited better marginal delineation.[15]

Additionally, Alam et al. evaluated the diagnostic accuracy of sonoelastography and B-mode sonography in assessing nodal swelling, concluding that the differentiation between reactive and cancerous lymph nodes was significantly improved with these imaging techniques. [16]

Salivary glands

Determining the pathology of salivary gland conditions can be challenging with conventional imaging techniques, despite the fact that salivary gland swellings are often superficial. However, elastography has proven useful in assisting surgeons with surgical decision-making by identifying cancerous lesions. Dana Dumitriu et al. assessed the effectiveness of real-time sonoelastography for the differential diagnosis of salivary gland tumors, using histopathological confirmation for comparison. Their study did not yield consistent results, revealing considerable variation in scores among different metastatic swellings. Ultimately, the study concluded that elastography has limited utility in distinguishing between benign and malignant salivary gland masses.[17]

Muscle stiffness

Measuring individual muscle force can provide insights into neuromuscular physiology, motor control, biomechanics, and robotics, as well as aid in diagnosing and managing both neurological and orthopedic diseases. Elastography plays a vital role in assessing muscle stiffness during contraction, which in turn allows for an estimation of muscle force.

It can be used to evaluate various muscle pathologies, including myospasm, myositis, myositis ossificans, hematomas, and muscle tumours.

Ariji et al. conducted a study using sonoelastography to assess the stiffness of the masseter muscle, exploring the relationship between muscle stiffness and the most pleasant massage pressure in patients with Myofascial Pain Dysfunction Syndrome.[18] Similarly, Takashima classified 26 females with bilateral masseter muscle pain as having temporomandibular disorder (TMD) and compared them to 24 healthy female controls. The findings indicated that TMD patients exhibited higher muscle stiffness. Furthermore, pain was found to have a positive correlation with muscle stiffness, while the maximum mouth opening was considered a negative correlation, both with and without pain. [19]

Detecting superficial neck masses

Ultrasound elastography can be readily conducted on the thyroid gland because of its accessible superficial location. Elastography measures the elastic properties of tissues by evaluating how they deform under applied pressure. Malignant tumours often exhibit increased stiffness compared to benign lesions, allowing for effective differentiation. As a non-invasive imaging modality, elastography allows for the assessment of neck masses without the need for surgical procedures or biopsies, reducing patient discomfort and risk. The information obtained from elastography can guide clinicians in selecting the most appropriate area for biopsy, improving the accuracy of sampling and increasing the likelihood of detecting malignancy.

Elastography can be used alongside traditional imaging techniques, such as ultrasound or MRI, enhancing diagnostic accuracy by providing additional information about the characteristics of neck masses. By evaluating changes in tissue stiffness over time, elastography can assist in monitoring the progression of known lesions, helping to assess treatment response or detect recurrence. The technique can improve the delineation of lesion margins, making it easier to characterize the masses and identify their relationship to surrounding tissues.[20]

ADVANTAGES[2]

1. Finer Marginal Delineation: Provides clearer delineation of tissue margins.
2. Differentiation of Lesions: Enables the distinction between benign and malignant lesions.
3. Enhanced Diagnostic Information: Offers additional diagnostic insights that complement B-mode ultrasound images.
4. Guidance for Procedures: Serves as guidance for percutaneous biopsies or nodal dissections.
5. Detection of Cancer Recurrence: Aids in determining the recurrence of cancer.

LIMITATIONS

1. Uncontrolled Compression: The transducer cannot control the compression of tissues and their extensions.
2. Suboptimal Imaging of Large Lymph Nodes: Large lymph nodes may produce suboptimal strain images due to inadequate probe contact over a larger area.

3. Artifacts from Motion: Artifacts can occur as a result of the motion of surrounding vessels and tissues.

RECENT TRENDS

• Vibroacoustography (VA): Combines mechanical vibrations and ultrasound to visualize and assess tissue properties. This technique that employs the acoustic response (acoustic emission) of an object to the harmonic radiation force of ultrasound for imaging and material characterization. This method generates acoustic emissions by focusing two ultrasound beams with slightly different frequencies at the same spatial point. The resulting vibration of the tissue occurs due to the ultrasound radiation force applied at a frequency equal to the difference between the two primary ultrasound frequencies.[21] It has a wide range of applications in both medical and industrial imaging and characterization. In the medical field, VA has been explored for imaging various tissues, including the breast, prostate, and thyroid. It has also been utilized to image mass lesions in excised human liver samples, as well as in arteries, bones, and microbubbles.[22]

• Supersonic Shear Imaging Elastography (SSI): A newer method that uses high-frequency ultrasound to provide real-time images of tissue stiffness with enhanced resolution. This method focuses the radiation force on a single location, then adjusts the depth of that focal point, allowing shear waves generated from multiple focal locations to constructively interfere, resulting in a conical shear wave. This technique is known as supersonic shear imaging (SSI), as the focal point of the radiation force moves faster than the shear wave speed within the medium. SSI has been effectively applied in assessing phantoms, liver, breast tissue, and skeletal muscle.[23]

CONCLUSION

Elastography has garnered significant attention for its inherent ability to provide mechanical contrast and its considerable diagnostic potential. Initial findings indicate that both strain and shear wave elastography could serve as valuable adjunctive tools in diagnosing pathologies affecting the maxillofacial region. However, the accuracy of these methods needs to be validated through studies involving larger patient groups. As technology continues to advance, elastography is expected to evolve into a non-invasive diagnostic complement to ultrasonography, enhancing the confirmation of certain tissue characteristics suggested by ultrasound.

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MANAGEMENT OF GAGGING IN CLINICAL PRACTICE -A REVIEW OF LITERATURE.

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ABSTRACT

The gag reflex is a normal, involuntary protective mechanism that prevents foreign objects from entering the pharynx, larynx, or trachea. However, in dental practice, an exaggerated gag reflex can present a significant challenge, often hindering routine clinical procedures . It can also lead to dental anxiety and treatment avoidance among patients. Understanding the etiology of gagging—whether psychological, physiological, or iatrogenic—is essential for effective management.

This article reviews the existing literature on the causes and management of gag reflex in dental practice and discusses various behavioral, pharmacological, and clinical strategies to help practitioners manage gagging efficiently and improve patient comfort and treatment outcomes.

INTRODUCTION

Every clinician, at some point in their practice, is likely to encounter a patient with a pronounced gag reflex. . It is one of the most common and distressing problems faced by both patients and practitioners. An exaggerated gag reflex can interfere with various dental procedures such as impression making, radiography, prosthesis fitting, and intraoral examinations, thereby affecting the quality of treatment and patient cooperation. Effective management of gagging requires a thorough understanding of its etiology, physiological basis, and the wide range of behavioral and clinical techniques available to control it.

This article reviews the literature on the management of gag reflex in clinical practise and discuss evidence based to minimize its impact on dental care.

THE BACKGROUND OF GAG REFLUX

Various stimulating or triggering factors of gag reflex are reported in literature ,but broadly the gag reflex has been classified into following groups :

Somatogenic Gagging-those in whom the physical stimulation produce the gag.

Example :insufficient retention of the prosthesis,thick posterior borders of the denture,inadequate posterior seal,lack of tongue space and malocclusion.

Psychogenic Gagging-those in whom the stimulation appears to be psychic in origin. Example :gagging triggered by fear,anxiety and taste.

Iatrogenic Gagging- caused by dentist himself .

Example :suction tip touching the pillars of fauces or mouth mirror contacting the posterior dorsum of tongue during check up,overextension of denture and abnormal thickness of the posterior palatal border.

Extra Oral Symptoms

These include excessive salivation, lacrimation, coughing, fainting, or in minority of patients ,a panic attack and sweating.the patient extends the head ,arms neck and back in an attempt to completely withdraw from the offending stimuli.[1]

Inra Oral Symptoms

The patient who gags may present with a range of disruptive reaction;from simple contraction of palatal or circumoral musculature to spasm of the pharyngeal structures,accompanied by vomiting.1

Trigger Zone Of Gag Reflex[2]

Gagging may be elicited by nontactile and tactile stimulation of certain intraoral structures.
Five intraoral areas is known as trigger zones:

- Palatoglossus & palatopharyngeal folds
- Base of tongue
- Palate
- Uvula and
- Posterior pharyngeal wall

Nontactile sensations such as

1. visual,
2. Auditory
3. Olfactory stimuli

This technique is known as supersonic shear imaging (SSI), as the focal point of the radiation force moves faster than the shear wave speed within the medium. SSI has been effectively applied in assessing phantoms, liver, breast tissue, and skeletal muscle.[23]

ETIOLOGY OF GAGGING[3]

Five factors that are believed to be important in etiology are-
1. Local and systemic disorders
2. Anatomic factors
3. Psychological factors
4. Physiologic factors
5. Iatrogenic factors
1. Local and systemic disorders-
1. Nasal obstruction
2. Postnasal drip
3. Sinusitis
4. Nasal polyp
5. Mucosal congestion of Upper respiratory tract
6. Dry mouth
7. Chronic Gastrointestinal disease
8. Chronic gastritis peptic ulceration
9. Carcinoma of stomach
10. Hiatus hernia
11. Uncontrolled diabetes

2. Anatomic factors-
Anatomic abnormalities, oral and pharyngeal sensitivity predispose a patient to gag when dentures are poorly constructed.
1. Along soft palate
2. Sudden drop at the junction of hard and soft palate .
3. An atonic and relaxed soft palate elicits gagging by allowing the uvula to contact the tongue and the soft palate to touch the posterior pharyngeal wall.

3. Psychological factors

Systemic conditions that have psychosomatic components are-

1. Temporomandibular paindysfunction syndrome
2. Atypical facial pain
3. Denture intolerance
4. Burning mouth syndrome.

Psychosomatic reaction may be active or passive. An active reaction is due to factors that currently have some functional purpose in the patient's life situation for various psychologic reasons patients may gag to gain attention from the dentist, and/or to avoid the outcome of treatment. In contrast, a passive reaction is the result of, conditioned reflexes established earlier in life for various reasons, the causes of which are no longer functionally important. Kamer and Braham stated that "fear is almost always the underlying factor influencing the psychological gagger." This fear may be generalized and vague or quite specific. Often the fear is not merely that of pain. Some patients gag because of an abnormal fear of swallowing a foreign object.

4. Physiologic factors

Extraoral stimuli: The mere sight of a mouth mirror or impression tray is stimulus enough to cause some patients to gag. Landa observed a deaf patient suffer a spasm of gagging while viewing the gagging of another patient.

Acoustic stimuli- The sound of the wife gagging was sufficient to precipitate an attack of gagging in the husband.

Olfactory stimuli - Certain smells may cause a patient to gag. The smell of various dental substances, cigarette smoke on the dentist fingers and even perfume have been reported as olfactory stimuli to the gag reflex.

Intraoral stimuli-The tactile response is roughly divided into two response regions : hyposensitive and hypersensitive regions. Line drawn through the fovea palatinae demarcates relatively hyposensitive anterior and hypersensitive posterior portion. The tongue was similarly divided into the hyposensitive anterior and hypersensitive posterior one third. Landa reported that the upper surface of the posterior one third of tongue is the most sensitive area in oral cavity.

5. Iatrogenic factors[4] -

In the otherwise non-gagging patient, poor execution of intraoral procedures may elicit the gag reflex. Sensitive tissues may be stimulated because of rough or careless technique and temperature extremes of instruments or because of-

1. Inadequate Posterior Palatal Seal and loose denture
2. Overloaded impression trays
3. Unstable & poorly retained prosthesis-produced movement of the denture base, which produces a tingling sensation and gagging
4. Overextended border of prosthesis particularly in the posterior area of palate and retromylohyoid space , distolingual part of mandibular denture- this impinges one or more of the trigger areas and thus produce gagging.
5. Placing maxillary teeth too far in a palatal direction and mandibular teeth too far lingually, so that dorsum of the tongue is forced into pharynx during the act of swallowing.

Assessment of gagging

Gagging is a clinical problem for the clinician or the patient may be either absent or reduced or exaggerated depending on the circumstance. Gagging may be assessed based on the Gagging Severity Index (GSI) and the Gagging Prevention Index (GPI), using five defined grades as proposed by Dickinson and Fiske. Grade one on the scale represents a person with a normal gag reflex and grade 5 represents a situation that cannot be clinically managed [5].

MANAGEMENT

A detailed case history should be recorded to identify the etiology of gagging.The management of gagging patient depends upon the severity and etiology of the gag reflux. The clinician should be calm,patient and always be willing to hear the problems of the treating patient,as attitude of the dentist will directly influence the outcome of the treatment.

Mild to moderate gagging problems can be effectively managed in the clinic itself,but in severe cases,patient should be refered to Hypnotherapist.The main objective of a dentist is to free the anxiety of the patient ,gain confidence in him and make him /her comfortable with our clinic and staffs. Prior to every procedure,patient should be informed about it.The prime duty of a clinician is to render the treatment to the patient .[6]

Various methods of management of gagging reflux are pharmacological treatment,behaviour modifications ,TENS,acupuncture,acupressure and surgical techniques .These methods can be employed either alone or in combination to overcome exaggerated gagging reflux.[7,8]

PHARMACOLOGICAL TREATMENT

a.Locally acting- peripherally acting drugs/Local anesthesia - The

Topical use of local anesthetics have shown to reduce the gag reflex.. It can be applied in the form of sprays gels lozenges,mouth rinses or injection.Topical lidocaine (flavoured) can be applied with a cotton roll on the palate and back of the tongue.Benzocaine spray can be used to briefly numb the areas for taking x-rays or taking an impression. The deposition of LA around the posterior palatine foramen can be used for gaggers. Injection of LA is not advisable as it can distend the tissue resulting in an accurate impression,which may inturn affect the retention of prosthesis.

Adding lignocaine solution(8ml of 2%lignocaine with 1 part in 100,0000 epinephrine) to measured volume of water and then add impression material(alginate) into it and mix thoroughly.Load the tray and place the tray in patient's mouth and press until it is set.[3]

B. Sprinkiling a little table salt on the tongue- placing table salt on the anterior portion of the tongue is said to stimulate the taste buds located there which subsequently activates the chorda tympani nerve leading to suppression of the gag reflex.. Rinsing the mouth with Normasol(0.9% saline)is also effective.

C. Electrolyte tablet - an electrolyte tablet administered and retained intraorally a few minutes before the start of procedure can suppress gag reflex .Tablets can be prescribed for home use to patients who cannot perform oral hygiene procedures properly.Severe gaggers may need to repeat a dose in 15 to 20 minutes,

2. Centrally acting drug- It is only a short term solution for severe gagging problem and should not used routinely.

a. Tranquilizers like chlopromazine are useful in patient under strain/stress.

b. Semi hypnotic-antihistamines,parasympatholytics.

c. General Anesthesia - it is not much adviced on regular basis,but can do it as a last to patients who do not respond to any form of sedation or behaviour therapy.

d. Conscious Sedation - this help the patient to achieve a state of total relaxation.It helps the clinician to work for hours with ease and comfortable.The use of conscious sedation with inhalation,oral or intravenousagents may temporarily eliminate gagging during the procedure while maintaining reflexes that protect the patients airway,.

Behaviour modification

a. Swallowing with teeth apart -It has been observed that all patients who gag swallow with their teeth clenched,using teeth,lips and cheeks as a buttress for the tongue to push against.Teaching the patient to swallow with the teeth apart ,the tip of the tongue placed anteriorly on the hard palate, and the orbicularis oris muscules relaxed has been advocated.

b. Breathing Through Nose - instruct the patient to breathe through their noses. If the nose is congested,it is advisable to use nasal decongestant ,to clear the nasal passage.

c. Humming- this method is used while taking x-rays. They will find it difficult to hum and gag at the same time with the intraoral film in mouth,

d. Beware of the gag reflux in the morning - few patient is more sensitive to gag in the early morning. So try to give appointments to gaggers in the late afternoon or evening and also advice them to report in empty stomach.

Other behaviour modifications like asking the patient to rinse with cold water before any procedure ,using moistened and increased speed films also help in depressing the gag reflux.

Desensitization

this technique consists of exposing the patient to the feared stimulus in a way that the intensity ,duration and frequency of the noxious stimuli is slowly increased ,thereby allowing the patient to gently habituate with the produre to be performed.

Various dental tools such as mouth mirror and imoression trays can be given to the patient to take home and try introducing then into their mouths themselves along with digital massaging of the palate.They should keep a check of how long they feel comfortable by timing it.Doing this a few times in twice a day ,they will become sensitive to gag reflex.

Singers marble technique[9] is a method useful in assuring so called hopeless gaggers.It is a method by which the gag reflux can be exhausted. It consist of seven visits.-

1st visit-no intraoral examinations done.patient is advised to keep 5 round glass marbles(approximately 1/2 inch diameter)in the mouth one at a time at his disposal till all all five marbles are present in his oral cavity.patient was instructed to keep all the five marbles in his mouth continuously for one week,except while eating or drinking. Continous assurance should be given to the patient that he will be able to wear the denture without any discomfort

2nd visit - patient will be motivated and his ability to wear dentures should be evaluated.

3rd visit - before impression LA applied on the gag reflux areas. Preliminary impression made with impression compound.Base plate of matte finish is to be prepared.

4th visit -the lower base plate is inserted and the patient was told to continue to keep 3 marbles in his mouth in addition to base plate. A training bead (a small bead of coloured acrylic resin) was placed on the lingual aspect of the lower central incisors.patient should be assured that he is making excellent progress.

5th visit -the upper base plate was inserted,he was asked to keep both of them in his mouth continuously ,except when eating the use of marble was discontinued.

6th visit -occlusal rims were used to establish the jaw relation.the patient should continue wearing upper and lower base plates while the dentures are being acrylized.

7th visit -at first the completed lower denture is inserted and used along with the upper base plate. The training bead was placed in the lower denture as a guide to tongue position.The patient is instructed to to keep the tip of the tongue always touching the bead,which wouls prevent the lower denture from lifting. Next the upper denture was inserted.

Similarly a toothbrush,radiograph,impression trays,acrylic discs,buttons and training devices can be used to overcome gagging in patients.

Relaxation

This technique helps to reduce or abolish gag reflex.example:ask the patient to tense and relax certain muscle groups,starting with legs and working upwards,while providing continous reassurance in a calm atmosphere. Sometimes the patient might had some traumatic experience at dentist office in the earlier days. So try to communicate with the patient and assure them that you will start the treatment only once he is comfortable.

Hynosis

Hynosis is defined as inducing a state of altered awareness in which the critical faculty of the conscious mind is partially or totally suppressed and selective thinking is established. It is done by standing in front of the patient and directing him to keep his gaze fixed continuously on your eye during the entire procedure.If his gaze wander,call to his attention and tell him to take deep breath and hold it while you count from 1 to 5 and then tell the patient to relax and repeat the process again.

Hypnosis is a very time consuming approach and cannot be used as an immediate solution. It is recommended to performed by or under the guidance of an experienced hypnotherapist.Patients with pre-existing strong doubts of the treatments effectiveness will have a lower chance of success.

Transcutaneous electrical nerve stimulation(TENS)

TENS is the use of electric current produced by a device to stimulate the nerves for therapeutic purposes. Using this non -invasive nerve stimulation,the gag reflex can be suppressed.

There is a little study on this topic but in one study it has been found out that stimulation of the cranial nerves belonging to the superior laryngeal nerve branch would block the physiological response of gagging..

This is achieved by using a nerve stimulation device attached to the wrist.this maintained contact of an electrode with the ventral aspect of the wrist and generated a stimulating signal which indirectly stimulated the cranial nerves associated with gagging.

Acupuncture

Acupuncture is a system of medicine in which a fine needle is inserted through the skin to a depth of a few millimeters, left in place for a time, sometimes manipulated and then withdrawn. Dental treatment was then carried out and the effectiveness of acupuncture in preventing gagging is assessed.

Method -

Ear acupuncture was selected for the following reasons-

- There is a specific, recognized anti gagging point on the ear. The needles are not disturbed during access to the mouth for dental treatment .
- The needles are out of the patient's line of vision - a bonus for anyone with a dislike of needles .
- The technique involves the insertion of one, fine, single-use disposable needle of 7mm length into the anti gagging point of each ear to a depth of 3 mm. The needles are manipulated for 30 seconds prior to carrying out dental treatment. The needles remain in Situ throughout treatment and are removed before the patient is discharged.

The patient does not require an escort and is not inconvenienced in anyway following treatment. [10]

Hypnopuncture

As the name suggest ,it is the combination of hypnosis and acupuncture. It provides a therapeutic treatment plan for long -term therapy for patients with a distinctive gag reflex.dentist need to be trained in these advanced patient management techniques.Acupuncture gives an immediate effect for the patient.The hypnosis part concentrates on hypnosedation as well as positive suppression of negative experiences linked to the gag reflex.

Although its effect is faster than hypnosis alone,a number of appointments is required before the patient is able to overcome their gag reflex.

Acupressure

This follows the same principle as acupuncture, but the former stimulate the points with gentle finger pressure rather than fine needles and therefore is a less invasive technique. Chengjiang (REN-24) is an effective acupressure point for controlling the gag reflex during impression making. To make use of it locate the REN-24 point. It is situated in the horizontal mentolabial groove. Approximately midway between the chin and the lower lip. Apply light finger pressure with the index finger progressively increase the finger pressure until the patient feels some discomfort and distension.

The acupressure should start at least 5 min before impression making, continue through the impression procedure, and should be terminated only after the impression has been removed from the patient's mouth.

Pressure can be applied by the patient, dental assistant, or dentist.

Surgical technique[3]

Leslie reported a surgical technique to relieve gagging for the patient unable to tolerate complete dentures. The basis for this technique stems from the observation that persistent gagging results from an atonic and relaxed soft palate, which is found in the nervous system. As a suspended organ, the palate is not in normal relation with the uvula and the pharyngeal wall. In such cases, the uvula touches the tongue and the soft palate rests back on the pharyngeal wall. This produces a tendency to gagging and nausea that often results in vomiting.

To correct this situation, Leslie advocated an operation to shorten and tighten the soft palate on healing after the removal of the uvula, This radical solution has not been widely accepted or used.

Other useful tips to prevent gag reflex

Use of a rubber dam is advocated.The basis of this is the physical blocking of anything getting close enough to trigger points in the mouth.But in patients with psychological basis to gag reflex,rubber dam is difficult to tolerate.

Use of roofless denture,matt finish denture,training bases,modification of edentulous maxillary custom tray by attaching a disposable saliva ejector to the base plate wax in the midline of the tray can be used to reduce gagging.

While taking maxillary impressions,posterior part of the tray should be seated first to direct any excess material to flow anteriorly and not towards the soft palate. The tray should not be overloaded and use fast setting impression material .[11]

Ear plugs can act as an external auditory meatus stimulator and suppress gagging reflex.

Closed mouth ID blocks may prove a successsful alternative in patients with pronounced gag reflex.

CONCLUSION

Effective management of the gag reflex is essential for providing quality dental care and ensuring patient comfort. A thorough understanding of its etiology enables clinicians to select the most appropriate management strategy. Combining behavioral techniques, desensitization methods, pharmacological aids, and modified clinical approaches can significantly reduce gagging episodes. Ultimately, a calm, empathetic attitude and good communication with the patient remain key to achieving successful treatment outcomes and maintaining trust in the dental setting.

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THE ORAL-SYSTEMIC AXIS: PERIODONTAL DISEASE AS A DRIVER OF CHRONIC SYSTEMIC INFLAMMATION: A REVIEW

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ABSTRACT

Periodontitis (PD), a chronic inflammatory disease initiated by a dysbiotic microbial biofilm, is no longer considered a localized oral condition. Mounting epidemiological, mechanistic, and intervention data robustly confirm its bidirectional relationship with numerous systemic diseases, profoundly impacting overall public health. This review critically synthesizes the latest evidence establishing periodontitis as a significant source of systemic inflammation, focusing on its core associations with Diabetes Mellitus (DM), Cardiovascular Disease, and emerging links with Neurodegenerative Disorders (NDs). We detail the central mechanistic pathways-systemic inflammation, microbial translocation, and immune cross-reactivity-that underpin this oral-systemic connection. Ultimately, we propose that integrating periodontal care into chronic disease management strategies is an imperative for improving systemic health outcomes and mitigating the global burden of non-communicable diseases.

K E Y W O R D S: Periodontitis, Systemic Diseases, Inflammation

INTRODUCTION

Periodontitis is a long-lasting, multifactorial inflammatory condition that begins with the buildup of a dysbiotic biofilm on the surfaces of teeth, triggering an immune-inflammatory response from the host[1]. Periodontitis affects a significant portion of the global population, with mild forms impacting about 62% and severe forms affecting roughly 23.6%, ranking it as the seventh most common human disease , Figure 1 represents global prevalence of periodontitis [2 ,3].

A systemic disease is defined as a condition that impacts multiple organs and systems throughout the body[4]. In 1900, British physician William Hunter was the first to propose that oral microorganisms could be linked to various systemic diseases. This theory was widely accepted in 1900-1940 . This theory was later disproven when treatments such as tooth extractions failed to improve systemic health (Journal of the American Medical Association, 1952) [5,6,7].

A detailed case history should be recorded to identify the etiology of gagging.The management of gagging patient depends upon the severity and etiology of the gag reflux. The clinician should be calm,patient and always be willing to hear the problems of the treating patient,as attitude of the dentist will directly influence the outcome of the treatment.

Mild to moderate gagging problems can be effectively managed in the clinic itself,but in severe cases,patient should be refered to Hypnotherapist.The main objective of a dentist is to free the anxiety of the patient ,gain confidence in him and make him /her comfortable with our clinic and staffs. Prior to every procedure,patient should be informed about it.The prime duty of a clinician is to render the treatment to the patient .[6]

Various methods of management of gagging reflux are pharmacological treatment,behaviour modifications ,TENS,acupuncture,acupressure and surgical techniques .These methods can be employed either alone or in combination to overcome exaggerated gagging reflux.[7,8]

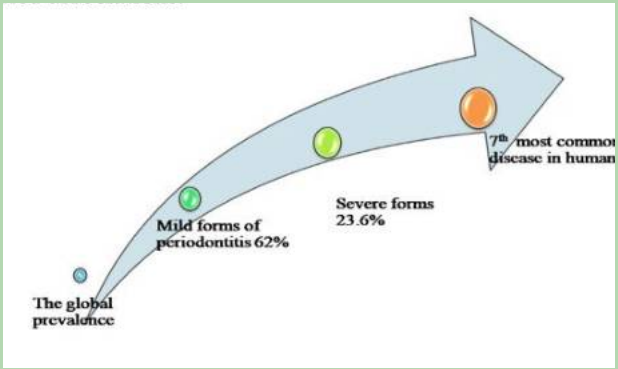

Figure 1. Global prevalence of Periodontitis [2]

2. CHRONIC INFLAMMATION: THE SHARED MECHANISM LINKING PERIODONTITIS TO SYSTEMIC DISEASES

The systemic impact of periodontitis is primarily mediated by three interconnected biological pathways [3, 11, 12]

2.1. Systemic Inflammatory Burden

Periodontal lesions act as a chronic, non-healing source of inflammation. Pro-inflammatory cytokines (e.g., TNF α, IL-1β, and IL-6) and acute-phase proteins (C-reactive protein (CRP)) produced locally in the periodontium enter the bloodstream at a measurable rate. Elevated systemic levels of these markers contribute to endothelial dysfunction, insulin resistance, and a pro-thrombotic state, which are central to various systemic diseases [3, 13].

2.2. Microbial Translocation (Bacteremia)

Periodontitis results in the ulceration of the pocket epithelium, providing a gateway for periodontal pathogens and their virulence factors (e.g., P. gingivalis lipopolysaccharide and gingipains) to enter the circulation during daily activities like chewing and brushing. These microbes and their components can disseminate and colonize distant sites, including atheromatous plaques and cerebral tissue [3, 14].

2.3. Immune Cross-Reactivity and Autoimmunity

A key hypothesis involves molecular mimicry.

P. gingivalis produces enzymes (gingipains) that can modify host proteins through a process called citrullination. In genetically susceptible individuals, these modified proteins can trigger an autoimmune response. This mechanism is strongly implicated in conditions like Rheumatoid Arthritis (RA), where anti-citrullinated protein antibodies (ACPAs) are a hallmark [3].

3. Periodontitis and Diabetes Mellitus

The connection between periodontitis and type 2 diabetes is bidirectional and complex. In people with periodontitis, bacteria entering the bloodstream, vascular inflammation, systemic oxidative stress, and chronic inflammation can impair insulin-producing beta cells and worsen blood sugar control. Conversely, in individuals with diabetes-especially those with poor glycemic control-high blood sugar contributes to increased inflammation, impaired immune function, and delayed wound healing in the gums, leading to more severe periodontal disease [15].

Poorly controlled diabetes significantly raises the risk and severity of periodontitis compared to those with well-controlled or no diabetes. On the other hand, severe periodontitis is linked to higher HbA1c levels, even in individuals without diagnosed diabetes, and increases the risk of developing prediabetes or diabetes. Table 1 shows the mechanism of birdirectional relationship between periodontitis and diabetes. The severity of periodontitis also correlates with diabetes-related complications, such as retinopathy, nephropathy, neuropathic foot ulcers, cardiovascular diseases, and increased mortality [13].

Some of the recent reports establishing the connections between DM and periodontitis can be seen in table 2.

4. Periodontitis and Cardiovascular Diseases

Periodontitis is now recognized as an independent risk factor for myocardial infarction (MI) and stroke. The association is consistent across large-cohort and case-control studies [19, 20]. The link between periodontitis and cardiovascular diseases (CVDs) is supported by evidence showing that periodontal bacteria can enter the bloodstream (bacteremia), triggering systemic inflammation(e.g., P. gingivalis DNA detected in atherosclerotic plaques) and accelerates the formation and destabilization of atherosclerotic lesions. Elevated inflammation due to periodontal lesions further contributes to this connection. Additionally, both conditions share common genetic and environmental risk factors, such as tobacco smoking [13]. Figure 2 shows the association between periodontal disease Cardiovascular Diseases and stroke. A very recent line of inquiry suggests a strong link between severe periodontitis and an elevated risk of cryptogenic ischemic stroke, particularly in younger adults without traditional risk factors[21]

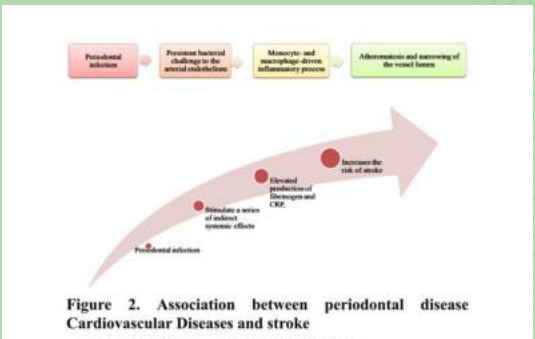

Figure 2. Association between periodontal disease Cardiovascular Diseases and stroke

Direction of Effect	Latest Findings and Mechanism	Clinical and Systemic Impact
DM to PD	Molecular & Inflammatory Amplification 1. Advanced Glycation End-products (AGEs): Hyperglycemia leads to AGE accumulation, which binds to RAGE receptors, hyper-amplifying inflammation and increasing oxidative stress in the periodontal tissues. 2. Immune Dysfunction: Impaired neutrophil function compromises the body's ability to clear oral bacteria, allowing the infection to flourish. [16].	Increased prevalence and severity of periodontitis, with greater attachment and bone loss, especially in poorly controlled DM (high HbA1c) [17].
PD to DM	Systemic Inflammation & Insulin Resistance 1. Cytokine Spillover: Chronic periodontal inflammation releases high levels of pro-inflammatory cytokines (e.g., TNF-α, IL-6) into the systemic circulation. 2. Promoting Insulin Resistance: These systemic cytokines interfere with the body's use of insulin, promoting insulin resistance in muscle and liver tissues[15,18]	Worsened Glycemic Control: Significant elevation of HbA1c (worsening of blood sugar control) in diabetic patients. Also linked to an increased incidence and risk for the development of pre-diabetes and Type 2 DM in non-diabetic individuals [16].

Table 1 Bidirectional relationship between periodontitis and diabetes.

Ying-Ying Wu et al	2015	Periodontal disorders and diabetes mellitus share characteristics with other chronic illnesses. Osteoclasts and osteoblasts are likely to be affected by inflammation in severe periodontitis that causes alveolar bone loss.
WencheS.Borgnakke	2019	Diabetes (dysglycemia) and oral health adversely affect each other in a vicious cycle.
Philip M. Preshaw1 and Susan M. Bissett	2020	Periodontitis is more common in people with diabetes, and the degree of glycaemic management is crucial in defining the risk. There is a two-way relationship between the two illnesses, with each disease having negative impacts on the other because to increased inflammation.
F.Barutta et al	2022	Anti-diabetic medications may potentially have pleiotropic effects in addition to decreasing blood sugar levels, which may be relevant in the context of periodontitis.
Simona Santonocitoet al	2022	The risk of developing kidney, heart, or other complications is significantly increased in people with periodontal disease and diabetes and can be reduced by lowering periodontal inflammation, which is brought on by both surgical and non-surgical periodontal therapy.
GrigoriosPlemmenos et al	2022	The simultaneous control of DM and AGE levels and the removal of dental plaque may lessen periodontal destruction.
Yuan su et al	2023	Periodontitis-derived virulence factors are involved in the pathological mechanism underlying Type2 Diabetes Mellitus
Alahmari, M. M et al	2023	Provides proof for the common occurence of periodontitis in DM patients.
Dhingra, K. etal	2023	Periodontitis treatment by subgingival instrumentation improves glycaemic control in diabetic patients
Thakkar-Samtani et al	2023	Undergoing periodontal treatment was associated with significant reductions in overall health care costs for patients with DM.
Ranbhise et al	2025	Periodontal therapy in DM patients has been shown to reduce systemic inflammatory markers and can lead to a clinically meaningful decrease in HbA1c often up to 0.4 in systematic reviews .

Table 2: Brief summary of recent reports revealing the connections between DM and periodontitis arranged chronologically.

5. Periodontitis and Respiratory diseases

Chronic Obstructive Pulmonary Disease (COPD) is marked by airflow obstruction caused by conditions like chronic bronchitis or emphysema. It involves the enlargement of bronchial mucus glands and an inflammatory process where neutrophils and mononuclear inflammatory cells accumulate in the lungs. COPD shares similar inflammatory mechanisms with periodontal disease.. This leads to an influx of neutrophils that release enzymes causing tissue damage. Additionally, monocytes and macrophages are recruited, leading to further inflammatory mediator release.

The connection between periodontal disease and COPD is less understood compared to other conditions like coronary heart disease. Some studies show that patients with COPD have deeper probing depths and more clinical attachment loss than those without COPD. Those with poor oral hygiene are at a higher risk for chronic respiratory conditions like emphysema and bronchitis. However, one large epidemiological study found no significant association between periodontal disease and COPD overall. It did show that severe periodontitis was linked to a higher risk of COPD in current smokers, suggesting that smoking may amplify the connection between the two diseases. A systematic review of 14 studies found a twofold increased risk of COPD in those with periodontal disease, though more research is needed to establish a clear relationship.. Studies have shown that scaling and root planing (a periodontal treatment) can reduce the frequency of COPD exacerbations compared to no treatment.

The oral cavity can harbor harmful organisms that may enter the respiratory system, contributing to respiratory infections. Though pneumonia is usually caused by bacteria like Streptococcus pneumoniae and Haemophilus influenzae, hospital-acquired pneumonia, or nosocomial pneumonia, is more severe and caused by different organisms, including anaerobic bacteria. These bacteria can originate from the oral cavity, particularly from subgingival biofilms.Hospitalized patients, especially those on ventilators or in intensive care units, are at high risk for nosocomial pneumonia. These infections often arise from the aspiration of oropharyngeal contents colonized by potential respiratory pathogens (PRPs), which are more common in prolonged hospital stays. Poor oral hygiene in these settings increases the likelihood of PRP colonization and pneumonia. The link between periodontal disease and COPD is still being explored, there is clear evidence that poor oral health can exacerbate respiratory diseases, especially in smokers and hospitalized patients. Maintaining oral hygiene could play a critical role in reducing the risk of both COPD and respiratory infections, particularly pneumonia. [12,22,23,24]

6. Periodontitis and cancers

Growing evidence indicates a strong link between periodontal disease and the development of several types of cancer. The cancer-causing potential of various periodontal pathogens has been confirmed both in vitro and in vivo. Cancer is a complex, multi-step disease influenced by genetic and environmental factors.

Periodontal pathogens contribute to chronic inflammation, immune response, cell invasion and growth, anti-apoptotic activity, and the presence of carcinogenic substances, all of which can lead to cancer. While linking periodontal pathogens directly to cancer development is challenging, certain pathogens like P. gingivalis, F. nucleatum, A. actinomycetemcomitans, T. forsythia, and T. denticola are known to activate signaling pathways that may play a role in cancer [Figure3]. Further research into how these pathogens influence cancer development could reveal critical mechanisms, offering new opportunities for diagnostic, preventive, and therapeutic strategies, ultimately improving treatment effectiveness and survival rates[25 ,26]. Figure 4 shows the causal role of pathogenic bacteria and microbial dysbiosis in cancer and the host.

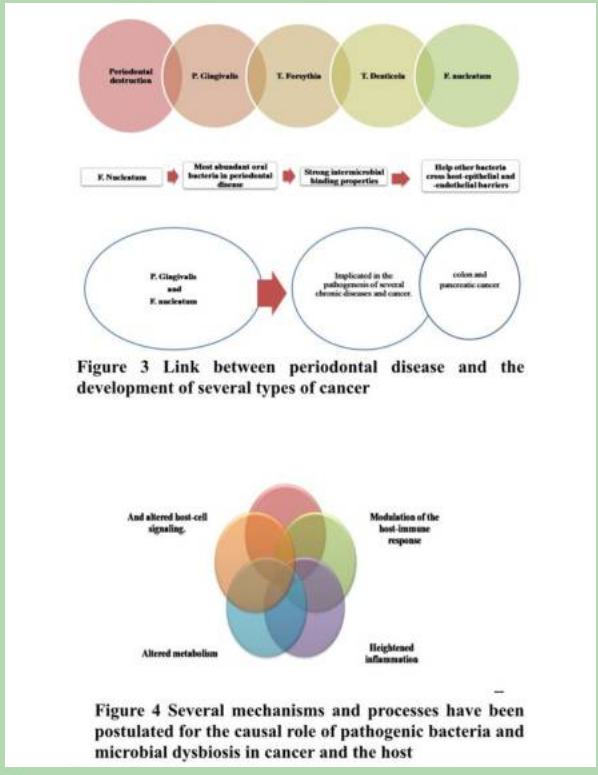

Figure 3 Link between periodontal disease and the development of several types of cancer

Figure 4 Several mechanisms and processes have been postulated for the causal role of pathogenic bacteria and microbial dysbiosis in cancer and the host

Periodontitis and Adverse Pregnancy Outcomes

Low birth weight (LBW) infants face significantly higher risks of neonatal death and long-term health complications, with preterm labor and premature rupture of membranes (PROM) being primary causes. While known risk factors like smoking, infections, and poor prenatal care explain many cases, others remain unexplained. Maternal infections, especially subclinical ones such as bacterial vaginosis, are linked to preterm birth and LBW through ascending or hematogenous spread of bacteria like Fusobacterium nucleatum, often originating from the oral cavity [27]. Periodontitis, a chronic oral infection, has been associated with adverse pregnancy outcomes due to systemic inflammation and bacterial translocation. Animal studies and human research suggest that oral pathogens can trigger elevated inflammatory mediators like TNF-α and PGE_2, contributing to fetal growth restriction, preterm labor, and even fetal death. Numerous studies and meta-analyses show a significant link between maternal periodontitis and risks of preterm birth, LBW, and preeclampsia, though not all findings are consistent. Some intervention trials show that treating periodontitis during pregnancy can reduce these risks, especially in high-risk populations, while others find limited impact. However, all agree that scaling and root planing during the second and third trimesters are safe and do not harm pregnancy outcomes, supporting their use as part of prenatal care [28]

Kidney function declines when the kidneys cannot filter water properly, and individuals with chronic kidney disease should pay close attention to oral hygiene, as oral pathogens contribute to periodontitis. Cognitive impairment, often associated with dementia or Alzheimer's, can be exacerbated by poor oral care, as periodontitis and cognitive decline are linked to a lack of patient awareness. Obesity, a marker of an unhealthy lifestyle, also increases the risk of periodontitis, as excess body fat is associated with poor oral health. Metabolic syndrome, which includes hypertension, hyperglycemia, and abdominal obesity, also correlates with periodontitis. Pathogenic bacteria in the oral cavity contribute to the development of periodontitis, highlighting the need for improved oral health to reduce these risks [29, 30].

9. Effect of Periodontal Treatment on Systemic Inflammation

Periodontal treatment for optimal outcomes improves diabetes outcomes and surrogate measures of cardiovascular risk [13]. The established oral-systemic links mandate a paradigm shift towards integrated healthcare. Effective treatment of periodontitis offers a tangible, modifiable target for reducing systemic inflammatory burden.

9.1 Impact of Periodontal Therapy:

Studies consistently show that non-surgical and surgical periodontal therapy significantly reduces systemic inflammatory biomarkers like CRP and IL-6 [4]. This reduction has been correlated with improved surrogate markers for systemic health, such as enhanced endothelial function and modest reductions in HbA1c levels in diabetic patients [15].

9.2 Interprofessional Collaboration:

The latest consensus guidelines strongly advocate for mandatory screening for DM in periodontitis patients and vice versa. Collaboration between dentists, cardiologists, diabetologists, and neurologists is crucial for developing cohesive and comprehensive patient management plans [3, 11,].

10. Conclusions and Future Outlook

Periodontal disease has been associated with an increased risk of several systemic conditions, including atherosclerotic cardiovascular disease, metabolic syndrome, diabetes, and respiratory disorders. Periodontitis can trigger systemic effects, such as heightened inflammation and a prothrombotic state, which are also found in conditions like rheumatoid arthritis, inflammatory bowel disease, and chronic obstructive pulmonary disease (COPD). These effects contribute to a higher likelihood of developing additional health complications and can significantly impact overall quality of life. Unlike factors such as age or genetics, periodontal disease is a modifiable risk factor. While research into how periodontal infections may influence other health conditions is ongoing, more studies are needed to fully understand its role in diseases like diabetes, heart disease, stroke, and respiratory issues. Periodontal medicine is advancing our understanding of the connection between oral health and systemic well-being.It's important to distinguish between scientific research and clinical practice. Research focuses on averages and statistical trends, such as the relationship between periodontal disease and poor blood sugar control in individuals with diabetes.

However, in clinical practice, we recognize that each patient is unique, and what holds true on average may not apply to every individual. Therefore, we tailor care to meet the specific needs of each patient to ensure the best outcomes.

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A CASE OF COMPOUND ODONTOME ASSOCIATED WITH AN IMPACTED MAXILLARY CENTRAL INCISOR IN A 14-YEAR-OLD GIRL

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ABSTRACT

Odontomas are the most common odontogenic tumors of developmental origin, frequently identified as incidental findings on radiographs. They are hamartomatous lesions comprising enamel, dentin, cementum, and pulp arranged in either an organized (compound) or disorganized (complex) pattern (1). This case report presents a 14-year-old female patient with a retained primary maxillary central incisor and a radiopaque mass in the anterior maxilla. Surgical removal of the odontome and exposure of the impacted permanent central incisor, followed by orthodontic alignment, resulted in successful eruption and restoration of esthetics and function.

Keywords: Odontome, Impacted tooth, Compound odontoma, Odontogenic tumor, Central incisor impaction

INTRODUCTION

Odontomas are benign odontogenic tumors or, more precisely, hamartomas composed of enamel, dentin, cementum, and pulp (2). They account for approximately 22% of all odontogenic tumors and are most frequently detected in the first two decades of life (3). According to the World Health Organization (WHO) classification (4), odontomas are categorized into two main types:

Compound odontomas: Composed of multiple small, tooth-like structures.

Complex odontomas: Irregular radiopaque masses representing a disorganized arrangement of dental tissues.

Odontomas are commonly associated with delayed eruption or impaction of teeth, especially in the anterior maxilla, where esthetics are of prime concern (2, 3).

Early radiographic evaluation and timely management are essential to prevent complications such as cystic changes, displacement of adjacent teeth, or malocclusion (1, 5).

CASE REPORT

14-year-old female reported to the dental clinic with a chief complaint of spacing in the upper front tooth region. The patient's medical and family history was non-contributory. On intraoral examination it was revealed that there was retention of the primary maxillary left central incisor (tooth 61) and absence of eruption of the permanent successor (tooth 21). The overlying mucosa appeared normal with no associated swelling or tenderness.

An orthopantomogram (OPG) revealed a well-defined radiopaque mass in the region of tooth 21, with the impacted permanent incisor positioned superiorly and slightly palatally. Cone-beam computed tomography (CBCT) confirmed the presence of multiple tooth-like radiopaque structures obstructing the eruption path of 21, consistent with a compound odontome (3, 4). The treatment plan was outlined to the patient. Following patient's consent, under local anesthesia, the odontome was surgically removed, and the impacted permanent incisor was surgically exposed. Orthodontic traction was initiated to guide the tooth into proper alignment within the dental arch. The postoperative course was uneventful, and satisfactory esthetic and functional outcomes were achieved.

Figure 2: Surgical site after removal of lesion and exposure of permanent left central incisor 21

Figure 3: Bracket placed on 21 for orthodontic traction for the eruption of the tooth

Figure 4: Excised calcified mass (odontome)

Figure 5: Flap repositioned and sutured

DISCUSSION

Odontomas are developmental anomalies (hamartomas) rather than true neoplasms (1).

The etiology is not fully established but may include local trauma, infection, hereditary influences, or genetic mutations affecting odontogenesis (5). Compound odontomas are most commonly located in the anterior maxilla, while complex odontomas tend to occur in the posterior mandible (4). Clinically, odontomas are usually asymptomatic and often discovered incidentally during investigations for unerupted or missing teeth (2).

Radiographically, compound odontomas appear as multiple miniature tooth-like structures surrounded by a radiolucent rim, whereas complex odontomas appear as a dense radiopaque mass with an irregular outline (3, 4). If left untreated, odontomas can cause eruption disturbances, root resorption, displacement of adjacent teeth, and occasionally cyst formation (1, 5). Early diagnosis and management are therefore critical to avoid long-term complications.

The present case is typical in its presentation—a young female patient with a retained deciduous incisor, a radiopaque mass in the anterior maxilla, and delayed eruption of the permanent central incisor. Surgical removal followed by orthodontic traction yielded a successful functional and esthetic result, corroborating outcomes reported in similar cases (2, 3, 5).

CONCLUSION

Odontomas, though benign, can significantly interfere with normal eruption patterns of permanent teeth, particularly in esthetically sensitive regions such as the anterior maxilla.

A multidisciplinary approach involving surgical removal, orthodontic alignment, and radiographic follow-up ensures predictable and satisfactory outcomes. Early detection and coordinated management remain the cornerstone of successful treatment.

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LASERS IN PERIODONTICS: SHEDDING LIGHT ON EVIDENCE, APPLICATIONS, AND MYTHS"- A NARRATIVE REVIEW

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ABSTRACT

Lasers have been advocated as adjuncts and alternatives to conventional periodontal therapy for over three decades. They promise bacterial reduction, improved healing, minimally invasive surgery, and patient comfort. However, the literature shows mixed results: some laser types and protocols yield modest short-term clinical benefits while others show no advantage over scaling and root planing (SRP) alone. This narrative review summarizes laser types used in periodontics (diode, Nd:YAG, Er:YAG/Er,Cr:YSGG, CO_2, low-level lasers), their mechanisms, clinical applications (nonsurgical pocket therapy, surgical periodontal therapy, peri-implantitis management, biostimulation), safety considerations, and recommendations for practice. We critically appraise randomized trials, systematic reviews, and consensus statements to provide an evidence-based, practitioner-focused synthesis. Key messages: (1) lasers are not a universal substitute for meticulous mechanical debridement; (2) adjunctive use may offer small improvements in some outcomes (BOP, short-term PD reduction) depending on laser type and protocol; (3) standardized protocols and long-term RCTs are still lacking; and (4) clinicians should weigh cost, training, and patient expectations when adopting laser therapy. Practical clinical protocols and research priorities are proposed.

Keywords
Lasers, periodontal therapy, diode laser, Er:YAG, Nd:YAG, scaling and root planning, adjunctive therapy, periodontal surgery

INTRODUCTION

Periodontitis is one of the most prevalent chronic inflammatory diseases of humans, characterized by the progressive destruction of the supporting structures of the teeth, ultimately leading to tooth loss if untreated (1).

Conventional non-surgical periodontal therapy, primarily scaling and root planing (SRP), has long been regarded as the cornerstone of periodontal management, aiming to eliminate bacterial deposits and create a biologically acceptable root surface (2).

In recent decades, advances in dental technology have introduced laser-assisted therapy as a potential adjunct or alternative to conventional treatment. Lasers are proposed to offer enhanced debridement, superior bacterial reduction, improved hemostasis, and accelerated wound healing (3,4).

Since their first application in dentistry in the 1960s, lasers have evolved from bulky and complex equipment to compact, clinician-friendly tools (5). Various wavelengths and laser systems — including Diode, Nd:YAG, Er:YAG, Er,Cr:YSGG, and CO_2 lasers — are now used in both non-surgical and surgical periodontal therapy (6).

However, despite their clinical appeal and increasing use, the literature remains divided on their true efficacy. Some randomized controlled trials demonstrate equivalent or marginally better outcomes compared with SRP, while others report no significant advantage (7,8).

This ongoing debate underscores the need for a comprehensive review of the current evidence, clarifying the realistic benefits, limitations, and common misconceptions surrounding laser use in periodontics.

HISTORY AND EVOLUTION OF LASERS IN DENTISTRY

The term LASER is an acronym for Light Amplification by Stimulated Emission of Radiation. The first working laser was developed by Theodore Maiman in 1960 using a ruby crystal (9). Within a few years, Goldman and colleagues pioneered its use on dental hard tissues, exploring its potential for caries removal and enamel surface modification (10).

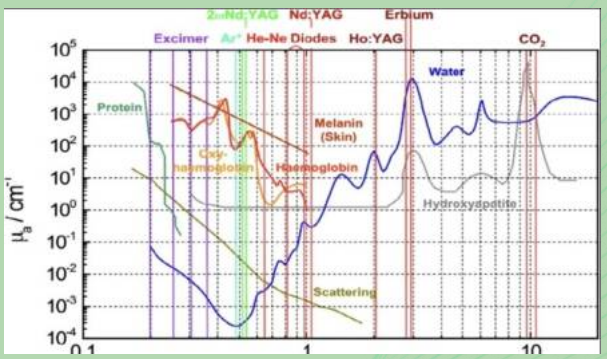
By the 1980s, specific laser wavelengths suitable for soft-tissue surgery, such as CO_2 and Nd:YAG lasers, were introduced, providing improved precision and hemostasis. Later, Er:YAG and Er,Cr:YSGG lasers were developed, offering selective ablation of both hard and soft tissues with minimal thermal damage (11).

In periodontics, laser use expanded from soft-tissue surgery and sulcular debridement to periodontal pocket disinfection, calculus removal, photobiomodulation, and regenerative applications (12).

Technological advancements have also made lasers portable, ergonomic, and cost-efficient, increasing their adoption in private practice settings (13).

CLASSIFICATION AND TYPES OF LASERS USED IN PERIODONTICS

Lasers are classified based on wavelength, active medium, and clinical application (14). The wavelength determines the laser's absorption characteristics in biological tissues such as water, hydroxyapatite, and hemoglobin (15).



3.1 Based on Wavelength and Active Medium

Laser Type	Active Medium	Wavelength (nm)	Tissue Target	Typical Applications
Diode Laser	Semiconductor (GaAs, GaAlAs)	810–980	Pigment, hemoglobin	Pocket disinfection, depigmentation, LLLT
Nd:YAG	Neodymium-doped Yttrium Aluminum Garnet	1064	Pigment, hemoglobin	Subgingival curettage, sulcular debridement
Er:YAG	Erbium-doped Yttrium Aluminum Garnet	2940	Water, hydroxyapatite	Root debridement, calculus removal
Er,Cr:YSGG	Erbium, Chromium-doped Yttrium Scandium Gallium Garnet	2780	Water, hydroxyapatite	Bone and soft-tissue surgery
CO_2 Laser	Carbon dioxide	10,600	Water	Gingivectomy, frenectomy, mucosal surgery

(Table adapted from Cobb 2010; Romanos 2013) (16,17)

Each laser type interacts differently with the target tissue, leading to varied clinical outcomes and thermal effects. For instance, diode and Nd:YAG lasers primarily target pigmented tissues, while Er:YAG and Er,Cr:YSGG lasers efficiently ablate mineralized tissue with minimal collateral damage (18).

LASER–TISSUE INTERACTION AND MECHANISMS OF ACTION

The biological effect of a laser on tissue depends primarily on the wavelength, power output, duration of exposure, and optical properties of the target substrate (19).

When laser energy contacts tissue, four major interactions may occur: reflection, transmission, scattering, and absorption (20). The absorbed energy leads to a variety of effects depending on the tissue composition—mainly water, hydroxyapatite, and chromophores such as hemoglobin and melanin (21).

4.1 Photothermal Effect

The photothermal mechanism is the most common in periodontal lasers. The absorbed light raises the local temperature, causing coagulation, vaporization, or carbonization of the target tissue (22). This effect is beneficial for hemostasis and soft-tissue ablation but must be controlled to avoid collateral thermal damage to underlying structures (23).

4.2 Photomechanical Effect

High-energy lasers such as Er:YAG and Er,Cr:YSGG create micro-explosions in water-rich tissues, enabling precise ablation with minimal heat accumulation. This photoacoustic effect is particularly useful for calculus removal and root surface debridement (24).

4.3 Photobiomodulation (PBM)

At low power densities, laser light can stimulate cellular activity—enhancing fibroblast proliferation, collagen synthesis, angiogenesis, and wound healing (25). This biostimulatory effect, also known as low-level laser therapy (LLLT), has significant potential in postoperative healing and pain reduction.

APPLICATIONS OF LASERS IN PERIODONTAL THERAPY

The clinical use of lasers in periodontology has evolved from simple soft-tissue ablation to complex regenerative procedures. Owing to their specific wavelengths and tissue-interaction characteristics, lasers can target pigments such as melanin and hemoglobin, vaporize water-containing tissues, and effectively reduce bacterial loads, thereby improving both surgical precision and patient outcomes (26).

The versatility of lasers has broadened their use across non-surgical, surgical, mucogingival, and peri-implant therapies, each with distinct clinical advantages (27).

1. Non-Surgical Periodontal Therapy

Scaling and root planing (SRP) remains the cornerstone of non-surgical periodontal treatment. However, SRP alone often fails to eradicate pathogenic bacteria within deep pockets or complex root anatomy. The adjunctive use of lasers, particularly diode and Nd:YAG lasers, enhances subgingival debridement by thermally disrupting bacterial cell walls and detoxifying root surfaces .

Cobb demonstrated that diode laser irradiation reduced Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans in periodontal pockets more effectively than SRP alone. In addition, laser energy stimulates fibroblast proliferation and collagen remodeling, improving epithelial reattachment and accelerating healing .

Clinical trials have reported that adjunctive diode or Er:YAG laser therapy achieves greater reductions in probing depth and gains in clinical attachment level compared with SRP alone, though results vary depending on parameters and operator skill

2. Surgical Periodontal Therapy

In periodontal flap surgery, lasers are utilized for incision, degranulation, and hemostasis. CO_2 and Er:YAG lasers exhibit strong absorption in water and hydroxyapatite, enabling efficient cutting with minimal collateral damage . The hemostatic and bactericidal properties of lasers ensure a blood-free operative field and reduce postoperative infection rates .

Romanos highlighted that laser-assisted flap surgery minimizes postoperative pain and edema while improving patient comfort compared with conventional scalpel or electrosurgical methods. Er,Cr:YSGG lasers can also perform selective osseous contouring and root conditioning without causing thermal necrosis.

Such precision and tissue preservation make lasers particularly useful in aesthetic crown lengthening, gingivectomy, and periodontal pocket reduction surgeries .

3. Laser-Assisted New Attachment Procedure (LANAP)

The Laser-Assisted New Attachment Procedure (LANAP) is a minimally invasive, FDA-approved laser protocol that facilitates periodontal regeneration without traditional flap reflection. It employs a free-running pulsed Nd:YAG laser (1064 nm) in a controlled sequence to selectively ablate diseased pocket epithelium while preserving underlying connective tissue .

The LANAP protocol comprises several critical steps:

1. Selective pocket de-epithelialization using short-pulse Nd:YAG irradiation.
2. Mechanical debridement with ultrasonic scalers to remove calculus.
3. Laser coagulation to achieve hemostasis and fibrin clot formation.
4. Clot stabilization to seal the pocket and create a regenerative environment (28).

Histologic studies by Yukna et al. (29) revealed true regeneration, with new cementum, periodontal ligament, and alveolar be formation following LANAP.

Nevertheless, the procedure remains operator-dependent, requiring extensive training and precise calibration. Systematic reviews emphasize that, while short-term outcomes are encouraging, more long-term randomized controlled trials are necessary to confirm its superiority over conventional therapy (31).

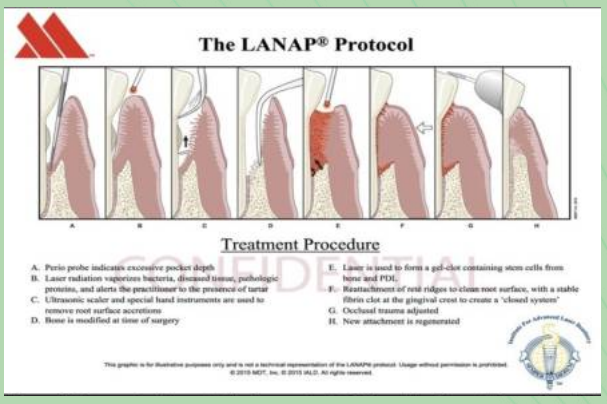


Figure X. Sequential steps in the Laser-Assisted New Attachment Procedure (LANAP) illustrating pocket debridement, degranulation, and fibrin clot formation (Source: San Diego Periodontics, 2024).

4. Mucogingival and Esthetic Applications

Lasers have proven highly effective in mucogingival surgeries such as Frenectomy, Gingivectomy, Depigmentation, and Esthetic crown lengthening. Their precision and excellent hemostasis minimize postoperative discomfort and eliminate the need for sutures (32).

Diode and CO_2 lasers are particularly suited for depigmentation because of their affinity for melanin, ensuring uniform results with minimal recurrence (33). In esthetic procedures, diode lasers allow precise sculpting of soft-tissue margins, yielding predictable contouring and rapid recovery (34).

5. Management of Peri-Implant Diseases

The management of Peri-implant mucositis and Peri-Implantitis often involves mechanical debridement and antimicrobial therapy, but laser use has shown promising adjunctive effects. Er:YAG lasers effectively remove biofilm and granulation tissue from implant surfaces without altering titanium morphology (35).

Furthermore, photobiomodulation (LLLT) enhances peri-implant healing by stimulating fibroblast proliferation and neovascularization (36). Nd:YAG lasers can assist in implant surface decontamination and re-osseointegration when used under controlled energy settings (37).

6. Postoperative Healing and Photobiomodulation

Beyond their ablative role, lasers exert biostimulatory effects that accelerate wound healing, enhance collagen synthesis, and reduce postoperative pain (38). LLLT operating between 600 and 900 nm increases ATP production and modulates inflammatory mediators, resulting in faster tissue repair.

These advantages make lasers a valuable adjunct in modern minimally invasive periodontal therapy, optimizing both clinical efficacy and patient satisfaction.

LOW-LEVEL LASER THERAPY (PHOTOBIOMODULATION) IN PERIODONTICS

Low-level laser therapy (LLLT), also known as photobiomodulation, uses light energy at sub-ablative doses to stimulate biological processes rather than destroy tissue (39).

Wavelengths in the range of 600–1000 nm are most effective, as they penetrate deeper into tissues and interact with mitochondrial chromophores such as cytochrome-c oxidase (40).

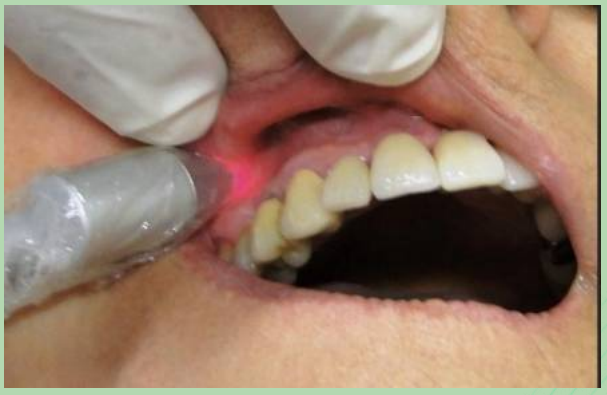
6.1 Mechanism

The absorbed photons enhance ATP production, reactive oxygen species signaling, and gene transcription, leading to improved cell metabolism, angiogenesis, and collagen remodeling (41).

6.2 Clinical Applications

In periodontics, LLLT is used to:

- Reduce postoperative pain and inflammation after surgery (42).
- Promote healing of mucogingival wounds and graft donor sites (43).
- Stimulate bone regeneration in intrabony defects (44).
- Improve healing after implant placement (45).



A recent clinical trial by Ribeiro et al. (2022) reported significant reduction in postoperative discomfort and enhanced soft-tissue healing following flap surgery with adjunctive LLLT (46).

CLINICAL EFFICACY AND EVIDENCE-BASED OUTCOMES

The effectiveness of laser therapy in periodontics remains a subject of debate.

A systematic review by Schwarz et al. (2015) concluded that Er:YAG and Nd:YAG lasers offer comparable clinical improvements to conventional SRP in probing depth reduction and attachment gain, but do not show statistically superior long-term outcomes (47).

Conversely, studies employing laser-assisted new attachment procedures (LANAP) have reported enhanced clinical attachment level (CAL) gains and reduced probing depth over 6–12 months (48). However, these studies often suffer from small sample sizes or lack of standardized parameters (49).

Meta-analyses also reveal that while bacterial reduction and patient comfort are significantly improved, attachment gain and bone regeneration remain inconsistent (50,51).

Therefore, lasers should be viewed as adjunctive tools rather than replacements for mechanical debridement.

DISCUSSION

The integration of laser technology into periodontal therapy marks a significant evolution in the way clinicians manage both soft and hard tissue conditions. Lasers offer precision, reduced patient discomfort, and better visibility, but their use must be grounded in biological understanding and evidence-based reasoning.

One of the key advantages observed in clinical practice is the ability of lasers to achieve hemostasis while performing delicate procedures. The photothermal coagulation effect seals small blood vessels and lymphatics, leading to a dry surgical field and less postoperative edema (52).

From the patient's perspective, this translates to shorter healing times and reduced analgesic requirements.

However, while lasers have enhanced surgical comfort and esthetic outcomes, their clinical superiority over mechanical debridement remains equivocal. Several well-designed studies have shown that SRP combined with diode or Er:YAG laser can reduce bacterial load and inflammation more effectively in the short term (47,48), yet attachment level gains and bone regeneration outcomes often mirror those achieved through traditional methods (49,50). This indicates that the laser should be viewed as an adjunct, not a replacement, in the periodontal toolkit.

A crucial factor influencing results is operator skill. The therapeutic window between effective ablation and thermal damage is narrow; improper settings may lead to surface melting, carbonization, or root sensitivity. Thus, clinical training and calibration are mandatory for safe and predictable outcomes (59).

It is also important to consider the economic and ethical implications. Laser devices represent a significant investment, and while patients may perceive laser treatment as more "advanced," clinicians must avoid overpromising outcomes. Ethical use demands that indications are chosen based on evidence rather than marketing appeal.

Despite limitations, the use of lasers has been revolutionary for procedures such as frenectomy, crown lengthening, gingival depigmentation, and peri-implant decontamination, where they offer precise and comfortable alternatives to conventional approaches.

The continuing development of hybrid and dual-wavelength systems may further enhance predictability and tissue selectivity.

MYTHS AND MISCONCEPTIONS IN LASER PERIODONTICS

Although lasers have gained popularity, several myths persist among clinicians and students alike.

1. Myth 1 – Lasers completely replace conventional scaling and root planning.
   - Reality: Lasers cannot remove all calculus deposits or create a smooth root surface comparable to mechanical instruments. They are best used as adjuncts, not substitutes.
2. Myth 2 – All lasers can be used on all tissues.
   - Reality: Each laser type interacts with tissue differently. For example, diode lasers are absorbed in pigmented tissues, while Er:YAG lasers target water and hydroxyapatite. Misuse can lead to tissue damage.
3. Myth 3 – Lasers guarantee faster bone regeneration.
   - Reality: Although bio stimulation may enhance healing, there is no consistent evidence that lasers alone induce true periodontal regeneration or new attachment formation.
4. Myth 4 – Higher power means better results.
   - Reality: Excessive energy increases the risk of thermal necrosis. Controlled, evidence-based parameters are essential for safe use.
5. Myth 5 – Lasers are painless and require no anesthesia.
   - Reality: While discomfort is reduced, certain deep or fibrotic tissues may still require local anesthesia for patient comfort.

FUTURE DIRECTIONS

The field of laser dentistry is rapidly evolving, with several promising avenues of research.

11.1 Integration with Regenerative Medicine

Future studies may combine laser irradiation with growth factors, stem cell therapy, or biomimetic scaffolds to achieve true periodontal regeneration. Early in-vitro findings suggest that PBM may stimulate osteogenic differentiation of mesenchymal stem cells (63).

11.2 Standardization of Protocols

One of the major limitations in current literature is the lack of standardized parameters. Establishing evidence-based clinical guidelines for power output, exposure time, and energy density is critical to ensure reproducibility and patient safety (64).

11.3 Advances in Laser Technology

The development of dual-wavelength systems, robotic-guided delivery, and AI-based energy modulation may enable more precise and tissue-specific therapy in the near future (65). Portable diode systems are also becoming more affordable, which may improve accessibility in general dental practice.

11.4 Long-Term Clinical Trials

More multicentric, randomized controlled studies with long-term follow-ups are required to assess true periodontal stability and cost-effectiveness. In India, where periodontal disease prevalence is high, such studies could help develop region-specific protocols (66).

CONCLUSION

Lasers have undoubtedly revolutionized the approach to periodontal therapy, providing clinicians with a minimally invasive, precise, and patient-friendly tool. When used judiciously, lasers can enhance treatment outcomes—particularly in terms of hemostasis, microbial reduction, and patient comfort.

However, the fundamentals of periodontal therapy remain rooted in mechanical debridement and biological healing principles. Lasers are valuable adjuncts, not replacements, and their benefits depend heavily on the clinician's understanding of physics, tissue response, and proper technique.

As research continues, the integration of lasers with biological and regenerative concepts promises to bring periodontal therapy closer to true tissue regeneration and improved patient experience. Until then, a balanced, evidence-driven approach remains the best practice.

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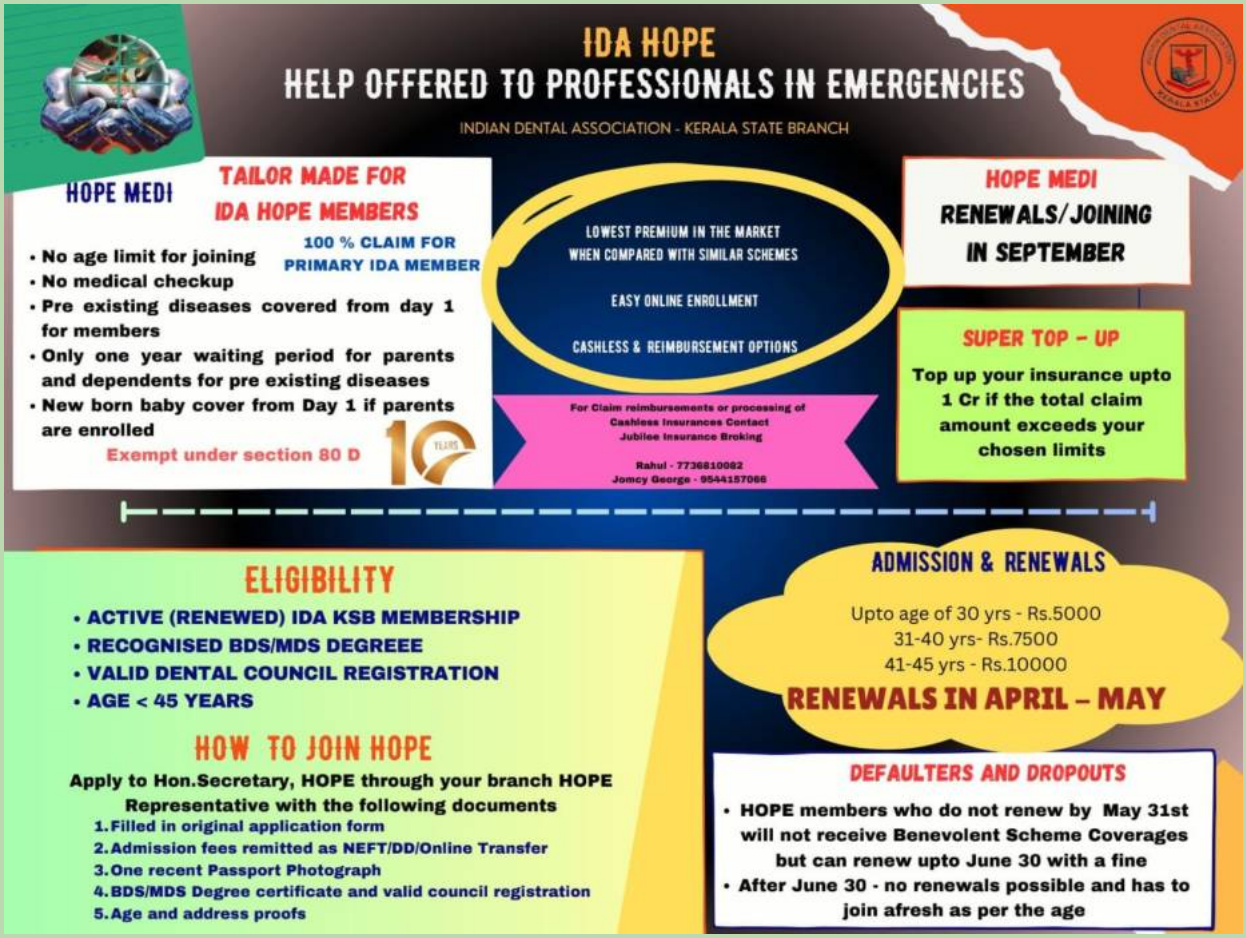
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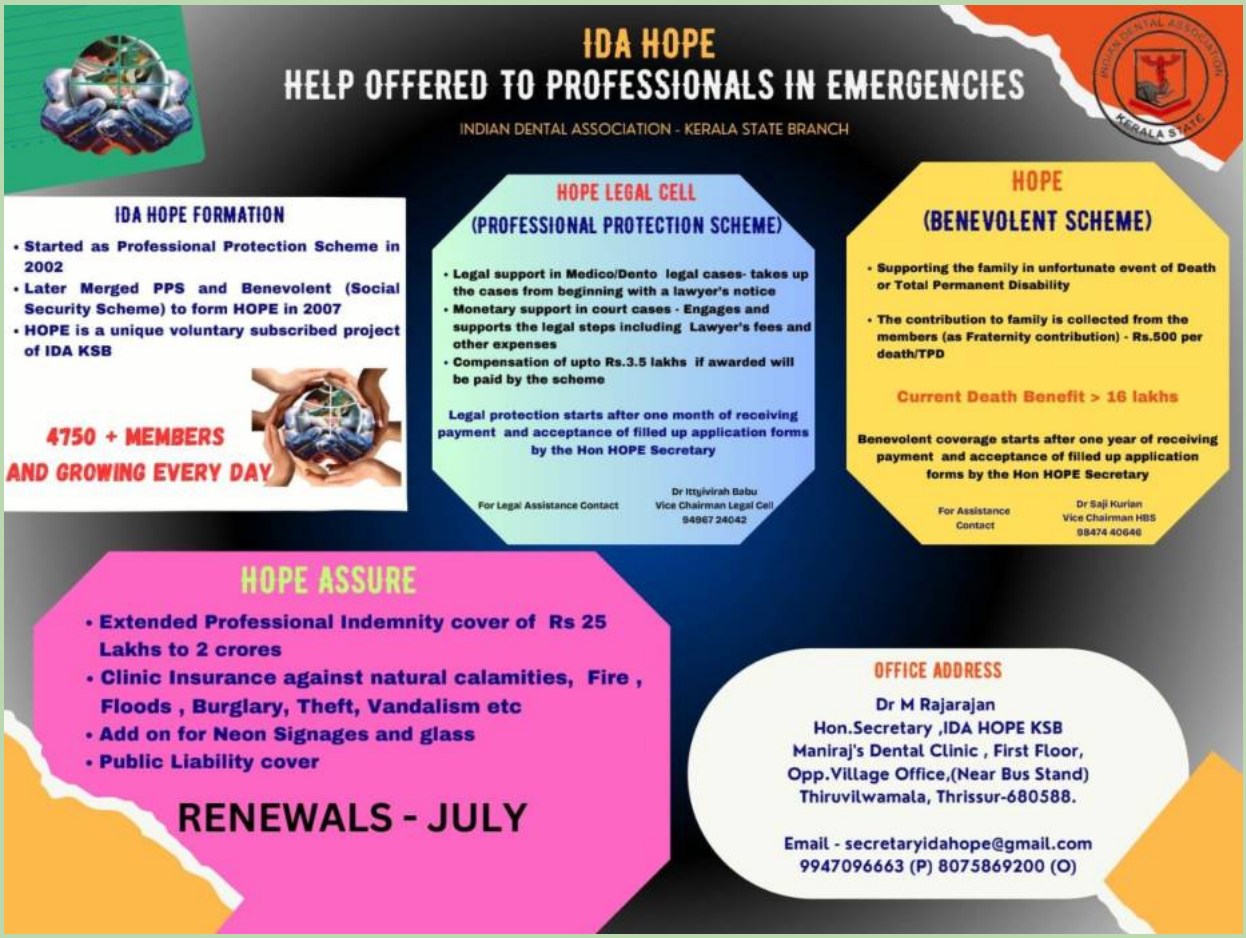
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• Monetary support in court cases - Engages and supports the legal steps including Lawyer's fees and other expenses
• Compensation of upto Rs.3.5 lakhs if awarded will be paid by the scheme
Legal protection starts after one month of receiving payment and acceptance of filled up application forms by the Hon HOPE Secretary
For Legal Assistance Contact
Dr Ittyivirah Babu
Vice Chairman Legal Cell
94967 24042
HOPE
(BENEVOLENT SCHEME)
• Supporting the family in unfortunate event of Death or Total Permanent Disability
• The contribution to family is collected from the members (as Fraternity contribution) - Rs.500 per death/TPD
Current Death Benefit > 16 lakhs
Benevolent coverage starts after one year of receiving payment and acceptance of filled up application forms by the Hon HOPE Secretary
For Assistance Contact
Dr Saji Kurian
Vice Chairman HBS
98474 40646
HOPE ASSURE
• Extended Professional Indemnity cover of Rs 25 Lakhs to 2 crores
• Clinic Insurance against natural calamities, Fire , Floods , Burglary, Theft, Vandalism etc
• Add on for Neon Signages and glass
• Public Liability cover
RENEWALS - JULY
OFFICE ADDRESS
Dr M Rajarajan
Hon.Secretary ,IDA HOPE KSB
Maniraj's Dental Clinic , First Floor,
Opp.Village Office,(Near Bus Stand)
Thiruvilwamala, Thrissur-680588.
Email - secretaryidahope@gmail.com
9947096663 (P) 8075869200 (O)

JOURNAL GUIDELINES

Manuscript type : accepted are i) research ii) case report iii) review iv) short study

Article should be typed in times new roman size 12 A4 size paper. Use 1.5 spacing through out with a significant margin. Authors are advised to retain a soft copy for the reference and a soft copy of the article has to be sent to the editors email

Ethical consideration: manuscript submitted for publication must comply with the following ethical consideration. Written informed consent must be obtained from the subject before their data included in the study. Any data from the patient must be submitted by hiding their identity. All research should be carried out with prior approval from institutional or national ethic committee and should be in accordance with Helsinki declaration of 1964. If animals are used for research, the authors must follow the institutional or national guidelines for the care of use of laboratory animals

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