PREVALENCE OF CHRONIC PULPITIS AMONG 13 TO 17 YEAR OLD PEDIATRIC PATIENTS VISITING PRIVATE DENTAL INSTITUTION IN CHENNAI - A RETROSPECTIVE STUDY

Running Title: A retrospective analysis of the prevalence of chronic pulpitis in pediatric patients within the age group of 13 to 17 years visiting private dental hospital in Chennai.

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 by knowing the prevalence and pathophysiology of chronic pulpitis, dental clinicians can prevent progression of this condition and

Keywords:

Prevalence; Chronic pulpitis, pediatric, prevention, innovative technology, novel method

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 this. 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 dominatingly 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, that 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.

Our team has extensive knowledge and research experience that has translate into high quality publications(16),(17),(18),(19),(20),(21),(22),(23),(24),(25),(26),(27),(28),(29),(30),(31),(32),(33),(34),(35)

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.

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 [Figure 1]. Almost over half of the sample population afflicted by chronic pulpitis (63.64%) were female and only 36.36% were male [Figure 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%). 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(Figure 3). In figure 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.

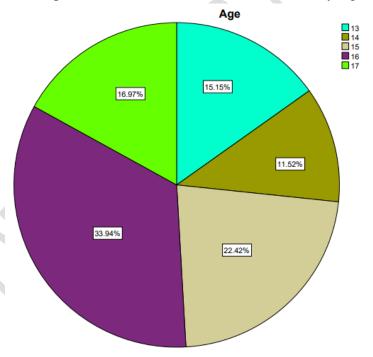


Figure 1: This pie chart represents the distribution of chronic pulpitis with regards to age. Most of the people who are likely to be affected with chronic pulpitis were 16 years of age (33.94%), 22.42% were 15 years old, 16.97% were 17 years old, 15.15% were 13 years old and 11.52% were 14 years old.

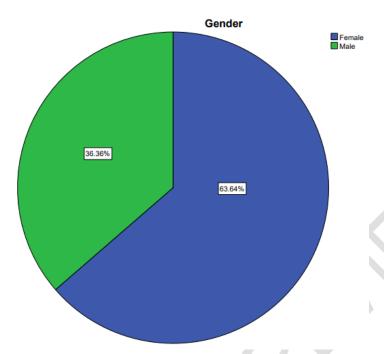


Figure 2: This pie chart represents the distribution of chronic pulpitis with regards to gender. Blue colour in the chart represents female and green represents male. Nearly more than half of the study population who are affected with chronic pulpitis (63.64%) were female and only 36.36% of the population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

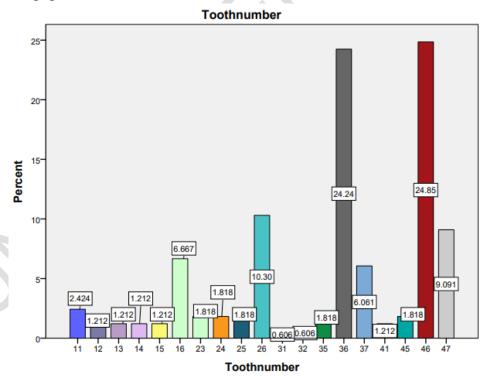
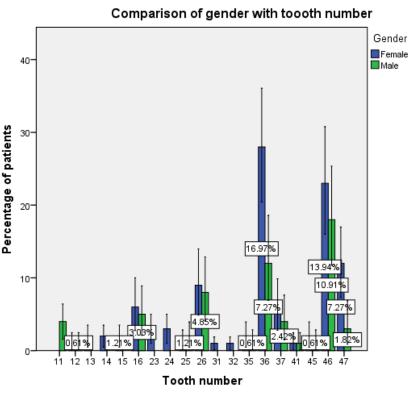


Figure 3: This bar chart represents the prevalence of chronic pulpitis with regards to tooth number. X-axis represents the tooth number and Y-axis represents the count of teeth affected with chronic pulpitis. The count of tooth number 46 affected with chronic pulpitis was found to

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.



Error Bars: 95% CI

Figure 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.

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% (figure 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 (36). 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) (37).

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 (38). 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 (figure 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 (39). 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" (40). 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 (41). 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 (42). 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 (43). 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 (44). This narrow groove, though often shallow, can host a small but active bacterial population that can easily cause cavitation (45). On comparison between gender and tooth number, it was revealed that the chronic pulpitis was most prevalent in females particularly in 36 and 46 (figure 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 (46).

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.

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.

REFERENCE:

- 1. Rechenberg D-K, Galicia JC, Peters OA. Biological Markers for Pulpal Inflammation: A Systematic Review [Internet]. Vol. 11, PLOS ONE. 2016. p. e0167289. Available from: 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 from: 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 Aug;12(Suppl 1):S49–56.
- 4. Giuroiu CL, Căruntu I-D, Lozneanu L, Melian A, Vataman M, Andrian S. Dental Pulp: Correspondences and Contradictions between Clinical and Histological Diagnosis. Biomed Res Int. 2015 May 11;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 from: 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; Available from: http://search.ebscohost.com/login.aspx?direct=true&profile=ehost&scope=site&authtype=c rawler&jrnl=09739122&AN=148410023&h=XYlwRUZ0p54Y6mZHESI7LVi228pkq%2F RhoPO4Jt4231oGPh6KuqA4bM6e9ZslMs9HCBZY6ZF8tl2TlR3cgyExXQ%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 from: 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 from: 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 Jan;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 Jan;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 from: https://www.medigraphic.com/cgibin/new/resumenI.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 Nov 13;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. Princeton B, Santhakumar P, Prathap L. Awareness on Preventive Measures taken by Health Care Professionals Attending COVID-19 Patients among Dental Students. Eur J Dent. 2020 Dec;14(S 01):S105–9.
- 17. Mathew MG, Samuel SR, Soni AJ, Roopa KB. Evaluation of adhesion of Streptococcus mutans, plaque accumulation on zirconia and stainless steel crowns, and surrounding gingival inflammation in primary molars: randomized controlled trial. Clin Oral Investig. 2020 Sep;24(9):3275–80.
- 18. Sridharan G, Ramani P, Patankar S, Vijayaraghavan R. Evaluation of salivary metabolomics in oral leukoplakia and oral squamous cell carcinoma. J Oral Pathol Med.

- 2019 Apr;48(4):299–306.
- 19. R H, Hannah R, Ramani P, Ramanathan A, Jancy MR, Gheena S, et al. CYP2 C9 polymorphism among patients with oral squamous cell carcinoma and its role in altering the metabolism of benzo[a]pyrene [Internet]. Vol. 130, Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology. 2020. p. 306–12. Available from: http://dx.doi.org/10.1016/j.oooo.2020.06.021
- 20. Antony JVM, Ramani P, Ramasubramanian A, Sukumaran G. Particle size penetration rate and effects of smoke and smokeless tobacco products An invitro analysis. Heliyon. 2021 Mar 1;7(3):e06455.
- 21. Sarode SC, Gondivkar S, Sarode GS, Gadbail A, Yuwanati M. Hybrid oral potentially malignant disorder: A neglected fact in oral submucous fibrosis. Oral Oncol. 2021 Jun 16;105390.
- 22. Hannah R, Ramani P, WM Tilakaratne, Sukumaran G, Ramasubramanian A, Krishnan RP. Author response for "Critical appraisal of different triggering pathways for the pathobiology of pemphigus vulgaris—A review" [Internet]. Wiley; 2021. Available from: https://publons.com/publon/47643844
- 23. Chandrasekar R, Chandrasekhar S, Sundari KKS, Ravi P. Development and validation of a formula for objective assessment of cervical vertebral bone age. Prog Orthod. 2020 Oct 12;21(1):38.
- 24. Subramanyam D, Gurunathan D, Gaayathri R, Vishnu Priya V. Comparative evaluation of salivary malondialdehyde levels as a marker of lipid peroxidation in early childhood caries. Eur J Dent. 2018 Jan;12(1):67–70.
- 25. Jeevanandan G, Thomas E. Volumetric analysis of hand, reciprocating and rotary instrumentation techniques in primary molars using spiral computed tomography: An in vitro comparative study. Eur J Dent. 2018 Jan;12(1):21–6.
- 26. Ponnulakshmi R, Shyamaladevi B, Vijayalakshmi P, Selvaraj J. In silico and in vivo analysis to identify the antidiabetic activity of beta sitosterol in adipose tissue of high fat diet and sucrose induced type-2 diabetic experimental rats. Toxicol Mech Methods. 2019 May;29(4):276–90.
- 27. Sundaram R, Nandhakumar E, Haseena Banu H. Hesperidin, a citrus flavonoid ameliorates hyperglycemia by regulating key enzymes of carbohydrate metabolism in streptozotocininduced diabetic rats. Toxicol Mech Methods. 2019 Nov;29(9):644–53.
- 28. Alsawalha M, Rao CV, Al-Subaie AM, Haque SKM, Veeraraghavan VP, Surapaneni KM. Novel mathematical modelling of Saudi Arabian natural diatomite clay. Mater Res Express. 2019 Sep 4;6(10):105531.
- 29. Yu J, Li M, Zhan D, Shi C, Fang L, Ban C, et al. Inhibitory effects of triterpenoid betulin on inflammatory mediators inducible nitric oxide synthase, cyclooxygenase-2, tumor necrosis

- factor-alpha, interleukin-6, and proliferating cell nuclear antigen in 1, 2-dimethylhydrazine-induced rat colon carcinogenesis. Pharmacogn Mag. 2020;16(72):836.
- 30. Shree KH, Hema Shree K, Ramani P, Herald Sherlin, Sukumaran G, Jeyaraj G, et al. Saliva as a Diagnostic Tool in Oral Squamous Cell Carcinoma a Systematic Review with Meta Analysis [Internet]. Vol. 25, Pathology & Oncology Research. 2019. p. 447–53. Available from: http://dx.doi.org/10.1007/s12253-019-00588-2
- 31. Zafar A, Sherlin HJ, Jayaraj G, Ramani P, Don KR, Santhanam A. Diagnostic utility of touch imprint cytology for intraoperative assessment of surgical margins and sentinel lymph nodes in oral squamous cell carcinoma patients using four different cytological stains. Diagn Cytopathol. 2020 Feb;48(2):101–10.
- 32. Karunagaran M, Murali P, Palaniappan V, Sivapathasundharam B. Expression and distribution pattern of podoplanin in oral submucous fibrosis with varying degrees of dysplasia an immunohistochemical study [Internet]. Vol. 42, Journal of Histotechnology. 2019. p. 80–6. Available from: http://dx.doi.org/10.1080/01478885.2019.1594543
- 33. Sarode SC, Gondivkar S, Gadbail A, Sarode GS, Yuwanati M. Oral submucous fibrosis and heterogeneity in outcome measures: a critical viewpoint. Future Oncol. 2021 Jun;17(17):2123–6.
- 34. Raj Preeth D, Saravanan S, Shairam M, Selvakumar N, Selestin Raja I, Dhanasekaran A, et al. Bioactive Zinc(II) complex incorporated PCL/gelatin electrospun nanofiber enhanced bone tissue regeneration. Eur J Pharm Sci. 2021 May 1;160:105768.
- 35. Prithiviraj N, Yang GE, Thangavelu L, Yan J. Anticancer Compounds From Starfish Regenerating Tissues and Their Antioxidant Properties on Human Oral Epidermoid Carcinoma KB Cells. In: PANCREAS. LIPPINCOTT WILLIAMS & WILKINS TWO COMMERCE SQ, 2001 MARKET ST, PHILADELPHIA ...; 2020. p. 155–6.
- 36. Ramani P, Gheena S, Karunagaran M, Hannah R. Clear-cell variant of oral squamous cell carcinoma: A rare entity. J Oral Maxillofac Pathol. 2021 Mar 1;25(4):22.
- 37. Settineri S, Tati F, Fanara G. Gender differences in dental anxiety: is the chair position important? J Contemp Dent Pract. 2005 Feb 15;6(1):115–22.
- 38. 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 May;40(5):1698–704.
- 39. 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 Jul;4(2):349–58.
- 40. 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. p. 15–8. Available from: http://dx.doi.org/10.31782/ijcrr.2020.122502
- 41. Behera A, Hannah R. Association of the Depth of Invasion wth Lymph Node Metastasis in Oral Squamous Cell Carcinoma Patients-A Retrospective Study. Indian Journal of [Internet]. 2020; Available from: http://search.ebscohost.com/login.aspx?direct=true&profile=ehost&scope=site&authtype=c rawler&jrnl=09739122&AN=148410053&h=nwnHkKvd5DGQXazZu6t5p6l8D0PbUqebP TLzhmdSl6FyXTWsSzGTNdBhQxTZUG9F%2BD8YcgHrW%2FSaXuV%2BCg9vCA%3 D%3D&crl=c
- 42. Sukumaran G, Ramani P, Ramasubramanian A, Karunagaran M, Ravikumar H. Implantation Dermoid Cyst [Internet]. [cited 2021 May 11]. Available from: https://www.researchgate.net/profile/DrAbilasha_Ramasubramanian/publication/339048652 _Implantation_Dermoid_Cyst/links/5fb254b4299bf10c36833e88/Implantation-Dermoid-Cyst.pdf
- 43. 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. p. 30–5. Available from: http://dx.doi.org/10.31782/ijcrr.2020.122503
- 44. 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.
- 45. 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 from: http://www.ijpronline.com/ViewArticleDetail.aspx?ID=17202
- 46. Ferraro M, Vieira AR. Explaining gender differences in caries: a multifactorial approach to a multifactorial disease. Int J Dent. 2010 Mar 16;2010:649643.