



Prevalence of Chronic Pulpitis among 13 to 17 Year Old Pediatric Patients Visiting Private Dental Institution in Chennai : A Retrospective Study

J. Dhivyadharshini ^a and Pratibha Ramani ^{b*}

^a Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India.

^b Department of Oral Pathology, Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India.

Authors' contributions

This work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.

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ABSTRACT

Introduction: Pulpitis is a debilitating inflammation of the pulp of the tooth. Bacteria infiltrate the pulp of the tooth, causing it to swell. It may affect one or more teeth. Pulpitis comes in two varieties - acute and chronic. Chronic pulpitis is a long-term inflammation of the pulp tissue that results in permanent damage to the reliability of the pulp tissue.

Aim: To assess the prevalence of chronic pulpitis among 13 to 17 year old pediatric patients visiting the private dental institution in Chennai.

Materials and Methods: Case sheets of patients were obtained from Record management system software for analysis. Patients with chronic pulpitis within the age group 13 to 17 were selected and the sample size was found to be n = 165 patients. The collected data was then tabulated for statistical analysis using SPSS.

Results: From the results obtained in our study, chronic pulpitis was most prevalent in females with age of 16 particularly in mandibular molars (16.97% in 36 and 13.94% in 46).

Conclusion: Within the limitations of the study it can be concluded that mandibular molars were commonly affected due to chronic pulpitis and by knowing the prevalence and pathophysiology of chronic pulpitis, dental clinicians can prevent the progression of this condition.

[≡] Professor and Head;

*Corresponding author: E-mail: pratibharamani@saveetha.com;

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1. INTRODUCTION

Blood vessels, nerves, interstitial fluid, fibroblasts, odontoblasts and other cellular elements make up the dental pulp, which is a connective tissue. Pulpitis is a debilitating inflammation of the pulp of the tooth to which one or many teeth could be affected [1]. Its mechanism of action reaction starts with vascular modifications induced by TLR 4/2 positive cells (Toll-like receptors) and involves the release of detectable inflammatory mediators including IL-8, IL-6, and IL-1. The vasculature is made up of core arteries that branch out in peripheries as plexus, namely the pulp horns, under normal conditions [2].

Dental hard tissues enclose the pulp, providing a weakly observant environment, in contrast to soft tissue-enclosed parts of the body. Local metabolites and sympathetic innervation, regulate the majority of dental blood vessels. The pulp's major cellular constituents are fibroblasts of the stroma and odontoblasts cells, which are found in the periphery. Immune cells and indistinct mesenchymal cells can also be present in the perivascular region [3]. Normally neutrophils will be predominant, even though dendritic cells and macrophages are present.

Pulpitis is the inflammation of the dental pulp that is thought to be a closely controlled series of vascular and cellular events regulated by molecular factors [4]. Pulpitis is usually caused by mutualistic oral microorganisms infecting the pulp on an opportunistic basis. Dental caries is the most natural way for microorganisms to enter the body. Bad oral hygiene is often the cause of a cavity. Foods high in sugar and starch cause bacteria throughout the oral cavity to destroy the teeth. Microbes in the oral cavity consume starch and sugar and produce an acid that eats away at the teeth. Tooth decay follows as a result of these microorganisms. Trauma, dentinal cracks, cracked tooth syndrome, uncovered dentinal tubules, and the central apical foramen are also possible sources of pulpal microbial infection [5]. Other findings suggest that adolescent caries is linked to a lower maternal academic background, irregular teeth brushing, lower fresh juice intake, and being female, while reduced milk and water consumption, as well as reduced residential water fluoride levels, may also be risk factors.

TLR-expressing cells in the dental pulp aids to cause immune reactions to microbes and their by-products. Odontoblasts, macrophages, dendritic cells and endothelial cells are all members of this group. To restrict infection, signal impairment, and ease the recovery, some of these cells like odontoblasts can develop mechanical barriers, sense and communicate impulses with the help of nerve fibers, or distinguish into a specific cell with the aid of dental pulp stem cells [6].

Four clinical pulpal disorders are characterized based on the patient's signs, symptoms, and examination: normal, reversible pulpitis, irreversible pulpitis or necrotic. The gold standard for determining the inflammatory condition of dental pulp is histology; moreover, it is widely accepted that histologic and clinical analysis of pulpal diagnosis are both in need of improvement [7]. Normal and necrotic pulps have a simple histological appearance. The problem is distinguishing between reversible and irreversible pulpitis. In view of histological reports, reversible pulpitis is portrayed by the shortfall of microbes and by confined coagulation and liquefaction quickly encompassing the aggravation, while irreversible pulpitis is described by the presence of the microscopic organisms and it's by products in the pulp and by a dominance of intense provocative cells dominantly neutrophils in the tissue underneath the sore recommending chemotactic action [8].

The fundamental clinical contrast among reversible and irreversible pulpitis is in the pulp reaction to a heat stimulus. Reversible pulpitis is characterized by an intense yet brief reaction to cold stimuli [9]. On the other hand, irreversible pulpitis is marked by a persistent, spontaneous ache which is exaggerated on facing a stimulus. However, enamel with permanent pulpitis can be painless in 40% of cases [10]. The pulp is expected to improve after the causative stimulus is removed in reversible pulpitis. If the pulp is irreversibly inflamed, however, recovery is unlikely, and pulpectomy (the complete removal of the dental pulp) is recommended [11].

Nowadays, case history, as well as medical and radiographic analysis, are used as diagnostic adjuncts to assess pulpal discomfort. Inspection, pulp exposure to thermal or electric stimulus, and discomfort on palpation or percussion are all part of the clinical assessment. Pulpitis can also be

classified as acute or chronic depending on the duration. Acute pulpitis is a debilitating disease which can be both reversible or irreversible, and is thought to be one of the leading causes of patients seeking emergency dental care outside of regular business hours [12].

Chronic pulpitis is an inflammation of the pulp tissue that lasts for a long time and causes permanent damage to the pulp tissue's reliability. Chronic pulpitis is caused mainly by chronic dental caries or repeated under fillings adjacent either with pulp chamber closed or exposed and also can be due to dental erosion or vital teeth primed for a fixed prosthetic crown [13]. The patient's dental experience of chronic pulpitis involves more incidents of dental irritation that did not necessitate a visit to the dentist. The clinical characteristics include a dull or irritating form of pain that was mainly insidious in onset, pain that could last from a few minutes to multiple hours (up to 2 hours) no and would be relieved on analgesics [14]. Chronic pulpitis also has other characteristics such as hyper-responsiveness at higher intensities of thermal stimulant during pulp testing and a negative reaction of the tooth to percussion [15].

The main purpose of our study was to assess the prevalence of chronic pulpitis in 13 to 17 year old pediatric patients visiting the private dental institution in Chennai. With the knowledge of prevalence of chronic pulpitis pertaining to gender and tooth, preventive measures can be initiated even before the caries or other factors

so as to avoid complications to the young pediatric patients.

2. MATERIALS AND METHODS

With the approval of the Institutional ethical committee, Retrospective study was done in the pediatric population within the age group of 13 to 17 years visiting Saveetha Dental College from June 2019 to February 2021 and the sample size n = 165 patients. Case sheets of patients were obtained from Record management software for data retrieval and analysis and patients with chronic pulpitis within the age group 13 to 17 were selected. Comparison of prevalence of chronic pulpitis with gender and teeth number was done. The collected data was then tabulated for statistical analysis using SPSS.

3. RESULTS

The current study evaluated the prevalence of chronic pulpitis among 13 to 17 year old pediatric patients visiting the private dental institution in Chennai. The first set of data estimated the age distribution of chronic pulpitis, which showed that the majority of those who were prone to have chronic pulpitis were 16 years old (33.94%), 22.42% were 15 years old, 16.97 % were 17 years old, 15.15 % were 13 years old, and 11.52 % were 14 years old [Fig. 1]. Almost over half of the sample population afflicted by chronic pulpitis (63.64%) were female and only 36.36% were male [Fig. 2]. The proportion of tooth number 46 affected by chronic pulpitis was higher (24.85%) than tooth number 36 which was found to be (24.24%).

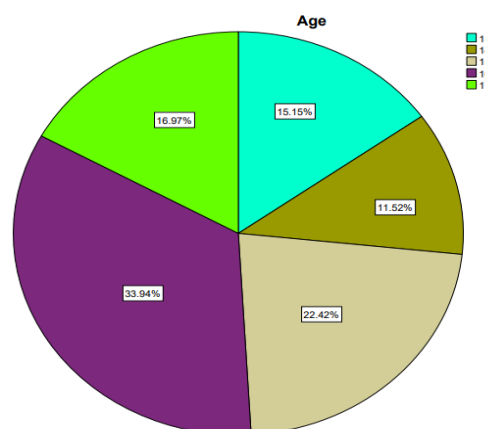


Fig. 1. This pie chart represents the distribution of chronic pulpitis with regards to age. Most of the people who are likely to be affected with chronic pulpitis were 16 years of age (33.94%), 22.42% were 15 years old, 16.97% were 17 years old, 15.15% were 13 years old and 11.52% were 14 years old

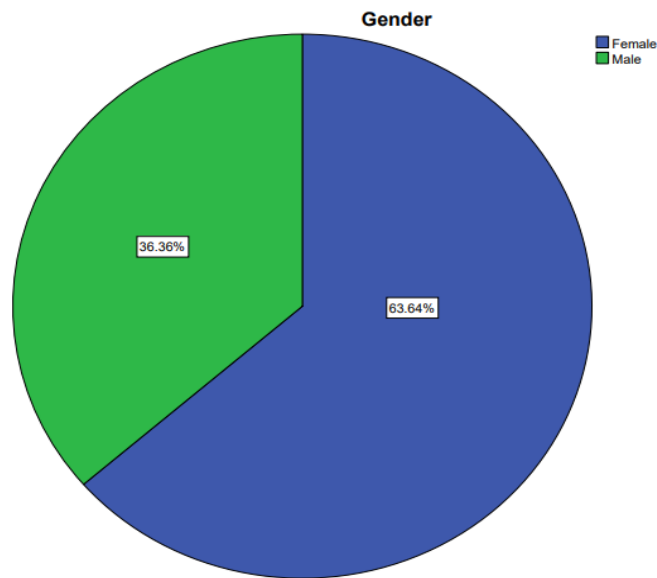


Fig. 2. This pie chart represents the distribution of chronic pulpitis with regards to gender. Blue colour in the chart represents female and green represents male. Nearly more than half of the study population who are affected with chronic pulpitis (63.64%) were female and only 36.36% of the population were males

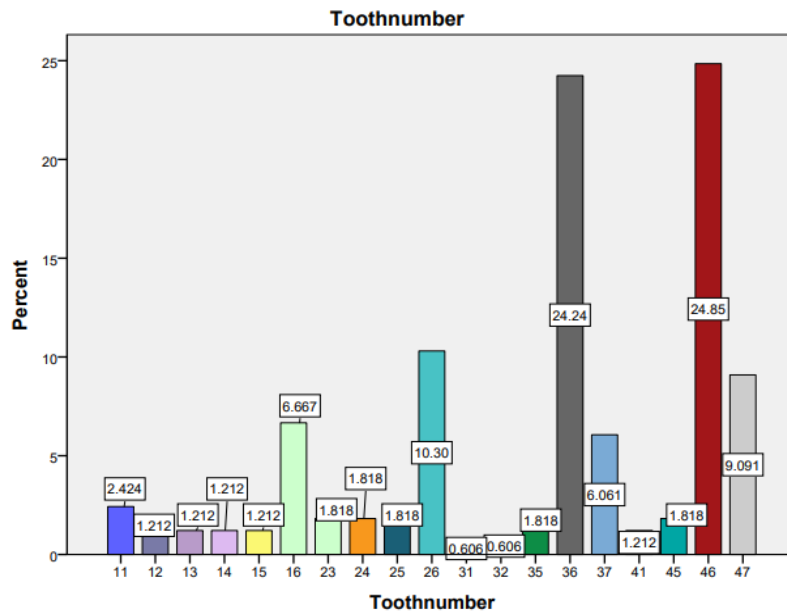


Fig. 3. This bar chart represents the prevalence of chronic pulpitis with regards to tooth number. X-axis represents the tooth number and Y-axis represents the count of teeth affected with chronic pulpitis. The count of tooth number 46 affected with chronic pulpitis was found to be higher (24.85%) than the other affected tooth, 24.24% was in tooth number 36, 10.30% was in tooth number 26, 9.09% was in tooth number 47, 6.66% was in tooth number 16 and 6.06% was in tooth number 37. From the graph we can interpret that the first molars, mainly first mandibular molars, are more prone to chronic pulpitis

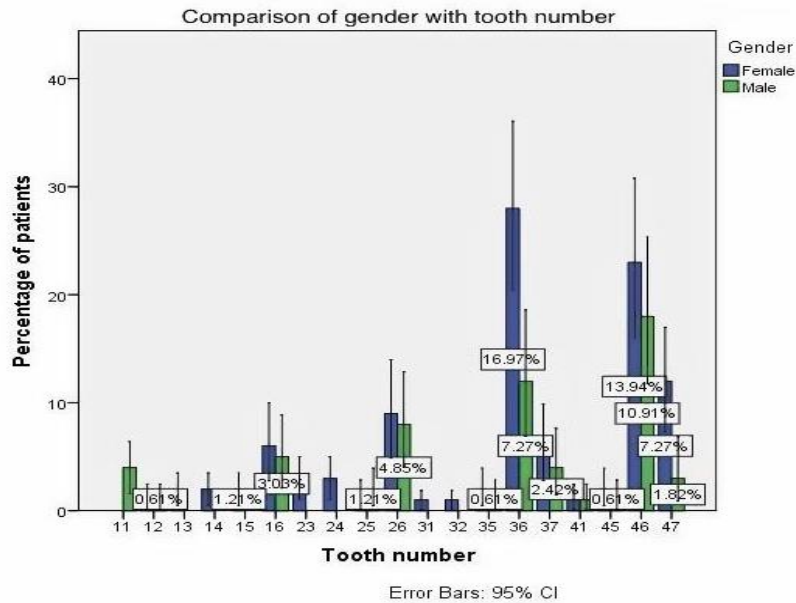


Fig. 4. This bar chart represents the comparison of gender with the tooth affected with chronic pulpitis. X-axis represents the tooth number and Y-axis represents the count of teeth affected with chronic pulpitis with regards to gender. Blue colour in the graph represents female and green represents male. The count of tooth number 36 affected with chronic pulpitis was found to be higher in case of females (16.97%) than the males (7.27%), secondly the females with tooth number 46 affected were found to be 13.94% while the count of male for the same tooth was found to be 10.91%. 7.27% of females had affected tooth number 47, 4.85% in females with tooth number 26 and 3.03% of females with tooth number 16. Chi square test shows Pearson Chi square value of 21.269a and p value of 0.266 ($P < 0.05$), hence association was found to be statistically insignificant. From the graph we can interpret that the tooth number 36 of the females are the most affected tooth

The count of other tooth numbers majorly affected with chronic pulpitis were 10.30% in tooth number 26, 9.09% in tooth number 47, 6.66% in tooth number 16 and 6.06% in tooth number 37 (Fig. 3). In Fig. 4, on comparing gender with the tooth affected with chronic pulpitis females were found to have a higher prevalence of chronic pulpitis in tooth number 36 (16.97%) than males (7.27%), and females with tooth number 46 were found to have a higher prevalence of 13.94% than males (10.91%). 7.27% of females had affected tooth number 47, 4.85% in females with tooth number 26 and 3.03% of females with tooth number 16. Chi square test showing the Pearson Chi square value of 21.269a and 0.266 p value ($P < 0.05$) which was not statistically significant.

4. DISCUSSION

Chronic pulpitis may develop with or without episodes of acute pulpitis and many pulps under large carious cavities die painlessly. From our study we found that the chronic pulpitis within the

age limits was found to be more prevalent in females (63.64%) when compared to males which was 36.36% (Fig. 2). Dental anxiety and negligence is more exhibited by females could be a reason. Infrequent visits to the dentist, lengthy waits in the dental clinic, past painful dental encounters, discomfort during dental surgery, the amount of care received, and intricacy of the procedure may all contribute to high levels of dental anxiety in females [16]. Common causes of this dental anxiety in females were found to be use of instruments such as needles and handpieces and the tilted-back position of the chair (Dental Anxiety Scale 1) [17].

It was also found in our study that the prevalence of chronic pulpitis was more frequent at the age of 16 (33.94%). There are no recent studies revealing the chronic pulpitis in this particular age group. This prevalence may be due to low awareness about dental hygiene. Also children in this age consume more foods rich in sugar and starch which causes the bacteria throughout the oral cavity to destroy the teeth. Microbes in the

oral cavity consume starch and sugar and produce an acid that eats away at the teeth [18]. Tooth decay follows as a result of this. This adolescence age group children are also prone to trauma which can also be a cause for chronic pulpitis.

It was found that the prevalence of chronic pulpitis was most common in mandibular first molars (24.85% in 46 and 24.24% in 36) when compared to other teeth (Fig. 3). Recent study showed that the prevalence of chronic pulpitis was 60.7%, 68.2% and 43.8% in molars, premolars and anterior teeth, respectively [19]. The reason could be due to anatomy of the tooth (the pits and fissures) and poor oral hygiene. When it comes to depressions in the occlusal portion of a tooth that are susceptible to dental caries, some scholars are using the phrase "groove-fossa system" [20]. In Pedodontics, this is especially important for distinguishing caries prone areas in deciduous teeth and first permanent molars. The pit created by the intersection of developmental grooves on the molar occlusal surface is a fascinating anatomical feature. It is also a very complicated region for dental caries on the occlusal surface [21]. Occlusal dentine caries that are missing on external inspection are referred to as concealed tooth decay. A relatively small open cavity is found between grooves in most cases of concealed caries. The point is formed when occlusal surface ridges meet at a focal place in the depression's bottom [22]. It's a juncture of grooves, also known as a minor physiological anatomical depression, that's easy to spot as a bacterial growth hotspot. A cavity may form beneath the resistant enamel as a result of continuous deep demineralization [23]. The mesiobuccal groove is a unique feature of mandibular first molars. It's a kind of sulcus that runs from the occlusal to buccal surfaces and is often unnoticed during dental examinations [24]. This narrow groove, though often shallow, can host a small but active bacterial population that can easily cause cavitation [25]. On comparison between gender and tooth number, it was revealed that the chronic pulpitis was most prevalent in females particularly in 36 and 46 (Fig. 4). This could be because girls may be exposed to the oral atmosphere, microbes and bacterial substrates for a longer period of time than a boy of the same age, creating more opportunities for caries [26].

The limitation of our study is minimum external validity and also the validity can be extended by

encompassing subjects of a wider demographic range. From our study it can be concluded that the chronic pulpitis was most prevalent in females in the age of 16 years particularly in mandibular molars. Hence, preventive measures can be done even before the caries could further progress to avoid complications to the young pediatric patients.

5. CONCLUSION

From our study it can be concluded that the chronic pulpitis was most prevalent in females in the age group of 16 years particularly in mandibular molars. So by knowing the prevalence and pathophysiology of chronic pulpitis, dental clinicians can prevent progression of dental caries.

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Rechenberg DK, Galicia JC, Peters OA. Biological Markers for Pulpal Inflammation: A Systematic Review [Internet]. PLOS ONE. 2016;11:e0167289. Available: <http://dx.doi.org/10.1371/journal.pone.0167289>
2. Ali SG, Mulay S. Pulpitis: A review. International of Dental and Medical Science [Internet]; 2015. Available: https://www.researchgate.net/profile/Gufaran_Syed/publication/281228037_Pulpitis_A_review/links/55dbffaf08aeb38e8a8b97a1/Pulpitis-A-review.pdf
3. Yamunadevi A, Pratibha R, Rajmohan M, Ganapathy N, Porkodisudha J, Pavithrah D, et al. Molecular Insight into Odontogenesis in Hyperglycemic

- Environment: A Systematic Review. *J Pharm Bioallied Sci.* 2020;12(Suppl 1):S49–56.
4. Giuroiu CL, Căruntu ID, Lozneau L, Melian A, Vataman M, Andrian S. Dental Pulp: Correspondences and Contradictions between Clinical and Histological Diagnosis. *Biomed Res Int.* 2015;2015: 960321.
 5. Govula K. Estimation of the Prevalence of Pulpitis in the Tertiary Care Hospital in Nellore district-A cross sectional study [Internet]. [Cited 2021 May 11]. Available: https://www.researchgate.net/profile/Kiranmayi_Govula/publication/338229885_Estimation_of_the_Prevalence_of_Pulpitis_in_the_Tertiary_Care_Hospital_in_Nellore_district-A_cross_sectional_study/links/5e09aeb9299bf10bc382cd15/Estimation-of-the-Prevalence-of-Pulpitis-in-the-Tertiary-Care-Hospital-in-Nellore-district-A-cross-sectional-study.pdf
 6. Suvarna K, Abilasha R, Gheena S. Analysis of Prevalence of Oral Squamous Cell Carcinoma in Patients with History of Chronic Irritation of Oral Tissues-A Retrospective Study. *Indian Journal of [Internet]*; 2020. Availabl: <http://search.ebscohost.com/login.aspx?direct=true&profile=ehost&scope=sit e&authtype=crawler&jrnl=09739122&AN=148410023&h=XYlwRUZ0p54Y6mZHESI7LVi228pkq%2FRhoPO4Jt4231oGPh6KuqA4bM6e9ZsIMs9HCBZY6ZF8tl2TIR3cgyExXQ%3D%3D&crl=c>
 7. Mitkova Damyanova D, Angelova S, Andreeva-Borisova R. Estimation of pulpitis prevalence in primary dentition. *Dent Res Oral Health.* 2018;01(03): 29–33.
 8. Umashankar K, Abilasha, Hannah, Ramani P, Gheena. Knowledge and attitude about COVID-19 pathogenesis among oral pathologists in Chennai. *Int J Curr Res Rev.* 2020;12(19):143–51.
 9. Monica K, Gheena S, Ramani P. In silico gene expression analysis of crucial cell cycle control gene *cdkn2a* and *cdkn2b* in head and neck squamous cell *Annals of [Internet]*; 2020. Available: https://www.journal.atmph-specialissues.org/abstract.php?article_id=8921
 10. Krishnan RP, Ramani P, Sukumaran G, Ramasubramanian A, Karunagaran M, Hannah R. Workplace violence among dental surgeons - A Survey; 2021 May 19 [Cited 2021 May 19]. Available: <https://www.ijdr.in/preprintarticle.asp?id=315914;type=0>
 11. Sinduja P, Ramani P, Gheena S, Ramasubramanian A. Expression of metallothionein in oral squamous cell carcinoma: A systematic review. *J Oral Maxillofac Pathol.* 2020;24(1):143–7.
 12. Ramani P, Krishnan RP, Karunagaran M, Muthusekhar MR. Odontogenic sarcoma: First report after new who nomenclature with systematic review. *J Oral Maxillofac Pathol.* 2020;24(1):157–63.
 13. Camposi FAT, Siqueira MFG, Ribeiro ILA. Prevalence of pulp therapy in deciduous teeth performed in UNIPÊ Clinical Dentistry School. *Revista Cubana de [Internet]*; 2016. Available: <https://www.medigraphic.com/cgi-bin/new/resumenl.cgi?IDARTICULO=72011>
 14. Tedesco TK, Reis TM, Mello-Moura ACV, Silva GS da, Scarpini S, Floriano I, et al. Management of deep caries lesions with or without pulp involvement in primary teeth: a systematic review and network meta-analysis. *Braz Oral Res.* 2020;35:e004.
 15. Tiwari S, Dubey A, Singh B, Avinash A. Clinical consequences of untreated dental caries evaluated with the pulpal involvement-roots-sepsis index in the primary dentition of school children from the Raipur and Durg districts, Chhattisgarh state, India. *Med Princ Pract.* 2015;24(2):184–8.
 16. Ramani P, Gheena S, Karunagaran M, Hannah R. Clear-cell variant of oral squamous cell carcinoma: A rare entity. *J Oral Maxillofac Pathol.* 2021;25(4):22.
 17. Settineri S, Tati F, Fanara G. Gender differences in dental anxiety: is the chair position important? *J Contemp Dent Pract.* 2005;6(1):115–22.
 18. Martin FE, Nadkarni MA, Jacques NA, Hunter N. Quantitative microbiological study of human carious dentine by culture and real-time PCR: association of anaerobes with histopathological changes in chronic pulpitis. *J Clin Microbiol.* 2002;40(5):1698–704.
 19. Ramasubramanian A, Ramani P, Sherlin HJ, Premkumar P, Natesan A, Thiruvengadam C. Immunohistochemical evaluation of oral epithelial dysplasia using

- cyclin-D1, p27 and p63 expression as predictors of malignant transformation. *J Nat Sci Biol Med.* 2013;4(2): 349–58.
20. E A, Aswani E, Gheena S, Pratibha R, Abilasha R, Hannah R, et al. Overexpression of HNRNPA2B1 is Associated with Poor Prognosis in Head and Neck Squamous Cell Carcinoma [Internet]. *International Journal of Current Research and Review.* 2020:15–8. Available: <http://dx.doi.org/10.31782/ijcrr.2020.122502>
21. Behera A, Hannah R. Association of the Depth of Invasion with Lymph Node Metastasis in Oral Squamous Cell Carcinoma Patients-A Retrospective Study. *Indian Journal of [Internet];* 2020. Available: <http://search.ebscohost.com/login.aspx?direct=true&profile=ehost&scope=site&authtype=crawler&jrnl=09739122&AN=148410053&h=nwnHkKvd5DGQXazZu6t5p6l8D0PbUqebPTLzhmdSl6FyXTWsSzGTNdBhQxTZUG9F%2BD8YcgHrW%2FSaXuV%2BCg9vCA%3D%3D&crl=c>
22. Sukumaran G, Ramani P, Ramasubramanian A, Karunakaran M, Ravikumar H. Implantation Dermoid Cyst [Internet]. [Cited 2021 May 11]. Available: https://www.researchgate.net/profile/DrAbilasha_Ramasubramanian/publication/339048652_Implantation_Dermoid_Cyst/links/5fb254b4299bf10c36833e88/Implantation-Dermoid-Cyst.pdf
23. Thamilselvan S, Abilasha R, Ramani P, Gheena S, Hannah R. Evaluation of Accuracy between Habit History and Incidence of Oral Squamous Cell Carcinoma [Internet]. *International Journal of Current Research and Review.* 2020:30–5. Available: <http://dx.doi.org/10.31782/ijcrr.2020.122503>
24. de Paiva MAA, Leite DFBM, Farias IAP, Costa A de PC, Sampaio FC. Dental anatomical features and caries: A relationship to be investigated. In: *Dental Anatomy.* Intechopen; 2017.
25. A study on the variability of drug responsiveness to anti inflammatory drugs - A pilot survey. *Int J Pharm Res [Internet].* 2020 Oct 2;12(02). Available: <http://www.ijpronline.com/ViewArticleDetail.aspx?ID=17202>
26. Ferraro M, Vieira AR. Explaining gender differences in caries: a multifactorial approach to a multifactorial disease. *Int J Dent.* 2010;2010:649643.

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