

GENERAL INFORMATION

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Editorial

Publication and Scientific Writing

Editors and researchers are looking for original and innovative research that will add to the field of study. The task of writing a research paper can be daunting. You may have completed ground breaking research, but unless the paper is correctly written, at best publication will be delayed, at worse never published. Scientific writing follows a rigid structure; a format developed over hundreds of years, and considered to be the most efficient means for communicating scientific findings to the broader research community.

Every finding released by a scientific publication is expected to provide an area of study with new solutions and insights. Researchers communicate their conclusions to their academic peers, by publication in a scientific journal. Scientific writing is a mode of communication that enables researchers to present conclusions effectively. However, this issue has only been treated with linguistic technicality worldwide. Practical rules have been imposed to ensure that a manuscript is written concisely and with clarity, and has objectivity and logic.

Our post graduate courses offer training in research methodology, but we have to incorporate into our curriculum the art of scientific writing. This can help maximize the accuracy and impact of a written document while enhancing its publication and success. Unless published, a research is incomplete.

Dr. Pradeep Kumar. C
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NUTRIGENOMICS IN PERIODONTICS-WILL YOUR GENE SAY WHAT YOU SHOULD EAT?

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Abstract

Health of an individual is reflection of his nutritional status. Nutrition may be important in redressing the balance between microbial challenge and the host response because it has been implicated in a number of inflammatory diseases and conditions, including type II diabetes mellitus, cardiovascular diseases and rheumatoid arthritis all of which have also been associated with periodontal diseases. The observed differences in the response of an individual to dietary modification can be attributed to differences in their genetic make-up, which emphasizes the importance of exploring the role of nutrient–gene interactions in the development of chronic diseases. Nutrigenomics has emerged as a rapidly developing research area with great potential to yield findings that could change the way dietary guidelines for populations and recommendations for individuals are established and advised in the future.

Keywords: Nutrigenomics, Metabolomics, Proteomics, Transcriptomics, Periodontitis

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Introduction

"Leave your drugs in the chemist's pot if you can heal the patient with food."

Hippocrates

Since the times of the renowned Greek physician Hippocrates, nutrition has clearly had a predominant and recognizable role in health management. The health of an individual is reflection of his nutritional status. It is not only naïve, but also likely dangerous, to assume all individuals will respond identically to the foods they consume. The observed differences in the response of an individual to dietary

modification can be attributed to differences in their genetic make-up. And this difference emphasizes the importance of exploring the role of nutrient–gene interactions in the development of chronic diseases.

Nutrition is identified as one of the key factors modulating host immune response. It has been studied for many years that nutritional intake has an effect on the levels of inflammation in various inflammatory diseases including periodontitis.¹ The interaction between genetic and dietary influences can result in a higher risk of disease in certain individuals and populations.²

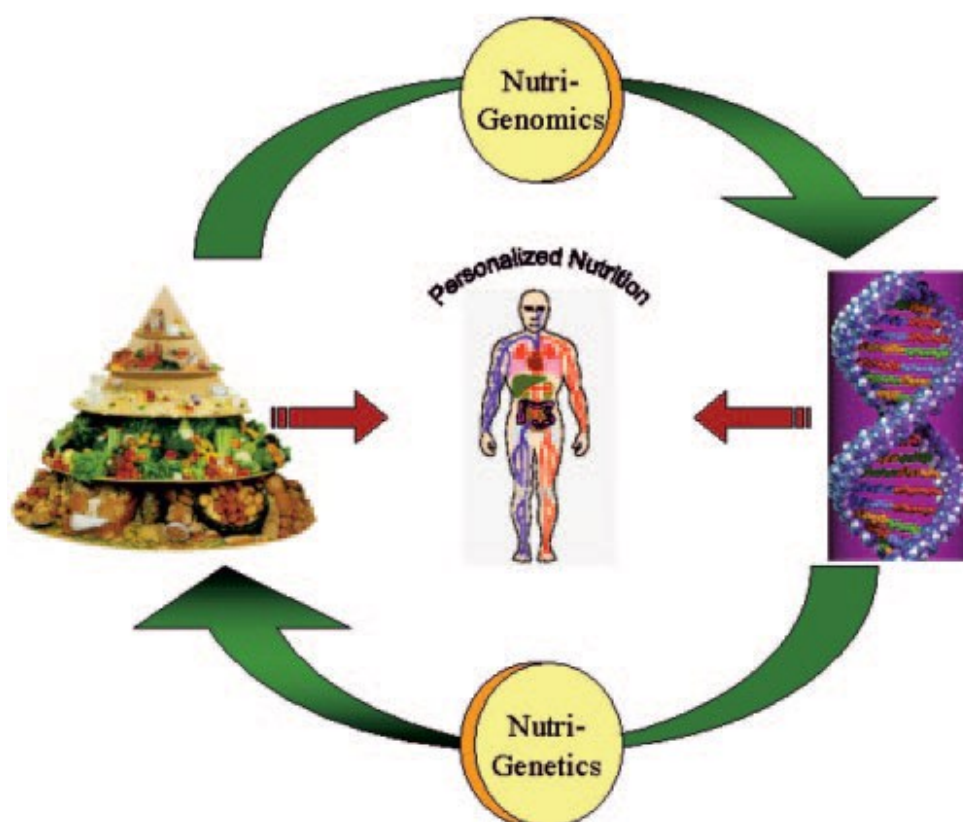


Fig 1: Nutrigenomics and nutrigenetics- two sides of a coin.

(Picture courtesy: *Nutrigenomics and nutrigenetics: the emerging faces of nutrition FASEB J. 19, (2005) pg.1603*)

Gingivitis and periodontitis are chronic infectious microbial diseases characterized by a dysregulated host inflammatory or immune response to plaque microorganisms in susceptible individuals. Many essential nutrients have immune modulatory properties; hence nutrition has correlation to the natural resolution of the periodontal diseases.³

The development of a personalising approach to nutrition for disease prevention and therapy will require a much more comprehensive understanding of nutrient-gene interactions and their impact on phenotype in order to identify, evaluate, and

prioritise appropriately targeted strategies for dietary intervention.²

What is nutrigenomics?

Nutrigenomics describes the scientific approach that integrates nutritional sciences and genomics and includes the application of other high-throughput 'omics' technologies such as transcriptomics, proteomics and metabolomics to investigate the effects of nutrition on health.²

Transcriptomics is the term used to describe the approach in which mRNA, and consequently gene expression, is analysed in a biological sample under certain conditions at a given point in time. Proteomics take this

analysis further and aims to characterize all proteins in a biological sample at the functional level. Metabolomics is used to describe quantitative analysis of all metabolites in a biological system such as cell, tissue or biological fluid (blood, plasma and saliva).⁴

Nutrigenomics is an umbrella term for two complimentary approaches: how nutrients affect gene function and how genetic variation affects nutrient response. The latter is sometimes referred to as nutrigenetics⁵ (Fig 1), and includes the study of how genetic variations affect food intake and eating behaviours.⁶

A major challenge of nutrigenomics, however, is to identify gene-nutrient interactions that affect complex polygenic disorders such as obesity, diabetes, cancer and cardiovascular disease that take several years – or even decades – to develop and have multifactorial etiologies. Thus the major goal of nutrigenomics is the prevention of the onset and progression of such chronic disease. Current research could lead to the development of personalized nutrition guidelines for individuals and specific sub-populations, which could decrease the risk of chronic diseases.²

Time line

- In 1997, the first nutrigenomics company was launched.
- In 1999, the name nutritional genomics was changed to genomics by Nancy Fogg-Johnson and Alex Merolli which provides powerful means of discovering hereditary factors in disease.

- The term “nutrigenomics” was first described in 2001 from Pelegrin and then, it appears in 2002 in a review by Van Ommen and Stierum.
- April 14, 2003-Human Genome Project was launched. The precise date of birth of the genomic era and the ‘omics’-revolution.
- In 2004, NuGo (European Nutrigenomics Organization) was born and funded until June 2010.⁷

Diet and genetics

There are many factors that can influence the response to diet such as age, sex, physical activity, smoking and genetics. The daily ingestion, absorption, digestion, transport, metabolism and excretion of nutrients and food bioactives involve many proteins such as enzymes, receptors, transporters, ion channels and hormones. Variations in genes encoding proteins that affect any of these processes can alter both the amount of the protein produced as well as how well that protein functions. If a genetic variation leads to altered production or function of these proteins, then nutritional status might be affected.²

The concept of diet-gene interactions involves a genetic variant modulating the effect of a dietary factor on a specific phenotype or health outcome measure such as serum lipid concentrations, high blood glucose or obesity. Conversely, diet-gene interactions can refer to the dietary modification of the effect of a genetic variant on the phenotype or health outcome measure.⁸

The fundamental hypotheses underpinning the science of nutrigenomics: First there is great diversity in the inherited genome between ethnic groups and individuals which affects nutrient bioavailability and metabolism. Second, people differ greatly in their food/nutrient availability and choices depending on cultural, economical, geographical and taste perception differences. Third malnutrition (deficiency or excess) itself can affect gene expression and genome stability; the latter leading to mutations at the gene sequence or chromosomal level which may cause abnormal gene dosage and gene expression leading to adverse phenotypes during the various life stages.⁹

Epigenetics

An important emerging aspect of nutrient-gene interaction studies with the potential for both intra- and transgenerational effects is epigenetics. Epigenetics refers to the processes that regulate how and when certain genes are turned on and off, while epigenomics pertains to analysis of epigenetic changes in a cell or entire organism.¹⁰

Diet on its own or by interaction with other environmental factors can cause epigenetic changes that may turn certain genes on or off. Epigenetic silencing of genes that would normally protect against a disease, as a result, could make people more susceptible to developing that disease later in life. The epigenome which is heritable and modifiable by diet is the global epigenetic pattern determined by global and gene-specific DNA methylation, histone modifications and chromatin-associated

proteins which control expression of house-keeping genes and suppress the expression of parasitic DNA such as transposons.⁹

In the past, epigenetics was focused on embryonic development, aging and cancer. Presently, epigenetics is exceptionally important, because nutrients and bioactive food components can alter the expression of genes at the transcriptional level. In recent years, epigenetics has become an emerging issue in broad range of diseases such as type 2 diabetes, obesity, periodontal diseases and neurocognitive disorders.⁷

Role of nutrigenomics in periodontal disease

Periodontitis represents a heterogeneous multifactorial group of infectious diseases with destructive inflammatory pathogenesis that lead to destruction of the supporting tissues of the teeth. If the inflammatory response fails to remove the causative pathogens, the prolonged release of neutrophils, proteolytic enzymes, proinflammatory mediators and reactive oxygen species occurs, which in turn destroy the periodontal attachment. Host based risk factors such as genetic background of an individual, socio-economic status, smoking and dietary habits have all been suggested to alter the innate susceptibility of the host to periodontal disease.¹¹

Improved understanding of the mechanism behind periodontal tissue destruction, the potential protective role of nutrients and the advent of modern genomic measurement tools have led to an increased interest in the association between nutrition and periodontal disease.

Recently it has been suggested that nutrition may be important in redressing the balance between microbial challenge and the host response because it has been implicated in a number of inflammatory diseases and conditions, including type 2 diabetes, cardiovascular disease, rheumatoid arthritis and inflammatory bowel disease, all of which have also been associated with periodontal disease.¹²

Specific nutrients (antioxidant vitamins A, C, E) and trace element selenium, copper, zinc can modulate the immune and inflammatory responses that maintain epithelial cell integrity and structure. The nutrients get depleted during inflammation with generation of Reactive Oxygen Species (ROS) causing damage to the cellular tissues.¹³

Moreover, selenium has further important redox functions, with selenium-dependent glutathione enzymes being involved in the reduction of damaging lipid and phospholipid hydroperoxides to harmless products.

A rodent model of zinc deficiency has shown to have an increased susceptibility to periodontal disease progression, as revealed by increased plaque and higher gingival index measurements.¹⁴ As an example of the potential of nutrigenomics tool for assessing the role of nutrition in periodontal disease, the role of the micronutrient zinc, a zinc transporter gene and the risk of developing type 2 diabetes was investigated. Recent genome-wide association studies have identified a genetic-susceptibility locus for type 2 diabetes comprising a nonsynonymous single nucleotide polymorphism (C/T;

rs13266634) in a β cell-specific zinc-transporter gene. This zinc transporter gene (SLC30A8, coding for ZnT8) may be important in insulin storage and release.¹⁵ In light of this new finding, the existing knowledge that zinc has a specific role in beta cell function takes on new significance with respect to potential strategies to prevent or treat type-2 diabetes and potentially periodontal disease.⁷

Vitamin C acts as a powerful scavenger of free radical. The association between low intake of vitamin C and occurrence of periodontitis has been demonstrated, in a study by Nissada 2010.¹²

Vitamin E terminates free radical chain reaction and stabilizes membrane structure. It is shown to have mitigatory effects on inflammation and collagen breakdown. A low level of vitamin E in gingival tissues of periodontitis patients has been reported.⁷

Omega 3 fatty acids such as n-3 PUFA (oily fish), increase the tissue concentration of eico-sapentaenoic acid, decosa-hexaenoic acid and down-regulate inflammation and inhibit bone loss in vitro.⁷

It has also been reported that the n-6 PUFA levels in the serum are higher in periodontitis patients, suggesting that an imbalance between n-6 and n-3 fatty acids may contribute to susceptibility to alveolar bone loss.⁷

Results from a prospective, observational study carried out over 14 years revealed that men with high consumption of wholegrain were 23% less likely to develop periodontitis than those who had the lowest consumption of wholegrain.⁷

The main functional value of pomegranate in oral health is its polyphenolic flavonoid content. The components of pomegranate juice were found to significantly inhibit cytokine IL-8, PGE₂, nitric oxide, human salivary α -amylase, α -glucosidase activity and found to reduce aspartate aminotransferase activity in saliva. The hydro-alcoholic extract from pomegranate fruit has shown to decrease the Colony Forming Unit (CFU) per milliliters of dental plaque by 84%. Local action and topical effects of antioxidant agents from pomegranate on the oral tissues have been hypothesized to have preventive effect against diseases of the oral cavity. One minute rinsing with a mouthwash containing pomegranate extract successfully reduced the amount of microorganisms cultured from dental plaque.¹⁶

The limited evidence linking the cause and prevention of periodontal disease, based on observational, cross-sectional or very small double-blind randomized supplementation trials, highlights the enormous scope for the use of nutritional genomics (nutrigenomics) to understand these links in more detail. Oral health scientists now have the opportunity to study nutrient–gene interactions and how diet affects the inflammatory mechanisms underlying severe periodontitis.

Nutritional modulation of periodontal inflammation

Periodontitis is initiated by the plaque biofilm, but most tissue destruction results from an abnormal inflammatory immune response in patients predisposed to the condition. The response is characterized

by hyperinflammation, which fails to eradicate the causative pathogens and generates prolonged release of neutrophil proteolytic enzymes, proinflammatory mediators and reactive oxygen species (ROS), which in turn destroy the periodontal attachment. The etiology of an inflated inflammatory immune response is complex, but several common features of hyperinflammation underlie the major chronic diseases of humans.¹⁷

Diets high in complex carbohydrates are generally healthy, whereas those rich in refined carbohydrates can be major causes of chronic inflammation. Elevated glucose and lipid levels generate ROS at a rate that exceeds endogenous antioxidant defenses, and oxidative stress results. Investigators have noted that this “postprandial dysmetabolism” plays a role in the genesis of inflammation. Multiple elevations in glucose eventually lead to chronic inflammatory pathologies.¹⁸ Diet-induced hyperlipidemia induces oxidative stress and downstream inflammation, and lipoproteins formed by liver hepatocytes can be converted to free fatty acids within the circulation and taken up by adipocytes, thus acting as a basis of proinflammatory adipokines. Furthermore, in states of oxidative stress, lipid peroxidation (a chain reaction induced by ROS attack on the polyunsaturated fatty acid [PUFA] side-chains of lipid membranes) arises, low-density lipoproteins are oxidized (oxLDL) and the oxLDLs bind to a group of pattern recognition receptors called “toll-like receptors” (TLR-2/4) on inflammatory cell membranes, triggering NF- κ B activation via the protein-kinase-C enzyme and other

related pathways. NF- κ B transcribes several proinflammatory cytokines.¹⁹

Researchers have proposed reduced glutathione (GSH), the key intracellular antioxidant redox-regulator of NF- κ B, as a novel approach to downregulation of hyperinflammatory events. GSH levels appear depleted in periodontitis and methods of enhancing intracellular GSH may prove beneficial.²⁰

PUFAs of the omega-3 form (ω -3PUFAs) found in fish oils lower postprandial triglyceride levels and confer anti-inflammatory and cardiovascular protective effects, inhibits lipid mediators of inflammation (such as prostaglandin E₂, arachidonic acid, 5-lipoxygenase and cyclooxygenase), modulate lymphokine production and upsurge antioxidant capacity, and are reported to decrease osteoclast activity. Gene expression echelons of IL-1 β and TNF α declined, and those of interferon- γ and intracellular antioxidant enzymes increased. PUFAs downregulates proinflammatory gene expression via the nuclear peroxisome proliferator-activated receptors (PPARs). Inflammation-resolving mediators (resolvins) are derived from ω -3 PUFAs.²¹

Micro nutritional approaches to periodontal therapy

Periodontitis is associated with low serum/plasma micronutrient levels that may result from dietary and/or life-style factors as well as nutrigenetic characteristics. Primary evidence suggests beneficial results from nutritional interventions; supporting the contention that daily intake of certain

nutrients should be at the higher end of recommended daily allowances.

For inhibition and management of periodontitis daily nutrition should include sufficient antioxidants, vitamin D, and calcium. Insufficient antioxidant levels may be managed by higher intake of vegetables, berries, and fruits (e.g. kiwi fruit), or by phytonutrient supplementation.¹² Antioxidant micronutrient deficiencies can be met by a higher intake of vegetables, fruits, and berries. The majority of patients are not antioxidant vitamin deficient and monovitamin supplements may be associated with toxicity effects.

Current evidence shows some benefit in reducing gingival inflammation from vitamin C supplementation, provided UL are not exceeded. The most appropriate sources of polyphenolicflavonoids and carotenoids are from natural fruit/vegetable/ berry intake or the use of whole fruit, vegetable, and berry concentrates. An initial intervention study with a powdered capsular form of the latter phytonutrients showed promise as an adjunctive approach to standard periodontal therapy in improving pocket depth reductions.¹⁹

The recommendations of 2011 European Workshop on Periodontology suggested that the dental team should consider including fish oils, fibre, fruits and vegetables and to reduce levels of refined sugars as part of a periodontal prevention / treatment regime and a general health benefit message.²²

Future of Nutrigenomics

It is becoming increasingly evident that nutrigenomics is taking a central stage in the investigation of the effect of nutrition on health outcomes, and that impacts of nutrients can be evaluated comprehensively by a multitude of 'omic' technologies and biomarkers. Some of these technologies are still in their infancy whilst others are much more mature and therefore differ significantly in their validation status with respect to health outcomes. It is becoming increasingly evident that genetic background, gender and life stage can have an impact on nutritional requirements; however, translation of this knowledge into recommendations based on genotype or at the individual level is only practical in those few cases (e.g. phenylketonuria or galactosemia) when the effect of genotype clearly overwhelms the impact of any other factor and is the ultimate determining factor of the nutritional and health status for an individual or genetic subgroup.¹⁹

The ultimate goal is to (i) match the nutriome (i.e. nutrient intake combination) with the current genome status (i.e. inherited and acquired genome) so that genome maintenance, gene expression, metabolism and cell function can occur normally and in a homeostatically sustainable manner, and (ii) provide better interpretation of data from epidemiological and clinical intervention studies regarding health impacts of dietary factors that may help to revise recommendations for personalised nutrition.²³

It is important to consider whether public health will be improved with individualised tailored recommendations.

How costly will personalised nutrition and counseling be? Will people be motivated to adhere to a tailored diet? Will this approach be a luxury for those with money and education? Moreover, researchers that are implicated into nutrigenomics should probably think more on the ethical aspects of applying nutrigenomics into everyday life.

Conclusion

Dietary intake of a nutrient does not necessarily result in the same concentrations in the blood or tissue because substantial individual variability in the absorption, distribution, metabolism and elimination can exist. The mechanisms responsible for the between-person differences in dietary response are very complex and have been poorly understood. Research to date has indicated that diet-gene interactions play a significant role in this between-person variability, and has clarified some of these genetic differences.

Periodontitis has a multifactorial etiology and is linked to various other chronic systemic diseases that are associated to nutritional imbalance. Several observational studies associates nutrition and periodontal health. Nutrigenomics has emerged as a rapidly developing research area with great potential to yield findings that could change the way dietary guidelines for populations and recommendations for individuals are established and advised in the future.

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ELECTRONIC APEX LOCATORS : A REVIEW

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****Dr. Ravi S.V, *****Dr. Sandeepal

Abstract

The establishment of a correct working length is one of the fundamental parameters for endodontic success. Traditionally this has been determined using radiography, but electronic apex locators are increasingly being used. Electronic apex locators reduce the number of radiographs required and assist where radiographic methods create difficulty. The use of an electronic apex locator in combination with the radiograph is greater precision in the determination of root canal length. They may also indicate cases where the apical foramen is some distance from the radiographic apex. Other roles include the detection of root canal perforation. The aim of this paper is to review the electronic determination of the length of the root canal.

Keywords: working length, endodontics, apex locators

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Introduction

The success rate of conventional root canal treatment is predictably high, as long as the basic principles of endodontic treatment are followed. Accurate determination of root canal length is particularly important to the success of root canal treatment. The outcome of treatment of roots with necrotic pulps and periapical lesions is influenced significantly by the apical level of the root filling.¹

Traditionally, the point of termination for endodontic instrumentation and obturation has been determined by taking radiographs. The development of the electronic apex locator has helped make the assessment of working length more accurate and predictable.² The electronic method eliminates many of the problems associated with radiographic measurements. It is most

important advantage over radiography is that it can measure the length of the root canal to the end of the apical foramen (AF), not to the radiographic apex.³

The aim of this review is to examine the variety of apex locators available and overview their development.

Morphology of the root canal terminus

Kuttler (1955) concluded that a root canal had two main sections, a longer conical section in the coronal region consisting of dentine and a shorter funnel-shaped section consisting of cementum located in the apical portion.⁴ The shape of this apical portion is considered to be an inverted cone (Fig. 1); its base being located at the major apical foramen. The apex of the inverted cone is the minor foramen that is often thought to coincide with the apical

constriction regarded as being at or near the cemento-dentinal junction (CDJ).⁵ In other words, the most apical portion of the root canal system narrows from the opening of the major foramen, which is within cementum, to a constriction (minor foramen) before widening out in the main canal to produce an hourglass shape (Fig. 1). Apex locators are used to locate the apical foramen by determining the apical constriction or cemento-dentinal junction.

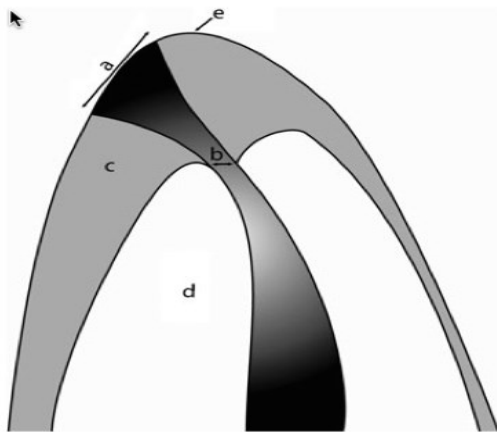


Fig 1: Idealized anatomy of apical portion of root: (a) major apical foramen (b) minor apical foramen (apical constriction) that may be coincident with CDJ (c) cementum (d) dentine and (e) root apex.

Electronic method of determining working length: electronic apex locators (EAL)

All EALs function by using the human body to complete an electrical circuit. One side of the apex locator's circuitry subsequently is connected to the oral mucosa through a lip clip and the other side to a file. When the file is placed into the root canal and advanced apically until its tip touches periodontal tissue at the apex, the electrical circuit is completed. With an apex

locator the working length is determined by comparing the electrical resistance of the periodontal membrane with that of gingiva surrounding the tooth, both of which should be similar. A probe, such as a file, is attached to an electronic instrument with an electric cord and is inserted through the root canal until it contacts the surrounding periodontal ligament. When the probe touches the soft tissues of the periodontal ligament, the electrical resistance of the EAL and the resistance between the file and oral mucosa are equal, which results in the device indicating that the apex has been reached. By measuring the depth of insertion of the probe, one may determine the exact working length of root canal. There is evidence that electronic devices measure mainly the impedance of the probing electrode (contact impedance with the same fluid) rather than tissue impedance itself.

History of electronic apex locators

The electronic method for root length determination was first investigated by Custer (1918).⁷ Suzuki revisited the idea in 1942 that studied the flow of direct current through the teeth of dogs. He registered consistent values in electrical resistance between an instrument in a root canal and an electrode on the oral mucus membrane and speculated that this would measure the canal length.⁸ Sunada took these principles and constructed a simple device that used direct current to measure the canal length. It worked on the principle that the electrical resistance of the mucous membrane and the periodontium registered 6.0 kX in any part of the periodontium regardless of the

persons age or the shape and type of teeth (Sunada 1962). Using direct current caused instability with measurement, and polarization of the file tip altered the measurement.).

Uses of apex locators

1. Can measure the pulp space exactly to the constriction.
2. Provide objective information with high degree of accuracy
3. Used when apical portion of canal is obstructed with impacted teeth, zygomatic arch, overlapping roots, excessive bone density.
4. Used in patients with gag reflex and cannot tolerate x-ray films
5. In pregnant ladies to reduce radiation exposure
6. Useful in children, disabled patients, heavily sedated patients, etc
7. Can be used in teeth with incomplete root formation, requiring apexification & to determine WL in primary teeth.

Advantages

1. Accurate
2. Easy and Fast
3. Reduction of exposure to radiation
4. Perforations can be detected

Limitations of Electronic Apex Locators

Touching a metallic restoration will affect performance of Electronic Apex Locators. Leakage of saliva through cervical caries or open margin will also affect Electronic Apex Locators. Immature “Blunderbuss” apex will give short readings

on Electronic Apex Locators. One issue of concern with EALs was their ability to work in the canal in the presence of various fluids such as blood and irrigants. This was a problem, but most EALs have resolved this issue. Another issue was the size of the root canal, in particular the size of the apical constriction and the use of various file sizes. For best results, the use of a file that closely approximates the width of the constriction is recommended.

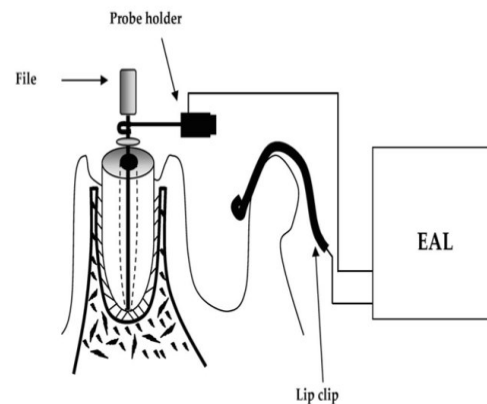


Fig 2: A typical circuit for electrical determination of working length

1. First Generation Electronic Apex Locators (1GEALs)⁹

First-generation EAL devices are also known as resistance apex locators. It measures opposition to the flow of direct current or resistance. When the tip of the file reached the apex in the canal, the resistance value is 6.5 kilo-ohms (current 40 mA). The disadvantage of 1GEAL devices is the pain was often felt due to high electric currents. Unreliable -Often yield inaccurate results in presence of electrolytes, excessive moisture, vital pulp tissue, exudates and blood .Today, most 1GEAL devices are off the market.



Fig 3: The Root Canal Meter (Onuki Medical Co., Tokyo, Japan) was developed in 1969.

2. *Second Generation Electronic Apex Locators (2GEALs)¹⁰*

Second-generation EALs, also known as impedance apex locator, measure opposition to the flow of alternating current or impedance. In 1972, Inoue developed Sono-Explorer (Hayashi Dental Supply, Tokyo, Japan), one of the earliest of the 2GEALs.

Advantages:

Does not require lip clip and no patient sensitivity, detects perforations. The major disadvantage of 2GEALs is that the root canal has to be reasonably free of electro conductive materials to obtain accurate readings. The presence of tissue and electro conductive irrigants in the canal changes the electrical characteristics and leads to inaccurate, usually shorter measurements.

There are several 2GEALs. These include

- a) Root Canal Meter (Onuki Medical Co., Tokyo, Japan).

- b) Endodontic Meter S II (Onuki Medical Co., Tokyo, Japan).
- c) Sono-Explorer Mark II (Hayashi Dental Supply, Tokyo, Japan).



Fig 4: Second generation EAL

3. *Third Generation Electronic Apex Locators (3GEALs)¹¹*

Third-generation EALs are similar to the 2GEALs except that they use multiple frequencies to determine the distance from the end of the canal. These units have more powerful microprocessors and are able to process the mathematical quotient and algorithm calculations required to give accurate readings. It worked on the principle that the electrical resistance of the mucous membrane and the periodontium registered 6.0 kX in any measuring canal.

Advantages:

It is easy to use, uses K file, audible indication, can operate in presence of fluids. It has an analog read out. The disadvantage of this generation sensitivity to canal fluid and the machine needs a fully charged

battery. Requires lip clip, chances of short circuit.

Several generations of EALs have been developed to refine their accuracy Examples- Endex'- Yamaoka et al., Neosomo ultimo, EZ apex locator, Mark V plus, Root ZX, Tri auto ZX, Endy 7000, SofyZX



Fig 5: Third generation EAL

4. The Fourth Generation Apex¹²

The fourth generation apex locator measures the impedance characteristics using more than two frequencies. The unit uses two separate frequencies 400 Hz and 8 kHz similar to the current third generation units.



Fig 6: Fourth generation EAL

The manufacturers claim that the combination of using only one frequency at a time and basing measurements on the root mean square values of the signals increases the measurement accuracy and the reliability of the device. The disadvantage includes needing to perform in relatively dry or in partially dried canals. Eg- SybronEndo elements apex locator

5. The Fifth Generation Apex¹³

The fifth generation apex locator was developed in 2003 which measure the capacitance and resistance of the circuit separately. It is supplied by diagnostic table that includes the statistics of values at different positions on the file is advantage, the disadvantage is that there is considerable difficulty in operating the dry canals. Eg- Propex® II Apex Locator from Maillefer



Fig 7: Fifth generation EAL

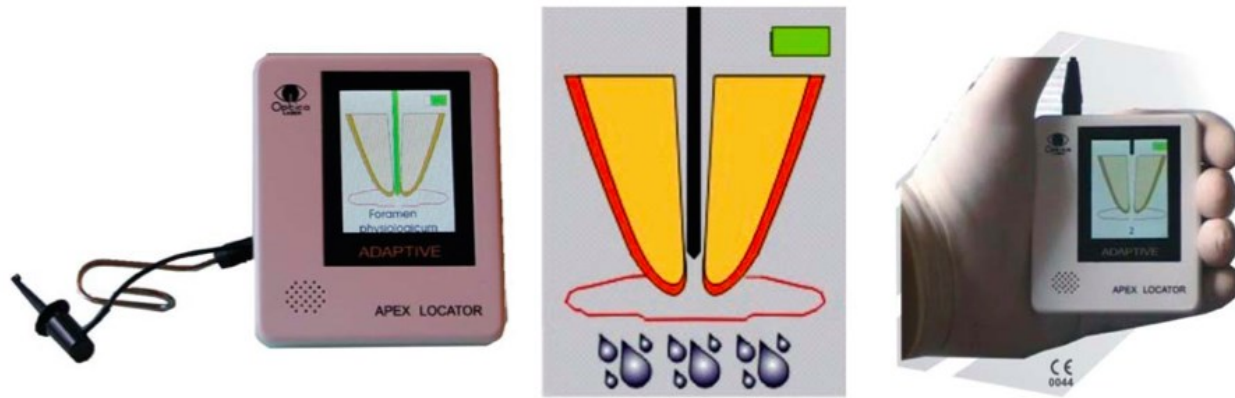


Fig 8: Sixth generation EAL

6. *The Sixth Generation Apex¹⁴*

Sixth generation is called the adaptive type. Adaptive apex locators overcome as the disadvantages of the popular apex locators in case of 4th generation low accuracy on working in wet canals. Adaptive Apex locators continuously defies humidity of the canal and immediately adapts to wet or dry canals, this way it is possible to use it for dry as well as wet canals with blood or exudates.¹³

Other uses for apex locators

Innovative uses for apex locators have been reported. All modern apex locators are able to detect root perforations to clinically acceptable limits and are equally able to distinguish both large and small perforations.¹⁵

Azabal et al. found the JustyII¹⁶ was able to detect simulated horizontal fractures but was unreliable when measuring simulated vertical fractures. Suspected all apex locators can confirm periodontal or

pulpal perforation during pinhole preparation, as a patent perforation will cause the instrument to complete a circuit and indicate the instrument is beyond the 'apex'.¹⁷ Any connection between the root canal and the periodontal membrane such as root fracture, cracks and internal or external resorption will be recognized by the apex locator which serves as an excellent diagnostic tool in these circumstances.

Multiple-function apex locators are becoming more common and several have vitality testing functions. Combination electronic apex locators and electric handpieces are also becoming common and are able to achieve excellent results with the same accuracy as the stand-alone units (Steffen et al. 1999). Kobayashi et al. (1996) reported the development of a combination ultrasonic unit the SOFYZX (J. Morita) which uses the Root ZX to monitor file length. Other handpiece combinations are the DentaportZX (J. Morita) and the Endy 7000 (Ionyx SA, Blanquefort, France).¹⁵

Problems associated with the use of apex locator.

Canal shape: Lack of patency, the accumulation of dentine debris and calcifications can affect accurate working length determination with electronic apex locators.¹⁶

The size of the apical foramen: The size of the apical foramen also has an influence on electronic length determination. Huang (1987) found that when the size of the major foramen was less than 0.2 mm measurements were not affected, even in the presence of conductive irrigants, but as it increased above 0.2 mm measured distances from the foramen increased. Immature or 'blunderbuss' apices tend to give short measurements electronically due to the instruments not touching the apical dentine walls.

Cardiac pacemakers: Electromagnetic interference from dental equipment including electronic apex locators as the potential to interfere with cardiac pacemakers. The manufacturers of electronic apex locators specifically warn against their use with patients with cardiac pacemakers.

Conclusion

No individual technique is truly satisfactory in determining endodontic working length. The CDJ is a practical and anatomic termination point for the preparation and obturation of the root canal and this cannot be determined radiographically. Modern electronic apex

locators can determine this position with accuracies of greater than 90% but still have some limitations. Knowledge of apical anatomy, prudent use of radiographs and the correct use of an electronic apex locator will assist practitioners to achieve predictable results.

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NITROUS OXIDE:OXYGEN INHALATION SEDATION IN PEDIATRIC DENTISTRY

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Abstract

Conscious sedation is a tool available to pediatric dentists in the outpatient setting that can improve patient tolerance and acceptability of unpleasant dental procedures. Relative analgesia has opened new vistas for managing uncooperative children and has rightly become an inseparable part of the dental setups in developed countries. Inhalation sedation as a technique for anxiety and pain control is not as popular with the busy modern day dentist in India. This article gives a brief review of the procedure which has the potential to attain cult status in future among dentists dealing with patients having dental anxiety.

Keywords: Conscious sedation, nitrous oxide,dental anxiety, pediatric dentistry

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Introduction

Delivering dental care to anxious and uncooperative pediatric population poses numerous challenges to a dentist. In challenging situations where behavior management techniques alone do not suffice, pharmacological management is considered as an alternative treatment modality to facilitate provision of dental care. Over the years, sedation has been used by pediatric dentists for successful provision of efficient dental treatment to the difficult children. This can also help to develop a long-term positive psychological response in these children towards dental care.

The American Academy of Pediatric Dentistry defines moderate sedation/analgesia (earlier termed conscious sedation) as “A minimally depressed level of consciousness

that retains the patient’s ability to maintain a patent airway independently and continuously, and respond appropriately to physical stimulation and/or verbal command”¹ Sedation drugs can be administered through various routes such as oral, nasal, intramuscular, intravenous (IV), subcutaneous, and inhalational routes.²

Inhalation is the most commonly advocated route of sedation in pediatric dentistry. It is also the most reliable in terms of onset and recovery.³

Nitrous oxide, commonly known as laughing gas or happy gas, is the only inhalation agent that meets the criteria as being the near ideal sedation agent for the discipline of pediatric dentistry.

Nitrous oxide is a sweet smelling, colorless, non-inflammable, inert gas⁴and is

compressed in cylinders at 750 psi as a liquid that vaporizes on release.³ It has a blood gas coefficient of 0.47 and has rapid onset and recovery time due to low solubility in blood. With doses typically provided by dental sedation units, nitrous oxide can be considered an inert, benign gas that has little influence on vital physiologic functions.

Table 1: Objectives of moderate sedation

Objectives of moderate sedation
➤ Reduce or eliminate anxiety ➤ Reduce untoward movements during dental treatment ➤ Enhance patient co-operation ➤ Raise pain threshold ➤ Reduce gagging ➤ Increase tolerance for long duration procedures

Indications for nitrous oxide sedation

- Children with mild-to-moderate anxiety.
- Patients with mild systemic disorders such as controlled hypertension, angina which may be exacerbated by the stress of dental treatment represent a medical indication.
- Patient with neuromuscular disorders such as spasticity, Parkinsonism & involuntary movement conditions often wishes to cooperate but physically cannot.
- Patient whose gag reflex interferes with dental treatment.

- Co-operative child undergoing lengthy dental procedure.

Contraindications

- Overall, nitrous oxide is a very safe drug with few absolute contraindications.
- In patients with significant respiratory compromise.
- Contraindicated in patients in whom expansion of these air-filled cavities could compromise patient safety (pneumothorax, pulmonary blebs, air embolism, bowel obstruction, and those undergoing surgery of the middle ear).
- Caution must be exercised in individuals with a history of stroke, hypotension, known cardiac conditions, conditions related to vitamin B12 deficiency and pregnancy.⁵
- Severe nasal congestion, those unable to wear a nasal mask.
- Children experiencing severe anxiety or extreme uncooperativeness, may not be good candidates for this milder therapy and may require more potent sedating intravenous or general anesthesia.

Equipment

Only dedicated dental nitrous oxide inhalation sedation delivery systems must be used. The standard delivery system is designed to prevent administration of nitrous oxide gas concentrations in excess of 70%, i.e. there is an assured minimum oxygen concentration of 30%.

There should be a fail-safe device which shuts down nitrous oxide delivery should the oxygen supply fail. The dentist sets the flow depending on the calculated

tidal volume of the patient and then uses a single valve to vary the percentage delivery of nitrous oxide against oxygen.



Fig 1: Equipment

Practical aspects of conscious sedation

Patient selection is done based on the classification system introduced by the American society of anesthesiologists (ASA).⁶

The classification uses six grades as follows:

ASA I	A normal healthy patient
ASA II	A patient with mild systemic disease.
ASA III	A patient with severe systemic disease
ASA IV	A patient with severe systemic disease that is a constant threat to life.
ASA V	A moribund patient who is not expected to survive without the operation.
ASA VI	A declared brain-dead patient whose organs are being removed for donor purposes.
E	Emergency operation of any variety (used to modify one of the above classifications, i.e., ASA III-E).

Patients who are in ASA classes I and II are frequently considered appropriate candidates for minimal, moderate, or deep sedation. Informed consent for sedation and/or analgesia and for the procedure should be obtained from the patient, or a person entitled to give consent on behalf of the patient, according to applicable legislation. The minimum duration of fasting prior to appointment.⁷

- 8 hours after a meal that includes meat, fried or fatty foods;
- 6 hours after a light meal (such as toast and a clear fluid), or after ingestion of infant formula or nonhuman milk;
- 4 hours after ingestion of breast milk; and
- 2 hours after clear fluids (such as water, fruit juices without pulp, carbonated beverages, clear tea, and black coffee).

The child must be wearing loose and comfortable clothing. Any change in health a week prior to operation, medical conditions and drugs used must be noted as postponement of procedure may be necessary. The pediatric patient should be accompanied to and from the treatment facility by a responsible adult.

A practitioner who uses sedation must have ready access to facilities, personnel, and equipment to manage emergency and rescue situations. Accurate entries and documentation must be made before sedation, at the time of sedation, during treatment and after treatment.⁸

Technique

Nasal hood of appropriate size is chosen to ensure adequate seal. After stabilization of the nose-piece and delivery of 100% oxygen for initial 3 minutes, Nitrous oxide level should be increased to 30-50 % gradually while monitoring the vital signs. Throughout the procedure, the dentist should talk gently to the child to promote relaxation, reinforce cooperative behavior and ensure responsiveness.

An indication of CNS effect would be minimal or slow movements of extremities in response to verbal commands. Upon termination of procedure, the child is allowed to inhale 100% oxygen for 3 to 5 minutes. This allows rapid diffusion of nitrous oxide from the venous blood into the alveolus while maintaining oxygenation, enabling the patient to return without incident to pretreatment activities.

Once dental treatment is complete, the patient enters the recovery phase. The vital signs are monitored and patient is fit for discharge only if he/she has achieved age and mental status appropriate normal level of responsiveness and orientation.⁹

Conclusion

Safe and adequate administration of sedative and analgesic medications can make painful and anxiety provoking situations tolerable. Moderate sedation/conscious sedation is a tool available to pediatric dentists in the outpatient setting that can improve patient tolerance and acceptability of unpleasant

procedures. Despite the numerous studies conducted worldwide, the search for the most efficient and safest sedative agent is yet in its 'ongoing phase'. N2O/O2 sedation is an excellent choice for managing the mildly fearful dental patient and should be integrated into every office's repertoire for delivery of care.

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PROSTHETIC REHABILITATION OF A PATIENT WITH NEUROFIBROMATOSIS: A CASE REPORT

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Abstract

The neurofibromatoses are a group of genetically distinct disorders that cause tumors in the nervous system characterized by the proliferation of Schwann's cells, perineural cells, and endoneurial fibroblasts. Neurofibromatosis 1 (NF1) occurs with a frequency of 1 in 3000 people. The national institutes of Health Consensus Development Conference formulated the diagnostic criteria for NF1, underlining the involvement of the skin, bone, and the nervous system in the condition. Although the systemic features of this disorder have been widely documented, the literature on the oral manifestations is vague. This case report discusses the prosthetic rehabilitation of a neurofibromatosis patient with complete dentures.

Keywords: Neurofibromatosis, Neurofibroma, Complete denture prosthesis

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Introduction

The neurofibromatoses are a group of genetically distinct disorders that cause tumors in the nervous system characterized by the proliferation of Schwann's cells, perineural cells, and endoneurial fibroblasts.¹ The heterogeneous nature of Neurofibromatosis (NF) has become well established in recent years. Riccardi recognizes at least seven types of NF, but of many proposed classifications of the disease, two distinct forms are generally accepted namely, a peripheral form known as NF1, and a central form known as NF2.^{2,3}

Neurofibromatosis type 1 (NF1) represents the classic form described by Recklinghausen in 1882.³ It is also known as Von Recklinghausen's disease, Peripheral

Neurofibromatosis, Von Recklinghausen's Neurofibromatosis, Recklinghausen's Phakomatosis, and Multiple Neurofibroma. Neurofibromatosis 1 (NF1) occurs with a frequency of 1 in 3000 people. Although many affected persons inherit the disorder, between 30 and 50 percent of new cases arise spontaneously through mutation (change) in an individual's genes. Once this change has taken place, the mutant gene can be passed on to succeeding generations.^{4,5} Peak age of presentation is said to be third decade of life; however, its occurrence between 10 months and 70 years of age has also been reported.⁶

The National Institutes of Health Consensus Development Conference formulated the diagnostic criteria for NF1,

underlining the involvement of the skin, bone, and the nervous system in the condition. They include 6 or more café-au-lait spots (≥ 0.5 cm prepuberty or ≥ 1.5 cm postpuberty), axillary or inguinal freckling, 2 or more neurofibromas or 1 plexiform neurofibroma, iris Lisch nodules, optic pathway tumor (optic glioma), sphenoidal dysplasia, and first degree relative (parent, sibling or offspring).⁷ A given case is diagnosed as NF1, when two of these clinical features are present. Additional features like precocious puberty, and delayed sexual development may be present. About 50% have specific learning disabilities in reading, spelling or mathematics, retarded growth, macrocephaly, scoliosis, hypertension and epilepsy.⁸

Genetic testing is not needed to diagnose the condition afterbirth because most people with NF1 will have enough signs of the condition by age of 5 years for a specialist to diagnose them with confidence. Genetic testing for NF1 is not widely available and is currently expensive, but it can be helpful in some situations, such as where prenatal diagnosis is requested. Prenatal genetic testing can be done where one of the parents is affected and wants to know if the fetus is affected, provided that the specific NF1 mutation in the affected parent has been identified.

Neurofibromas can cause cosmetic problems and wrap around or penetrate the nerves causing pain. There is about a 5% increase in the risk for various cancers, including brain tumour. Hypertension is more common in NF1 patients than in the

general population. There is also an increased rate of scoliosis in NF1 which commonly presents and progresses around the time of puberty.^{7,8}

Case Report

The patient, a 65 year old unmarried lady, came to the department out-patient section for prosthodontic rehabilitation with complete dentures. Patient claims no history of similar condition in the family. She is hypertensive and is on medication since 4 years. Patient also gives a history of spinal cord lesion and quadriplegia. On general examination, all vital signs were within the normal limits. The patient seemed to be well-oriented with time, place, and person.

Extra oral examination revealed multiple nodular mass all over the body including face, hands, feet, and trunk, suggestive of neurofibromas (Fig 1). She gives a history of the appearance of the extra oral nodules, starting at the age of 10 years. No associated pain or redness observed.

Intraoral examination showed completely edentulous maxillary and mandibular arches (Fig 2). Patient had undergone extraction due to caries and periodontal involvement two months back. Soft tissue examination revealed a 0.6 x 0.6 cm nodule on the dorsum of the tongue. Multiple soft nodular masses were found on both the arches.

Radiological examination with the help of an orthopantomograph revealed enlargement of the mandibular canal (Fig 3).



Fig 1: Neurofibromas on the face, back, and arm of the patient

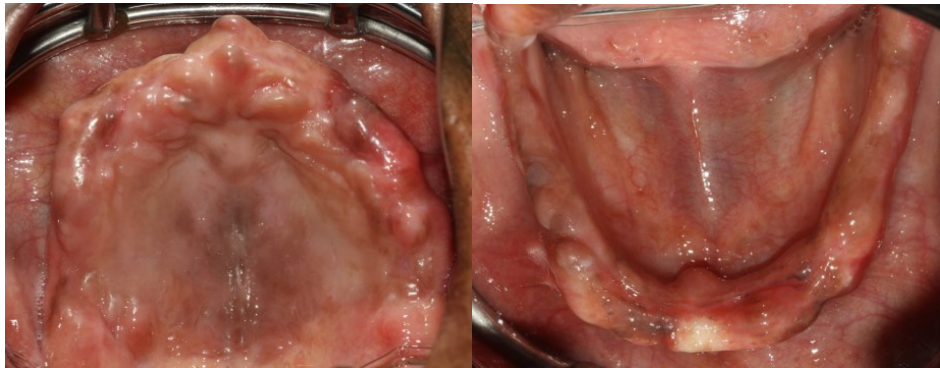


Fig 2: Neurofibromas on the edentulous ridges of the patient

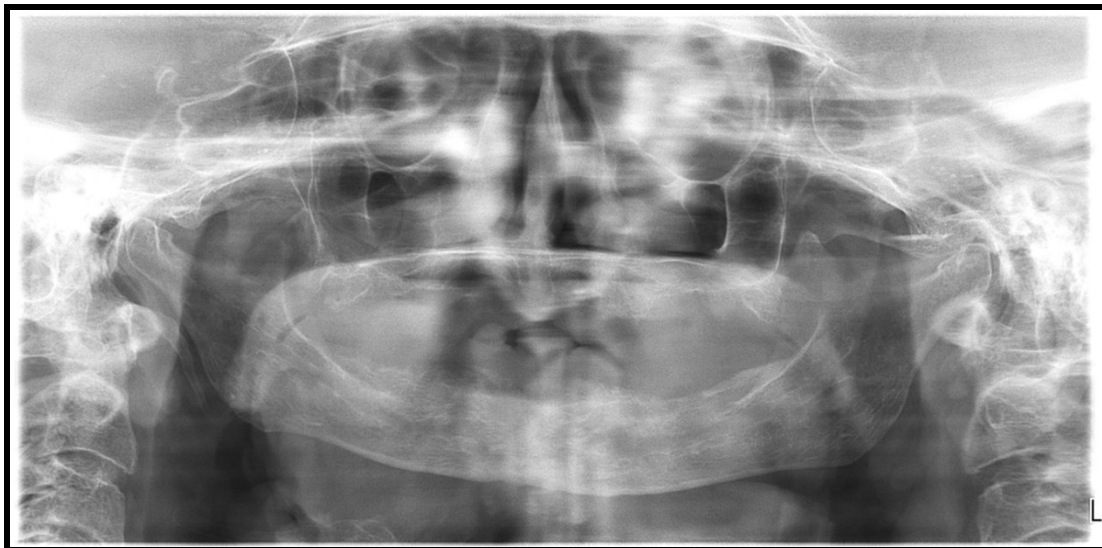


Fig 3: Orthopantomograph showing widening of mandibular canal

Treatment

The patient was given explanation regarding the advantages and limitations of the prosthesis to avoid unrealistic

expectations. She was informed about the need for preprosthetic surgery to remove the nodular masses on the edentulous arches to improve the denture foundation. The

excision of the nodular masses was done under local anesthesia and sent for biopsy to confirm neurofibromatosis. A waiting period of 45 days was given for proper healing and to check for recurrence of the nodules.

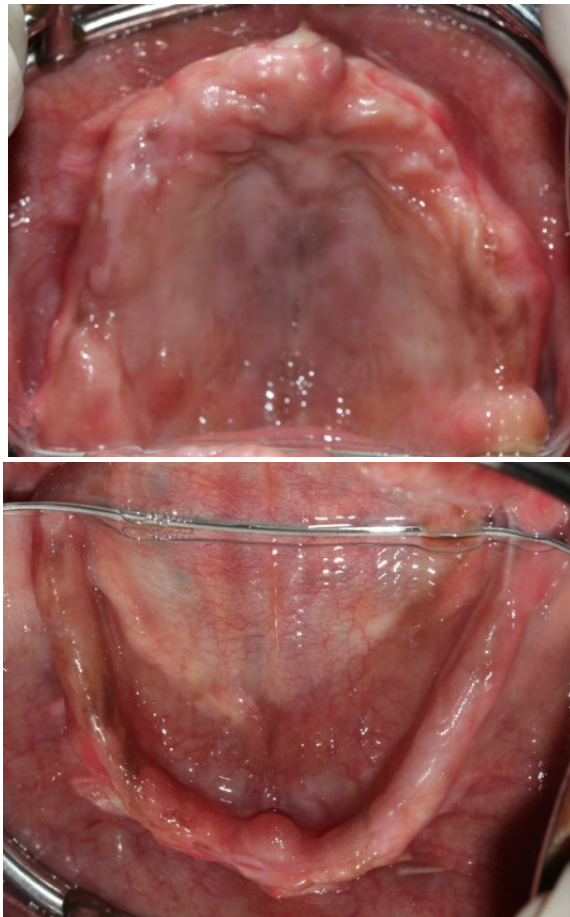


Fig 4: Edentulous ridges after preprosthetic surgery

Tray selection and primary impression: After preliminary examination of the patient's oral cavity, stock trays were selected for maxillary and mandibular arches. Primary impressions were made using alginate as impression material. Primary casts were made and the areas with compressible tissues were relieved with wax. Special trays with complete spacer

design were fabricated with autopolymerising resin.

Secondary impression: The special trays were checked in the patient's mouth and the overextensions if any were trimmed and smoothened. Border moulding was done using low fusing green stick compound and the tissues were displaced within physiological limits in order to allow maximum extension of denture bearing area. Wax spacers were removed and the secondary impressions were made with low viscosity alginate impression material due to the presence of bilateral undercuts in maxillary tuberosity region.

The master cast obtained from the secondary impression was used to fabricate a temporary denture base of autopolymerising resin, on which an occlusal rim was made out of modelling wax.

Jaw relation: The occlusal rims were adjusted to provide proper facial fullness, anterior and posterior plane, and visibility. The vertical dimension and freeway space were determined using Niswonger's technique, phonetics and esthetics. The facial midline, canine lines and the smile line were recorded on the occlusal rim. Finally, the patient was guided into centric relation and the rims were sealed in this position. Teeth selection was done according to various criteria such as shape of the face, gender and complexion of the patient. The master casts along with the occlusal rims were mounted on a mean value articulator and the teeth arrangement was done considering the weaker mandibular arch.

Try in: Once the teeth arrangement was completed, wax up and carving was done and try in was carried out in the patient's mouth. The facial fullness, midline, and occlusal plane were inspected and corrections done accordingly. The phonetics and esthetics were also assessed and the required corrections were made. Then the patient's satisfaction and approval were obtained prior to processing of the dentures. After processing the complete denture prosthesis were retrieved and trimming, finishing, and polishing were carried out.

Complete denture insertion: The prostheses were inserted into the patient's mouth and retention, stability, support, function and esthetics were evaluated. Necessary corrections and adjustments were made. Occlusal adjustments and selective grinding were also done (Fig 5 & 6).



Fig 5: complete denture in mouth

Post insertion instruction and recall: The post insertion instructions about the care of denture and denture bearing tissues were explained to the patient. The patient was recalled after 48 hours for the initial follow up and then after 7 days for evaluation and then subsequently for long term follow up.



Fig 6: Patient with the complete denture prosthesis

Discussion

NF1 is a progressive disorder, which means most symptoms will worsen with time. It isn't possible to predict the course of an individual's disorder. Most people with NF1 have a normal life expectancy. Neurofibromas on the skin can cause cosmetic and psychological issues. The presence of compressible tissues over the denture bearing areas slightly compromises the retention and support of the complete denture. However the patient is content with the complete denture. Periodic and annual recall of patients is necessary as neurofibromas have high recurrence chances.

Conclusion

Neurofibromatosis changes the general appearance of the patient and due to the fibromatous growth within the oral cavity can lead to compromised retention and stability of the removable prostheses. However within the boundaries of the

treatment options available and with interdisciplinary help we can provide the best possible option to improve the appearance and function of the patient.

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A NEWER APPROACH IN EXCISION OF FIBROMA: A CASE REPORT

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Abstract

Fibromas are the most common benign oral soft-tissue neoplasm of mesenchymal origin that are composed of fibrous connective tissue. The current paper presents a case where an intraoral fibroma was successfully managed in child using a new approach of pre surgical suture placement. Conservative surgical excision was performed. Considering the size and location of the lesion, excisional biopsy was performed and sent for histopathological analysis which confirmed the lesion as fibroma. A follow-up at 4 months revealed uneventful healing of the site without reoccurrence of the lesion.

Keywords: fibroma, fibroepithelial polyp, surgical removal, newer technique

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Introduction

Fibromas are the most common benign oral soft-tissue neoplasm of mesenchymal origin that are composed of fibrous connective tissue.¹ They can occur in any body organ and are known as irritation fibroma, traumatic fibroma, focal fibrous hyperplasia, fibrous nodule, or fibroepithelial polyp.² Fibromas commonly occur due to trauma or local irritation. Found in 1.2% of adults, it is the most common oral mucosal mass submitted for biopsy and is composed of Types I and III collagen.²⁻⁴

Case report

A 11 year old girl had reported to the Department of Paediatric and Preventive dentistry, KMCT Dental College, Calicut, Kerala with a chief complaint of a soft tissue

growth which was positioned in the right buccal mucosa with no associated pain since 4 months (Fig 1). The patient gave a history of the swelling being small at first with a slow enlargement. No relevant medical history was revealed.



Fig 1: Soft tissue growth on the buccal mucosa

Extra orally, there was no deformity detected and the regional lymph nodes were not palpable. Intra orally, there was presence

of a solitary, sessile, pale pink coloured lesion on right side of buccal mucosa 1 cm away from corner of mouth. Lesion appeared as an elevated nodule with a smooth surface measuring 9 mm x 7 mm in diameter.

The lesion was non scrapable, and non-tender and clinical diagnosis of irritation fibroma was made. After topical anesthetic agent application, a suture was placed around the lesion in an interrupted manner which will serve to control the bleeding after excision (Fig 2).



Fig 2: Suture being placed around lesion before excision

The advantages of placing a suture around the lesion are better control of bleeding and easy placement of suture over the excisional site. The soft tissue growth was grasped with tissue forceps and retracted with minimum tension. The lesion was excised with BP blade number 12. The excised tissue was immersed in a 10% formalin solution and sent for histopathological examination. The suture was then tightened with surgical knot 4 months post surgical review was done. The area healed uneventfully without any scarring (Fig 3a-e).



Fig 3a: Lesion is held with tissue forceps



Fig 3b: Lesion being excised



Fig 3c: Interrupted suture placed



Fig 3d: Excised lesion



Fig 3 e: 4 month post-surgical view

Histopathological examination

The H and E stained soft tissue section shows stratified squamous parakeratinised epithelium with an underlying moderately collagenous connective tissue. Overlying epithelium is hyperplastic and shows atrophy towards one end. The connective tissue shows plump spindle shaped fibroblast arranged parallel to collagen bundles. Endothelial lined vascular channels with extravasated RBC's can be appreciated. Deeper in the connective tissue muscle bundles and nerve fibers are evident (Fig 4).



Fig 4: Histologic section

Discussion

Fibrous growths of the oral soft tissues are fairly common and include a diverse group of reactive and neoplastic conditions. Tissue enlargement of the oral cavity often presents a diagnostic challenge because adverse group of pathologic processes can produce such lesions.³

Fibroma is the most common benign soft tissue tumor in the oral cavity. Most fibromas represent reactive focal fibrous hyperplasia due to trauma or local irritation. A fibroma occurs as a result of a chronic repair process that includes granulation tissue and scar formation resulting in a submucosal fibrous mass.⁴ Hyperplasia is a self-limiting process unlike neoplasia and hyperplastic cells sometimes show regression after removal of the stimulus.⁵

The general literatures have cited the reason for a few of the oral lesions like irritation fibroma and mucocoele, due to oral habits such as lip biting/sucking in childhood.⁶ Rare association of reactive

hyperplasia or traumatic fibroma with a natal tooth in a 4 year and 6 month old infant has been reported.⁷

The signs and symptoms of Irritation Fibroma include: It usually presents as a single nodule in the mouth (tongue, cheek, and lips). Rarely, the tumor can occur as multiple nodules too. The nodule is typically less than 2 cm in size (along the larger dimension), and normally painless and slow-growing. The skin nodules are not painful. The skin over the nodule may be brownish-red to white, but is usually intact. The masses are usually firm to touch and well-defined. Most tumors are asymptomatic; however, in others, a wide-range of behavior is observed.⁸⁻¹⁰

Irritation Fibroma may cause bleeding from the mouth, ulceration of tongue, cheek, or lips. Irritation Fibromas may occur as new growths that develop over weeks to months. A fibro-epithelial polyp is diagnosed based on the location of soft tissue swelling. If swelling is located on the tongue, the possibility of a neurofibroma, neurilemmoma, or granular cell tumor must be considered.¹¹ Swelling on the lower lip or buccal mucosa may be a mucocele, lipoma, or salivary gland tumor.¹¹

Another important distinguishing feature is that a traumatic fibroma exhibits two different patterns of collagen arrangement, a radiating pattern and a circular pattern, depending on the amount of irritation and the site of the lesion. An irritation fibroma with a radiating pattern is associated with sites that are immobile in nature (e.g. the hard palate) and with more severe trauma while an irritation fibroma

with a circular pattern is associated with sites that are flexible in nature (e.g. the cheeks) and with less severe trauma, but a true fibroma exhibits neither of those patterns¹¹.

A thorough physical examination and a complete medical history are very crucial. Tissue biopsy of the tumor: Examination of the biopsy under a microscope by a pathologist is considered to be gold-standard in arriving at a conclusive diagnosis.^{8,9,10} In addition to surgery, other treatment modalities are electrocautery, an Nd:YAG laser, a flash lamp-pumped pulsed dye laser, cryosurgery, intra lesional injection of ethanol or corticosteroids, and sodium tetra decyl sulfate sclerotherapy.¹²

Excision under local anesthesia using B.P blade is the simplest method that can be used in any clinic. The profuse bleeding after excision makes suturing difficult and time consuming. The method discussed here solves the problem of controlling the bleeding during suturing procedure. An effective use of this method in excisional biopsy is presented here.

Conclusion

Fibromas are generally rarely occurring lesions in oral cavity of children. If occurring they are mainly as a result of habits and trauma leading to irritation fibromas. Pedodontist should be familiar with the different types of fibrous hyperplasias they may encounter during patient treatment and should prevent the lesion by surgical excision in toto along with elimination of the source of irritation that led to the lesion.

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