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Dr. Sandeep Kumar
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Editorial



The Editor of the " UPDATES IN DENTISTRY" had the audacity to publish my views from dentistry. As I write this column, we are living under the shadow of the corona-virus pandemic. The morbidity and mortality statistics are truly frightening at this point and are supposed to get much worse. Amidst this pandemic, this journal continues its normal publication schedule, thanks to the production team they are doing right now in the face of this global crisis.

With the concern of infection control in health care settings, personal protective equipments (PPE) have been given to many individuals. American Dental Association advised all the dental practices to cease non-emergent in-person care to reduce the infection rate and started to rebuild stockpiles of PPE for health care providers.

Many people today enjoy excellent oral health and are keeping their natural teeth throughout their lives. But for some, caries are still the most prevalent chronic disease of childhood. Too many people mistakenly believe that they need to see a dentist only if they are in pain or something is wrong.

Dentistry promotes continuity of care that is comprehensive, convenient, cost effective and efficient. Their responsibilities include diagnosing of oral diseases and promoting oral health and its prevention. Even the routine procedures such as tooth extractions, preparing and placing fillings, carry potential risks of complications such as infection, temporary or even permanent nerve damage, prolonged bleeding, pain etc. Dentists can spot early warning signs in t he mouth that may indicate disease elsewhere in the body. Regular dental visits and care will help maintain and improve optimal health throughout their lifetimes.

With people around the world wondering what the future will hold after this pandemic, I remain confident that our profession will not only survive but thrive. My confidence is even deeper, with a passion for symmetry, perfection and beauty to unlock each patient's epitome of a perfect smile.

Going forward with the most rewarding thing, the patient's happiness and satisfaction and the stability of the results.

Dr. Sandeep Kumar
Editor in chief
Director Principal
Professor & Head
Department of Prosthodontics
Surendera Dental College & Research Institute
Sriganganagar

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FACTORS ASSOCIATED WITH EARLY CHILDHOOD CARIES AMONG PRESCHOOL CHILDREN IN SRI GANGANAGAR, RAJASTHAN: A CROSS-SECTIONAL ANALYSIS

Dr. Manu Batra, Dr Deeksha Gijwani

ABSTRACT

Objectives: To assess a proposed age-related pattern of early childhood caries (ECC) in preschool children in Sri Ganganagar, Rajasthan, and to investigate correlations with parental, child behavioral, and household-level factors.

Materials and Methods: A cross-sectional dataset was developed for the city of Sri Ganganagar for 720 children in age group of 2-5 years of preschool. The outcome was ECC, defined as the presence of one or more decaying, missing due to caries or filled primary teeth/surfaces in children below 72 months of age. The exposures were age, sex, parental education, household income, sweet snack/drink frequency, night-time bottle feeding, toothbrushing frequency, maternal dental check-up attendance, sibling caries, weaning/breastfeeding beyond 24 months and pet contact. clinical recording according to the WHO oral health survey principles. Associations were investigated using chi-square testing and multivariable logistic regression analysis.

Results: Overall, the simulated ECC prevalence was 32.1% (231/720). The prevalence increased from 23.1% at 2 years to 43.7% at 5 years. In the multivariable analysis, age 5 years, frequent sugary snacks/drinks, toothbrushing less than once daily, night-time bottle feeding, poorer household income, lower maternal education, and sibling caries were associated with greater adjusted chances of ECC.

Conclusions: The proposed Sri Ganganagar pattern indicates the age-related increase in ECC and emphasizes modifiable family-level behaviors, particularly sugar exposure and frequency of brushing. The findings are suggestive and need to be confirmed by carrying out an actual school/Anganwadi-based epidemiological survey.

KEYWORDS

Early childhood caries; preschool children; Sri Ganganagar; Rajasthan; oral epidemiology; dental caries; simulated data

INTRODUCTION

Early childhood caries (ECC) continues to be one of the most common avoidable chronic illnesses of young children. The American Academy of Paediatric Dentistry (AAPD) defines ECC as the presence of one or more decaying, missing owing to caries, or filled tooth surfaces in any primary tooth in a child younger than 72 months of age. Severe ECC is defined as smooth surface caries in children younger than 3 years of age or age-specific thresholds for decaying, missing, or filled surfaces in children aged 3-5 years [1].

Dental caries is a dysbiosis disease of the dental biofilm characterized by frequent fermentable carbohydrate exposure, poor oral hygiene and inconsistent fluoride protection. The World Health Organization indicates that free sugars are a key common risk factor for caries and suggests that the intake of free sugars should be less than 10% of total energy, ideally below 5% [2]. Pain, illness, disturbed sleep, difficulties eating, school absences and impaired quality of life can be the result of untreated caries in children [3-5].

India has a high burden of preschool caries. The prevalence of ECC in India has been estimated to be about one-half of preschool children in recent systematic reviews, although the figures vary substantially depending on the region, sampling method, diagnostic threshold, age and socioeconomic background [6,7]. A recent study at a preschool in Sri Ganganagar revealed an increase in caries prevalence with age and higher caries prevalence among children from lower socioeconomic backgrounds, thereby emphasizing the necessity of locally adapted preventive interventions [8].

The study done by Lennon et al. employed cross-sectional parent reported caries data and evaluated parental, child and environmental factors across preschool ages [9]. The broad risk-domain approach is appropriate to adapt in the setting of Sri Ganganagar, as the risk for ECC in early childhood may be affected by family food habits, oral hygiene behaviours, parental education, access to dental services, and household environment [10-12].

Hence, the present study was done to assess a proposed age-related pattern of early childhood caries (ECC) in preschool children in Sri Ganganagar, Rajasthan, and to investigate correlations with parental, child behavioral, and household-level factors.

MATERIALS AND METHODS

Study design and setting

The cross-sectional study conducted among preschool children aged 24-71 months in Sri Ganganagar, Rajasthan, India. In actual practice, children can be enrolled from Anganwadi centres, private preschools and outpatient/community dental screening camps after

Institutional ethical permission and consent from parents/guardians

Participants

The simulated analytical sample consisted of 720 children: 160 aged 2 years, 180 aged 3 years, 190 aged 4 years and 190 aged 5 years. Children with severe systemic disease, developmental dental defects unrelated to caries or inadequate parent questionnaire data would be eliminated in a genuine study.

Outcome variable

ECC was modelled as a dichotomous outcome. ECC was defined as the presence of one or more primary teeth/surfaces documented as decaying, missing due to caries or filled. The dmft index was created as a descriptive count outcome. Field survey examinations should be conducted using standard WHO oral health survey methodologies, natural or artificial light, disposable mouth mirrors, CPI probes, where appropriate, infection control precautions and examiner calibration [13].

Potential influencing factors

The factors that were simulated in the exposure were the following: parental socioeconomic status (e.g., a mother's level of education being less than 10 years or her household income being lower), child and oral-health behaviours (e.g., consuming sugary snacks or drinks frequently, nursing at night, not brushing teeth more than once a day, continuing to wean or breastfeed after 24 months), the pattern of dental care for the mother (e.g., not getting a dental checkup more than once a year), environmental factors (e.g., frequent contact with pets). These domains are similar to those used in the modern ECC literature but have been tailored to the preschool environment of Sri Ganganagar [9-12].

Statistical analysis

All analyses were done on the simulated complete-case data. Age-specific prevalence was computed as percentages; Chi-square tests were used to examine ECC prevalence between binary exposure groups. Unadjusted odds ratios (OR) and 95% confidence intervals (CI) were calculated from 2 x 2 tables. Adjusted ORs for ECC were calculated using multivariable logistic regression model. Statistical significance was assessed at $p<0.05$. The analyses were performed in python with pandas, scipy, stats models and matplotlib.

Ethical statement

This draft does not include human participants as the data is simulated. Any legitimate survey in Sri Ganganagar necessitates clearance from an institutional ethics committee, informed consent from parents or guardians, child assent when applicable, confidentiality measures, and referral mechanisms for children needing dental care.

RESULTS

Study population and prevalence of ECC

The simulated dataset contained 720 preschool children, with 382 (54.9%) boys. The overall estimated frequency of ECC was 32.1% (231/720) with mean dmft of 1.06 (SD 1.75). The prevalence of ECC rose with age, from 23.1% at age 2 years to 43.7% at age 5 years (Table 1).

Univariable associations

In univariable analyses, ECC prevalence was higher among children exposed to frequent sugary snacks/drinks, toothbrushing less than once daily, night-time bottle feeding, lower maternal education, lower household income, maternal dental check-up less than once yearly, sibling caries, weaning/breastfeeding beyond 24 months, and regular pet contact. In these simulated data there was only a tiny difference between males (Table 2).

Multivariate Model

The strongest simulated relationships were for age 5 years, frequent sugary snacks/drinks, toothbrushing < once daily, night-time bottle feeding, lower household income, lower maternal education and sibling caries, and were all mutually adjusted. In the adjusted model, maternal dental check-up less than once per year and weaning beyond 24 months indicated weaker but directionally greater odds (Table 3).

TABLE

Table 1. Age-wise distribution and hypothesized ECC prevalence.

Age group	N	ECC cases	ECC prevalence (%)	Mean dmft (SD)	Male (%)
2 years	160	37	23.1	0.71 (1.49)	56.9
3 years	180	51	28.3	0.92 (1.68)	52.8
4 years	190	60	31.6	1.06 (1.78)	54.2
5 years	190	83	43.7	1.50 (1.92)	55.8
Total	720	231	32.1	1.06 (1.75)	54.9

ECC: early childhood caries; dmft: decayed, missing due to caries, filled primary teeth. Values are from simulated data.

Table 2. Univariable associations between selected factors and hypothesized ECC.

Factor	Exposed n	ECC % exposed	Reference n	ECC % reference	Unadjusted OR (95% CI)	p value
Male sex	395	31.4	325	32.9	0.93 (0.68-1.28)	0.721
Maternal education <=10 years	295	40.7	425	26.1	1.94 (1.41-2.66)	<0.001*
Lower household income	342	38.6	378	26.2	1.77 (1.29-2.43)	<0.001*
Sugary snacks/drinks >=2 times/day	339	36.9	381	27.8	1.52 (1.11-2.08)	0.012*
Bottle feeding at night	203	37.4	517	30	1.40 (0.99-1.97)	0.066
Toothbrushing <1 time/day	330	39.4	390	25.9	1.86 (1.36-2.55)	<0.001*

TABLE

Maternal dental check-up <once/year	359	39	361	25.2	1.90 (1.38-2.61)	<0.001*
Sibling with caries	148	42.6	572	29.4	1.78 (1.23-2.59)	0.003*
Weaning/breastfeeding >24 months	183	43.2	537	28.3	1.92 (1.36-2.72)	<0.001*
Regular pet contact	228	39	492	28.9	1.58 (1.13-2.19)	0.008*

Reference group is absence of the named exposure. OR: odds ratio. P values are chi-square based. Values are simulated, *= Statistically Significant.

Table 3. Multivariable logistic regression for hypothesized ECC.

Variable	Adjusted OR	95% CI	p value
Age 3 vs 2 years	1.58	0.93-2.68	0.091
Age 4 vs 2 years	1.7	1.01-2.86	0.046
Age 5 vs 2 years	3.33	2.00-5.55	<0.001*
Maternal education <=10 years	1.8	1.27-2.54	<0.001*
Lower household income	1.75	1.24-2.47	0.002*
Sugary snacks/drinks >=2 times/day	1.61	1.14-2.27	0.007*
Bottle feeding at night	1.45	1.00-2.11	0.051
Toothbrushing <1 time/day	1.85	1.31-2.62	<0.001*
Maternal dental check-up <once/year	1.81	1.28-2.56	<0.001*
Sibling with caries	2.1	1.40-3.16	<0.001*
Weaning/breastfeeding >24 months	2.04	1.39-2.99	<0.001*
Regular pet contact	1.88	1.30-2.71	<0.001*

Model includes all variables listed in the table. Age 2 years is the reference group. Values are simulated and illustrative, *= Statistically Significant.

DISCUSSION

This research shows a reasonable pattern of Sri Ganganagar preschool ECC with increasing incidence for ages 2-5 years. The age gradient is consistent with the cumulative nature of caries exposure in primary teeth and with earlier Indian and local preschool findings of increased caries at older preschool ages [8,14].

The most changeable behavioral signals in the simulated model were frequent sugary snacks/drinks, toothbrushing less than once a day, and night-time bottle feeding. This is physically feasible, as chronic sugar exposure consistently lowers plaque pH and supports cariogenic biofilm activity.

WHO sugar recommendations and broader caries literature support decreasing free sugars and avoiding sugar-sweetened beverages in very young children [5,13].

In the simulated data, poorer maternal education and lower household income were related with greater risks of ECC. This corresponds with Indian and worldwide research associating caries with social deprivation, parental education, oral-health knowledge and affordability/access to preventative dental services [15-16]. Pre-school children in Sri Ganganagar may reside in urban, peri-urban and rural settings, where family-level socioeconomic factors may influence food, brushing supervision, toothpaste use and care-seeking.

Sibling caries was maintained as a risk indicator. This could be associated with a common food, common oral hygiene, common caregivers' behaviours and probable microbial transmission in families. Similarly, infrequent maternal dental visits may reflect less preventative attitude or impediments to dental availability. These factors are easy to question at school or in Anganwadi visits by health professionals or dental interns, so they are effective in screening.

The uploaded KUNO-Kids reference paper revealed parent, child and environmental domains linked with parent-reported preschool caries including socioeconomic variables, dental health behaviour, weaning age, sibling caries and pet contact [9]. The current Sri Ganganagar draft is based on a similar conceptual structure, but adapts the outcome and prevalence level to a higher-burden Indian preschool setting.

The local study in 2018 from Sri Ganganagar similarly noted the age-related increases and socioeconomic inequalities that guided the direction of the present results [8].

A big strength of the draft is that all references are real and it is evident where the simulated data are. The biggest restriction is the absence of any real field observations. Hence, the prevalence numbers, p values and odds ratios should not be regarded as evidence about children in Sri Ganganagar. A true survey should include probability sampling, examiner calibration, measurement of water fluoride where possible, recording of plaque/oral hygiene, assessments of food frequency, and referral of children with untreated caries [13].

CONCLUSION

This study demonstrates a Sri Ganganagar preschool ECC article with an age-related increase in caries and increased odds for children exposed to frequent sugary snacks/drinks, poor toothbrushing, night-time bottle feeding, lower maternal education, lower income, and sibling caries, using a simulated transparent dataset. These hypothesized findings inform the development of a genuine, ethics-approved survey and focused prevention program including parents, Anganwadi workers, preschool instructors, pediatrics dentists and primary care providers.

PUBLIC HEALTH IMPLICATIONS

1. Counselling parents to reduce free sugars, especially sweet snacks, bottled drinks, sweetened milk and feeding from a bottle at bed time.
2. Carers should supervise brushing with an age-appropriate amount of fluoride toothpaste, at least twice daily, where possible.
3. Children with sibling caries, low parental education or sporadic dental visits could be targeted for screening and fluoride varnish referral.
4. Private preschools and Anganwadi institutions should be included in a field study in Sri Ganganagar so as to prevent under-representing lower income children

REFERENCES

1. American Academy of Pediatric Dentistry. Policy on early childhood caries (ECC): consequences and preventive strategies. The Reference Manual of Pediatric Dentistry. Chicago (IL): American Academy of Pediatric Dentistry; 2025.
2. World Health Organization. Sugars and dental caries. Geneva: World Health Organization; 2025.
3. World Health Organization. Global oral health status report: towards universal health coverage for oral health by 2030. Geneva: World Health Organization; 2022.

4. Petersen PE, Bourgeois D, Ogawa H, Estupinan-Day S, Ndiaye C. The global burden of oral diseases and risks to oral health. Bull World Health Organ. 2005; 83(9): 661-9.
5. Sheiham A, James WP. Diet and dental caries: the pivotal role of free sugars reemphasized. J Dent Res. 2015; 94(10): 1341-7. doi:10.1177/0022034515590377.
6. Devan I, Ramanarayanan V, Janakiram C. Prevalence of early childhood caries in India: a systematic review and meta-analysis. Indian J Public Health. 2022; 66 Suppl: S3-S11. doi:10.4103/ijph.ijph1078_22.
7. Ganesh A, Muthu MS, Mohan A, Kirubakaran R. Prevalence of early childhood caries in India - a systematic review. Indian J Pediatr. 2019; 86(3): 276-86. doi:10.1007/s12098-018-2793-y.
8. Nabi S, Kavikumar V, Tahir A. Incidence and prevalence of early childhood caries in preschool children-our experience. Eur J Biomed Pharm Sci. 2018; 5(8): 253-5. doi:10.20959/ejbps201808-5977.
9. Lennon AM, Fluch-Maier K, Hiller KA, Schenke I, Gade N, Buchalla W, et al. Factors associated with parent-reported caries in preschool children from the KUNO-Kids Health Study-repeated cross-sectional analysis. Community Dent Oral Epidemiol. 2026; 0: 1-17. doi:10.1111/cdoe.70078.
10. Prakash P, Subramaniam P, Durgesh BH, Konde S. Prevalence of early childhood caries and associated risk factors in preschool children of urban Bangalore, India: a cross-sectional study. Eur J Dent. 2012; 6(2): 141-52.
11. 1ose B, King NM. Early childhood caries lesions in preschool children in Kerala, India. Pediatr Dent. 2003; 25(6): 594-600.
12. Leong PM, Gussy MG, Barrow SY, de Silva-Sanigorski A, Waters E. A systematic review of risk factors during the first year of life for early childhood caries. Int J Paediatr Dent. 2013; 23(4): 235-50. doi:10.1111/j.1365-263X. 2012.01260. x.
13. World Health Organization. Oral health surveys: basic methods. 5th ed. Geneva: World Health Organization; 2013.
14. Kundu H, Patthi B, Singla A, Jankiram C, Jain S, Singh K. Dental caries scenario among 5, 12 and 15-year-old children in India-a retrospective analysis. J Clin Diagn Res. 2015; 9(7): ZE01-5. doi:10.7860/JCDR/2015/12439. 6150.
15. Folayan MO, El Tantawi M, Aly NM, Al-Batayneh OB, Schroth RJ, Castillo JL, et al. Association between early childhood caries and poverty in low- and middle-income countries. BMC Oral Health. 2020; 20(1): 8. doi:10.1186/s12903-019-0997-9.
16. Folayan MO, El Tantawi M, Aly NM, Al-Batayneh OB, Schroth RJ, Castillo JL, et al. Association between early childhood caries and parental education and the link to the sustainable development goal 4: a scoping review. BMC Oral Health. 2024. doi:10.1186/s12903-024-04291-w.

AUTHORS: -

Dr Manu Batra
Professor & Head
Department of Public Health Dentistry
Surendera Dental College & Research Institute
Email id: drmanubatra@aol.com
Mobile No: 9719522272

Dr Deeksha Gijwani
Reader
Department of Public Health Dentistry
Surendera Dental College & Research Institute
Email id: deeks09@gmail.com
Mobile No: 8769718770

CORRESPONDING AUTHOR: -

Dr Manu Batra
Professor & Head
Department of Public Health Dentistry
Surendera Dental College & Research Institute
Email id: drmanubatra@aol.com
Mobile No: 9719522272

EARLY ORTHOPEDIC MANAGEMENT OF SKELETAL CLASS III MALOCCLUSION USING ALT-RAMEC AND REVERSE-PULL FACEMASK THERAPY

Dr. Sakshi Garg, Dr. Eenal Bhambri, Dr. Ankit Bharadwaj

ABSTRACT

Prompt orthopaedic intervention is the favoured strategy for addressing skeletal Class III malocclusion in developing patients with maxillary insufficiency. This case study outlines the orthopaedic treatment of a 9-year-old girl with skeletal Class III malocclusion, relative maxillary insufficiency, anterior crossbite, slight anterior crowding, and a hypodivergent growth pattern. The evaluation of skeletal maturity (SMI Stage 3) suggested an opportune moment for growth alteration. The intervention utilised the Alternate Rapid Maxillary Expansion and Constriction (ALT-RAMEC) protocol using a bonded Hyrax device, succeeded by reverse-pull facemask therapy and retention thru a modified transpalatal arch and chin cup. Post-orthopedic assessment revealed effective maxillary advancement, enhancement of the sagittal skeletal connection and facial profile, rectification of the anterior crossbite, and positive cephalometric alterations while preserving vertical facial proportions. Preliminary orthopaedic intervention with the ALT-RAMEC regimen demonstrated efficacy in attaining skeletal alignment and laying a solid groundwork for future extensive orthodontic therapy.

KEYWORDS

Skeletal Class III malocclusion, ALT-RAMEC, reverse-pull facemask, early orthopaedic intervention, maxillary advancement, chin cup stabilisation.

INTRODUCTION

Skeletal Class III malocclusion represents a difficult dentofacial irregularity that may stem from a lack of maxillary development, mandibular advancement, or both factors combined.2. Prompt orthopaedic treatment during the active growth stage provides the most significant opportunity for beneficial skeletal adjustment and could lessen the requirement for future surgical interventions. The utilisation of reverse-pull facemask therapy is prevalent for maxillary protraction in adolescent Class III patients. Liou's ALT-RAMEC protocol improves circum-maxillary suture mobilisation thru cycles of expansion and constriction, thereby optimising the orthopaedic efficacy of facemask therapy in comparison to standard rapid maxillary expansion. This study outlines the effective orthopaedic intervention for a growing skeletal Class III patient thru the application of the ALT-RAMEC protocol, followed by reverse-pull facemask therapy and chin cup retention, showcasing the advantageous skeletal alterations realised after the orthopaedic phase.

PATIENT INFORMATION

A healthy child aged 9, arrived with the primary concern of a protruding mandible. Her medical and dental backgrounds were not significant.

EXTRAORAL EXAMINATION

The extraoral assessment indicated a symmetrical visage including a linear profile, pronounced jaw, adequate lip closure, and an oblique nasolabial angle.

INTRAORAL EXAMINATION

The patient was in mixed dentition with Angle's Class I molar relationship, anterior crossbite, minor anterior crowding in both arches, and 2-mm mandibular dental midline deviation to the left.



PRE- TREATMENT PHOTOS

CEPHALOMETRIC FINDINGS

Cephalometric analysis indicated skeletal Class III malocclusion with relative maxillary deficit, slight mandibular prognathism, hypodivergent growth pattern, and compensatory proclination of maxillary incisors with retroclination of mandibular incisors. Profile of soft tissue was straight with competent lips indicating favourable scenario for early orthodontic treatment.

GROWTH ASSESSMENT

Skeletal maturity examination showed SMI Stage 3, indicating active growth and favourable timing for orthopaedic intervention.

DIAGNOSIS

The individual was identified as having an evolving skeletal Class III malocclusion attributed to relative maxillary insufficiency, accompanied by an anterior crossbite, slight anterior crowding, a 2-mm deviation of the left mandibular dental midline, and a hypodivergent growth pattern.

TREATMENT OBJECTIVES

The goals of the treatment included maxillary protraction, rectification of the front crossbite, establishment of a favourable overjet, enhancement of the sagittal skeletal relationship while preserving the vertical development pattern, and ensuring a secure basis for extensive fixed orthodontic therapy.

TREATMENT ALTERNATIVES

Options for treatment encompassed postponed orthodontic intervention combined with orthognathic surgery, traditional rapid maxillary expansion succeeded by facemask therapy, and bone-anchored maxillary protraction. Given the patient's age, promising development potential, and the superior orthopaedic efficacy of the ALT-RAMEC regimen, early intervention with ALT-RAMEC in conjunction with reverse-pull facemask therapy was chosen.

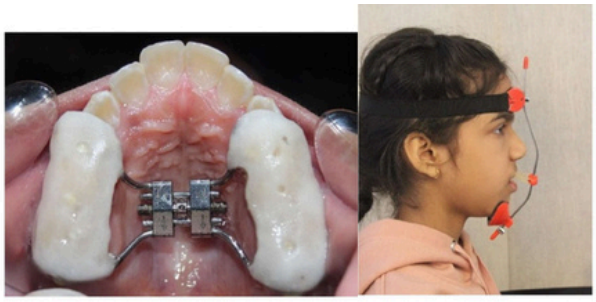
TREATMENT PLAN

The intervention comprised:

- ALT-RAMEC employing a bonded Hyrax device.
- Reverse-pull facemask intervention for maxillary advancement.
- Preservation utilising an altered transpalatal arch and chin cup.
- Thorough fixed orthodontic intervention following the orthopaedic stage.

TREATMENT PROGRESS

Pretreatment documentation was acquired prior to commencing therapy. A bonded Hyrax device was utilised to execute the ALT-RAMEC procedure via alternating weekly dilation and constriction. This was succeeded by reverse-pull facemask treatment (about 400 g per side, 12–14 hours daily) until the anterior crossbite was rectified and the sagittal skeletal connection enhanced. Following the active orthopaedic phase, the Hyrax device was taken out, a revised transpalatal arch was installed, and chin cup therapy commenced to preserve the orthopaedic adjustment over the subsequent growth phase.



BONDED HYRAX AND FACEMASK

TREATMENT RESULTS

The orthopaedic phase yielded advantageous enhancements in skeletal, dental, and face structures. The external examination revealed improved midfacial prominence and a more harmonious facial contour. Intraorally, the front crossbite was rectified by achieving a positive overjet, hence creating a secure occlusal base for future fixed orthodontic intervention. Post-treatment cephalometric analysis revealed effective maxillary advancement, enhancement of the sagittal skeletal connection, and preservation of the advantageous vertical growth pattern with little adverse dentoalveolar consequences. The orthopaedic adjustment remained consistent throughout the retention phase utilising a modified transpalatal arch and chin cup.



POST-TREATMENT PHOTOGRAPHS

CEPHALOMETRIC ANALYSIS

The juxtaposition of cephalometric results before and after therapy revealed advantageous alterations in skeletal, dental, and soft tissue structures. Maxillary advancement led to enhanced sagittal jaw alignment with little alterations in the mandible and consistent vertical facial ratios. Analysis of the soft tissue indicated enhanced support for the top lip and an improved facial profile. The results validated the effective orthopaedic rectification of the skeletal Class III malocclusion and established a reliable basis for future extensive orthodontic intervention.

Variable	Pretreatment	Post-Orthopedic	Clinical Significance
SNA	78°	81°	Maxillary advancement
ANB	-2°	0°	Improved sagittal relationship
Wits appraisal	-6 mm	-2 mm	Reduced skeletal discrepancy
Co-A	72 mm	89 mm	Increased effective maxillary length
Co-Gn	99 mm	107 mm	Normal mandibular growth
Maxillomandibular differential	27 mm	23 mm	Improved jaw relationship
Overjet	-1 mm	+2 mm	Anterior crossbite corrected



CHIN CUP FOR RETENTION

CONCLUSION

This example illustrates that prompt orthopaedic intervention employing the ALT-RAMEC regimen, succeeded by reverse-pull facemask therapy, is a successful strategy for treating developing patients with skeletal Class III malocclusion resulting from maxillary insufficiency. Beneficial enhancements in skeletal, dental, and facial structures were attained with consistent vertical ratios, establishing a robust basis for future extensive orthodontic intervention.

REFERENCES

1. Liou EJ, Tsai WC. A new protocol for maxillary protraction in cleft patients: repetitive weekly protocol of alternate rapid maxillary expansions and constrictions. Cleft Palate Craniofac J. 2005 Mar;42(2):121-7
2. Isci D, Turk T, Elekdag-Turk S. Activation – deactivation rapid palatal expansion and reverse headgear in Class III cases. Eur J Orthod 2010; 32: 706–15
3. Liou EJ. Toothborne orthopedic maxillary protraction in Class III patients. J Clin Orthod. 2005; 39: 68–75.
4. Baik HS. Clinical results of the maxillary protraction in Korean children. American Journal of Orthodontics and Dentofacial Orthopedics. 1995 Dec 1;108(6):583-92.
5. Burstone CJ, Marcotte MR, Taylor TD. Problem solving in orthodontics: Goaloriented treatment strategies. Chicago, IL: Quintessence; 2000 Oct 1.
6. Westwood PV, McNamara JA Jr, Baccetti T, Franchi L, Sarver DM. Long-term effects of Class III treatment with rapid maxillary expansion and facemask therapy followed by fixed appliances. Am J Orthod Dentofacial Orthop 2003;123:306-20.

AUTHORS: -

Dr. Sakshi Garg
Post Graduate Student
Department of Orthodontics and Dentofacial Orthopedics
Surendera Dental College & Research Institute
Email id: drsakshigarg0105@gmail.com
Mobile No: 7973414841

Dr. Eenal Chhabra Bhambri
Professor & Head
Department of Orthodontics and Dentofacial Orthopedics
Surendera Dental College & Research Institute
Email id: dreenal700@gmail.com
Mobile No: 8769829700

Dr. Ankit Bharadwaj
Reader
Department of Orthodontics and Dentofacial Orthopedics
Surendera Dental College & Research Institute
Email id: drankitbharadwaj@gmail.com
Mobile No: 9602355012

CORRESPONDING AUTHOR: -

Dr. Sakshi Garg
Post Graduate Student
Department of Orthodontics and Dentofacial Orthopedics
Surendera Dental College & Research Institute
Email id: drsakshigarg0105@gmail.com
Mobile No: 7973414841

NONSURGICAL HEALING OF PERIAPICAL LESION WITH INTRACANAL MEDICAMENT OF CALCIUM HYDROXIDE AND CHLORHEXIDINE

Dr. Manjusha Gopalakrishnan, Dr. Neetu Jindal, Dr. Renu Aggarwal

ABSTRACT

This case report presents a conservative, non surgical endodontic approach for the healing of periapical lesion in relation to 24 using a freshly prepared paste made from calcium hydroxide powder mixed with 2% chlorhexidine solution as intracanal medicament. Patient was recalled at 1 month, 3 months and 6 months for follow up. Radiographic examination was done during follow up for assessment of healing of periapical lesion. Six months follow up radiographs showed significant increase in the radiopacity in the periapical area suggestive of clinically significant effect of mixture of calcium hydroxide and 2% chlorhexidine to resolve periapical pathology without surgical intervention.

KEYWORDS

Periapical lesion, Calcium hydroxide, 2% Chlorhexidine, Non-surgical root canal treatment.

INTRODUCTION

Pulpal inflammation often arising from trauma or extensive tooth decay can progress to pulp necrosis if left untreated.[1] This necrotic environment promotes bacterial colonization in the root canal system.[2] These micro-organisms release toxins into the periapical area that initiates an inflammatory response, leading to the formation of periapical lesion. [1,2]

Periapical lesions are one of the most common pathological conditions that are commonly encountered in dental practice.[3] Periapical lesions can subside if non-surgical root canal therapy completely eliminates the microbial etiology.[4] However, due to the complex and polymicrobial nature of the root canal infection, mechanical debridement alone may not ensure complete disinfection.[2] Therefore, effective chemomechanical preparation combined with potent antimicrobial intracanal medicaments is crucial for successful non surgical endodontic therapy.[1,4,5]

Among the various intracanal medicaments available, calcium hydroxide has remained a preferred choice due to its high pH and anti microbial properties.[6] However, its effectiveness is limited against certain pathogens such as Enterococcus faecalis, which are commonly found in persistent infections.[7]

Recent advancements recommend combining calcium hydroxide with chlorhexidine, a broad-spectrum antimicrobial with residual activity for enhanced efficacy. This case report illustrates the healing of periapical lesion using freshly prepared paste of calcium hydroxide powder mixed with 2% chlorhexidine solution.

CASE REPORT

A 26-year-old male patient reported to the Department of Conservative dentistry and Endodontics at Surendera Dental College and Research Institute with chief complaint of decay in upper left back tooth region of the jaw. Clinical and radiographic examination was done with respect to 24. Clinically, deep class 2 caries was seen involving the distal surface of tooth. Tooth was non-tender on percussion. Thermal test was done for assessing the vitality of tooth. There was negative response to thermal test which states that tooth was non vital. Pulp exposure of the involved tooth with a well circumscribed radiolucency in relation to the apical region of 24 was seen on an intraoral periapical radiograph.

Under rubber dam isolation, access cavity was prepared using Endo Z Fu Burs (Dentsply Maillefer, Ballaigues, Switzerland). Working length determination was done using apex locator (Root ZX Mini, J Morita Corp. USA) to ascertain the length of the root canal and confirmed with a radiograph. Biomechanical preparation was done using a combination of stainless steel hand k files (MANI, Inc Japan) and rotary NiTi files, progressively enlarging the canals upto size 25/4%. Copious irrigation with 3% NaOCl (KMC, pharmacy, India) using 27-gauge side vented needle (Navi Tip, ultra dent products. inc USA) was done in between each instrumentation.

After final rinse with 2% chlorhexidine (Zodenta Safe Plus, NeelKanth health Care), canals were dried using absorbent paper points. A paste was freshly prepared by mixing Calcium hydroxide powder (PrevestDenPro) with 2% chlorhexidine solution. This intracanal medicament was delivered into the canals using a lentulospiral (Mani inc Japan). Access cavity was temporarily sealed using Cavit (3M ESPE, St Paul, MN, USA). At the next visit after 14 days, temporary restoration was removed and the intracanal medicament was thoroughly flushed out from the canals using hand files along with copious irrigation using sodium hypochlorite and EthyleneDiaminetetraacetic acid (Merck Germany) irrigant solutions. After proper drying of the canals, closed dressing of calcium hydroxide powder and 2% chlorhexidine mixture was applied again.

One week later, the patient was recalled again. Since the tooth was asymptomatic, the canals were obturated by

lateral condensation technique with gutta percha and AH Plus sealer (Dentsply, Maillefer, Swizerland) and restored post endodontically with interim restoration. At the six month follow up visit, the patient reported with no symptoms and the radiographic evaluation showed significant healing with reduction in the size of periapical radiolucency (Fig 2). Due to insufficient remaining tooth structure, a metal post was placed (Fig 3) and composite core build up was done. Tooth preparation was done. A porcelain fused to metal crown was fabricated and cemented at the final appointment restoring both function and aesthetics (Fig 5).

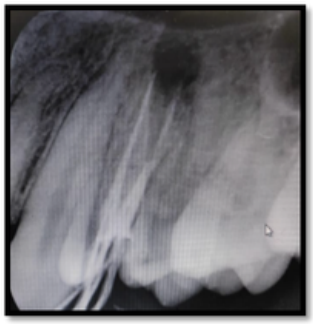


Fig 1: Post Obturation Radiograph

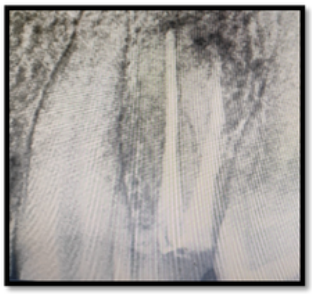


Fig 2: Follow up radiograph after 6 months



Fig 3: Metal post placement done

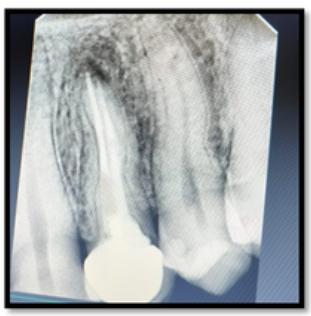


Fig 4: Crown Placement done



Intraoralphotographs showing PFM crown with respect to 24.

DISCUSSION

Periapical lesions often result from chronic infections to pulpal necrosis.[7] Traditionally, surgical intervention was considered as an option for large or persistent lesions. However, advances in endodontic techniques and materials have shifted the focus towards conservative approach. Numerous studies have demonstrated that proper chemomechanical debridement combined with effective intracanal medicament can resolve periapical lesions without surgical intervention.

A study by Del Fabbro et al (2007) found that after one year, there was no significant difference in success rate between surgical and non-surgical treatment approaches for periapical lesions.[1] However, surgical procedures come with several disadvantages such as the risk of damaging adjacent vital teeth, injury to surrounding anatomical structures, and the potential for post-operative pain and discomfort.[1,3] Because of these risks, nonsurgical management is generally preferred and should be considered the first line of treatment. Surgical intervention is to be considered only when non-surgical root canal therapy does not succeed.

To achieve proper periradicular region sterilization i.e. complete elimination of microorganisms and healing, various intracanal medicaments and irrigation techniques have been used in adjunct with root canal therapy for better treatment outcomes.

Calcium hydroxide is the most commonly used intracanal medicament in endodontics due to its alkaline nature and its ability to induce hard tissue formation. Its high pH disrupts bacterial membranes and neutralizes endotoxins released by anaerobic microorganisms.[8] However, certain resistant bacteria like Enterococcus faecalis can survive in treated canals even in the presence of calcium hydroxide. This limitation of calcium hydroxide can be overcome by combining it with chlorhexidine.[7]

Chlorhexidine binds to dentin and provide long term antimicrobial action due to its substantivity property. Chlorhexidine due to its cationic nature disrupts bacterial cell membranes leading to leakage of cytoplasmic contents and cell death. Studies by Evans et al. and Gomes et al (2002) have shown that the calcium hydroxide and chlorhexidine mix is significantly more effective in eliminating E. faecalis than calcium hydroxide alone.[9]

In this case, the mixture led to substantial healing of the lesion over a six-month period without surgical intervention.

CONCLUSION

This case demonstrates that a conservative, non-surgical endodontic approach using a combination of calcium hydroxide and chlorhexidine intracanal mixture can effectively heal even large periapical lesions. Proper disinfection combined with effective medicament can often eliminate the need of surgical intervention for large lesions. Clinicians should consider such conservative approach as the first line of treatment reserving surgery only for cases where non - surgical therapy fails.

REFERENCES

1. Ramesh A, Pillai R, Afzal A, Santhosh A, Arunima GS, Sandeep KV. Healing of periapical lesion by non-surgical endodontic therapy: A case series. IP Indian Journal of Conservative and Endodontics. 2024;9(2):99–102. doi: 10.18231/2024.022.
2. Mohanty AK, Kumar D, Non-surgical management of a large periapical lesion using triple antibiotic paste: A case report. Journal of Oral Medicine, Oral Surgery, Oral Pathology and Oral Radiology. 2022;8(1):49–52. doi: 10.18231/2022.010.
3. Alghamdi F, Alkhattab O. Effectiveness of intracanal calcium hydroxide medicament in treating periapical lesions: a systematic review.Jos.2022;75(1):44-54. doi:10.5114/jos.2022.114502.
4. Kriplani S, Sedani S, Mishra A, Umre U. Non-surgical management of periapical lesions with the use of newer modalities in adjunct to the conventional: A case series. Cureus2024;16(3):57314;10.7759;57314.
5. Bansode PV, Bhalerao VU, Pathak SD, Wavdhane MB, Rana H, Rai S. Nonsurgical healing of large periapical lesion with nonsurgical root canal treatment and intracanal medicament of calcium hydroxide and chlorhexidine: A case report. IOSR Journal of Dental and Medical Sciences. 2019;18(1):40–43;10.9790/0853-1801084043.
6. Kala M, Sindhuri AR. Comparison of healing of periapical lesion with different intracanal medicament using mineral trioxide aggregate as apical barrier. IOSR Journal of Dental and Medical Sciences. 2017;16(5):17–22.
7. Sonigra H, Patel N, Vachhani K, Attur K. Calcium hydroxide and chlorhexidine: Synergy in non-surgical endodontic approach for extensive periapical lesion: New traditional approach. Journal of Advanced Oral Research. 2015;6(1):39–43. doi:10.1177/2229411220150108.
8. Evans MD, Baumgartner JC, Khemaleelakul SU and Xia T. Efficacy of calcium hydroxide: chlorhexidine pastes as an intracanal medication in bovine dentin. J Endod 2003; 29:338-9; 2003;29(5):338–339.
9. Gomes BP, Vianna ME, Sena NT, Zaia AA, Ferraz CC and de Souza Filho FJ. In vitro evaluation of the antimicrobial activity of calcium hydroxide combined with chlorhexidine gel used as intracanal medicament. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006; 102: 544-50.

AUTHORS: -

Dr. Manjusha Gopalakrishnan
Post Graduate Student
Department of Conservative Dentistry and Endodontics
Surendera Dental College & Research Institute
Email id: manjuprejith10@gmail.com
Mobile No: 8589018196

Dr. Neetu Jindal
Professor & Head
Department of Conservative Dentistry and Endodontics
Surendera Dental College & Research Institute
Email id: drneetujindal@gmail.com
Mobile No: 9929854146

Dr. Renu Aggarwal
Professor
Department of Conservative Dentistry and Endodontics
Surendera Dental College & Research Institute
Email id: drrenu.hmh@gmail.com
Mobile No: 9460956567

CORRESPONDING AUTHOR: -

Dr. Manjusha Gopalakrishnan
Post Graduate Student
Department of Conservative Dentistry and Endodontics
Surendera Dental College & Research Institute
Email id: manjuprejith10@gmail.com
Mobile No: 8589018196

MODIFICATION IN TREATMENT OF ZYGOMATICOMAXILLARY COMPLEX FRACTURE AS PER AESTHETIC CONCERNS

Dr. Manisha Solanki, Dr. Piyush Pathak, Dr. Devyendu Garg, Dr. Jadhav Chetan,Dr. Darshan R. Chouhan

ABSTRACT

Zygomaticomaxillary complex (ZMC) fractures commonly result in functional impairment and facial asymmetry, making both functional and aesthetic restoration essential. This case report describes the management of a 28-year-old female with a right-sided ZMC fracture following a road traffic accident. Although open reduction and internal fixation (ORIF) through an extraoral approach under general anesthesia was initially planned, the patient preferred to avoid visible facial scarring and general anesthesia. An intraoral vestibular approach under local anesthesia was therefore performed, with reduction of the fracture and fixation using an L-shaped miniplate at the zygomaticomaxillary buttress. The patient achieved satisfactory functional recovery with restoration of facial symmetry and no external scar. This case highlights the importance of individualized treatment planning and demonstrates that an intraoral approach can provide excellent aesthetic and functional outcomes in selected ZMC fractures.

KEYWORDS

ZMC Fracture, ORIF, Intraoral approach, Maxillofacial trauma, Facial aesthetic, vestibular incision

INTRODUCTION

Zygomatic fractures also known as Zygomatic complex fracture, are one of the most common fracture in the orofacial region due to the prominent and vulnerable position of the zygomatic bone. While these fracture leads to functional difficulty such as trismus, diplopia or even sensory disturbance, the aesthetic component of the injury is one of the main concern of the patient [1]. The zygoma is one of the main structural element of the midface, which contribute heavily to contour and facial symmetry, forming the cheek prominence and overall structure of the face. Thus any injury to the face on this region can have profound effect on facial appearance, making cosmetic concerns a major priority for patients.

The two primary surgical approaches used in the management of zygomatic fractures are the extraoral and intraoral approaches. The extraoral approach, provides direct access to the fracture for precise reduction and fixation, particularly in cases with significant displacement or comminution. While effective, this approach carries the risk of visible scarring. On the other hand, the intraoral approach, allows for the reduction of fractures with minimal visible scarring, making it a preferred option for patients concerned about their facial appearance.[2]

Zygomaticomaxillary Complex (ZMC) fractures can be treated using one-point, two-point, or three-point fixation techniques. One-point fixation is simple and quick, suitable for minimally displaced fractures but lacks stability. Two-point fixation offers better support and is ideal for moderately displaced fractures, providing more control over alignment. Three-point fixation, considered the gold standard, offers maximum stability, restoring the three-dimensional structure of the face and minimizing the risk of malunion, making it the best choice for complex or severely displaced fractures. The choice of fixation method depends on fracture severity and desired functional and aesthetic outcomes[3]

This case report discusses the clinical presentation of the patient, the surgical rationale for approaching the treatment which provides both functional and aesthetic outcomes that are acceptable to both the patient and operating surgeon.

CASE REPORT

A 28yr old female reported to the Department of Oral and maxillofacial surgery at Surendera Dental College and Research Institute with a chief complaint of Pain in the right cheek bone region.

Patient gave history of road traffic accident 10 days back with an impact on the right lateral side of the face, after which patient was rushed to near by hospital at which patient was administered emergent medical care. Patient then reported to their family doctor where she was administered analgesics as well as antibiotics for the injury. She then reported to department with classical symptoms of zygomatic fracture including facial asymmetry due to flattening of the right side along with sub conjunctival hemorrhage in the right eye. Patient also showed abrasion on the right side of the face with visible scarring as well as edema on the FZ region. Tenderness was present on palpation with respect to the right side of the face localized to the infraorbital as well as zygomatic region of the face. A step deformity was also palpated on the right infraorbital region. On radiographic examination fracture line was visible in the zygomatic region as well as the frontal-zygomatic region.

Open Reduction with Internal Fixation (ORIF) under General anaesthesia with an extraoral approach through

infraorbital region was considered as the most appropriate treatment option, however on discussion with the patient she expressed her concern about the extraoral scar associated with the extraoral incision. Along with this the patient was not willing to under general anesthesia for the treatment. Thus keeping the patient concern in mind, ORIF under Local Anesthesia with an intraoral approach through the vestibular incision was opted as the treatment option.

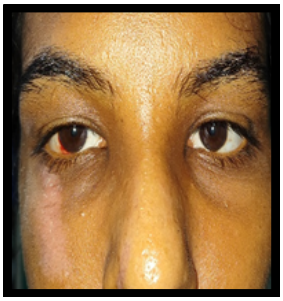


Fig 1: showing sub conjunctival hemorrhage as well as abrasion on the right side



Fig 2: Birds eye view showing depression of malar prominence on right side.

Before undergoing the procedure, patient was given I.V. Medication which included amoxicillin, metrogyl and rantec; as well as an intramuscular injection of diclofenac.

Patient was asked to do a chlorhexidine mouthwash after which the patient's was draped as well the incision site painted with butadiene. Posterior superior alveolar as well as infraorbital nerve block on the right side, which was administered with Lidocaine Hcl with 1:80000 adrenaline.

A 3cm incision was given in the vestibular region from canine to first molar by the help of stainless steel no 15 blade on a bard parker no 4 handle. Full thickness mucoperiosteal flap was raised by the help of molt no 9 periosteal elevatore to expose the fracture site. After exposure of the fracture site the two fractured segements were reduced by the help of Bristow's elevator intraorally. The fracture site was assessed for proper union. The site was then irrigated and the incison closed in layers with 3-0 silk.

Patient was then monitored for any complications. Patient was ordered to take soft diet as well as keep the incision site clean. Pt was advised cold pack compression for first 24 hrs after the surgery to reduce the post-operative swelling, if any. Pt was ordered to not sleep on the fractured site for atleast 3 weeks after the surgery. Pt was recalled for regular follow up at 1 week, 3 week, 6 week and 6 months to check for healing.



Fig 3: shows the occlusion as well as the site for incision for vestibular approach



Fig 4: shows the fracture site at zygomaticomaxillary buttress region.



Fig 5: L shaped plate is placed at the buttress region for fixation at ZMC region



Fig 6: Closure of the fracture site done in layers with 3-0 silk sutures.



Fig 7: Post Op vs Pre Op showing no scaring as well deformity on the right side.

DISCUSSION

Zygomaticomaxillary Complex (ZMC) fractures, also referred to as tripod fractures, are one of the most common facial fractures encountered in trauma cases. These fractures typically involve the zygomatic bone's connections to the maxilla, the lateral orbital wall, and the temporal bone.[1] Proper treatment is crucial not only for restoring facial appearance but also for maintaining he functionality of the eye socket, nasal passages, and other key facial structures. The fixation method used for these fractures can significantly impact the healing process, influencing both the aesthetic and functional outcomes. Three common fixation techniques for ZMC fractures are one-point, two-point, and three-point fixation.[2] Each of these methods has its own unique advantages and limitations, making the choice of fixation strategy highly dependent on the nature and severity of the fracture.

One-point fixation is the simplest and least invasive of the

three methods. In this technique, the fracture is secured at just one point, typically in an area that is easily accessible, such as the zygomatic arch or orbital rim. This method is most commonly employed in cases of minimally displaced fractures or when the fracture is not complex[3,4]. Because it involves only one fixation point, it offers a relatively quick and straightforward approach to stabilizing the fracture. The advantages of this technique include its simplicity, shorter surgical time, and lower risk of complications like nerve damage or infection, as it typically requires less dissection of surrounding tissues. However, one-point fixation also has significant limitations. Since it only stabilizes the fracture at a single point, it may not provide sufficient support to hold the fractured pieces in place during the healing process.[3] As a result, there is a higher risk of the fracture displacing again, particularly in cases where the fracture is more than minimally displaced or if the surrounding bone structures are not well aligned. For this reason, one-point fixation is generally reserved for less complicated fractures.

Two-point fixation, as the name suggests, involves the use of two fixation points to stabilize the fracture. This method typically includes fixation at the zygomaticomaxillary buttress and the zygomatic arch or at the inferior orbital rim. Two-point fixation is generally used for fractures that are more displaced than those treated with one-point fixation but still not so complex as to require three-point fixation. The key advantage of two-point fixation is the added stability it offers compared to the one-point technique. [5]By securing the fracture at two points, the surgeon has more control over the alignment of the fractured pieces, reducing the likelihood of secondary displacement. The two-point technique also allows for better anatomical restoration of the zygoma, which is essential for both aesthetic and functional recovery. However, two-point fixation is not without its downsides. While it is more stable than one-point fixation, it still does not offer the same level of support as three-point fixation. As a result, it may not be suitable for more severely displaced fractures or for fractures with multiple fragments. Additionally, the procedure is more invasive than one-point fixation, requiring a longer surgical time and a slightly higher risk of complications such as infection, nerve damage, or damage to surrounding soft tissues. Nevertheless, two-point fixation is a solid option for many ZMC fractures that are not as severe as those requiring three-point fixation.

Three-point fixation is considered the gold standard for the majority of ZMC fractures, particularly those that are severely displaced or involve multiple fracture lines.[6] This technique stabilizes the fracture at three distinct points: the zygomaticomaxillary buttress, the zygomatic arch, and the orbital rim. By securing the fracture at three points, the surgeon ensures that the fractured zygoma is fully stabilized, which is critical for both functional and cosmetic outcomes. This method restores the zygoma's three-dimensional shape, ensuring proper alignment with the orbital floor, maxillary arch, and other facial structures. The key advantage of three-point fixation is its superior stability, which significantly reduces the risk of malunion or non-union. In cases where the fracture is complex or involves multiple fragments, this technique provides the necessary support to hold the bone fragments in place during the healing process. [7]Additionally, three-point fixation allows for precise anatomical restoration of the facial structures, which is especially important for patients concerned about aesthetic outcomes following facial trauma.

However, three-point fixation comes with its own set of challenges. It is a more technically demanding procedure, requiring a high level of surgical skill and experience. The surgery tends to be longer and more complex, as it involves securing the fracture at multiple sites.[7] As a result, the risk of complications such as nerve injury, infection, or excessive scarring may be higher compared to the one-point or two-point methods. In addition, the procedure may involve more extensive dissection of the surrounding tissues, which can increase recovery time and postoperative discomfort. Despite these potential drawbacks, the benefits of three-point fixation in terms of fracture stability and long-term outcome often outweigh the risks, particularly in cases of severe or complex fractures.

CONCLUSION

In conclusion, the choice between one-point, two-point, and three-point fixation for ZMC fractures depends on several factors, including the severity of the fracture, the degree of displacement, and the specific goals of the surgery. One-point fixation remains a useful technique for minimally displaced fractures, offering a quicker and less invasive approach. Two-point fixation is suitable for fractures with moderate displacement, providing better stability than one-point fixation without the complexity of three-point fixation. For more complex, severely displaced fractures, three-point fixation is generally considered the gold standard, offering superior stability and a higher chance of restoring both function and appearance. Each case must be evaluated individually, with the surgeon considering the fracture's complexity, the patient's needs, and the potential risks of each technique. Ultimately, the goal is to achieve optimal healing, restore facial aesthetics, and preserve the functionality of the affected facial structures.

REFERENCES

1. Moreira Marinho RO, Freire-Maia B. Management of fractures of the zygomaticomaxillary complex. Oral Maxillofac Surg Clin North Am. 2013;25(4). doi:10.1016/j.coms.2013.07.011
2. Ellis E, Kittidumkerng W. Analysis of treatment for isolated zygomaticomaxillary complex fractures. Journal of Oral and Maxillofacial Surgery. 1996;54(4). doi:10.1016/S0278-2391(96)90107-X
3. Balakrishnan K, Ebenezer V, Dakir A, Kumar SK, Prakash D. Management of tripod fractures (zygomaticomaxillary complex) 1 point and 2 point fixations: A 5-year review. J Pharm Bioallied Sci. 2015;7. doi:10.4103/0975-7406.155937
4. Shokri T, Sokoya M, Cohn JE, Bahrami A, Inman J, Ducic Y. Single-Point Fixation for Noncomminuted Zygomaticomaxillary Complex Fractures—A 20-Year Experience. Journal of Oral and Maxillofacial Surgery. 2020;78(5). doi:10.1016/j.joms.2019.12.030
5. Kim JH, Kim YS, Oh DY, Jun YJ, Rhie JW, Moon SH. Efficacy of Altered Two-Point Fixation in Zygomaticomaxillary Complex Fracture. Biomed Res Int. 2020;2020. doi:10.1155/2020/8537345
6. Nasr WF, ElSheikh E, El-Anwar MW, Sweed AH, Bessar A, Ezzeldin N. Two- versus Three-Point Internal Fixation of Displaced Zygomaticomaxillary Complex Fractures. Craniomaxillofac Trauma Reconstr. 2018;11(4). doi:10.1055/s-0037-1604199
7. Widodo DW, Dewi DJ, Ranakusuma RW, Irawati Y. Evaluation of 3 and 2-point internal fixation in the management of zygomaticomaxillary complex fractures: Case report. Annals of Medicine and Surgery. 2021;67. doi:10.1016/j.amsu.2021.102539

AUTHORS

Dr. Manisha Solanki
Professor and Head
Department of Oral and Maxillofacial Surgery
Surendera Dental College and Research Institute
Email id: drmanisha80@gmail.com
Mobile No: 9460956567

Dr. Piyush Pathak
Post Graduate Student
Department of Oral and Maxillofacial Surgery
Surendera Dental College and Research Institute
Email id: piyushpathak53@gmail.com
Mobile No: 9501445012

Dr. Devyendu Garg
Post Graduate Student
Department of Oral and Maxillofacial Surgery
Surendera Dental College and Research Institute
Email id: devyendugarg31@gmail.com
Mobile No: 9464120460

Dr. Jadhav Chetan
Post Graduate Student
Department of Oral and Maxillofacial Surgery
Surendera Dental College and Research Institute
Email id: cjadhav.338@gmail.com
Mobile No: 7620168151

Dr. Darshan R. Chouhan
Post Graduate Student
Department of Oral and Maxillofacial Surgery
Surendera Dental College and Research Institute
Email id: chouhandarshan697@gmail.com
Mobile No: 7414830082

CORRESPONDING AUTHOR: -

Dr. Manisha Solanki
Professor and Head
Department of Oral and Maxillofacial Surgery
Surendera Dental College and Research Institute
Email id: drmanisha80@gmail.com
Mobile No: 9460956567

Dr. Sadhika Sadhar, Dr. Suruchi Juneja

ABSTRACT

The presence of teeth at birth (natal teeth) or within a month after birth (neonatal teeth) is relatively a rare phenomenon. Natal and neonatal teeth are conditions of significant importance to pediatric dentists and pediatricians. This report discusses two cases in which infants less than a month old required extraction of a mobile mandibular natal tooth to avoid the risk of aspiration and interference with feeding. Also, a review of the literature was conducted to discuss the aetiology, clinical features, complications, and management of natal and neonatal teeth.

KEYWORDS

Natal teeth, Neonatal teeth.

INTRODUCTION

Normal eruption of primary teeth is seen with the eruption of mandibular incisors at around 6 months of age [1]. Premature eruption of these teeth is addressed with various phrase such as congenital teeth, pre deciduous teeth, fetal teeth, and dentitia praecox [2]. Massler and Savaral [3] proposed two different terms as tooth/teeth present at birth as "natal teeth" and those erupting during the first month of life as "neonatal teeth. Natal and neonatal teeth are seen in the mandibular incisor region with a 66% predilection for females [4]. The presence of teeth in newborns is uncommon, varying from 1:6000 to 1:800 cases, occurring in general with incidences of two or three teeth [5].

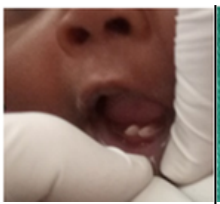
The exact aetiology is not known. Several sources suggest a possible hereditary component [4].An autosomal dominant gene was co related by a report of a family of 5 siblings who were born with natal teeth [6]. In a study that was conducted on Alaskan Tlingit Indians, concluded the prevalence of natal or neonatal teeth to be 9% of their newborns [7].

Environmental factors, particularly including exposure to polychlorinated biphenyls, have appeared to have increased incidence of natal teeth [8]. Natal teeth have been associated with a number of developmental abnormalities and various syndromes, including cleft lip and palate, Pfeiffer, Ellis-van Creveld (chondroectodermal dysplasia), Rubinstein-Taybi, steatocystoma multiplex, pachyonychia congenita (Jadassohn-Lewandowsky), cyclopia, Hallermann-Streiff (Mandibulo-oculo-facial dyscephaly with hypotrichosis), Pierre-Robin, Wiedeman- Rautenstrauch (neonatal progeria), Pallister-Hall, ectodermal , craniofacial dysostosis, multiple adrenogenital, Sotos, steatocystoma, epidermolysis bullosa simplex, and Walker-Warburg syndrome [1, 7,].

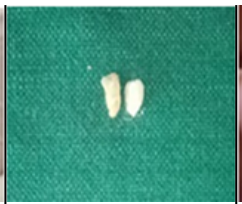
However, in most cases the pathogenetic factors are impossible to identify. Therefore, careful evaluation of these infants is highly recommended. When the tooth/teeth are excessively mobile, extraction is indicated preventing accidental risk of and swallowing or aspiration. However, when reviewing the literature, no reported case of aspiration of natal or neonatal teeth has been documented. The most common complaint of natal and neonatal teeth was found to be trauma to the tongue on the tip or ventral surface, a complication referred to as Riga-Fede syndrome reported in 6–10% of cases [4].

CASE PRESENTATION 1

A 20 days male newborn was referred to the Department of Paediatric and Preventive Dentistry with loose teeth in the lower front region of the jaw with a complaint of discomfort during suckling mother's milk (Figure 1a). The infant was (2.5 kg) and was delivered by normal vaginal delivery, with normal prenatal history of the mother. On intraoral examination, there was presence of natal teeth in the lower front gum region. Moderate mobility (Grade II) was associated with the teeth posing a threat of aspiration, so it was decided to extract the teeth. Hence, prophylactic administration of vitamin K was done before extraction. Extraction of a shell shaped crown (Figure 1b) was done under topical anaesthesia with epinephrine. Careful curettage of the sockets was conducted to remove any odontogenic cells remnants that might be left at the extraction site otherwise. Post extraction haemostasis was obtained (Figure 1c). Postoperative instructions were given to the mother and a follow-up visit was scheduled a week later.



(Figure 1a) – Natal teeth in lower anterior region



(Figure 1b) - Extraction of a shell shaped crown



(Figure 1c)- Post extraction haemostasis obtained

CASE PRESENTATION 2

A 28-day-old female newborn was referred to the Department of Paediatric and Preventive Dentistry with the complain of loose teeth in the lower front tooth region

and discomfort during breastfeeding. The medical history was not relevant. Intraoral examination revealed small shell shaped crowns in the anterior lower gum region exhibiting grade III mobility and lesion on ventral surface of tongue (Figure2a). It was decided that the teeth needs to be extracted as there was risk of aspiration and it had also injured the ventral surface of tongue. Prophylactic administration of Vitamin K was not required as the mother was previously administered Vitamin K. The teeth with a faint opaque whitish color, smaller than a primary tooth were extracted (Figure 2 b). The extraction site was carefully curetted for any remnants. Post extraction hemostasis was obtained (Figure2 c). Postoperative instructions were given to the mother and a follow-up visit was scheduled a week later.



(Figure2a)- Natal teeth in lower anterior region



(Figure 2b) - Extraction of a shell shaped crown



(Figure 2c)- Post extraction haemostasis obtained

DISCUSSION

The aetiology of natal and neonatal teeth remains unclear however it is related to various factors, including superficial position of the tooth germ, increased eruption rate due to pyretic incidents, hormonal stimulation, developmental abnormalities, syndromes, heredity, and osteoblastic activity within the germ zone related to the remodelling phenomenon [1, 7, 8,]. These teeth can have various alterations in morphology can be either conical or of normal shape may have an opaque yellow, brown color and usually have smaller crown than of primary teeth [1, 8,].

Hebling et al. [9] suggested clinical classification according to tooth morphology during eruption into the oral cavity: (1) Shell-shaped crown that is poorly fixed to the alveolus by gingival tissue with root absence. (2) Solid crown that is poorly fixed to the alveolus by gingival tissue with little or no root. (3) Eruption of the incisal margin of the crown through the gingival tissues.(4)Gingival edema with palpable but unerupted tooth.

The enamel in these teeth is normal .However, once the teeth erupt prematurely; the uncalcified enamel matrix wears off due to incomplete mineralization leading to teeth becoming yellow brown in color [6]. Furthermore, the increased mobility leads to dentin and cementum cervical changes and possible ensuing of Hertwig's sheath degeneration preventing root formation [10].

Histological data on natal and neonatal teeth have found that varying degrees of hypoplastic enamel cover the crowns of these teeth. The enamel thickness for natal teeth is 300mm and for neonatal teeth it is 135 mm, whereas in normal primary teeth the enamel layer is between 1000 and 1200mm [11].The dentinal area did not reveal any significant differences compared to normal primary teeth; however some SEM studies of these teeth have shown large interglobular spaces with abnormal cell inclusions.

The correct diagnosis of natal and neonatal teeth is important so as to determine if these teeth are supernumerary or normal dentition. Bohn nodules and dental lamina cyst are additional oral manifestations that may be confused with these dental conditions; but they can be differentiated by radiographic examination. Several factors should be considered before a treatment plan is decided: (1) degree of mobility and implantation, (2) convenience during suckling, (3) interference with breastfeeding, and (4) if the tooth is supernumerary or is part of the normal dentition.

Indications for removal include risk of dislocation, subsequent aspiration, and traumatic injury to the tongue and/or the maternal breast [12]

Natal/neonatal teeth that show mobility of more than 1mm are indicated for extraction; this is due to the probability of aspirating or ingesting natal teeth. Another reason for the removal of the natal/neonatal tooth is to alleviate feeding difficulties or complications like Riga- Fede disease. If extraction is the treatment of choice, it can be deferred till the child is 10 days of age or more and has appropriate blood levels of vitamin K. This ten-day waiting period is to allow the normal flora of the intestine to become established to produce vitamin K, an essential factor for prothrombin production in the Liver [1,6]

Since parenteral vitamin K prevents a life threatening haemorrhagic disease of the newborn, the American Academy of Pediatrics recommends that all newborns be given a single intramuscular dose of 0.5 to 1mg of vitamin K [13].

Once extraction is performed, it is essential to remove the underlying dental papilla and Hertwig's epithelial root sheath during the extraction of natal tooth/teeth to prevent the development of root structure that could continue if these structures are left in situ.

CONCLUSION

Natal and neonatal teeth are rare occurrences in the oral cavity and proper evaluation and diagnosis are crucial to provide the best treatment option. Paediatricians are usually the first to detect these teeth and early consultation with the dentist can prevent complications. The decision to maintain or remove these teeth should be assessed in each case independently. Radiographic examination is an essential diagnostic tool. Thus far, no studies confirmed the cause and effect relationship with any of the proposed theories so far. The aetiology of natal and neonatal teeth still required further investigations.

REFERENCES

1. Leung AK, Robson WL. Natal teeth: a review. Journal of the national medical association. 2006 Feb;98(2):226.
2. Portela MB, Damasceno L, Primo LG. Unusual case of multiple natal teeth. Journal of Clinical Pediatric Dentistry. 2005 Sep 1;29(1):37-9.
3. Massler M, Savara BS. Natal and neonatal teeth: a review of twenty-four cases reported in the literature. The Journal of pediatrics. 1950 Mar 1;36(3):349-59.
4. Kates GA, Needleman HL, Holmes LB. Natal and neonatal teeth: a clinical study. Journal of the American Dental Association (1939). 1984 Sep 1;109(3):441-3.
5. Gonçalves FA, Birman EG, Sugaya NN, Melo AM. Natal teeth: review of the literature and report of an unusual case. Brazilian Dental Journal. 1998 Jan 1;9(1):53-6.
6. Cunha RF, Boer FA, Torriani DD, Frossard WT. Natal and neonatal teeth: review of the literature. Pediatric dentistry. 2001 Dec 1;23(2):158-62.
7. Mayhall JT. Natal and neonatal teeth among the Tlinget Indians. Journal of dental research. 1967 Jul 1;46(4).
8. Rogan WJ. PCBs and cola-colored babies: Japan, 1968, and Taiwan, 1979. Teratology. 1982 Dec 1;26(3):259-61.
9. Hebling J, Zuanon AC, Vianna DR. Dente Natal—a case of natal teeth. Odontol Clin. 1997;7:37-40.
10. Spouge JD, Feasby WH. Erupted teeth in the newborn. Oral Surgery, Oral Medicine, Oral Pathology. 1966 Aug 1;22(2):198-208.
11. Bigeard L, Hemmerle J, Sommermater JI. Clinical and ultrastructural study of the natal tooth: enamel and dentin assessments. ASDC journal of dentistry for children. 1996 Jan 1;63(1):23-31.
12. Buchanan S, Jenkins CR. Riga-Fedes syndrome: Natal or neonatal teeth associated with tongue ulceration. Case report. Australian dental journal. 1997 Aug;42(4):225-7.
13. Bhutta ZA, Darmstadt GL, Hasan BS, Haws RA. Community-based interventions for improving perinatal and neonatal health outcomes in developing countries: a review of the evidence. Pediatrics. 2005 Feb 1;115(Supplement_2):519-617.

AUTHORS

Dr. Sadhika Sadhar
Reader
Department of Paedodontics and Preventive Dentistry
Surendera Dental College and Research Institute
Email id: sadhika94sadhar@gmail.com
Mobile No: 7087929878

Dr. Suruchi Juneja
Professor and Head
Department of Paedodontics and Preventive Dentistry
Surendera Dental College and Research Institute
Email id: suruchisukhija@hotmail.com
Mobile No: 9829365880

CORRESPONDING AUTHOR: -

Dr. Suruchi Juneja
Professor and Head
Department of Paedodontics and Preventive Dentistry
Surendera Dental College and Research Institute
Email id: suruchisukhija@hotmail.com
Mobile No: 9829365880

GUIDED ENDODONTIC TREATMENT FOR DISCOLOURED CALCIFIED CENTRAL INCISOR

Dr. Priya Jindal, Dr. Neetu Jindal, Dr. Renu Aggarwal, Dr. Grusha Gupta

ABSTRACT

Teeth with calcific metamorphosis pose a challenge to the endodontist in achieving proper access, identification, and debridement of the root canal. With the conventional technique, radiographs and bur orientation have dictated the access cavity preparation, which leads to the removal of more sound tooth structure and iatrogenic errors. To overcome these issues, the recent diagnostic imaging techniques such as cone beam computed tomography followed by three-dimensional fabrication of template have been proposed, which aid the clinician to accurately locate and negotiate the obliterated canal. The present case report describes the importance of conservative access cavity preparation with the use of the guided endodontic technique for the treatment of calcified canals in the maxillary central incisor.

KEYWORDS

Cone-beam Computed Tomography, Dental Pulp Calcification, Guided Endodontics, Pulp Canal Obliteration

INTRODUCTION

Dental trauma results in various complications, which pose an enigma to the practitioner. Calcification is one among the sequelae after trauma. According to the American Association of endodontists, it is defined as "A pulpal response to trauma that is characterized by deposition of hard tissue within the root canal space".[1] It is also referred to as pulp canal obliteration, dystrophic calcification, diffuse calcification, and calcific degeneration. The mechanism for calcification is unclear. According to Robertson, it may be caused due to the disturbance in the neurovascular circulation of the pulp.[2]

The incidence of calcification in canals was approximately 4%–24%. Endodontic treatment in calcified teeth is contradictable. Some studies suggest that these teeth need to be observed and a clinical follow up is needed before the endodontic procedure.[3] Teeth with symptomatic periapical pathology are considered to be ideal for intervention.

Teeth with canal obliteration can complicate easy access to the pulp canal leading to the difficulty in negotiation of the root canal, which can result in

CASE PRESENTATION

A 32-year-old female was referred to the Department of Endodontics, complaining of discoloration in the left maxillary central incisor. She had a history of dental trauma 10 years ago. Her medical history was non-contributory. The patient was classified as ASA I. The clinical examination showed discoloration in the left maxillary central incisor (Figure 1A). The tooth didn't respond to the pulp sensibility tests, there was no symptom in percussion and palpation. Mobility and probing depth were normal. An initial periapical radiographic examination of teeth showed pulp canal calcification and rarefaction in the periapical area (Figure 1B). The tooth was diagnosed as pulpless infected canal and asymptomatic periodontitis.

High-resolution CBCT images with 100-micron voxel size, 110 kV, and 6 cm × 6 cm ield of view (NewTom Vgi, Verona, Italy) were acquired. CBCT images showed obliteration in the coronal and middle thirds (15 mm) of the canal and the only patent canal segment was the apical portion 6 mm above the apex (Figure 1C). Also, a periapical lesion with a diameter of 2 mm diameter was noticed (Figure 1D). Once the patient was clarified about the treatment approach, informed consent was obtained. To access the root, a virtual customized dimensions of 5 mm in length, 3 mm in external diameter, and 1 mm in internal diameter was used. It was merged into the template to serve as a drill guide during the orifice shaping and planning process to access the canal.

The template was placed on the anterior teeth, and the stability and accordance of the template were checked. The tooth was isolated with the split dam technique (Figure 2 A and B). The access guide was fixed and positioned under local anesthesia with lidocaine and 1/100000 epinephrine (Persocaine; Daroupakhsh, Tehran, Iran) with an infiltration technique. Via the template sleeve, a mark was made to show the precise location of the endodontic access cavity. First, a hole in the enamel of the access area was prepared by a round diamond bur (Meisinger, Dusseldorf, Germany) with a high-speed hand-piece (NSK, Tokyo, Japan), then the template was placed, and mounce bur #1 (Meisinger, Germany) was used with a low-speed handpiece (NSK, Japan) to penetrate the canal through a correct direction. In this way, we could perform a minimally invasive access cavity that kept the incisal edge of the tooth intact.

Drilling was performed with saline irrigation. After drilling 10 mm inside the tooth, a K-file #10 was placed in the canal, and the direction of the prepared hole was checked with periapical radiographs with different horizontal angulations, then drilling resumed 16 mm into

the tooth. A K-ile #10 was used to negotiate the canal, and the apex locator (Morita, Tokyo, Japan) was applied to determine the working length which was conirmed by peri-apical radiography. A glide path was prepared with manual K-ile #15. Passive ultrasonic (Woodpecker, Guangxi, China) with a solution of 2.5% NaOCl was used for root canal irrigation. Cleaning and shaping were performed by RaCe rotary iles up to size 25, 4% (FKG Dentaire, La Chaux de Fonds, Switzerland). A paper cone was used to dry the root canal and the root canal was obturated using gutta-percha cones (Meta-Biomed, Cheongju City, Korea) and AH-26 sealer (Dentsply Maillefer, Ballaigues, Switzerland) by cold lateral condensation method (Figure 2C).



Figure 1A



Figure 1B

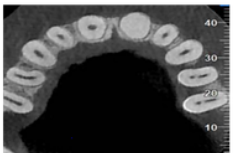


Figure 1C

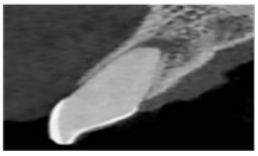


Figure 1D

Figure 1 A) The photograph shows discoloration in the maxillary left central incisor; B) Periapical radiograph shows pulp canal calcification and a periapical lesion; C) CBCT image, the axial reconstruction shows pulp canal obliteration in the cervical third; D) CBCT image, the sagittal reconstruction shows pulp canal obliteration in cervical and middle third.



Figure 2(A)

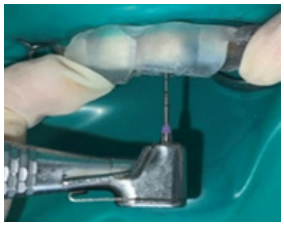


Figure 2(B)



Figure 2 (C)

Figure 2 A) The printed resin template; B) The template was adapted to the maxillary teeth and stabilized manually to avoid vertical movement during drill insertion; C) Peri-apical radiograph after obturation

DISCUSSION

This paper emphasizes the advantage of treatment of patients with canal calcification using new technologies like CBCT and guided endodontics. CBCT imaging is a helpful tool for identifying root canal anatomy, detecting periapical diseases, recognizing vertical root fractures, planning surgical procedures, and estimating the degree of canal obliteration.[7] According to the statement of the American Association of Endodontists and the American Academy of Oral and Maxillofacial Radiography (AAE/AAOMR) on the application of CBCT imaging in endodontics, CBCT is recommended for locating calcified root canals.[8] Holcomb and Gregory's[9] investigation, reported the prevalence of partial or complete canal calcification to be 4% for incisors, and these teeth also have a 7%–27% probability of developing a periapical lesion. There have been reports of a poor prognosis for RCT in teeth that don't succeed during instrumentation.[10] Despite calcified tooth that shows the tooth without any pulp canal space, histologically, pulp space containing pulp tissue is found in majority of them.[11] So guided endodontics makes root canal treatment more predictable for clinicians to perform and helps them minimize iatrogenic consequences, which enhances the prognosis.[6] Pulp canal obliteration (PCO) itself is not considered an endodontic disease, it may occur as a healing sign.[12] Root canal treatment is only indicated in the presence of acute symptoms and apical periodontitis or when Pulp canal obliteration complicates the execution of treatment and deteriorates prognosis.[9] According to the AAE, Pulp canal obliteration is considered to be an endodontic case with a high degree of difficulty which should be considered for referral. With the use of microscopes and ultrasonic tips, skilled endodontists were able to complete root canal treatment in PCO cases with 80% success rate.[13] Even when employed by general practitioners or inexperienced dentists, successful RCTs became significantly more common using the guided endodontic method.[5] This technique can help perform endodontic treatment in teeth with calcified canals without a dental microscope.[14] There is virtually minimal risk of perforation, ledge formation, and missed calcified canal with this procedure, regardless of the degree of calcification, unless there are errors in design or insufficient stability in the guide.[7,15] A case report by Krastl et al. (2016) as well as numerous other studies[19-22] explain how conservative access cavity preparation is feasible if it's created parallel to the tooth's long axis through the incisal edge.[4,16-19] Involvement of the incisal edge makes aesthetic results compromised. Lara-Mendes et al. (2018) reported modified endodontic access that doesn't invade the incisal edge and showed that it is possible to prepare access up on the cingulum in guided endodontics.[20] In this report, to preserve the tooth structure and to perform a minimally invasive RCT, the guided access preparation was modified, so the access was prepared on the conventional area instead of the incisal edge. Guided access is a reliable and predictable method that enhances both the short-term and

long-term outcomes since it enables more conservative approaches and maintaining tooth structure.

CONCLUSION

This case report explains root canal treatment with an endodontic guide for a heavily calcified central incisor of the maxilla in which the access was modified to preserve the incisal edge. The long-term prognosis can be enhanced by this conservative technique. To improve the success rate and reduce iatrogenic accidents, this case study recommends using CBCT and an endodontic guide while treating calcified canals.

REFERENCES

1. American Association of Endodontics. Glossary of Endodontic Terms. 8th Ed. Chicago, IL: American Association of Endodontics, 2012.
2. Robertson A. A retrospective evaluation of patients with uncomplicated crown fractures and luxation injuries. Endod Dent Traumatol. 1998;14(5):245–56.
3. McCabe PS, and Dummer PMH. Pulp canal obliteration: an endodontic diagnosis and treatment challenge. Int Endod J. 2011;45(2)177–97.
4. Krastl G, Zehnder MS, Connert T, et al. Guided endodontics: a novel treatment approach for teeth with pulp canal calcification and apical pathology. Dent Traumatol. 2016; 32(3)240-46.
5. Connert T, Krug R, Eggmann F, Emsermann I, ElAyouti A, Weiger R, Kuhl S, Krastl G. Guided Endodontics versus Conventional Access Cavity Preparation: A Comparative Study on Substance Loss Using 3-dimensional-printed Teeth. J Endod. 2019;45(3):327-31.
6. Dąbrowski W, Puchalska W, Ziemlewski A, Ordyniec-Kwasnica I. Guided Endodontics as a Personalized Tool for Complicated Clinical Cases. Int J Environ Res Public Health. 2022;19(16):1-19.
7. Van Der Meer WJ, Vissink A, Ng YL, Gulabivala K. 3D Computer aided treatment planning in endodontics. J Dent. 2016;45:67–72.
8. AAE. Use of Cone Beam Computed Tomography in Endodontics 2015 Update. Oral Surg Oral Med Oral Pathol Oral Radiol. 2015;120(4):508–12.
9. Holcomb JB, Gregory WB Jr. Calcific metamorphosis of the pulp: its incidence and treatment. Oral Surg Oral Med Oral Pathol. 1967;24(6):825–30.
10. Cvek M, Granath L, Lundberg M. Failures and healing in endodontically treated non vital anterior teeth with post traumatically reduced pulpal lumen. Acta Odontol Scand. 1982;40(4):223–8.
11. Schindler WG, Gullickson DC. Rationale for the management of calcific metamorphosis secondary to traumatic injuries. J Endod. 1988;14(8):408–12.
12. Robertson A, Andreasen FM, Bergenholtz G, Andreasen JO, Noren JG. Incidence of pulp necrosis subsequent to pulp canal obliteration from trauma of permanent incisors. J Endod. 1996;22(10):557–60.
13. Wu D, Shi W, Wu J, Wu Y, Liu W, Zhu Q. The clinical treatment of complicated root canal therapy with the aid of a dental operating microscope. Int Dent J. 2011;61(5):261- 66.
14. Ishak G, Habib M, Tohme H, Patel S, Bordone A, Perez C, Zogheib C. Guided Endodontic Treatment of Calcified Lower Incisors: A Case Report. Dent J(Basel). 2020;8(3):1-8.
15. Fonseca Tavares WL, de Carvalho Machado V, Oliveira Fonseca F, Canal Vasconcellos B, Cruz Guimaraes L, Diniz Viana AC, Feitosa Henriques LC. Guided Endodontics in Complex Scenarios of Calcified Molars. Iran Endod J. 2020;15(1):50–6.
16. Connert T, Zehnder MS, Weiger R, Kuhl S, Krastl G. Microguided Endodontics: Accuracy of a Miniaturized Technique for Apically Extended Access Cavity Preparation in Anterior Teeth. J Endod. 2017;43(5):787–90.
17. Connert T, Zehnder MS, Amato M, Weiger R, Kuhl S, Krastl G. Microguided Endodontics: a method to achieve minimally invasive access cavity preparation and root canal location in mandibular incisors using a novel computer-guided technique. Int Endod J. 2018;51(2):247–55.
18. Nabavi S, Navabi S, Mohammadi SM. Management of Pulp Canal Obliteration in Mandibular Incisors with Guided Endodontic Treatment: A Case Report. Iran Endod J. 2022;17(4):216–9.
19. Moreira Maia L, Maria Toubes K, Moreira Junior G, Quadros Tonelli S, de Carvalho Machado V, Ferreira Silveira F, Nunes E. Guided Endodontics in Nonsurgical Retreatment of a Mandibular First Molar: A New Approach and Case Report. Iran Endod J. 2020;15(2):111–6.
20. Lara-Mendes STO, Barbosa CdFM, Machado VC, Santa-Rosa CC. A New Approach for Minimally Invasive Access to Severely Calcified Anterior Teeth Using the Guided Endodontics Technique. J Endod. 2018;44(10):1578–82.

AUTHORS: -

Dr. Priya Jindal
Post Graduate Student
Department of Conservative Dentistry and Endodontics
Surendera Dental College & Research Institute
Email id: drpriyajindal33@gmail.com
Mobile No: 6378001816

Dr. Neetu Jindal
Professor & Head
Department of Conservative Dentistry and Endodontics
Surendera Dental College & Research Institute
Email id: drneetujindal@gmail.com
Mobile No: 9929854146

Dr. Renu Aggarwal
Professor
Department of Conservative Dentistry and Endodontics
Surendera Dental College & Research Institute
Email id: drrenu.hmh@gmail.com
Mobile No: 9460956567

Dr. Grusha Gupta
Senior Lecturer
Department of Conservative Dentistry and Endodontics
Surendera Dental College & Research Institute
Email id: grushagupta37@gmail.com
Mobile No: 7837190883

CORRESPONDING AUTHOR: -

Dr. Priya Jindal
Post Graduate Student
Department of Conservative Dentistry and Endodontics
Surendera Dental College & Research Institute
Email id: drpriyajindal33@gmail.com
Mobile No: 6378001816

3D BIOPRINTING IN PERIODONTAL REGENERATION – A NEW ERA IN DENTISTRY

Dr. Khushi Khatri, Dr. Rajni Aggarwal

ABSTRACT

Periodontitis is a major cause of tooth loss and results in the destruction of the tooth-supporting structures, including the periodontal ligament, cementum, alveolar bone, and gingival tissues. Conventional regenerative approaches, including guided tissue regeneration, bone grafts, and growth factor therapy, have shown promising clinical outcomes; however, their ability to achieve complete and predictable periodontal regeneration still remains limited. In recent years, three-dimensional (3D) bioprinting has become an important part of tissue engineering due to its ability to create highly precise and patient-specific tissue constructs that closely resemble natural tissue architecture. Bioprinting combines different biomaterials, cells and growth factors to create tissue structures. Developments in scaffold design, the addition of stem cells, the creation of bioinks, and the use of controlled growth factor delivery system have significantly increased the interest in using 3D bioprinting for periodontal regeneration. This review discusses the principles, techniques, biomaterials, applications, advantages, limitations, and future prospects of 3D bioprinting particularly in the field of periodontal regeneration. Findings from current experimental and clinical studies indicate that 3D bioprinting may become a significant advancement in regenerative dentistry.

KEYWORDS

3D bioprinting, periodontal regeneration, scaffolds, bioinks.

INTRODUCTION

Periodontitis is a chronic inflammatory disease that progressively damages the supporting structures of the teeth including the gingiva, periodontal ligament, cementum and alveolar bone.[1] It is one of the primary causes of tooth loss and affects oral function and overall quality of life. Conventional periodontal treatment mainly aims to control infection and prevent further tissue damage. However, achieving complete and predictable regeneration of the lost periodontal structures still remains a major challenge in clinical practice. In recent years, various regenerative techniques including guided tissue regeneration (GTR), bone grafts, enamel matrix derivatives, platelet concentrates, and growth factor therapies have been developed to enhance healing results. While these techniques can promote tissue repair to a certain extent, they often fail to completely restore the natural structure and function of periodontal tissues because of the highly complex and organized architecture of the periodontium.[2,3] Tissue engineering has created new opportunities in regenerative dentistry through the combined use of scaffolds, cells, and signalling molecules for the repair and regeneration of damaged tissues. Among the latest developments in this field, 3D bioprinting has attracted considerable attention due to its ability to produce customized tissue constructs with highly accurate structural organization. Unlike traditional scaffold fabrication methods, 3D bioprinting enables precise layer-by-layer deposition of cells, biomaterials, and growth factors, thereby closely mimicking the natural extracellular matrix and architecture of periodontal tissues.[4]

Although several technical and regulatory challenges still exist, ongoing research continues to highlight the promising potential of 3D bioprinting in periodontal regeneration. Therefore, this review focuses on the principles, techniques, biomaterials, applications, advantages, limitations, and future prospects of 3D bioprinting in periodontology.[4,5]

PERIODONTAL TISSUE ENGINEERING

Periodontal tissue engineering is based on three important components:

1. Cells
2. Scaffolds
3. Bioactive signalling molecules

The periodontium consists of both hard and soft tissues that are arranged in a highly organized structure. Effective periodontal regeneration requires the simultaneous formation of alveolar bone, cementum, periodontal ligament fibres, and gingival connective tissue. One of the major advantages of 3D bioprinting is its ability to recreate this multilayered arrangement with a high degree of precision.[4,6]

WHAT IS 3D BIOPRINTING?

3D bioprinting is an advanced bio fabrication technique in which bioinks containing living cells, biomaterials and bioactive substances are deposited layer-by-layer to create tissue-like structures.[3]

The general workflow of 3D bioprinting includes:

1. Imaging and defect assessment using CBCT or CT scans
2. Computer-aided design (CAD)

3. Preparation of bioinks
4. Layer-by-layer printing of constructs
5. Tissue maturation in bioreactors

This approach enables fabrication of customized structures that closely mimic the natural anatomy of periodontal tissues.[6]

COMPONENTS OF 3D BIOPRINTING

1. Bioinks

Bioinks are printable biomaterials that contain living cells and biologically active substances.[4,6]

Characteristics of Ideal Bioinks

- Biocompatibility
- Printability
- Mechanical stability
- Biodegradability
- Cell viability support
- Controlled release of growth factors

Commonly Used Bioinks

- Alginate
- Gelatin methacrylate (GelMA)
- Collagen
- Hyaluronic acid
- Chitosan
- Fibrin
- Silk fibroin

These materials provide favourable conditions for cellular adhesion, proliferation, and differentiation.[4,6]

2. Cells Used in Periodontal Bioprinting

Stem cells play a major role in tissue regeneration.[4,6]

Commonly Used Cells

- Periodontal ligament stem cells (PDLSCs)
- Dental pulp stem cells (DPSCs)
- Bone marrow mesenchymal stem cells (BMMSCs)
- Gingival fibroblasts
- Osteoblasts

PDLSCs are particularly promising because they possess osteogenic and cementogenic differentiation capabilities.[4,6]

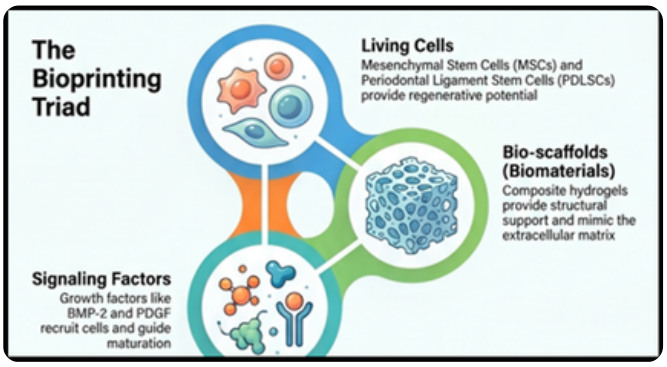
3. Growth Factors

Growth factors stimulate cellular proliferation and differentiation.[4,5]

Commonly Used Growth Factors

- Bone morphogenetic protein-2 (BMP-2)
- Platelet-derived growth factor (PDGF)
- Transforming growth factor-beta (TGF-β)
- Vascular endothelial growth factor (VEGF)

These molecules promote angiogenesis, osteogenesis, and periodontal ligament formation.[4,5,6]



TYPES OF 3D BIOPRINTING TECHNIQUES

Inkjet Bioprinting

Inkjet bioprinting is one of the earliest and most commonly used techniques in tissue engineering. It functions by depositing small droplets of bioink onto a substrate using either thermal or piezoelectric mechanisms. In thermal systems, heat creates air bubbles that push droplets through the nozzle, whereas piezoelectric systems use electrical impulses to generate pressure for droplet release.[3,5]

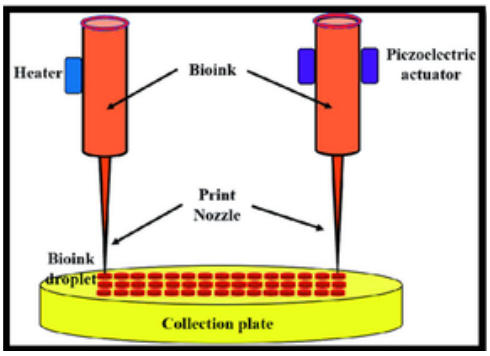
This technique is mainly suitable for low-viscosity bioinks and offers rapid printing with relatively low operational cost. Because of its non-contact printing mechanism and high speed, it is widely used for printing cell-laden hydrogels and incorporating growth factors in periodontal regeneration.[5]

Advantages

- Rapid printing speed
- Cost-effective and widely accessible
- Good cell viability due to reduced mechanical stress
- Precise deposition of small bioink droplets
- Useful for gradient printing and growth factor delivery

Limitations

- Restricted to low-viscosity bioinks
- Possibility of nozzle clogging
- Lower mechanical strength of printed constructs
- Limited vertical stability
- Less suitable for large tissue constructs



Inkjet Bioprinting

Extrusion-Based Bioprinting

Extrusion-based bioprinting is the most frequently used technique in regenerative dentistry and periodontal tissue engineering. In this method, bioink is continuously extruded through a nozzle using pneumatic, piston-driven, or screw-driven pressure systems.[3,6]

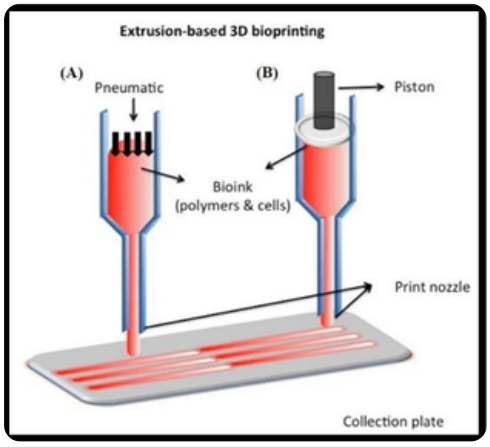
This technique is highly versatile because it can process a wide range of biomaterials with different viscosities, including hydrogels, ceramics, polymer blends, and cell-loaded matrices. Due to its ability to produce mechanically stable scaffolds with high cell density, extrusion bioprinting is widely used for periodontal ligament and alveolar bone regeneration.[6]

Advantages

- Suitable for highly viscous bioinks
- Capable of printing high cell concentrations
- Better structural integrity and scaffold strength
- Compatible with multiple biomaterials
- Useful for large and complex tissue constructs

Limitations

- Shear stress may reduce cell viability
- Lower resolution compared to laser-based methods
- Risk of nozzle blockage
- Longer printing time for complex structures



Laser-Assisted Bioprinting

Laser-assisted bioprinting (LAB) is a nozzle-free technology that uses laser energy to transfer bioink droplets from a donor surface to a receiving substrate. The system typically consists of a laser source, an energy-absorbing layer, and a bioink-coated ribbon.[3,6]

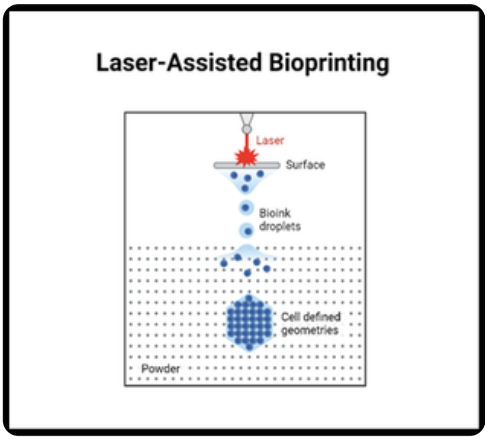
When the laser pulse strikes the absorbing layer, localized vaporization generates pressure that pushes the bioink toward the collector surface. Because this method eliminates nozzle-related issues and minimizes mechanical stress, it provides excellent cell viability and high printing precision.

Advantages

- Extremely precise printing
- No nozzle clogging
- High cell survival rate
- Excellent spatial resolution
- Suitable for delicate cell types

Limitations

- Expensive equipment and maintenance
- Technically complex procedure
- Limited availability in routine clinical research
- Time-consuming fabrication process



Stereolithography (SLA)

Stereolithography is a light-based bioprinting technique that uses photopolymerization to fabricate highly accurate three-dimensional structures. In this method, ultraviolet or visible light selectively polymerizes photosensitive bioinks layer-by-layer according to a CAD model.[5]

SLA is considered one of the most precise bioprinting techniques because it provides smooth surface architecture, excellent resolution, and accurate structural control. It is particularly useful for creating complex scaffold geometries with interconnected porosity required in periodontal tissue engineering.[4,5]

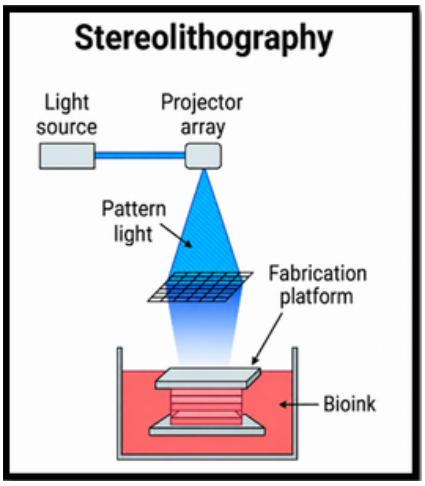
Hydrogels such as GelMA and polyethylene glycol diacrylate (PEGDA) are commonly used in SLA-based bioprinting.

Advantages

- High printing accuracy
- Smooth surface architecture
- Excellent structural precision
- Ability to create complex microstructures
- Faster fabrication compared to some traditional methods

Limitations

- Limited biomaterial compatibility
- Potential cytotoxicity from photo initiators and UV exposure
- Restricted use of highly viscous bioinks
- High equipment cost



SCAFFOLD DESIGN IN PERIODONTAL REGENERATION

Scaffolds provide structural support for tissue regeneration.[7]

Characteristics of Ideal Scaffold

- Biocompatibility
- Porosity
- Mechanical strength
- Controlled degradation
- Cell adhesion capability

Recent advances have enabled fabrication of multiphasic scaffolds capable of simultaneously regenerating bone, PDL, and cementum.[6,7]

APPLICATIONS OF 3D BIOPRINTING IN PERIODONTOLOGY

Periodontal Ligament Regeneration

Bio printed scaffolds containing periodontal ligament stem cells (PDLSCs) have shown promising results in regenerating periodontal ligament tissues. These scaffolds help in achieving organized PDL fibre orientation and improved attachment formation, thereby supporting functional periodontal regeneration.[7]

Alveolar Bone Regeneration

Bio ceramic-based scaffolds incorporated with materials such as hydroxyapatite and β-tricalcium phosphate have demonstrated significant osteogenic potential. These constructs promote bone formation and enhance the regeneration of alveolar bone defects.[8]

Gingival Tissue Engineering

Hydrogel-based bio printed constructs provide a favourable environment for gingival fibroblast growth and proliferation. They contribute to improved soft tissue healing and support regeneration of gingival tissues.[8]

Customized Guided Tissue Regeneration Membranes

Using CBCT-derived data, patient-specific guided tissue regeneration membranes can be fabricated with high precision. These customized membranes adapt accurately to periodontal defects, improving regenerative outcomes.[7]

Drug Delivery Systems

Bio printed scaffolds can also serve as advanced drug delivery systems. They allow controlled and sustained

release of antimicrobial agents, growth factors, and other therapeutic molecules directly at the defect site, thereby enhancing tissue healing and regeneration.[8]

RECENT ADVANCES IN 3D BIOPRINTING

Multiphasic Scaffolds

Recent developments have led to the fabrication of multiphasic scaffolds that closely resemble the natural organization of periodontal tissues. These scaffolds contain separate compartments designed specifically for bone, periodontal ligament, and cementum regeneration.[9,10]

Smart Bioinks

Smart bioinks are an emerging innovation in tissue engineering. These bioinks can respond to environmental stimuli and release bioactive molecules in a controlled manner, improving tissue regeneration efficiency.[9]

4D Bioprinting

4D bioprinting is an advanced extension of 3D bioprinting in which printed constructs can alter their shape or function over time in response to external stimuli such as temperature, moisture, or pH changes.[9,10]

AI and CAD Integration

The integration of artificial intelligence with computer-aided design (CAD) technology has improved the precision and personalization of scaffold fabrication. This combination enables the development of highly accurate, patient-specific regenerative constructs.[10]

Advantages of 3D Bioprinting in Periodontal Regeneration

- Precise spatial control during tissue fabrication
- Ability to produce customized patient-specific constructs
- Improved cell survival and viability
- Better tissue integration and regeneration
- Reduced surgical morbidity
- Controlled and sustained release of growth factors
- Closer resemblance to the natural architecture of periodontal tissues

CHALLENGES AND LIMITATIONS

Vascularization Issues

One of the major challenges in tissue engineering is achieving adequate blood supply within large bio printed constructs, which is essential for long-term tissue survival.[3]

Mechanical Weakness

Many hydrogel-based scaffolds lack sufficient mechanical strength, limiting their stability and long-term clinical application.[8]

High Cost

The equipment, biomaterials, and maintenance associated with 3D bioprinting are expensive, which restricts its widespread use.[5,10]

Regulatory Concerns

Clinical application of bio printed tissues requires strict regulatory approval and extensive validation to ensure safety and effectiveness.[1,3,9]

Limited Long-Term Clinical Evidence

Most current studies are limited to laboratory and animal models, and long-term human clinical data are still insufficient.[1.3..9]

FUTURE PERSPECTIVES

Future research in 3D bioprinting is primarily focused on:

- Development of vascularized tissue constructs
- Introduction of smart and responsive biomaterials
- Integration of nanotechnology into scaffold design
- Use of gene-activated bioinks
- Clinical translation and commercialization
- Personalized regenerative therapies

The combined advancements in stem cell biology, nanotechnology, artificial intelligence, and 4D bioprinting are expected to further transform periodontal regeneration and regenerative dentistry in the future.[1,3,9]

CONCLUSION

3D bioprinting has emerged as a groundbreaking advancement in regenerative dentistry and periodontology. Its capability to fabricate customized, biomimetic, and multilayered tissue constructs offers considerable advantages over conventional regenerative techniques. Current research indicates that this technology can effectively support regeneration of alveolar bone, periodontal ligament, cementum, and gingival tissues.

Although several technical and regulatory challenges still need to be addressed, continuous progress in biomaterials, stem cell engineering, and bioprinting technologies is expected to accelerate its clinical application. In the coming years, 3D bioprinting may play a transformative role in personalized periodontal therapy and redefine the future of regenerative dentistry.

REFERENCES

1. Chen H, et al. Advances of 3D bioprinting technology for periodontal tissue regeneration. iScience. 2025;28(6):112532.
2. Gul M, et al. Role of three-dimensional printing in periodontal regeneration. J Indian Soc Periodontol. 2019;23(6):504-510.
3. Zhao F, et al. The 3-dimensional printing for dental tissue regeneration. Front Bioeng Biotechnol. 2024;12:1356580.
4. Almeida ND, et al. 3D bioprinting techniques and bioinks for periodontal tissue regeneration. Biomimetics. 2024;9(8):480.
5. Mohd N, et al. Current advances of three-dimensional bioprinting for dental applications. Int J Mol Sci. 2022;23:11234.
6. Miao G, Liang L, Li W, Ma C, Pan Y, Zhao H, et al. 3D bioprinting of a bioactive composite scaffold for cell delivery in periodontal tissue regeneration. Biomolecules. 2023;13(7):1062.
7. Davidopoulou S, et al. Multidimensional 3D-printed scaffolds and regeneration in periodontal defects. Biomaterials. 2024;15(2):44.
8. Ostrovidov S, et al. Bioprinting and biomaterials for dental alveolar tissue engineering. Front Bioeng Biotechnol. 2023;11:991821.
9. Daghrery A, et al. Advances in 3D printed scaffolds for periodontal regeneration. Curr Oral Health Rep. 2026.
10. Acharya R, et al. 3D bioprinting shaping the future of periodontal tissue regeneration and disease management. Cureus. 2025;17(4):e82432.

AUTHORS: -

Dr. Khushi Khatri
Post Graduate student,
Department of Periodontology & Oral Implantology,
Surendera Dental College and Research Institute
Email : khushikhatri790@gmail.com
Mobile No.: 6375858955

Dr. Rajni Aggarwal
Professor and Head
Department of Periodontology & Oral Implantology
Surendera Dental College and Research Institute
Email : rajniperio@gmail.com
Mobile No.: 9979470144

CORRESPONDING AUTHOR: -

Dr. Khushi Khatri
Post Graduate student,
Department of Periodontology & Oral Implantology,
Surendera Dental College and Research Institute
Email : khushikhatri790@gmail.com
Mobile No.: 6375858955

EMERGING HERBAL ANTI-INFLAMMATORY AGENTS: MECHANISMS OF ACTION, THERAPEUTIC POTENTIAL, AND FUTURE PERSPECTIVES

Dr. Mohit Dadhich, Dr. Harshita Pandey

ABSTRACT

Inflammation is an essential physiological response that protects the body against infection, tissue injury, and harmful stimuli. While acute inflammation is a tightly regulated process that promotes healing and restoration of tissue integrity, persistent or chronic inflammation contributes to the pathogenesis of numerous disorders, including periodontitis, rheumatoid arthritis, cardiovascular diseases, diabetes mellitus, neurodegenerative disorders, and cancer. Conventional anti-inflammatory drugs such as non-steroidal anti-inflammatory drugs (NSAIDs) and corticosteroids remain the cornerstone of inflammation management. However, their long-term use is frequently associated with adverse effects, including gastrointestinal ulceration, renal impairment, hepatotoxicity, cardiovascular complications, immunosuppression, and delayed wound healing. These limitations have stimulated growing interest in herbal medicines as safer and biologically active alternatives.

Medicinal plants contain a wide range of phytochemicals, including polyphenols, flavonoids, terpenoids, alkaloids, and glycosides, that exert anti-inflammatory effects by targeting multiple molecular pathways simultaneously. Unlike conventional drugs that often inhibit a single pathway, herbal compounds regulate inflammatory signaling through suppression of nuclear factor-kappa B (NF-κB), cyclooxygenase (COX), lipoxygenase (LOX), mitogen-activated protein kinase (MAPK), pro-inflammatory cytokines, matrix metalloproteinases (MMPs), and oxidative stress. Among the emerging herbal agents, curcumin, quercetin, resveratrol, epigallocatechin gallate (EGCG), Boswellia serrata, Aloe vera, ginger, neem, licorice, garlic, and Ashwagandha have demonstrated promising anti-inflammatory, antioxidant, antimicrobial, and immunomodulatory properties in both experimental and clinical studies.

Recent advances in nanotechnology, local drug delivery systems, phytosome formulations, hydrogels, and biodegradable biomaterials have further enhanced the therapeutic potential of these herbal compounds by improving their stability, bioavailability, and targeted tissue delivery. Such innovations are particularly relevant in periodontology, where sustained local drug release may improve periodontal healing while minimizing systemic adverse effects.

This review discusses the biological basis of inflammation, the molecular mechanisms through which emerging herbal anti-inflammatory agents exert their therapeutic effects, their advantages and limitations compared with conventional drugs, current clinical applications, and future research directions. Although available evidence supports the potential of these phytochemicals as adjuncts in inflammatory disease management, further large-scale randomized controlled trials and standardized formulations are required before their routine integration into evidence-based clinical practice.

KEYWORDS

Herbal medicine, Anti-inflammatory agents, Phytochemicals, NF-κB, Cytokines, Oxidative stress, Periodontitis, Curcumin, Polyphenols.

INTRODUCTION

Inflammation is a highly coordinated biological response that enables the body to defend itself against infection, trauma, toxins, and tissue injury.[1] It represents one of the most fundamental protective mechanisms of the immune system, designed to eliminate harmful stimuli, remove damaged cells, and initiate tissue repair. Under normal circumstances, inflammation is self-limiting and resolves once healing is complete. However, when inflammatory responses become excessive, persistent, or poorly regulated, they contribute to progressive tissue destruction and the development of chronic diseases.[1,2]

The burden of chronic inflammatory diseases has increased dramatically over the past few decades and now represents one of the leading causes of morbidity worldwide. Persistent inflammation plays a central role in the pathogenesis of cardiovascular diseases, metabolic syndrome, diabetes mellitus, rheumatoid arthritis, inflammatory bowel disease, Alzheimer's disease, osteoporosis, and several malignancies.[3] In dentistry, chronic inflammation is the hallmark of periodontal diseases, where a dysregulated host immune response against microbial biofilms leads to progressive destruction of the gingiva, periodontal ligament, cementum, and alveolar bone. Instead of the microorganisms alone causing tissue damage, it is often the exaggerated host inflammatory response that drives disease progression.

At the molecular level, inflammation involves a complex network of immune cells, inflammatory mediators, cytokines, chemokines, prostaglandins, leukotrienes, reactive oxygen species (ROS), and proteolytic enzymes. Signalling pathways such as nuclear factor-kappa B

(NF-κB), mitogen-activated protein kinase (MAPK), Janus kinase/signal transducer and activator of transcription (JAK/STAT), and the arachidonic acid cascade orchestrate these inflammatory events. While these pathways are essential for host defence, their sustained activation results in continuous production of pro-inflammatory mediators that perpetuate tissue injury and delay healing.[1]

For decades, conventional pharmacological agents such as non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids, selective cyclooxygenase-2 (COX-2) inhibitors, and immunosuppressive drugs have been used to suppress inflammatory responses. These medications are effective in reducing pain, swelling, and inflammatory tissue damage; however, prolonged administration is frequently associated with undesirable adverse effects. NSAIDs may cause gastric ulceration, gastrointestinal bleeding, renal dysfunction, and cardiovascular complications, whereas corticosteroids can lead to osteoporosis, adrenal suppression, delayed wound healing, hyperglycaemia, increased susceptibility to infection, and other systemic complications. Furthermore, most synthetic anti-inflammatory drugs primarily target a single inflammatory pathway, whereas chronic inflammatory diseases often involve multiple overlapping molecular mechanisms.[3]

These limitations have encouraged researchers to explore naturally occurring bioactive compounds capable of modulating inflammation through multiple biological targets simultaneously. Herbal medicines have been used in traditional healthcare systems such as Ayurveda, Traditional Chinese Medicine, and Unani medicine for centuries to manage inflammatory disorders. Advances in phytochemistry and molecular biology have identified numerous plant-derived compounds with significant anti-inflammatory potential. These phytochemicals include flavonoids, polyphenols, terpenoids, alkaloids, saponins, tannins, lignans, and essential oils, many of which exhibit additional antioxidant, antimicrobial, immunomodulatory, and wound-healing properties.

Unlike many synthetic drugs, herbal compounds often regulate inflammation by acting on several interconnected signalling pathways. They suppress activation of NF-κB and MAPK, inhibit cyclooxygenase and lipoxygenase enzymes, reduce the production of inflammatory cytokines such as tumour necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6), decrease oxidative stress, inhibit matrix metalloproteinases responsible for connective tissue degradation, and promote resolution of inflammation. This multitarget mechanism makes herbal agents particularly attractive for chronic inflammatory diseases, including periodontitis, where numerous inflammatory mediators contribute simultaneously to tissue destruction.

Among the wide range of medicinal plants investigated over the last decade, curcumin, quercetin, resveratrol, epigallocatechin-3-gallate (EGCG), Boswellia serrata, Aloe vera, ginger, neem, licorice, garlic, and Ashwagandha have emerged as some of the most promising candidates. Experimental studies have demonstrated that these phytochemicals not only suppress inflammatory signalling but also enhance antioxidant defence, improve collagen synthesis, stimulate angiogenesis, inhibit osteoclast-mediated bone resorption, and promote tissue regeneration. Recent innovations such as nanoparticle-based drug delivery systems, liposomes, phytosomes, biodegradable hydrogels, and local drug delivery devices have further improved their clinical applicability by overcoming limitations related to poor solubility and low bioavailability.[1]

Therefore, a comprehensive understanding of the molecular mechanisms underlying the anti-inflammatory effects of medicinal plants is essential for translating laboratory evidence into clinical practice. This review aims to provide an updated overview of emerging herbal anti-inflammatory agents, their mechanisms of action, therapeutic applications, advantages over conventional anti-inflammatory drugs, current limitations, and future prospects, with particular emphasis on their potential role in managing periodontal and other chronic inflammatory diseases.

CURCUMIN (CURCUMA LONGA)

Curcumin is the principal bioactive polyphenol isolated from the rhizome of Curcuma longa (turmeric), a medicinal plant belonging to the family Zingiberaceae. It possesses a wide range of pharmacological properties, including anti-inflammatory, antioxidant, antimicrobial, and immunomodulatory activities. These properties have made curcumin one of the most extensively studied herbal compounds for the management of chronic inflammatory diseases, including periodontitis.[4]

The anti-inflammatory effects of curcumin are primarily mediated through the inhibition of the NF-κB signaling pathway, a key regulator of inflammation. Under inflammatory conditions, NF-κB stimulates the production of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6, along with inflammatory enzymes including COX-2 and iNOS. Curcumin suppresses NF-κB activation, thereby

reducing the expression of these mediators and limiting inflammatory cell recruitment and tissue destruction. It also inhibits the COX-2 and 5-LOX pathways, leading to decreased synthesis of prostaglandins and leukotrienes, which are responsible for pain, edema, and vascular changes associated with inflammation.

In addition to its anti-inflammatory activity, curcumin acts as a potent antioxidant by scavenging reactive oxygen species (ROS) and enhancing the activity of endogenous antioxidant enzymes such as superoxide dismutase and catalase. It also suppresses matrix metalloproteinases (MMPs), thereby reducing collagen degradation and connective tissue destruction. Furthermore, curcumin inhibits RANKL-mediated osteoclast differentiation, which helps prevent inflammatory bone resorption and supports periodontal tissue preservation.

Several in vitro, animal, and clinical studies have demonstrated the therapeutic potential of curcumin in inflammatory diseases. In periodontology, curcumin has been evaluated as a mouthwash, gel, and local drug delivery agent, where it has shown significant improvements in gingival inflammation, probing pocket depth, clinical attachment level, and bleeding on probing when used as an adjunct to scaling and root planing. Although its clinical application is limited by poor bioavailability, recent advances in nanoparticle- and hydrogel-based delivery systems have considerably enhanced its therapeutic efficacy.[4]

QUERCETIN

Quercetin is a naturally occurring flavonoid found in fruits and vegetables such as onions, apples, berries, grapes, broccoli, and citrus fruits. It is well known for its potent antioxidant, anti-inflammatory, antiviral, and immunomodulatory properties. Due to its ability to regulate multiple inflammatory pathways, quercetin has emerged as a promising phytochemical for the management of chronic inflammatory diseases, including periodontal disease.

Quercetin exerts its anti-inflammatory effects by inhibiting the NF-κB and MAPK signaling pathways, thereby reducing the production of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6. It also suppresses the expression of COX-2 and iNOS, resulting in decreased synthesis of prostaglandins and nitric oxide that contribute to pain, vasodilation, and tissue inflammation.

Additionally, quercetin stabilizes mast cells and inhibits histamine release, further limiting the inflammatory response.[3]

As a potent antioxidant, quercetin scavenges reactive oxygen species (ROS) and enhances endogenous antioxidant defense, thereby preventing oxidative stress-induced tissue damage. It also inhibits matrix metalloproteinases (MMPs), reducing extracellular matrix degradation and protecting periodontal tissues. Experimental studies have demonstrated that quercetin decreases inflammatory cell infiltration and supports tissue healing. In periodontology, it has shown promising results in reducing periodontal inflammation and alveolar bone loss. However, its poor water solubility and limited bioavailability remain challenges, which are being addressed through nanoformulations and advanced drug delivery systems.

RESVERATROL

Resveratrol is a naturally occurring polyphenol present in grapes, berries, peanuts, and red wine. It has gained significant attention due to its anti-inflammatory, antioxidant, cardioprotective, and bone-protective properties. Recent studies suggest that resveratrol may serve as a beneficial adjunct in the management of chronic inflammatory and periodontal diseases.

The anti-inflammatory activity of resveratrol is primarily mediated through inhibition of the NF-κB pathway and activation of Sirtuin-1 (SIRT1), which regulates inflammatory and oxidative stress responses. By suppressing NF-κB activation, resveratrol reduces the production of TNF-α, IL-1β, IL-6, COX-2, and iNOS, thereby limiting inflammatory signaling. It also inhibits osteoclast differentiation by downregulating RANKL expression, reducing inflammation-associated bone resorption.[3]

Resveratrol possesses strong antioxidant activity by neutralizing reactive oxygen species and enhancing the activity of antioxidant enzymes. It further suppresses matrix metalloproteinase activity, thereby preventing collagen degradation and connective tissue destruction. Experimental and clinical studies have demonstrated its ability to reduce inflammatory markers and improve tissue healing. In periodontology, resveratrol has shown potential in controlling periodontal inflammation and preserving alveolar bone. However, its rapid metabolism and low bioavailability remain limitations that may be overcome through novel delivery systems.

EPIGALLOCATECHIN-3-GALLATE (EGCG)

Epigallocatechin-3-gallate (EGCG) is the major catechin found in green tea (Camellia sinensis) and is considered one of the most biologically active plant-derived polyphenols. It exhibits potent anti-inflammatory, antioxidant,

antimicrobial, and anticancer properties, making it a promising therapeutic agent for inflammatory diseases.

EGCG suppresses inflammation primarily by inhibiting the NF-κB and MAPK signaling pathways, leading to reduced production of TNF-α, IL-1β, IL-6, and other inflammatory mediators. It also inhibits COX-2 activity, thereby reducing prostaglandin synthesis. In addition, EGCG suppresses matrix metalloproteinases, particularly MMP-2 and MMP-9, helping to preserve connective tissue integrity and prevent periodontal destruction.

The potent antioxidant activity of EGCG protects tissues by scavenging reactive oxygen species and reducing oxidative stress. It also exhibits antimicrobial activity against key periodontal pathogens, contributing to reduced bacterial load and inflammation. Clinical studies have shown that green tea polyphenols improve gingival health, reduce probing pocket depth, and decrease inflammatory biomarkers when used as an adjunct to conventional periodontal therapy. Although its stability and bioavailability remain concerns, recent nanoformulations have improved its therapeutic potential.

BOSWELLIA SERRATA

Boswellia serrata, commonly known as Indian frankincense, is an important medicinal plant used in Ayurvedic medicine for the treatment of chronic inflammatory disorders. Its therapeutic effects are mainly attributed to boswellic acids, particularly 3-O-acetyl-11-keto-β-boswellic acid (AKBA), which exhibit potent anti-inflammatory activity.

Unlike conventional NSAIDs that mainly inhibit cyclooxygenase, boswellic acids selectively inhibit the 5-lipoxygenase (5-LOX) pathway, resulting in reduced leukotriene synthesis. Since leukotrienes play a major role in leukocyte recruitment and chronic inflammation, their inhibition significantly decreases inflammatory cell infiltration and tissue damage. Boswellia also suppresses the production of TNF-α, IL-1β, and IL-6, further limiting inflammatory responses.[5]

In addition to its anti-inflammatory effects, Boswellia exhibits antioxidant properties that reduce oxidative stress and support tissue healing. Experimental studies have demonstrated its ability to decrease edema, inflammatory cell infiltration, and bone destruction. Emerging evidence suggests that Boswellia may also have beneficial effects in periodontal therapy by reducing gingival inflammation and promoting tissue repair. Further clinical studies are required to establish standardized therapeutic protocols.

ALOE VERA

Aloe vera (Aloe barbadensis Miller) is a medicinal plant widely recognized for its wound-healing, antimicrobial, antioxidant, and anti-inflammatory properties. Its therapeutic effects are mainly attributed to bioactive compounds such as acemannan, anthraquinones, flavonoids, vitamins, and polysaccharides, which promote tissue repair and modulate inflammatory responses.

Aloe vera exerts its anti-inflammatory activity by inhibiting the COX-2 pathway, thereby reducing prostaglandin synthesis. It also suppresses the production of pro-inflammatory cytokines including TNF-α, IL-1β, and IL-6, leading to reduced inflammatory cell infiltration and tissue damage. Additionally, its antioxidant activity neutralizes reactive oxygen species, limiting oxidative stress and promoting tissue healing.

Another important property of Aloe vera is its ability to stimulate fibroblast proliferation, collagen synthesis, and angiogenesis, thereby accelerating wound healing. In periodontology, Aloe vera has been evaluated as a gel, mouthwash, and local drug delivery agent, demonstrating improvements in gingival inflammation, plaque accumulation, and periodontal healing when used as an adjunct to scaling and root planing. Its excellent biocompatibility and minimal adverse effects make it a promising herbal alternative for periodontal therapy.[6]

GINGER (ZINGIBER OFFICINALE)

Zingiber officinale, commonly known as ginger, is one of the oldest medicinal herbs used in traditional medicine for the treatment of inflammatory and gastrointestinal disorders. The major bioactive constituents of ginger include 6-gingerol, 6-shogaol, zingerone, and paradols, which possess significant anti-inflammatory, antioxidant, and antimicrobial properties. These compounds have attracted considerable attention for their potential role in managing chronic inflammatory diseases.

The anti-inflammatory effects of ginger are mainly mediated through inhibition of the cyclooxygenase (COX) and 5-lipoxygenase (5-LOX) pathways, resulting in reduced production of prostaglandins and leukotrienes. Ginger also suppresses activation of the NF-κB signaling pathway, thereby decreasing the release of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6. Additionally, its antioxidant activity scavenges reactive oxygen species (ROS), reducing oxidative stress and preventing further tissue injury.

Several experimental studies have demonstrated that ginger

reduces inflammatory cell infiltration, protects connective tissue, and promotes healing. In periodontology, ginger extracts have shown promising antimicrobial activity against periodontal pathogens and have been reported to improve gingival inflammation when used as an adjunct to conventional periodontal therapy. Although further clinical trials are required, ginger represents a promising natural anti-inflammatory agent with a favorable safety profile.[1]

NEEM (AZADIRACHTA INDICA)

Azadirachta indica (Neem) is a well-known medicinal plant widely used in Ayurveda because of its antimicrobial, anti-inflammatory, antioxidant, and immunomodulatory properties. Various parts of the plant, including its leaves, bark, seeds, and oil, contain biologically active compounds such as nimbidin, azadirachtin, quercetin, and nimbolide, which contribute to its therapeutic effects.

Neem exerts its anti-inflammatory action primarily by inhibiting the NF-κB pathway, leading to reduced production of inflammatory cytokines including TNF-α, IL-1β, and IL-6. It also suppresses the expression of COX-2 and inducible nitric oxide synthase (iNOS), thereby reducing prostaglandin and nitric oxide production. In addition, its antioxidant properties help neutralize reactive oxygen species, limiting oxidative stress and tissue destruction.

Apart from modulating inflammation, neem exhibits broad-spectrum antimicrobial activity against several oral pathogens, making it particularly beneficial in periodontal therapy. Clinical studies have shown that neem-based mouthwashes and gels effectively reduce plaque accumulation, gingival inflammation, and bleeding on probing. Its combined antimicrobial and anti-inflammatory actions make neem a valuable adjunct in the prevention and management of periodontal diseases.[6]

LICORICE (GLYCYRRHIZA GLABRA)

Glycyrrhiza glabra, commonly known as licorice, has been used for centuries in traditional medicine to treat inflammatory, respiratory, and gastrointestinal disorders. The principal active constituents, glycyrrhizin and glabridin, possess potent anti-inflammatory, antioxidant, antimicrobial, and immunomodulatory properties.

Licorice suppresses inflammation by inhibiting activation of the NF-κB signaling pathway, thereby reducing the release of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6. It also inhibits COX-2 expression and decreases prostaglandin synthesis, resulting in reduced pain and inflammation. Furthermore, licorice protects tissues against oxidative stress by scavenging free radicals and enhancing endogenous antioxidant defense mechanisms.

Studies have demonstrated that licorice exhibits antimicrobial activity against important periodontal pathogens while also reducing inflammatory tissue damage. In dentistry, licorice has been incorporated into mouthwashes, gels, and chewing gums, where it has shown beneficial effects in reducing plaque formation, gingival inflammation, and periodontal disease progression. However, prolonged excessive consumption should be avoided because glycyrrhizin may produce systemic adverse effects such as hypertension and hypokalemia.

ASHWAGANDHA (WITHANIA SOMNIFERA)

Withania somnifera (Ashwagandha) is an important adaptogenic herb extensively used in Ayurvedic medicine for promoting general health and managing stress-related disorders. Its pharmacological activity is mainly attributed to withanolides, steroidal lactones that exhibit anti-inflammatory, antioxidant, immunomodulatory, and regenerative properties.

Ashwagandha reduces inflammation by suppressing activation of the NF-κB and MAPK signaling pathways, thereby decreasing the production of inflammatory mediators including TNF-α, IL-1β, IL-6, COX-2, and iNOS. In addition, it enhances the activity of antioxidant enzymes such as superoxide dismutase, catalase, and glutathione peroxidase, protecting tissues against oxidative stress and limiting inflammation-induced cellular damage.[3]

Experimental studies have shown that Ashwagandha reduces inflammatory cell infiltration, promotes tissue repair, and supports bone health by regulating osteoclast activity. Although evidence in periodontology is still emerging, its anti-inflammatory and antioxidant properties suggest potential applications as an adjunctive therapeutic agent in the management of periodontal disease. Further well-designed clinical trials are required to establish its long-term efficacy and safety in dental practice.

ADVANTAGES OF HERBAL ANTI-INFLAMMATORY AGENTS OVER CONVENTIONAL DRUGS

- **Multi-target mechanism of action**: Unlike conventional anti-inflammatory drugs that usually target a single pathway, herbal compounds modulate multiple inflammatory pathways simultaneously, including NF-κB, MAPK, COX-2, LOX, and pro-inflammatory cytokines.
- **Potent antioxidant activity**: Most herbal agents neutralize reactive oxygen species (ROS) and enhance endogenous antioxidant defense, thereby reducing oxidative stress and preventing tissue damage.

- **Antimicrobial properties**: Several phytochemicals, such as neem, green tea, and garlic, possess antimicrobial activity against pathogenic microorganisms, providing additional therapeutic benefits in infectious inflammatory conditions like periodontitis.
- **Immunomodulatory effects**: Herbal compounds help regulate the host immune response by balancing pro-inflammatory and anti-inflammatory cytokines rather than causing generalized immunosuppression.
- **Reduced adverse effects**: Compared with long-term NSAID and corticosteroid therapy, herbal medicines are generally associated with fewer gastrointestinal, renal, hepatic, and cardiovascular complications when used appropriately.
- **Promotion of tissue healing**: Many herbal agents stimulate fibroblast proliferation, collagen synthesis, angiogenesis, and bone regeneration, thereby accelerating wound healing and tissue repair.
- **Suitable for adjunctive therapy**: Herbal formulations can be effectively combined with conventional treatments such as scaling and root planing or systemic medications to improve therapeutic outcomes.
- **Cost-effective and readily available**: Many medicinal plants are easily available, relatively inexpensive, and have a long history of traditional use.

LIMITATIONS AND CHALLENGES

- **Poor bioavailability**: Many phytochemicals, including curcumin and resveratrol, exhibit poor aqueous solubility, rapid metabolism, and limited systemic absorption.
- **Lack of standardization**: Variations in plant source, cultivation conditions, extraction methods, and formulation result in inconsistent concentrations of active constituents.
- **Limited high-quality clinical evidence**: Although numerous in vitro and animal studies are available, large multicenter randomized controlled clinical trials remain limited.
- **Dose variability**: The optimal therapeutic dose and duration of treatment have not been established for many herbal compounds.
- **Drug–herb interactions**: Certain herbal products may interact with prescription medications, potentially affecting their efficacy or safety.
- **Quality control issues**: Contamination, adulteration, and variability in herbal preparations remain significant concerns.
- **Regulatory limitations**: Many herbal products lack uniform regulatory guidelines regarding manufacturing standards, quality assurance, and clinical evaluation.
- **Limited long-term safety data**: The safety of prolonged use of several herbal agents has not been adequately established through long-term clinical studies.

FUTURE PERSPECTIVES

The future of herbal anti-inflammatory therapy is promising, driven by advances in phytochemistry, molecular biology, and drug delivery technologies. Although many herbal compounds have demonstrated significant anti-inflammatory potential in experimental studies, further research is required to establish their routine clinical use. The following areas represent the major future directions:

- **Development of novel drug delivery systems**: Nanoparticles, liposomes, phytosomes, hydrogels, and biodegradable scaffolds can improve the bioavailability, stability, and targeted delivery of herbal compounds.
- **Local drug delivery in periodontology**: Incorporation of herbal agents into gels, chips, fibers, microspheres, and injectable hydrogels may provide sustained drug release within periodontal pockets while minimizing systemic side effects.[7]
- **Standardization of herbal formulations**: Establishing standardized extraction methods, dosage, and quality control measures is essential to ensure consistent therapeutic efficacy and safety.
- **Large-scale randomized clinical trials**: More multicenter, well-designed clinical studies with long-term follow-up are needed to validate the efficacy and safety of herbal anti-inflammatory agents.
- **Combination therapy**: Herbal compounds may be used in combination with conventional anti-inflammatory drugs or antibiotics to enhance therapeutic outcomes while reducing drug dosage and adverse effects.
- **Identification of novel phytochemicals**: Continued exploration of medicinal plants may lead to the discovery of new bioactive compounds with superior anti-inflammatory activity.
- **Artificial intelligence and computational drug discovery**: AI-based phytochemical screening, molecular docking, and network pharmacology can accelerate the identification of promising herbal molecules and their molecular targets.
- **Personalized herbal medicine**: Advances in pharmacogenomics and precision medicine may enable individualized herbal therapies based on a patient's genetic profile and inflammatory status.[8]
- **Expansion of clinical applications**: Future research may establish the role of herbal anti-inflammatory agents in implant dentistry, peri-implant diseases, oral mucosal disorders, and regenerative periodontal therapy.

CONCLUSION

Inflammation is a complex biological process that plays a central role in the pathogenesis of numerous chronic diseases, including periodontitis. Although conventional anti-inflammatory drugs remain effective, their long-term use is often limited by undesirable adverse effects. This has led to increasing interest in plant-derived bioactive compounds as safer and more versatile therapeutic alternatives.

Emerging herbal anti-inflammatory agents such as curcumin, quercetin, resveratrol, epigallocatechin-3-gallate (EGCG), Boswellia serrata, Aloe vera, ginger, neem, licorice, and Ashwagandha* exhibit significant therapeutic potential by targeting multiple inflammatory pathways. These phytochemicals suppress key mediators of inflammation, including NF-κB, COX, LOX, MAPK, pro-inflammatory cytokines, and matrix metalloproteinases, while also providing antioxidant, antimicrobial, and immunomodulatory benefits. Their multitarget mechanism of action makes them particularly attractive for the management of chronic inflammatory conditions where inflammation, oxidative stress, and microbial infection coexist.

Despite encouraging preclinical and clinical evidence, challenges such as poor bioavailability, lack of formulation standardization, and limited high-quality clinical trials continue to restrict their widespread clinical application. Advances in nanotechnology, local drug delivery systems, and standardized herbal formulations are expected to overcome many of these limitations and enhance therapeutic efficacy.

Overall, herbal anti-inflammatory agents represent a promising adjunct to conventional therapy rather than a complete replacement. Continued translational research and robust clinical evidence will be essential to establish their role in evidence-based medicine and periodontal practice, ultimately contributing to safer, more effective, and patient-centered management of inflammatory diseases.

REFERENCES

1. Newman MG, Takei HH, Klokkevold PR, Carranza FA. Carranza's Clinical Periodontology. 14th ed. St. Louis: Elsevier; 2024.
2. Kumar V, Abbas AK, Aster JC. Robbins and Cotran Pathologic Basis of Disease. 11th ed. Philadelphia: Elsevier; 2023.
3. Rang HP, Ritter JM, Flower RJ, Henderson G. Rang & Dale's Pharmacology. 10th ed. London: Elsevier; 2023.
4. Brunton LL, Hilal-Dandan R, Knollmann BC. Goodman & Gilman's The Pharmacological Basis of Therapeutics. 14th ed. New York: McGraw-Hill; 2023.
5. Medzhitov R. Origin and physiological roles of inflammation. Nature. 2008;454(7203):428-35.
6. Chen L, Deng H, Cui H, Fang J, Zuo Z, Deng J, et al. Inflammatory responses and inflammation-associated diseases. Oncotarget. 2018;9(6):7204-7218.
7. Gupta SC, Patchva S, Aggarwal BB. Therapeutic roles of curcumin: lessons learned from clinical trials. AAPS J. 2013;15(1):195-218.
8. Kunnumakkara AB, Bordoloi D, Padmavathi G, Monisha J, Roy NK, Prasad S, et al. Curcumin, the golden nutraceutical: multitargeting for multiple chronic diseases. Br J Pharmacol. 2017;174(11):1325-1348.
9. Li Y, Yao J, Han C, Yang J, Chaudhry MT, Wang S, et al. Quercetin, inflammation and immunity. Nutrients. 2016;8(3):167.
10. Salehi B, Mishra AP, Nigam M, Sener B, Kilic M, Sharifi-Rad M, et al. Resveratrol: A double-edged sword in health benefits. Biomedicines. 2018;6(3):91.

AUTHORS: -

Dr Mohit Dadhich
Senior Lecturer
Department of Pharmacology
Govt. Medical College, Hanumangarh
Email id: -
Mobile No: -

Dr Harshita Pandey
Senior Lecturer
Department of Microbiology
Govt. Medical College, Hanumangarh
Email id: -
Mobile No: -

CORRESPONDING AUTHOR: -

Dr Mohit Dadhich
Senior Lecturer
Department of Pharmacology
Govt. Medical College, Hanumangarh
Email id: -
Mobile No: -

ARTIFICIAL INTELLIGENCE IN PERIODONTOLOGY: TRANSFORMING DIAGNOSIS, TREATMENT PLANNING, AND PERSONALIZED PERIODONTAL CARE

Dr Ruchika, Dr Rajni Aggarwal, Dr Archana Bhatia

ABSTRACT

Artificial Intelligence (AI) has emerged as one of the most transformative technologies in healthcare, offering unprecedented opportunities to enhance diagnostic precision, clinical decision-making, and patient management. In periodontology, where disease progression is multifactorial and influenced by biological, behavioral, and environmental factors, AI provides a powerful platform for integrating complex datasets into meaningful clinical insights. Recent advances in machine learning, deep learning, natural language processing, and computer vision have facilitated the development of intelligent systems capable of detecting periodontal disease, predicting treatment outcomes, assessing implant prognosis, and supporting personalized care strategies. The integration of AI with digital dentistry, radiographic interpretation, genomic analysis, and telehealth services has further expanded its clinical utility. This article reviews the current applications of AI in periodontology, explores emerging innovations, discusses ethical and practical challenges, and highlights future directions for the development of intelligent periodontal healthcare systems.

KEYWORDS

Artificial Intelligence, Periodontology, Machine Learning, Deep Learning, Precision Dentistry, Periodontal Disease, Dental Implants

INTRODUCTION

Periodontal diseases remain among the most prevalent chronic inflammatory conditions affecting populations worldwide. Despite significant advances in diagnostic methodologies and therapeutic approaches, early identification of disease progression and accurate prediction of treatment outcomes continue to challenge clinicians. Traditional periodontal assessment relies heavily on clinical measurements, radiographic interpretation, and practitioner experience, which may introduce variability and limit predictive capabilities[1].

Artificial Intelligence represents a paradigm shift in healthcare by enabling machines to mimic human cognitive functions such as learning, reasoning, pattern recognition, and decision-making. Through sophisticated algorithms capable of analyzing large volumes of clinical and biological data, AI has demonstrated remarkable potential in improving diagnostic accuracy and optimizing patient-specific treatment strategies.

The increasing digitization of dental records, widespread use of cone-beam computed tomography (CBCT), digital radiography, intraoral scanners, and electronic health records has created a fertile environment for AI applications in periodontology[2]. These developments are paving the way for precision periodontal medicine, where prevention and treatment can be tailored to the unique characteristics of individual patients.

FUNDAMENTALS OF ARTIFICIAL INTELLIGENCE

Artificial Intelligence encompasses a broad spectrum of computational technologies designed to perform tasks that traditionally require human intelligence. Key components include:

Machine Learning

Machine Learning (ML) enables computers to learn from data without explicit programming. By identifying hidden patterns within datasets, ML algorithms can generate predictive models that support clinical decision-making[3].

Deep Learning

Deep Learning is a subset of machine learning that utilizes artificial neural networks with multiple layers. These systems are particularly effective in image recognition and radiographic analysis, making them highly relevant to periodontal diagnostics.

Natural Language Processing

Natural Language Processing (NLP) allows computers to interpret and analyze human language. In dentistry, NLP can extract clinically relevant information from electronic health records and research databases.

Computer Vision

Computer vision enables machines to analyze visual information from radiographs, photographs, and intraoral scans. This technology forms the basis of many AI-driven diagnostic tools in periodontology.

APPLICATIONS OF ARTIFICIAL INTELLIGENCE IN PERIODONTAL DIAGNOSIS

Automated Detection of Periodontal Bone Loss

One of the most extensively studied applications of AI in periodontology involves the automated assessment of

alveolar bone loss using radiographic images. Deep learning algorithms can identify anatomical landmarks, quantify bone levels, and classify disease severity with impressive accuracy[4].

AI-assisted radiographic analysis offers several advantages:

- Reduction of observer variability
- Improved diagnostic consistency
- Faster interpretation of large datasets
- Enhanced early disease detection

Recent studies have demonstrated that convolutional neural networks can identify periodontal bone defects with accuracy levels approaching those of experienced specialists.

Classification of Periodontal Disease

AI systems can analyze multiple clinical parameters simultaneously, including:

- Probing pocket depth
- Clinical attachment loss
- Bleeding on probing
- Plaque scores
- Radiographic findings

By integrating these variables, machine learning models can classify periodontal disease stages and grades according to contemporary periodontal classification systems, thereby supporting evidence-based treatment planning.

Early Disease Prediction

A significant advantage of AI lies in its predictive capability. Rather than merely identifying existing disease, AI models can estimate an individual's future risk of periodontal breakdown by evaluating demographic, behavioral, genetic, microbial, and systemic health factors.

Predictive algorithms may help clinicians identify high-risk patients before substantial tissue destruction occurs, facilitating preventive intervention strategies[5].

ARTIFICIAL INTELLIGENCE IN PERIODONTAL TREATMENT PLANNING

Personalized Treatment Approaches

Periodontal disease exhibits considerable heterogeneity among patients. AI enables the development of individualized treatment plans based on patient-specific risk profiles.

Factors incorporated into AI-driven treatment recommendations may include:

- Smoking status
- Diabetes control
- Genetic susceptibility
- Oral hygiene behavior
- Microbial composition
- Previous treatment response

Such precision-based approaches can improve therapeutic efficiency while minimizing unnecessary interventions.

Prognostic Assessment

Predicting the long-term survival of teeth affected by periodontal disease remains a complex clinical challenge. AI algorithms can evaluate multiple prognostic variables simultaneously and generate objective predictions regarding tooth retention, disease recurrence, and treatment success[6].

These predictive models assist clinicians in making informed decisions regarding periodontal therapy, regenerative procedures, or extraction.

Artificial Intelligence and Periodontal Regeneration

Regenerative periodontal therapy requires accurate assessment of defect morphology and healing potential. AI systems can support regenerative procedures through:

- Automated defect classification
- Evaluation of regenerative outcomes
- Prediction of healing responses
- Selection of optimal biomaterials

Future AI platforms may integrate radiographic, molecular, and clinical data to identify patients most likely to benefit from regenerative interventions.

ARTIFICIAL INTELLIGENCE IN IMPLANTOLOGY AND PERI-IMPLANT CARE

Implant Treatment Planning

Successful implant therapy requires comprehensive evaluation of bone quality, bone quantity, anatomical structures, and prosthetic considerations. AI-powered systems can analyze CBCT scans to assist clinicians in:

- Implant site selection
- Implant size determination
- Assessment of anatomical limitations
- Surgical risk evaluation

Prediction of Implant Success

Machine learning algorithms have demonstrated potential in predicting implant survival by analyzing factors such as:

- Bone density
- Systemic health conditions
- Smoking habits
- Implant characteristics

- Oral hygiene practices

These predictive models contribute to more reliable treatment outcomes and improved patient counseling.

Detection of Peri-Implant Diseases

AI-based image analysis systems are increasingly being investigated for the detection of peri-implant mucositis and peri-implantitis. Automated identification of crestal bone loss and peri-implant inflammatory changes may facilitate earlier intervention and improve implant longevity.

Integration of Artificial Intelligence with Precision Periodontology

The concept of precision periodontology seeks to provide individualized care based on a patient's unique biological profile. AI serves as a critical enabler of this approach by integrating diverse sources of information, including:

- Clinical measurements
- Radiographic findings
- Salivary biomarkers
- Genetic profiles
- Microbiome analysis
- Systemic health data

Through comprehensive data integration, AI can generate highly personalized diagnostic and therapeutic recommendations.

EMERGING INNOVATIONS AND FUTURE DIRECTIONS

Digital Twins in Periodontology

One of the most promising future applications involves the development of digital twins—virtual replicas of patients that simulate disease progression and treatment responses. These models may allow clinicians to evaluate multiple treatment scenarios before implementing therapy[7].

AI-Powered Salivary Diagnostics

Advances in biosensor technology and machine learning are facilitating the development of AI-driven salivary diagnostic systems capable of detecting inflammatory biomarkers associated with periodontal disease.

Smart Periodontal Monitoring Systems

Wearable and mobile health technologies integrated with AI may enable continuous monitoring of oral health behaviors and treatment compliance. Such systems could provide real-time feedback to both patients and clinicians.

Generative Artificial Intelligence

Generative AI tools are increasingly being explored for clinical documentation, patient education, literature synthesis, and research support. When used responsibly and under professional supervision, these systems may significantly improve workflow efficiency within periodontal practice[8].

Challenges and Ethical Considerations

Despite its considerable potential, several challenges must be addressed before widespread clinical adoption of AI in periodontology.

Data Quality and Standardization

AI models require large, high-quality datasets for training and validation. Variability in clinical recording methods and imaging protocols may affect model performance.

Algorithm Transparency

Many deep learning systems function as "black boxes," making it difficult to understand the rationale behind their decisions. Greater transparency is necessary to promote clinician trust and regulatory acceptance.

Data Privacy and Security

The use of patient data raises important concerns regarding confidentiality, informed consent, and cybersecurity. Robust governance frameworks are essential to ensure ethical implementation.

Human Oversight

AI should augment rather than replace clinical expertise. Final diagnostic and treatment decisions must remain under the supervision of qualified dental professionals.

CONCLUSION

Artificial Intelligence is redefining the landscape of modern periodontology by enhancing diagnostic accuracy, facilitating personalized treatment planning, improving prognostic assessment, and supporting preventive care strategies. The convergence of AI with digital dentistry, biomarker research, and precision medicine has the potential to revolutionize periodontal healthcare in the coming decades. Although challenges related to ethics, data quality, and clinical integration remain, the future of periodontology is increasingly intertwined with intelligent

technologies. As research continues to advance, AI is expected to become an indispensable component of evidence-based periodontal practice, ultimately improving patient outcomes and transforming the delivery of oral healthcare.

Conflict of Interest

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REFERENCES

1. Asokan S, Emmadi P, Chamundeswari R. Effect of oil on plaque-induced gingivitis: a randomized, controlled, triple-blind study. Indian J Dent Res. 2009;20(1):47–51.
2. Peedikayil FC, Sreenivasan P, Narayanan A. Effect of coconut oil in plaque-related gingivitis: a preliminary report. Niger Med J. 2015;56(2):143–147.
3. Shino B, Peedikayil FC, Jaiprakash SR, Bijapur GA, Kottayi S, Jose D. Comparison of antimicrobial activity of chlorhexidine, coconut oil, probiotics, and ketoconazole on Candida albicans: an in vitro study. Scientifica. 2016;2016:7061587.
4. Asokan S, Rathan J, Muthu MS, Rathna PV, Emmadi P, Raghuraman R. Effect of oil pulling on Streptococcus mutans count in plaque and saliva using sesame oil. J Indian Soc Pedod Prev Dent. 2008;26(2):82–87.
5. Thaweboon S, Nakaparksin J, Thaweboon B. Effect of oil pulling on oral microorganisms in biofilm models. Asia J Public Health. 2011;2(2):62–66.
6. Zanatta FB, Antoniazzi RP, Rösing CK. Staining and calculus formation after 0.12% chlorhexidine rinses in plaque-free and plaque-covered surfaces: a randomized trial. J Appl Oral Sci. 2010;18(5):515–521.
7. Peng TR, Cheng HY, Wu TW, et al. Effectiveness of oil pulling for improving oral health: a meta-analysis. Healthcare (Basel). 2022;10(10):1991.
8. Naseem M, Khiyani MF, Nauman H, Zafar MS, Shah AH, Khalil HS. Oil pulling and importance of traditional medicine in oral health maintenance. Int J Health Sci (Qassim). 2017;11(4):65–70.

AUTHORS: -

Dr Ruchika
Post Graduate Student
Department of Periodontology & Oral Implantology
Surendera Dental College & Research Institute
Email id: ruchikakataria16@gmail.com
Mobile No: 9462907457

Dr. Rajni Aggarwal
Professor and Head
Department of Periodontology & Oral Implantology
Surendera Dental College and Research Institute
Email : rajniperio@gmail.com
Mobile No: 9979470144

Dr. Archana Bhatia
Professor
Department of Periodontology & Oral Implantology
Surendera Dental College & Research Institute
Email id: bhatiaarchana16@yahoo.com
Mobile No: 9888370446

CORRESPONDING AUTHOR: -

Dr Ruchika
Post Graduate Student
Department of Periodontology & Oral Implantology
Surendera Dental College & Research Institute
Email id: ruchikakataria16@gmail.com
Mobile No: 9462907457

STRESS AND PERIODONTIUM: UNDERSTANDING THE MIND–GUM CONNECTION

Dr. Saurav Aggarwal, Dr. Mohit Dadhich, Dr. Harshita Pandey, Dr. Khushi Khatri

ABSTRACT

Stress is a normal physiological response to challenging situations. However, when stress becomes prolonged or poorly managed, it can affect several systems of the body, including the immune, endocrine, and nervous systems. The periodontium is also influenced by these changes. Periodontitis is primarily initiated by a dysbiotic microbial biofilm, but its progression and severity are strongly influenced by the host response and various modifying factors. Psychological stress may act as one such factor. It can influence periodontal health through activation of the hypothalamic–pituitary–adrenal axis and the sympathetic nervous system, alterations in cortisol and other stress mediators, changes in immune regulation, impaired healing, and behavioural changes such as poor oral hygiene and smoking. Several clinical studies and systematic reviews have reported an association between psychological stress and periodontal disease, although the relationship is complex and affected by multiple confounding factors. Understanding this relationship is important for dental professionals because successful periodontal therapy involves not only control of dental plaque and periodontal inflammation but also recognition of patient-related behavioural and psychological factors. Stress assessment and appropriate counselling may therefore be useful components of comprehensive periodontal care.

KEYWORDS

Psychological stress, periodontium, periodontitis, cortisol, inflammation, psychoneuroimmunology, periodontal health.

INTRODUCTION

The mouth is not an isolated part of the body. Oral tissues continuously interact with systemic physiological, behavioural, and environmental factors. Among these factors, psychological stress has received increasing attention in periodontal research.

Stress can be described as the body's response to a perceived physical or psychological challenge. A short period of stress may help an individual respond to an immediate threat. In contrast, persistent stress may result in prolonged activation of the body's stress-response systems. This can influence hormonal balance, immune function, inflammation, sleep, nutrition, and health-related behaviours.

Periodontitis is a chronic inflammatory disease associated with dysbiotic subgingival microbial communities and an altered host inflammatory response. Although bacterial biofilm remains the essential initiating factor, the clinical expression of periodontal disease varies considerably between individuals. Factors such as smoking, diabetes, genetic susceptibility, age, socioeconomic conditions, and psychological stress can modify the host response and disease progression.[1]

Research over the past several decades has suggested a relationship between psychological stress and periodontal disease. A systematic review by Peruzzo et al. reported that most of the studies included in their analysis showed a positive relationship between psychosocial stress and periodontal disease, although the authors emphasized the methodological limitations of the available evidence.[1] More recent systematic reviews have also reported an association between psychological stress, anxiety, cortisol levels, and periodontitis.[2,3]

Therefore, stress should not be considered an independent primary cause of periodontitis. Rather, it may influence the biological and behavioural environment in which periodontal disease develops and progresses.

HOW DOES STRESS AFFECT THE BODY?

The response to stress involves a coordinated interaction between the nervous, endocrine, and immune systems. Two major pathways are particularly important:

1. Hypothalamic–pituitary–adrenal (HPA) axis
2. Sympathetic–adrenal–medullary (SAM) system

When an individual experiences stress, the hypothalamus stimulates the release of corticotropin-releasing hormone. This subsequently promotes adrenocorticotropic hormone secretion from the pituitary gland, resulting in the release of cortisol from the adrenal cortex.

At the same time, sympathetic stimulation leads to the release of catecholamines such as adrenaline and noradrenaline.

These mediators are useful during an acute stress response. However, prolonged activation may disturb normal immune regulation and inflammatory control. Since periodontal disease is strongly influenced by the interaction between microorganisms and the host immune response, these systemic changes may affect periodontal tissues.

This interaction between psychological processes, the nervous system, endocrine responses, and immunity is

commonly discussed under the concept of psychoneuroimmunology.[4]

POSSIBLE MECHANISMS LINKING STRESS AND THE PERIODONTIUM

1. Increased cortisol levels

Cortisol is one of the major hormones involved in the stress response. Chronic psychological stress can alter the normal regulation of the HPA axis and influence cortisol secretion.

Cortisol has important immunoregulatory effects. Persistent alterations in cortisol exposure may interfere with normal immune surveillance and inflammatory regulation. In periodontal tissues, this may modify the host response to the microbial challenge.

Several studies have investigated salivary cortisol as a possible biological marker of stress. A meta-analysis found evidence of altered cortisol levels in patients with periodontitis, although the authors emphasized that the available studies were observational and that several confounding factors must be considered.[5]

A more recent meta-analysis published in 2025 also reported higher salivary cortisol levels in periodontitis patients experiencing stress, although the evidence was considered to have limitations.[6]

2. Alteration of immune response

The periodontal tissues are constantly exposed to microorganisms. In health, the immune system maintains a controlled response that protects the tissues without causing unnecessary damage.
During chronic stress, neuroendocrine mediators may modify immune-cell activity and cytokine production. This can disturb the balance between protective and destructive inflammatory responses.

An altered immune response may result in increased production of inflammatory mediators involved in periodontal tissue destruction. These mediators can contribute to connective tissue breakdown and alveolar bone resorption when inflammation becomes persistent.

Thus, stress may influence periodontal destruction indirectly by modifying the host response rather than directly damaging periodontal tissues.

3. Effect on periodontal inflammation

Periodontitis is characterized by chronic inflammation in response to a dysbiotic microbial challenge. The severity of tissue destruction depends not only on the microbial burden but also on the nature and magnitude of the host inflammatory response.

Stress-related neuroendocrine changes may alter inflammatory signalling and the regulation of cytokines. This may influence the intensity and persistence of periodontal inflammation.

A systematic review assessing stress, depression, inflammation, and periodontal disease found relationships between stress-related conditions and biomarkers such as cortisol, dehydroepiandrosterone, chromogranin A, and inflammatory mediators. However, the available biomarkers cannot currently be used alone to determine periodontal disease severity.[3]

4. Impaired wound healing

Successful periodontal healing requires coordinated inflammatory, proliferative, and tissue-remodelling processes.

Chronic stress can interfere with normal healing through neuroendocrine and immune pathways. Alterations in immune-cell function, vascular responses, and inflammatory regulation may delay tissue repair.

This is clinically relevant because periodontal patients frequently undergo procedures such as scaling and root planing, periodontal surgery, and regenerative procedures. Adequate healing is essential for achieving favourable treatment outcomes.

5. Behavioural changes

The relationship between stress and periodontal disease is not purely biological. Behavioural changes may be equally important.

A stressed individual may:

- Pay less attention to oral hygiene.
- Brush irregularly or inadequately.
- Miss periodontal maintenance appointments.
- Smoke or increase tobacco consumption.
- Change dietary habits.
- Experience disturbed sleep.
- Consume alcohol more frequently.
- Have reduced motivation to follow professional advice.

These behaviours can increase plaque accumulation or introduce additional risk factors for periodontal disease.

Therefore, the effect of stress on periodontal health may occur through a combination of biological and behavioural pathways.

STRESS AND PERIODONTAL DISEASE: WHAT DOES THE EVIDENCE SAY?

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- Pay less attention to oral hygiene.
- Brush irregularly or inadequately.
- Miss periodontal maintenance appointments.
- Smoke or increase tobacco consumption.
- Change dietary habits.
- Experience disturbed sleep.
- Consume alcohol more frequently.
- Have reduced motivation to follow professional advice.

These behaviours can increase plaque accumulation or introduce additional risk factors for periodontal disease.

Therefore, the effect of stress on periodontal health may occur through a combination of biological and behavioural pathways.

STRESS AND PERIODONTAL DISEASE: WHAT DOES THE EVIDENCE SAY?

The association between stress and periodontal disease has been investigated through observational studies, clinical studies, and systematic reviews.

An early systematic review by Peruzzo et al. found that 57.1% of the included studies reported a positive association between psychosocial factors/stress and periodontal disease. However, the authors noted differences in study design and methods of measuring stress and periodontal disease.[1]

A later systematic review specifically examining psychological stress and periodontitis found a possible positive association but concluded that more high-quality studies were needed to establish the relationship more clearly.[7]

A meta-analysis published in 2022 reported a pooled odds ratio of approximately 1.78 for the association between psychological stress and periodontitis. However, substantial heterogeneity was present among the included studies, indicating that the results should be interpreted cautiously.[2]

More recent evidence has also focused on the effect of stress on periodontal treatment. A 2025 systematic review reported that stressed patients were more likely to demonstrate higher probing pocket depths and bleeding on probing following periodontal treatment, although the number of available studies was limited.[8]

These findings suggest that stress may influence periodontal health and treatment response, but stress alone cannot be considered a direct cause of periodontitis. The disease is multifactorial, and microbial dysbiosis remains central to its initiation and progression.

STRESS AS A PERIODONTAL RISK INDICATOR

It is important to distinguish between a causal factor, a risk factor, and a risk indicator.

Stress does not independently initiate periodontitis in the same manner as the microbial biofilm. Instead, it may modify the host response and influence behaviours that affect periodontal health.

The effect of stress may also vary from person to person. Two individuals experiencing similar stressful circumstances may have very different periodontal outcomes because of differences in genetics, immune response, oral hygiene, smoking habits, socioeconomic conditions, systemic health, and coping mechanisms.

Therefore, psychological stress is best considered a potential modifying factor or risk indicator for periodontal disease, rather than a sole etiological factor.

STRESS AND RESPONSE TO PERIODONTAL THERAPY

Periodontal treatment primarily aims to control the microbial challenge and reduce periodontal inflammation. However, treatment success also depends on patient-related factors.

Patients experiencing high levels of stress may have:

- Poorer oral-hygiene compliance.
- Irregular maintenance attendance.
- Reduced adherence to postoperative instructions.
- Increased tobacco use.
- Greater difficulty maintaining lifestyle modifications.
- Potential alterations in wound healing and inflammatory regulation.

Recent evidence suggests that psychological stress may negatively influence the clinical response to non-surgical periodontal treatment.[8]

This highlights an important concept: periodontal therapy should be patient-centred rather than limited to mechanical plaque control.

ROLE OF THE PERIODONTIST

The dental professional does not need to diagnose psychological disorders to recognize the possible influence of stress.

Simple clinical observations and patient communication can help identify individuals who may benefit from additional support.

The periodontist can:

1. Ask about major lifestyle or occupational stressors when appropriate.
2. Explain the possible relationship between stress and periodontal health.
3. Reinforce the importance of effective oral hygiene.
4. Encourage regular periodontal maintenance.
5. Discuss smoking and other harmful coping behaviours.

6. Encourage adequate sleep, balanced nutrition, and healthy coping strategies.
7. Refer the patient to an appropriate healthcare professional when significant psychological problems are suspected.

Stress management should therefore be viewed as an adjunct to, and not a replacement for, conventional periodontal therapy.

PRACTICAL STRESS-MANAGEMENT MEASURES

Simple lifestyle measures can support general and oral health:

- Regular physical activity.
- Adequate and consistent sleep.
- Balanced nutrition.
- Relaxation or breathing exercises.
- Healthy social interaction.
- Avoidance of tobacco and excessive alcohol.
- Regular dental and periodontal maintenance.
- Seeking professional psychological support when required.

These measures do not directly eliminate periodontal pathogens, but they may improve overall health, treatment adherence, and the patient's ability to maintain good oral hygiene

FUTURE PERSPECTIVES

The relationship between stress and periodontal disease is an emerging area of periodontal research. Future studies should use standardized methods for measuring psychological stress, cortisol, periodontal inflammation, microbial changes, and treatment outcomes.

Longitudinal studies are particularly important because they can help determine whether chronic stress precedes periodontal deterioration or whether periodontal disease itself contributes to psychological stress.

Further research into psychoneuroimmunology may also improve our understanding of how the nervous, endocrine, and immune systems interact with periodontal tissues.

CONCLUSION

Stress and periodontal health are connected through a complex interaction of neuroendocrine, immune, inflammatory, and behavioural mechanisms. Chronic psychological stress may increase susceptibility to periodontal inflammation, influence disease progression, and potentially affect treatment response. However, stress should not be considered a primary cause of periodontitis; microbial dysbiosis and the host inflammatory response remain central to periodontal disease.

For the modern periodontist, understanding the patient's psychological and behavioural environment can contribute to more comprehensive patient care. Recognizing stress, improving patient motivation, promoting healthy coping strategies, and maintaining effective periodontal therapy may together help achieve better long-term periodontal outcomes.

REFERENCES

1. Peruzzo DC, Benatti BB, Ambrosano GMB, Nogueira-Filho GR, Sallum EA, Casati MZ, et al. A systematic review of stress and psychological factors as possible risk factors for periodontal disease. J Periodontol. 2007;78(8):1491-504.
2. Aggarwal K, Gupta J, Kaur RK, Bansal D, Jain A. Effect of anxiety and psychologic stress on periodontal health: a systematic review and meta-analysis. Quintessence Int. 2022;53(2):144-54.
3. Castro MML, Ferreira RO, Fagundes NCF, Almeida ACG, Maia LC, Lima RR. The assessment of stress, depression, and inflammation as a collective risk factor for periodontal diseases: a systematic review. J Periodontal Res. 2020;55(2):249-59.
4. Li Y, Zhang Y, et al. Psychological stress: neuroimmune roles in periodontal disease. J Med Dent Sci. 2022.
5. Kaur S, White S, Bartold PM. Periodontal disease and stress: a systematic review and meta-analysis of observational studies. Arch Oral Biol. 2018;93:181-91.
6. Al-Ak'hali MS, Al-Moraissi EA, Fageeh HN, Alakhali H, Peeran SW, Khurayzi TK, et al. Are salivary cortisol levels elevated in periodontitis patients experiencing stress compared to those without stress? A systematic review and meta-analysis. J Contemp Dent Pract. 2025;26(2):206-16.
7. Araujo MM, Martins CC, Costa LC, Cota LOM, Faria RL, Cunha FA, et al. Association between psychological stress and periodontitis: a systematic review. J Evid Based Med. 2020.
8. Villafuerte KRV, Vieira LHP, Santos KO, Rivero-Contreras E, Lourenço AG, Motta ACF. Psychological stress reduces the effectiveness of periodontal treatment: a systematic review. J Clin Med. 2025;14(5):1680.
9. Parwani R, Parwani SR. Does stress predispose to periodontal disease? Dent Update. 2014;41(3):260-4, 267-8, 271-2.
10. Impact of stress on periodontal health: literature revision. Healthcare (Basel). 2023.

AUTHORS: -

Dr. Saurav Aggarwal
Department of Medicine
RIMT University, Mandi - Gobindgarh, Punjab
Email id: aggarwalsaurav475@gmail.com
Mobile No: -

Dr Mohit Dadhich
Senior Lecturer
Department of Pharmacology
Govt. Medical College, Hanumangarh
Email id: -
Mobile No: -

Dr Harshita Pandey
Senior Lecturer
Department of Microbiology
Govt. Medical College, Hanumangarh
Email id: -
Mobile No: -

Dr. Khushi Khatri
Post Graduate student,
Department of Periodontology & Oral Implantology,
Surendera Dental College and Research Institute,
Sri Ganganagar (Rajasthan) 335001
Email : khushikhatri790@gmail.com

CORRESPONDING AUTHOR: -

Dr. Saurav Aggarwal
Department of Medicine
RIMT University, Mandi - Gobindgarh, Punjab
Email id: aggarwalsaurav475@gmail.com
Mobile No: -

Dr. Yashashri Pramod Nehete, Dr. Suruchi Juneja, Dr. Sadhika Sadhar, Dr. Prachi, Dr. Kajal Tayal

ABSTRACT

Sleep disorders are increasingly recognized as an important public health concern because of their adverse effects on growth, cognitive development, behavior, cardiovascular health, and oral health. Paediatric dentists are uniquely positioned to identify early signs of sleep-related disorders during routine dental examinations, allowing timely referral and multidisciplinary management.

This narrative review summarizes the current evidence regarding the classification, risk factors, clinical manifestations, diagnosis, and management of sleep disorders relevant to paediatric dentistry. Particular emphasis is placed on sleep-disordered breathing, obstructive sleep apnea, sleep bruxism, and the role of craniofacial abnormalities in airway compromise. Contemporary treatment approaches, including behavioural therapy, continuous positive airway pressure, oral appliance therapy, myofunctional therapy, orthodontic interventions, and surgical management, are discussed. Recent advances in artificial intelligence, digital dentistry, precision medicine, and wearable technologies that support early diagnosis and personalized treatment are also highlighted.

Paediatric dentists play an essential role in the early recognition of sleep disorders through routine screening, identification of craniofacial risk factors, and collaboration with sleep physicians and other healthcare professionals. Incorporating sleep assessment into routine dental practice may facilitate earlier diagnosis, improve treatment outcomes, and enhance both oral and systemic health.

KEYWORDS

Sleep disorders, Paediatric dentistry, Obstructive sleep apnea, Dental sleep medicine, Oral appliance therapy, Artificial intelligence.

INTRODUCTION

Sleep is a fundamental biological process essential for maintaining physical health, cognitive performance, emotional well-being, immune function, and normal growth and development. Inadequate or disturbed sleep has been associated with an increased risk of cardiovascular disease, diabetes mellitus, obesity, depression, impaired cognitive function, and reduced quality of life. Among the various sleep disorders, sleep-disordered breathing (SDB), particularly obstructive sleep apnea (OSA), represents one of the most prevalent and clinically significant conditions.[1,2]

Increasing evidence demonstrates a close relationship between craniofacial morphology, upper airway anatomy, and sleep-disordered breathing, thereby expanding the role of dentistry in sleep medicine.[3]

SLEEP PHYSIOLOGY

Normal sleep consists of two major states: non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep, which occur cyclically every 90–110 minutes throughout the night. NREM sleep is divided into three stages (N1, N2, and N3), progressing from light sleep to deep slow-wave sleep. Muscle tone of the upper airway decreases, particularly during REM sleep, increasing susceptibility to airway collapse in predisposed individuals.[4]

CLASSIFICATION OF SLEEP DISORDERS RELEVANT TO DENTISTRY

The six major categories (ICSD-3)

- Insomnia disorders
- Sleep-related breathing disorders
- Central disorders of hypersomnolence
- Circadian rhythm sleep–wake disorders
- Parasomnias
- Sleep-related movement disorders

These conditions are commonly associated with craniofacial abnormalities, mouth breathing, malocclusion, tooth wear, temporomandibular disorders, and altered facial growth patterns.[5]

Sleep-disordered breathing (SDB) comprises a spectrum of respiratory disorders ranging from primary snoring and upper airway resistance syndrome (UARS) to obstructive sleep apnea (OSA).[5] Untreated OSA is associated with cardiovascular disease, hypertension, metabolic dysfunction, and insulin resistance.[6] Major risk factors include obesity, adenotonsillar hypertrophy, craniofacial abnormalities, nasal obstruction, male sex, and family history. Craniofacial features such as retrognathia, maxillary constriction, a high-arched palate, increased lower facial height, and reduced upper airway dimensions further predispose individuals to airway obstruction.[5]

Sleep bruxism involves rhythmic or non-rhythmic masticatory muscle activity during sleep, resulting in tooth wear, fractured restorations, muscle fatigue, and temporomandibular disorders. Occlusal splints may protect the dentition but do not treat the underlying sleep

disorder. Insomnia is characterized by difficulty initiating or maintaining sleep and may impair healing, increase pain perception, and adversely affect oral health-related quality of life.[7] Parasomnias, including sleepwalking, sleep terrors, nightmares, and REM sleep behavior disorder, may occasionally cause oral or facial injuries.[1-4] Circadian rhythm sleep–wake disorders and sleep-related movement disorders, such as restless legs syndrome and periodic limb movement disorder, can impair sleep quality, patient compliance, and recovery following dental treatment.[6]

CLINICAL FEATURES

Clinical manifestations vary according to the type of sleep disorder and patient age. Patients with SDB commonly present with habitual snoring, witnessed apneas, gasping, restless sleep, nocturnal sweating, and mouth breathing.[6] In children, hyperactivity, behavioural disturbances, attention deficits, enuresis, poor academic performance, and impaired growth are more frequent than excessive daytime sleepiness. Sleep bruxism presents with tooth wear, fractured restorations, tooth sensitivity, masseter hypertrophy, tongue indentations, linea alba, temporomandibular discomfort, and morning jaw soreness.[8] Insomnia is associated with fatigue, irritability, anxiety, impaired concentration, and increased pain sensitivity.[8] Circadian rhythm disorders lead to altered sleep timing and daytime fatigue, whereas restless legs syndrome and periodic limb movement disorder cause sleep fragmentation and excessive daytime sleepiness. Parasomnias may occasionally result in unexplained oral or facial trauma, warranting careful evaluation by dental professionals.[9]

DIAGNOSIS AND EVALUATION

Diagnosis begins with a comprehensive history including habitual snoring, witnessed apneas, gasping during sleep, mouth breathing, restless sleep, excessive daytime sleepiness, morning headaches, behavioural problems, poor academic performance, and family history of sleep disorders.[9] Overnight polysomnography (PSG) remains the gold standard for diagnosing OSA, providing objective assessment of sleep architecture, respiratory events, oxygen saturation, and the Apnea–Hypopnea Index (AHI) for disease severity.[10] Screening tools such as the STOP-Bang Questionnaire, Epworth Sleepiness Scale (ESS), and Paediatric Sleep Questionnaire (PSQ) aid in identifying high-risk individuals but do not replace PSG. Lateral cephalometry and cone-beam computed tomography (CBCT) help evaluate craniofacial morphology and upper airway dimensions. Early recognition and timely referral by dental professionals are essential for multidisciplinary management and prevention of long-term complications.[11]

MANAGEMENT OF SLEEP DISORDERS

Management requires a multidisciplinary, individualised approach involving paediatric dentists, sleep physicians, paediatricians, otolaryngologists, orthodontists, neurologists, psychologists, and pulmonologists, with the goals of restoring normal sleep, relieving symptoms, improving quality of life, and preventing systemic complications.[8,10]

NON-SURGICAL MANAGEMENT

Cognitive behavioural therapy for insomnia (CBT-I) is the preferred first-line treatment for chronic insomnia because of its superior long-term outcomes compared with pharmacotherapy. Continuous positive airway pressure (CPAP) remains the standard treatment for moderate-to-severe OSA by preventing upper airway collapse during sleep. Mandibular advancement devices (MADs) enlarge the upper airway by advancing the mandible and are the most widely used oral appliances due to good patient acceptance and long-term compliance. Tongue-retaining devices (TRDs) may be considered in edentulous patients or those unable to tolerate MADs.[12] Myofunctional therapy, including tongue, lip, soft palate, and nasal breathing exercises, improves orofacial muscle tone and serves as an effective adjunct in reducing upper airway collapsibility..[9]

Orthodontic interventions benefit children with maxillary constriction, high-arched palate, posterior crossbite, or mandibular retrognathia. Rapid maxillary expansion (RME) increases maxillary width, enlarges the nasal cavity, reduces airway resistance, and improves nasal breathing. Functional orthopedic appliances, such as Twin Block and Herbst appliances, may further enhance airway dimensions by advancing the mandible during growth.[13]

SURGICAL MANAGEMENT

Surgical treatment is indicated when conservative therapy is ineffective or when anatomical airway obstruction is present.[3,14] Adenotonsillectomy is the first-line surgical treatment for paediatric OSA associated with adenotonsillar hypertrophy and significantly improves AHI, oxygen saturation, behaviour, and quality of life.[14,15]In adults with craniofacial skeletal deficiency, maxillomandibular advancement (MMA) is among the most effective procedures for enlarging the upper airway. Other procedures include uvulopalatopharyngoplasty (UPPP), septoplasty, turbinate reduction, tongue-base

reduction, genioglossus advancement, hyoid suspension, and hypoglossal nerve stimulation in selected patients.[12,16]

Management of sleep bruxism aims to prevent dental complications and relieve symptoms. Patients with significant tooth wear, morning jaw discomfort, or habitual snoring should be evaluated for coexisting OSA, while occlusal splints may protect the dentition and reduce muscle discomfort.[17] Insomnia is primarily managed with cognitive behavioural therapy for insomnia (CBT-I), including stimulus control, sleep restriction, cognitive restructuring, relaxation techniques, and sleep hygiene, with pharmacotherapy reserved for selected cases.[12,18] Parasomnias are managed through patient safety, reassurance, trigger avoidance, and treatment of underlying sleep disorders; dentists should recognize associated oral injuries such as tongue biting, lip trauma, fractured restorations, and dental trauma.[19] Circadian rhythm sleep–wake disorders are managed by maintaining regular sleep schedules, timed light exposure, limiting evening screen time, and reinforcing sleep hygiene, with referral for persistent disturbances when indicated.[15,16,20,21]

RECENT ADVANCES IN DENTAL SLEEP MEDICINE

Recent advances in dental sleep medicine have enhanced the diagnosis and management of sleep disorders through digital technologies and personalized care. Three-dimensional (3D) imaging, cone-beam computed tomography (CBCT), and digital intraoral scanners enable accurate assessment of craniofacial structures and upper airway anatomy.[22,23] Home sleep apnea testing (HSAT), wearable monitoring devices, telemedicine, and artificial intelligence (AI) facilitate early diagnosis, risk assessment, treatment planning, and continuous monitoring. Precision medicine further supports individualized management by integrating genetic, anatomical, clinical, imaging, and lifestyle factors to optimize treatment outcomes[12,14]

ROLE OF THE PAEDIATRIC DENTIST

Paediatric dentists play a key role in the early detection of sleep disorders by recognizing clinical signs such as habitual snoring, mouth breathing, enlarged tonsils, high-arched palate, maxillary constriction, mandibular retrognathia, and sleep bruxism during routine dental examinations.[17] They facilitate timely referral, provide patient and caregiver education, fabricate and monitor oral appliances, support interceptive orthodontic treatment, and collaborate in multidisciplinary care to improve both oral and systemic health outcomes.[24,25]

CONCLUSION

Sleep disorders are increasingly recognized as an integral component of paediatric oral and systemic health. Paediatric dentists play a crucial role in the early detection of sleep-disordered breathing and other sleep-related conditions through routine clinical examination and assessment of craniofacial development. Timely screening, appropriate referral, and multidisciplinary management are essential for improving both short- and long-term health outcomes. With ongoing advances in digital imaging, artificial intelligence, and precision medicine, dental sleep medicine is evolving toward more accurate, personalized, and preventive care. Future research should focus on strengthening the evidence base for dental interventions and establishing standardized clinical protocols to further integrate paediatric dentistry into comprehensive sleep healthcare.

REFERENCES

1. Marcus CL, Brooks LJ, Draper KA, Gozal D, Halbower AC, Jones J, Schechter MS, Sheldon SH, Spruyt K, Ward SD, Lehmann C, Shiffman RN. Diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics. 2012;130(3):576-584.
2. Gozal D, Kheirandish-Gozal L. Childhood obstructive sleep apnea: A comprehensive review. Sleep Med Rev. 2009;13(5):345-358.
3. Huynh NT, Desplats E, Almeida FR. Orthodontic treatments for managing obstructive sleep apnea syndrome in children: A systematic review and meta-analysis. Sleep Med Rev. 2016;25:84-94.
4. Villa MP, Evangelisti M, Martella S, Barreto M, Del Pozzo M. Respiratory sleep disorders in children and role of the paediatric dentist. Eur J Paediatr Dent. 2019;20(1):9-14.
5. Anwer HMM, Albagieh HN, Kalladka M, Chiang HK, Malik S, McLaren SW, Al-Hamdan RS. The role of the dentist in the diagnosis and management of pediatric obstructive sleep apnea. Saudi Dent J. 2021;33(7):424-433.
6. Lo Giudice A, Malgioglio A, Maniaci A, La Mantia I, Bianchi A, Cocuzza S. Current evidence on the role of pediatric dentists in the multidisciplinary management of pediatric obstructive sleep apnea. Diagnostics (Basel). 2024;14(5):506.
7. Sateia MJ. International Classification of Sleep Disorders—Third Edition: Highlights and modifications. Chest. 2014;146(5):1387-1394.

8. Kaditis AG, Alvarez MLA, Boudewyns A, Abel F, Alexopoulos EI, Ersu R, Joosten K, Larramona H, Miano S, Narang I, Trang H, Van Waardenburg D, Verhulst S. Obstructive sleep disordered breathing in 2- to 18-year-old children: Diagnosis and management. Eur Respir J. 2016;47(1):69-94.
9. Lobbezoo F, Ahlberg J, Raphael KG, Wetselaar P, Glaros AG, Kato T, Santiago V, Winocur E, De Laat A, De Leeuw R, Svensson P. International consensus on the assessment of bruxism: Report of a work in progress. J Oral Rehabil. 2018;45(11):837-844.
10. Camacho M, Certal V, Abdullatif J, Zaghi S, Ruoff CM, Capasso R, Certal VF. Myofunctional therapy to treat obstructive sleep apnea: A systematic review and meta-analysis. Sleep. 2015;38(5):669-675.
11. Chervin RD, Hedger K, Dillon JE, Pituch KJ. Pediatric Sleep Questionnaire: Validity and reliability. Sleep Med. 2000;1(1):21-32.
12. Johns MW. A new method for measuring daytime sleepiness: The Epworth Sleepiness Scale. Sleep. 1991;14(6):540-545.
13. Riemann D, Baglioni C, Bassetti C, Bjorvatn B, Dolenc Groselj L, Ellis JG, Espie CA, Garcia-Borreguero D, Gjerstad MD, Gonçalves M, Hertenstein E, Jansson-Fröjmark M, Jennum PJ, Leger D, Nissen C, Parrino L, Paunio T, Pevernagie D, Verbraecken J, Weeß HG, Wichniak A, Zavalko I, Arnardottir ES, Deleanu OC, Strazisar B, Zoetmulder M, Spiegelhalder K. European guideline for the diagnosis and treatment of insomnia. J Sleep Res. 2017;26(6):675-700.
14. Patil SP, Ayappa IA, Caples SM, Kimoff RJ, Patel SR, Harrod CG. Treatment of adult obstructive sleep apnea with positive airway pressure: An American Academy of Sleep Medicine clinical practice guideline. J Clin Sleep Med. 2019;15(2):335-343.
15. Ramar K, Dort LC, Katz SG, Lettieri CJ, Harrod CG, Thomas SM, Chervin RD, Carden KA, Aurora RN. Clinical practice guideline for the treatment of obstructive sleep apnea and snoring with oral appliance therapy: An update for 2015. J Clin Sleep Med. 2015;11(7):773-827.
16. Sutherland K, Vanderveken OM, Tsuda H, Marklund M, Gagnadoux F, Kushida CA, Cistulli PA. Oral appliance treatment for obstructive sleep apnea: An update. J Clin Sleep Med. 2014;10(2):215-227.
17. Guilleminault C, Huang YS, Monteyrol PJ, Sato R, Quo S. Critical role of myofunctional therapy in pediatric sleep-disordered breathing. Sleep Med. 2013;14(6):518-525.
18. Pirelli P, Saponara M, Guilleminault C. Rapid maxillary expansion in children with obstructive sleep apnea syndrome. Sleep. 2004;27(4):761-766.
19. Caples SM, Rowley JA, Prinsell JR, Pallanch JF, Elamin MB, Katz SG, Harwick JD. Surgical modifications of the upper airway for obstructive sleep apnea in adults: A systematic review and meta-analysis. Sleep. 2010;33(10):1396-1407.
20. Li KK, Riley RW, Powell NB, Troell RJ, Guilleminault C. Maxillomandibular advancement for obstructive sleep apnea syndrome. J Oral Maxillofac Surg. 2000;58(10):1181-1186.
21. Irish LA, Kline CE, Gunn HE, Buysse DJ, Hall MH. The role of sleep hygiene in promoting public health: A review of empirical evidence. Sleep Med Rev. 2015;22:23-36.
22. Edinger JD, Means MK. Cognitive behavioral therapy for primary insomnia. Clin Psychol Rev. 2005;25(5):539-558.
23. Allen RP, Picchietti DL, Garcia-Borreguero D, Ondo WG, Walters AS, Winkelman JW, Zucconi M, Ferri R, Trenkwalder C, Lee HB; International Restless Legs Syndrome Study Group (IRLSSG). Restless legs syndrome/Willis-Ekbom disease diagnostic criteria: Updated International Restless Legs Syndrome Study Group (IRLSSG) consensus criteria—history, rationale, description, and significance. Sleep Med. 2014;15(8):860-873.
24. Chung F, Yegneswaran B, Liao P, Chung SA, Vairavanathan S, Islam S, Khajehdehi A, Shapiro CM. STOP Questionnaire: A tool to screen patients for obstructive sleep apnea. Anesthesiology. 2008;108(5):812-821.
25. Kapur VK, Auckley DH, Chowdhuri S, Kuhlmann DC, Mehra R, Ramar K, Harrod CG. Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: An American Academy of Sleep Medicine clinical practice guideline. J Clin Sleep Med. 2017;13(3):479-504.

AUTHORS: -

Dr. Yashashri Pramod Nehete
Post graduate student
Department of Paediatric and Preventive Dentistry
Surendera Dental College and Research Institute
Email: yashashrinehete@gmail.com

Dr. Suruchi Juneja
Professor and Head
Department of Paediatric and Preventive Dentistry
Surendera Dental College and Research institute
Email: drsuruchijuneja@gmail.com

Dr. Sadhika Sadhar
Reader
Department of Paediatric and Preventive Dentistry
Surendera Dental College & Research Institute
Email id: sadhika94sadhar@gmail.com

Dr. Prachi
Post graduate student
Department of Paediatric and Preventive Dentistry
Surendera Dental College & Research Institute
Email id: prachiichhabra1999@gmail.com

Dr. Kajal Tayal
Post graduate student
Department of Paediatric and Preventive Dentistry
Surendera Dental College & Research Institute
Email id: kajaltayal2000@gmail.com

CORRESPONDING AUTHOR: -

Dr. Yashashri Pramod Nehete
Post graduate student
Department of Paediatric and Preventive Dentistry
Surendera Dental College and Research Institute
Email: yashashrinehete@gmail.com

IMPORTANCE OF LIP PRESSUREIN ORTHODONTICS

Dr. Monika Singla, Dr. Eenal Bhambri, Dr. Ankit Bharadwaj, Dr. Khushmita, Dr. Ajay. K, Dr. Atul Aggarwal

ABSTRACT

Lip pressure is crucial to orthodontics, markedly affecting teeth alignment, jaw development, and the enduring stability of treatment outcomes. Optimal dental alignment is preserved thru a subtle equilibrium among the lips, tongue, and cheeks, collectively constituting the orofacial muscle balance. Any impairment in lip functionality can disrupt this equilibrium, leading to malocclusion, recurrence after orthodontic treatment, or the emergence of functional challenges. Abnormal lip pressure patterns may specifically lead to incorrect alignment of the anterior teeth. Altered arch configuration and adverse skeletal development throughout maturation. Furthermore, insufficient lip closure or excessive lip movement can negatively influence speaking, swallowing, and respiration, further complicating therapeutic results. Comprehending the significance of lip pressure is crucial for practitioners in attaining stable, functional, and aesthetic outcomes. This evaluation emphasises the importance of lip pressure in orthodontics and its role in achieving both successful treatment outcomes and enhanced facial aesthetics.

KEYWORDS

Labial force, Orofacial musculature, Balance theory, Orthodontic regression, Craniofacial development, Facial aesthetics, Oral behaviors, Orthodontic devices.

INTRODUCTION

The lips constitute a vital component of the human visage and serve a significant function in facial expression, oral proficiency, phonation, sensibility, and mastication. The lip is a crucial element influencing the aesthetics of the lower portion of the face. According to Hulsey1, "A smile serves as a significant medium thru which individuals express their emotions. The majority of orthodontic research and assessments have relied on the patient's profile and lips in a relaxed state.

Lip pressure denotes the exertion of force by the lips on the teeth, gums, and orthodontic devices such braces, aligners, and retainers.

The lips apply force on the teeth during routine activities like speaking, eating, and smiling. This pressure is moderate yet exerts a considerable influence on the movement of teeth over time. Lip pressure influences the alignment of teeth, contingent upon the tonicity of the lips, since both hypotonic and hypertonic lip pressure are influenced by the competency of the lips, which can be classified as either competent or non-competent. Lip pressure is influenced by the angulation of the incisors and muscular activity. All of these elements influence lip pressure and significantly impact the success of orthodontic treatment and face aesthetics.

ANATOMY

The perioral region contains the Orbicularis oris muscle, a concentric, sphincter-like muscle responsible for the closure of the upper lip.The movement of the lower lip closure is regulated by the Mentalis muscle. Additional muscles include the Levator labii superioris, Zygomaticus major and minor, Depressor labii inferioris, Buccinator, among others.

ROLE OF LIP PRESSURE IN ORTHODONTICS:

1. **Equilibrium theory inOrthodontics:**

This theory asserts that the balance of pressure exerted by the tongue, lips, and cheeks is crucial for maintaining the proper alignment of the teeth. Elevated pressure from the lips induces a lingual inclination of the incisors, leading to overcrowding and a pronounced bite. If the pressure decreases, the incisors will protrude, resulting in an increased overjet and a larger anterior overbite. Proffit WR's study suggested that a consistent tooth alignment necessitates a balance among the muscles, whereas Tomes' research demonstrated that the perioral muscles, in conjunction with the lips and tongue, primarily affect tooth placement. The balance of these muscles is essential for the growth of the craniofacial region.

2. **Lip pressure and jaw growth:**

The pressure exerted by the lips can affect both the teeth and the growth of the jaw in individuals of all ages. If inadequate lip function applies inconsistent force on the teeth, it disrupts the natural growth of the jawbone, resulting in malocclusion. This was corroborated by the research conducted by Janson T and Ingervall B. They also determined that lip pressure was associated with arch width, cranial base measurements, and maxillary tilt. A further investigation supporting this was conducted by Jung MH, Yang WS, and Nahm DS, who also identified a relationship between lip pressure and craniofacial development.

3**. Lip pressure and facialaesthetics:**

The pressure of the lips affects the contour and aesthetics of the face. Excessive pressure from the lips results in

retroclination of the upper incisors observed in Class II Division 2, causing facial asymmetry. Insufficient pressure causes the upper incisors to be proclined, leading to a convex profile and resulting in long face syndrome. Excessive retraction of anterior teeth during orthodontic therapy results in a diminished facial profile. A study conducted by Jung indicated that lip incompetency results in muscular hypertrophy, contributing to long face syndrome.

4. **Lip pressure and smile**

Uneven pressure on the lips may lead to an irregular smile. Excessive pressure from the lips leads to a diminished smile line, obscuring the top incisors and imparting an older look. If lip pressure is diminished, it results in an exaggerated gingival show or gummy smile. This was corroborated by research carried out by Venugopal A, Manzano P6. This research determined that lip pressure can influence oral motor activities, such as smiling. If there is adequate lip strength, it amplifies emotional reactions such as smiling.

5. **Lip pressure and habits:**

The pressure exerted by the lips is crucial in influencing oral behaviours and general orofacial well-being. Excessive or insufficient lip pressure can aggravate prevalent oral behaviours such as mouth breathing and tongue thrusting, hence affecting dental alignment, speech, and dentofacial growth. Lambrechts H and Baets E7 corroborated that a correlation exists between oral habits and lip pressure, noting less lip pressure in an open lip connection.

6.**Lip pressure and Post Orthodontic relapse:**

Lip pressure significantly influences post-orthodontic relapse, since uneven lip pressure can exert persistent stresses on teeth, hence impacting the durability of orthodontic results. An article by Fazio DD corroborated this, indicating that pressure from the lips and cheeks affects relapse following orthodontic treatment and highlighting the importance of muscle training to enhance retention methods.

DEVICES FOR MEASURING LIP PRESSURE:

Numerous techniques have been developed to assess the pressure exerted by the lips within the mouth cavity. A range of instruments, including the Pommeter with a mouthpiece linked to a dynamometer, Electromyography for capturing muscle activity, the Y-meter featuring a Slimline sensor with a compact load cell to assess vertical resistor device (flexiforce low type) with a transducer thickness under 1mm, are employed to evaluate lip position at rest and during swallowing. The Myometer 160 is utilised for assessing lip pressure, featuring a probe composed of two plates fastened together on one side, with force quantified by an electronic apparatus situated between the plates and displayed via a bar graph. A bulb containing a fluid and linked to pressure sensing devices, as well as an Oral Performance instrument and various other technologies, has been employed to quantify lip force (Fig.1).

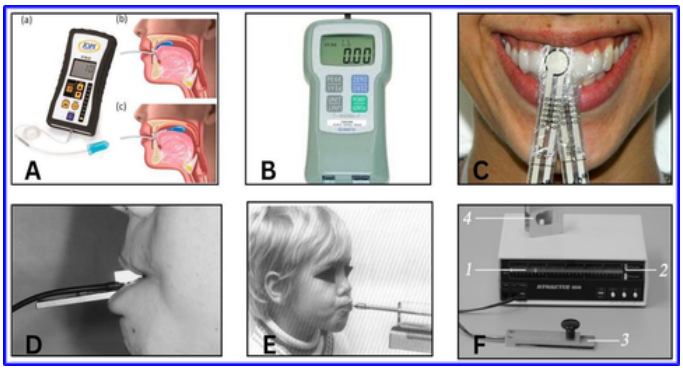


FIG.1 A-IOPI , B-Bulb filled , C-Flexiforce , D-Y meter , E-Pommeter, F- Myometer 160

DISCUSSION

A thorough assessment of lip pressure should involve the observation of lip competence, strain, and posture. The assessment of lip pressure ought to be conducted utilising various instruments and techniques. Unusual pressure, whether excessive or insufficient, signifies the necessity for orthodontic treatment. Recognising atypical behaviours such as thumb sucking, mouth breathing, or tongue pushing might influence lip pressure or the reverse.Early identification of these habits facilitates thorough treatment planning that include both oral and muscle aspects. Orthodontic devices might be required

Customisation predicated on lip pressure, akin to Frankel appliances, can be employed to alter muscle function and enhance lip competence. Lip bumpers serve to alleviate excessive lip pressure on the lower incisors, facilitating appropriate dental development. Orthodontists frequently evaluate the impact of dental alignment on lip support and facial symmetry.5 Following orthodontic therapy Retainers can be engineered to address certain lip pressure concerns; for instance, Hawley's retainer can be modified to facilitate appropriate lip positioning. Post-treatment surveillance must be conducted to mitigate the likelihood of relapse thru regular assessment of lip pressure.

CONCLUSION

Lip pressure is an essential element in orthodontic assessment, treatment strategy, and enduring stability. Comprehending its significance in dental alignment, face aesthetics, and functionality might assist orthodontists in formulating more efficient treatment approaches. The

equilibrium of pressure from the lips, tongue, and cheeks dictates the inherent alignment of the teeth within the dental arches. When this equilibrium is disturbed by either excessive lip pressure or inadequate lip tone, it may result in several malocclusions, including proclined or retroclined anterior teeth, dental crowding, open bites, or an increased overjet. Insufficient lip pressure is frequently linked to behaviours such as mouth breathing and tongue thrusting, which can aggravate dental misalignment and impede appropriate jaw growth. Adequate lip support enhances a harmonious facial profile, whereas excessive dental retraction during orthodontic procedures results in a diminished aesthetic. Consequently, comprehending the correlation between lip functionality and facial aesthetics is crucial when devising treatment plans. Thus, sustaining equilibrium in lip pressure enhances the results of orthodontic therapy. Additional research is necessary to measure lip pressure and establish standardised techniques for including soft tissue evaluation into digital orthodontic planning.

REFERENCES

1. Almashhadan SM. A Lip exercises in Orthodontics. Academicia Globe 2022;3(4):65-. 74.
2. Proffit WR. Muscle pressureand tooth position.Aust Orthod J 1973;3(3):104-08.
3. Fazio DD, Lombardo L, Gracco A, Amico PD, Siciliani G. Lip pressure at rest and during function in 2 groups of patients with different occlusions. Am J Orthod Dentofacial Orthop 2011;139:e1-e6.
4. Janson T, Ingervall B. Relationship betweenlip strength and lip functionin posture and chewing. Eur J Orthod 1982;4:45-53.
5. Jung MH, Yang WS, Nahm DS. Maximum closingforce of mentolabial muscles and type of malocclusion. Angle Orthod 2010;80:72-79.
6. Venugopal A, Manzano P, Ahmed F, Vaiid N, Bowman SJ. Gummy smiles: Etiologies, diagnoses & formulating a clinically effective treatment protocol. Seminarsin Orthodontics 2024;30:482-501.
7. Lambrechts H, Baets ED, Fieuws S, Willems G. Lip and tongue pressurein orthodontic patients . Eur J Orthod 2010;32:466-471.
8. Theur U, Ingervall B, Odont. Pressure from the lips on the teeth and malocclusion. Am J Orthod Dentofacial Orthop 1986;90:234-242.
9. Jung MH, Yang WS, Nahm DS. Effects of upper lip closing force on craniofacial structures. Am J Orthod Dentofacial Orthop 2003;123:58-63.
10. Valentim AF, Moreira RM, Furlan. Evaluation of the force applied by the tongue and lip on the maxillary central incisor tooth. CoDAS 2014;3:26.
11. Lee YS, Ryu J, Baek SH. Comparative analysis of the differences in dentofacial morphology according to the tongue and lip pressure. Diagnostics 2021;11:503.
12. Tomes CS. The bearing of the development of the jaws on irregularities. Dental Cosmos 1873;15:292

AUTHORS: -

Dr. Monika Singla
Postgraduate Student,
Department of Orthodontics and Dentofacial Orthopaedics,
Surendera Dental College and Research Institute

Dr. Eenal Bhambri
Professor and HOD,
Department of Orthodontics and Dentofacial Orthopaedics,
Surendera Dental College and Research Institute

Dr. Ankit Bharadwaj
Senior Lecturer,
Department of Orthodontics and Dentofacial Orthopaedics,
Surendera Dental College and Research Institute

Dr. Khushmita
Postgraduate Student,
Department of Orthodontics and Dentofacial Orthopaedics,
Surendera Dental College and Research Institute

Dr. Ajay. K
Postgraduate Student,
Department of Orthodontics and Dentofacial Orthopaedics,
Surendera Dental College and Research Institute

Dr. Atul Aggarwal
Senior Lecturer,
Department of Oral and Maxillofacial Surgery,
Surendera Dental Collegeand Research Institute

CORRESPONDING AUTHOR: -

Dr. Monika Singla
Post Graduate Student
Department of Orthodontics and Dentofacial Orthopedics
Surendera Dental College & Research Institute
Email id: msmonikasingla0@gmail.com
Mobile No: -

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