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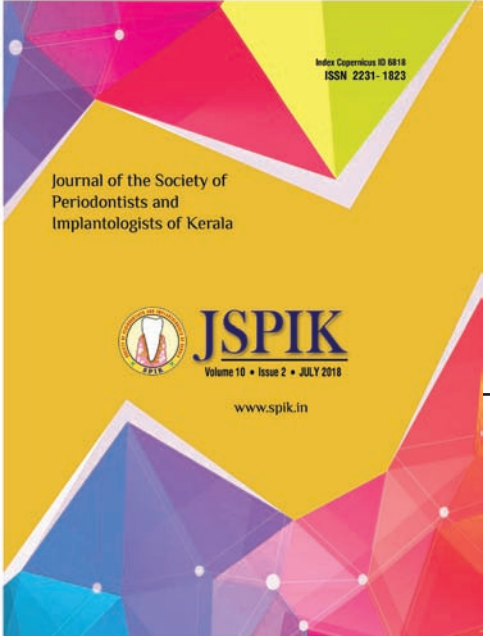

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President's message

Greetings dear SPIK members……

I consider it an honour to be the president of this society and thank each and everyone of you for bestowing this honour on me.

We had the SPIK annual family meet at hotel lake palace, Trivandrum on the 5th and 6th of May 2018.It was a well organised event but it was disheartening to see the poor turnout of our SPIK members. Our association should be a platform that serves to enhance our knowledge and skills and also to interact and improve our friendship.

I am confident that with the active involvement of our members, we can scale greater heights in pursuit of dental excellence. For this,, both academic and professional advancement of our members is a must. Hence more and more academic oriented training programmes should be conducted by our respected association

I take this opportunity to thank our energetic editor, Dr.Plato for the excellent job he has done for the successful outcome of our journal.

For every malayalee, rains are an integral part of their life and they enjoy every aspect of it. But last month, the unprecedented rains ended up in one of the biggest natural disaster of our times. All through this tragedy, we saw the unity and the positive spirit of our people to overcome the crisis which was the biggest lesson we learned. Now it is time for rehabilitation and I would also urge all SPIK members to become a part of it and also to conduct oral health programs in the affected areas.

This year should become a model for the power of team work and reflect the strength of our association .I take this opportunity to urge all members to work together with unity and a positive spirit during the coming year so that we can scale great heights.

Thanking you,

Dr Seema Thampi
President, SPIK

Editorial

SPIK with a New Team ….

Welcome to the Aug edition of JSPIK!

As I take over as editor of JSPIK, my main goal was to regularize the issues. I am glad to have the support of All – Authors, Reviewers and Office bearers of SPIK 2017 Team by Dr Anto Joseph. We at SPIK are planning 2 future editions viz December and April during my term. JSPIK in its past three issues tried its best to include all the good clinical cases of 2017. My job as an editor is to make our journal as useful and informative to you as possible. Also I humbly request all of you to send interesting articles to JSPIK, so that we can widen our knowledge and manage clinical problems more effectively. If you have an article to be published I would be delighted to hear from you.

Dr Plato Palathingal
Editor
editorspik@gmail.com

Secretary's Message

Hand scaling….then and Now

"Dentistry is an art and science.. " we have been taught right from the initial days in dental colleges. Most successful dentists are gifted artists with great skills who are capable of sculpting beautiful charming smiles. In the 1700s, Pierre Fouchard pioneered the modernization of dentistry with the advent of improved and refined instruments and procedures. Ever since, the profession had grown by leaps and bounds, it embraced the technological advances in biomedical engineering and material science. It always followed the medical profession and adapted the advancement in diagnostic techniques including newer imaging and surgical procedures. Over the years it has become less traumatic, less invasive, less fearsome and more patient friendly. The fear of the dental drill is slowly becoming a thing of the past and the eugenol smelly dental clinics have given way to swanky dental spas with plush interiors where people go to unwind and become good looking.

The dental drill has become less noisy, less jerky and smooth. Injections are being administered after topical anesthesia and the needles have become thin and less painful. Surgical procedures have become minimally invasive and less traumatic. Dentists now have lasers that cut flesh like butter and teeth like cakes. We have piezo surgery that cuts bone with little fuss. And for scaling teeth we have ultrasonic or piezo scalers that are so smooth and imparts only some tingling sensation in the worst situation. The above mentioned advancements are now available in most countries and affordable to many.

But why do we still rely on hand scalers to treat the majority of patients that attend the Periodontics department at Dental colleges in India. I strongly believe that the undergraduate Periodontics clinics of the 500 odd dental colleges of our country probably are the only places where the thousands of BDS students still perform all their periodontal treatments to their patients right from preschool children to the very old using the CRUDE NON standardized hand held scalers.

Why is it still like this?

Why are we not offering the power driven scalers to our poor undergraduate students?

Why are we insisting that the students should use these hoes and chisels which perhaps they might never use it in their life time?

Why are we denying them the chance to touch and feel the comfort of the ultrasonic scalers about which they have to learn and perhaps, write an essay to get a pass in their examination?

I do understand that the students need to learn the grip and the grasp and the tactile feeling by using hand held scalers in order to practice better dentistry. But for that should they continue using it to treat all their patients they get to see in third year and final year perio postings and most of the time, without anaesthesia. Times have changed. I wonder how patients tolerate this ordeal of undergoing hand scaling by an inexperienced dental student without even topical anaesthesia. It is a difficult feeling as a clinical teacher to look into the eyes of those poor fellows who most of the time end up with bruised bleeding gingiva after having undergone hand scaling in a UG clinic. In my experience as a clinical teacher I have hardly found standard qood quality refined supragingival hand instruments with undergraduate students. Those instruments they usually possess are heavy, blunt and traumatic. In this era of painless dentistry with lasers, veneers and clear aligners done under magnification we need to think beyond poorly designed hand scalers to teach periodontal treatment to the BDS student. We need to use them with prudence. If limiting its use improves patients and students (treatment providers) acceptability and comfort, then why hesitate?.

But perio still thrills

Dr. Baiju R.M.
Secretary, SPIK

Oral Hygiene Awareness among Hemodialysis Patients–An Epidemiological Cross-Sectional Survey

Sajna H.R[1], Amitha Ramesh[2], Biju Thomas[3]

ABSTRACT

Background: The number of patients undergoing hemodialysis due to chronic renal disease is increasing year by year. These patients are predisposed to present a lot of dental problems. They tend to neglect their oral hygiene and dental care which can further aggravate their systemic illness. Therefore a survey was conducted to evaluate the level of knowledge and awareness about oral hygiene among these patients.

Aim: To assess the extent of oral hygiene awareness among patients undergoing hemodialysis.

Materials and methods: A hospital-based questionnaire survey was conducted among 151 patients undergoing hemodialysis in K S Hegde Medical Academy. Eligible participants were selected. A questionnaire-based survey was used to assess their oral hygiene practice, the frequency of visiting dentist and knowledge about the relationship between oral health and general health.

Results: Out of 151 patients, only 36% brush their teeth twice daily of which 59% use toothbrush and toothpaste as cleansing aid and 10%use charcoal. Most of them used the medium toothbrush with a horizontal brushing technique. Majority of them experienced bleeding gums and 52% experienced bad breath. Our data showed that nearly half of the patients never visited a dentist at any point in time. Only 17% of the patients were aware of the fact that oral health may affect kidney disorder.

Conclusions: The results of this survey showed that only a few patients undergoing hemodialysis were aware of the importance of maintaining good oral hygiene. Efforts should be made to educate the patients regarding the complications that may affect the general health due to the negligence of oral health.

Keywords: oral hygiene awareness, kidney disorders, hemodialysis

Introduction

The oral cavity is considered as the mirror of systemic health. Majority of the systemic diseases presents hard tissue and soft tissue manifestations in the oral cavity. Oral cavity can act as the site of origin for pathogenic organisms which can spread to distant parts of the body, especially in immunocompromised hosts. Oral infection like periodontitis can act as a potential contributing factor to a variety of clinically important systemic diseases.[1]

Chronic renal failure (CRF) is one such disease which presents with a spectrum of oral manifestations, including xerostomia, uremic stomatitis, and various mucosal and periodontal lesions. The renal system disorders are considered as a major cause of morbidity and mortality worldwide.

The major function of kidney in our body includes (1)excretion of the end products of metabolism (2) regulation of erythrocyte production in the bone marrow (3) regulation of electrolyte concentration and blood volume (4) participation in calcium homeostasis through hydroxylation of vitamin D3 into active or

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inactive metabolites.[2]

Any pathologic process that results in decreased renal function would, therefore, be expected to have the serious effect on the body.[3]

Chronic Kidney Disorder (CKD) is considered as a clinical situation in which internal renal functions stops and the patients require alternative treatments like renal transplantation or hemodialysis up to his/her death. CKD is the 12th leading cause of death and 17th cause of disability[4]. The method of removing the metabolic waste products mechanically from the blood is called as dialysis. There are two types of dialysis: Extracorporeal or hemodialysis and Intracorporeal or peritoneal dialysis.[5]

Among all the oral signs and symptoms related to the chronic kidney disorder, chronic periodontitis plays an important role. Periodontal pathogens may invade, and proliferate in coronary endothelial cells leading to atheroma formation and impaired vasculature relaxation[6]. Periodontal disease may exert similar effects within the vasculature of the kidney since both cardiovascular and renal disease share many risk factors in common. Therefore chronic periodontitis increases the overall systemic inflammatory burden in the body. It can inversely affect the systemic condition of an immunocompromised host like hemodialysis patients and can lead to the difficulties in the management of such patients.[7]

Table 1

Variables	Number	Percentage
Type of brush		
Neem stick	-	-
Charcol	15	10
Toothbrush/paste	88	59
Finger and tooth powder	47	31
Method of brushing		
Horizontal	71	48
Circular	11	7
Vertical	35	23
Combined	33	22.0
Frequency of brushing		
Occasionally	-	-
Once daily	94	63.0
Twice daily	54	36.0
More than twice	2	1.0
Type of the brush		
Hard	37	28
Medium	81	62
Soft	13	10

Since a large number of chronic renal disease patients presents pathologies associated with oral cavity and the interrelationship that both conditions can have on each other,it is important to make awareness in these patients to maintain good oral hygiene.[8]

Promoting good oral hygiene reduces the overall inflammatory load and oral infections that may predispose a patient to septicemia, endocarditis and possible endarteritis of the vascular access or line for hemodialysis or of catheters for peritoneal dialysis.[9] Therefore, preventive dental care should be considered very important for this population.

The aim of the present cross-sectional survey was to assess the extent of oral hygiene awareness among chronic kidney disorder patients undergoing hemodialysis treatment.

Materials & Methods

This self-prepared, questionnaire-based survey was conducted for a period of 3 months. Patients reporting to the hemodialysis unit K S Hegede medical academy were included in the survey. A verbal consent was taken from all the individuals who were willing to participate.

Data Source

A total of 151 participants were included in the survey. They were selected by stratified random sampling technique.

Inclusion and Exclusion Criteria

Subjects with chronic kidney disorder attending the hemodialysis unit K S Hegede Medical Academy on a regular follow up were included. Patients who were not interested to participate and,patients under the age of 16 years were excluded.

Questionnaire:

The data was collected in the form of 19 questionnaires which were similar to the one used by previous researchers with few additional questions. Patients were asked about their oral hygiene

practices such as the method of brushing, frequency, change of toothbrush, use of interdental aids and mouthwashes. Patients were also asked questions about the frequency of visiting dentist, awareness about the interrelationship between oral health and systemic health. A brief medical history including hypertension, diabetes and prosthetic devices (heart valves, grafts) were recorded.

Data Analysis:

The data were analyzed in Microsoft Excel using average and percentage.

Results

A total of 151 participants were included in the survey. Out of which, males were slightly predominant than female. Almost 60% of participants were in the age group of 31 years to 60 years. About 23.3% were diabetic whereas 64% had a history of hypertension. 57% were undergoing hemodialysis for 5 years. 46% of them had an income less than 1 lakh per annum. 15.1% were unemployed and only 30% were having high school education.

Table 1 shows the oral hygiene practice among the studied group. It clearly indicates that only 59% of them use toothbrush and paste. The survey showed that majority used the vertical method of brushing and brushed once daily. It also shows that only 10% of them use soft bristle brush when compared with medium or hard. When asked about the use of any interdental aids 81.7% said they do not use interdental aids and about 14% said they use floss at times.

About 30% of the patients experienced bad breath and mobility of teeth. 22% had some form of swellings in the mouth. Most of the patients experienced bleeding from the gums. (fig 1)

29% of the patients think that there is no relationship between systemic diseases and oral health. Only 17% were aware of the fact that there exists a relationship between oral health and kidney disorder. (fig 4)

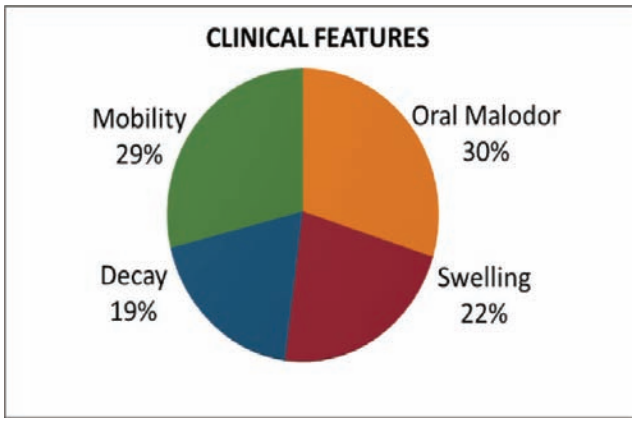

Fig. 1

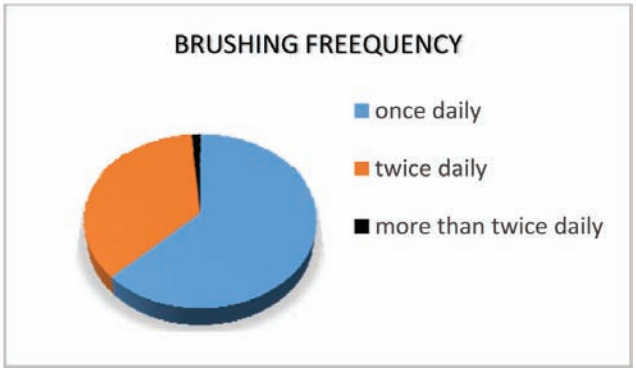

Fig. 2

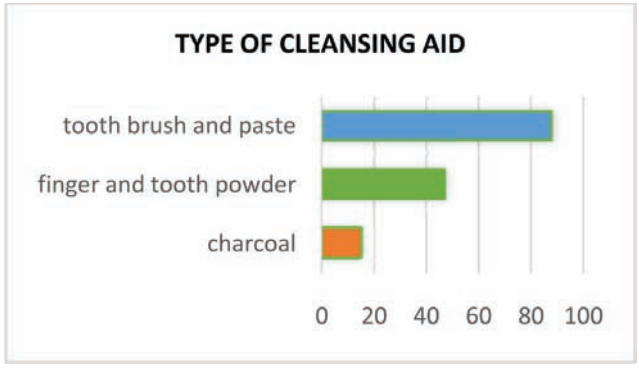

Fig. 3

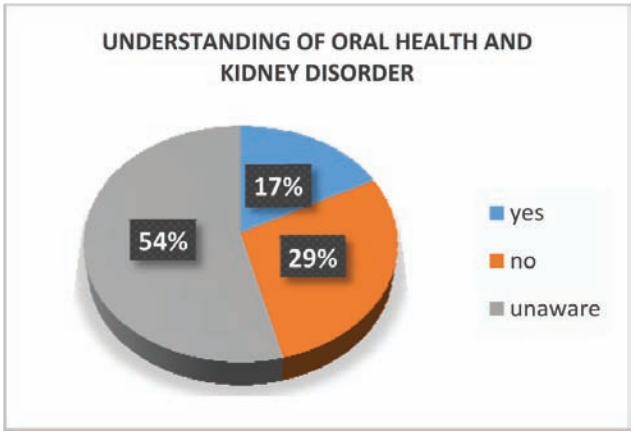

Fig. 4

Discussion

Numerous studies have suggested that when compared to the general population, oral hygiene of chronic kidney disorder patients is very poor. Naugle et al[10] conducted a study regarding the oral health status of hemodialysis patients and the results concluded that almost all of them had some form of periodontal disease. Moreover, 64% had severe gingivitis and a higher-than-normal score for the decayed, missing and filled index.

The data from our survey revealed that the patients on hemodialysis are more likely to ignore their oral health. Dialysis economically burdens and stresses the patients as well as their families. Oral hygiene practices like brushing and flossing were irregular amongst these patients. This may be due to the stress caused by the long-term hospitalization,poor socioeconomic status or due to the lack of awareness. This lack of care may put these patients at higher risk of oral infections which can predispose them to a variety of tooth related problems.

The results disclosed that these patients seek dental treatment only in emergency situations. The possible reason may be due to the poor socioeconomic status and psychological stress caused by the course of hemodialysis treatment.

Chronic renal disorder patients exhibit decreased bone mineral density that would increase the risk for alveolar bone loss resulting in mobility of tooth and eventually tooth loss. Oral infections, especially periodontitis may affect the host's susceptibility to systemic disease in three ways: by shared risk factors; subgingival biofilms acting as reservoirs of gram-negative bacteria, and the periodontium acting as a reservoir of inflammatory mediators[1].

Dentists should emphasize the importance of maintaining oral hygiene for patients undergoing hemodialysis and educate them about the inter-relationship between oral health and systemic health. These patients should be motivated to seek sufficient dental care on a regular basis.

In interpreting the findings of the present survey, it's important to outline the possible limitations. Majority of the patients recruited for the study were from a very low socioeconomic status, which might have influenced their oral hygiene habits. Secondly, because of the self-reported aspect of the data and patients interviewed, it is difficult to determine whether the response is affected by the vulnerability.

Conclusion

The results of this survey among the selected sample of patients attended the hemodialysis unit, K S Hegede Medical Academy (Mangalore), showed that only a few patients were concerned about maintaining the good oral hygiene. Dentist plays a pivotal role in educating patients about the dental care needed. It is also necessary to make the patients aware that there is a bidirectional relationship between systemic health and oral health. Hence awareness about the oral hygiene practices among chronic renal disorder may decrease the morbidity and mortality of the disease. As this survey was conducted in a limited group of people, further studies are needed to be conducted amongst a larger population.

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Management of isolated gingival recession using modified lateral pedicle graft: a case report

Dhivya R.[1], Ramakrishnan T.[2], Indumathi P.[1], Vidya Sekhar[3], Ebenezer M.[3], Ramnath E.[4]

ABSTRACT

Gingival recession is a common mucogingival deformity and should be treated at its earliest detection. Exposed root surfaces are more likely to develop root sensitivity, root caries and cause esthetic problems. Several techniques have been proposed to cover the denuded root looking for satisfactory outcomes both esthetically and functionally. Among various surgical procedures, laterally positioned pedicle graft is widely used successfully to cover Miller's class-I and Class-II recession defects. This article highlights on a case report in which a modified laterally positioned flap had been used for root coverage in isolated Miller's class-I recession defect in relation to mandibular anterior region. Significant root coverage was obtained with no change in the position of gingiva at the donor site.

Keywords: Gingival recession, Modified lateral pedicle graft, Periodontal esthetics, Root coverage.

Introduction:

Location of the gingival margin apical to cemento-enamel junction is called Gingival recession.[1] Periodontology has been concerned with the commonly occurring problem of isolated gingival recession and root exposure for a long period.[2] The etiology of gingival recession is multi-factorial. The most significant factors causing gingival recession are plaque-induced gingival inflammation; with some other precipitating factors such as a prominent root surface,thin gingiva,tooth malposition, frenal pull, mechanical trauma caused by tooth brushing, and iatrogenic factors such as uncontrolled orthodontic movement of teeth and faulty restorations.[3] The various consequences are clinical crown lengthening, esthetic problem, hypersensitivity, root caries, abrasion and patient's concern of tooth loss. The gingival recessions have been successfully treated by several periodontal plastic surgery procedures. The ultimate goal of these surgical procedures is the complete restoration of all anatomical structures in the area of recession and an optimal aesthetic outcome.[4] These can be divided into three main groups: pedicle soft tissue grafts, free soft tissue grafts and regenerative techniques.[5] The pedicle graft was the first periodontal plastic surgery procedure. The displaced pedicle flap technique was originally described by Grupe and Warren in 1956 as a laterally repositioned full thickness flap which is one of the most predictable method.[6] Healing studies have confirmed that plastic repair of denuded root surfaces is possible when the laterally positioned flap is used, where the "bridging" phenomenon is effected by new connective tissue and epithelial attachment to the previously exposed cementum.[2] Several modifications have been suggested to the original Laterally positioned flap technique of Grupe and Warren. Grupe in 1966

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suggested performing a submarginal incision at the donor site in order to preserve the marginal integrity of the tooth adjacent to the recession defect. The objective of this case report was to describe a case where root coverage was achieved by Modified lateral pedicle flap.

Case report:

A patient named Ms. Swetha 18 year's old female came to our college with a chief complaint of sensitivity in lower front region and receding gums for past 1 year. Patient was referred to the Department of Periodontology and Oral Implantology Unit for the management. On intra oral examination 31 was found with Miller's class I gingival recession (Figure 1). Patient's medical and dental histories were noncontributory. Thorough Scaling and Root Planing was performed and patient was recalled after 4 weeks for evaluation. After departmental discussion and patient's consent, it was decided to surgically treat the recession with laterally positioned flap with submarginal incision at donor site.

Presurgical therapy:

The surgical procedure was explained to the patient and an informed consent was obtained. The clinical parameters such as probing depth, recession depth and width were recorded before the surgery (Figure 2). The patient was asked to rinse with 10 ml of 0.12% chlorhexidine, prior to surgery to reduce the bacterial load.

Surgical procedure:

The surgical site was properly isolated and anasthetised, Root planing was performed in 31. The recipient bed was prepared by de-epithelizing the area adjacent to the recession site. Since the papilla was intact in the recipient site with #15 BP blade a submarginal horizontal incision was given extending from the distal line angle of 31 to the mesial line angle of 33 without involving the interdental papilla. Vertical incision was then given extending to the mucosal tissue,the base of the flap being wider for adequate vascularity. The vertical incision was extended far enough apically to ensure the adequate mobility of the flap. The donor flap was then reflected using a periosteal elevator. The flap was sufficiently wider than the recipient site to cover the root. The flap was slid laterally onto the adjacent denuded root,making sure that it is adapted properly without excess tension (Figure 3). Flap was then secured with sling suture (5-0 Vicryl suture) (Figure 4). After suturing we identified a little frenal pull in the surgical site so frenum relief was given and a periodontal dressing was placed to protect the surgical site (Figure 4).

Figure 1: Preoperative

Figure 2:Clinical parameters measurement
Fig 2a: Recession depth(3 mm) Fig 2b: Recession width(3 mm) Fig 2c: Probing depth(1 mm)

Figure 3:Submarginal insicion at donor site and transfer of flap

Figure 4:Sutures placed

Figure5:Periodontal dressing

Postoperative instructions:

For postoperative pain control Analgesics was prescribed twice daily for three days. 0.2% chlorhexidine digluconate was also prescribed to be used twice daily for 3 weeks. The patient was asked to refrain from tooth brushing at the surgical site for 2 weeks.

Result:

10 days after surgery periodontal dressing and sutures were removed,and the site irrigated with normal saline. Healing was uneventful and there is a good amount attached gingiva with complete recession coverage and excellent gain in the clinical attachment. (Figure 6).

Discussion:

The treatment of gingival recession for aesthetics or root sensitivity is a frequent demand in patients with high standards of oral hygiene.[7] Gingival recession can be successfully treated using several surgical procedures,regardless of the technique used, the ultimate goal of a root coverage procedure is the complete coverage of the recession defect and an optimal integration of the soft tissue. Complete root coverage will not only lead to an esthetic correction but also helps in resolution of hypersensitivity and prevention of root abrasion (Chambrone et al., 2012)[8]. Lateral pedicle graft, introduced by Grupe and Warren in 1956, is one of the first surgical procedure of various mucogingival surgeries designed to cover exposed root surfaces.[5] The laterally positioned flap can be used to cover isolated, denuded root surfaces that have adequate donor tissue laterally.[9] The advantages of the lateral pedicle graft are that it is relatively easy technique, it produces excellent esthetic results and no second surgical site is involved for donor harvesting. The disadvantages are that it is applicable only for single-site recession, there is danger of gingival recession at the donor site. Various modifications were done in original lateral pedicle flap to overcome the disadvantages. In 1966, Grupe done a modification in the lateral pedicle technique using submarginal incision at the donor site so that no denuded osseous surfaces would be created.[10] This technique was evaluated by many investigators (McFall, 1967[11], Smukler, 1976[2]), and the success of this root coverage procedure was found to be in the range of 69% to 72%. Staffelino in 1964 did a split thinkness flap to minimize recession at donor site, Corn in 1964 did a cutback incision at the base of the flap to provide a tension free flap.[3]

For several years lateral pedicle graft was one of the surgical procedure available that could predictably produce root coverage. The success of lateral or horizontally placed grafts depends on various factors. Its limitations that may contraindicate its use such as[12]:

- An insufficient amount of gingival tissue available for positioning
- A shallow vestibule
- Secondary frenal attachment(s) at the donor site: and
- Multiple adjacent recessions.

In the present case, lateral pedicle flap with submarginal incision was chosen to prevent the denuded osseus surface and to retain the integrity of the marginal gingiva and interdental papilla of the adjacent teeth. This procedure gave successful

Evidence of Healing was good and root coverage was satisfactory at three month post operative period.

Figure 6: Postoperative (10 days)

Preoperative Post operative- 3 month

outcome,wherein the patient was relieved of sensitivity. Even though the tooth was in malalignment, as there was adequate tissue available from the adjacent site, there was no problem in achieving primary closure at the donor site, hence it healed uneventfully. Since the Lateral pedicle graft carries its own blood supply, excellent healing and high survival rate were established.

Conclusion:

The loss of gingiva in anterior region can often lead to esthetic and functional challenge for clinician and patient. It is important to provide optimum functional and esthetic solution for the missing gingival tissue and simultaneously to preserve periodontal health. In the present case a laterally positioned flap technique with submarginal incision was used to cover Millers CLASS I defect in mandibular anterior regions. This technique has been demonstrated to be a reliable and predictable treatment modality for obtaining root coverage in Miller class- I and class II recession defect for complete root coverage. However careful case selection and surgical management is critical if a successful outcome has to be achieved. Marked esthetic and functional results can be obtained with lateral pedicle grafts for replacing marginal tissue there by increasing the width of attached gingiva.

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Treatment of multiple gingival recession using mother cell derivative with coronally advanced flap technique

Indumathi P.[1], Ramakrishnan T.[2], Dhivya R.[1], Vidya Sekhar[3], Ebenezer M.[3], Mejalla M.A.[4]

ABSTRACT

Gingival recession is one of the most common soft tissue problems, especially when it hinders the esthetics of a patient, it can further leads to root caries, sensitivity, and difficulty in plaque control. Gingival recession is treated to restore the gingival margin to the cement enamel junction (CEJ). The use of amniotic membrane is a novelty in the dentistry field, as it reduces the drawbacks of other materials. The aim of this case report was using amniotic membrane with coronally advanced flap technique without vertical incision in the treatment of multiple gingival recession defects. Amniotic membrane was effective in providing root coverage and an effective alternative to autograft tissue in the treatment of Miller's Class I and II recession defects.

Key words : Gingival recession, Amniotic membrane, Coronally advanced flap

Introduction:

Exposure of tooth by the apical migration of gingiva is called gingival recession,or atrophy.[1] Gingival recession occurs either due to a direct mechanical or physical injury to the gingival tissues or indirectly due to an inflammatory reaction in the gingival tissues.[2] Exposed root surface cause problems like unesthetic appearance, dentinal hypersensitivity, pulpal hyperemia besides being prone for caries.[3] Hence root coverage has become a very important procedure in periodontics. Various periodontal plastic surgical techniques have been used for its treatment. Dino et al.[4] revealed for the 1st time that amniotic membrane could be separated, sterilized and safely used at a later date. Amnion-derived cells have attracted lot of attention in the regeneration of periodontal tissues.

Recent data suggest that amniotic membrane provides positive results in terms of increased tissue thickness, root coverage, increased attached gingival tissue, as a barrier membrane for intrabony defects, healing of soft and hard tissue and provides excellent esthetic results in term of color and texture match. As the membrane is considerably thin, along with having a self adherent nature, it intimately adapts to the contours around the roots and defects, eliminating the need to use sutures so the surgical time is significantly reduced.[5] Amnion lines the innermost portion of the amniotic sac of the placenta. Its structure consists of a single layer of epithelium cells, thin reticular fibers (basement membrane), a thick compact layer, and a fibroblast layer. The basement membrane contains collagen type III, IV, and V and cell-adhesion bioactive factors including fibronectin and laminins.[6] According to various studies the amnion basement membrane closely mimics the basement membrane of human oral mucosa. The use of amniotic membrane is a novelty in the dentistry field, as it reduces the drawbacks of other materials. The aim of this case report was using amniotic membrane with coronally advanced flap without vertical incision in the treatment of multiple gingival recession defects.

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Case report

A 32-year-old systemically healthy male patient was referred to the Department of Periodontology at Adhiparasakthi dental college and hospital Melmaruvathur, with the chief complaint of receding gums with hypersensitivity. The patient had a Miller class I recession defect on the mandibular anteriors (figure 1). The patient had positive tactile and airblast sensitivity. Any etiology that may contribute to the progression of recession was identified and ruled out. Also, instructions for proper toothbrushing and oral hygiene were given to ensure maintenance before, during, and after therapy. The procedure was explained to the patient, and informed consent was obtained. A root coverage with coronally advanced flap utilizing amnion membrane was planned after Phase I therapy (after 4 weeks).

Surgical procedure

Measurements were recorded, and the surgical area was prepared with adequate anesthesia using 2% lignocaine hydrochloride containing 1:80,000 adrenaline. With the use of no .15 B.P blade, crevicular incision was given in relation to 31,32,41&42 region. A full thickness flap was elevated (figure:2) and thorough root planing was done. Amniotic membrane was placed on the denuded root surface(figure:3) and flap was repositioned coronally and sutured [figure:]. Care was taken to prevent the movement of the membrane during flap closure. Periodontal dressing given (figure:5)

Post surgical procedure:

Patients were advised not to brush on the treated site for 2 weeks and instead 0.2% chlorhexidine rinse

Figure 1 : Preoperative view

Figure 2 : Flap elevation

Figure 3: Amniotic membrane placed

Figure 4: Suturing

Figure 5 : Periodontal dressing

Figure 6 : 1 Week post-operative view

Figure 7: 3 Month post-operative view

Pre operative view

Post operative view

was prescribed for 3 weeks. Antibiotics and analgesics were prescribed postoperatively. The patient was examined at 1st and 4th week to assess healing and then followed-up at 3 months for reassessment of clinical parameters.

Results

Postoperative results showed a decrease in recession depth , recession width and gain in clinical attachment level. Increased width of keratinized gingiva with 100% root coverage was achieved in this case (Figure 6,7). There was significant increase in WKG and gingival thickness from baseline to 3 months which may be due to the presence of mitogenic factors and anti-inflammatory proteins present in the placental membranes

Discussion

The ultimate goal of periodontal plastic surgical procedure for recession coverage is the complete regeneration of the supporting components of the periodontium, resulting in complete coverage of the denuded root surfaces in an esthetic as well as a functional manner.[4] The recent resorbable amniotic membrane not only maintains the regenerated tissues but also it enhances gingival wound healing, provides a rich source of stem cells. Hence , amniotic membrane is the choice of material nowadays in augmenting the better results in various periodontal procedures. Coronally advanced flap technique provides optimum root coverage with good colour blending and complete recovery of the original soft tissue marginal morphology.[7]

Properties of Amniotic Membrane[8]

Epithelialization, Anti-inflammatory,Anti-viral and anti-microbial, Anti-Scarring

Angiogenesis (formation of new blood vessels), Immunomodulatory properties

Reduction of pain, Increased extracellular matrix deposition

Advantages

Amniotic membrane cells having following advantages;[9]

- Readily available
- Normally discarded organ so fewer ethical concerns
- Non- invasive procurement procedure
- Abundant cells that are sufficient for cell replacement therapies
- Possess immunomodulatory properties so reduced risk of rejection
- No tumorigenicity
- Relatively easy isolation so cost effective
- Possess stem cell like characteristics

Gurinsky[10] stated that dehydrated amnion allograft provides good results in terms of root coverage, increased tissue thickness, and increased attached gingival tissue.

Velez et al.[11] stated that cryopreserved amniotic membrane (CAM) aids in cicatrization and wound healing after dental implant surgery. CAM supports the growth of epithelium thus facilitating migration and reinforcing adhesion.

Shetty et al.[12] stated that using amniotic membrane as a novel approach to root coverage is more advantageous than PRF owing to the laboratory preparation of the autologous biomaterial and use of the amniotic membrane as an additive material alternate to subepithelial connective tissue in reducing the need for a second surgical site is better advocated.

There has been an increase in the use of the amniotic membrane clinically as a soft tissue regeneration graft and as a barrier membrane. It is easily available and it is cost effective to preserve. The material provides a natural scaffold for wound healing and contains various important growth factors and biological macromolecules important in wound healing and serve as a scaffold for cell proliferation and differentiation as a result of its antimicrobial properties. AM is an excellent candidate to use as a native scaffold for tissue engineering.[13]

Owing to results in this study, amniotic membrane can be a reliable alternative to autogenous CTG in the treatment of gingival recession for, the former eliminates donor site morbidity, reduces the need for multiple surgeries and expense, saves time that would be required for harvesting a CTG, and offers unlimited graft availability of uniform thickness.

Conclusion

The present case report demonstrated CAF with amniotic membrane were effective in providing

complete root coverage and an excellent alternative to autograft tissue in the treatment of Miller's Class I and II recession defects.

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JSPIK

Comparative evaluation of efficacy of two pre-procedural mouth rinses in preventing the aerosol contamination in dental operatory

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ABSTRACT

Background and Objectives: Aerosol contamination during dental treatment is most common and can be the potential source of acute or chronic respiratory/systemic disease. Certain pre-procedural oral rinse may help in reducing the aerosol contamination. This study was conducted to assess and compare the efficacy of stabilized chlorine dioxide (SCD) and chlorhexidine gluconate (CHX) in preventing the aerosol contamination.

Materials and methods: 30 systemically healthy subjects were selected. They were allocated in three groups (A, B, C). Group A was given water whereas group B and C were given 0.2% CHX and SCD respectively as pre procedural mouth rinse. Three agar plates were kept at 1, 3 and 6 feet distance from patients' mouth. Then, ultrasonic scaling was done. Agar plates were incubated for 24 hours and then number of colony forming units (CFU) was counted.

Results: Group A showed significantly more number of CFU in agar plates at three positions. Among B & C groups, no significant difference was seen in number of CFU in agar plates at 3 and 6 feet ($p>0.05$). But at one foot distance, group C had statistically more no. of CFU than group B ($p< 0.05$).

Conclusion: CHX is more effective oral rinse in preventing the aerosol contamination.

Introduction

In our daily clinical practice of dentistry, there may be an inevitable environmental contamination through aerosol. Particles consisting of or conveying microorganisms, irritants, allergens, or other toxic substances can be atomized into the air during dental procedures and are a potential source of acute or chronic respiratory disease. Depending on their size, these particles remain suspended as aerosols or fall rapidly and splatter on objects in their trajectory. Airborne particles larger than 50 to 100 micrometers (μm) in diameter have inertial forces greater than the frictional forces of air and are ballistic in nature. True aerosol particles usually are less than 50 μm in diameter, are invisible, and remain airborne for long periods. Of particular interest as infective agents are bacterial aerosol particles in the 0.5- to 10-μm diameter range (median particle diameter 5 μm), which can be inhaled and impinged on the terminal bronchioli and alveoli of the human lung.[1] According to Stokes law, particles of this size fall very slowly.[2]

Aerobiology is the study of seasonal atmospheric diffusion of phytopathic fungus spores and aeroallergens over long distances and aerosolized bacteria and viruses associated with respiratory diseases diffused over short distances, mostly indoors.[3]

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Dental aerobiology is the study of airborne particles in the dental office and laboratory environment and the relationship of these particles to the health of personnel and patients. The dissemination of microorganisms from the mouth of dental patients during dental operations can occur by three routes: (1) by direct contact with the dentist's instruments and fingers, (2) indirectly by aerosolized microorganisms suspended in air, and (3) indirectly by droplet infection (eg. splatter from the coolants used in ultrasonic scalers).[4] Size of aerosols and splatter differs in aerosol being lesser than 50 microns in diameter.

Belting et al.[5] have found living tubercle bacilli in splatters generated by use of the air turbine handpiece during treatment of dental patients. These splatters landed six inches to four feet from the mouth of the patient and easily encompassed the area occupied by the dentist and his assistant.

Table 1

		N	Mean	Std. Deviation	Statistic	df1	df2	Sig.
1 feet	GROUP A	10	22.25	3.76571	170.786	2	12.781	<0.001
	GROUP B	10	2.02	0.70836				
	GROUP C	10	13.92	3.88496				
	Total	30	12.73	8.97407				
3 feet	GROUP A	10	9.78	4.23367	18.309	2	15.621	<0.001
	GROUP B	10	1.66	0.38644				
	GROUP C	10	1.46	0.52111				
	Total	30	4.3	4.60794				
6 feet	GROUP A	10	6.51	2.68181	22.204	2	13.29	<0.001
	GROUP B	10	3.13	2.5206				
	GROUP C	10	0.92	0.63034				
	Total	30	3.52	3.12944				

Table 2

Multiple Comparisons					
TukeyHSD					
Dependent Variable	(I) groups	(J) groups	Mean Difference (I-J)	Std. Error	Sig.
1 feet	GROUP A	GROUP B	20.23000	1.40891	<0.001
		GROUP C	8.33000	1.40891	<0.001
	GROUP B	GROUP C	-11.90000	1.40891	<0.001
3 feet	GROUP A	GROUP B	8.12000	1.10589	<0.001
		GROUP C	8.32000	1.10589	<0.001
	GROUP B	GROUP C	.20000	1.10589	.982
6 feet	GROUP A	GROUP B	3.38000	.96412	.004
		GROUP C	5.59000	.96412	<0.001
	GROUP B	GROUP C	2.21000	.96412	.074

So, this study assesses and compares the quantity of aerosol contamination using an ultrasonic scaler at one, four and six feet of distance from patient's mouth, with or without pre procedural mouth rinse. Thus, the primary objective of the study included counting the number of colony forming units on the agar plates after ultrasonic scaling in patients who pre rinse with CHX/SDC mouth wash preoperatively (case groups) as compared to water as pre rinse (control group). Secondary objective was to compare the efficacy of CHX and SDC

Subjects and methods:

The study was conducted in the Department of Periodontics, KVG Dental College, Sullia after the ethical clearance was obtained from the Institutional Review Board. Thirty participants with at least 20 permanent teeth and a mean plaque index (Silness and Loe[6]) score between one and three were considered for inclusion. Also, gingival index of score (Loe and Silness[7]) between one and three were included in the study. It was made clear to the potential subjects that participation was voluntary and written informed consent was obtained from those who agreed to participate. Patients with medical conditions contraindicating the use of ultrasonic scaler (communicable disease) and patients who were under systemic or topical antibiotics were excluded from the study. The subjects were divided into group A, group B, and group C. Study design is shown in the line diagram (Fig.1)

Three standardized locations of the operatory were chosen to evaluate for each patient using blood agar plates placed at - patient's chest area, and at a distance of three and six feet from the operating area on a standardized chair with controlled frequency and water pressure during treatment procedure Johnston et al.[8] has proved that blood agar plates are a valid medium for culturing airborne bacteria.

The average distance between the patient's mouth and his own chest was one foot (12 inches). The treatment for all the study patients was carried out by

Table 3

GROUPS			Mean	N	Std. Deviation	t	df	Sig. (2-tailed)
GROUP A	Pair 1	1 feet	22.25	10	3.76571	7.029	9	**<0.001**
		3 feet	9.78	10	4.23367			
	Pair 2	1 feet	22.25	10	3.76571	11.82	9	**<0.001**
		6 feet	6.51	10	2.68181			
	Pair 3	3 feet	9.78	10	4.23367	2.948	9	**0.016**
		6 feet	6.51	10	2.68181			
GROUP B	Pair 1	1 feet	2.02	10	0.70836	1.53	9	0.16
		3 feet	1.66	10	0.38644			
	Pair 2	1 feet	2.02	10	0.70836	-1.274	9	0.235
		6 feet	3.13	10	2.5206			
	Pair 3	3 feet	1.66	10	0.38644	-2.062	9	0.069
		6 feet	3.13	10	2.5206			
GROUP C	Pair 1	1 feet	13.92	10	3.88496	10.612	9	**<0.001**
		3 feet	1.46	10	0.52111			
	Pair 2	1 feet	13.92	10	3.88496	11.467	9	**<0.001**
		6 feet	0.92	10	0.63034			
	Pair 3	3 feet	1.46	10	0.52111	8.734	9	**<0.001**
		6 feet	0.92	10	0.63034			

the same dentist on all the days and only one patient was treated per day to allow the room to be free of aerosol. Before each appointment all the operatory surface was cleaned and fumigated.

Prophylaxis was carried out with a piezoelectric scaler (Satelac® P5 Booster). The procedure was carried out for 30 minutes. The speed of the scaler and the flow of coolant were standardized for all the patients. Treatment was carried out by placing three sterile coded agar plates uncovered at pre designated sites to collect samples of aerosolized bacteria. The agar plates are shown in Fig. 2.

After the samples are collected, they were taken to the Department of Microbiology, KVG Medical College, Sullia for further analysis. All the samples were incubated for 24 hours and then the counting of CFU was done using manual method. Grid square of six square centimeters was drawn with each square of one square centimeter. The CFUs in each small square was counted. If the CFU overlapped with border of the square, then only those were counted which had contributed >75% to that square. Once the CFUs were counted in all the squares, the average was taken out by dividing the total sum of CFUs by the number of squares. This gave the average CFUs/cm2.

Results

Statistical analysis was done using one way ANOVA Welch test. Also, post-hoc analysis was done using Tukeys test.

Table 1 shows that at one feet, two feet and six feet there is significant reduction of CFU when chlorhexidine or chlorine dioxide were used as mouth rinse compared to when no mouth rinse was used. The contents of the table 1 are graphically presented in fig. 3, fig. 4, and fig. 5 at one feet, three feet and six feet respectively.

Table 2 shows various group comparisons at different distances. At one foot when group A is compared to group B and C, there is highly significant reduction of CFUs. Also, when group B and C are compared to each other, there is significant difference in reduction of aerosol contamination by chlorhexidine mouth wash as pre rinse. At three feet, similar results were obtained when we compare group A with group B and C. But when group B and C are compared to each

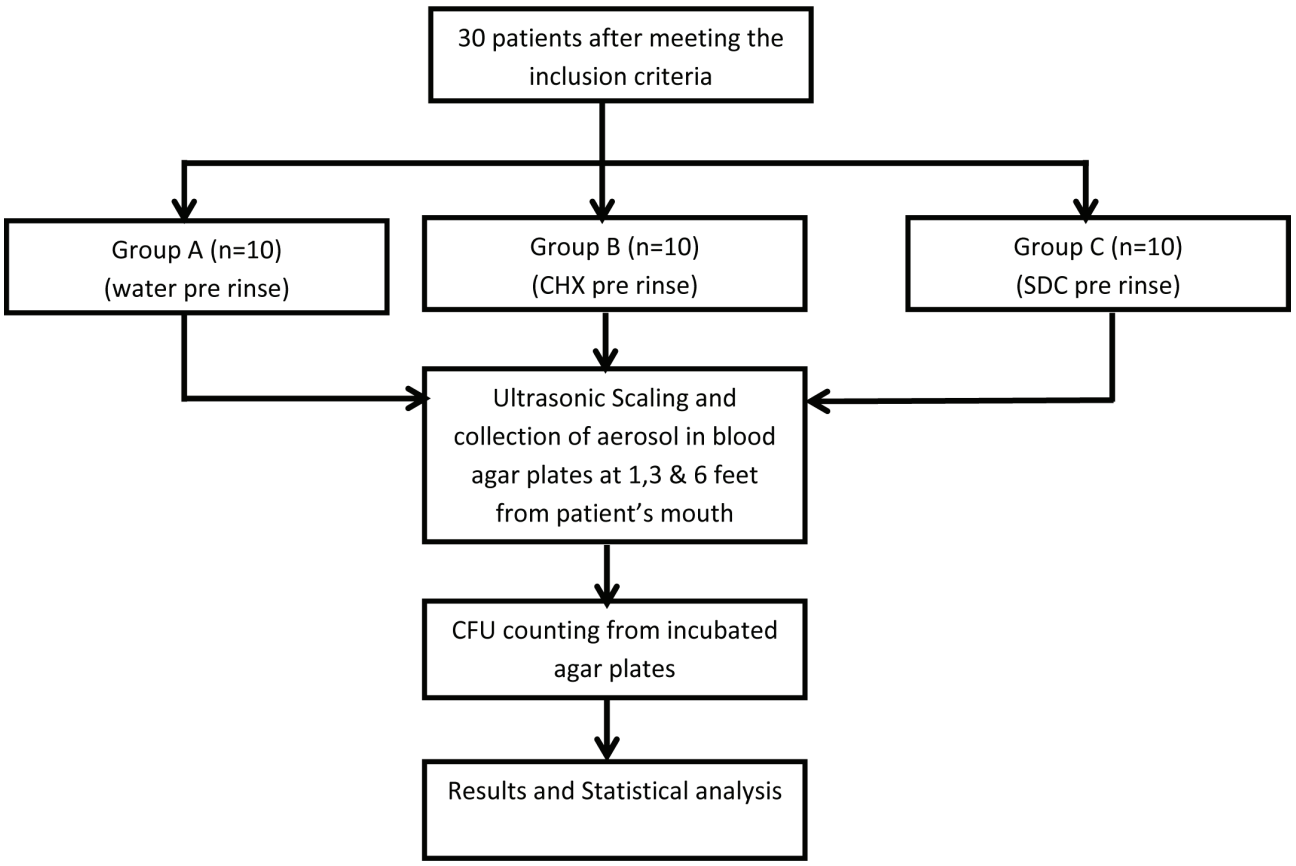

Figure 1

other, there is no significant difference in the reduction of the CFUs. At six feet, when group A is compared to group B and C, there is significant reduction in CFU counts. On the other hand, group B is compared to group C there is no significant difference obtained in CFU counts.

Table 3 shows comparison of the CFU reduction within a group at various distances. In group A, in which no mouth rinse was used, the mean CFUs at a distance significantly decrease with the increase in the distance. In group B, there is no significant difference in CFU counts when comparisons were drawn between one and six feet or at three and six feet. But, there is significant difference when CFUs at one foot were compared to CFUs at three feet.

Discussion

The compound chlorine dioxide, now commercially important, is not in fact a recent discovery. The gas was first produced by Humphrey Davy in 1811 when reacting hydrochloric acid with potassium chlorate. This yielded "euchlorine", as it was then termed. Watt and Burgess, who invented alkaline pulp bleaching in 1834, mentioned euchlorine as a bleaching agent in their first patent. Chlorine dioxide then became well known as bleach and later a disinfectant.

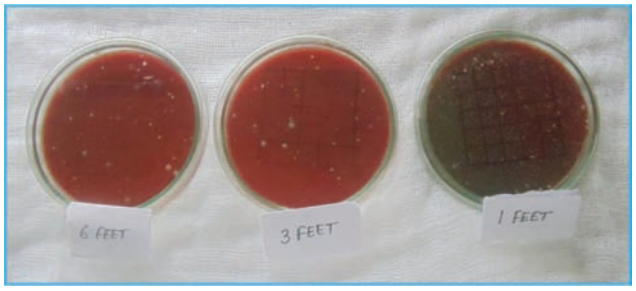

Figure 2

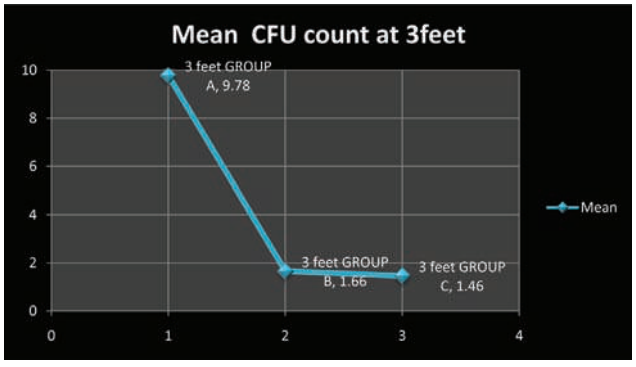

Figure 4

Stabilized chlorine dioxide is an aqueous solution comprising sodium chlorite and stabilizers. When the pH of stabilized chlorine dioxide is lowered from a neutral pH, molecular chlorine dioxide is released from the aqueous solution. This mechanism of action of stabilized chlorine dioxide has bactericidal and bacteriostatic effects on the microbial ecology of aerobic, facultative and anaerobic pathogenic bacteria.

In literature, there has been reported the marked bactericidal activity of chlorine dioxide mouth rinse. Two published clinical studies were performed to assess the effect of a stabilized chlorine dioxide oral rinse and toothpaste on both gingivitis and oral soft tissue.[9,10]

Studies have shown that chlorhexidine mouth wash was effective pre rinse in reducing the amount of aerosol contamination when compared to water as pre rinse.[11] The results of the present study indicate that chlorhexidine significantly reduces the CFU counts when it was compared to chlorine dioxide and water mouth rinse groups.

Table 2 shows that there is significant difference in each group except chlorhexidine and chlorine dioxide group in the 3 and six feet variables.

But when compared, chlorine dioxide oral rinse

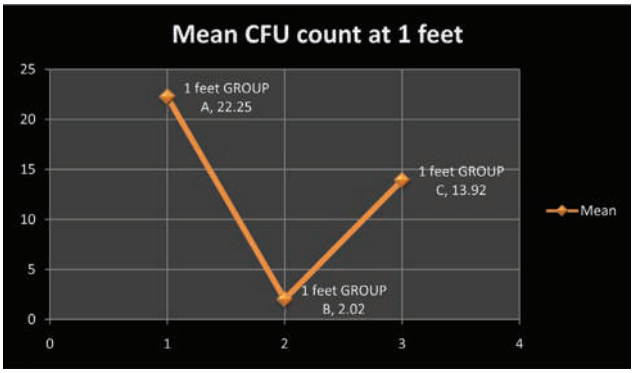

Figure 3

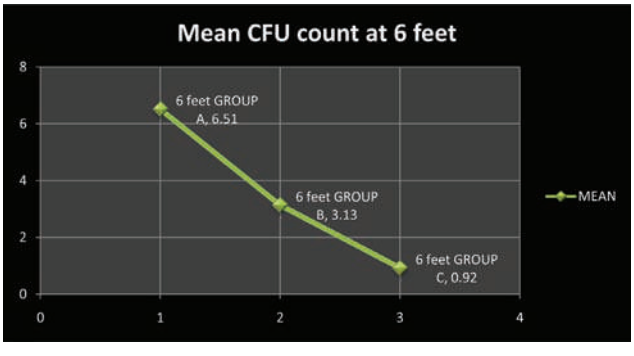

Figure 5

exhibited less bactericidal activity against some of the test organisms than chlorhexidine.[12]

The results of the present study indicate that both chlorhexidine and chlorine dioxide mouth wash can reduce the aerosol contamination compared to water when given as mouth rinse. Importantly, at one feet distance chlorhexidine was significantly more effective in controlling the aerosol contamination than chlorine dioxide. This becomes further relevant considering the fact that most of the operators and assistants will be at this distance during the treatment.

Conclusion

Within the limitations of the study, it can be concluded that chlorhexidine can be effectively used as a pre procedural mouth rinse. Chlorine dioxide can be used too. But, chlorhexidine remains the gold standard.

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Microbial and clinical effects of orthodontic treatment on periodontium

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ABSTRACT

Active orthodontic treatment in oral cavity results in clinical and microbial changes which include shift in microbial flora towards being more pathogenic. Gingival index, plaque index, bleeding index and probing depth shows transient change during treatment. This review summarizes the microbial and clinical effects of orthodontic treatment on periodontium.

Keywords: Microbial change, Periodontium, Orthodontic treatment, Attachment loss

Introduction

The interdigitation of orthodontic treatment and periodontal changes has invited the attention of both researchers and clinicians since decades. This article reviews the microbial changes and clinical effects of orthodontic treatment on the periodontium.

Microbial changes

The bands, brackets, orthodontic wires and accessories like coil springs, tubes, ligature ties and elastomeric chains create a unique environment for plaque accumulation. The very same components challenge the dexterity of patients in maintaining oral hygiene. All these compounds change over periodontal microbiology, making it pathogenic. The general shift is from being aerobic to anaerobic bacteria. There is an increase in spirochetes, fusiform bacteria,facultative anaerobes, Lacto bacilli and Prevotella intermedia.[1-4] The amount of cocci decreases and motile rods are increased. These changes are initiated within 12 days and by 3 months, red and orange complexes are established. The subgingivally placed orthodontic bands invariably develops red complex.[5] Campylobacterrectus, Veillonella, Peptostreptococcus, Actinomycesviscosus and Bacterionemarothia are also found to increase significantly.[6] Jewtuchowiczetal[7] reported a 3 fold increase in the prevalence of yeast like Candida albicans, C.parapsilopsis, C.dubliniensis, C.tropicalis, C.guilliermondii, C.sake and Rhodotorula species.

Clinical effects

An increase in inflammatory response due to the microbial shift result in concomitant increase in gingival index (GI) and bleeding on probing index (BOP) or gingival bleeding index (Table 1).[8] Though the tremendous increase in plaque retentive sites present a highly conducive environment for inflammation and periodontal problem, this appear to be transient in many cases followed by significant improvement in oral hygiene status.[9-11] This improvement has direct correlation with the reinforcement of oral hygiene instructions and measures taken by orthodontist and the compliance level of patient.[12]

The increase in probing depth due to the hyperplastic gingiva as a result of appliance irritation is mostly not associated with attachment loss. This type of inflammatory change is shown to have direct relation with the band placement and usually resolves within 48 hours of band removal.[14] True attachment loss in orthodontic treatment cases is often due to the combination of heavy force and active periopathogenic bacterial activity.[17] This finding underlines the significance of proper oral hygiene and periodontal care during active orthodontic treatment.

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The recession associated with tooth movement occurs when the tooth is moved out of its alveolar housing. Thus, injudicious use of mechanics could result in dehiscence and fenestration which becomes more pronounced if the tooth is already periodontally compromised in terms of recession or morphology. Apart from the above mentioned clinical effects, orthodontic appliance placement can cause gingival erosion, contusion, ulceration and even chemical burns from the bonding materials.[13,15,16]

Conclusion

The clinical and microbial changes effected by orthodontic appliances and treatment are largely reversible (Table 2).[8] Yet their influence in a comprehensive treatment cannot be neglected especially in periodontally compromised population. Since the number of adults seeking orthodontic correction is increasing, the chances of encountering periodontally compromised malocclusion is high. A proper protocol must be observed in treating such

Table 1: Clinical Parameter Change During Active Orthodontic Therapy (Davis et al 2014)[8]

References	Plaque index	Gingival index	BOP/GBI	PPD
Zacchrisson and Alnaes	Not reported	Not reported	Not reported	increased
Kloehn and Pfeifer	Decreased	Not reported	Not reported	Not reported
Karkhanechi et al	Increased	Increased	Increased	Increased
Sinclair et al	No change	Increased at bonded sites	Not reported	No change
Paolantonio et al	decreased	Not reported	Decreased	No change
Naranjo et al	Increased	Increased	Increased	No change
Ristic et al	Increased	Increased	Increased	Increased
Lo et al	decreased	decreased	Not reported	Not reported
Liu et al	Increased	Increased	Not reported	No change
Van Gastel et al	Not reported	Not reported	Increased	Increased

BOP: bleeding on probing; GBI: gingival bleeding index; PPD: probing pocket depth

Table 2: post treatment clinical changes in periodontium[8]

References	PI	GI	BOP/GBI	PPD
Kloehn and Pfeifer	Decreased	Not reported	Not reported	Decreased
Sallum et al	Decreased	Decreased	Not reported	Decreased
Liu et al	Decreased	Decreased	Not reported	Decreased
Van Gastel et al	Not reported	Not reported	Decreased	Decreased

PI: plaque index; GI: gingival index; BOP: bleeding on probing; GBI: gingival bleeding index; PPD: probing pocket depth

patients to ensure quality care which is vital (Geisinger et al 2014).[18]

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A comprehensive approach for management of amilodipine induced gingival overgrowth - a case report

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ABSTRACT

Gingival overgrowth/hyperplasia is an abnormal growth of the gingival tissue associated with multiple factors including inflammatory (acute and chronic), idiopathic, drug-induced, neoplasia (benign and malignant tumours), hormonal disturbances, ascorbic acid (vitamin C) deficiency and with dental eruption. Drug-induced gingival hyperplasia arising secondarily to prolonged use of drugs is the most common gingival overgrowth. Currently, more than 15 drugs have been identified as possible causative agents, including oral contraceptives. However, there are 3 classes of drugs that are well-established causes of gingival enlargement, being responsible for most cases: Anti-epileptic drugs, antihypertensive calcium antagonists and immunosuppressant like cyclosporine etc. Amlodipine, a long-acting calcium channel blocker, an anti-hypertensive drug was considered a "safe drug" having relatively less adverse drug reactions. However with an increase in the usage of amlodipine an adverse effect of gingival overgrowth has been reported. This article reviews about amlodipine induced gingival enlargement with a case report and its management with comprehensive approach.

Key Words: Amlodipine induced Gingival Hyperplasia, Diode LASER,Gingivectomy

Introduction:

Gingival hyperplasia is defined as an abnormal growth of the gingival tissue.[1] It is incongruous to use the term "gingival hyperplasia" because enlargement of the gingival tissue is the result of an increase in the volume of extracellular tissue , rather than an actual increase in the number of cells.[2] Gingival enlargement results in aesthetic changes and clinical symptoms such as pain, tenderness, bleeding, periodontal disorders, abnormal tooth movement, enhancement of caries development, dental occlusion problems and speech disturbances.[3] It may be caused by medications (antiepileptic drugs)[4-7], genetic abnormalities (hereditary gingival fibromatosis, proliferative lesions, etc).[8-9]

Gingival hypertrophy related to medication, has been described as one of the important enlargement. It is well established that the drugs most frequently induce gingival enlargement primarily falls under the following classes such as antihypertensive, antiepileptic, calcium antagonists and immunosuppressants. These groups of drugs possess one property in common that directly affect cellular calcium metabolism. Fibroblasts from patients treated with these drugs may produce an inactive form of collagenase due to the alteration of cellular calcium metabolism, results in subsequent lack

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of collagen and extracellular matrix degradation which eventually results in hypertrophy of gingival tissue.

Case report:

A 45-year-old female patient came to the department with the chief complaint of loose teeth in upper and lower front jaw regions since 1 year with swollen and bleeding gums. Bead like nodular growth over the gums were the first noted signs by the patient which progressively enlarged to the present size covering almost entire teeth interfering with further cleaning of teeth. The family history was not substantial. The history of present illness rendered that the patient noticed the enlargement for the past 6 months which was painless and progressive in nature.

The patient was hypertensive since 5 years and was under medication Coronol-AM (atenolol, 50 mg + amlodipine, 5 mg) once daily. The patient was moderately built and nourished with no signs of anaemia and jaundice and cyanosis. Patient's vital signs were within the normal range.

Clinical Presentation:

Intraoral examination revealed generalized enlargement of attached gingiva extending up to marginal and interdental gingiva. The gingival surface appears lobulated with loss of scalloping and stippling. Poor oral hygiene status of patient was assessed by the presence of local irritating factors surrounding the teeth.

A provisional diagnosis of combined gingival enlargement was arrived on the basis of drug history and clinical examination of the patient. Complete hemogram of the patient was carried out. All the parameters were under the normal range. An orthopantomogram taken, revealed generalized bone loss.

Fig 1,2,3- Pre-Operative Photographs

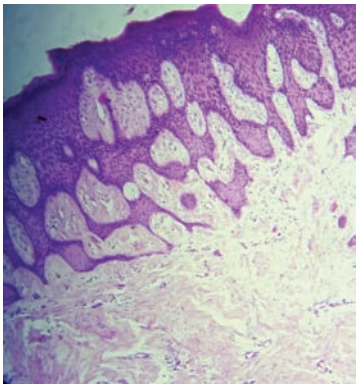

Fig 4-Parakeratinised stratified squamous epithelium with underlying dense connective tissue stroma(Mag. 4x)

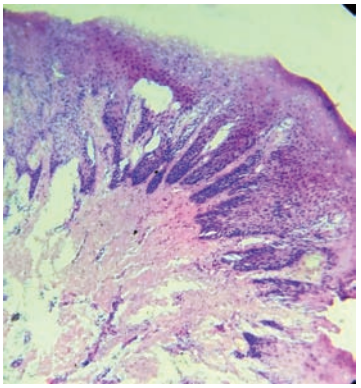

Fig 5- Epithelium showing hyperplastic changes, slender elongated rete ridges and areas of koilocyticchanges(Mag. 10x)

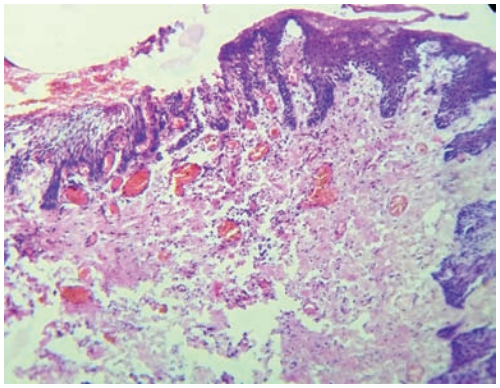

Fig 6- Connective tissue showing vascular channels filled with RBCs and areas of Chronic inflammatory cells(Mag. 10x)

Histological Presentation:

Histopathological examination of an incisional biopsy taken from the upper and lower anterior gingivae reveals parakeratinised stratified squamous epithelium with under lying dense fibrous collagenous stroma (Fig 4) exhibiting the presence of plump fibroblasts and dilated vascular channels filled with RBCs (Fig 6). Presence of perivasculitis and dense chronic inflammatory cell infiltration chiefly of lymphocytes are seen in the connective tissue. The overlying epithelium shows hyperplastic changes with slender long rete ridges extending deep in to the connective tissue stromakoilocytic changes are seen in the cells of surface epithelium (Fig 5). Presence of mitosis is also evident in few areas.

Diagnosis:

A final diagnosis of combined gingival enlargement (amlodipine induced and inflammatory) was arrived at after correlating the history, clinical findings, and investigations. Preoperative photographs were taken (Fig 1, 2, 3). Planned sessions of scaling and root planning along with a change in drugs were carried out under the patient's physician consent. Patient was under medication (tablet Normadate 100 mg twice daily) and was evaluated after a period of 6 weeks. There was an exorbitant change in the clinical picture of gingiva with complete loss of inflammatory component.

Treatment:

Definitive treatment involves surgical elimination of the excess gingival tissue through implementation of either the gingivectomy procedure or periodontal flap method. The choice of the clinician among these two surgical techniques should be made on an individual basis, considering the following aspects: the extent of area requiring surgery; the presence of periodontitis and osseous defects; the diode laser

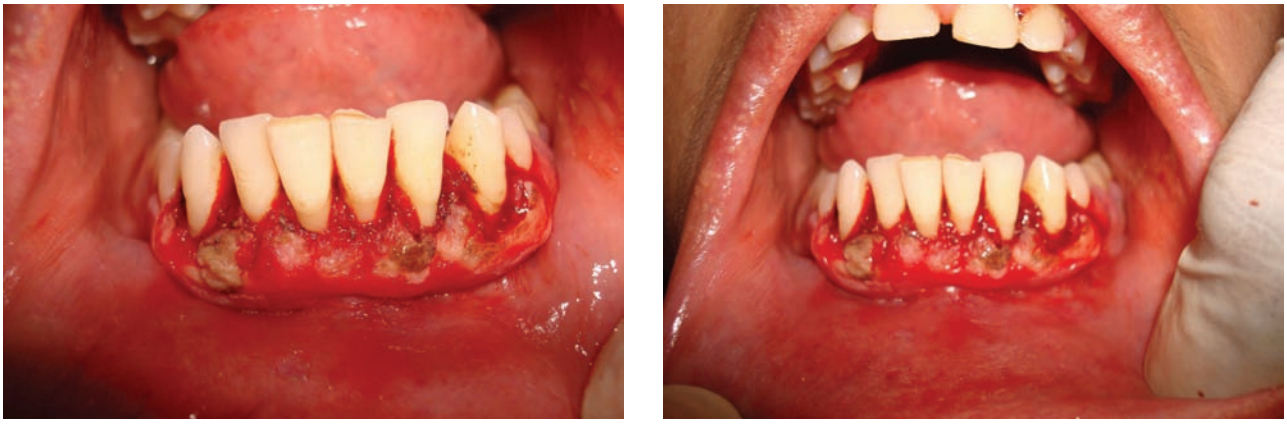

Fig 7,8- Immediate Post Operative photograph after diode LASER gingivectomy

Fig 9,10,11- Two Months Post Operative Photograph

surgical therapy is also becoming more common in treatment of gingival overgrowth due to its advantages in postoperative haemostasis.[10]

Given the significance of plaque and calculus as risk factors for exacerbation of gingival enlargement, preliminary periodontal therapy was aimed at non-surgical periodontal treatment which included scaling and root planing, usage of 0.12% chlorhexidine mouthwash and regular application of metronidazole gel.

After obtaining the physician's approval regarding patient's medical condition for the surgery, the complete surgical procedure was elucidated to the patient and an informed consent was received. The gingivectomy procedure was intended for one quadrant in every appointment at an interval of 1 week after achieving adequate anesthesia using 2% xylocaine with adrenaline (1:2,00,000). Precise identification of the pockets on all the surfaces of all teeth within the surgical field was done with the help of a pocket marker at three points/tooth (mesial, mid-radicular and distal line angles) on the labial /buccal and lingual/palatal aspects. An external bevel incision was made with the help of a No.15 Bard Parker blade and Kirkland surgical periodontal knife. Interdental incisions were made using Orbans knife and the bulk of tissue was removed using curettes. Tissue tags were excised by means of Goldman fox surgical scissors. The surgical spot was then examined for any residual debris. The patient was given post-operative instructions and was instructed to take analgesics (combination of paracetamol-500 mg and diclofenac-50 mg) thrice daily for duration of 3 days. The surgical procedure was repeated for the rest of the quadrants. Healing was uneventful. Diode Laser gingivectomy also performed to remove the excess bulk of tissue after periodontal flap surgical procedures. The use of diode laser in the present case effectively removed small amounts of remaining hyperplasic gingival tissue (Fig 7,8,) and resulted in fast healing and mild discomfort.

Regular follow-up of the patient was maintained and oral hygiene instructions were reinforced at every appointment in a gap of 3 weeks (Fig: 9,10,11).

Management:

Although recurrence can occur in some cases, meticulous home care, chlorhexidine gluconate rinses, and close 3-monthly maintenance and professional debridement following surgery will minimize this possibility. Relapse may occur 3-6 months after surgical treatment, but in general maintenance of surgical results for at least 12 months is reported.[11]

Treatment consists of stopping the offending drug if possible with the patient's physician consent. Reduction in the size of the gingival overgrowth has been reported within a week of drug withdrawal and may lead to full resolution.[12]

Discussion:

The American Academy of Periodontology 2001 defines drug-induced gingival enlargement as an overgrowth or increase in size of gingiva resulting in whole or part from systemic drug use.[13] Currently, there are over 20 medications from three pharmaceutical categories including calcium channel blockers, anticonvulsants and immunosuppressants that are associated with gingival enlargement.[14]

Amlodipine is a dihydropyridine calcium antagonist that inhibits the transmembrane influx of calcium ions into cardiac and vascular smooth muscles. It is often used for the treatment of angina and as an anti-hypertensive.[15]

The mechanisms by which calcium antagonists induce gingival hyperplasia have yet to be fully explained.[16] Amidst the numerous projected mechanisms, the best hypothesis Hitherto is that calcium antagonists inhibit the influx of calcium ions which is needed for the degradation and synthesis of collagen.[17] The accumulated collagen and extracellular matrix not degraded owing to inhibition of calcium influx by calcium antagonists is suggested to cause gingival hyperplasia. Furthermore, the significance of good oral hygiene for prevention of gingival hyperplasia is emphasized.[18]

The first case of amlodipine-associated gingival overgrowth was reported by Ellis in 1993.[19] The clinical appearance of gingival overgrowth frequently appears within 2-3 months after starting treatment with

amlodipine, alike other calcium channel blockers.[20, 21]

Several factors may influence the relationship between the various implicated drugs and components of the gingival tissues, which includes: age, pharmacokinetic variables, genetic predisposition,ultra structural factors, drug-induced alterations in gingival connective tissue homeostasis, and inflammatory changes, drug-induced action on growth factors, etc.[22]

Three significant factors which are important in the expression of these gingival changes can be considered: plaque-induced inflammatory changes in the gingival tissues, drug variables,and genetic factors – the latter determining the heterogeneity of the gingival fibroblasts.[22] Some drugs induce a direct effect on a subgroup of fibroblasts, named "responders", that are apparently genetically determined to be sensitive to the drug causing gingival enlargement. These drugs produce a decline in calcium influx (due to alterations in calcium-sodium exchange), hereby producing a fall in cellular folic acid uptake (producing a localized folate deficiency) thus, restricting the synthesis of the collagenase-activating enzyme (the active form of collagenase). Also, since the presence of inflammation secondary to dental plaque causes proliferative increase in connective tissue, it saturates the catabolic strength of collagenase, thereby causing a local accumulation of this matrix owing to the inhibited degradation of the extra cellular matrix.[3,23]

There is an inconclusive relationship between the drug dose and the severity of the gingival enlargement, drug concentrations in serum and time span of therapy, saliva and gingival crevicular fluid.[24] Several other factors may be involved in Drug induced gingival enlargement such as androgenic hormones.[25] Brown et al. (1991) have pointed out in this entity several factors: increase of sulphate glycosaminoglycans, immunoglobulins, epithelial growing factor; calcium and sodium rupture efflux in fibroblasts, folic acid and colagenase deficiency.[23]

Although one of the foundations for treatment of all drug-induced gingival overgrowths is discontinuation of the medication along with drug substitution, it was not possible however, in the present case, as those drugs were lifesaving drugs for the patient according to the patient's physician. Therefore we proceeded with the best possible treatment in order to enable the patient to maintain his oral hygiene

Conclusion:

Gingival hyperplasia could occur with amlodipine even at a small dose (5 mg). Physicians and dentists should be aware of the etiologic medications that can induce gingival hyperplasia and be able to identify changes in the oral cavity in such patients and to prevent, diagnose, and successfully manage them. It can be treated locally and systemically with combined effort of medical and dental physician. So, cooperative teamwork between the patient, his physician, and the dental health care professional is mandatory to minimize and successfully treat such unwanted side effects of drugs.

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Assessment of myeloperoxidase levels in gingival crevicular fluid and serum in patients with aggressive periodontitis-a clinico biochemical study.

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ABSTRACT

Aim (Background and objective): In aggressive periodontitis, amplified activity of PMN s is linked with elevated levels of myeloperoxidase (MPO) enzyme in gingival Crevicular fluid when compared with chronic periodontitis. The aim of the study was to assess the levels of myeloperoxidase enzyme in GCF and serum in patients with aggressive periodontitis.

Materials and method: The study included thirty otherwise systemically healthy subjects and divided into three groups as; Group 1(10) Periodontally healthy, Group 2 (10) Generalized aggressive periodontitis and Group 3(10) Localized aggressive periodontitis. Clinical parameters like plaque and gingival bleeding index, and clinical attachment loss were recorded. The GCF and blood samples were collected and subjected to ELISA to estimate the levels of MPO enzyme.

Statistical analysis: The data obtained were subjected to ANOVA and Student t test.

Results: The mean plaque and Bleeding index scores and CAL as well the MPO levels in serum and GCF were significantly higher in aggressive periodontitis than healthy controls (Plaque index-p <0.001, Gingival bleeding index p= 0.001,

CAL-p= 0.001, MPO Serum-p= 0.002, MPO GCF -p= 0.004). However, there was no significant difference in MPO levels between generalized and localized aggressive periodontitis (MPO Serum-p =0.15 & MPO GCF-p= 0.57).

Conclusion: The assessment of PMN MPO enzyme levels in GCF and Serum could help to determine the disease severity and risk for future disease progression.

Key words – Aggressive periodontitis, Gingival crevicular fluid, Myeloperoxidase, PMN s, Serum

Key message: Aggressive periodontitis is a rapidly progressive form of periodontitis for which neutrophil deficiency has been quoted as etiologic factor. However recent evidence suggests that chemotaxis defective neutrophils can be hyperactive and this is linked to elevated levels of myeloperoxidase enzyme which is a potent antimicrobial component of neutrophils and serve as a promising marker of periodontal inflammation.

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Introduction

Periodontitis is an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms or group of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession, or both.[1]

Aggressive periodontitis generally affects systemically healthy individuals less than 30 years old, although patients may be older.[1]

In 1971, Baer defined aggressive periodontitis as "a disease of the periodontium occurring in an otherwise healthy adolescent who is characterized by a rapid loss of alveolar bone about more than one tooth of the permanent dentition. The amount of destruction manifested is not commensurate with the amount of local irritants.[2]

In 1989, the world workshop in clinical periodontics[3] categorized this disease as localized juvenile periodontitis (LJP), a subset of the broad classification of early onset periodontitis (EOP).

In the AAP International workshop for classification of periodontal diseases 1999 the disease was renamed as 'Aggressive Periodontitis'. The disease usually affects young individuals at or after puberty and may be observed during the second or third decade of life (i.e, 10 to 30 years of age). The disease may be localized or generalized.[4]

Armitage GC in 1999 redefined Aggressive periodontitis as "a complex disease exhibiting microbial alteration and cellular dysfunction that differentiate the underlying molecular mechanisms of its pathogenesis from chronic periodontal disease".[4]

According to Lang and Bartold et al 1999, the primary features of aggressive periodontitis are non-contributory medical history, rapid attachment loss and bone loss, familial aggregation of cases, and the secondary features are amounts of microbial deposits inconsistent with the amount of periodontal destruction, elevated proportion of Aa, phagocytic abnormalities and hyper responsive macrophage phenotype.[5]

Aggressive periodontitis has been associated with various abnormalities in host cell function. Kantarci and Van Dyke et al conducted a study on neutrophil induced tissue injury in periodontal disease and concluded by saying "Localized aggressive periodontitis is a result of chronic hyperactive or primed state of neutrophils".[6]

Neutrophils have the potential to protect the tissue from pathogenic bacteria by various bactericidal mechanisms. These include intracellular and extracellular killing by enzymes and reactive oxygen metabolites. On the other hand, the same mechanism have the potential of inducing tissue damage and may contribute to local periodontal breakdown by direct destruction of connective tissue and bone and amplification of inflammatory process.[7]

The neutrophils contain granules which are important for their functioning. The granules are generated during differentiation of neutrophils and for storage. On the basis of function and enzyme content they are classified as azurophils, specific and small storage granules.[8]

The function of neutrophil granules is not just to provide hydrolytic substrate degradation but also to kill the ingested bacteria. Among the various azurophilic granulesmyeloperoxidase is a critical enzyme for the conversion of hydrogen peroxidase to hypochlorous acid. Together with hydrogen peroxide and a halide cofactor it forms the most effective microbicidal and cytotoxic mechanism of leukocytes (MPO system).[8]

The concept of neutrophil deficiency as a simple etiologic factor in localized aggressive periodontitis has recently been challenged by a considerable amount of evidence suggesting that the chemotaxis-defective neutrophils can be hyperactive in respect to some of their functions.[9]

In generalized aggressive periodontitis, amplified activity of PMN s is linked with elevated levels of MPO enzyme in GCF when compared to chronic periodontitis.[10]

Even though their function is to combat pathogenic bacteria, the activated neutrophils also promote cell destruction and MPO enzyme activity measurements have been used as predictors of periodontal disease severity. Thus, the present study was aimed at assessment of myeloperoxidase enzyme levels in GCF and serum in patients with generalized and localized aggressive periodontitisand the objectives were to compare the same with Periodontally healthy controls and also to correlate the MPO levels with the clinical parameters.

Subjects and Methods:

The study was conducted in the Department of Periodontics;The study period was between Nov 2011 and Dec 2012.

The aim of the present study was to assess the levels of myeloperoxidase enzyme in GCF and serum in patients with aggressive periodontitis. The study was approved by institutional human ethical committee of Medical and Dental College and Hospital.

The sample size for the present study was determined by systat 12 power analysis for serum and GCF MPO levels. Accordingly the study included 30 systemically healthy subjects in the age group between 17 and 35 years and were divided into three groups based on the inclusion and exclusion criteria as follows ;

Group 1 (n-10); Periodontally healthy subjects with fully functioning dentition as assessed by probing sulcus depth ≤ 3mm without clinical evidence of attachment loss.

Group 2 (n-10); Clinically diagnosed as generalized aggressive periodontitis characterized by generalized interproximal attachment loss affecting at least three permanent teeth other than first molars and incisors.[5]

Group 3 (n-10); Clinically diagnosed as localized aggressive periodontitis characterized by interproximal attachment loss in at least two permanent teeth, one of which is a first molar, and involving no more than two teeth other than first molars and incisors.[5]

Exclusion criteria

1. Patients with any systemic factors that could influence the outcome of the

Study.

2. History of hospitalization or intake of systemic medications in the last

Six months.

3. Smokers

4. History of periodontal therapy within preceding six months.

Clinical parameters

The following periodontal clinical parameters were recorded by a single examiner 'TN' for all patients.

1. Plaque index (silness & Loe 1964).[11]
2. Bleeding on probing (Ainamo & Bay).[12]
3. Probing pocket depth measurements.[13]
4. Clinical attachment level.[13]

Collection of blood samples: Subject consent was taken prior to collection of blood samples and 3ml of blood was collected from antecubital fossa by venepuncture using 20 gauge needles with a 5ml syringe. 2 ml of blood was transferred to test tubes and were allowed to clot at room temperature and after one hour, serum and plasma were extracted from the blood by centrifuging at 3000 rpm for five minutes. Extracted serum and plasma were immediately transferred to Eppendorf vials and stored at -20□ till the time of assay.

Collection of GCF: In order to eliminate the risk of contamination with saliva, maxillary sites were preferred for GCF sampling. The GCF collection was done in the morning hours between 9 and 11 am using WHATMAN's no 1 filter paper strips. The paper strips were placed into the sulcus for approximately 30 seconds and GCF samples were collected from multiple sites.[14]

The Serum pooled GCF samples were subjected to assessment of MPO enzyme using a specific ELISA kit® (AESKULISA MPO®) (Integrated sciences: Willoughby NSW Australia). The kit contained positive and negative controls, antihuman immunoglobulins conjugated to horseradish peroxidase, TMB substrate, IM hydrochloric acid and microtiterplate. The serum and GCF MPO enzyme levels were measured using ELISA technique which is an enzymatically amplified "two step" sandwich type immunoassay where the standard control and test samples were incubated in micro titration wells coated with MPO enzyme antibody.

Statistical methods

All clinical and Biochemical parameters were tabulated and subjected to statistical analysis by

1.Analysis of variance (ANOVA) test to compare the mean values of the three

Groups.

2. Student t test for comparing mean and standard deviation of biochemical

Parameters and Periodontal parameters between the groups.

Results:

Table 1 One way ANOVA test showing the mean and standard deviation of Plaque index scores-Group

wise

The Plaque scores were found to be significant between the groups (healthy 0.62±0.17) (GAgP 1.41±0.10) and (LAgP 1.36±0.19) with a p value <0.001. One way ANOVA test showed mean plaque score higher in Group 2 followed by Group 3 than the Group 1. (Table 1)

Table 2 One way ANOVA test showing the mean and standard deviation of Percentage of Gingival bleeding sites-Group wise

The percentage of bleeding sites were found to be 0.58 ±0.19 for Group1(healthy), 38.40±6.06 for group 2(GAgP) and 61.50±8.93 for group 3 (LAgP). One way ANOVA revealed that the mean percentage of bleeding sites were found to be different between the groups and higher in Group 3 compared to other groups which was statistically significant (P value 0.001).(Table 2)

Table 3 One way ANOVA test showing the mean and standard deviation of Probing pocket depth(mm)-Group wise

Table 3 shows the mean probing depth between the groups (Group1- 1.68 ±0.25, Group 2-6.01±0.31 and Group 3- 5.90±0.59) were significantly different. One way ANOVA test revealed that the mean probing depth was higher in group 2 compared with other groups which was statistically significant (P <0.001)

Table 4 One way ANOVA showing the mean and standard deviation of clinical Attachment loss (CAL) in mm-Group wise

In table 4 the mean and standard deviation of clinical attachment loss was found to be 0 for group 1 (healthy), 6.60±0.60 for group 2(GAgP) and 6.25±0.89 for group 3(LAgP). One way ANOVA test revealed that the mean CAL was found to be different between

Table 1

Groups	N	Minimum	Maximum	Mean	Standard dev	F Value	P Value
1 (Healthy)	10	0.40	1.00	0.62	0.17	83.58	<0.001(S)
2. (G AgP)	10	1.30	1.60	1.41	0.10		
3. (L AgP)	10	1.10	1.60	1.36	0.19		

Table 2

Groups	N	Minimum	Maximum	Mean	Standard dev	F Value	P Value
1 (Healthy)	10	0.00	0.78	0.58	0.19	36.61	0.001(s)
2. (G AgP)	10	51.00	64.00	38.40	6.06		
3. (L AgP)	10	50.00	75.00	61.50	8.93		

Table 3

Groups	N	Minimum	Maximum	Mean	Standard dev	F Value	P Value
1 (Healthy)	10	1.18	1.93	1.68	0.25	537.56	<0.001(s)
2. (G AgP)	10	5.60	6.40	6.01	0.31		
3. (L AgP)	10	5.10	6.80	5.90	0.59		

Table 4

Groups	N	Minimum	Maximum	Mean	Standard dev	F Value	P Value
1 (Healthy)	10	0.00	0.00	0.00		560.65	0.001(s)
2. (G AgP)	10	6.10	7.10	6.60	0.60		
3. (L AgP)	10	6.00	7.20	6.25	0.89		

the groups and was higher in group 2 compared to other groups which was statistically significant (P value 0.001).

Table 5 ANOVA test showing mean and standard deviation of levels of MPO enzyme in serum (µ/ml)-group wise

The mean and standard deviation of MPO enzyme levels in serum was found to be 7.66±2.23 in group 1(healthy), 11.16±2.24 in group 2(GAgP) and 9.65±1.62 in group 3(LAgP). One way ANOVA test revealed that the mean levels of MPO enzyme in serum was found to be different between the groups and was higher in group 2 compared with other groups which was statistically significant (P value 0.002).(Table 5)

Table 6 ANOVA test showing mean and standard deviation of levels of MPO enzyme in GCF (µ/ml)-group wise

The mean and standard deviation of MPO enzyme levels in GCF was found to be 14.17±2.33 for group 1 (healthy), 17.73±2.73 for group 2(GAgP) and 16.97±2.37 for group 3(LAgP). One way ANOVA test revealed that the mean levels of MPO enzyme in GCF was found to be different between the groups and were higher in Group 2 compared to other groups which was statistically significant (P value 0.004).(Table 6)

Table 7 Student t test to compare MPO enzyme levels in serum(µ/ml)

There was no significant difference in serum MPO levels between group 2 (11.16±2.24) and group 3(9.65±1.62) (P= 0.57).

Similarly there was no significant difference in GCF MPO levels also between group 2 (17.73±2.73) and group 3(16.97±2.37) (P=0.15) (Table 7)

Discussion:

Periodontal disease results from the interaction between the host defence mechanisms and microorganisms in the dental plaque biofilm.

Table 5

Groups	N	Minimum	Maximum	Mean	Standard dev	F Value	P Value
1 (Healthy)	10	1.5	10.1	7.66	2.23	7.82	0.002(s)
2. (G AgP)	10	8.5	15.0	11.16	2.24		
3. (L AgP)	10	8.0	12.4	9.65	1.62		

Table 6

Groups	N	Minimum	Maximum	Mean	Standard dev	F Value	P Value
1 (Healthy)	10	10	18	14.17	2.33	6.74	0.004(s)
2. (G AgP)	10	13	21	17.73	2.73		
3. (L AgP)	10	14	20	16.97	2.37		

Table 7

	Groups	N	Mean	Standard dev	P Value
MPO Serum	2. (G AgP)	10	11.16	2.24	0.15 (NS)
	3. (L AgP)	10	9.65	1.62	

	Groups	N	Mean	Standard dev	P Value
MPO GCF	2. (G AgP)	10	17.73	2.73	0.57(NS)
	3. (L AgP)	10	16.97	2.37	

Polymorphonuclear leukocytes represent the first line of these host defence mechanisms. Patients with altered numbers or altered function of circulating PMNs tend to have severe periodontal disease. Enhanced neutrophil responses play a critical role in the activation of immune system and causation of aggressive periodontitis.[15]

Locally PMNs release antimicrobial and inflammatory mediators, which act synergistically to protect and maintain tissues free of pathogens. Studies by Kantarci and Van Dyke proposed that neutrophils are not hypofunctional or deficient but hyperactivated or in primed state which leads to excess activity and release of toxic enzymes that are responsible for tissue destruction.[6]

Myeloperoxidase (MPO) enzyme is a constituent of azurophilic granules of PMNs that oxidizes chlorine ions to the potent bactericidal oxidant hypochlorous acid (HOCL). Due to its importance during inflammatory processes and for being an indicator of PMN presence in tissues, MPO enzyme has been widely used as an inflammatory marker of both acute and chronic conditions.

The aim of the present study was to assess the levels of MPO enzyme in GCF and serum in patients with aggressive periodontitis and compare the same between periodontal health and in generalized and localized aggressive periodontitis patients. In the present study, mean gingival bleeding scores were found to be higher in patients with aggressive periodontitis- LAgP(61.50±8.93), GAgP (38.40±6.06) when compared with periodontally healthy subjects (0.58±0.19) which was statistically significant (P<0.001). The bleeding on probing (BoP) is a widely used clinical sign as indicator of the periodontal condition and disease progression.[16] Our finding is in accordance with Rong Kun Liu et al[17] who in their study observed heavy PMN infiltration in both aggressive and chronic periodontitis with strong expression of IL 1β and TNF α showing more extensive inflammatory cell infiltration than in healthy controls.

In our study the probing pocket / sulcus depths were significantly higher in group 2 (6.01±0.31) than the group 3 (5.90±0.59) and group 1(1.68±0.25) with a significant P value (0.001). Similarly the CAL was significantly higher in Group 2 (6.60±0.60) than group 3(6.25±0.89) and group 1 (1.68±0.25) with a significant P value (0.001). The above results were similar to that of study by Nibali et al[18] where the probing pocket depth were greater in patients with aggressive periodontitis than healthy controls.

The control of potentially periodontopathic micro organisms by host neutrophils is crucial to periodontal health. Delivery of antimicrobial substances by neutrophils involves respiratory burst activity, phagocytosis, secretion or cytolysis / apoptosis. Neutrophils contain a number of antimicrobial components including calprotectin complex, lysozyme, defensins, myeloperoxidase enzyme and a NADPH oxidase system.[10]

Myeloperoxidase (MPO) enzyme is considered as a promising marker of periodontal inflammation which was confirmed by Cao CF and Smith QT.[19]

In the present study, mean MPO levels in serum were found to be significantly higher in patients with Generalized aggressive periodontitis (11.16±2.24) and localized aggressive periodontitis (9.65±1.62) than periodontally healthy controls (7.66±2.23) (P value 0.001). The above results were in accordance with a histological study by Sukhdeepsingh et al who observed that the intensity of MPO granule staining was greater (Grade 3) in aggressive periodontitis than the chronic periodontitis and healthy controls.[8]

Also in our study, mean MPO levels in GCF were found to be significantly higher in patients with Generalized aggressive periodontitis (17.73±2.73) and localized aggressive periodontitis (16.97±2.37) than in periodontally healthy controls (14.17±2,33) (P value 0.001). Our findings were similar to that of NerminYamaliket al.[20]

However, in the present study there was no significant difference in serum and GCF MPO levels (P=0.15) and (P=0.57) respectively between Generalized and localized aggressive periodontitis.

Most of the increased activity of MPO enzyme in periodontally diseased patients could be attributed to increase in number of neutrophils and MPO enzyme measurements have been used as predictors of periodontal disease severity.[21]

Few limitations of our study were not comparing patients with chronic periodontitis and non inclusion of microbial assay. However within the limitations of the study we conclude that the patients with aggressive periodontitis exhibited higher serum and GCF MPO levels than periodontally healthy controls suggesting the significance of primed hyperactive neutrophils in

the etiopathogenesis of aggressive periodontitis.

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Official News Letter of Society of Periodontists and Implantologists of Kerala

INAUGURATION OF 11TH ANNUAL CONFERENCE AND FAMILY GET TOGETHER ON 5-6 MAY 2018 AT LAKE PALACE HOTEL, KADINAMKULAM, TRIVANDRUM

PREZ' Message

.... Looking back...at the eve of SPIK Annual Conference, as the President, I had fully enjoyed every moment of this togetherness. We had started the year putting emphasis on 'spreading Periodontics in Kerala and Strengthening Periodontists inKerala. I believe we had done good justice to this goal; thanks to the collective efforts of Team SPIK '17-'18, timely advices and guidance of past office bearers, advisors and senior members. Let us keep this spirit in the years to come. Thank you dear members of the SPIK family for making my tenure a memorable one

Let us work together towards excellence.

Dr Anto Joseph Puthanangady

WORLD NO TOBACCO DAY AWARENESS PROGRAMME




The release of pamplheton No Tobacco Day programme was done by Dr NandakumarKat Kollam railway station in association with Azeezia Dental College on May 31st .

Dr SantoshSreedhar releasing the pamphlet on May 31stat Kannur railway station on behalf of No Tobacco Day Awareness Day Programme.

Dr Siby T Chennakarareleasing the pamphlet at Ernakulamrailway station on behalf of No Tobacco Day Awareness Day Programme.

Anti Tobacco awareness pamplhet prepared by SPIK

SCIENTIFIC PROGRAMME REPORT


At the outset I would like to thank the President, Secretary and the office bearers of Society of Periodontists&Implantologists of Kerala for giving an opportunity to host SPIK Midterm Conference 2017 at Hotel Avenue Regent , Kochi

The programme started with inaugural function at 09.30 AM with collaring of the President by theSecretary. SPIK followed by invocation.Dr.Baiju R M welcomed the gathering. Dr.Anto Joseph Puthangady, President. SPIKinaugurated the programmed by lighting the lamp.Dr.Biju Philip, Scientific Convenor detailed about the programme. Dr. Jayan Jacob delivered the vote of thanks.

A total of 60 delegates attended the programme. Scientific session presentation was on '' ''IMPLANTS IN AESTHETIC ZONE – Predictability and Pitfalls by Renowned Implantologist Dr T V Narayan. Hands on programme on sinus lift was also done in association with the session

Executive committee meeting of SPIK was conducted after the scientific programme.

REPORT OF 8TH MIDTERM CONFERENCE OF SPIK

8th midterm conference of SPIK was hosted by the Department Of Periodontics, Malabar Dental College & Research Centre, Edappal and was held at Casino Hotel , Thrissur on 20, 21 January 2018. The event was inaugurated by famous script writer of Malayalam Cinema, Sri JonePaul Puthusseryand presided by Dr Anto Joseph, President of SPIK. Dr Mahesh Narayanan, Organising Chairman welcomed the gathering and Dr Baiju RM, secretary elaborated on the activities of SPIK of this year. The scientific session was coordinated by Dr Jeethu John Jerry , Dr Muhammed Haris introduced the chief Guest and Registration desk was handled by Dr Neeta Joseph. Dr Shabeer Ahamed, organising Secretary proposed the vote of thanks.

3rd issue of JSPIK was also released on the occasion by Dr Plato Palathingal, the editor. Apart from Free paper and Poster presentations , a new category was introduced for the postgraduate students- Table display demonstration and a Clinical workshop on Bone Grafting . Dr Jose Paul, Dr Bindu R Nayar, Dr Harish Kumar, DrBaiju RM, Dr Jayan Jacob Mathews, Dr Biju Thomas and Dr Bini Raj were the key note Speakers and clinical workshop on Grafting was conducted by Dr ArunSadasivan. Cash prizes and certificates were awarded to the winners by Dr Vinod Kumar RB- Principal and Adv Shihab Mecheri- CEO of Malabar Dental College.

REPORT OF SPIK PERIODONTOLOGY SCHOLARSHIP EXAMINATION 2018 HELD AT AMRITA SCHOOL OF DENTISTRY ON 23RD OF MARCH 2018

The Society of Periodontists and Implantologists of Kerala in association with Amrita School of Dentistry, Kochi, organized and conducted the 5th SPIK Periodontology Scholarship Examination on 23rd of March 2018. Dr Biju Philip, Scientific Program convener- SPIK, DrJayachandran P, HOD –Department of Periodontics, Amrita school of Dentistry and Dr Angel Fenol, Professor-Department of Periodontics and Exam Convener, Amrita School of Dentistry along with DrBaiju RM, Secretary SPIK, Dr Tony Kurien, Exam Coordinator SPIK,DrJayan Jacob Mathew, DrAmbili R and DrSameera G Nath worked towards the success of the program. They were assisted expertly by other members of the faculty of Department of Periodontics, Amrita School of Dentistry.Dr Maya Rajan Peter and Dr Lakshmi P provided technical support during the conduct of the program. Nine participants from nine different Dental Colleges of the State of Kerala and Mahe, who had proven to be the best in the subject in their respective college, participated in the exam.

The exam commenced at 9:00 am. The panel of examiners comprising of Dr Angel Fenol, DrAmbili R, DrJayan Jacob Mathew and DrSameera G Nath had compiled a series of tasks for the students to test their competence.The examinees were given spotters to identify and explain followed by Multiple Choice Questions, Clinical case scenarios, Pedagogy and Viva.

The exam concluded at 2:00pm. The students and the accompanying persons were provided refreshments during the course of the day.

At the completion of the rigorous evaluation process four students emerged winners with the third prize being shared by two participants. Malavika R Nair of PMS College of Dental Sciences and Research Center secured the first place. Surumi Salam of Annoor Dental College, Muvattupuzha won the second place. Revathy of Government Dental College, Kottayam and Meeralakshmi S of Amrita school of Dentistry shared the third place. Results were announced and all the participants were given Certificate of Participation during the Valedictory function.

JSPIK REPORT2017-18

The First (Aug) issue of JSPIK was released during scientific programme on Aug 20th, 2017 by DR T V Narayan, Renowned Implantologistand Prof Jayan Jacob in presence of SPIK President Dr AntoJoseph , Secretary Dr Baiju RM and Editor Dr Plato Palathingalat Hotel Avenue Regent, M G Road Kochi.

The second (Dec)issue of JSPIK was released duringspikmidterm conference on Jan 21st, 2018 by Famous Cine Writer John Paul and SPIK President Dr Anto Joseph in presence of SPIKSecretary Dr Baiju RM and Editor Dr Plato Palathingalat Hotel Casino International, Thrissur.

The third (Mar)issue of JSPIK was released duringspikannual conference on May 6th, 2018 by Prof Justin Padamadan and Dr RezyCheru in presence of Dr Anto Joseph, Dr SanthoshSreedhar, Dr SeemaThampi and Dr Presanthilajanamat Lake View International Hotel, Trivandrum.

My Sincere & Special Thanks to Reviewers of JSPIK 2017-18 who supported me to release three issues of JSPIK on time in 2017-18.

Dr.Anniekitty George, Dr.Arunsadasivan, Dr. Baiju R M, Dr.BijuBalakrishnan, Dr. BinduNayar, Dr.Binitta Paul, Dr.Devisree, Dr.Divyasree, Dr.Jayan Jacob, Dr. Johnson

Dlima, Dr. Jose Paul, Dr. Mahesh Narayanan, Dr.Priya Jose, Dr. Sameer, Dr.Sameera G Nath, Dr.Senny Thomas, Dr.Sanjeev R, Dr.Santhosh VC, Dr.Seba Abraham, Dr.Shyamala Devi, Dr.SrikanthPuthalath, Dr.Vivek Narayan

I also thank Mr Sudheer for his tremendous support for Designing and printing the journal at its best design and standards

Editor – JSPIK

Dr Plato Palathingal