Indian Dental Association
Madras Branch

E - MIDAS JOURNAL

"An Official Journal of IDA - Madras Branch"

Chennai/Volume: 5/Issue: 2/Pages: 1-16/June 2018
www.idamadras.com
eISSN 2454 - 8928

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Prof. Dr. V. Shankar Ram, M.D.S., Ph.D.,
President
IDA Madras Branch

President's Message

Dear Members,

Greetings from IDA Madras Branch!

IDA the premier organization of dental professionals secures the dignity and honour of its members, besides enhancing the image of the profession. Out of more than 450 local branches IDA Madras branch bear more members than any other branch.

Research is an integral part of science and dental science is not an exception. I am hereby invite you all to send us well researched articles along with case reports, reviews and professional experience to enrich our scientific knowledge.

My warm regards and good luck is always with dynamic editorial team pioneered by Dr. C.K. Dilip Kumar our editor. May the team continue their saga of continuous publication for the years to come.

Prof. Dr. V. Shankar Ram, M.D.S., Ph.D.,

Dr. H. Thamizhchelvan
Hon. Branch Secretary
IDA - Madras Branch
Hon. Secretary National CDH
IDA (Head Office)

Secretary's Message

Dear Collegues,

" We always feel that life of others is better than us, but we feel that we are also other for someone else, be content in financial or lifestyle, but don't settle in developing your skills".

"More skills you learn, more money you make". Water at 99 degree centigrade is hot and also at 100 degree centigrade, but that 1 degree centigrade difference push the steam forward.

So it is that one degree or smartness in our field will push us forward.

H. Thamizhchelvan

Dr. H. Thamizhchelvan

e-MIDAS Chennai/Volume: 5/Issue: 2/Pages 1-16/June 2018

Dr. C.K. Dilip Kumar
Editor-in-Chief
IDA - Madras Branch

Letter from the Editor

Salutations from the editorial desk.

It's been our endeavour to ensure that the publication Mirrors effectively the latest trends in dentistry, in order to provide quality dental services to the community. Each year the number & quality of the articles have increased tremendously. I would like to thank my editorial board members and panel of reviewers for helping me in the preparing this issue.

Hence, I conclude this short editorial with the saying "Man can have nothing, but what he Strives For."

Dr. C.K.Dilip Kumar

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Original

Prevalence of Oral Mucosal Lesions in Relation to Tobacco and Alcohol Usage

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Introduction

Premalignant lesions of the oral cavity represent an important target for cancer prevention. They can be detected by visual inspection and their importance derives from the high proportion of cases in which biopsy reveals dysplasia or even frank carcinoma. The strongest risk factors for these oral lesions are use of inhaled tobacco, chewing substitutes that usually include tobacco such as pan masala and betel nut quid and alcohol, Smoking and Chewing Tobacco.

The prevalence of oral precancerous lesions varies in different countries and suggests a variance from as much as 25% to as little as 0.2%.[1] In India it is estimated that 195 million people use tobacco, 62.46 million use alcohol. The incidence of oral mucosal lesions depends on the method, duration, frequency and intensity of use.[2]

Smokeless tobacco, which is chewed alone or with betel quid / paan, has a significant detrimental impact on the oral cavity. A wide variety of mucosal changes have been noted in habitual users of smoked and smokeless tobacco.[3] These changes most likely result from the many irritants, toxins, and carcinogens found naturally in cured or burned tobacco leaves, but may also arise from the mucosal drying effects, the high intraoral temperatures, intraoral pH changes, local alteration of membrane barriers and immune responses, or altered resistance to fungal and viral infections.

Alcohol could contribute to oral lesions, either directly or indirectly. Chronic exposure to ethanol may be associated with carcinogenic cytological changes in the oral mucosa, even in the absence of tobacco smoking.[4] In this study, subjects who visited dental outpatient clinic with the habit of smoking, chewing tobacco and consuming alcohol were examined for the prevalence of oral mucosal lesions

Materials and Method

The study group constituted 998 patients, examined over a period of 1 year attending Sri Venkateswara Dental College and Hospital, Kanchipuram, South India.

Patients who visited the Dental outpatient department with the history of tobacco habits and alcohol consumption were selected for this study. Patients were explained orally about this study and those who were willing to reveal their personal habits and willing to undergo oral examination were taken as subjects. Patients who came for medical ailments met the physician first for their chief complaint and were later taken for oral examination in the dental clinic.

Inclusion Criteria

1. Both male and females patients in the age of 18 years and above.
2. Patients who consent to reveal the tobacco and alcoholic habits and consent to subject themselves for oral examination.
3. Individual who practiced the habit for a minimum period of 6 months and still actively continuing the habit.

Exclusion Criteria

1. Patients who were not willing to reveal the habits and/or subject themselves for oral examination.
2. Patients admitted for systemic diseases.
3. Individual who gave up the habit during the past 6 months.

A preformed case sheet, which included detailed recording of the patient's habits, was used for each individual. The oral habits section included questions about regular use of tobacco smoking, tobacco chewing, areca quid and alcohol consumption. All the patients were examined with the help of a mouth mirror and probe under adequate illumination. The lesions if present were photographed with the patient's consent. The collected data were compiled and statistical analysis was made.

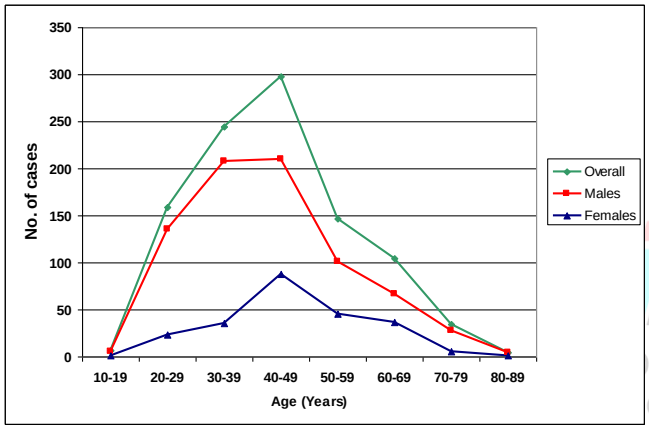
Results

In this study of 998 individuals with habits, 760 (76.2%) were males and 238 (23.8%) were females, with the age range from 18 years to 80 years. The mean age of the study population was 42.7, in which it was 41.6 for males and 45.9 for females. (Graph 1) The different types of habit seen among them was shown in Table 1

Out of the 998 individuals examined 275 of the individuals were found to have 334 lesions. Among

them, 227 (82.5%) of the males and 48 (17.5%) of the females had lesions. Based on the age, 2.5% of the individuals had lesions less than 30 years of age, 16.4% of them between 30 – 39, 40% of the between 40 – 49, 19.6% between the age group of 50 -59 and 21.5% of them were above the age of 60.(Graph 2)

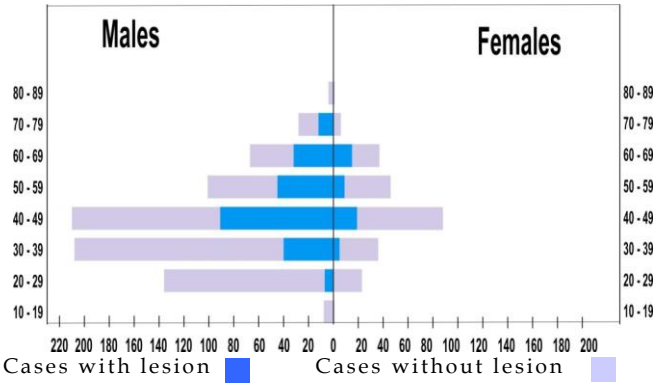
Lesions were present in 30% of the smokers, 25% of tobacco chewers, 51% in alcoholics, 39% of individuals had alcohol and smoking habit, 15% of the individuals consumed alcohol and chewed tobacco, 33% of the individuals smoked and chewed tobacco and 48% of the individuals smoked, chewed tobacco and consumed alcohol. (Graph 3) The mean frequency and duration of various habits among different habit groups are tabulated. The lesions found in 275 individuals and the distribution of different lesions by habits is shown in Graph 4. Assessment of odds ratio and risk estimate for individual habits were done as shown in Table 2.



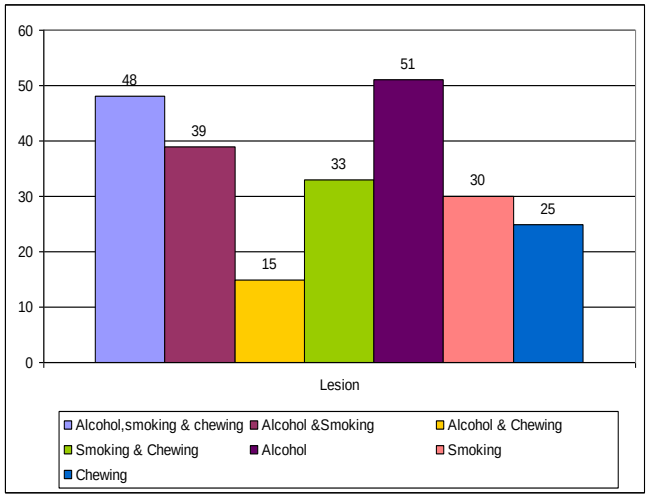
Graph: 1 Age and Sex of the study population

Table: 1 Habits in the study population

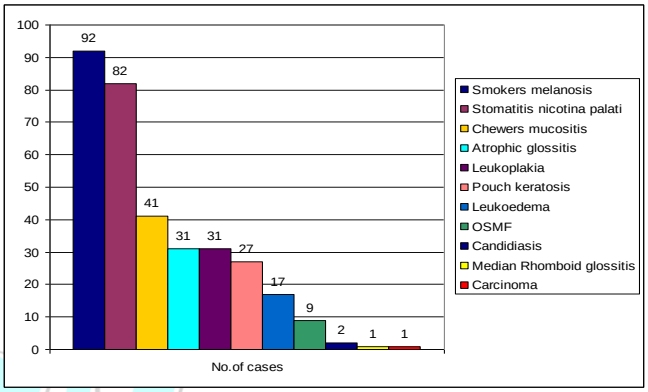
Habits	Number
Smoking	208
Chewing tobacco	273
Alcohol	37
Alcohol & Chewing	33
Smoking & Chewing	99
Smoking & Alcohol	248
Smoking, Alcohol & Chewing	100



Graph 2.Study Population with and without lesion



Graph 3: Prevalence of lesions based on habits



Graph 5: Distribution of different lesions

Table: 2 Odds ratio and risk estimate for individual habits

	Mean	Lesion Present	Lesion Absent	OR	95% CI Lower	95% CI Upper
Smoking Duration	> 7	40 (19.2%)	50 (27.9%)	11.95	4.782	29.883
	< 7	6 (2.9%)	104 (50%)			
Alcohol Duration	> 15	11 (29.7%)	5 (13.5%)	3.5	0.903	14.153
	< 15	8 (21.6%)	13 (35.1%)			
Tobacco Duration	> 10	46 (16.8%)	60 (22.6%)	20.57	8.356	50.646
	< 10	6 (2.2%)	161 (59.0%)			

Discussion

Study on the prevalence of oral mucosal lesions in tobacco habits and alcohol consumption has been done in different parts of the world, differences do exist as the methodologies and sample size vary. The prevalence of oral mucosal lesions in this study (27.6%) is close to the prevalence observed in Thailand (28.4%) and in South London (28.1%). [5]

The various risk habits found in this study are tobacco smoking, chewing, alcohol consumption and a combination of two or three of these. There was correlation between oral mucosal lesions like smokers melanosis, stomatitis nicotina palate, betel chewers mucosa, leukoplakia, leukoedema, oral submucous fibrosis, atrophic glossitis, pouch keratosis, median rhombhoid glossitis, candidal infection, cancer to the above habits and found to be consistent with other studies.[2]

Smokers melanosis (27.5%) had the highest incidence. Hedin CA and Axell T. found smokers had significantly more oral surfaces pigmented than non tobacco users.6 Tobacco smoking stimulates oral melanocytes to a higher melanin production along with genetic factors.

The second most prevalent oral mucosal lesion was stomatitis nicotina palate (24.5%). This finding was much higher than in Ljubljana and in Sweden which were population based.[7,8] The third most prevalent lesion was betel chewers mucositis (12.2 %), consistent with that of a Northern hill tribe of Thailand (13.1%).[9] In our study, this lesion was totally associated with tobacco chewing and was seen more prevalent among females.

The fourth most prevalent lesions were leukoplakia and atrophic glossitis (9.2% respectively).The result of leukoplakia was slightly higher than that of Ching – Hung Chung[1] (7.44%) 1 and Rooban T et al. [2] (6.6%). In India, oral leukoplakia was reported in people who either smoked and/or had a betel quid chewing habit. Alcohol has been found to increase the risk of oral leukoplakia in the presence of tobacco but the independent association between alcohol and leukoplakia remains unclear. The findings in our study showed that there is a strong association between leukoplakia and toxic habits.

Atrophic glossitis (9.2%) seen in our study group was higher than the finding of Axell T , who showed the prevalence of 3% in Thai, 1.3% in Malaysian [6] and 1.1% in Swedish populations.[8] Chronic consumption of alcohol causes oral mucosal atrophy. It has systemic effects such as malnutrition and immunosuppression also

Regarding pouch keratosis (8%), our finding was consistent with that of Axell T 6. The development of this lesion is most strongly influenced by habit duration and also by the brand of tobacco used, early onset of spit tobacco use, total hours of daily use, amount of tobacco consumed daily, and number of sites routinely used for placement.

Oral submucous fibrosis was found in nine individuals (2.6% of our study) and is strongly associated with tobacco chewing habits. The predominant use of areca nut results in comparatively an earlier onset of the disease and fibrous bands formation, whereas chewing of areca nut with tobacco, betel leaves and lime results in later onset of the disease. [10]

Candidal infection (0.5%) and median rhomboid glossitis (0.2%) were associated with smokers in our study[11]. Oral cancer was seen in one individual in the buccal mucosa and tongue.

Comparison of the frequency of development of lesion among the various habit groups showed the Atrophic glossitis was the only lesion and has highest prevalence of lesion (51%) among "only alcohol consumptiongroup". But the prevalence of atrophic glossitis reduced when the individuals had multiple habits. The "only chewers" group presented with 25% of chewer's mucositis. Betel quid habit i.e leaf with areca nut, slaked lime and tobacco was more when compared to the commercial forms of areca nut. Females presented with chewer's mucositis and pouch keratosis and males presented with oral submucous fibrosis.

Areca nut is the most important etiologic factor of submucous fibrosis. The nut contains many alkaloids, arecoline being the most abundant, is shown to stimulate collagen synthesis by fibroblasts. According to Reichart PA and Philipsen HP, chewer's mucositis may be a precursor lesion of oral submucous fibrosis.[12]

A high prevalence of lesions of 48% was seen among individuals with all the three habits. The effect of individual habit in causation of various lesions showed either increased frequency or increased duration, supported by a higher mean age of individuals with lesion when compared to individuals without lesions. Assessment of odds ratio showed smoking for more than 7 years had an 11.95 times higher risk when compared to those who smoked for less than 7 years. Individuals who consumed alcohol for more than 15 years had an 3.56 times higher risk of developing a lesion and chewers who had used tobacco for more than 10 years had an 20.57 times higher risk of developing a lesion.

Conclusion

Oral health professionals should incorporate prevention and cessation services in their routine and daily practice and prevalence study of oral mucosal lesion related to habits will help dentist to provide more effective community based health promotion programs.

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Original

Periodontal Inflammation and Infections: Systemic Implications

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Abstract

The emergence of PERIOMEDICINE made it explicit that a bidirectional link exists between periodontal diseases and systemic health. For more than 3000years now, this association is being investigated. Starting from the proposal of Focal infection theory, numerous paradigm shifts have been witnessed in the periodontal science. Enormous numbers of research studies supporting the bidirectional link are documented in the literature. However similar amount of evidence against it also exists. This article gives and insight into the various forms of evidence in literature that have been documented to prove an association or causal link or otherwise between periodontal disease and systemic implication.

Key Words: Evidence, Focal infection, Periodontitis, Systemic Health.

Introduction

"Take care of your teeth and they'll take care of you."

This dictum is of unknown origin, yet the relation between oral health and general health has been inquisitive for more than 3000 years. Hippocrates, the father of medicine advocated teeth extraction as means to cure arthritis. [1]

The rise and decline of the Focal Infection Theory

In the late 19th century, it was only after the acceptance of the germ theory and principles of Bacteriology, that the role of infections as etiological agents of diseases was being recognized. Willoughby D Miller attributed oral diseases to infections and general diseases to oral diseases. [2,3,4] British surgeon William Hunter accused oral sepsis to either be the origin or deleterious catalyst of certain grave diseases. This idea gradually crystallized into the Focal Infection Theory which stated that "circumscribed foci of bacteria, localized to various parts of the body can result in myriad systemic diseases". [5,6]

The era of focal infection began with the proselytising efforts of its most visible proponent Frank Billings, claiming to cure infections of distant organs by extraction of teeth and tonsillectomies. Though pus within the bodily compartment was considered a systemic threat, its drainage into the mouth was interpreted to be inconsequential systemically.[8,9] One school of thought concluded that dissemination of infection from the focus was prevented by the immune response – but failure of the immune components was considered in dissemination leading to systemic disease. Exclusion of focal infections was considered a rational form of therapy and unresolved cases were attributed to unrecognized foci in the internal organs. Moreover a number of poorly understood diseases were explained by the focal infection theory including psychiatric diseases. This lead to a boom in tooth extractions and tonsillectomies to such an extent that one contemporary quoted "If the craze for violent removal goes on, it will come to pass that we will have a gutless, glandless, and toothless, and I am not sure we may not have, thanks to false psychology and surgery, a witless race''.[10]

At the turn of the century, with the dawn of Bacteriology, it appeared that most, if not all diseases might be infectious in origin. In time it became clear that the theory of focal infection carried this concept to an extreme. The elegance of this theory was easy application; but, it resulted in meagre cure rates, occasional deterioration of disease and inconsistencies in experimental results. Ultimately, it was demonstrated that the science on which the theory was based was flawed. [10]

Re-emergence of focal infection theory

But in the recent years, there has been immense interest in possible associations between periodontal disease and various systemic conditions. This has lead authors to adopt a cautious approach, some seeking intervention to determine causality. The inability of Epidemiology to confirm causality has been emphasized, categorizing the phenomena as a progressive invasion of local tissues distinguishing it from the former Focal Infection Theory. Others have found the scientific evidence of the theory to be slim but have conceded that it may be established by evolving science. Yet, select authors affirm the return of a modest focal theory.

Periodontal health

The oral ecosystem serves as a habitat for about more than 1014 microbes. These pose a constant threat to the defence mechanism. Moreover, the unique anatomy of the tooth which is partly exposed to the oral environment and partly rooted to the connective tissue presents as a vulnerable entity. The imbalance between the host factors and the microbial community initiates

the disease. The influence of systemic disease on the pathogenesis of periodontal destruction and vice-versa has been a topic of debate for years together necessitating the need to establish directionality. [11]

Periodontal health and systemic health

Modest associations between periodontitis and some, though not all, of the diseases and conditions reviewed, is supported by published evidence. [12,13,14] Ample mechanisms have been implicated in literature signifying the bidirectional relationship between periodontitis and a range of systemic conditions establishing a causal link. Amongst these, the theory of three mechanisms put forward by The den Van Velzen at al was awarded much credibility. The theory corroborated that systemic effects could be attributed to metastatic infections, spread of bacterial toxins and immune mediated injury. [15] The above-mentioned concept was reinforced by Van Dyke, who emphasized that periodontal disease causes inflammation and the resultant bacteraemia can metastasize to different organs with production of inflammatory mediators and activation of adaptive immunity having far reaching influence on systemic health. [16] Yet, there exists a lacuna in establishing a definitive link to correlate these mechanisms aforesaid for pathogenesis of various diseases. [17-33]

Definition of disease state

There is a striking heterogeneity in the definition of Periodontitis across and within each disease condition. Moreover, not all studies met a stringent threshold for periodontitis. [17,21-26] Hence it is difficult to compare and identify size of any associations between periodontal disease and systemic diseases. In this context, the presence or absence of associations depend on the definitions adopted. Hence there is a need to conclude a consensus on the threshold to be used to define periodontitis as well as systemic diseases. [17]

Study Designs

There is a need for a paradigm shift from using cross sectional studies to longitudinal studies. This may enable the observation of disease progression and treatment results. [17,21,25,28,30]

Use of surrogate measures

Epidemiological studies predominantly did not use clinical measurements but rather surrogate measures of disease producing intriguing results. The examination of evidence based surrogate markers, do not justify the definite disease event. Studies should rather aim to analyse health outcomes. [25,27-28,31]

Test of hypotheses

In a vast majority of studies, identification of association is followed by suggestion of hypotheses which can be tested. The difficulties posed by the future study designs to investigate the hypotheses should not be underestimated. [24]

Concept of causation

The concept of causation is difficult and any given disease can be caused by more than a single mechanism and every causal mechanism involves the combined action of many component causes. The association of periodontitis with certain systemic disease cannot offer explanation or understanding of the disease. The associations in many cases may be weak but a causal link cannot be totally dismissed. The principles of disease causation and causal theory are beyond the scope of discussion. [17]

Role of shared risk factors

According to the risk factor hypothesis, periodontal diseases share a series of common risk factors with a range of systemic conditions. These may be accountable for the increased risk of systemic complications. These factors present the issue of confounding and bias. [18,24,26,29]

Applying the Bradford Hill criteria

Scientific studies reveal an association between a given factor and a health effect. This cannot be inferred to indicate that the factor causes the specific disease. Researchers are suggesting the application of the Bradford Hill (1965) criteria to establish the strength of evidence for complex conditions for infective aetiology. [17]

This necessitates the evaluation of the body of existing proof for the following:

(a) Statistical strength of association
(b) Consistency
(c) Specificity
(d) Temporal relationship
(e) Biological gradient or dose response relationship
(f) Biological plausibility
(g) Coherence
(h) Experimental reversibility
(i) Analogy / other precedents

Evolving dynamics of systemic diseases

With the advent of predictive, preventive and personalized medicine, it has become even more relevant to integrate these concepts with periodontics. [34]

Prevention

The known methods by which periodontal disease can be prevented are more efficient than those available for

any other chronic diseases. Periodontal disease prevention employs uncomplicated procedures. But taking into consideration the high prevalence of the disease, it can be concluded that it is hardly being used. The approach of prevention has to be embraced. Neglect is the principle cause of periodontal disease. Neglect of oral health is the primitive cause; neglect of systemic health can be merely contributory. [35]

Prediction

Patients with systemic diseases should be placed at a higher risk for periodontal health deterioration and vice-versa. Though the relation between periodontal pathologies and systemic disorders is complex, status of deteriorating periodontal health can be an early indicator as well as a risk factor for a variety of multi-factorial diseases. This includes pre-term birth, a spectrum of vascular pathologies, stroke, heart and lung disease, diabetes mellitus with co-morbidities, some types of cancer, neurological disorders and several mental disorders such as depression, anxiety, anorexia, bulimia, Alzheimer's disease and so on. [34]

Personalization

The patient in question and the wide of range of other systemic contributory factors should be comprehensively analysed. Applying the concept of individual medicine - the great strength of individualized treatment is to offer a holistic and integrative approach comprising of curative, rehabilitative and preventive examination as well as treatment methods tailored for the individual. The multidimensional interaction of risk factors – both internal and external including genetic background, age, gender, environmental risk factors, lifestyle, culture and beliefs as well as social status in the overall predisposition of individuals to disease is recognized .other aspects like development of disease, course of the disease and response to therapeutic intervention is to be considered. The fact that this varies from individual to individual is to be highlighted. [34]

Conclusion

There is a need to use concrete and community agreed case definitions of periodontal disease status. The systematic implementation of the same is essential to decipher the relationship of periodontitis and systemic diseases. Moreover these associations are uncertain since periodontal disease is a heterogeneous mix of conditions. This issue is exaggerated by the ill-defined systemic outcomes in the target disease. A clear cut and narrower definition of diseases may enable identification of profound relationships. Shared genetic susceptibility and cross genetic susceptibility have been considered in correlating certain diseases though there is lack of substantial evidence to support this notion. This is significant as individual variation in disease experience observed clinically can be influenced by a range of factors -genetic, epigenetic and environmental.

Moreover the systemic virulence potential of one's oral micro biome and immune response may be a completely different issue in assessing the nature of the challenge. Hence one size fits all intervention should be replaced by a more customized approach. In order to understand the associations of systemic diseases and periodontal diseases, well designed observational studies should be an integral component of future research. Longitudinal studies to assess the risk would be valuable. Further research is essential for the translation of basic research into clinical studies and practice. Such focused research modalities could go a long way in unravelling the dogma that plaques this dubious association between periodontal disease and various systemic infections.

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